No. 123895 & No. 124002
(Consolidated)

IN THE SUPREME COURT OF ILLINOIS

JOHN JONES and DEBORAH JONES, Plaintiffs-Appellees, v. PNEUMO ABEX LLC and OWENS-ILLINOIS, INC., Defendants-Appellants.	)	On Petition for Leave to Appeal from the Appellate Court of Illinois, Fifth Judicial District, No. 5-16-0239. There on Appeal from the Circuit Court of the Second Judicial Circuit, Richland County, Illinois, No. 13-L-21, Hon. William C. Hudson, Judge Presiding.

BRIEF OF PLAINTIFFS-APPELLEES AS TO OWENS-ILLINOIS, INC.

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POINTS AND AUTHORITIES

NATURE OF THE CASE

Nine Illinois appellate court justices from three different districts have reviewed the evidence at issue in this case, and all nine have concluded the evidence is sufficient—using the clear and convincing evidence standard—for a jury to determine Owens-Illinois and Owens Corning conspired to suppress knowledge of the hazards of asbestos.

This case is not about vicarious liability. This case is not about holding Owens-Illinois (O-I) accountable for the conduct of other companies. This case is about holding Owens-Illinois accountable for its own conduct. As this Court has recently affirmed, "[T]he fundamental purposes of tort law are to hold wrongdoers liable for foreseeable consequences of their actions and to deter wrongful conduct." *Beaman v. Freesmeyer*, 2019 IL 122654, ¶ 45. That is what this case is about—holding Owens-Illinois accountable for the foreseeable consequences of facilitating, for decades, the manufacture, sale, and proliferation of a product it was told was toxic and hazardous to workers as early as 1943.

This is not the same case as *McClure*. The parties are not the same. The specific allegations are not the same. The evidence is not the same. The plaintiffs in *McClure* alleged a conspiracy between Owens-Illinois and Johns-Manville and Unarco. Here, Plaintiffs have alleged a conspiracy between Owens-Illinois and the company it created and owned, Owens Corning (OC). This is a big distinction, and, correctly, every Illinois court of review to look at the facts of this case has understood that distinction, despite O-I's best efforts to confuse the issue by pretending this case is the same as *McClure*.

McClure found there was insufficient evidence to establish Owens-Illinois conspired with Johns-Manville and Unarco (plaintiffs later discovered Owens-

Illinois misrepresented their contacts with Johns-Manville and Unarco to this Court in that case). But as to whether there is sufficient evidence Owens-Illinois and Owens Corning ever conspired with each other to suppress knowledge of the hazards of asbestos, the vote among Illinois appellate court justices is now 9-0 across three districts that there is. If this Court finds there is not clear and convincing evidence of a conspiracy between Owens-Illinois and Owens Corning to suppress knowledge of the hazards of asbestos, it will be the first Illinois court of review to do so. The appellate panels agreed that *whether* O-I and OC conspired to suppress knowledge of the hazards of asbestos was a question for the jury. The only dispute in the appellate court is *when* the conspiracy ended. That question, too, is a question for the jury, not for a court of review. *United States v. Steele*, 685 F.2d 793, 804 (3d Cir. 1982). The Fifth District's decision should be affirmed.

ISSUE PRESENTED FOR REVIEW

Is the question of whether and when Owens-Illinois conspired, or withdrew from the conspiracy, with Owens Corning a question of fact for the jury? Did Owens-Illinois withdraw from the civil conspiracy with Owens Corning in 1958 (the end of the distributorship agreement) by operation of law even though O-I (1) had massive financial entanglements with OC for decades after, (2) continued to falsely advertise Kaylo as "non-toxic," (3) continued to provide the packaging for Kaylo through the late 1960s, (4) continued to profit off of asbestos use in its own facilities well through the 1980s, and (5) filed its own $1,000,000,000 asbestos civil conspiracy lawsuit alleging similar facts as Plaintiffs here?

PLAINTIFFS' SUPPLEMENTAL APPENDIX

The documents and exhibits in this litigation are many and voluminous. The record in this case is not "identical" or "literally the exact same" as prior cases. In fact, each trial is unique and out of the hundreds and hundreds of potential exhibits, not every exhibit is used in every trial or in the same way in every trial.

It would be highly impractical, nigh impossible, for Plaintiffs to attach every potential trial exhibit to their summary judgment responses in these cases because it would total in the thousands of pages. Due to that reality, Plaintiffs provided excerpts of key exhibits at summary judgment in the trial court to cut down on the bulk of printed material, with the understanding the parties in this litigation are well-versed on the body of evidence. Through this process a small number of pages/exhibits were inadvertently omitted from Plaintiffs' written summary judgment opposition. This was not a problem at the trial court, or even observed, because the parties stipulated to the available body of evidence. C09292 (Defense counsel: "The evidence offered by the plaintiff here is the same as *Gillenwater*...the arguments are the same"); C09194 (The trial court's order: "Counsel for Plaintiffs and Defendant readily admitted at argument that the body of evidence in the instant matter is the same as that in *Gillenwater*."); C09197 (The trial court's order: "Counsel for Plaintiff and Defendant readily admitted at argument that the body of evidence in the instant matter is the same as that in *Rodarmel* . . . with one exception, the testimony of Dr. Frank.").

It is only now that Defendants are claiming there is no record evidence of key facts the parties stipulated to in the lower courts—for example, the fact Owens-Illinois manufactured and sold the packaging for Kaylo to Owens

Corning through the late 1960s, a fact Plaintiffs raised in their summary judgment opposition without any dispute from Owens-Illinois. C07262. Another example would be Abex's selective references to the United States Public Health Service exhibits. Plaintiffs have included those exhibits in their Supplemental Appendix, which Plaintiffs have attached to each brief in accordance with Supreme Court Rule 342.

Plaintiffs assume neither Abex nor Owens-Illinois will object to the Court considering this evidence since (1) both Defendants stipulated in the courts below the record in this case is the same as in other cases that includes those exhibits and (2) both Defendants claim the record in this case is, respectively, "virtually identical," "indistinguishable," "the same," and "static" to prior conspiracy cases and that, here, "all evidence is before the court," "no new facts can come to light at trial," and the trial court here was "faced with literally the identical factual record" as a previous conspiracy case. *Office Electronics, Inc. v. Adell*, 228 Ill. App. 3d 814, 819 (1992) (Court can consider attachments to brief if the parties so stipulate).

Certainly, Abex and Owens-Illinois would not make the claim that all the evidence is before this Court or that the trial court here was "faced with literally the identical factual record" as other courts if they knew certain relevant evidence was not in the summary judgment record but would be introduced at trial before any judgment *n.o.v.* motion, as that would destroy the entire premise of their argument that there is no difference between summary judgment and judgment *n.o.v.*

Regardless, the Court can take judicial notice of this small amount of material. It is true, and acknowledged by the parties, that these exhibits/excerpts

have all been included in the records of other conspiracy cases that were decided in the appellate court and eventually the subject of petitions for leave to appeal to this Court. This Court can take judicial notice of records in this Court or other courts. *American Federation of State, County, and Municipal Employees, Council 31 v. Illinois Labor Relations Board*, 2017 IL App (5th) 160229, ¶ 20.

All "A" citations in this brief are to Plaintiffs' Supplemental Appendix.

STATEMENT OF FACTS

A. Asbestos Conspiracy Jurisprudence As To Owens-Illinois

1. The Three Recent Cases Discussing The Clear And Convincing Evidence Of Owens Corning's Conspiracy With Owens-Illinois

No Illinois court of review has ever determined there is insufficient evidence under a clear and convincing standard for a jury to conclude Owens-Illinois (O-I) and Owens Corning (OC) conspired to suppress knowledge of the hazards of asbestos. On the contrary, three panels from three different districts of the Appellate Court (including this case) have reviewed the evidence of conspiracy as to Owens Corning and Owens-Illinois, and all have unanimously concluded the evidence is sufficient for a jury finding of conspiracy. *Gillenwater v. Honeywell Intern., Inc.*, 2013 IL App (4th) 120929, ¶ 96; *Johnson v. Pneumo Abex LLC*, 2018 IL App (3d) 160406-U, ¶ 55; *Jones v. Pneumo Abex LLC*, 2018 IL App (5th) 160239, ¶¶ 16-18.

The only difference between *Gillenwater*, *Johnson* (Plaintiffs will address *Johnson*, despite it being a Rule 23 order, because both Abex and O-I cited to it repeatedly in their briefs and petitions), and this case is that the *Gillenwater* panel and the majority in *Johnson* concluded the conspiracy ended in 1958. The dissent in *Johnson* and the unanimous opinion below both determined there is sufficient

evidence under the clear and convincing standard for a jury to conclude the conspiracy lasted beyond 1958 through the 1960s and into the 1970s. *Johnson*, ¶¶ 63-64 (Holdridge, J. dissent). The plaintiff in *Johnson* and Plaintiff here were both first exposed to Kaylo asbestos insulation in the 1960s. *Johnson*, ¶ 63 (Holdridge, J. dissent); C05730-31. In short, whereas *Gillenwater* and the *Johnson* majority held O-I "withdrew" from the conspiracy in 1958 as a matter of law, the *Johnson* dissent and the Fifth District below determined that whether and when O-I withdrew from the conspiracy is an issue of fact for the jury.

2. The *McClure* Decision Acknowledged But Did Not Consider The Relationship Between Owens-Illinois And Owens Corning, The Basis Of This Lawsuit.

Based on a limited record, this Court looked at whether Owens-Illinois and Owens Corning, respectively, conspired with Johns Manville and Unarco to suppress knowledge of the hazards of asbestos. *McClure v. Owens Corning Fiberglas Corp.*, 188 Ill. 2d 102 (1999). In that case, the Court explicitly stated it was *not* considering whether Owens-Illinois and Owens Corning conspired with each other:

> [P]laintiffs also presented evidence pertaining to the relationship between Owens Corning and Owens-Illinois. This evidence is only tangentially related to the essential question in this case, which is whether plaintiffs proved the existence of an agreement between defendants and Unarco or Johns-Manville. Proof of a relationship between *defendants themselves* does not establish the required agreement with *Unarco or Johns-Manville*.

188 Ill. 2d at 150 (emphasis in original). In other words, this Court acknowledged the relationship between Owens-Illinois and Owens Corning but concluded it had little, if any, bearing on that case. Any suggestion this case and the record in this case is identical to *McClure* is not true.

3. This Court's First Asbestos Conspiracy Decision, *Adcock*, Laid Out Plaintiffs' Evidentiary Burden.

The seminal Illinois asbestos conspiracy case is not *McClure*, it is *Adcock v. Brakegate, Ltd.*, 164 Ill. 2d 54 (1995). Owens Corning was the defendant in *Adcock*, and Owens-Illinois filed an amicus brief in support of OC. In that case, this Court explained a plaintiff's evidentiary burden in the context of the inherent difficulties of establishing evidence of a conspiracy: namely, that conspirators generally do not reduce their plans to detailed writings and advertise them. *Adcock*, 164 Ill. 2d at 66 ("[C]onspiracies are often purposefully shrouded in mystery."). With that in mind, this Court wrote that civil conspiracies, "by their very nature, do not permit the plaintiff to allege, with complete particularity, all the details of the conspiracy or the exact role of the defendants in the conspiracy. In fact, a conspiracy is rarely susceptible of direct proof; instead, it is established from circumstantial evidence and inferences drawn from the evidence, coupled with commonsense knowledge of the behavior of persons in similar circumstances." *Id.*

According to *Adcock*, a plaintiff does not need to produce evidence of an explicit agreement. *Id.* at 64. Rather, "A defendant who understands the general objectives of the conspiratorial scheme, accepts them, and agrees, **either explicitly or implicitly** to further those objectives . . . is liable as a conspirator." *Id.* (emphasis added).

B. How Owens-Illinois And Owens Corning Exposed Their Workers And Customers To A Lethal Product

Around the same time Johns-Manville, Abex, and their partners were scheming on how to manipulate the unfavorable report from the Saranac

Laboratory (see Statement of Facts in Plaintiffs' Abex brief), Defendant Owens-Illinois was itself receiving bad news from Saranac.

In 1943, Owens-Illinois began manufacturing and selling an asbestos thermal insulation product it branded "Kaylo." That same year, O-I engaged the services of Dr. Gardner at Saranac to test the product for dust release. Pl.'s Ex. (hereafter PX) 567, A222. Even before testing it, Dr. Gardner warned O-I in 1943 that because the company used asbestos and quartz (silica) in Kaylo, O-I had "all the ingredients for a first class hazard." PX 567.

Saranac provided O-I with an update on the Lab's progress in 1948, after Dr. Vorwald had taken over as a result of Dr. Gardner's death. Vorwald wrote to O-I that Saranac's testing to that point revealed, "In all animals sacrificed after more than 30 months of exposure to Kaylo dust unmistakable evidence of asbestosis has developed, showing that Kaylo on inhalation is capable of producing asbestosis and must be regarded as a potentially-hazardous material." PX 263, A102.

Dr. Vorwald, to his credit, assumed he was delivering news that would affect O-I's manufacturing of Kaylo. "I realize that our findings regarding Kaylo are less favorable that anticipated," he wrote. "However, since Kaylo is capable of producing asbestosis, it is better to discover it now in animals rather than later in industrial workers." PX 263, A104. Vorwald re-iterated this warning in a progress report sent to O-I in 1950: Kaylo dust "does produce the asbestotic type of reaction in the lungs and, therefore, we believe every precaution should be taken to minimize exposure of industrial employees." PX 576, A224.

Saranac finished its Kaylo study in 1952. Vorwald wrote to O-I that, once again, the tests showed "Kaylo dust is capable of producing a peribronchiolar

fibrosis typical of asbestosis." PX 278, A107. Vorwald indicated the Kaylo study results might be published. PX 278. But, now more familiar with the asbestos industry than he was in 1948, re-assured O-I that even if the study results were published, "reference will be made only to hydrous calcium silicate and not to 'Kaylo;' thus the interest of your Company will be safe-guarded." PX 278, A107. Still, Vorwald stated again—as he had in 1948 and in 1950—"the results of the study indicate every precaution should be taken to protect workers against inhaling the dust." PX 278, A107.

Throughout the nine years (1943-1952) Owens-Illinois received clear and persistent warnings from Saranac, it continued to ramp up production of Kaylo. In 1943, its sales were around $5,000. PX 705, A260. By 1948, sales had jumped to over $360,000. PX 705, A260. And by 1952, sales of its asbestos product totaled $3,335,841.65. PX 705, A260. That is a nearly ten-fold increase after receiving Vorwald's "less favorable" 1948 report.

'For Where Your Treasure Is, There Will Your Heart Be Also'

While O-I was building the Kaylo market in the late 40s and early 50s, the company O-I created in 1938, Owens Corning (OC), was also prospering. Upon the creation of OC in 1938, O-I owned 49.77% of OC common stock. C07357, A382. By 1956, O-I's shares of OC stock were worth over $84 million dollars. C07371, A384. In 1968, approximately when John Jones was exposed to Kaylo, O-I owned 25.1% of OC—an investment worth $143 million dollars accounting for almost 97% of O-I's total investment in public companies. C07376, A387. As late as 1978, O-I still owned over 750,000 shares of OC stock. C07380, A389. OC and O-I had many corporate officers and directors in common from OC's creation through the 1940s, until forced to stop that practice by the Department of Justice.

PX 710, A253-54. In 1995 arbitration proceedings between the two companies, counsel for OC said it was as difficult for OC to initiate the proceedings "as it would be for one member of a family to sue another member of a family." C07391 at 12:12-13, A306.

That familial relationship often came in handy for OC. In 1941, union insulators voiced concerns to OC over the skin irritation they were experiencing from OC's fiberglass insulation. PX 66, A19-20. After a year of reacting locally in 1941, OC's plan for 1942 was to "take the offensive" against the workers and create an "impressive file" of all the medical and scientific literature concerning the hazards of asbestos. PX 66, A20. OC believed the file would be "five or six hundred pages" and could be deployed as a "weapon-in-reserve" to show the insulators how good they had it working with fiberglass instead of asbestos. PX 66, A19. OC thought threatening workers with the risk of asbestos exposure could "promote dissension in the ranks that conceivably could bring about the over-throw of present Union leadership." PX 66, A20. O-I helped OC develop its "weapon-in-reserve" and fortified OC's arsenal with articles discussing the health hazards of asbestos to industrial workers. PX 265, A106.

As it happened, OC never deployed its "weapon-in-reserve." This restraint proved beneficial to both companies in 1953 when O-I and OC entered into a distributorship agreement for the asbestos insulation Kaylo. C07291-310. Under the agreement, O-I would continue to manufacture Kaylo, and OC would distribute it. *Gillenwater*, ¶ 55. The agreement lasted until 1958, when O-I sold its Kaylo division to OC. *Id.* at ¶ 57. Throughout the period of the agreement, neither company placed any warnings on Kaylo packaging. *Id.* In fact, both companies did just the opposite—they advertised Kaylo as "non-toxic." PX 33,

A15, C07343. Owens Corning's Medical Director, John Konzen, testified that the advertisement was false. C07335. Konzen admitted that at the time of the advertisement, 1956, Owens Corning had known for at least 13 years that asbestos was toxic. C07335. Owens Corning did not warn its workers of the hazards of asbestos until "1977 or '78." C07329 at 117:20-119:22.

After O-I sold its Kaylo division to OC in 1958, the companies remained close. O-I continued to provide packaging for Kaylo until the late 1960s. PX 696, A243. The companies continued to keep the packaging warning-free. PX 708, A296. As referenced above, O-I maintained a major investment in OC well into the 1970s. OC's profits and earnings were a frequent topic of conversation at O-I directors' meetings from the 1940s through the '70s. C07398-425.

O-I's Failure To Warn Post-Kaylo

In 2003, four years **after** this Court decided *McClure*, O-I's then CEO Joseph Lemieux gave a deposition in which he provided a detailed history of O-I's asbestos use in O-I plants and the company's indifference to workers. C07792-C07827. Lemieux's testimony revealed O-I's custom of suppressing knowledge of the hazards of asbestos from its workers and customers continued long after it sold the Kaylo division to OC. C07792-C07827.

O-I utilized asbestos products in its manufacturing processes throughout the 1960s and '70s. C07807. Yet, Lemieux, who was a plant-level managerial employee from 1957-1973, had no recollection of ever seeing signs or other information indicating asbestos posed a hazard. C07798-99. Lemeiux did not learn of the hazards of asbestos from his employer, O-I, until 1974 upon advancing within the company to a VP position at corporate headquarters. C07799.

When Lemieux arrived in his new position, O-I had no program to control asbestos dust in its plants. O-I's first iteration of an "asbestos-control program" was not initiated until the summer of 1974, when O-I finally started scrambling to bring its plants up to compliance with federal regulations. C07872-73. Lemieux, in his role as vice-president in charge of the glass container division, sent a Teletype to plant managers in his division stating there was a "serious problem concerning our asbestos control program." C07814, C07904. Although Lemieux sent a message to the plant managers informing them of the dust problem, he could not recall ever sending a similar message to the plants' blue collar workers. C07814. Evidence introduced in the *Gillenwater* trial showed O-I allowed its plants to skirt the company's "asbestos-control program" into the 1990s. C07820-21.

C. O-I And OC Knowingly Misrepresented Their Contacts With Johns-Manville And Unarco To This Court In *McClure*.

In the trial court, counsel for O-I stated during his oral argument, "[I]f [Plaintiffs' counsel] has something to say about what Owens-Illinois represented to the Illinois Supreme Court in *McClure*, of course, he ought to go to the Illinois Supreme Court and talk about that. It's been 17 years since *McClure* has been decided and he hasn't done that yet." R. Vol. 22 at 75:3-7, C09307.

O-I and OC represented to this Court in *McClure* that their contacts with Johns-Manville and Unarco were "scant and benign," that OC had "nothing to do with Unarco, the Bloomington plant or the plaintiffs during the 1950s or 1960s," and that O-I had "nothing to do with Unarco, its plant, [or] the asbestos used there." C07460-61, 64; C07541.

Documents discovered by plaintiffs post-*McClure* prove these representations were untrue. C07265. According to O-I itself, in the 1950s, OC sold Unarco's asbestos insulation product, Unibestos. C07652, A317. Also according to O-I, beginning in 1958 OC "entered into a rebranding agreements with other manufacturers pursuant to which OCF bought the asbestos-containing products of others (e.g., Johns-Manville) and resold them under the OCF Kaylo label." C07653 (A318), C07788-89. The rebranded Johns-Manville products had an even higher asbestos content than Kaylo. PX 708 (A285), C07765. John McCallister, a former O-I employee, testified in 1983 in front of O-I's lawyers that O-I received the asbestos it used in Kaylo from both Johns-Manville and Unarco. C07741. None of this information was before this Court in *McClure* when it held O-I and OC had at most "isolated" contacts with Unarco and Johns-Manville. 188 Ill. 2d at 151.

D. O-I's $1,000,000,000 Asbestos Conspiracy Lawsuit Alleges Similar Facts As Plaintiffs' Complaint.

O-I agrees with Plaintiffs that Johns-Manville participated in an asbestos conspiracy. In fact, in 1999—the same year this Court decided *McClure* (in which O-I told this Court there was no asbestos conspiracy involving Johns-Manville)—O-I filed a complaint in the United States District Court for the Eastern District of Texas seeking to recover more than $1,000,000,000 in damages. C07605-39. The charge? O-I complained it was the victim of an asbestos conspiracy by Johns-Manville and others. C07605-39.

O-I's allegations are very similar to Plaintiffs'. O-I alleged the conspirators had "formed an international asbestos cartel," whose purpose was to "suppress[] information about the health risks posed by exposure to asbestos, and

maximiz[e] demand for and profits from the sale of asbestos fiber." C07615. The conspirators, according to O-I, "knew that, if their customers (the manufacturers of asbestos-containing products) learned that users of finished insulation products were at risk of contracting asbestos related disease, demand for asbestos fiber would decrease or disappear entirely." C07615.

According to O-I, a central part of the conspiracy included the conspirators "work[ing] together actively to suppress publication of scientific research concerning the potential risks posed by exposure to asbestos dust." C07618. The conspirators "monitored and edited scientific research results prior to publication to eliminate references to unfavorable results, withheld information about asbestos-related illnesses from their own employees and the public, and attempted to suppress publication of scientific research." C07618. This would be the same conduct Plaintiffs allege Abex of participating in with Johns-Manville in this case. Part of the *Gillenwater* record is the testimony of O-I's Vice President and former general counsel, Phil McWeeny, who said he believed Johns-Manville was in an asbestos conspiracy.

ARGUMENT

I. SUMMARY OF ARGUMENT

At each stage of this case, Owens-Illinois has advanced a different principal argument, each one flimsier than the last. O-I urged the trial court to simply adopt all the findings and conclusions of *Gillenwater*, which the trial court did. C09286-96; C09305-09; C09193-95. Then, in the Appellate Court, O-I argued Plaintiffs had failed to raise a fact question on causation under *Thacker v. UNR Industries, Inc.*, 151 Ill. 3d 343 (1992). Def.'s App. Br. 10-14. O-I admitted it never raised that argument in the trial court.

Now, in this Court, O-I has scrapped its *Thacker* argument. In fact, O-I does not even cite *Thacker* a single time in its brief. Instead, O-I advances another brand new argument, which is that this case is "for all practical purposes" the same as *McClure*. O-I Br. 26. Whereas O-I claimed to the trial court this case was the same as *Gillenwater*, it is now claiming, actually, forget *Gillenwater*, this case is the same as *McClure*. Nothing could be further from the truth or more readily disproved.

This case is not the same as *McClure*. This fact is so easily established it is a wonder O-I tries so desperately to convince the Court otherwise. The issues in *McClure* were that Owens-Illinois and Owens Corning each conspired with Johns-Manville and Unarco (the sources of the *McClure* plaintiffs' alleged asbestos exposure). In this case (and in *Gillenwater*), the issue as to Owens-Illinois and Owens Corning is if they conspired *with each other*. Owens-Illinois understands this distinction. Under the headline "Plaintiffs' Claimed Conspiracy," O-I explained to the Fifth District, "As to Abex, Plaintiffs point to a conspiracy with 'others, including Johns Manville,' **but as to OI, Plaintiffs now urge a conspiracy only with OCF.**" O-I App. Br. 4 (emphasis added).

This Court understood the distinction when it decided *McClure*. In that case, the Court explicitly stated it was not considering whether Owens-Illinois and Owens Corning conspired with each other:

> [P]laintiffs also presented evidence pertaining to the relationship between Owens Corning and Owens-Illinois. This evidence is only tangentially related to the essential question in this case, which is whether plaintiffs proved the existence of an agreement between defendants and Unarco or Johns-Manville. Proof of a relationship between *defendants themselves* does not establish the required agreement with *Unarco or Johns-Manville*.

188 Ill. 2d at 150 (emphasis in original). It is bewildering why O-I would make its principal argument to this Court that the facts here are "virtually identical" to *McClure*; that Plaintiffs "rely on the same evidence as *McClure*"; and that (in all caps in the original), "This Court has already conclusively decided, on indistinguishable evidence, that no reasonable jury could find that Owens-Illinois conspired." O-I Br. 5, 26-27, 44. In the trial court, counsel for O-I said, "The record in *McClure* was very similar, **but not identical**, to the record here before your Honor." C09287 (emphasis added). Now O-I claims the record here is "virtually identical" to *McClure*. O-I Br. 5, 44.

Nothing can explain O-I's barrage of misinformation at this late stage other than desperation. O-I does not want this Court to learn why nine justices from three districts of the appellate court have unanimously found a jury question exists on whether O-I and OC conspired as Plaintiffs claim. None of those justices found that this case is "for all practical purposes" the same as *McClure*. O-I does not want this Court to consider the fact it filed a $1,000,000,000 asbestos conspiracy lawsuit alleging Johns-Manville was a member of an asbestos conspiracy *in the same year it argued to this Court there was no conspiracy with Johns-Manville.*

O-I does not want this Court to consider the fact O-I and OC misrepresented their contacts with Johns-Manville and Unarco in *McClure*, telling this Court they had "scant and benign" contacts with those companies and that they had "nothing to do" with Unarco or its Bloomington plant, when in fact O-I and OC purchased asbestos fiber from Johns-Manville, purchased fiber from Unarco's Bloomington plant, rebranded Johns-Manville's and Unarco's asbestos insulation under the Kaylo name, and directly sold Unarco's asbestos

insulation, Unibestos. C07265, 07652, 07653, 07741. This is not "scant and benign" contact, nor is it merely "parallel conduct." *Dukes*, 386 Ill. App. 3d at 440 ("Plaintiff presented evidence Unarco and J-M had been suppliers to O-I throughout the 1940s and 1950s. This evidence was not in *McClure*. It is contrary to what O-I represented to the supreme court in *McClure* and is at odds with that court's determination of isolated contacts between those companies.").

O-I's misinformation campaign has yet to win a single convert. Although it "emerged with a judgment" in *Gillenwater*, it did so on a technicality only after the court acknowledged O-I and OC were in a civil conspiracy by clear and convincing evidence. *Gillenwater*, ¶ 96. O-I tries to claim this holding is "plainly *dicta*." O-I Br. 37. Whether it is *dicta* or not is irrelevant—the Fourth District's opinion is not binding on this Court anyway. What is important is the court reviewed the evidence and concluded the evidence was clear and convincing that Owens-Illinois and Owens Corning conspired to suppress knowledge of the hazards of asbestos just as the plaintiffs claimed.

O-I's support further eroded in *Johnson*, the Rule 23 order O-I repeatedly cites. In that case, only two justices (deferring to *Gillenwater*) determined O-I withdrew from the conspiracy in 1958. The third, in dissent, wrote that plaintiffs evidence "could support a finding of a continuing conspiracy after 1958 by clear and convincing evidence." ¶ 63. In this case, O-I could not convince any of the three justices that it would be unreasonable for a jury to find by clear and convincing evidence it conspired with Owens Corning to suppress or misrepresent the hazards of asbestos beyond 1958.

Owens-Illinois makes a passionate appeal to *stare decisis* to open its brief. But since O-I's call to action in defense of *stare decisis* is based on the false

premise that this case is the same as *McClure*, it is hard to see how *stare decisis* helps O-I or Abex here at all. *McClure* has no direct application to these facts and to these defendants. *McClure* is largely silent as to O-I and OC's relationship and does not discuss at all whether a defendant's purported withdrawal from a civil conspiracy is an issue of fact for jurors. Cases that do apply here—*Burgess* and *Dukes*—certainly do not offer Abex or O-I any relief, especially the section of *Dukes* discussing how O-I made false representations to this Court in *McClure*. 386 Ill. App. 3d at 440.

Finally, like Abex, O-I tries to eliminate the distinction between facts and evidence in arguing the Fifth District below erred by not weighing the evidence at summary judgment to resolve questions of fact in O-I's favor. The Fifth District was correct not to do so. Owens-Illinois faults the Fifth District for "not cit[ing] or otherwise discuss[ing] this Court's ruling in *Fooden*." The Fifth District can hardly be blamed for not citing *Fooden* because (1) *Fooden* only applies in cases where the facts are not in dispute and (2) O-I itself only cited *Fooden* once in passing—in a string cite—in its 33-page brief to the Fifth District, a brief that focused mostly on whether Plaintiffs' evidence met the requirements of *Thacker* (an argument O-I has now abandoned). O-I's App. Br. 24. The Fifth District cannot be blamed for not addressing an argument O-I did not make.

II. THIS CASE DOES NOT HAVE A 'PREORDAINED OUTCOME,' NOR IS IT 'ON THE ROAD TO *JNOV*' BECAUSE COURTS SHOULD NOT WEIGH THE EVIDENCE AT SUMMARY JUDGMENT OR AT JUDGMENT *N.O.V.*

Abex and Owens-Illinois both argue that because the summary judgment and judgment *n.o.v.* standard are the same they should receive summary judgment. Plaintiffs agree the standards are essentially the same—insofar that at

each stage the courts are not supposed to weigh the evidence and usurp the role of the jury. Regardless of a case's procedural posture, the jury is the fact-finder, not the court. *Gatlin v. Ruder*, 137 Ill. 2d 284, 294 (1990) ("A court cannot decide factual disputes as a matter of law.").

The difference between this case and the cases Defendants cite now, *Fooden v. Board of Governors* and *Cohen v. Chicago Park Dist.* (Abex did not cite *Fooden* below and O-I cited it only once in the middle of a string cite), is in those cases the material facts were *not* in dispute. In this case, the material facts *are* in dispute. That distinction is the dispositive element missing from Defendants' argument.

A. Courts Are Not The Triers Of Fact At Any Stage.

It is true the summary judgment standard and judgment *n.o.v.* standard are the same in key respects. Summary judgment is "a drastic means of disposing of litigation and should be allowed only when the right of the moving party is clear and free from doubt." *Beaman v. Freesmeyer*, 2019 IL 122654, ¶ 22. In instances where "reasonable minds could draw divergent inferences from the undisputed material facts or where there is a dispute as to a material fact, summary judgment should be denied and the issue decided by the trier of fact." *Id.* In ruling on a motion for summary judgment, "the court must construe the pleadings, depositions, admissions, and affidavits strictly against the movant and liberally in favor of the opponent." *Id.*

Likewise, "judgment *n.o.v.* is properly entered in those limited cases where all of the evidence, when viewed in its aspect most favorable to the opponent, so overwhelmingly favors movant that no contrary verdict based on that evidence could ever stand." *Maple v. Gustafson*, 151 Ill. 2d 445, 453 (1992). In

ruling on a motion for judgment *n.o.v.*, "a court does not weigh the evidence, nor is it concerned with the credibility of the witnesses; rather it may only consider the evidence, and any inferences therefrom, in the light most favorable to the party resisting the motion." *Id.* It is a basic principle of appellate review that "the appellate court should not usurp the function of the jury and substitute its judgment on questions of fact fairly submitted, tried, and determined from the evidence which did not greatly preponderate either way." *Id.* at 452.

Judgment *n.o.v.* and summary judgment are both rare forms of relief granted in the most narrow and compelling circumstances. A court has "no right to enter judgment *n.o.v.*" when the evidence raises a factual dispute. *Id.* at 454. Summary judgment is "an extraordinary remedy" that "should be denied" where there is a dispute as to a material fact. *Forsythe v. Clark USA, Inc.*, 224 Ill. 2d 274, 280, 298 (2007).

B. The *Evidence* In This Case Is 'Static'; The *Facts* Are In Dispute.

Courts do not act as the fact-finder when the material facts are in dispute—like they are in this case—either before the trial at summary judgment or after the trial at judgment *n.o.v. Gatlin*, 137 Ill. 2d at 294. In an attempt to side-step this basic tenet of Illinois law, Abex and O-I blur the distinction between *evidence* and *facts*. Abex claims the "primary reason for denying a summary judgment [is] the concern that it will prematurely deprive the plaintiff of an opportunity to develop the facts fully at trial." Abex. Br. 26. Abex then claims there is no reason to deny summary judgment here because "[t]he full facts surrounding the conspiracy claim are not only known, they are static; they occurred 70 years ago and are repeated in each trial of this conspiracy claim." Abex Br. 26. O-I tries the same thing. O-I writes, "[W]here the *factual* record is

complete, the standards for summary judgment and *JNOV* are the same." O-I Br. 40 (emphasis added).

Abex and O-I's argument is deceptive because it conflates facts and evidence. The *evidence*, which is open to interpretation, is "static" or "complete" (as it largely would be in any case at the close of discovery). The *facts* are determined by the fact-finder after hearing the evidence. The court is not the fact-finder. The jury is the fact-finder. And when the facts are in dispute, summary judgment is improper. *Beaman*, 2019 IL 122654 at ¶ 22 ("[W]here there is a dispute as to a material fact, summary judgment should be denied...."). In the words of this Court, "The purpose of summary judgment is not to try a question of fact, but to determine if one exists." *Robidoux v. Oliphant*, 201 Ill. 2d 324, 335 (2002). The Fifth District was correct below when it held weighing the evidence "results in our appellate court, in effect, trying the case." *Jones*, ¶ 23. In that respect, this case is no different than any other case at summary judgment.

C. Summary Judgment Should Be Denied When The Facts Are In Dispute.

In addition to blurring the distinction between facts and evidence, Abex and O-I also fail to recognize the distinction between summary judgment in cases where the facts are in dispute and in cases where the facts are not in dispute. No two cases illustrate this crucial distinction better than the two cases relied upon by Abex and Owens-Illinois, *Fooden v. Board of Governors of State Colleges and Universities*, 48 Ill. 2d 580 (1971) and *Cohen v. Chicago Park Dist.*, 2017 IL 121800. In both of those cases, unlike here, the material facts were not in dispute. As such, neither case is applicable here.

In *Fooden*, the defendant moved for summary judgment and provided an *uncontested* affidavit in support. 48 Ill. 2d at 587. This Court noted that in instances where a summary judgment motion is supported by affidavit "**and** where such facts are **uncontradicted** by counteraffidavit, they must be taken as true, notwithstanding contrary averments in the adverse party's **pleadings** which merely purport to establish bona fide issues of fact." *Id.* (emphasis added). Plaintiffs here are not resting on their pleadings, like the plaintiffs in *Fooden* did. *Id.* at 588. Plaintiffs here are not admitting to Defendants' versions of the facts, like the plaintiffs in *Fooden* did. *Id.* Plaintiffs here are not claiming that their complaint alone raises issues of material fact, like the plaintiffs in *Fooden* did. *Id.* Here, Plaintiffs vigorously dispute Defendants' versions of the facts, meaning *Fooden* does not apply.

Cohen v. Chicago Park Dist. does not apply here for the same reason. In that case, too, the material facts were undisputed. There, it was undisputed the plaintiff fell off his bike after hitting a small crack in a city pathway. 2017 IL 121800 at ¶ 6. It was undisputed that a week later, while riding his bike again, the plaintiff observed the crack had been repaired. *Id.* at ¶ 7. It was undisputed that upon first learning of the crack, defendant's employee inspected the crack and put it on the repair list and collected bids. *Id.* at ¶ 11-12. It was undisputed repair of the crack began less than a month later and was completed less than two months later. *Id.* at ¶ 12. In the words of this Court, it was "undisputed that defendant in this case took corrective action" almost immediately after first learning of the crack. *Id.* at ¶ 32. Although the plaintiff "emphasize[d]" and argued that the defendant "could have done more," the plaintiff did not dispute any of the material facts. *Id.* at ¶ 33. This Court held that, under those undisputed

facts, the defendant's activities did not rise to "willful and wanton" conduct. *Id.* at ¶ 34. This case is not like *Cohen* because the facts here are in dispute.

A third case cited by Abex, *Baldwin v. Twin Rivers Club*, 262 Ill. App. 3d 516 (1994) is also inapposite. In *Baldwin*, a case involving an injured worker who fell off a wooden plank, the Third District affirmed summary judgment awarded to defendant. In doing so, the court noted, "In the instant case, **there is no genuine factual dispute** as to the type of device in question—a plank spanning an area between wooden beams or rafters." *Id.* at 521 (emphasis added). Because there was no factual dispute, the court could rule, as a matter of law, whether the plank fit within the meaning of the Structural Work Act. *Id.* Defendants have not cited one case where a court stepped in and usurped the function of the jury to determine issues of triable fact at summary judgment.

1. The Facts As To Abex Are In Dispute.

The facts in this case are in dispute. Abex claims their participation in the Saranac agreement, including the decision with other asbestos companies to suppress information about the hazards of asbestos from the Saranac report, is not evidence that Abex agreed with other asbestos companies (either explicitly or implicitly) to suppress information about the hazards of asbestos. Plaintiffs claim it is. Abex claims it "did not perceive" asbestos as a "problem in brake manufacturing"; Plaintiffs claim Abex had intimate knowledge of the hazards, which is why it only allowed testing at their plants if the testing agencies promised to not inform "the employees, their lawyer or physician" of the results. PX 181, A64. Abex claims Dr. Lynch's statement that Dr. Gardner's research, including the cancer findings, was "valuable and publishable as it stands" (AX 711, A13) "expresses no opinion" about the "scientific validity" of Dr. Gardner's

work. Plaintiffs argue that if a contemporary expert like Dr. Lynch says another doctor's work is "valuable and publishable," that is expressing an opinion as to its "scientific validity." Abex claims it had an "effective dust control program." Abex. Br. 22. In response, Plaintiffs point to the many air sampling studies showing Abex plants with serious dust problems. PX 208, A82-97. These are just a few of the many disputed material facts in this case. *See Jones*, ¶ 14 ("[W]e find the record is replete with genuine issues of material fact....").

These are not just claims Plaintiffs make in their complaint or through argument by counsel—in the words of Justice Cook, "We have meetings, conferences, telephone calls, and cooperation in this case." *Menssen*, 2012 IL App (4th) at ¶ 62 (Cook, J. dissenting). We also have correspondence, company newsletters, and test results that all dispute Abex's version of events. In other words, Plaintiffs have *evidence*, both direct and circumstantial, to support their claim that Abex agreed to suppress information related to the hazards of asbestos and committed overt acts in furtherance of that agreement. Under these circumstances, summary judgment is improper, and judgment for Abex is not "preordained." The Court should affirm the Fifth District.

2. The Facts As To Owens-Illinois Are In Dispute.

Owens-Illinois spends a considerable amount of space in its brief disputing the evidence in this case. O-I Br. 5-16, 26-40. After doing so, O-I then claims that because it has told its version of the story, this Court can adopt O-I's spin on the events and grant summary judgment. That is not the law, and, furthermore, O-I's version of events is tethered very loosely to reality.

O-I claims its 1953 agreement with OC to mass produce and distribute Kaylo is not evidence of conspiracy because it is just a "bulk sales transaction,"

and if their agreement raises an inference of conspiracy "then *every* bulk sales transaction or distribution agreement would permit the same inference." O-I Br. 38-39 (emphasis in original). O-I ignores the fact that not *every* "bulk sales transaction" is for a product the parties to the transaction know can kill its users. Plaintiffs contend it is very hard to team up with another company to mass produce and sell a product both companies know is toxic without a mutual understanding among the two companies to conceal that fact. In their arbitration dispute, both O-I and OC made very compelling cases that each company knew well before 1953 about the hazardous properties of asbestos—yet they then still entered into an agreement to mass produce and distribute the product, in part by advertising it as "non-toxic." PX 696, PX 708, PX 33. Every court to consider the distribution agreement has found it is clear and convincing evidence of a conspiracy between O-I and OC. *E.g.*, *Gillenwater*, ¶ 96.

O-I claims no "inference of a conspiracy could rationally be made after 1958, when O-I sold its Kaylo division to OCF." O-I Br. 32. Plaintiffs claim O-I's continued massive investment in OC after 1958, O-I's preoccupation with OC's profits, the fact O-I manufactured and sold the packaging for Kaylo through the 1960s, and the fact O-I continued to use asbestos in its own operations for almost 40 years after 1958, all raise the inference O-I and OC's mutual understanding or implicit agreement to suppress information on the hazards of asbestos continued beyond 1958. O-I did not stop using asbestos and asbestos products (which it used in glass production) in 1958, so it did not stop having an interest in suppressing knowledge of the hazards of asbestos after 1958.

Owens-Illinois says advertising Kaylo as "non-toxic" many years after receiving word from Saranac that Kaylo was a "first class hazard" that causes

disease in industrial workers merely "comports with the usage of the term . . . in the published scientific literature." O-I Br. 12. O-I says interpreting those acts any other way means the conspiracy "extends to the highest reaches of government and the medical profession." O-I Br. 28. But the ads were meant to reach the end users, people like John Jones, not to "the highest reaches of government and the medical profession." For blue collar workers without Ph.D.'s, like John Jones, to read something that says "non-toxic" means the product will not harm or kill him. Indeed, in arbitration against OC, after the two companies finally had a falling out, O-I blamed OC for placing warnings on Kaylo that "somebody with a Ph.D would have difficulty understanding." PX 708, A288, A296 (stating warnings "would have required a graduate degree to understand").

Even Owens Corning's Medical Director, John Konzen, testified the "non-toxic" advertisement was false and Owens Corning knew at the time it was false. C07335-39. Konzen admitted that at the time of the advertisement, 1956, Owens Corning had known for at least 13 years that asbestos was toxic in the common understanding of the term. C07335. Did Owens Corning know something in the 1950s that Owens-Illinois did not? No, recall that in 1941 Owens-Illinois provided articles on the hazards of asbestos to Owens Corning so the latter could build its "weapon-in-reserve." PX 66, A19-21.

As for those articles, O-I claims it is just "sharing copies of published information." But the point is not the humdrum activity of sharing an article. The point is that sharing those articles in 1941 for the express purpose of building a "weapon-in-reserve" is evidence of at least four important facts: (1) O-I and OC *both* knew about the hazards of asbestos, and *both* knew users did not (that is the "in-reserve" part); (2) O-I and OC *both* knew asbestos was specifically dangerous

to workers (that's the "weapon" part); (3) O-I and OC *both knew the other company knew* asbestos was hazardous to workers; and (4) O-I and OC *both* knew those things at least 12 years before they joined forces to mass produce and distribute Kaylo.

The probative value of sharing the 1941 "weapon-in-reserve" articles is not just the sharing part—it is what it says about what O-I and OC knew, when they knew it, when and what they knew the other company knew, and what they decided to do with that knowledge 12 years later when they *entered into an agreement* to mass produce and distribute their asbestos product, Kaylo. What is says, of course, is that they *both* decided, together, to say nothing about the hazards of asbestos. The 1941 "weapon-in-reserve" articles make it impossible for O-I to claim it was acting unilaterally or "parallel" to Owens Corning in 1953 when they teamed up to sell Kaylo without informing anyone of the hazards. They both knew the other knew how dangerous the product was and both said nothing. At a minimum, that is what is called an "implicit agreement" or "mutual understanding" under *Adcock*, which makes Owens-Illinois potentially liable as a conspirator. 164 Ill. 2d at 66.

Owens-Illinois argues the 1953 agreement "nowhere provides for agreed limits on what can or cannot be said about the health effects of the product." O-I Br. 28. O-I's suggestion that the conspiratorial agreement must be reduced to writing is a non-starter. It is "not necessary that defendant admit the conspiracy; evidence of an implicit agreement is enough." *Menssen v. Pneumo Abex*, 2012 IL App (4th) 100904, ¶ 61 (Cook, J. dissenting); *cf. Adcock*, 164 Ill. 2d at 66; *McClure*, 188 Ill. 2d at 134 ("A defendant who understands the general objectives of the

conspiratorial scheme, accepts them, and agrees, either explicitly or implicitly to do its part to further those objectives . . . is liable as a conspirator.").

O-I invented Kaylo. Then O-I was told Kaylo had "all the ingredients for a first class hazard" to workers. PX 567, A222. Upon receiving that news, O-I ramped up the manufacture and sale of Kaylo. PX 705, A260. Then in 1953 O-I teamed up with Owens Corning to mass distribute Kaylo (marketing it as "non-toxic"). C07291-310; PX 33; PX 696, A243. O-I then provided OC with the packaging for Kaylo through the late 1960s. PX 696, A243. Through all of this, O-I stayed silent about the known hazards of the product as Owens Corning continued to sell it throughout the country and Owens-Illinois continued to protect its massive investment in OC. Owens-Illinois is just as responsible for Kaylo as Owens Corning. *Adcock*, 164 Ill. 2d at 64 (a defendant "may be held liable for any tortious act committed in furtherance of the conspiracy").

As to O-I's investment in OC, O-I claims the Fifth District held that "just buying stock in another company satisfies the clear and convincing evidence standard." O-I Br. 31. This statement is farcical. That is not what the Fifth District held. This is another example of O-I stripping context from the evidence. Buying stock in another company may not always be clear and convincing evidence of a conspiracy. But O-I did not just randomly buy stock in OC at some point in the 1960s. O-I created OC in 1938. In 1948, O-I had to be told by the Department of Justice to disentangle itself from OC's board of directors, but then nonetheless maintained an investment in OC that accounted for its biggest domestic investment through the 1970s. C07371, C07380; A381-89 (O-I annual reports). In 1968, approximately when John Jones was exposed to Kaylo, O-I owned 25.1% of OC—an investment worth $143 million dollars accounting for almost 97% of

Owens-Illinois's total investment in public companies. C07376, A387. Maintaining an investment of that magnitude in a company O-I teamed up with to mass distribute O-I's lethal product raises a question of fact as to whether O-I really "withdrew" from the conspiracy in 1958. *Steele*, 685 F.2d at 804. That is what the Fifth District held, not that putting some OC stock in your company retirement plan is evidence of a conspiracy.

Finally, O-I claims the fact it filed a $1,000,000,000 asbestos conspiracy lawsuit does not mean O-I itself was in a conspiracy. O-I Br. 34. It is not surprising that O-I chose to not sue itself in its $1,000,000,000 asbestos conspiracy lawsuit. What is surprising is that it would allege in that case Johns-Manville was a member of an asbestos conspiracy at the very same time it was arguing to this Court there was no asbestos conspiracy involving Johns-Manville, even though O-I's lawsuit makes similar allegations and references some of the same evidence as plaintiffs did in *McClure*.

D. The Fourth District's Recent History Of Frequently Awarding Judgment *n.o.v.* In Asbestos Cases.

One thing all the parties to this appeal agree on is that judgment *n.o.v.* is supposed to be rare. Owens-Illinois told the court below, "Illinois appellate courts, including the Supreme Court, almost never reverse jury verdicts." O-I App. Br. 15. Respectfully, the Fourth District has reversed, in decisions often creating new law, numerous verdicts on behalf of asbestos victims in the last decade, and it has done so frequently by granting the defendant judgment *n.o.v.* One justice has indicated the Fourth District is using a "sufficiency-of-the-evidence" standard to do so. *Menssen*, 2012 IL App (4th), ¶ 57 (Turner, J. specially concurring). A list follows:

- *Dukes v. Pneumo Abex Corp.*, 386 Ill. App. 3d 345 (2008) (finding certain evidence, including memo from defendant's employee stating its workers "have had a good life" from asbestos and so "might as well die from it" as unfairly prejudicial to defendant and ordering new trial, vacating jury verdict);

- *In Re Estate of Dukes*, 2011 IL App (4th) 100805-U (retrial of *Dukes I*, this time just granting **judgment *n.o.v.*** to defendants after second plaintiff's verdict achieved without the "unfairly prejudicial" exhibit);

- *Estate of Holmes v. Pneumo Abex*, 2011 IL App (4th) 100462 (majority decision awarding defendant **judgment *n.o.v.*** reversing plaintiff's verdict, creating new law on duty in take-home mesothelioma case);

- *Rodarmel v. Pneumo Abex*, 2011 IL App (4th) 100463 (awarding defendants **judgment *n.o.v.*** after plaintiff's verdict and declining to follow its own precedent in *Dukes I* and *Burgess I & II*);

- *Menssen v. Pneumo Abex Corp.*, 2012 IL App (4th) 100904 (majority decision awarding defendants **judgment *n.o.v.*** after plaintiff's verdict);

- *Dunham v. Honeywell Intern., Inc.*, 2013 IL App (4th) 120608-U (Rule 23 order awarding defendant **judgment *n.o.v.*** on plaintiff's conspiracy claim and reversing jury verdict for plaintiff on plaintiff's negligence claim, ordering a new trial);

- *Gillenwater v. Honeywell Intern., Inc.*, 2013 IL App (4th) 120929 (awarding defendants **judgment *n.o.v.*** after plaintiff's verdict);

- *Smith v. Illinois Central R. Co.*, 2015 IL App (4th) 140703 (jury verdict for plaintiff reversed and new law established as to ability of asbestos defendants to introduce "other exposures" evidence from multiple sources despite lack of any expert testimony linking other exposures as the sole proximate cause of plaintiff's disease);

- *Sondag v. Pneumo Abex Corp.*, 2016 IL App (4th) 140918 (In this case, the Fourth District majority adopted authority from out-of-state intermediate courts (¶ 21) to create new Illinois law as to what is a compensable asbestos disease and awarded defendant **judgment *n.o.v.*** after jury returned verdict for plaintiff.);

- *McKinney v. Hobart Brothers Company*, 2018 IL App (4th) 170333 (Again, the Fourth District awarded defendant **judgment *n.o.v.*** after plaintiff received a jury verdict. Similar to *Rodarmel*, the court

> gave defendant **judgment *n.o.v.*** on unbriefed and unargued issues. The court held defendant Hobart owed no duty to plaintiff because although its corporate representative admitted at trial Hobart knew asbestos caused disease in humans for well over a decade prior to plaintiff's exposure, since Hobart never tested its products, plaintiff could not present evidence Hobart knew its asbestos product in particular caused disease. This is in direct conflict with *Startley v. Welco Manufacturing Company*, 2017 IL App (1st) 153649.).

This Court should affirm the Fifth District below and re-affirm that summary judgment and judgment *n.o.v.* are not opportunities for courts of review to sit as second juries. *Gatlin*, 137 Ill. 2d at 294.

III. *STARE DECISIS* SUPPORTS AFFIRMING THE FIFTH DISTRICT.

O-I's appeal to the "bedrock principle" of *stare decisis* is badly misplaced. If the Court is given to following *stare decisis*, it should affirm the conclusions of *Burgess I & II* and *Dukes I*. After all, those decisions predate *Rodarmel* and *Gillenwater*. As this Court has noted, "Where a court of review reexamines an issue already ruled upon and arrives at an inapposite decision, the straight path of *stare decisis* is affected, as well as the reliance interests of litigants, the bench, and the bar." *O'Casek v. Children's Home and Aid Soc. of Illinois*, 229 Ill. 2d 421, 440 (2008). Although one panel of the appellate court is not strictly bound by a prior decision from a different panel, the spirit of *stare decisis* "is the means by which courts ensure that the law will not merely change erratically, but will develop in a principled and intelligible fashion." *Chicago Bar Ass'n v. Illinois State Bd. of Elections*, 161 Ill. 2d 502, 510 (1994).

In *O'Casek*, this Court held *stare decisis* was not a reason, in itself, to overrule the appellate court after the appellate court departed from its own precedent in *Cargill v. Czelatdko*, 353 Ill. App. 3d 654 (2004). 229 Ill. 2d at 440. However, this Court also noted that in the four years since *Cargill*, the legislature

passed a new law directly addressing the issue of the case. 229 Ill. 2d at 440 ("Public Act 94-677 . . . was not in existence when *Cargill* was decided."). Intervening authority from a higher court, a change in the facts, or a change in the law by the legislature are legitimate reasons to reexamine prior precedent. *See Chicago Bar Ass'n*, 161 Ill. 2d at 510 ("[A] court will detour from the straight path of *stare decisis* only for articulable reasons, and only when the court must bring its decision into agreement with experience and newly ascertained facts."); *Decker v. Union Pacific R. Co.*, 2016 IL App (5th) 150116, ¶ 27 (declining to follow prior Fifth District decision which relied on outdated Illinois Supreme Court precedent).

In this litigation, there were no "newly ascertained facts" in the three years between *Dukes* and *Rodarmel*. The only new evidence in this litigation is that which Plaintiffs have discovered since *McClure*. *Dukes*, 386 Ill. App. 3d at 440. Yet, Plaintiffs did not observe O-I, or Abex for that matter, making any impassioned appeals to *stare decisis* when the Fourth District in *Rodarmel* decided to torpedo *Burgess* and *Dukes*. *Rodarmel*, ¶ 137 (Turner, J. specially concurring) ("I find unnecessary the majority's analysis on whether the evidence was sufficient...."). O-I did not file an amicus brief in *Rodarmel* urging the court to follow *stare decisis*.

In short, the only changes that occurred between *Dukes* and *Rodarmel* were changes in the panels of the court. *See Menssen*, 2012 IL App (4th), ¶ 57 (Turner, J. specially concurring) ("[A] majority of the Fourth District Appellate Court justices has adopted *Rodarmel's* sufficiency-of-the-evidence analysis as Fourth District precedent). But that is not reason to depart from prior precedent from different panels. In fact, *stare decisis* is meant to "permit[] society to presume that fundamental principles are established in the law rather than in the proclivities

of individuals." *Chicago Bar Ass'n*, 161 Ill. 2d at 510. *Stare decisis* "contributes to the integrity of our constitutional system of government both in appearance and fact." *Id.*

The disingenuousness of O-I's appeal to *stare decisis* is betrayed by the fact O-I asks the Court to apply *stare decisis* to *McClure*, but not to *Gillenwater*. It is understandable why: *Gillenwater* concluded O-I and OC were in a conspiracy. Even applying *stare decicis* a la carte to *Gillenwater* is a non-starter for O-I because doing so would contradict O-I's claim that this case is "for all practical purposes" the same as *McClure*, a claim O-I has not made until now.

O-I's claim that this Court should apply *stare decisis* here because this case and *McClure* are "identical" or "the same" is without merit. If O-I's claim about this case and *McClure* were true, then why did the Fourth District write a lengthy opinion in *Gillenwater*? If this case was "for all practical purposes" the same as *McClure*, and if this Court has "already conclusively decided" this case "on indistinguishable evidence" as O-I boldly claims (O-I Br. 20, 26, 34), then the Fourth District in *Gillenwater* could have just said, "This case has already been conclusively decided on indistinguishable evidence by the supreme court in *McClure*. Case closed." Obviously, that is not what happened in *Gillenwater*, that is not what happened in *Johnson*, and that is not what happened in any of the courts below because O-I's claim that this case is indistinguishable from *McClure* is patently incorrect. Yet, O-I makes the claim anyway; perhaps O-I has concluded its only hope to prevail in this case is to convince this Court it has already decided it.

O-I's appeal to *stare decisis* is curious for another reason: O-I fails to cite this Court's seminal asbestos conspiracy case, *Adcock*, even once in its brief. One

would think any demand to apply *stare decisis* would include at least one reference to the seminal case on the issue. O-I is certainly aware of *Adcock*—O-I filed an amicus brief in that case. No doubt O-I has realized *Adcock* is strikingly unhelpful to several of O-I's arguments, such as O-I's claim there is no conspiracy here because O-I has never explicitly admitted to conspiring (*Adcock* holds an explicit agreement is not required) and its claim that conspiracy liability is "outside the proper scope of a manufacturer's liability." O-I Br. 34. *Adcock*, in which manufacturer Owens Corning was the defendant, holds civil conspiracy is a "recognized" and important cause of action in Illinois. 164 Ill. 2d at 62.

IV. THIS COURT SHOULD AFFIRM THE FIFTH DISTRICT BECAUSE JURY ISSUES EXIST AS TO WHETHER OWENS-ILLINOIS CONSPIRED WITH OWENS CORNING TO SUPPRESS INFORMATION ABOUT THE HAZARDS OF ASBESTOS.

O-I raised its *stare decisis* and judgment *n.o.v.* arguments to distract from the truth of this case: evidence exists for a jury to find clearly and convincingly that Owens-Illinois and Owens Corning conspired to suppress knowledge of the hazards of asbestos.

The same elements of a civil conspiracy described in Plaintiffs Abex brief are at issue in Plaintiffs' case against Owens-Illinois. But whereas Plaintiffs allege Abex conspired with Johns-Manville and others, Plaintiffs also have evidence that Owens-Illinois conspired with the company it created, Owens Corning. All Illinois courts of review that have surveyed Plaintiffs' evidence have found Owens-Illinois and Owens Corning both engaged in reprehensible parallel and direct conduct throughout many decades. *E.g.*, *McClure*, 188 Ill. 2d at 146; *Gillenwater*, 2013 IL App (4th) at ¶ 18. The *Gillenwater* court said plaintiffs' portrayal of defendants as "caring only about making money, even at the

expense of people's lives" was "deserved." *Gillenwater*, ¶ 139. The *Gillenwater* court also acknowledged plaintiffs had met the clear and convincing evidence burden on the issue of whether O-I and OC were coconspirators in a scheme to suppress information about the hazards of asbestos in order to ensure continued sales of Kaylo. *Id.* at ¶ 96. The disagreement between the appellate courts is *when* the conspiracy ended. In answering that question, as is always the case at summary judgment, the court "has a duty to construe the record strictly against the movant and liberally in favor of the nonmoving party." *Beaman v. Freesmeyer*, 2019 IL 122654, ¶ 22.

A. The 'Scope' Of the Conspiracy: Yes, Owens-Illinois 'Cared' About Owens Corning's Kaylo Sales.

This case is ultimately about whether O-I's conspiracy with OC ended by operation of law in 1958 or whether Plaintiffs have produced sufficient evidence to create an issue of material fact as to if and when O-I withdrew from the conspiracy. The Fourth District, in *Gillenwater*, held the former. ¶ 96. The Fifth District, below, held the latter (as did the dissenting justice in *Johnson*). *Jones*, ¶¶ 17-18. The Fifth District is correct.

In *Gillenwater*, the Fourth District held O-I and OC were in a civil conspiracy from 1953 to 1958. *Gillenwater*, ¶ 96. Those are the years during which O-I and OC "had a distributorship agreement whereby Owens-Illinois manufactured Kaylo and Owens Corning distributed it." *Id.* at ¶ 90. O-I claims this holding is *dicta*. O-I Br. 37. Whether it is *dicta* has no bearing on whether the Fourth District meant what it said, and whether the holding is *dicta* has no relevance here because the Fourth District does not control this Court. *AFM Messenger Service, Inc. v. Dept. of Employment Sec.*, 198 Ill. 2d 380, 406 (2001)

(appellate court opinions "may provide some guidance, [but] they are not binding on this court").

Kaylo is the asbestos-containing pipe insulation at issue in this case due to John Jones's exposure to it. As is well known now, in 1958 Owens-Illinois sold its Kaylo division to Owens Corning, who continued manufacturing and selling Kaylo, which always contained asbestos through the 1960s and into the 1970s. *Gillenwater*, ¶ 64-65. The *Gillenwater* court held the conspiracy between O-I and OC ended upon O-I's sale of the Kaylo division to OC because "[t]he record gives no basis for supposing that, after 1958, Owens-Illinois cared whether Owens-Corning sold any more Kaylo." *Id.* at ¶ 107.

The Fourth District's analysis is wrong. The record in *Gillenwater*—and the record in this case—is replete with evidence showing that even after 1958 Owens-Illinois cared very much about "whether Owens-Corning sold any more Kaylo." *Id.* The *Gillenwater* court was wrong to dismiss—in one sentence—the financial entanglement of the two companies that lasted from OC's creation throughout the 1970s. *Id.* at ¶ 88. As the court notes, in 1959, O-I's ownership of OC stock had a value of over $110 million dollars, by far O-I's largest investment. *Id.; see* C07371, A384 (1956 annual report). In 1968, approximately when John Jones was exposed to Kaylo, O-I owned 25.1% of OC—an investment worth $143 million dollars accounting for almost 97% of O-I's total investment in public companies. C07376, A387. As late as 1978, O-I still owned over 750,000 shares of OC stock. C07380, A389. OC's profits and earnings were a frequent topic of conversation at O-I directors meetings from the 1940s through the '70s. C07398-425. OC's earnings, profits, and—by extension—the value of its stock were linked with its success in selling Kaylo. Furthermore, Owens-Illinois continued to

provide the packaging for Kaylo until the late 1960s, meaning its own sales revenues were impacted by Kaylo sales. PX 696, A243. With a financial stake totaling in the hundreds of millions of dollars (its biggest investment), of course Owens-Illinois cared "whether Owens-Corning sold any more Kaylo" after 1958. *Adcock*, 164 Ill. 2d at 66 (holding a conspiracy is established from "circumstantial evidence and inferences drawn from evidence, coupled with common sense knowledge of behavior of people in persons in similar circumstances"). After all, by their own description, the two companies were like family. C07391 at 12:12-13, A306.

B. Owens-Illinois Did Not 'Withdraw' From The Conspiracy By Selling Its Kaylo Division To Owens Corning.

The *Gillenwater* court's holding that Owens-Illinois "withdrew from the conspiracy" in 1958 by simply selling its Kaylo division to Owens Corning is based on reductive and flawed reasoning. *Id.* at ¶¶ 110-118. The court decreed that the "object" of the conspiracy between O-I and OC was for OC to sell O-I's Kaylo without an adequate warning. *Id.* at ¶¶ 107, 111. Thus, the court's reasoning goes, when O-I sold Kaylo to OC, Kaylo was *technically* no longer "O-I's product," and, so, O-I could no longer participate in a conspiracy to sell "O-I's Kaylo" because "O-I's Kaylo" no longer existed. In short, after the sale, Owens-Illinois Kaylo became Owens Corning Kaylo. *Id.* at ¶ 111.

The court's reasoning is flawed because even after O-I sold Kaylo to OC it still had a powerful incentive to participate in the conspiracy. In addition to the reasons explained above concerning O-I's continued financial entanglement with OC, Owens-Illinois *continued to utilize asbestos in its own operations*. C07807. The difference between the conspiracy here and the conspiracy alleged by the

government in the case *Gillenwater* cites, *United States v. Steele*, 685 F.2d 793 (3d Cir. 1982), is that once the defendant employee in *Steele* resigned from his position at General Electric, he truly had no reason to care whether the conspiracy continued. Imagine if, instead of retiring from General Electric, he had gone to work for a co-conspirator where he also performed work in furtherance of the conspiracy. Would his defense that he "withdrew" from the conspiracy by resigning from General Electric still be persuasive?

The Third Circuit recognized this distinction. Unlike the *Gillenwater* court, the *Steele* court held the question of whether the defendant withdrew from the conspiracy was a question of fact, not a question of law. *Steele* states that evidence of withdrawal can be rebutted "by going forward with evidence of some conduct in furtherance of the conspiracy subsequent to the act of withdrawal." *Steele*, 685 F.2d at 804. Had the prosecution in *Steele* done that, instead of "standing on its proof," the question of whether the defendant had truly withdrawn from the conspiracy "properly would have gone to the jury." *Id.*

Likewise, the fact O-I sold its Kaylo division to OC in 1958 is merely *evidence of withdrawal*, it is not, as the *Gillenwater* court would have it, withdrawal by operation of law. Owens-Illinois's continued financial stake in Owens Corning after 1958, the fact it continued to provide the packaging for Kaylo throughout the 1960s, and the fact it continued to use asbestos in its own products and facilities and never warned its consumers or workforce is all evidence rebutting the contention Owens-Illinois "withdrew" from the conspiracy in 1958 simply by selling Kaylo to Owens Corning, as the court below understood. *Jones*, ¶¶ 17-18. It is not the Court's duty to weigh this evidence at

summary judgment; instead, the question should go to the jury. *Beaman*, ¶ 22; *see also Steele*, 685 F.2d at 804. The Court should affirm the decision below.

C. The Evidence Of Conspiracy As To Owens-Illinois And Owens Corning Is Not Limited To Parallel Conduct.

Throughout this appeal, Owens-Illinois and Abex have invoked *McClure* to argue that parallel conduct is insufficient to raise a fact issue of conspiracy and, as a result, they should both receive summary judgment. Their argument is based on the false premise that the evidence in this case is limited to parallel conduct. It is not. It is based on intersecting conduct, both as to Abex and Johns-Manville (see Plaintiffs' Abex brief) and as to Owens-Illinois and Owens Corning.

Owens-Illinois and Owens Corning did not just act independently-yet-parallel to each other. They interacted with each other. Owens-Illinois was massively invested in Owens Corning. Owens-Illinois and Owens Corning shared directors until the Department of Justice intervened. Owens-Illinois provided Owens Corning with the packaging for Kaylo asbestos insulation through the 1960s. PX 696, A243. O-I shared articles about the hazards of asbestos with Owens Corning, but neither company ever shared those articles with their employees or customers. O-I and OC agreed to team up to mass produce and distribute Kaylo. This is not parallel conduct. This is acting in concert.

D. The 'Innocent Explanation Rule' Does Not Apply To Owens-Illinois' Parallel Conduct With Owens Corning.

In the instances where O-I and OC's conduct is parallel, the "innocent explanation" rule should not apply. In order for parallel conduct to be subject to the "innocent explanation rule," the conduct in question must be "*as consistent*

with innocence as with guilt." *McClure*, 188 Ill. 2d at 147-48 (emphasis added). The crux of the innocent explanation rule as applied to this case is the question of whether these asbestos companies all acted in the same wrongful way because of a mutual understanding, or whether they just *happened to* because, as members of the same industry, they would all encounter "the same business problems, the same consumer demands, and the same competitive pressures." *McClure*, 188 Ill. 2d at 141.

Although the *McClure* court decided the evidence showed the latter as to O-I and OC's parallel conduct with Johns-Manville and Unarco, the evidence presented in *McClure* is not the same as the evidence in this case. Since *McClure*, as discussed above, plaintiffs have uncovered additional evidence illuminating the shared interests of the companies. Statement of Facts, *supra*, 12-13. In fact, Owens-Illinois and Owens Corning misrepresented the extent of their contacts with each other and with other conspirators in that case. *Id*; *Dukes*, 386 Ill. App. 3d at 440. Additionally, because the plaintiff in *McClure* was not exposed to Kaylo, this Court only decided whether O-I and OC's parallel conduct with Johns-Manville and Unarco was "innocent." Until this case, *Gillenwater* was the only published decision to take up whether O-I and OC's parallel and intersecting conduct *with each other* shows a conspiratorial agreement between the two of them. Although the *Gillenwater* court correctly found O-I and OC were in a conspiracy, the court usurped the jury's role by making its own determination of when the conspiracy ended.

Just as the court in *Rodarmel* misapprehended the nature of plaintiffs' conspiracy allegations, so too did the court in *Gillenwater*. The *Gillenwater* court, which admitted to having a "problem . . . with the theory of conspiracy" seems to

believe the conspiracy plaintiffs allege in these cases is just a conspiracy to make money, and what's wrong with trying to make money? *Gillenwater*, 2013 IL App (4th) at ¶ 138-39. The court asks,

> [W]hy would one need to posit a conspiratorial agreement to explain these companies' continuing to do the wrongful things whereby they were each making a lot of money? They each, individually and independently, had a powerful economic incentive to conceal the hazards of their own asbestos-containing products. Consequently, it would seem that a conspiratorial agreement is a fifth wheel, which could be lopped off by Occam's razor.

Id at ¶ 139. The flaw in this view, especially in light of the evidence in this case, is it requires the cynical assumption that each corporate member of every industry in a capitalist economy will naturally, and independently, turn to knowingly exposing its consumers and workforce to a lethal product without warning in order to increase profits. This outcome is not a public policy Illinois courts should want to encourage.

Moreover, that assumption is not borne out in typical corporate conduct. For example, just because one car company saves money by making cars with exploding gas tanks does not mean every other car company will do so as well. Just because one car company saves a few pennies on each car by using a faulty ignition switch does not mean every other car company will do so as well. Just because one car company cheats on its emissions tests does not mean every other car company will do so as well. Indeed, in its $1,000,000,000 asbestos conspiracy lawsuit, Owens-Illinois alleges if it had only known asbestos was hazardous by 1953, the company would have "foregone commercial production of Kaylo" and "redirected its resources to other ventures." C07627.

On the other hand, if a group of car companies came together, tested the ignition switches they were all using, found them to be faulty and hazardous, and then each did their part to suppress that information from consumers so that they could all keep "making a lot of money," you might say that conduct is not "as consistent" with innocence as with guilt. You might say it shows—clear and convincingly—the companies had a mutual understanding or implicit agreement to suppress information about the hazardous ignition switch. Wouldn't the conduct in this example be *more* consistent, instead of *as* consistent, with guilt instead of innocence? That is the conclusion jury after jury after jury has reached in the over 20 years asbestos conspiracy cases were tried in this state, from the late 1980s until the Fourth District in *Rodarmel*, *Menssen*, and *Gillenwater* substituted its judgment for not only the judgment of all those juries, but also the judgment expressed in *Burgess I*, *Burgess II*, and *Dukes I*.

There is nothing inherent in making thermal pipe insulation, or glass products, or packaging products, or brake pads and brake linings that requires the suppression of information about the lethal hazards encountered while making those products or working with them. Similar business problems, consumer demands, and competitive pressures do not provide an "innocent explanation" for the uniform decision to suppress information about the hazards of asbestos from workers and consumers for decades "even after the human cost [becomes] evident." *Gillenwater*, ¶ 18. As O-I astutely pointed out, the companies in the conspiracy could have simply "foregone commercial production" of asbestos products and "redirected [their] resources to other ventures." That is normal business conduct when a business knows it has a "first class hazard" on its hands, like O-I did in 1943. Normal business conduct is not mass-producing

the first class hazard and engaging your former subsidiary to mass distribute it with an advertisement that says "non-toxic." Normal business conduct is not staying silent while a company in which you have an ownership interest totaling hundreds of millions of dollars exposes an entire generation of tradesmen to a lethal product you developed.

V. EXPOSING A GENERATION OF WORKERS TO A LETHAL PRODUCT IS 'EXTRAORDINARY.' HOLDING THE WRONGDOERS RESPONSIBLE IS JUSTICE.

Owens-Illinois dedicates the first paragraph of its brief to fearmongering about expanded liability. That O-I conjures up a bogeyman in its first paragraph should tell the Court everything it needs to know about how O-I perceives the strength of its case based on the evidence and the law.

O-I continues its fearmongering on page 34 of its brief. O-I says the "elephantine mass" of asbestos litigation is ever-growing, and O-I warns that holding it accountable for its own tortious conduct will result in "unending streams of wasteful trials," meaning the "extraordinary traffic" of asbestos conspiracy claims will only continue. O-I's Br. 34-36.

Of course, when O-I felt it had been the victim of an asbestos conspiracy, it had no problem adding to the "elephantine mass" of asbestos litigation by filing a $1,000,000,000 asbestos conspiracy lawsuit. Without a hint of self-awareness, Owens-Illinois even crassly suggests that plaintiffs like John Jones should just forget O-I, the company that created both Kaylo and Owens Corning, and petition bankruptcy trusts instead. O-I Br. 35. But that was not what Owens-Illinois did when it demanded $1,000,000,000 in its own asbestos conspiracy lawsuit. Maybe instead of filing a $1,000,000,000 asbestos conspiracy lawsuit, Owens-Illinois should have just followed its own advice and asked Johns-

Manville to settle its claims for $4,845, which is the most the Manville Trust compensates claimants like John Jones with terminal lung cancer.

There is nothing "extraordinary" about holding a company liable for exposing a generation of workers and their families to a deadly product. O-I's argument is perverse: because its wrongdoing was so pervasive and the fallout has affected so many for so long, it can no longer be held accountable. O-I is wrong. The continued devastation from O-I's misconduct is more reason to hold O-I accountable, not less. *Beaman v. Freesmeyer*, 2019 IL 122654, ¶ 45.

A hallmark of O-I's briefs in these cases has been O-I's willingness to steadfastly claim it never did anything wrong at all in the face of a mountain of evidence to the contrary. Indeed, in the court below, O-I claimed its 1953 agreement with Owens Corning to mass produce and distribute a product they both knew was deadly constituted nothing more than "the epitome of ordinary business conduct." Def.'s App. Br. 30.

"Ordinary business conduct" does not result in thousands of deaths per year for decade, after decade, after decade. The "elephantine mass" of asbestos cases is not a result of plaintiffs expanding liability to places where it should not lie. It is a result of companies—two of the foremost being Owens-Illinois and Owens Corning—pushing to utilize and sell ever more asbestos products in the face of a public health crisis of their own making.

Owens-Illinois knew its product "had all the ingredients for a first class hazard" in 1943. PX 567. But instead of "forego[ing] commercial production" of Kaylo and "redirected[ing] its resources to other ventures," O-I ramped up production for 10 years and then in 1953 teamed up with Owens Corning for the express purpose of selling even more Kaylo. C07652, A317 ("In 1953, Owens-

Illinois turned to OCF, which had substantial experience in the business of manufacturing and installing heat insulation products, to sell some Kaylo products.").

Instead of warning workers and customers, O-I sought to ensure the interests of its company would be safe-guarded. PX 278. Instead of warning consumers its product was toxic, Owens-Illinois and Owens Corning together advertised Kaylo as "non-toxic." PX 33. Instead of warning its workers about the hazards of asbestos, O-I helped OC develop a dossier of medical articles as a "weapon in reserve" to deploy only if necessary to "over-throw" union leadership. PX 66. Neither company warned its workers for decades after first learning asbestos causes disease. C07329 at 117:20-119:22; C07814, 20-21.

In sum, O-I's complaints of having to defend lawsuits brought by victims of this conduct should not find a sympathetic audience in this Court. O-I is here because of its own misconduct—not just because Owens Corning exposed John Jones to asbestos Kaylo in the 1960s, but because Owens-Illinois, through its own actions, did "its part... to further [the] objective" of the conspiracy." *McClure*, 188 Ill. 2d at 134. This is not a case of vicarious liability. This is a case of affording a trial to a man with terminal lung cancer because Owens-Illinois wanted to make more money. This Court should affirm.

CONCLUSION

Plaintiffs have presented sufficient evidence to raise triable issues of material fact as to whether Owens-Illinois and Owens Corning entered into a civil conspiracy and whether Owens-Illinois withdrew from the conspiracy by 1969. Plaintiffs respectfully request this Court affirm the Fifth District's reversal of the trial court's grant of summary judgment to Owens-Illinois.

JOHN JONES and DEBORAH JONES
Plaintiffs-Appellees,

By: _____

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RULE 341 CERTIFICATION

I certify that this brief conforms to the requirements of Rule 341(a) and (b). The length of this brief, excluding the pages or words contained in the Rule 341(d) cover, the Rule 341(h)(1) statement of points and authorities, the Rule 341(c) certificate of compliance, the certificate of service, and those matters to be appended to the brief under Rule 342(a), is 13,480 words.

CHARLES LYNN CORWIN,
ARDC, #6312586

No. 123895 & No. 124002
(Consolidated)

IN THE SUPREME COURT OF ILLINOIS

JOHN JONES and DEBORAH JONES, Plaintiffs-Appellees, v. PNEUMO ABEX LLC and OWENS-ILLINOIS, INC., Defendants-Appellants.	On Petition for Leave to Appeal from the Appellate Court of Illinois, Fifth Judicial District, No. 5-16-0239. There on Appeal from the Circuit Court of the Second Judicial Circuit, Richland County, Illinois, No. 13-L-21, Hon. William C. Hudson, Judge Presiding.

SUPPLEMENTAL APPENDIX

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June 7, 1965

Mr. R. S. Parker
New York Office

Subject: Asbestos Study

You have a copy of Dr. Blackwell's letter of May 25 to Dr. W. S. Lainhart of the U. S. Public Health Service and a letter of the same date to me.

Bill Terry has suggested we might want to refer this matter as to the "general idea" to our insurance people, our labor people, and possibly our legal department to get an expression from them in the event they have been exposed to this sort of survey and what their experience indicates.

We have an exploratory meeting set up at Winchester for June 23d --- this is discussion only and we will not commit ourselves to anything until after the meeting.

Dr. Lainhart called me the other day and we arranged this meeting --- at that time he pointed out they, as a Federal Agency, liked to work through the State and, consequently, asked if a representative of the State of Virginia Health Dept. could be in attendance.

I agreed on the premise that the State man would be as discreet as the Federal men are described to be.

We have no fear of the survey since we know of no instance where we have a work (asbestos) related health hazard, or lung damage, etc.

I've gone into some detail here so all parties are up to date.

Your comments on Bill Terry's suggestion will be appreciated before the June 23d meeting.

/hm

D. K. Rennie

cc: NBTerry
CCBlackwell, M.D.
LHEvans
CBMallory

JWK007022

A1

DEFENDANT'S EXHIBIT
A&LX 602

MEMORANDUM OF AGREEMENT

November 20, 1936

THE UNDERSIGNED hereby agree to underwrite certain experiments with asbestos dust to be conducted by Dr. LeRoy U. Gardner at the Saranac Laboratory, Saranac Lake, New York.

The general nature of these experiments and the cost thereof were explained at the meeting of certain brake lining manufacturers held in New York City on November 19, 1936.

The purpose of this memorandum is to confirm the agreement between the Undersigned to underwrite the cost of these experiments amounting to $5,000 per year for a period of three (3) years, or a total of $15,000.

The Undersigned agree to share equally the costs of said experiments. For example, if ten parties sign this Agreement, the annual contribution of each will be $500. It may be more or less, depending on whether the number of those signing is more or less than ten.

It is understood that payments to Dr. Gardner will, for the sake of convenience, be made directly by Johns-Manville Corporation or by Raybestos-Manhattan, Incorporated, and that the others of the Undersigned will reimburse Johns-Manville

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R. M. 337 D. 5-2-77

Corporation or Raybestos-Manhattan, Incorporated for their pro rata share of such payments as made.

AMERICAN BRAKE BLOCK CORPORATION
(Limited to $250.00 per annum)
By _____
Vice President

ASBESTOS MANUFACTURING COMPANY
By _____

GATKE CORPORATION
(Limited to $250.00 per annum)
By _____ Pres.

JOHNS-MANVILLE CORPORATION
By _____
Vice-President

KEASBEY & MATTISON
By _____
Vice President

RAYBESTOS-MANHATTAN, INCORPORATED
By _____

RUSSELL MANUFACTURING COMPANY
By _____

THERMOID RUBBER COMPANY
By _____

UNION ASBESTOS & RUBBER COMPANY
(Limited to $250.00 per annum.)
By _____
Secretary-Treasurer

UNITED STATES GYPSUM COMPANY
(Limited to $250.00 per annum)
By _____
Secretary-Treasurer

_____

By _____

_____

By _____

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THERMOID COMPANY

By _____

SOUTHERN ASBESTOS COMPANY

By Geo. L. Fakel, Pres.

06818

November 20, 1936

LeRoy U. Gardner, M. D., Director,
The Saranac Laboratory,
Saranac Lake, New York.

Dear Dr. Gardner:

At a meeting yesterday attended by certain brake lining manufacturers, Mr. Simpson and I were able to present to a fairly large group of interested corporations the proposal that one of your dusting chambers be engaged for further experimentation with asbestos dust.

The proposal was very well received and it appears that not less than eight, and perhaps ten or more, corporations will participate in financing these further experiments along the lines discussed when we met with Dr. Lanza and Dr. McConnell a few days ago.

Accordingly, you may consider this letter as an authorization to you to commence the contemplated experiments with asbestos dust for the purpose of determining more definitely the causes and effects of asbestosis. It is my understanding that, among other questions which it is anticipated these experiments will answer, are the following:

(1) What concentration of dust is necessary to produce the fibrosis of the lungs which is designated as asbestosis.

(2) Whether exposure to asbestos dust will produce asbestosis without the existence of previous infection and whether the X-ray changes found in advanced human asbestosis can be reproduced in animals without infection.

(3) Whether the fibrosis produced by asbestos is of the progressive type, that is, will the fibrosis increase (once it has started) after exposure to the dust has ceased.

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(4) Whether the fibrosis resulting from exposure to asbestos dust is occasioned by the silicon content of the asbestos or by its fibrous structure.

(5) Whether the presence of "asbestos bodies" has any diagnostic significance.

It is also our understanding that these experiments will require approximately three years, which will mean that they will not be concluded until about the end of the year 1939; and that they will cost $5,000 annually, or a total of $15,000.

It is our further understanding that the results obtained will be considered the property of those who are advancing the required funds, who will determine whether, to what extent and in what manner they shall be made public. In the event it is deemed desirable that the results be made public, the manuscript of your study will be submitted to us for approval prior to publication.

I shall appreciate your advising me if the foregoing accurately expresses the proposition you had in mind. Also, let me know the manner in which you would like to have the funds advanced to you. Would you, for example, desire to have $1250 paid quarterly in advance, or just what arrangement would suit you best? Any method you suggest will be satisfactory.

Since the experiments are being underwritten by a relatively large group, it has been suggested that, as a matter of convenience, any communications regarding the work be addressed to me and I will, where necessary, communicate the contents thereof to Mr. Simpson and the other parties. You will be provided with funds by either Johns-Manville Corporation or Raybestos-Manhattan, Incorporated and we or they will secure from the other parties their pro rata contribution.

I will advise you later of the names of the other contributing parties.

I shall appreciate your informing me at the earliest possible moment whether the arrangement as outlined herein meets with your approval.

Sincerely yours,

Vandiver Brown

Vandiver Brown
General Attorney

VB:T

004738

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THE SARANAC LABORATORY FOR THE STUDY OF TUBERCULOSIS
OF
THE EDWARD L. TRUDEAU FOUNDATION

November 23, 1936

Mr. Vandiver Brown
Johns-Manville Corporation
22 East 40th Street
New York City

Dear Mr. Brown:

Your authorization to proceed with the proposed asbestosis experiments at the Saranac Laboratory is acknowledged with satisfaction.

We believe that such experiments can be expected to furnish answers to the questions which you have outlined. The estimated cost of $5,000 annually for a period of three years, making a total of $15,000, is correct.

Your suggestion to make advanced quarterly payments of $1,250 will be entirely satisfactory to the Laboratory. If this is not convenient any other arrangement that you may care to make will be acceptable.

The Saranac Laboratory agrees that the results of these studies shall become the property of the contributors and that the manuscripts of any reports shall be submitted for approval of the contributors before publication.

I would recommend that the experiments be performed with pure Canadian asbestos fibre containing relatively little serpentine rock dust. The "Kings Floats" used in our first experiment was unsatisfactory owing to the fact that it contained such a high concentration of non-fibrous rock dust. If this meets with your approval I would suggest that a sample be submitted to the Laboratory for chemical and petrographic analysis.

Having mutually satisfied ourselves as to the material to be used I would request that the companies agree to supply a sufficient quantity of asbestos, ground to the finest state of subdivision possible. I am not aware whether any of the contributors have apparatus capable of reducing fibres to inhalable dimensions. If this request should not

prove practical some arrangement for its final reduction will have to be made by the Laboratory. But at least the material received by us should be broken to powder.

It is impossible to estimate the amount of material necessary for the experiments at the present time as its behavior in our dusting apparatus has never been determined. With the "Kings Floats" tests approximately 1200 pounds a year were required. It is conceivable that a pure fibrous dust might be dissipated into the atmosphere more rapidly and hence even a larger quantity would be necessary.

We are ready to start the work at any time and will be pleased to examine samples of dust as soon as they can be procured.

Thanking you for your cooperation in arranging this program, I am

With best regards,

Sincerely yours,

Leroy U. Gardner, M.D.
Director cc

LUG:CC

JOHNS-MANVILLE CORPORATION
TWENTY-TWO EAST FORTIETH STREET
NEW YORK, N.Y.

EXECUTIVE OFFICES

February 27, 1937

Mr. Sumner Simpson,
Raybestos-Manhattan, Inc.,
Bridgeport, Conn.

Re: Asbestos Dust Experiments -
Dr. L. U. Gardner

Dear Mr. Simpson:

The United States Gypsum Company signed the Memorandum of Agreement relating to the underwriting of Dr. Gardner's experiments with asbestos dust, limiting its participation to the sum of $250 per annum, or a total of $750 for the three-year term of the experiments.

I believe that we have now fully canvassed the industry. The co-operation of the following companies has been obtained on a basis whereby their respective contributions are limited to $250 per annum:

(1) American Brake Block Corporation
(2) Gatke Corporation
(3) Union Asbestos & Rubber Company
(4) United States Gypsum Company.

The participation of the above named companies will provide $1,000 a year, leaving the sum of $4,000 per year to be pro-rated between the following companies who have not limited their participation in the enterprise:

(1) Asbestos Manufacturing Company
(2) Johns-Manville Corporation
(3) Keasbey & Mattison
(4) Raybestos-Manhattan, Incorporated
(5) Russell Manufacturing Company
(6) Thermoid Company and Southern Asbestos Company.

The six companies last above named will, therefore, be called upon to contribute the sum of $666.67 per year for the three-year period.

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I am enclosing herewith, for your records, a photostatic copy of the Memorandum of Agreement and photostatic copies of my letter to Dr. Gardner of November 20, 1936 and his reply of November 23, 1936, which set forth the substance of the arrangement between Dr. Gardner and the group.

Copies of this letter, together with copies of the enclosed photostats, are being sent to each of the other participants.

Last December Johns-Manville Corporation sent Dr. Gardner its check for $1250, constituting advance payment for the first quarter of 1937 on account of his annual retainer. Your Company and others of the group will be billed shortly for your share of this payment under the terms of the Memorandum. Johns-Manville Corporation is willing to continue making these advances to Dr. Gardner at the commencement of each succeeding calendar quarter during 1937, unless you or others prefer the matter be handled differently. I believe, however, that Dr. Gardner will prefer, in the matter of payment, to deal with one member of the group and it is my opinion, in which I believe you concur, that this represents the most satisfactory procedure.

Johns-Manville Corporation has likewise provided Dr. Gardner with 1200 pounds of asbestos fibre which he considers satisfactory for his purposes, although it has been found necessary for him to subject it to a "ball milling" process in order to reduce it to the desired degree of fineness. He estimates that this quantity will be sufficient for the first year's experiments and we will be very glad, at our expense, to provide him with similar quantities for the other two years.

I have also sent Dr. Gardner a photostatic copy of the Memorandum of Agreement so that he is fully advised as to the identity of those companies whose support he is receiving. At the same time I advised Dr. Gardner that those concerns which had limited their contributions were to participate just as fully in the benefits resulting from his experiments as were the others.

Very truly yours,

Vandiver Brown

Vandiver Brown
General Attorney

VB:T
Enclosures

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A10

December 18, 1939.

Dr. Leroy U. Gardner,
The Saranac Laboratory,
Saranac Lake, New York.

Dear Dr. Gardner:

I have received yours of December 14th and the very interesting "Interim Report on Experimental Asbestosis" which was enclosed therewith.

As usual, I plan to distribute copies of your report to the various members of the group which has been sponsoring these experiments - ten in number. For this purpose I will require four (4) additional sets of the illustrations and I will appreciate your furnishing them at your earliest convenience.

The matter of financial support for another year of experimentation will have to be considered by the group and for that reason I cannot, of course, give you any definite advice at this time.

With very best regards, I am

Sincerely yours,

VB:y

Vandiver Brown,
General Attorney.

Cc Mr. Sumner Simpson,
Raybestos-Manhattan Inc.,
Bridgeport, Conn.

C04745
4-21-77 JK
RA 1:25 PM

BC-Saranac 053

November 12, 1946

Mr. Manfred Bowditch
Field Director
The Saranac Laboratory
Saranac Lake, N. Y.

Dear Mr. Bowditch:

As a member of the group who advanced certain sums of money to The Saranac Laboratory for asbestos dust experiments conducted by Dr. Gardner and which were completed some time ago, we are interested in knowing whether other members of your staff are in a position to assemble the accumulated data and prepare a final report thereon. As you are aware from earlier conversations, the preparation of this report was unavoidably delayed during the war period, although our most recent correspondence indicated that Dr. Gardner believed it would be completed this winter. Several interim reports were prepared by Dr. Gardner and distributed to various members of the underwriting group and under date of February 24, 1943 he submitted an "Outline of Proposed Monograph on Asbestosis" which indicated that his development of his thesis was fairly well matured. These lead me to believe that anyone fairly closely associated with him in the conduct of these experiments or who, being an expert in the field, might review the accumulated data could give the Industry a report that would be valuable.

At your convenience, I would appreciate receiving your comments on the foregoing and I would also like to refer you to my letter of November 20, 1936 to Dr. Gardner and his reply of November 23 which initiated these experiments, with particular reference to the paragraphs which state that the results of these studies are to be the property of the contributors and that manuscripts of any reports thereon are to be submitted for approval before publication. Although you will note from these letters the arrangement originally contemplated three years of experimental work at $5,000 per year, they were actually prolonged for two additional years.

Sincerely yours,

Vandiver Brown
Secretary

VB:S
CC Mr. Ernest Muehlock
Keasbey & Mattison Co.
Ambler, Pa.

Mr. Sumner Simpson
Raybestos-Manhattan, Inc.
Bridgeport, Conn.

July 23, 1947.

Dr. A. J. Lanza,
Associate Medical Director,
Metropolitan Life Insurance Company,
New York, 10, N. Y.

Dear Doctor Lanza:

I am sending herewith Gardner's manuscripts as I have arranged, joined and titled them in a proposed single publication.

The experimental report is left as it was, except for the heading. Please pass judgment on the title and footnote.

His monograph outline of human asbestosis contains little if any significant material---No one else can write what he intended without it becoming the writer's instead of Gardner's.

On the other hand, the outline on experimental asbestosis is more than that. It is really a summary of Gardner's observations and is valuable and publishable as it stands. I have put a heading on that part, deleted section 1, on 'Methods', and renumbered the other sections.

The whole makes somewhat of a bulky publication, but I believe that you can induce the Journal of Industrial Hygiene to take it in this form. If not, let's split it into two.

I am sending the detached Part I of the Outline also, in order that you may see what I mean.

Please consult with such others as Sarsany as you should and with the Asbestos people, if that is in order. Feel free to reject any idea of mine which does not seem best and criticize freely.

With best personal regards, I am

Sincerely yours,

Kenneth M. Lynch, M. D.

KML:khp.

J-4

C06886

A13

July 23, 1947.

P. S. I am retaining for the present the other material sent me. Can someone at Saranac readily supply the references in the manuscript? I am a bit short on time. Should Cummings be in this?

KKL.

K120a

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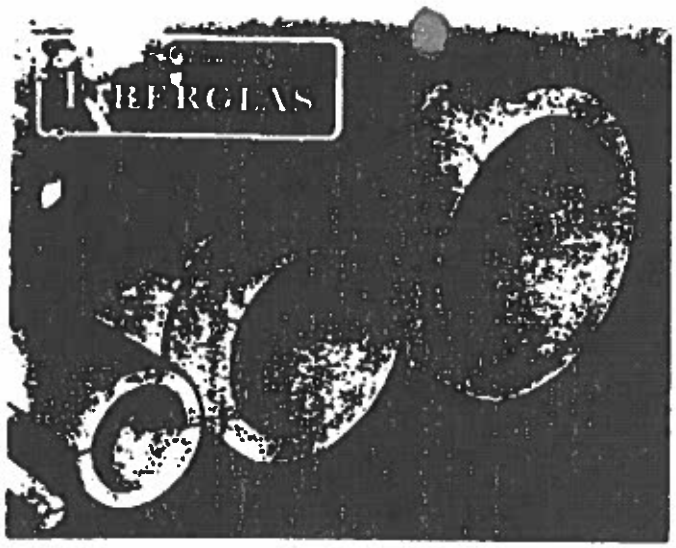
A14



piping ve [illegible]

IN1.C5

October, 1956

high temperature insulation for indoor and outdoor piping up to 1200°F. operating temperature

Kaylo pipe insulation

Kaylo *Pipe Insulation, a white, rigid hydrous calcium silicate heat insulation, manufactured by Owens-Illinois Glass Company and distributed nationally by Owens-Corning Fiberglas Corporation, effectively insulates indoor and outdoor piping up to 1200°F.*

This thoroughly tested and field-proven material contains the physical properties most desired for efficient, durable insulation work. Kaylo Pipe Insulation's thermal effectiveness, high strength and moisture resistance are unattained by most other insulating materials.

A chemically reacted material, Kaylo Pipe Insulation contains no added binder. For mechanical effects, a small amount of asbestos fiber is included at the time of manufacture.

jackets

canvas—Kaylo Sectional Pipe Insulation includes standard canvas jackets, at no additional charge, on thicknesses up to and including 2½". Standard canvas is available on segmental forms at a slight additional cost. Bands are available for use with this product at slight additional charge and will be furnished on request.

6 oz. and 8 oz. canvas jackets are available for all types, sizes and thicknesses of Kaylo Pipe and Tube Insulation at an additional charge. These jackets are factory applied on Sectional Pipe and Tube Insulation. On Multi-segmental forms, they are furnished but not adhered.

sizes

thickness—Kaylo Pipe Insulation is available in thicknesses from 1" to 3" depending upon pipe size. Kaylo Tube Insulation is available in thicknesses of 1" and 1½".

pipe sizes—Kaylo Pipe Insulation is available to fit pipe from ½" to 39" in diameter. Kaylo Tube Insulation will fit copper tubing from ½" to 3⅛" in diameter.

forms—Kaylo Pipe Insulation is available in sectional or multisegmental form depending upon pipe size. All insulation is furnished in 3' sections.

*T. M. Owens-Illinois Glass Co

offers . . .

high efficiency—Among the most efficient insulations for temperatures up to 1200°F. Low "k" results from extremely small and numerous insulating air spaces.

simplified dimensional standards—Sizes are designed to permit nesting of one size over another to produce greater thicknesses than available in single layer or to produce "broken joint" concept.

water resistance—Kaylo Pipe Insulation retains an appreciable percentage of its strength even after complete saturation in water. After a cycle of soaking and drying, it returns to its original thermal efficiency and strength with no shrinkage or warping.

high strength—High flexural strength, compressive strength and resistance to abrasion far above normal requirements for heat insulation, greatly reduce installation and service breakage.

easy fabrication—Ordinary tools of the trade sufficient for all cutting, sawing or scoring. Non-irritating to the skin and non-toxic. Offers pleasant handling characteristics.

long life—Remains strong and efficient over the years. Dimensionally stable with little shrinkage. Has superior life characteristics over other types of high temperature insulations.

versatility—Performs efficiently at temperatures through the hot water and low pressure steam range in addition to temperatures in the superheated steam range. One high temperature material to do the job usually requiring combinations of two different insulating materials.

limitations

Kaylo Pipe Insulation is designed for temperatures up to 1200°F. and is not recommended when operating temperatures of piping exceed this limit.

NEW YORK UNIVERSITY
COLLEGE OF MEDICINE
(UNIVERSITY AND BELLEVUE HOSPITAL MEDICAL COLLEGE)
477 FIRST AVENUE, NEW YORK 16, N.Y.

DEPARTMENT OF PREVENTIVE MEDICINE
Institute of Industrial
Medicine

TELEPHONE: [redacted]
MU 3-1800

December 14, 1948

Dr. Arthur Vorwald
Saranac Laboratory
Saranac Lake, New York

Dear Arthur:

With respect to the asbestos report, Part I, September 30, 1948, a meeting of the representatives of the underwriting companies was held in New York. The report was favorably received and it was the general feeling that it was a most satisfactory and excellent job. It was the feeling of this group that all references to cancer or tumors should be omitted (Paragraph 75, Page 31 - the introductory paragraph under "COMPLICATIONS" - Paragraph 92, Page 39). This request would likewise call for the elimination of any tables relating to this subject matter.

The group felt that there might be included under "CONCLUSIONS" a reference to the non-progressive character of the fibrosis produced by the asbestos.

If practicable, they would like to see a more definite statement with respect to the size of the fibres which do the damage. Inquiry was also made as to whether there was any relation to the number of fibres per cubic foot of air.

It was felt that the language in Conclusion D, that is, that a typical fibrosis could be produced by an atmospheric suspension of asbestos dust containing only "an extremely small proportion of long fibres" was too vague and should be defined more specifically.

In addition to the above comments there are other suggestions which relate more to form and emphasis:

To the extent permitted by the results of the experiments make the "CONCLUSIONS" and the introductory "ABSTRACT" more definite, and reduce the number of words whose meanings are not precise. For example, in the "ABSTRACT" the words "seems", "relatively" , "apparently", "is believed" and in "CONCLUSIONS", page 40, the words "primarily", "extremely small proportion".

As far as practicable conform the "ABSTRACT" to "CONCLUSIONS". For example, "CONCLUSIONS" states that "short asbestos fibres do not produce fibrosis", whereas "ABSTRACT" says that "short fibres are relatively inert".

J-3

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Where experimental results indicate that asbestos fibre produces a different action than that caused by free silica, emphasize these points by reference to the differences as, for instance, particle size, mechanical rather than chemical action, non-progression, and predisposition to tuberculosis.

In CONCLUSION E, page 41, reference is made in the fourth line to "our usual experience" and in the last line, bottom of page, "previous experience". Just what was meant by this reference was not clear to the group and they would like to see that item clarified.

It was felt that the report would be somewhat easier to follow if the paragraphs dealing with the various types of experiments were separated by headings. For example, Paragraphs 11 to 24 inclusive, deal with the early King's Floats experiments; Paragraphs 25 to 44 deal with the 200-mesh material to which was added one part in four of unground material; Paragraphs 45 to 57 inclusive, deal with the 100% ball-milled dust experiments, and Paragraphs 58 to 76 inclusive, deal with the long fibre experiments. There was confusion as to the experiments referred to in some of the numbered paragraphs. It would be helpful if the four experiments were headed up in some manner other than with the regularly numbered paragraphs which precede and follow them.

It was decided that after these revisions have been concluded the report of these experimental studies should be published as promptly as possible, preferably in the Journal of Industrial Hygiene. Any report on human asbestosis should be separate and not a part of this report.

The above comprises the various comments and suggestions which I do not think involves any material change in the report. Please let me know if you wish any further information or discussions of the points raised in this letter.

Best regards.

Sincerely yours,

Tony.

A. J. Lanza

CC6890

A17

[redacted]

December 14, 1948

A. J. Lanza, M.D.
Associate Medical Director
Metropolitan Life Insurance Co.
1 Madison Avenue
New York 10, N.Y.

Dear Tony:

In furtherance of our telephone conversation of this morning, I am enclosing a copy of my letter of December 13 to Andy. I feel as you do about his report to G.H.Q. and was somewhat disturbed by his reaction to your memorandum to him.

As for the asbestos report, I am happy to know that it was so well received. We, of course, want to get it into publication as soon as possible; however, with the changes introduced which the asbestos group believe are essential. Having your summary of their comments will enable us here to review the article for review when I come to New York, which I shall tentatively schedule on my calendar for January 4.

As always, I was glad to hear your voice this morning. With kindest personal regards and best wishes for a Merry Christmas and Happy New Year,

Sincerely yours,

Arthur J. Vorwald, M.D.
Director

AJV:LB
Enc.

J-7

C06894

A18

OWENS-CORNING FIBERGLAS CORPORATION

INTRA-COMPANY CORRESPONDENCE

General Offices - Toledo, Ohio
January 7, 1942

Attention of: Mr. E. J. Marshall

cc: Mr. Harold Boeschenstein
Mr. W. P. Zimmerman

Subject: ASBESTOS WORKERS UNION

Review of 1941

1. The issue was joined.
2. Our health story was drawn together.
3. Our strategy has been to attack each situation locally.

Immediate Developments Ahead for 1942

1. Siebert article to appear in January issue of INDUSTRIAL MEDICINE.
2. Gardner article in preparation for JOURNAL OF INDUSTRIAL HYGIENE.
3. Sulzberger program under way.

Strategy for 1942

Should it not be to take the offensive?

The following plan is suggested:

1. Gather as a weapon-in-reserve an impressive file of photostats of medical literature on asbestosis. Available are two bibliographies covering medical literature to 1938, citing references to scores of publications in which the lung and skin hazards of asbestos are discussed. This file would cover five or six hundred pages, which can be microphotographed in the library of the Surgeon General in Washington or in some other medical library.

2. (a) Explore through Aetna the feasibility of working out a plan whereby our products liability coverage could be extended to all members of the Asbestos Workers Union wherever and whenever they are handling Fiberglas products. This would involve some 2,000 workers all of whom would have to be X-rayed (X-rays to be interpreted at Trudeau by Dr. Sampson), and in all probability given a physical examination before group coverage became effective on a set date.

   (b) If feasible, approach Union leaders with offer presenting plan as follows:

      (1) A demonstration of Owens-Corning's willingness to work

PLAINTIFF'S EXHIBIT 66

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A19

OWENS-CORNING FIBERGLAS CORPORATION

INTRA-COMPANY CORRESPONDENCE

General Offices - Toledo, Ohio

Attention of

Subject

with A.F. of L. Union Labor.

(2) A means of extending to Union members on construction jobs the same kind of insurance protection and industrial hygiene precautions now available to workers in private industry.

(3) An irrefutable demonstration of the willingness of a reputable commercial insurance company to underwrite such a risk and of the manufacturer to carry the premium cost.

(c) If reaction is favorable, arrange for Aetna representatives to present plan at Union district conferences and/or meetings of locals or at general convention of Union in fall of 1942. Presentation to be accompanied by distribution of Aetna leaflets explaining the insurance and telling the Fiberglas health story.

(d) If reaction is unfavorable, use the asbestosis weapon-in-reserve to let them stew. We may be sure that word of the proposal will reach competition and may give us a lever with which to go direct to the locals under the union's professed law of local autonomy. This procedure may provide an opportunity to promote dissension in the ranks that conceivably could bring about over-throw of the present Union leadership.

(e) The proposal can be exploited publicity-wise to our advantage either way.

If the reaction is favorable, it is a step unprecedented in the industry, taken by a manufacturer so confident of the absence of unusual hazard in his materials that he has joined forces with Aetna - at a rate more favorable than could be granted to any competitive manufacturer - to protect and reassure labor and assume liability attaching to any occupational hazard attributable to his materials.

If the reaction is unfavorable, the way is opened to spread word among the locals about the refusal of the Union officials to make this protection available to the members and to play all the stops on asbestosis. Implied is the threat to distribute to all members of the Union copies of the U.S. Public Health Bulletin #241 on Asbestosis.

Principal advantages of the whole plan are that it would take out of the realm of rumor and gossip all the stories of injuries that are floating around, would enable us to document such stories, and would enable us to take the offensive in telling the health story of Fiberglas where it would do the most good.

See

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A20

Reprinted, with additions, from the A. M. A. Archives of Industrial Hygiene and Occupational Medicine, January 1951, Vol. 3, pp. 1-43.

EXPERIMENTAL STUDIES OF ASBESTOSIS

ARTHUR J. VORWALD, Ph.D.(Path.), M.D.
THOMAS M. DURKAN
AND
PHILIP C. PRATT, M.D.
SARANAC LAKE, N. Y.

ASBESTOSIS is a form of pneumonoconiosis resulting from prolonged inhalation of asbestos dust. The name "asbestos," literally "unburnable," is not that of a specific mineral but is a term applied to a number of different minerals whose characteristic feature is a structure composed of long, parallel, flexible fibers. This structure is unique because the fibers are capable of repeated longitudinal subdivision to units of molecular proportions. In length the fibers vary from a few microns to 6 or more inches (15 or more cm.). Some varieties are stiffer than others, but many are sufficiently flexible to be spun into yarn and woven on modified textile machinery.

The asbestos minerals are silicates of variable composition and belong to the serpentine and the amphibole groups. Listed below are the more common varieties.

Amphibole group: actinolite, amosite, anthophyllite, crocidolite and tremolite.

Serpentine group: chrysotile.

The bulk of the asbestos of commerce is chrysotile, $3MgO.2SiO_2.2H_2O$, which is mined on this continent principally in the Thetford region of the Province of Quebec, Canada, and in Vermont. Crocidolite and amosite also are used commercially but in much smaller amounts. Chrysotile occurs as veins in serpentine, a mineral of similar chemical composition, which exists in massive form and is made up of microscopic fibers without the parallel orientation characteristic of chrysotile. The massive, bluish black serpentine, which is smooth and soapy to the touch, is traversed by veins of fibrous chrysotile varying in width from a barely perceptible line to 6 (15 cm.) or more inches. The fibers run across the vein and not lengthwise with the formation.

From the Saranac Laboratory of the Edward L. Trudeau Foundation.

This series of studies of asbestosis, initiated at the Saranac Laboratory more than twenty years ago by the late Dr. Leroy U. Gardner, director of the laboratory, was nearly completed at the time of his death in October 1946. Although partial reports and informal reviews of some of the experiments had been given from time to time by Dr. Gardner, this paper presents for the first time a complete survey of the entire experimental investigation.

EX. C

C06641

Attention is directed to the mineral brucite, $Mg(OH)_2$, which is often found in the same formations with serpentine and chrysotile and may be fibrous in structure. Except for the manufacture of magnesium, brucite has no commercial value at present because its fibers are not sufficiently flexible to be used in textiles, but they are capable of repeated longitudinal subdivision. Unlike other asbestiform minerals, brucite is not a silicate, and for this reason it has been a valuable tool in an experimental evaluation of the action of fibrous minerals on lung tissue.

EXPERIMENTAL ASBESTOSIS

For many years studies[1] have been carried on at the Saranac Laboratory in an investigation of the cause, nature and development of asbestosis. The present paper is devoted to experimental asbestosis,

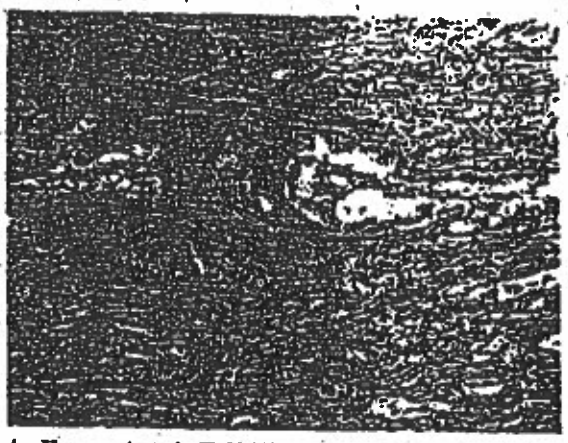

Fig. 1.—Human asbestosis (P-36-144). The photomicrograph reveals a bronchiole (right center) with a smooth muscle bundle at its inferior margin and with an extensive zone of collagen deposition largely obliterating the surrounding alveolar structure. The black foci are macrophages containing incidental pigment. Asbestosis bodies are present but are not apparent at this magnification (× 200).

and in it are described the experiments made on animals with various kinds of asbestos dust. Another report, to be prepared and issued at a future date, will be concerned with human asbestosis and will cover the health aspects of workers who have been exposed to asbestos dust in an industrial environment.

Although in man asbestosis is a chronic disease with diffuse pulmonary fibrosis which requires years to develop, it is possible to reproduce

1. (*a*) Gardner, L. U., and Cummings, D. E.: Studies on Experimental Pneumoconiosis: VI. Inhalation of Asbestos Dust: Its Effect upon Primary Tuberculous Infection, J. Indust. Hyg. 13:65 and 97, 1931. (*b*) Gardner, L. U.: Chrysotile Asbestos as an Indicator of Subtile Differences in Animal Tissues. Am. Rev. Tuberc. 46:762, 1942.

in one or more species of animal characteristic tissue changes which are similar to the lesions of human asbestosis (fig. 1). Since the life span of the experimental animal is relatively short, it is not possible to produce the characteristic lesions in animals under conditions identical with the usual industrial environment. Consequently, to obtain a complete evaluation of the tissue response to inhaled particulate and fibrous material, it is necessary to accelerate the reaction by employing higher concentrations of dust than would ordinarily be encountered in industry. While conditions of exposure are thus different, the information yielded by animal experiments is invaluable in furnishing a better understanding of the reaction of the human organism to inhaled asbestos dust.

Experimental Methods

For investigating the tissue reactions of experimental animals to the various asbestos minerals, two types of technic have been employed, namely, the inhalation method and the injection method. In inhalation experiments, groups of animals—up to 100 or more guinea pigs and sometimes smaller numbers of rabbits, cats, dogs, rats or mice—are kept for eight hours a day in a cubical dust room, 8 ft. (2.5 M.) in dimension, in which a cloud of asbestos dust is maintained by a rotating paddle in a dust hopper.[13] At intervals during the experiment a few animals are killed and the tissues examined to determine the nature and the extent of the dust reaction. Some animals are exposed for periods up to three years. The injection experiments are used to determine in as short a time as possible whether or not a particular dust has a potential capacity to produce inflammatory reaction when in direct contact with tissues of the body. The method involves injecting the dust, either dry or suspended in fluid, into the animal by the intravenous, the intraperitoneal, the intratracheal or another route.

Long term inhalation experiments furnish information on which great reliance is placed when estimating the degree to which a dust might constitute a respiratory hazard to industrial workers. Even though an atmospheric dust may be potentially dangerous, as indicated by injection experiments, only inhalation procedures will reveal whether the dust can be inhaled, pass the natural defense barriers of the body and reach the pulmonary tissue in quantities sufficient to cause damage. Injection methods are useful, however, because they make certain that contact occurs between the dust particles and tissues and because they allow accurate estimation of the dosage and of the potential capacity of that dose to produce reaction. The intratracheal method is particularly valuable when one is dealing with fibrous minerals like asbestos, since it permits observation of the effect of the fibers on pulmonary tissue.

Tissue Susceptibility

Unlike free silica, asbestos does not produce specific effects in all organs of all species of animals. The comparative data presented in table 1 are based on completed observations and therefore differ slightly from a preliminary report.[14] Fine quartz introduced into various organs

of various animals (guinea pig, rabbit, rat, mouse, cat, dog, chicken and even tadpole) eventually will produce silicotic nodules but at different rates. Similar introduction of long fiber asbestos has resulted in a fibrous reaction in the lung and, to a lesser extent, in the peritoneum but not in other organs of the guinea pig, the rabbit, the cat and the white rat. In our experience the lungs of the dog and the white mouse failed to respond with fibrosis, although Schuster [2] has reported such changes in a dog that lived in an asbestos-fabricating plant. This variation in species and in organ susceptibility is yet to be accounted for [3]; it is presumed that in the susceptible animals the greater reaction of the lung to asbestos, far exceeding the reaction of other organ tissues, is due principally to the greater mobility of the lung.

Peculiar Characteristics of Asbestos

Experience has demonstrated that most of the nonfibrous dust particles inhaled into the lungs of man and animal are 10 microns or less

Table 1.—Reaction to Long Fiber Chrysotile in Lungs of Man and Other Species of Animal

Species	Mode of Exposure	Fibrosis *	Asbestosis Bodies
Man	Inhalation	4+	Numerous
Guinea pig	Inhalation and injection	2+	Moderately numerous
Rabbit	Inhalation and injection	+	Rare and atypical
Cat	Inhalation and injection	+	Rare and atypical
White rat	Inhalation and injection	+	Very rare
White mouse	Inhalation	0	Rare and atypical
Dog	Injection	0	None

* The symbols 0 to 4+ refer to the degree of tissue reaction.

in maximum dimension. Larger particles apparently do not gain access to the lungs, because, first, large particles settle in air so rapidly that few remain suspended in the atmosphere breathed and, second, large particles are more effectively removed by the protective mechanisms of the upper respiratory tract. In the case of fibrous materials these factors have less influence and fibers 100 and even 200 microns in length have been found in the terminal air spaces of human lungs. In small laboratory animals exposed to asbestos dust the maximum length of fiber found in the lung rarely exceeds 60 microns.

A large proportion of nonfibrous particulate dust inhaled into the lung is found in the terminal air spaces (alveolar ducts, atriums, alveoli) in all parts of the organ; in contrast, inhaled asbestos fibers are first discovered in the respiratory bronchioles. These small passages are immediately distal to bronchioles lined by ciliated epithelium.[4] Their

2. Schuster, N. H.: Pulmonary Asbestosis in a Dog, J. Path. & Bact. 36 (pt. 2):751, 1931.

3. Vorwald, A. J.: Variations in Individual Susceptibility to Industrial Dusts Inhaled into the Lungs, Am. Rev. Tubere. 62: (IB) 13, 1950.

4. Miller, W. S.: The Lung, Springfield, Ill., Charles C Thomas, Publisher, 1937.

own essential lining is a low cuboidal type of epithelium but, as their name implies, they actually function in respiration through lateral alveoli distributed along their walls. Either these alveoli or the abrupt change in the character of the lining epithelium, or the small diameter of the respiratory bronchiole, or the combination of all three factors is responsible for retention of the fiber at this site. Only after asbestosis is well established are appreciable numbers of fibers seen in the more peripheral air spaces. Further explanation is required to clarify this observation.

Rate of Tissue Reaction to Asbestos Fibers

The affected tissues react much more rapidly to asbestos than to quartz dust. For example, in rats receiving asbestos fibers by intratracheal injection fibrosis of a characteristic type is visible as early as one month after injection; for quartz dust the latent period is two months or more. Thus, the development of nodular fibrosis due to inhaled silica lags behind the deposition of dust to a greater extent than does the evolution of the diffuse reaction to asbestos. This results in a difference in the degree of progression which follows termination of exposure to dust. For example, on discontinuance of exposure the nodules of silicosis become larger, to a limited extent, for a considerable period of time, whereas the fibrosis of asbestosis increases for only a short time. Subsequently, the asbestotic fibrous tissue contracts; this process often distorts the adjacent pulmonary tissue and may, as a result, progressively interfere with cardiorespiratory function.

Asbestosis Bodies

The peculiar structure known as the asbestosis body or "curious body" is a specific concomitant of asbestosis.[5] The typical body is a golden yellow, beaded or haustrated rod, which may be either straight or curved (fig. 2). Often one or both ends are bulbous like a dumbbell. The bodies vary considerably in length, and dimensions up to 250 microns have been recorded.

It is believed that asbestosis bodies are inhaled fibers on which protein and iron pigment of tissue origin have been deposited.[6] Gloyne[5b] observed reproduction of these bodies in guinea pigs nine months after subcutaneous injection of fibers rendered free of iron. The bodies are abundant in man and in the guinea pig (table 1) but are much larger in the former, probably because the larger-sized air passages admit fibers of greater dimension. In guinea pigs they form after about 70 days

5. Gloyne, S. R.: (a) The Formation of the Asbestosis Body in the Lung, Tubercle 12:398, 1931; (b) The Asbestosis Body, Lancet 1:1351, 1932. (c) Gardner and Cummings.[5a]

6. Lynch, K. M., and Smith, W. A.: Asbestosis Bodies in Sputum and Lung, J. A. M. A. 95:659 (Aug. 30) 1930. Simson, F. W., and Strachan, A. S.: Asbestosis Bodies in the Sputum: A Study of Specimens from 30 Workers in an Asbestos Mill, J. Path. & Bact. 34:1, 1931. Gardner and Cummings.[5a] Gardner.[5b] Gloyne.[5a, b]

of contact with the tissue. In cats, rabbits and mice a few of the fibers show an atypical coating after much longer residence in the lungs. In rats the bodies are rarely seen, and in dogs none could be found. Although the evidence is incomplete, it appears that the formation of the asbestosis body prevents the fiber from damaging the tissue. Many of the points mentioned above will be elaborated on in subsequent para-

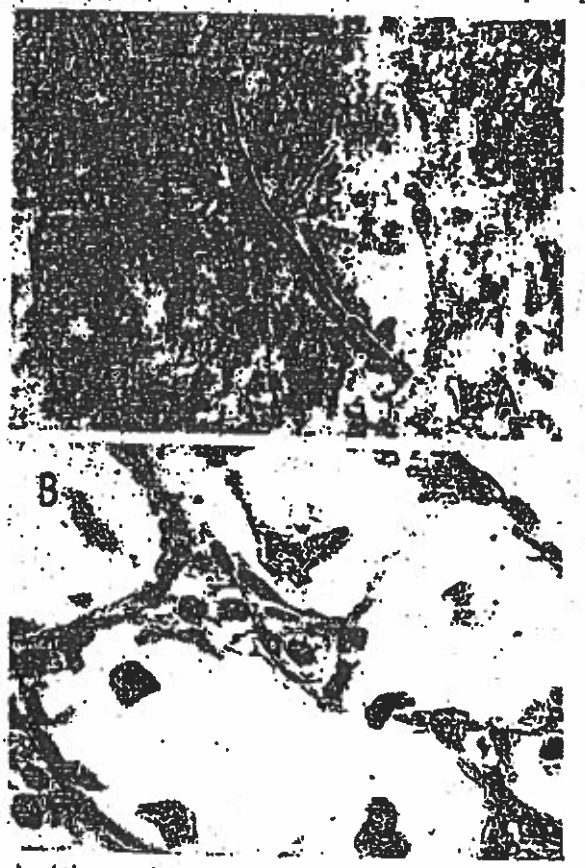

Fig. 3.—*A*, human asbestosis bodies. This collection of asbestosis bodies was found in the lung shown in figure 1. The usual variations of size and configuration are represented (× 400).

B, guinea pig asbestosis body. This one is similar to some of those shown in *A* (× 400).

graphs dealing with the actual experiments. For presentation our investigation is divided into two sections, one dealing with inhalation experiments and the other with injection experiments.

C.C6646

INHALATION EXPERIMENTS

Four large scale inhalation experiments have been conducted in this laboratory with various forms of asbestos dust. In each of these investigations, more than 160 animals were used, and the experiments were carried on for periods ranging from two to more than five years. The four kinds of asbestos dust employed are designated as King's floats, short fiber, 100 per cent ball-milled, and long fiber asbestos dust.

KING'S FLOATS ASBESTOS DUST

The first inhalation experiment conducted at the Saranac Laboratory with asbestos dust was begun in 1928. Animals inhaled the dust for

TABLE 2.—*Chemical Analysis of Asbestos Dusting Materials*

Type of Asbestos	SiO_2	Fe_2O_3	Al_2O_3	Cr_2O_3	MgO	CaO	MgO	Na_2O	K_2O	CO_2	Ignition Loss	Total
King's floats.....	39.82	6.84		*	*	0.07	37.36	*	*	*	12.73	97.12
Short fiber.....	37.17	9.09	1.40	0.34	0.69	0.85	38.96	0.16	0.09	0.88	11.00	100.73
Long fiber.....	40.60	5.82	0.78	*	0.09	0.31	40.34	0.06	0.06	0.07	11.00	99.78

* Not determined.

TABLE 3.—*Petrographic Analysis of Asbestos Dusting Materials*

King's floats *: The approximate composition, based on particles (except chrysotile) smaller than 10 microns and reported as percentages obtained from particle counts, was chrysotile 14, serpentine 69, magnetite 12, carbonates 10, talc 12, other minerals 4. Few chrysotile fibers up to 200 microns long were included.

Short fiber †: The material, before being ball milled, contained a preponderance of fibrous chrysotile and platy (nonfibrous) serpentine. The approximate composition, by percentage, was chrysotile 17, serpentine 58, magnetite 10, quartz 2, brucite 2, other minerals, including dolomite, actinolite and tremolite, 11.

Long fiber †: The material consisted principally of the fibrous asbestos mineral chrysotile. Shreds of nonseparated fibers 5 to 15 microns in diameter and up to 80 microns in length were present. The approximate composition, by percentage, was chrysotile 73, serpentine 16, magnetite 5, brucite 2, other minerals, among which were calcite and chloritic and pleonectic minerals, 3. Only a trace of quartz was observed.

* The analysis of the King's floats asbestos, made by Dr. C. S. Hurlbut Jr., of Harvard University, has been reported elsewhere (Hurlbut, C. S., Jr., and Williams, C. K.: The Mineralogy of Asbestos Dust, J. Indust. Hyg. & Toxicol. 17: 201, 1935).

† For the short fiber asbestos and the long fiber asbestos the petrographic analysis was supplemented with x-ray diffraction examination.

periods up to 33 months. Some guinea pigs with six and nine months' exposure lived for an additional three years after cessation of their exposure. A preliminary report [18] presented observations after 29 months of exposure. At that time observations covered a period of only 2¼ years and the conclusions as to the ultimate effects of inhaled asbestos dust were provisional. Those conclusions are substantiated by results of the completed study, which is reported as follows.

Composition and Atmospheric Concentration of the Dust.—The dusting material, a commercial variety of asbestos known as King's floats, was composed of short fibers, ranging in length from 1 mm. to 1 micron or less, and of particles which also varied in size. It was obtained from the Thetford, Quebec, plant of the Asbestos Corporation of America, and analyses (tables 2 and 3) reveal that the amount of fibrous chrysotile was only 14 per cent, a rather low value.

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Impinger samples taken soon after the experiment was started indicated that the dust concentration was at first quite low, the average dust count being only 6.0 million particles per cubic foot of air by the standard light field technic and 0.8 million for particles and fibers greater than 10 microns. After the inhalation experiment had been under way for about two years, the speed of the rotating paddle in the dusting machine was increased and for the remaining 10 months of the experiment considerably more dust was dispersed into the atmosphere. The average dust count of impinger samples collected after this change was 93.7 million by the usual light field method and 1.5 million for particles and fibers larger than 10 microns. It is probable, however, that the true values of the dust concentration were higher than the counts given in this paragraph. The impinger samples for the King's floats experiment were collected in water, but later studies[7] have shown that counts of impinger samples of asbestos dust taken in water are not reliable. Ethyl alcohol instead of water was used as the collecting fluid in all subsequent experiments.

TABLE 4.—*Summary of Inhalation Experiment with King's Floats Asbestos Dust*

Nature of Experiment	Animals	Maximum Dust Exposure, Mo.	Maximum Survival After Dust Exposure, Mo.	Results
Dust exposure continuous throughout life	44 guinea pigs	33	0	Typical peribronchiolar fibrosis after 10 months
	6 rabbits	10	0	Foreign body bronchitis
	12 rats	6	0	Little or no reaction
Dust exposure followed by prolonged residence in normal air	28 guinea pigs	6	28	Nonprogressive fibrosis
	28 guinea pigs	6	27	Nonprogressive fibrosis
	1 rabbit	6	30	
	1 rabbit	10	24	Absorption of foreign body reaction
Tuberculous infection * at start of dust exposure	40 guinea pigs	33	0	Temporary progression of infection, followed by healing with fibrosis
Controls to infection: no dust exposure	33 guinea pigs	0	25 †	Healing by resolution (one exception)
Tuberculous infection * after 28 mo. of dust exposure, then residence in normal air	12 guinea pigs	28	14	No appreciable increase in susceptibility to tuberculous infection; healing with fibrosis
Controls to infection: no dust exposure	12 guinea pigs	0	12 †	Healing by resolution

* The guinea pigs were infected with low virulence R1 strain of tubercle bacillus.
† This means the survival period following infection.

Results of the investigation, briefly summarized in table 4, show that inhalation of King's floats asbestos dust produced a typical peribronchiolar fibrosis in guinea pigs but not in rabbits or rats.

Reaction in Normal Guinea Pigs.—Guinea pigs inhaling this dust for periods up to 33 months had a characteristic fibrosis occurring in conical patches about the respiratory bronchioles. During this exposure the peripheral alveoli were not involved. The particulate elements of the dust were transported through the lymphatic system to the bronchial nodes, causing no significant reaction in either site; the fibrous elements remained fixed at the points of original localization and were seldom detected in the lymph nodes.

After exposure of approximately a year a small amount of cellular reaction had been produced about many respiratory bronchioles (fig. 3 *A*). As more dust was inhaled, it continued to accumulate in the same location, and later stages of the disease (fig. 3 *B*) consisted of extensions of the original lesions.

Apparently, the inhaled fibers were caught in the pocket-like alveoli that are given off from the lateral walls of the respiratory bronchioles. There they

7. Fulton, W. B.; Houtz, R. L.; Dooley, A., and Mathews, J. L.: Asbestosis: I. The Collection and Counting of Asbestos Dust Encountered in Asbestos Fabricating Plants, Special Bulletin 37, Pennsylvania Department of Labor and Industry, Harrisburg, 1934.

were phagocytosed, and many of them were carried into the wall by migratory cells. Mononuclear leukocytes attracted to the area caused an appreciable thickening of the bronchiolar wall. After 16 months a delicate fibrosis made its appearance. The process evolved gradually, and the number of fine intercellular collagenous fibers steadily increased. As this fibrous deposit contracted, it partially closed and

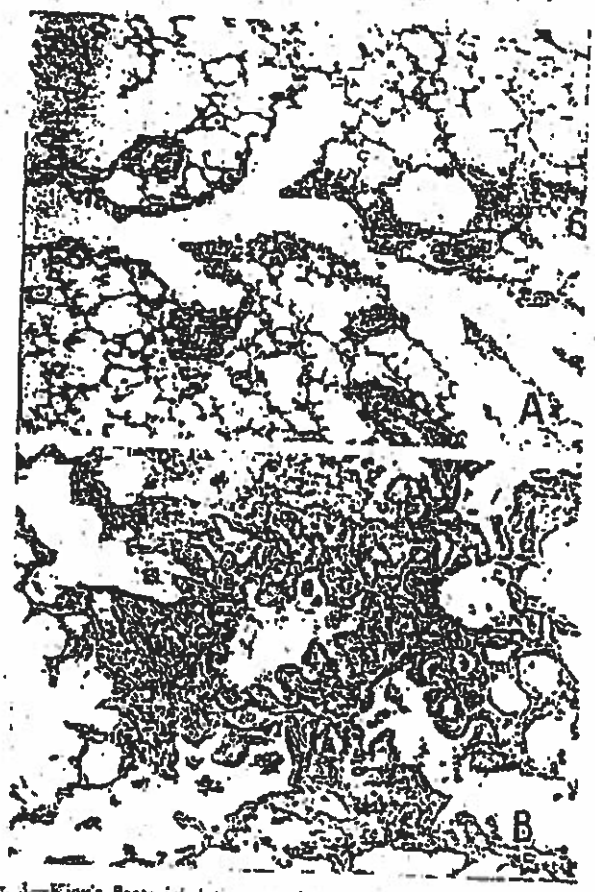

Fig. 3.—King's floats inhalation experiment: *A*, lung of a guinea pig with 12 months' exposure. It includes a respiratory bronchiole, at the left, branching and becoming an alveolar duct, at the right. Note the accumulation of cells in the wall of the bronchiole and in adjacent alveoli (× 130). *B*, lung of a guinea pig with 23 months' exposure. The field includes a bronchiole, at the center, with peribronchial fibrosis extending into the walls of adjacent alveoli. Note the cuboidal epithelium lining these alveoli. This is the so-called "adenomatoid" appearance (× 200).

distorted the alveoli, and with this change the alveoli became lined with cuboidal cells. The result was an adenoma like appearance which frequently accompanies

chronic pulmonary inflammation resulting from many causes. Willis[8] described a similar structure in the lungs of guinea pigs inhaling silicon carbide. The longer asbestos exposures resulted only in more thickening of the walls of the air spaces, largely due to an increase in the amount of fibrosis. The fibrous tissue always remained cellular and failed to show the hyalinization characteristic of silicosis.

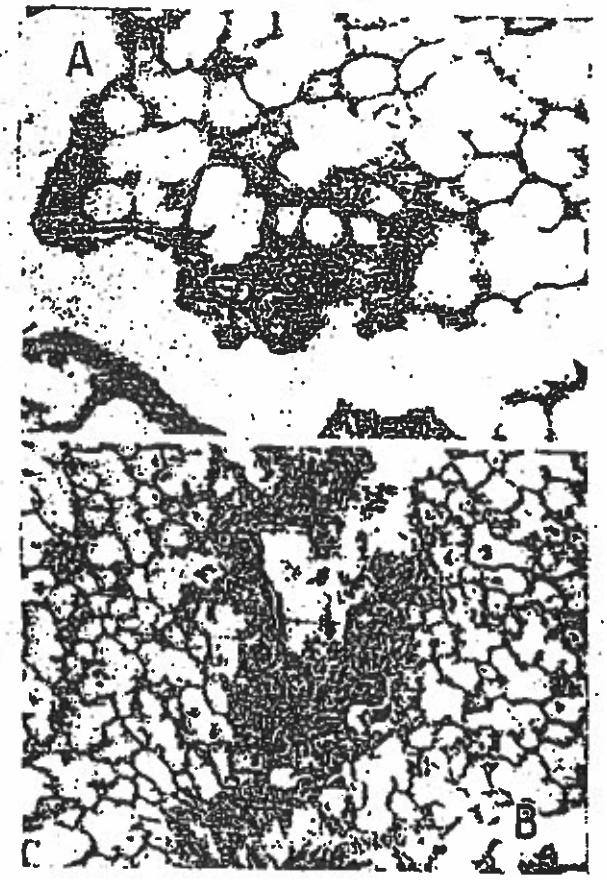

Fig. 4.—King's floats inhalation experiment: *A*, lung of a guinea pig with six months' dust exposure followed by 35 months' inhalation of normal air. The reaction is rather slight, but distinct fibrosis is present (× 200). Note that 23 months of continuous exposure (fig. 3 *B*) produces much more extensive reaction.

B, lung of a guinea pig exposed to the asbestos dust for nine months and living thereafter in normal air for 37 months. The reaction shown is more than that in *A* but much less than the reaction in figure 3 *B* (× 200).

8. Willis, H. S., and Brutsaert, P.: Tumor-like Structures in the Lungs of Guinea Pigs Artificially Exposed to Silica Dust, Am. Rev. Tuberc. 17:268, 1928.

Asbestosis bodies (fig. 2 *B*), first seen in the lungs of the guinea pigs that had inhaled dust for about two months, became more numerous and more distinctly segmented with increasing exposure.

The reaction produced in guinea pigs exposed for six and nine months did not progress significantly during a subsequent period of 35 and 37 months when the animals lived in a normal atmosphere (fig. 4). Between eight and 11 months after exposure ceased, the cellular reaction in the lung had been completely replaced by thin strands of fibrous tissue. At later periods the scar tissue was less in amount, but in the last animal killed, 37 months after discontinuing dust exposure, some fibrosis was still visible.

Reaction in Guinea Pigs Infected with Tubercle Bacilli at the Onset of Dust Inhalation.—Of the group of 40 guinea pigs infected with attenuated tubercle bacilli, R[9] strain,[9] at the time that dust exposure was begun, 31 died or were killed before the completion of two years of the exposure and were reported in the paper by Gardner and Cummings.[1a] Seventeen of these died from intercurrent pneumonia. Briefly, the results were as follows: Ten revealed some evidence of spread of the tuberculous process (fig. 5 *A*); in 6 of these it was confined to the lungs, and in the other 4 the abdominal viscera also were involved. Extension of the infection was first seen after seven months of dust inhalation; during the next 20 months more than half of the animals showed actively spreading tuberculosis, and in 3 of them small cavities had developed. During the last eight months no animals exhibited any evidence of active infection although in half of them the healed fibrous scars of previous spreads were obvious. The scars were more extensive than is characteristic of either tuberculosis or asbestosis alone.

The nine animals which were still alive after two years of dust exposure were killed at intervals during the following year. In four of them the primary foci of infection were healed with fibrosis and even calcification, and there was no evidence of progression (fig. 5 *B*). In the remaining five the tuberculous foci showed evidence of having previously spread locally; in four of them, by the time of autopsy, the foci were healed, with excessive fibrosis; in the fifth animal there was a generalized chronic tuberculous pneumonia in one lobe, and in the other lobes there were isolated primary tubercles, which were still active but had not spread.

Reaction in Guinea Pigs Infected with Tubercle Bacilli After Establishment of Asbestosis.—Twelve guinea pigs, after inhaling King's floats asbestos dust for 26 months, were infected with tubercle bacilli and then removed to normal air. Six of these animals died within seven weeks, five from intercurrent nontuberculous infection. The remaining six animals were killed at intervals up to 14 months after infection. The subpleural tubercles were no more numerous in the dusted animals than in the nondusted controls, but a considerable number were found in the depths of the lung about foci of asbestosis. The tuberculous component of the combined reaction showed only slight local extension about lesions in the lungs and tracheobronchial lymph nodes. Caseation was found in tubercles 1½ months old, but by 5½ months it had completely disappeared, leaving only scar tissue. Foci of fibrosis still persisted in the last animal, which was killed 14 months after infection.

Reaction in Rabbits.—Rabbits exposed to the asbestos dust for periods up to 19 months showed a foreign body type of reaction of low grade, but no fibrosis. Although their lungs contained particulate elements of the dust, fibers were not present, indicating that the upper respiratory mechanism of the rabbit is adequate to exclude fibrous foreign bodies. Two rabbits, after inhaling dust for six and 19

9. Steenken, W., Jr., and Gardner, L. U.: R Strain of Tubercle Bacillus; Its Dissociation and Virulence of Variants in Normal and Silicotic Guinea Pigs, Am. Rev. Tuberc. 54:51, 1946.

months, lived in normal air for more than two years. At autopsy neither animal showed any evidence of cellular reaction or fibrosis in the terminal bronchioles, nor were there any asbestosis bodies.

Reaction in White Rats.—All the rats had acquired an infection, resulting in the formation of pulmonary abscesses, before they came to autopsy. Apparently, so much heavy mucus obstructed their bronchi that very few fibers could have entered

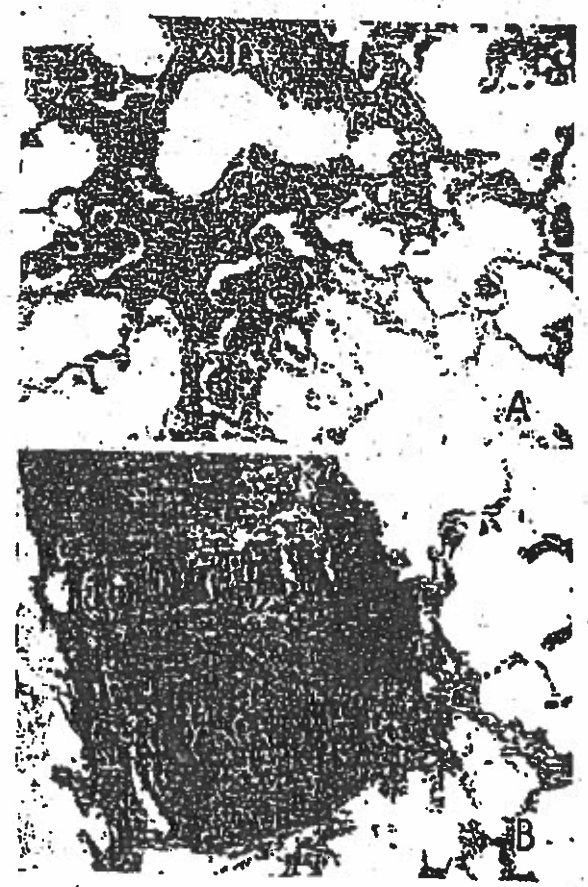

Fig. 5.—King's float inhalation experiment: *A*, lung of guinea pig infected with R_1 tubercle bacilli and then exposed to dust for 24 months. A bronchiole is shown just above center. Surrounding it is some collagen deposition, together with typical epithelioid cell infiltration of the wall. Note the lack of encapsulation and the peripheral, epithelioid cell pneumonia, which illustrate a spreading tuberculous process (× 200).

B, lung of a guinea pig infected with R_1 tubercle bacilli and then exposed to dust for 33 months. Note the subpleural distinctly encapsulated caseous focus, the calcification at the right border of the lesion and the absence of cells in adjacent alveoli, all of which illustrate a healing tuberculous process (× 200).

their lungs. In a few of the rats, an occasional asbestosis body was discovered, but there was no fibrosis. This phase of the experiment was considered unsuccessful.

Summary and Interpretation of Inhalation Experiment with King's Floats Dust.—The findings in the experiment with King's floats dust can be summarized under two headings:

1. Effect of the inhaled dust on normal animals. The King's floats dust caused a characteristic peribronchiolar fibrosis in guinea pigs but not in rabbits or rats. The fibrosis did not increase significantly in extent after the dust exposure was discontinued.

2. Effect of the inhaled dust on tuberculosis in guinea pigs. In guinea pigs infected with attenuated tubercle bacilli and then placed in the dust room, the results were more variable than is usual in an experiment of this type. A few animals showed no sign of progression of the infection; in most of them there was evidence of temporary progression with subsequent healing; in one animal the tuberculous process remained active to death. In contrast, when guinea pigs after being infected are exposed to quartz dust instead of asbestos dust, the infectious process continues to progress and eventually causes the death of the animals. On the other hand, infected animals exposed to a harmless dust like iron oxide do not show any progression of the infection.[10] Guinea pigs infected with attenuated tubercle bacilli after the termination of two years' asbestos dust exposure did not show progressive disease. The only modification of the infection was in its localization, a few bacilli being retained in the peribronchiolar fibrous tissue, with tubercles forming there in addition to the usual tubercles beneath the pleura.

In view of the variability of the results, the unusual nature of the response and the high proportion of deaths due to intercurrent pneumonia, it is felt that only tentative conclusions as to the influence of asbestos dust on the course of tuberculous infection are justified by this experiment.

Short Fiber Asbestos Dust

Since hazardous dusts like quartz are most effective in producing fibrosis when the particles are 3 microns and less in size, an inhalation experiment was performed to determine whether this condition is true for asbestos dust. It was thought that a short fiber asbestos dust consisting almost entirely of fibers and particles smaller than 3 microns would initiate an accelerated tissue response and produce an advanced reaction in a shorter time than did the King's floats dust, which contained fibers from 1 mm. to 1 micron and less in length as well as much particulate matter.

Composition and Atmospheric Concentration of the Dust.—The dusting material for this experiment was the remains of fibers collected in dust bins of an asbestos-fabricating plant after a carding operation and screened to pass 200 mesh. Since

10. Vorwald, A. J.; Pratt, P. C.; Durkan, T. M.; Delahant, A. B., and Bailey, D. A.: Siderosis: A Benign Pneumoconiosis Due to the Inhalation of Iron Dust, Indust. Med. & Surg. 19:170, 1950.

the material as received contained many long fibers, it was ground in a steel ball mill to reduce practically all the particles to 3 microns or less in size. When used alone in the standard dusting machine, this finely ground asbestos tended to pack in the hopper, and it became necessary to mix one volume of the unground material with three volumes of the ground to generate a satisfactory dust cloud. It is pertinent to mention here that the addition of the small quantity of unground asbestos was unfortunate, because it confused the interpretation of results.

The composition of the short fiber asbestos as received is disclosed by the chemical and petrographic analyses given in tables 2 and 3. Samples taken before and after grinding yielded about the same values on analysis, indicating that there was no contamination from the mill or loss of water content.

The dust concentration varied during the experiment, the light field counts for atmospheric samples collected inside the animal cages with the impinger apparatus ranging from 83 million to 182 million. The average of counts was 130 million for the first year of the experiment, 134 million for the second year and 140 million for the third year.

Size-frequency measurements of air-floated dust from inside the cages at a magnification of 1,300 × revealed a great preponderance of fine particles, nearly

TABLE 5.—*Summary of Inhalation Experiment with Short Fiber Asbestos Dust*

Nature of Experiment	Animals	Maximum Dust Exposure, Mo.	Maximum Survival After Dust Exposure, Mo.	Results
Dust exposure continuous throughout life	48 guinea pigs	34	0	Rate of reaction about the same as in experiment with King's floats asbestos but extent of involvement very much less †
	12 rats	22	0	Characteristic patches of peribronchiolar fibrosis; no asbestosis bodies
	13 cats	44 *	0	Subpleural reaction only
	7 rabbits	42 *	0	No fibrosis seen grossly; microscopic evidence of alveolar wall thickening after 40 months' exposure
Dust exposure followed by prolonged residence in normal air	13 guinea pigs	20	44	Progression after removal from dust doubtful—neither clearly established nor definitely excluded
	2 cats	21	74	Same as for continuous exposure
	1 rabbit	42 *	0	Similar to continuous exposure; evidence of slight regression

* After 22 months the animals were exposed to 100 per cent ball-milled asbestos.
† The reaction was probably due to long fibers in the unground material which was mixed with the ground asbestos dust to produce a satisfactory dust cloud.

90 per cent of the particles seen being smaller than 3 microns. It was estimated that approximately 1 per cent of the dust was in the form of fibers greater than 10 microns in length.

Four species of animals—guinea pigs, white rats, cats and rabbits—were used in this experiment. The results of the dust exposure, summarized in table 5, are presented in greater detail below.

Reaction in Guinea Pigs.—Eighty guinea pigs were originally placed in the dust room, but 21 of them were later eliminated from the experiment and killed because of enlargement of the cervical lymph nodes thought to be due to intercurrent infection of the upper respiratory tract. Of the other 59 animals, 46 remained in the dust room until they were killed or died at periods up to 34 months, and 13 animals were transferred to normal air after being exposed to the dust for 20 months.

The type of tissue reaction provoked by the inhaled short fiber asbestos was essentially the same as that already observed in the experiment with King's floats asbestos. The rate of reaction also was approximately the same, but the extent of involvement was very much less. After 16 to 24 months of exposure only a very few small foci of reaction, which generally required microscopic examination for detection, had been produced in the guinea pigs.

Only after exposures had continued for approximately one year was there an appreciable tendency for dust-containing phagocytes to gather into clumps. By 16 months phagocytes had collected about the walls of a few of the respiratory bronchioles which revealed a little proliferation or infiltration of mononuclear cells. There were also some multinucleated cells, but they were of the inert, foreign body type. At 20 to 24 months the cellular clumps were sometimes quite prominent, and sometimes changes in the epithelium resulted in the adenoma-like or "adenomatoid" appearance (fig. 3 *B*) previously described in the section reviewing the experiment with the King's floats dust. In most of the subsequent members of the series the reaction remained cellular, but a few exhibited pronounced development of fibrous tissue. In these few members of the series, the collagen was pale in color and tenuous, with no appearance of being hyalinized. Diffuse chronic pleurisy was prevalent in a few animals without evidence of pul-

TABLE 6.—*Analyses of Lungs of Guinea Pigs After Prolonged Inhalation of Short Fiber Asbestos Dust*

Exposure to Dust, Mo.	Period in Normal Air, Mo.	Amount of Ash, % of Dried Lung	Total SiO_2, % of Dried Lung	Total SiO_2, % of Ash	Tissue Reaction *
Dust Exposure Continuous During Life					
12	0	5.02 4.83 5.16	0.51 0.49 0.44	10.21 10.09 10.24	±
18	0	5.09 4.76 4.95	0.49 0.45 0.43	9.66 9.09 10.00	±
20	0	5.02 6.43	0.53 0.90	14.16 14.07	2+
24	0	5.12 5.50	0.78 0.78	14.16 14.20	3+
28	0	5.25 6.39	0.93 1.37	17.89 20.66	4+
31	0	5.06 6.25	0.78 0.86	12.87 15.13	4+
Dust Exposure Followed by Prolonged Residence in Normal Air					
20	4	5.16 5.13	0.45 0.80	9.29 7.36	2+
19	16	6.31 5.98	0.62 0.34	10.71 5.61	2+
20	14	4.77 5.28 4.77	0.43 0.35 0.22	9.31 6.69 4.69	2+

* The symbols averaging the tissue reaction in each group of guinea pigs represent merely the relative degree of reaction, ranging from ± (questionable) to 4+ (the maximum for this experiment). The relationships apply only within this table and cannot be compared with symbols in other tables.

monary infection. This suggests that pleurisy may be a specific concomitant of asbestosis, but the evidence is not adequate to establish this point. The reaction of the tracheobronchial lymph nodes was more pronounced than in the previous experiment with King's floats asbestos, probably because more fine particles had been transported to the nodes in animals inhaling short fiber asbestos. The nodal reaction was essentially an increase in reticulum, rather than a fibrosis, with the original cells being preserved between the thickened reticular fibers.

In the group removed to normal air after 20 months' inhalation of dust, progression of disease was not definitely demonstrated, but neither could it be absolutely disproved, owing to the variability of the response in different animals. The reactions, from mild to severe, occurred sporadically and bore no relationship to the length of time after cessation of exposure. The differences were attributed to variation in individual susceptibility. This view received support from the chemical analyses (table 6), which revealed comparable amounts of ash and silica in lungs with widely different amounts of tissue change. For example, the ash

and silica values were quite similar for three animals living in dust 20 months and then in normal air for 14 months, yet the tissue reaction was severe in one animal, mild in another and only doubtful in the third.

The formation of asbestosis bodies was at first extremely limited in both groups. After five months' exposure only a very rare short body could be found, usually inside a cell. Some of the finest intracellular particles were surrounded by yellow deposits having the same color as the asbestosis body. One year's exposure had permitted an accumulation of many longer fibers, a number of which were counted and seen as typical asbestosis bodies. Most of these were still short enough to be partially or entirely within phagocytic cells. By the twentieth month and thereafter they

TABLE 7.—*Analyses of Lungs of White Rats That Had Inhaled Short Fiber Asbestos Dust*

Duration of Exposure, Mo.	Amt. of Ash, % of Dried Lung	Total SiO_2, % of Dried Lung	Total SiO_2, % of Ash	Duration of Exposure, Mo.	Amt. of Ash, % of Dried Lung	Total SiO_2, % of Dried Lung	Total SiO_2, % of Ash
0*	2.9	0.00	0.0	6	3.3	0.07	2.1
	4.2	0.00	0.0		5.4	0.05	1.0
	2.6	0.00	0.0		3.7	0.04	1.1
	2.8	0.00	0.0				
	3.5	0.00	0.0				
	3.0	0.00	0.0		3.9	0.03	1.0
	3.4	0.00	0.0		3.8	0.10	2.8
				8	7.3	0.16	2.2
	3.5	0.05	1.4		4.4	0.17	4.0
2	5.6	0.12	2.2		3.7	0.16	4.2
	3.9	0.09	2.3				
	5.3	0.08	1.5				
					4.9	0.13	2.6
	3.3	0.08	2.3	10	4.6	0.13	2.9
4	7.5	0.11	1.5		5.3	0.13	2.5
	5.4	0.07	1.2		4.7	0.13	2.8

* Normal controls (no dust exposure).

TABLE 8.—*Average Values of Ash and Total Silica for Lungs of White Rats Inhaling Various Dusts for Various Periods (Lungs Only, Without Included Lymph Nodes)*

Duration of Exposure, Mo.	Amt. of Ash, % of Dried Lung				Total SiO_2, % of Dried Lung				Total SiO_2, % of Ash			
	Short Fiber Asbestos	Quartz	Ferruginous Chert	Gypsum-Quartz Mixture	Short Fiber Asbestos	Quartz	Ferruginous Chert	Gypsum-Quartz Mixture	Short Fiber Asbestos	Quartz	Ferruginous Chert	Gypsum-Quartz Mixture
2	3.3	4.3	5.3	2.9	0.09	0.01	0.15	0.05	2.9	11.7	3.0	2.6
4	7.4	4.5	5.6	3.5	0.09	0.21	0.32	0.07	2.5	11.4	5.8	2.0
8	3.5	7.1	9.0	3.4	0.05	2.94	1.48	0.11	1.3	41.5	24.4	3.4
9	9.8	4.6	9.0	3.6	0.13	1.44	1.40	0.25	2.3	29.4	26.5	9.1
10	4.9	7.5	14.1	4.1	0.15	6.10	6.09	0.23	3.2	59.8	43.2	5.7

were relatively numerous although still rare in comparison with the findings in the King's floats experiment.

Reaction in White Rats.—Seventy-three white rats were exposed to atmospheric short fiber asbestos dust for periods up to 32 months. During the first 10 months animals were killed bimonthly and for the remainder of the experiment at less frequent intervals. Up to eight months the dust cells were widely scattered and existed in foci only sporadically. Reaction was limited to occasional slight thickening of the septums about small accumulations of dust cells. At 10 months there was a suggestion of early fibrosis in a few rats, but the change was so slight that it would probably have been overlooked without the clump of dust cells which attracted attention to the area. Only 10 animals were exposed for from 12 to 32 months. In each of them the lungs contained minute foci of well defined fibrosis distributed like that of asbestosis but without asbestosis bodies. The lesions, visible only at a magnification of 150 diameters or more, consisted of patches along

alveolar ducts in which the walls of the associated air spaces were very thick, owing to swollen collagen framework. Connective tissue and Foot-Bielschowsky silver preparations revealed complete loss of capillary bed locally. Outside the collagen was a thin layer of epithelial cells. This did not resemble the "adenomatoid" change characteristic of guinea pig asbestosis. Near the lesions the air spaces were filled with phagocytes containing gray to yellow particulate dust and a rare, long, naked asbestos fiber. Careful search failed to reveal even a suggestion of an asbestosis body. Pleurisy was absent. The tracheobronchial nodes showed compact focal collections of monocytic cells at 12 months and, at 20 months, some diffuse thickening of the reticulum. In a few rats there was definite fibrosis along the margins of the node, extending into the mediastinal areolar tissue.

Results of chemical analyses made on the white rats are given in table 7, and the average values have been recorded in table 8 for comparison with similar values for rats inhaling other dusts. It will be noted that the values for asbestos are lower than those for quartz or chert but approximate those for the gypsum-quartz mixture, in which atmospheric agglutination tended to reduce the amount of dust inhaled. This condition prevailed even though the atmospheric concentration of asbestos dust was essentially the same as that of the quartz, was one-half that of the gypsum-quartz mixture and was one-fifth that of the ferruginous chert. Since the values for asbestos are low, it might be inferred that the total quantity of that dust actually inhaled was small or that it had been eliminated from or dissolved within the lungs. Evaluation of these possibilities is not feasible on the basis of the observations derived from this study.

Reaction in Cats.—Twenty cats were used in this inhalation experiment with the short fiber asbestos. Eighteen were kept in the dust room continuously until put to death, the exposure period ranging from one month to nearly 54 months. The other two were removed to normal air after a dust exposure of 31 months; one of these was killed five months, and the other 24 months, later. In general, the tissue response was confined to microscopic foci of fibrosis, which were in the walls of groups of subpleural alveoli rather than in the peribronchiolar areas. In one animal the change was extensive enough to be visualized on gross inspection of the section. Only in the animal with the longest exposure—54 months—did the roentgenogram reveal definitely abnormal shadows. A roentgenogram made after 30 months revealed no abnormality; after 45 months, a faint mottling could be detected throughout both lungs. At autopsy, nine months later, there was only microscopic fibrosis in the subpleural zone plus heavy lymphocytic infiltration about small bronchioles. Asbestosis bodies were rare. On prolonged search a few yellow atypical bodies, smooth and without haustrations, were found in two animals exposed for more than a year.

Reaction in Rabbits.—Eight rabbits were exposed to dust for periods extending from one to more than five years; the last animal was removed from the dust room and left in normal air six months before being killed. There was never enough pulmonary fibrosis to be detected grossly, and there was no chronic adhesive pleurisy. Microscopic evidence of alveolar wall thickening was first detected in one animal after about three years of exposure and was seen in all five animals examined thereafter, including the one removed to normal air. One animal that died of paralysis after nearly four years of exposure exhibited a reaction visible on gross inspection of tissue sections. The possibility of pulmonary infection in this animal could not be excluded. In another animal dying two years later the focal fibrosis was not nearly as obvious or as advanced. Areas of involvement, which were largely visualized because of phagocytic reaction within the air spaces, tended microscopically to become more fibrous with the passage of time, but there was never much encroachment on the lumen of air spaces and the structure of the lung was preserved. Asbestosis bodies were not detected in rabbits that died early in the experiment but were seen in all animals that had been exposed to the dust for more than three years.

Summary and Interpretation of Inhalation Experiment with Short Fiber Asbestos Dust.—The original purpose of the experiment was to evaluate the role of short asbestos fibers in the genesis of asbestosis. It was felt also that if the tissues reacted more rapidly and more extensively to short fiber asbestos than to King's floats there would be a basis for believing that the action of asbestos is in part, at least, a chemical one as postulated for quartz. This experiment, in which the tissue reaction was slower and less extensive than that in the previous experiment with King's floats dust, indicates that the capacity of inhaled asbestos fibers to produce fibrosis is determined primarily by factors not chemical in nature.

Of the four species exposed in this experiment, only the guinea pig and to a lesser extent the white rat responded with characteristic peribronchiolar fibrosis. The cat reacted with atypical subpleural fibrosis and the rabbit with only slight parenchymal fibrosis.

Ball-Milled Asbestos Dust

In the inhalation experiment with short fiber asbestos dust a small quantity of unground short fiber asbestos was mixed with the ball-milled product in order to generate a suitable dust cloud. When that experiment failed to produce an accelerated tissue reaction, in comparison with the response initiated by King's floats, it became apparent that the biologic activity of asbestos is not increased by a reduction of fiber size. Thus the possibility arose that the tissue reaction observed was due solely to the relatively few long fibers of the unground asbestos and that the short fibers of asbestos had no more than a very insignificant role in the production of asbestosis, a concept not in accord with previous experiments concerning pneumonoconiosis. Consequently another inhalation experiment was started in which only ball-milled asbestos was used.

Composition and Atmospheric Concentration of the Dust.—The dusting material was the ball-milled, short fiber asbestos used in the previous inhalation experiment, but unground material was not mixed with it. Owing to the tendency of the material to form small spherules which prevented much of the fibrous portion from floating out of the dusting machine, the dispersal of the dust was not entirely satisfactory. Therefore, after an initial seven months of operation, steel wire brushes were attached to the inside surface of the hopper and to the rotating paddle to disintegrate the spherules and release the fibers. This arrangement gave satisfactory results and was used for the remaining 21 months of the experiment.

The composition of the raw asbestos used is shown in tables 2 and 3. Petrographic and x-ray diffraction examination of atmospheric dust, collected in the dust room with an electrostatic precipitator after the installation of wire brushes, indicated that about 15 per cent of the air-suspended material was chrysotile, and about 60 per cent, serpentine; of the balance, magnetite comprised 10 per cent, brucite 3 per cent, quartz 2 per cent and other minerals 10 per cent. During the seven month period before the wire brushes were used, the chrysotile content of the atmospheric dust was somewhat lower than 15 per cent, but reliable values were not obtained.

The dust concentration during the first seven months of the experiment was about 100 million particles per cubic foot of air. After the wire brushes were

installed, the dust counts were higher, and the over-all average for the remaining 21 months was about 150 million.

Size-frequency studies of atmospheric dust collected inside the animal cages revealed that nearly 99 per cent of the components suspended in the air could be classified as clumps or particles; only about 1 to 1.5 per cent was fibers. One third to one half of the fibers were longer than 10 microns, indicating a concentration of long fibers of about 0.8 million. This figure is about one-half the estimated value of 1.4 million for the short fiber experiment.

Guinea pigs, rats and mice were used in the inhalation experiment with the 100 per cent ball-milled asbestos dust. The results are summarized in table 9.

Reaction in Guinea Pigs.—The experiment was started with 100 guinea pigs. As the dust exposure proceeded, there were 39 accidental deaths, 32 of these being due to pneumonia in an epidemic. The 61 pigs remaining exposed to the dust were killed at intervals during exposure, except for 16 guinea pigs transferred to normal air after 28 months of dusting. For the first year of exposure practically the only reaction to the dust was the presence of scattered phagocytes and an occasional minute asbestosis body. At 16 and 20 months no gross response was visible on the tissue section, but microscopically peribronchiolar foci of inflammatory cells

TABLE 9.—*Summary of Inhalation Experiment with 100 per Cent Ball-Milled Asbestos Dust*

Nature of Experiment	Animals	Maximum Dust Exposure, Mo.	Maximum Survival After Dust Exposure, Mo.	Results
Dust exposure continuous throughout life	84 guinea pigs	24	0	No appreciable pulmonary reaction
	49 rats	28	0	No suggestion of asbestosis
	28 mice	12	0	No suggestion of asbestosis
Dust exposure followed by prolonged residence in normal air	28 guinea pigs	28	32	Fibrosis typical of asbestosis was present at mo. after exposure ceased in an amount sufficient to be visible grossly; smaller foci could be seen microscopically at 8 mo. and 8 mo. after termination of exposure

could be seen. At 24 months (fig. 6 *A*) there was still no change large enough to be seen with a hand lens, although microscopic examination revealed cellular accumulations about terminal bronchioles and many more asbestosis bodies, chiefly within cells. The lungs of animals exposed for the full dusting period of 28 months and afterward living in normal air for two months revealed the changes described above and also very slight peribronchiolar fibrosis. For exposed animals living eight months in normal air the findings were similar, but at 12 months three of four animals showed grossly visible characteristic peribronchiolar fibrosis with adenomatoid change (fig. 6 *B*).

The tracheobronchial nodes were essentially normal until exposure had been continued for more than a year and a half. Animals killed at 12 months and at 16 months revealed a few minute collections of phagocytes containing particles but practically no fibers large enough to be recognized as such. After 20 months of exposure many monocytes filled with yellow granules were present. At 30 months there had been a slight increase in reticulum but no fibrosis. No further changes occurred in the nodes. Asbestosis bodies were not seen in the nodes of any of the guinea pigs.

Minute asbestosis bodies were observed in the lungs as early as three months after exposure began, but they did not become numerous until 16 months had elapsed. The bodies were short and practically all were intracellular, although at 20 months some were long enough to project beyond the cell borders. It is

important to note that in the later months of exposure there was a distinct increase in the number of long fibers, up to 70 microns in length, in the lungs with the formation of characteristic long asbestosis bodies.

Chemical analyses (table 10) of the lungs revealed that considerable dust had been retained in the lungs. After 24 months of continuous exposure the average

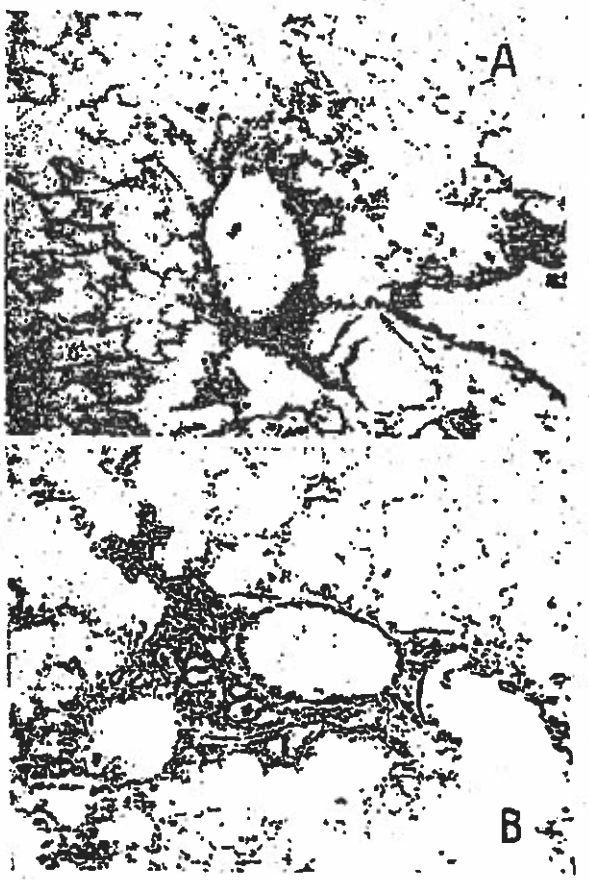

Fig. 6.—Ball-milled asbestos inhalation experiment: *A*, lung of a guinea pig with 24 months' dust exposure. A bronchiole is shown at the center, with a slight accumulation of phagocytic cells but without the formation of collagen (× 200).

B, lung of a guinea pig with 28 months' dust exposure and then 12 months' inhalation of normal air. The reaction is much like that shown in *A*, but there is a slight deposition of collagen, most apparent at the left (× 200).

value for total silica, per cent of ash, was 25.37. This should be contrasted with the average value of 14.34 (table 6) for animals exposed 24 months to the short fiber asbestos dust.

In view of the high values for silica obtained with the animals exposed to 100 per cent ball-milled dust, it is important to note that their pulmonary response was much less than that of animals exposed for 24 months to the short fiber asbestos in the previous experiment. This again indicates that the biologic activity of asbestos inhaled into the lung is not increased by a reduction in size of the fibers.

Reaction in White Rats and Mice.—In this experiment 40 rats were exposed for periods up to 20 months and 24 mice for periods up to 12 months. In neither species did even a suggestion of asbestosis develop, and reaction was limited to phagocytosis of inhaled particles by widely scattered dust cells which remained free in air spaces or were transported to the tracheobronchial lymph nodes. No asbestosis bodies were found in the rats, but in the mice there were a very few small, nonbeaustrated forms within phagocytes.

TABLE 10.—*Analyses of Lungs of Guinea Pigs Exposed to Dust in Inhalation Experiment with 100 per Cent Ball-Milled Asbestos Dust*

Exposure to Dust, Mo.	Period in Normal Air, Mo.	Amt. of Ash, % of Dried Lung	Total SiO_2, % of Dried Lung	Total SiO_2, % of Ash	Tissue Reaction *
Dust Exposure Continuous During Life					
7	0	4.35 4.30 4.25	0.21 0.30 0.23	4.85 7.05 5.30	0
8	0	3.66 3.60 3.03	0.22 0.21 0.01	4.80 7.00 12.01	0
8	0	3.60 3.07 3.74	0.70 0.42 0.26	13.47 6.80 6.77	0
8	0	3.10 3.03 3.02	0.23 0.25 0.20	7.61 7.47 7.12	0
8	0	2.05	0.22	11.00	0
12	0	6.01 6.71	1.23 1.15	22.36 22.03	0
16	0	5.10 5.01	1.02 1.11	19.90 21.03	±
20	0	5.05 5.20	1.05 1.20	20.50 21.01	±
24	0	5.20 5.04	1.05 1.21	19.06 21.70	±
Dust Exposure Followed by Prolonged Residence in Normal Air					
20	2	7.20 6.17	1.31 2.10	21.03 33.24	+
24	8	5.23 5.00	0.05 0.57	12.00 14.53	+
20	12	6.35 5.17	0.84 0.04	13.05 15.11	2+

* The symbols averaging the tissue reaction in each group represent merely the relative degree of reaction, running from 0 to ± (questionable) to 2+ (the maximum observed in this experiment). The relationships apply only within this table and cannot be compared with symbols in other tables.

Summary and Interpretation of Inhalation Experiment with 100 per Cent Ball-Milled Asbestos Dust.—The tissue reaction observed in this experiment was not as intense as that in the previous investigation with short fiber asbestos. The reaction was slower in development and less extensive even though more dust accumulated in the lungs. Since there were fewer fibers longer than 3 microns in the material used in this experiment, the results tend to confirm the interpretation made in the summary of the previous short fiber experiment that the reaction is not primarily chemical in nature, and to support the impression that reduction in size of asbestos fibers does not increase the biologic activity of asbestos inhaled into the lung.

The finding of long asbestosis bodies in animals that had inhaled the ball-milled material is an example of the difficulty of completely eliminating long fibers from a large volume of asbestos as required for an inhalation experiment.

In regard to the progression of the tissue reaction after the animals had been removed from the dust, observed in this experiment but not in the others, the following interpretation is offered: When the reaction is well developed at the termination of exposure, the contraction of the fibrous tissue obscures any progression that may have occurred; in this experiment, however, since the reaction observed was less mature, its subsequent progress was more readily apparent.

Long Fiber Asbestos Dust

Since inhalation of short fiber and of 100 per cent ball-milled asbestos dust did not result in acceleration of the tissue reaction in comparison with that produced by King's floats, the hypothesis that short fibers of asbestos were of minor importance in the etiology of asbestosis was given added support, and attention was directed to the view that the long fibers were of primary significance in that etiology. The King's floats asbestos used in the first inhalation experiment had a rather low content of fibrous chrysotile and contained considerable serpentine and other impurities. Therefore, it was decided to conduct a new inhalation experiment with a purer form of chrysotile which would be richer in long fibers.

Composition and Atmospheric Concentration of the Dust.—The dusting material employed in this investigation was obtained from an asbestos fabricating plant. Samples of several varieties of long fiber asbestos dust were first submitted to the Saranac Laboratory for examination, and one of these, which was low in magnetite and chromite and had a fibrous content estimated to be about 75 per cent, was selected as most suitable. Steel wire brushes, fastened to the inside surface of the hopper and to the rotating paddle as in the preceding inhalation experiment, were used to open up the bundles of asbestos and liberate more fibers into the atmosphere.

The composition of the long fiber asbestos used is indicated by the chemical and petrographic analyses given in tables 2 and 3. Analysis of air-suspended material from the dust room disclosed that about 60 per cent of the long fiber dust was chrysotile and about 20 per cent serpentine; as already noted, the composition of a similar air-floated sample of ball-milled, short fiber dust was 15 per cent chrysotile and 60 per cent serpentine.

The dust concentration as revealed by impinger samples taken inside the animal cages was much lower than the concentration for the experiments with short fiber or ball-milled dust. For the first year of the experiment with long fiber asbestos the average of the light field counts was 32 million particles per cubic foot of air; for the second year, 48 million; for the third year, 39 million, and for the fourth year, 43 million.

The size-frequency of atmospheric samples of the long fiber asbestos dust and of the ball-milled dust is shown in table 11. Both samples were collected with the electrostatic precipitator. It will be noted that there was far more fibrous material in the long fiber dust.

Guinea pigs, cats, rats and mice were employed in this inhalation experiment. The results, summarized in table 12, are described in greater detail below.

Reaction in Guinea Pigs.—The experiment was started with 100 guinea pigs. After exposure had been carried on for a year, a severe epidemic of pneumonia arose in the dust room and about one third of the animals died or were killed. To replace them, 38 more guinea pigs were added to the surviving group. Histological examination revealed lesions in the lungs after eight months of dust exposure, consisting of cellular connective tissue about the terminal bronchioles (fig. 7 *A*). At 12 months there were adenomatoid changes in the adjacent parenchymal areas, and by the sixteenth month (fig. 7 *B*) definite fibrosis was present in these areas as well as around the bronchioles. The fibrous lesion could be seen macroscopically at 20 months. From this time on the reaction increased in extent and in the amount of collagen, and by the thirty-fourth month, it had fanned out

TABLE 11.—*Size-Frequency of Atmospheric Long Fiber and 100 per Cent Ball-Milled Asbestos Dust Collected Inside Cages*

Type of Asbestos	Grains, %			Fibers, %		Clumps, %	Total
	< 3 Microns	3-10 Microns	> 10 Microns	< 10 Microns	> 10 Microns		
Long fiber	69.4	3.1	0.9	23.8	0.7	1.0	100
Ball-milled	90.8	4.8	0.6	0.8	0.6	3.2	100

TABLE 12.—*Summary of Inhalation Experiment with Long Fiber Asbestos Dust*

Nature of Experiment	Animals	Maximum Dust Exposure, Mo.	Maximum Survival After Dust Exposure, Mo.	Results
Dust exposure continuous throughout life	127 guinea pigs	36	0	Definite fibrosis in 10 mo.
	4 cats	42	0	Nearly developing fibrosis first seen at 24 mo.
	20 rats	25	0	Marked peribronchiolar fibrosis first seen at 21 mo.
	20 mice	25	0	Limited reaction: no fibrosis
Dust exposure followed by prolonged residence in normal air	12 guinea pigs	20	14	Clearing of inflammatory reaction and definite contraction of fibrous tissue
	9 guinea pigs	27	0	Clearing of inflammatory reaction and slight contraction of fibrous tissue
	3 cats	18	21	Similar to continuous exposure group; suggestion of progression in one of the two animals

considerably into the parenchyma (fig. 8 *A*). The lesions were rather sharply localized and the extensions from different bronchioles showed no tendency to fuse, even in animals exposed for the maximum period of three years. Although the intrapulmonary reaction sometimes reached the pleura, there was no involvement of that membrane. Emphysema was not detected at any point. Some thickening of the larger bronchi with a chronic inflammatory infiltration was revealed, but it was considered no more than would be produced by a similar period of inhalation of any dust.

In guinea pigs exposed to the dust for 20 months and then removed to normal air, there was a marked tendency for cellular inflammatory reaction to clear. This effect, accompanied by contraction of the fibrous tissue, resulted in a diminishing size of the focal lesions. None of these animals, killed at various periods up to 14 months after exposure, revealed lesions as large as those in the group killed at the end of the 20 month exposure period or those in animals which remained in the dust room for more than 20 months. Fourteen months after dust exposure ceased, the foci in four of the six remaining guinea pigs were so small that they were visible only with a hand lens (fig. 8 *B*).

In the group exposed for 27 months and then transferred to a normal atmosphere the response was quite similar to that in the 20 month exposure animals mentioned above. Small foci were always visible on gross inspection of sections of all guinea pigs of the 27 month series, but in no instance was there evidence of the reaction.

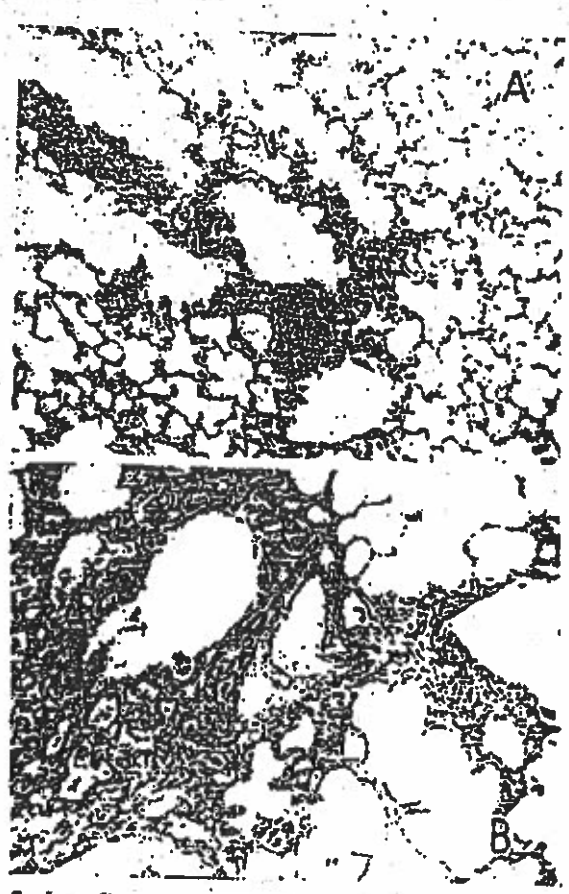

Fig. 7.—Long fiber asbestos inhalation experiment: *A*, lung of a guinea pig with eight months' dust exposure. The bronchiole at the center already shows an accumulation of phagocytic cells, and there is a slight deposition of collagen. Compare with figure 6 *A*, showing the reaction to ball-milled asbestos after 24 months (× 200).

B, lung of a guinea pig with 16 months' dust exposure. Again note a bronchiole with its surrounding reaction, consisting of fibrosis and atelectatoid change. Collagen deposition is now seen in the walls of adjacent alveoli, at the right (× 200).

In the tracheobronchial lymph nodes reaction was first visible at the third month of exposure. By the eighth month patches of cellular connective tissue

began to appear in the medulla, and by the fourteenth month most of the node had been replaced by cellular connective tissue. This picture, which resembled that in early silicosis, persisted to the end of the experiment. Some animals showed, as a variant, heavy sheets of diffusely distributed monocytes and large active giant cells, but there was never any necrosis or hyaline formation. The spindle-shaped

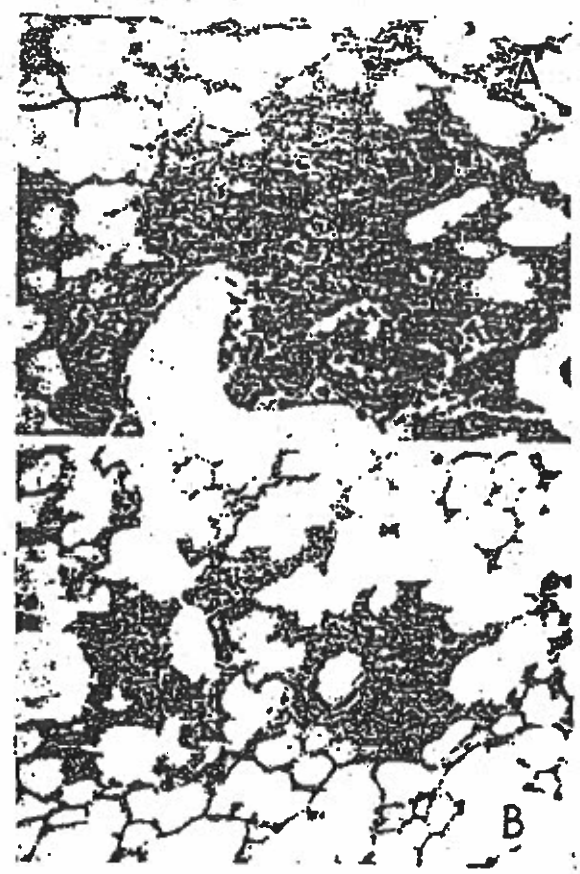

Fig. 8.—Long fiber asbestos inhalation experiment: *A*, lung of a guinea pig with 34 months' dust exposure. A bronchiole is seen at the lower center: the large area above it represents the involvement of alveolar walls. Compare with figure 7 *B* and note the increased extent of reaction (× 200).

B, lung of a guinea pig with 20 months' dust exposure and then 14 months' living in normal air. The reaction is essentially like that shown in figure 7 *B*: The bronchiole at the right center is surrounded by fibrous tissue with adenomatoid change at the right. There is residual scarring in the walls of adjacent alveoli at the left. It is apparent that no progression has occurred (× 200).

new cells were yellowish from fine pigment granules that stained for iron. No fibers or asbestosis bodies were seen.

Although asbestosis bodies were found in the lung as early as one month after exposure began, they were rare and hard to find. At five months more were visible, chiefly coiled inside giant cells, and at eight months many bodies

TABLE 13.—*Analyses of Lungs of Guinea Pigs Exposed to Dust in Inhalation Experiment with Long Fiber Asbestos Dust*

Exposure to Dust, Mo.	Period in Normal Air, Mo.	Amt. of Ash, % of Dried Lung	Total SiO_2, % of Dried Lung	Total SiO_2, % of Ash	Tissue Reaction *
		Dust Exposure Continuous During Life			
1	0	4.35 4.23 4.37	0.04 0.09 0.04	1.10 2.11 0.93	0
3	0	4.45 4.48 4.55	0.05 0.05 0.06	1.23 1.18 1.40	±
4	0	4.38 4.35 4.51	0.05 0.05 0.06	1.20 1.22 1.48	±
5	0	4.77 4.64 5.06	0.06 0.11 0.09	1.25 2.67 1.77	±
6	0	4.72 4.51 4.34	0.10 0.09 0.07	2.21 1.40 1.58	+
12	0	4.87 5.02 5.34	0.25 0.20 0.21	4.80 4.08 4.99	+
18	0	2.98 2.83 2.38	0.48 0.33 0.34	13.70 11.59 10.01	3+
20	0	3.14 3.00 3.56	0.43 0.48 0.53	[illegible] 11.83 14.89	2+
24	0	3.45 2.02	0.15 0.28	20.13 8.29	3+
27	0	2.40 2.74	0.39 0.42	11.81 13.15	3+
30	0	2.55 2.03 2.88	0.37 0.36 0.34	10.85 11.22 4.10	4+
34	0	5.65 6.70	0.50 0.44	4.05 13.47	4+
36	0	4.10 2.71	0.37 0.35	9.11 12.69	4+
		Dust Exposure Followed by Prolonged Residence in Normal Air			
22	0	3.64 3.42 3.88	0.48 0.49 0.53	13.22 13.63 14.59	2+
20	4	2.92 2.81	0.11 0.27	7.23 9.60	3+
20	10	4.18 4.40	0.24 0.23	5.73 5.07	2+
20	14	5.07 5.04	0.21 0.16	4.19 3.08	+
27	0	2.40 2.74	0.39 0.40	11.61 13.15	3+
27	3	3.46 2.84	0.21 0.13	1.80 4.06	2+
27	7	2.19 3.16	0.25 0.28	1.70 4.78	2+
27	9	3.21 2.35	0.10 0.12	3.04 5.21	2+

* The symbols averaging the tissue reaction in each group represent merely the relative degree of reaction, ranging from 0 to ± (questionable) to 4+ (the maximum for this experi-[ment]). The relationships apply only within this table and cannot be compared with symbols in other tables.

were free in connective tissue. They became fairly abundant as exposure continued, although in some later animals the asbestosis bodies were only moderately numerous.

It is important to note from analyses of the lungs (table 13) that even though the tissue response at any given period of time was much greater in the guinea

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pigs of this experiment than in those exposed to either short fiber or ball-milled asbestos, the amount of mineral matter in the lung ash was much less.

Reaction in Cats.—Four cats inhaled the long fiber asbestos dust for periods of 14, 25, 33 and 42 months, respectively, and were immediately killed. Two other cats, after being exposed to dust for 18 months, lived in a normal atmosphere for an additional 24 months. Fourteen months' exposure was sufficient to produce cellular accumulations of phagocytes around terminal bronchioles and peripheral arterioles together with compact collections of similar cells in the tracheobronchial lymph nodes. At that time there were no typical asbestosis bodies, but smooth, pointed, yellow fibers were seen very rarely. With continued exposure, up to 42 months, reaction in the locations noted progressed to the formation of cellular connective tissue which made well defined sheaths about the respiratory bronchioles and arterioles, marked lymphoid hyperplasia and lymphoid infiltration of bronchiolar walls (fig. 9). Typical asbestosis bodies were not formed, although there was

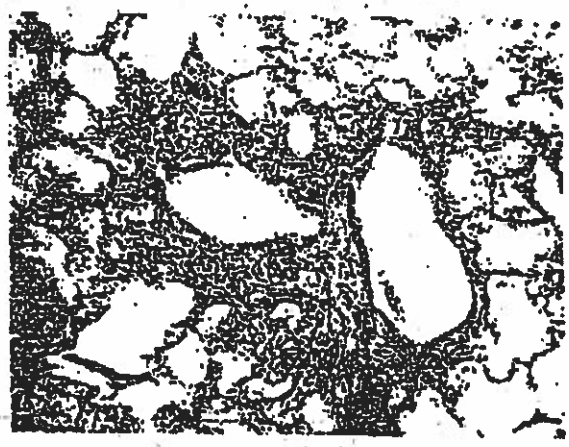

Fig. 9.—Long fiber asbestos inhalation experiment: Lung of a cat with 42 months' dust exposure. Two bronchioles are shown with adjacent cellular reaction and collagen deposition (× 200).

an occasional fiber, smooth, yellow and pointed. Pleurisy was not present. The reaction was similar in location to that in the guinea pigs, but fibrosis was much slower in development. Roentgenograms of cats made after exposure periods of 25, 33 and 42 months, respectively, failed to demonstrate evidence of pulmonary lesions.

Reaction in Rats.—Although 20 rats were placed in the dust room, many died from pneumonia and were not suitable for study. Five animals, of which one was exposed for 19 months and four for 25 months, were free from pulmonary infection and offered a basis for tentative conclusions. In the 19 month animal, the reaction was just beginning. All four animals killed at 25 months showed a well marked peribronchiolar fibrosis. After a long search, only two small, smooth asbestosis bodies were found in the 19 month animal and none was found in the 25 month animal. Thus these animals exhibited fibrosis without asbestosis bodies or fibrosis accompanied by only a very infrequent asbestosis body.

Reaction in Mice.—Out of 20 white mice used in this experiment, 11 lived a year or more in dust and died or were killed without showing an appreciable degree of pulmonary infection. The reaction to the inhaled dust was limited to phagocytosis by mononuclear cells. Usually these were widely scattered through the air spaces; a limited number were grouped about the terminal bronchioles, producing some thickening of their walls. There was no suggestion of fibrosis.

Numerous asbestosis bodies were observed in animals killed late in the experiment. Thus these animals exhibited asbestosis bodies without fibrosis.

Summary and Interpretation of Inhalation Experiment with Long Fiber Asbestos Dust.—The purpose of this experiment was to evaluate the importance of long fibers in the tissue response to inhaled asbestos. The results, in comparison with those of previous investigations, indicate strongly that long fibers are chiefly responsible for asbestosis. Thus, the reaction in guinea pigs developed earlier and became more extensive in this experiment than in previous experiments in spite of a smaller concentration of atmospheric dust and a lower mineral content of the lungs. Furthermore, typical peribronchiolar fibrosis was produced in cats, although in a previous experiment with short fiber dust peribronchiolar fibrosis did not develop in this species.

The cause of the cellular fibrosis in the lymph nodes of the guinea pigs is not clear. It did not occur in other inhalation experiments with asbestos.

INJECTION EXPERIMENTS

Since the inhalation experiments reported above strongly suggested that long fibers of asbestos are the significant factor in the causation of asbestosis, a series of injection experiments was inaugurated wherein the dosage and the length of the fibers could be controlled more precisely. Also, by the use of controlled dosages, the relative capacities of various asbestos minerals to produce reaction could be compared. In these injection experiments, guinea pigs, rabbits, rats and dogs were used, and the mineral dust was injected by the intratracheal, the intraperitoneal and the intravenous technic, but not all the technics were used for each species. For the purpose of simplification the findings in each series of tests, except for dogs, have been condensed and reported in tables, to which reference will be made later. In the case of dogs, only one test was made, and since the findings were negative, no detailed report is included.

Experiments Using Intratracheal Technic

As the asbestos minerals do not cause typical advanced fibrosis in extrapulmonary tissue, the intratracheal technic is the preferred way of introducing fibrous dust into the experimental animal. In this method the dust suspension is injected by means of a special needle or catheter deep into the trachea, from which it flows into the lungs.

Comparison of Fibrous and Nonfibrous Dusts.—To demonstrate that the ability of asbestos to produce fibrosis resides in its fibrous character, the series of injection experiments reported in table 14 were performed.

TABLE 14.—*Comparison of Reactions to Chrysotile and Serpentine Injected Intratracheally*

Dosage: Each animal was given an intratracheal injection of 0.5 cc. of a 5 per cent suspension of the dust. Two weeks later another similar injection was given. Total amount of dust injected was 50 mg.

Animals used: Six groups of 9 guinea pigs each (one group for each type of dust).

Periods at which animals were killed: One or two animals in each group at 1, 3, 6, 9½ and 12 months after last injection.

Preparation of dust: Chrysotile (ball milled) unheated: Ball milled for 1,175 hr., dried and reground in agate mortar.
Chrysotile (ball milled) ignited: Ball milled chrysotile heated for 2 hr. at about 700 C., then ground in agate mortar 2 or 3 min.
Chrysotile (fibrous) unheated: Ground in agate mortar to pass 200 mesh.
Chrysotile (fibrous) ignited: 200-mesh material heated for 2 hr. at about 700 C. No further grinding.
Serpentine (ball milled) unheated: Ball milled for 1,175 hr., dried and reground in agate mortar.
Serpentine (ball milled) ignited: Ball milled serpentine heated for 2 hr. at about 700 C., then ground in agate mortar 2 or 3 min.

Material	Size of Dust Particles	Results
Chrysotile (ball milled) unheated	3 microns and less	Granular destroyed capacity to cause fibrosis. At 2 mo. considerable inflammatory edema and cellular proliferation and localization of dust particles about bronchioles; at 3 mo., only a very slight proliferative reaction; at 6, 9½ and 12 mo., widely scattered small mononuclear phagocytes. At 12 mo., a few microscopic patches of thin alveolar wall thickening with some adenomatoid change in portion of air spaces abutting on thickened bronchi. No asbestosis bodies seen.
Chrysotile (ball milled) ignited	3 microns and less	Reaction limited to large foreign body giant cells without production of fibrous tissue.
Chrysotile (fibrous) unheated	20-40 microns approx.	A distinct fibrosis. Reaction localized to connective tissue about terminal bronchioles; little within these tubes. Contraction caused adenomatoid appearance of air spaces given off directly from terminal bronchioles. Reaction area became smaller with progress of time; no new regions involved. No fibrous pleurisy even at points abutting intrapulmonary spaces. At 1 mo., considerable inflammatory edema and loss of cellular proliferation; at 2 mo. well marked cellular proliferation and fibrosis occurring mostly about respiratory bronchioles. This reaction developed before asbestosis bodies had formed and was as advanced as that produced by 2 yr. inhalation of asbestos dust. At 6 mo., reaction less extensive than at 3 mo., apparently due to contraction of fibrous tissue; asbestosis bodies were abundant. At 9½ mo., reaction still less extensive, confined to the immediate vicinity of the small terminal bronchioles, where the scar tissue was quite dense and was becoming hyaline in character. Sometimes it even obliterated the bronchiole. Asbestosis bodies had become scarce. At 12 mo., the well developed peribronchial and interbronchial adenomatoid areas of fibrosis had produced considerable distortion. Here peripherally were patches of pneumonitis with eosinophilic infiltration, some of which was being transformed into fibrous tissue. These seemed to be precursors of the localized, diffuse patches of thin alveolar wall fibrosis seen elsewhere.
Chrysotile (fibrous) ignited	20-40 microns approx.	Reaction limited to large foreign body giant cells without proliferation. Fracture the fibers, which made them brittle, destroyed their capacity to produce subsequent reaction.
Serpentine (ball milled) unheated	3 microns and less	Dust relatively inactive. At 1 and 2 mo., simple phagocytosis without proliferation; at 6 mo., no change except possibly lymphoid cell infiltration; at 9½ mo., a slight chronic pneumonitis; at 12 mo., only a little pneumonitis without suggestion of fibrosis.
Serpentine (ball milled) ignited	3 microns and less	Dust relatively inactive. Reaction exactly the same as for unheated serpentine. With ignited serpentine, less tendency for dust to be carried to bronchial nodes.

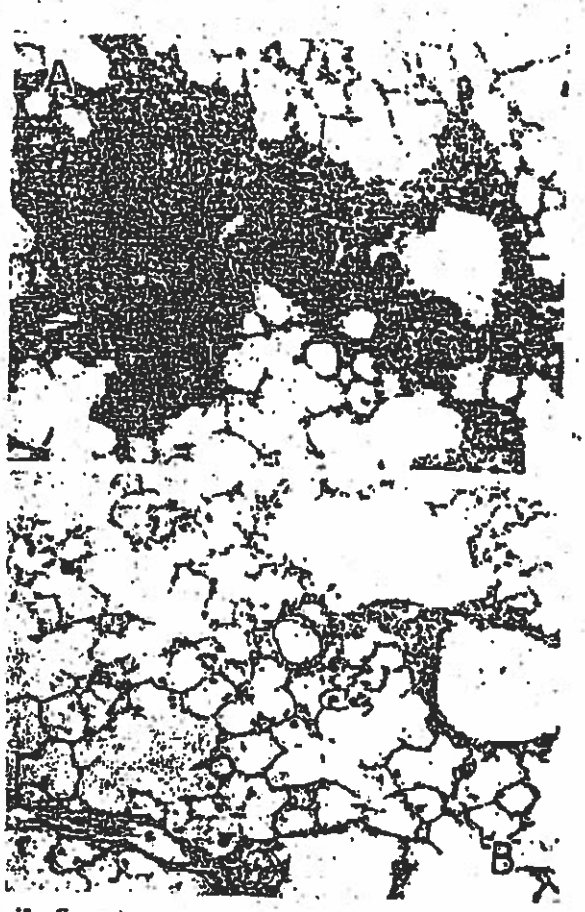

Fig. 10.—Comparison of reactions provoked by injected long fiber and ball-milled asbestos dusts: *A*, lung of a guinea pig which four months before had received an intratracheal injection of long fiber asbestos dust. Note the peribronchiolar accumulation of cells with collagen deposition. The bronchiole chiefly involved is in the midst of the reaction (× 200).

B, lung of a guinea pig which four months before had received an intratracheal injection of ball-milled asbestos dust. A bronchiole is shown at the right. In contrast with *A*, note that only a few cells have accumulated about the bronchiole and that collagen deposition is absent (× 200).

The tests were made with long fiber chrysotile, unheated, and with chrysotile that had been ignited to destroy its flexible structure or ball milled to reduce the length of fiber to 3 microns and less. At the same time control tests were made with serpentine, which has the same chemical composition as chrysotile but is nonfibrous. A review of the findings reveals that only the unheated, long fiber chrysotile produced typical peribronchiolar fibrosis and that ball-milled material containing only fibers less than 3 microns in length failed to cause fibrosis (figs. 10 and 11). Fibers subjected to ignition also had lost their capacity to cause serious tissue damage. Ignition produced important changes in the chrysotile fibers, among them being loss of water, an alteration from a flexible to a brittle structure and possibly other changes. Experi-

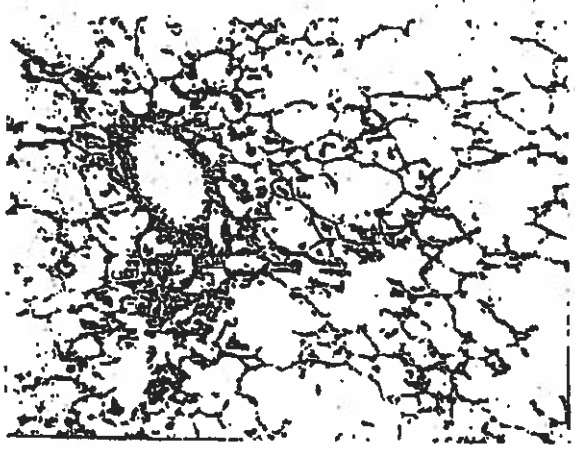

Fig. 11.—Serpentine injection experiment: Lung of a Guinea pig that had received an intratracheal injection of this dust four months before. A bronchiole is shown at the left center. The phagocytic cells exhibit little predilection for the bronchiole and collagen deposition is absent (× 200).

mental studies concerning this observation will be reported in a separate publication.

Comparison of Various Long Fiber Dusts.—Some very interesting findings are disclosed by the results of the experiments recorded in table 15. First, all the long fiber asbestos minerals tested, with the exception of anthophyllite, produced typical fibrosis. The characteristic peribronchiolar reaction caused by three representative long fiber asbestos minerals—chrysotile, amosite and crocidolite—is shown in figures 10 *A* and 12. Why anthophyllite behaved differently from the other asbestos minerals is not entirely clear.

Second, with the mineral brucite, which is not a silicate but is a fibrous form of magnesium hydroxide, a characteristic fibrosis like

TABLE 15.—*Comparison of Reactions to Various Long Fiber Dusts Injected Intratracheally*

Dosage: Two injections of 0.5 cc. of a 5 per cent suspension given two weeks apart. Total dose was 50 mg.

Animals used: From 6 to 9 guinea pigs for each dust.

Periods at which animals were killed: Usually at 1, 4, 8 and 12 months after last injection.

Size of dust particles: Separated so that most fibers were from 20 to 50 microns long.

Mineral	Results
Chrysotile (Thetford)	Distinct fibrosis. Additional information given opposite chrysotile (fibrous) unheated, in table 14.
Chrysotile (Arizona; low iron content: 0.3% Fe_2O_3)	Reaction virtually identical with that to Thetford chrysotile. Both fibrosis and asbestosis bodies produced with an asbestos containing very little iron. Fibrosis occurred as plugs within terminal bronchioles and as finer deposits at periphery. Fibrosis developed before asbestosis bodies were seen and was in cellular state well formed at one month. With age, fibrous tissue contracted and occupied smaller area but was more dense. Adenomatoid changes similar to those with Thetford chrysotile. Pleurisy limited to immediate vicinity of early reaction about areas of massive localization. Asbestosis bodies formed but were few. At 1 mo., after injection, minute foci of mononuclear proliferation about bronchioles and in areas of atelectasis; at 1½ mo., heavy peribronchiolar patches of fibrosis often with papillary projections partially closing lumen of bronchiole; adenomatoid appearance marked; connective tissue reaction showed heavy collagen but no hyalinization; at 3 mo., minute foci of well matured fibrosis about bronchioles; at 6 mo., mature asbestosis fibrosis with evidence of contraction; considerable chronic pneumonitis with infiltration of lymphocytes and eosinophils. At 9 mo., small intrabronchiolar fibrous plugs with foci of more delicate fibrosis at periphery.
Amosite	Typical fibrous endobronchiolitis and peribronchiolitis with formation of atypical asbestosis bodies. Maturation of bodies begun before 4th mo. after injection, well developed by 8th mo. Bodies persist after 12th mo. Reaction at 1 mo., heavy endobronchiolitis and peribronchiolitis already showing fibrous changes; atelectasis and fibrosis with some necrosis at site of massive localization of dust. At 4 mo., heavy, widely scattered endobronchiolitis and peribronchiolitis, now fibrous, with marked deformity of bronchioles and with an adenomatoid appearance. At 8 and 10½ mo., reaction in lung essentially the same as at 4 mo. At 12 mo., foci of fibrous endobronchiolitis and peribronchiolitis still larger, with more dense scar tissue and more deformity of bronchial tubes but no extension into, or atelectasis of, peripheral parenchyma.
Crocidolite (Bolivia)	Advanced fibrous endobronchiolitis and peribronchiolitis. Scaried asbestosis bodies noted at 8 mo. At 1 mo., early fibrous endobronchiolitis and peribronchiolitis; many giant cells and some lymphocytic reaction. At 4 mo., small areas of endobronchiolitis scattered throughout the lung; cellular fibrosis. At 8 and 12 mo., areas of bronchiolitis smaller because of contraction of dense scar tissue; at 12 mo., marked lymphocytic infiltration and adenomatoid appearance.
Crocidolite (S. Africa)	Typical advanced fibrous endobronchiolitis and peribronchiolitis produced by 0.5% suspension (1 cc. total dose); most animals would not tolerate usual 5% suspension. Fibrosis well developed before asbestosis bodies seen. At 4 mo., well developed fibrous bronchiolitis with lymphocytes and giant cells and adenomatoid change. At 8 mo., typical bronchiolitis not quite as extensive or as heavily fibrous as with a 5% suspension, otherwise the same. Many deeply stained fibers with a good proportion of macerated asbestosis bodies. At 12 mo., heavy fibrous bronchiolitis, more peribronchiolitis and endobronchiolitis, with lymphocytes and giant cells; very marked adenomatoid appearance.
Anthophyllite	Lymphocytic infiltration and giant cells but no fibrosis. A very few atypical asbestosis bodies. At 1 mo., many scattered foci of intrabronchiolar dust without massive localization; lymphocytic infiltration of walls and a few giant cells. At 8 and 12 mo., little evidence of dust; a few bronchioles and bronchi with giant cells in adjacent alveoli and with lymphocytic infiltration of walls.
Tremolite	Fibrosis about bronchioles. At 1 mo., areas of dust localization with collapse of alveoli and infiltration with acute inflammatory cells, macrophages and giant cells. Within the area were a few foci of fibrous tissue and numerous areas of hypertrophy of alveolar epithelium. Many bronchioles packed with fibers. At 4 mo., general appearance of lesion unchanged; pleura slightly thickened over heavy localizations of dust. An occasional segmented asbestosis body seen. At 4 mo., many foci of fibers in bronchioles and alveolar ducts with cellular reaction as before; also, some foci showed distinct collagen deposition. At 12 and 13 mo., reaction as before with fibrosis about bronchioles more apparent because of contracting and decrease of inflammation. Giant cells prominent. Pleura markedly involved.
Brucite	Typical fibrous endobronchiolitis and peribronchiolitis like reaction to asbestos minerals. At 1 mo., extensive endobronchiolitis and peribronchiolitis with giant cells; dense fibrous loops within bronchioles and cellular fibrosis about them; adenomatoid change present. At 3 mo., heavy intrabronchiolar and peribronchiolar fibrosis producing marked deformity with distortion of tubes and obliteration of surrounding air spaces; fibrosis pale without hyalinization but with few nuclei; no necrosis. Typical asbestosis bodies seen. At 4 and 8 mo., little change; fibrous tissue contracting. At 10½ mo., dense fibrous bronchiolitis with asbestosis bodies. No pleurisy. No extension to surrounding lung.
Glass wool	No fibrosis within a year. At 1 mo., no reaction inside bronchioles; in peripheral air spaces clumps of giant cells packed with fine spicules of glass with lymphocytic infiltration of adjacent walls; no asbestosis bodies. At 3 mo., reaction less intense than at 1 mo.; fair-sized clumps of elongated giant phagocytes containing spicules and particles of glass; no endobronchiolitis. At 4 and 8 mo., reaction still diminishing. At 12 mo., local areas of [illegible] with no fibrosis or endobronchiolitis; moderate number of smooth iron-staining fibers.

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that produced by the asbestos minerals was obtained (fig. 13 *A*). Since the brucite used contained only 0.90 per cent silica as an impurity, it is obvious that a siliceous component is not an essential factor in the development of asbestosis.

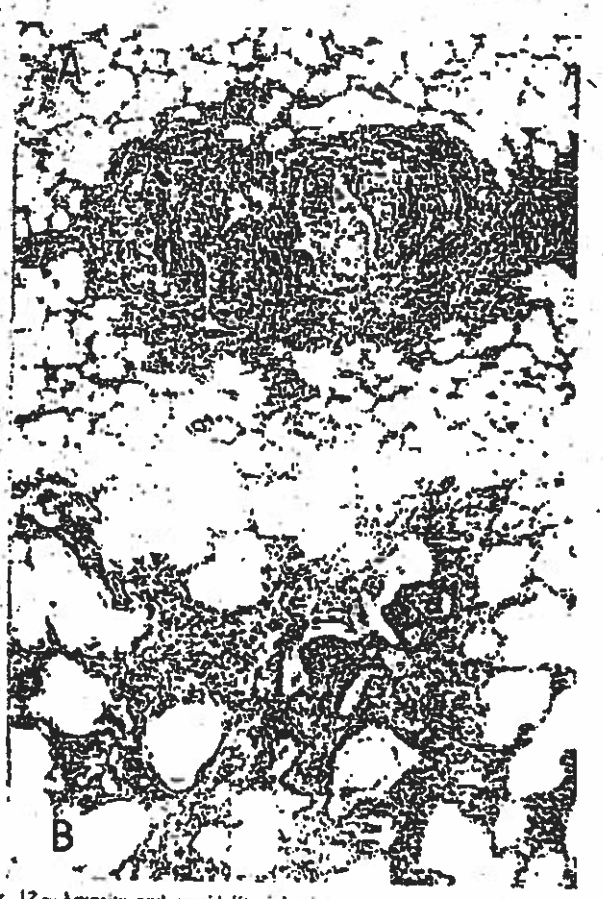

Fig. 12.—Amosite and crocidolite injection experiments: *A*, lung of a guinea pig four months after an intratracheal injection of amosite. The inflammatory reaction exhibits pronounced accumulation of cells and collagen deposition (× 200).

B, lung of a guinea pig four months after an intratracheal injection of crocidolite. As in *A*, peribronchiolar accumulation of cells and deposition of collagen are shown (× 200).

Third, no fibrosis resulted from the injection of glass wool fibers (fig. 13 *B*), even though glass wool resembles asbestos in many ways. However, there are fundamental differences. A glass wool fiber 3 microns

in diameter is a solid rod which in short lengths is fairly rigid, while an asbestos fiber of the same diameter is a bundle of extremely fine filaments which impart to the fiber a high degree of flexibility. It would seem that this structure and the associated flexibility are important factors governing the capacity of a mineral to produce peribronchiolar

Fig. 13.—Brucite and glass wool injection experiments: *A*, lung of a guinea pig which four months before had received an intratracheal injection of brucite. Even with this nonsiliceous fibrous mineral there is peribronchiolar accumulation of cells and deposition of collagen similar to that shown in *A* and *B* of figure 12 (× 200).

B, lung of a guinea pig which four months before had received an intratracheal injection of glass wool. Two bronchioles are shown, one in cross section and the other in longitudinal section. Below the latter is a thick-walled blood vessel. The bronchioles are without reaction and can be considered normal for comparison with other figures. Glass wool fibers are present in this field but cannot be seen at this magnification (× 200).

fibrosis. Experimental studies concerning this observation will be reported in a separate publication.

TABLE 16.—*Comparison of Reactions Produced by Long Fiber and Short Fiber Dusts Injected Intratracheally*

Dosage: Two injections of 0.5 cc. of a 5 per cent suspension given two weeks apart. Total dose was 50 mg.
Animals used: Six groups of guinea pigs.
Periods at which animals were killed: 1, 3, 6, 6½ and 12 months after injection.

Mineral	Size of Dust Particles	Results
Chrysotile (Thetford)	Long fiber, 20-60 microns	A distinct fibrosis. Refer to chrysotile (fibrous) unheated in table 14.
	Short fiber, 5 microns and less	No fibrosis. Refer to chrysotile (ball milled) unheated in table 14.
Amosite	Long fiber, 20-60 microns	Typical fibrous endobronchiolitis and peribronchiolitis. Refer to table 13.
	Short fiber, 20 microns and less	Reaction limited to phagocytosis with lymphocytic infiltration of adjacent walls. Short fibers packed inside swollen phagocytes; longer ones free; some coated to form typical asbestosis bodies. At 1 mo. after injection, alveoli contained good-sized giant cells; most phagocytes were within air spaces and had not migrated to walls. At 4 mo., free intracellular fibers had worked themselves into interstitial tissue, where there was extensive proliferation of lymphoid cells and monocytes but no fibrosis. At 9 mo. foreign body reaction with some pneumonitis, no bronchiolitis. Typical asbestosis bodies present.
Crocidolite (Bolivia)	Long fiber, 20-60 microns	Advanced fibrous endobronchiolitis and peribronchiolitis. Refer to table 13.
	Short fiber, 20 microns and less	No fibrosis. At 1 mo., air spaces unobserved and largely filled with giant cells packed with dust needles. Walls heavily infiltrated with monocytes and lymphoid cells. At 4 mo., a moderate degree of cellular infiltration of walls; small giant cells packed with dust spicules. At 6 and 9½ mo., masses of giant cells, containing mineral particles, in small bronchi but not in respiratory bronchioles; monster cells widely scattered in terminal air spaces. Numerous asbestosis bodies. No reaction in connective tissue. No endobronchial proliferation. At 12 mo., many scattered small monocytes packed with dust. No endarteritis. No peripheral fibrosis. In lymph node, slight reticulosis; no fibrosis.
Anthophyllite	Long fiber, 20-60 microns	Lymphocytic infiltration and giant cells but no definite fibrosis. Refer to table 14.
	Short fiber, 5 microns and less	No fibrosis and practically no asbestosis bodies. At 1 mo., focal collections of dust-filled monocytes and a few giant cells; at 4 mo., some adenomatoid epithelial reaction; at 9 mo., simple pneumonitis with phagocytosis of short fibers; at 12 mo., isolated and sharply localized collections of dust cells inside air spaces about terminal arterioles. Reaction in walls limited to lymphoid cell infiltration. No fibrosis. In lymph node, reaction limited to slight prominence of reticulum.
Tremolite	Long fiber, 20-60 microns	Fibrosis about bronchioles. Refer to table 13.
	Short fiber, 20 microns and less	Simple foreign body reaction. No acute inflammation. No accumulation of dust in or about terminal bronchioles. No endobronchiolitis. At 1 mo., scattered small giant cells and considerable infiltration of adjacent walls with monocytes and lymphoid cells. At 4 mo., little change except more cellular infiltration of connective tissue. At 6 mo., lymphoid infiltration and thickening of walls about some but not all terminal bronchioles.
Erucite	Long fiber, 20-60 microns	Typical fibrous endobronchiolitis and peribronchiolitis like reaction to asbestos minerals. Refer to table 15.
	Short fiber (made by crushing long fibers with rubber policeman)	Inert type of reaction. At 1 mo. after injection, small monocytes widely scattered through air spaces; focus of atelectasis with lymphoid infiltration of compressed air-space walls. No endobronchial reaction as with chrysotile. At 3 mo., reaction similar to that at 1 mo.; typical asbestosis bodies seen. At 12 mo., small clumps of inactive dust-filled phagocytes; no fibrosis. No reaction in lymph nodes

Comparison of Long Fiber and Short Fiber Dusts.—With quartz dust it has been demonstrated that the smaller the particles the more

intense is the tissue reaction and that particles larger than 3 microns in diameter cause little reaction. In the case of asbestos, however, the reverse is true and apparently only long fibers have any specific effect, as was suggested by the inhalation experiments. This is confirmed by the data of table 16, in which a series of tests with fibrous minerals is reported. When the injected dust consisted of fibers 20 to 50 microns long, all the fibrous minerals tested except anthophyllite, as noted in the preceding section, produced fibrosis; when the material was prepared by first grinding the fibrous dust until the length of fibers was reduced to 20 microns and less or, in some cases, to 3 microns and less, none of the injected dusts caused fibrosis.

These results differ from those of King, Clegg and Rae,[11] who reported the production of reticulosis comparable to the experimental silicotic nodule in rabbits receiving monthly intratracheal injections of 100 mg. of Rhodesian asbestos fibers, 15 microns long, and the production of diffuse interstitial fibrosis in rabbits receiving similar injections of short fibers, 2.5 microns in length. We believe this dose, especially in the long term rabbits, is highly excessive. In our experiments the dosage was kept low in order to minimize untoward reactions which might obscure the peribronchiolar type of fibrosis which characterizes early human asbestosis.

Experiments Using Intravenous Technic

The experiments summarized in table 17, in which the intravenous method of injection was employed, show that the asbestos minerals are far different from quartz in their action on tissue. It has been repeatedly demonstrated that intravenous injection of quartz particles 3 microns and less in diameter will cause a typical tissue reaction with the development of hyalinized fibrotic lesions in extrapulmonary sites, such as the liver and the spleen. Asbestos minerals, however, on intravenous injection generally produce only an inert type of reaction, as is revealed by the results given in the table. The reason for the early deaths in the experiment with chrysotile particles is not clear.

Experiments Using Intraperitoneal Technic

The results of injection experiments with the intraperitoneal technic are given in table 18. It will be noted that the long fiber dusts produced a fibrous reaction while dusts composed of particles 3 microns and less in size caused only an inert type of response. These experiments indicate also that the fibrosis initiated by the irritation of asbestos fibers is not restricted to the lungs, as was formerly assumed, but can be produced in the peritoneum as well.

Other Experiments with Asbestos Minerals

A number of additional experiments were conducted to throw more light on specific phases of the asbestosis problem.

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11. King, E. J.; Clegg, J. W., and Rae, V. M.: Effect of Asbestos, and of Asbestos and Aluminum, on Lungs of Rabbits, Thorax 1:138, 1946; abstracted, Indust. Hyg. Digest, 1947, vol. 11 (Feb.), no. 234.

TABLE 17.—*Summary of Injection Experiments by Intravenous Technic*.

Dosage: Total amount of dust was 1.0 Gm., divided into 20 equal doses (each dose was 5 cc. of a 1 per cent suspension) which were given twice a week for 10 weeks.

Mineral	Size of Dust Particles	Rabbits Used	Maximum Survival After Last Injection, Mo.	Results
Chrysotile (Thetford)	3 microns and less (ball milled 132 hr.)	6	..	The rabbits did not tolerate intravenous injections of finely ground chrysotile; and 5 of the 6 died after 1 to 5 injections of even diluted suspensions; the other animal died after 17 injections of one-quarter strength suspension (52 days after first injection). Reaction limited to few large giant phagocytes of inactive type in liver, spleen and lungs. No thrombi of dust cells seen in pulmonary capillaries. No definite explanation for fatalities discovered, but material may have been retained in heart, causing fatal thrombi.
Amosite	3 microns and less (ground in agate mortar)	8	47	Advanced pulmonary infection killed 3 animals at 9, 11 and 17 mo. after last injection, shortening intended duration of experiment and complicating picture. However, rabbits killed earlier (2 and 6 mo.) showed only inert phagocytosis with no progression in the 6 mo. animal. The last two (11 and 17 mo.) were probably the same although focal necrosis of the liver and amyloid of the spleen made interpretation difficult.
Crocidolite	3 microns and less (ground in agate mortar)	4	12	Reaction was that to an inert substance with no change in 12 mo. (Other observations at 2, 4 and 6 mo.) Simple phagocytosis of particles. No tendency to agglomerate and no change in adjacent tissues. Grinding the dust to sizes of 3 microns and under destroyed the fibrous structure of this mineral, and the injected material resembled plates rather than fibers.
Anthophyllite	3 microns and less (ball milled 1,100 hr.)	4	21	Reaction essentially that of an inert mineral. Observations made at 3, 6, 12 and 21 mo. Only suggestion of irritating properties manifested in spleen and lymph nodes, but not liver, of the 21 mo. rabbit. In this animal there had been proliferation of mononuclear and giant cells that was not present in either spleen or lymph node of 12 mo. animal. The absence of associated fibroblastic reaction in these organs and of any change in the liver condition justifies the classification of anthophyllite as an inert silicate. No fibers were retained in lung to demonstrate whether asbestosis bodies would develop.
Tremolite (soda-iron)	3 microns and less (ball milled 140 hr.)	4	20	Reaction essentially that of an inert mineral. Last animal killed showed a little proliferation and lymphocytic infiltration in liver, but none earlier (at 3, 6 and 12 mo.). No evidence of any activity in lesions in other organs.
Tremolite (soda)	3 microns and less (ball milled 45 hr.)	4	24	An inert foreign body reaction with no change in 24 mo. Observations made at 3, 6, 12 and 24 mo.

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TABLE 18.—*Summary of Injection Experiments by Intraperitoneal Technic*

Dosage: Each animal * received a single intraperitoneal injection of 2 cc. of a 10 per cent dust suspension. Total amount of dust injected was 0.2 Gm.

Mineral	Size of Dust Particles	Guinea Pigs Used	Maximum Survival After Injection, Mo.	Results
Chrysotile (Thetford)	3 microns and less (ball milled 210 hr.)	15	30	No fibrosis or asbestosis bodies. Dust particles ingested by phagocytes, chiefly multinucleated variety. Six mo. after injection fibrous elements of dust appear to have dissolved leaving only the insoluble magnetite, a contaminant. No reaction in surrounding fat or areolar tissue. No transporting of dust to regional lymph nodes. Observations made at intervals from 1 to 30 mo. after injection.
Chrysotile (Thetford)	Long fiber (through [illegible] mesh)	4	2	Definite fibrous reaction produced, delicate and nonhyaline. Atypical asbestosis bodies developed, but all were unusually small. No evidence of extra long fibers seen. Observations only at 2 mo.
Amosite	5 microns and less (ground in agate mortar)	6	12	Infection interfered with interpretation. Dust reaction appeared to be of inert type and limited to phagocytosis with a moderate tendency to lymphocytic infiltration. Observations at 1, 4, 8 and 12 mo.
Crocidolite	5 microns and less; also some long spicules (ground in agate mortar)	7	12	Dust foci consisted only of large mononuclear and giant phagocytes surrounded by a minimum amount of cellular connective tissue. The injected dust contained not only fine material that in grinding had been mashed into irregular plates but also many long spicules 10 microns or more in length. No asbestosis bodies seen, although the longer spicules appeared slightly swollen and granular. Observations at 1, 4, 8 and 12 mo.
Anthophyllite	3 microns and less (ball milled 1,400 hr.)	9	26	Essentially inert foreign body reaction. In early animals (1, 4 and 8 mo.) focus of monocytes and small giant cells and a little central necrosis. In the 8 mo. animal there was also slight peripheral fibrosis. At 12 and 26 mo., nonprogressive mass of monocytes and giant cells; no fibrosis.
Anthophyllite (originally labeled talc)	Mostly 3 microns and less; some fibers 20 microns or more long (ground in agate mortar)	8	13	Reaction, which consisted of very large giant cells surrounded by a variable number of lymphocytes, was much heavier to these unintentionally long fibers than to the fine dust in the experiment above. There was more or less proliferation of fibroblasts producing cellular connective tissue visible in areas where the quantity of foreign particles was not so great that it obscures the reaction. Observations at 1, 4, 8 and 13 mo.
Tremolite (soda-iron)	3 microns and less (ball milled 15 hr.)	6	12	Inert type of response never progressing beyond the stage of very slight lymphocytic reaction about masses of dust-filled phagocytes. No fibrosis. Observations at 1, 4, 8 and 12 mo.
Tremolite (soda)	3 microns and less (ball milled 48 hr.)	6	18	Inert nonprogressive foreign body type of reaction. No fibrosis. Observations at 1, 4, 8, 12 and 18 mo.
Anthophyllite	100 microns and less	6	12	Distinct early fibrosis produced by anthophyllite and fibrous pyrophyllite with subsequent regression; crystalline pyrophyllite inert throughout. At 1 mo., giant cells about long [illegible] spicules; at 4 mo., definite fibrosis replacing giant cells of anthophyllite and fibrous pyrophyllite reaction; at 8 mo., fibrosis which started at 4 mo. had decreased, especially with fibrous pyrophyllite. At 12 mo., reaction to all three dusts consisted of foreign body giant cells with lymphocytes but without necrosis or fibrosis. No asbestosis bodies.
Pyrophyllite (fibrous)	100 microns and less	8	12	
Pyrophyllite (crystalline)	100 microns and less	6	12	

* Each animal receiving long fiber chrysotile was given an injection of 2 cc. of a 0.5 per cent dust suspension.

Protective Action of Aluminum Compounds

When colloidal aluminum hydroxide had been added to a suspension of long fiber chrysotile prior to injecting this suspension intratracheally into rats, the aluminum compound did not prevent the irritation of tissue due to chrysotile. If anything, the acute inflammatory response evoked by the injected fibrous mineral was accelerated. One month after the last injection of the dust suspension the bronchiolitis was becoming fibrous. King and his associates also found that aluminum failed to protect pulmonary tissue from the irritation caused by asbestos fibers[12]; in their experiments metallic aluminum was used instead of the hydroxide.

Formation of Asbestosis Bodies

The iron in the coating of the asbestosis body appears to be derived from blood or tissue elements and not, as has been suggested, from the mineral fiber. After two kinds of chrysotile were injected subcutaneously into the groin of a guinea pig—one kind containing 2 per cent and the other 0.2 per cent ferric oxide—the asbestosis bodies were equally numerous at both sites of injection and showed no difference in their reaction to prussian blue, the reagent which stains iron. This finding is in agreement with that of Giroux.[12]

Tissue Reaction to Asbestosis Bodies

Asbestosis bodies recovered from human lung tissue and injected intratracheally into guinea pigs failed to produce a fibrous reaction. The material for injection was obtained by digesting with sodium hypochlorite solution the lung tissue removed at autopsy from an asbestos worker. The asbestosis bodies could be seen in the guinea pigs for at least a year after injection. This experiment shows that the asbestosis body has a rather resistant coating which is not destroyed by moderate hypochlorite treatment, which may be maintained in vivo for a year or longer and which renders the fiber incapable of producing fibrosis. It thus appears that the coating is a protective mechanism. This thought was expressed by Beintker as early as 1934.[13]

THEORY OF IRRITANT ACTION

Two hypotheses have been proposed to explain the tissue irritation and reaction caused by asbestos fibers: the chemical and the mechanical. In the chemical theory, which is based on experience with quartz, it is assumed that the asbestos minerals dissolve in the body fluids and that in this process their bases are leached away to leave silica in a form capable of irritating tissues. According to this hypothesis asbestosis is merely an indirect silicosis. Several facts make the chemical theory untenable: Intratracheal injection of brucite fibers, which had a silica

12. Giroux, M.: Amiantose expérimentale: valeur pathognomonique du "corps l'amiante," Laval méd. 8:239, 1943.

13. Beintker, E.: Über die Asbestosiskörperchen: Bemerkungen zu der Arbeit von Beger, Virchows Arch. f. path. Anat. 293:527, 1934.

content of only 0.90 per cent, caused typical fibrosis like that produced by the asbestos minerals; free silica particles increase in potency as the particle size becomes less, but asbestos fibers shorter than about 10 to 20 microns are relatively innocuous; aluminum hydroxide neutralizes the irritating effect of quartz but not of asbestos; serpentine has the same chemical composition as long fiber chrysotile, but it produced only an inert type of tissue reaction; there is a wide range in the chemical composition of the minerals which do cause asbestosis (table 19). In view of this evidence it seems more likely that asbestosis is caused by an unusual mechanical irritation due to long asbestos fibers, this irritation being related to the peculiar filamented structure of the fiber and the associated flexibility, which are possessed by no other foreign body studied. Thus, ignition of chrysotile fibers changed their structure and made them inert, although the same fibers, before being heated, would have produced fibrosis (table 14). Further support for the theory of mechanical irritation is that asbestosis occurs in an organ of high mobility —the lung—and that a fibrous reaction can be produced by injecting

TABLE 19.—*Analyses of Fibrous Minerals*

Fibrous Minerals	SiO_2 %	Fe_2O_3 %	FeO %	Al_2O_3 %	CaO %	MgO %	Na_2O %	K_2O %	Ignition < 105° C. %	Loss > 105° C. %	Total %
Amosite	50.88	4.06	30.85	1.89	2.01	0.25	0.07	0.20	0.06	0.28	99.97
Amphibole	53.04	3.06	.....	1.62	12.22	23.20	0.23	0.32	0.82	3.80	99.77
Anthophyllite	59.80	0.87	.....	0.82	0.44	28.37	0.21	0.16	0.27	4.08	99.52
Bruciti	0.09	0.78	0.86	0.16	0.01	31.80	0.01	0.06	0.03	26.80	99.70
Chrysotile	38.54	2.28	.....	0.34	0.02	34.01	0.20	0.05	1.48	14.89	99.89
Crocidolite	54.90	18.27	4.29	1.91	0.59	12.21	6.91	0.67	0.02	0.34	99.69
Tremolite	58.20	7.22	.....	0.46	6.16	29.02	0.15	0.09	0.34	1.78	99.82

asbestos fibers into the peritoneum, where there is also a degree of mobility, but not by injecting them into other extrapulmonary organs such as the liver, the spleen and subcutaneous tissue.

COMPLICATIONS

The experimental investigations with asbestos minerals were concerned primarily with the effect of the dust on normal tissue, but some attention was given to other phases, such as susceptibility to infection. The only experiment in which the effect of asbestos dust on a pulmonary infection was studied was the first inhalation experiment, carried on with King's floats dust. It is unfortunate that, owing to the lack of adequate facilities at that time, infection studies could not be made in the other inhalation experiments also.

SUSCEPTIBILITY TO TUBERCULOUS INFECTION

The development of a tuberculous process initiated at the beginning of exposure to dust, and also of a tuberculous infection superimposed on an established asbestosis, was described in preceding sections of this paper. It may be stated that asbestos when classified according to the effect of a dust on tuberculous infection would be placed below an active

dust like quartz but above an inert dust such as iron oxide. In animals infected with attenuated tubercle bacilli, quartz causes the infectious process to progress until the animal dies of tuberculosis. Inert dusts have no effect on the infection, and the lesions usually heal and the disease disappears. Asbestos dust is in a different category. In the experimental investigation, when the fibrous dust was being inhaled during the evolution of the infection, there was spreading of the tuberculous process for a time, but usually the stimulus for continued proliferation of the tubercle bacilli was not sustained, the progression was arrested and healing followed. In guinea pigs infected with attenuated tubercle bacilli after being exposed to asbestos dust for slightly more than two years, progressive disease did not develop. The only modification of the infection was one of localization, a few bacilli being retained in the fibrous terminal bronchioles and forming tubercles there, in addition to the usual foci beneath the pleura. Such tubercles healed in a few months.

Susceptibility to Nontuberculous Infection

There was no specific experiment concerning the effect of inhaled asbestos dust on nontuberculous infection. Intercurrent pneumonia was rather common among animals exposed to asbestos dust, the frequency in guinea pigs exposed in the four inhalation experiments ranging from 16 to 39 per cent. This incidental evidence suggests the possibility of an effect of asbestos dust on nontuberculous infection. Nevertheless, since such epidemics are not uncommon in inhalation experiments with other dusts and even in the colony of normal animals, it is felt that the inhalation of asbestos dust does not exert a significant effect on the susceptibility to nontuberculous pulmonary infection.

Comment and Summary

Owing to the vast amount of data included in this investigation, it seems most convenient to summarize and to state as concisely as possible the various observations which emerged from the experiments and to follow each with a brief résumé of the evidence.

A. Various species of animals, including the guinea pig, the rat and the rabbit, but not the mouse and the dog, develop peribronchiolar fibrosis of the lung similar to human asbestosis after being exposed by inhalation or intratracheal injection to long chrysotile asbestos fibers.

Both inhalation and injection experiments provide ample support for this statement. Figure 8.1 reveals the cellular fibrosis that occurs in guinea pigs following inhalation of long fiber asbestos; figure 9 shows the fibrosis caused in the cat by inhalation of long fiber asbestos dust. Similar but less extensive fibrosis occurred also in rats and rabbits (table 1). Mice and dogs failed to respond. This variation in response of different species to identical dust exposures is still to be accounted for.

B. Long asbestos fibers are essential in the production of the peribronchiolar fibrosis; short fibers are incapable of producing this reaction

Inhalation experiments with asbestos dust suggest, and intratracheal injection experiments confirm, that peribronchiolar fibrosis is produced by asbestos fibers between 20 and 50 microns in length but not by particles shorter than 20 microns (tables 16 and 18). This indicates that the minimum length of fiber possessing the capacity to produce the typical peribronchiolar fibrosis in animals is somewhere between 20 and 50 microns. Pointed studies have not been carried out to determine the upper limit of effective fiber length. It appears, however, that that limit will be determined by the inhalability of the fiber.

C. The mode of action of the long asbestos fiber in the production of asbestosis is primarily mechanical rather than chemical in nature.

The evidence for this conclusion has been reviewed in a preceding section, page 39. The flexible filamented structure of asbestos fibers plays an essential part in the irritating action, since the solid, inflexible fibers of glass wool do not produce fibrosis (fig. 13 *B*).

D. Typical experimental asbestosis was produced by the inhalation of an atmospheric suspension containing an average of 138 million asbestos particles per cubic foot of air by light field count, of which less than 1 per cent consisted of fibers longer than 10 microns.

In the inhalation experiment with 100 per cent ball-milled asbestos dust containing 0.6 per cent of fibers longer than 10 microns (table 11) typical fibrosis was obtained (table 9). The evidence presented shows at least that an atmospheric concentration of asbestos dust containing less than 1 million (0.6 per cent × 138 million) fibers longer than 10 microns per cubic foot of air is capable of producing experimental asbestosis in guinea pigs. The actual lower limit of concentration of long fibers necessary to produce asbestosis in animals cannot be established from these studies.

E. The duration of exposure required to develop the pulmonary reaction to inhaled asbestos dust is inversely proportional to the concentration of long fibers in the atmosphere; as the concentration is increased, the reaction develops in shorter time.

The basis for this statement appears in the data of the inhalation experiment with long fiber asbestos. For that experiment the average concentration of the atmospheric dust was about 40 million particles per cubic foot of air, and size-frequency determinations disclosed that 6.7 per cent of the air-suspended material consisted of fibers longer than 10 microns (table 11). Thus, by calculation, it is estimated that the concentration of the longer fibers was 2.7 million (6.7 per cent × 40 million). The lungs of animals exposed to the long fiber asbestos dust revealed that the pulmonary reaction developed in approximately one-half the exposure time required for its development in animals inhaling the ball-milled product, for which the concentration of the longer fibers was only 0.8 million (0.6 per cent × 138 million).

F. Established experimental asbestosis ceases to progress on discontinuance of dust exposure.

The experimental investigation shows, in fact, that on discontinuance of exposure there was an appreciable clearing of the mature pulmonary

lesions, due to contraction of the fibrous tissue. In contrast, an immature tissue response, evidenced primarily by cells with little or no fibrosis, continued to progress. It is assumed that, following attainment of fibrotic maturity, the same process of contraction would ensue as was noted for the mature lesion.

G. The formation of asbestosis bodies represents a coating of the fibers by blood and tissue elements, which results in loss of ability of the fiber to produce fibrosis.

Intratracheal injection of asbestosis bodies failed to produce the typical asbestotic tissue reaction in experimental animals. The cessation of progressive reaction observed soon after exposure terminates may be due to the formation of asbestosis bodies.

H. Aluminum hydroxide failed to neutralize the fibrosing action of the long fiber asbestos.

Aluminum hydroxide added to the suspension of chrysotile asbestos prior to intratracheal injection did not retard or prevent the development of asbestosis in rats.

I. Inhalation of asbestos dust did not alter significantly the final outcome of experimental tuberculosis in two series of guinea pigs exposed to the dust.

The apparently mild influence of asbestos dust is in distinct contrast to the stimulating effect exerted by inhaled quartz on a tuberculous process in the lung. The interpretation must remain tentative, however, since it is based on an investigation limited to two series of guinea pigs exposed to only one kind of asbestos, namely, King's floats: Table 4 shows that when the infection was coincidental with the onset of dust exposure, there was temporary progression of the infectious process, with subsequent healing; when infection was initiated after 26 months of dust exposure, the course of the tuberculosis was not appreciably altered. The latter finding is quite different from our usual experience with quartz dust or with mixed dusts containing quartz, wherein the adverse influence of quartz on a tuberculous infection is manifested most strikingly when infection is initiated after a period of dust exposure, viz., superimposed on a background of established silicosis. As indicated above, this more sensitive test, when applied to asbestos dust, failed to demonstrate that the latter had an adverse influence on a tuberculous infection. The inability of asbestos dust in that experiment to affect unfavorably the tuberculous process furnishes strong support for the interpretation that inhaled asbestos dust has no more than a mildly unfavorable effect on pulmonary tuberculosis.

Acknowledgment is gratefully made to the group of companies of the asbestos industry whose generous financial support made this investigation possible.

Printed and Published in the United States of America

MEDICAL DEPARTMENT

L H. Evans

Pls bring CTBM up to date on this & indicate we have tentatively agreed to participate -- after we meet with govt people in Washington

DT 5/27

May 25, 1965

ASBESTOS STUDY

Phone Cincinnati Area 513, 684-2689

Mr. Donald K. Rennie
Vice President
Brakeblok - Troy Office

Dear Don:

As I mentioned to you on the telephone today, the U.S. Public Health Service visited with the Medical and Hygiene Departments on 5-24-65. Their representatives, William S. Lainhart, M.D. and Lewis J. Cralley, Ph.D., presented their problem, namely, the study of asbestos among the major users of it. They have already studied the textile industry in some detail and are now pursuing the realm of the friction materials group.

They have expressed a desire to study our Brakeblok operation with a detailed in-plant environmental or industrial hygiene survey. They indicate that they would like to cooperate with the Hygiene Department of Brake Shoe so that they could double check each other's results. After a thorough investigation including study of the chest x-rays of the employees, they would formulate their own conclusions and hope to determine whether or not there is any increased causal relationship between those exposed to asbestos in their normal work and those subsequently developing cancer of the lung. The results of the survey will be published in scientific journals but these articles will not identify the company or the plant. The results of their investigation will be made known to local management and our industrial hygiene group, but will not be divulged to the individual employee, his lawyer or physician.

I explained to Drs. Cralley and Lainhart that I would approach the American Brakeblok Division regarding this. They indicated that if the Brakeblok Division management had any questions, they would be happy to come and see you. I believe that these men are truly dedicated to their work and that they are pursuing this in a proper scientific fashion in an attempt to solve a rather controversial problem.

Meet'g set up for June 23rd 9:00 AM at Wind. with Messrs Drs Lainhart - Cralley and rep of State of Virginia Health Dept

DKR 6/1

PLAINTIFF'S EXHIBIT 101

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A64

MEDICAL DEPARTMENT

I shall write to Dr. Lainhart and indicate that we have discussed the subject and request him to find a mutually convenient time for you to become acquainted and explore the problem further and to your complete satisfaction. I hope that we will be able to cooperate fully and I feel there may be benefits to be had not only by such cooperation and increased liaison, but by furthering scientific knowledge.

They will require a questionnaire be completed on each employee. A lot of the data is undoubtedly present in the medical file at the plant and this would most certainly be better than the employee's recall of such events in his past. This could be completed on Company time or off the job. They indicated to me that approximately 20 minutes would be consumed in the completion of their Bureau of Budget #68-6402.

I am also enclosing, in addition to the above mentioned form, a resume of the proposed asbestos study. I would like to add that on page 4, "In-plant Medical Studies," that this would perhaps be an ideal way to conduct such a study but this is not necessarily nor by any means what will be required or performed insofar as such extensive testing.

I shall be interested in any further comments which you might like to make and developments that may occur.

C. C. Blackwell, Jr., M.D.
Medical Director
Enc.
CCB:mp
cc: Mr. M. B. Terry
Mr. R. B. Parker - NYO

JWK007730 010812

A65

INTERNAL CORRESPONDENCE

AMERICAN BRAKEBLOK DIVISION

TROY OFFICE

June 7, 1965

Mr. C. B. Mallory
Winchester

Subject: Asbestos Study by U. S. Public Health Service

Dear Charlie:

The American Brakeblok Division has been approached by the U. S. Public Health Service, through the Medical Department, to participate in an environmental or industrial hygiene survey that is being conducted in friction material industries using asbestos.

It has been tentatively agreed that we would participate, although it is not compulsory; however, before we set up dates we have made arrangements to meet in Winchester, June 23d, at 9 a.m. There will be Drs. Lainhart and Cralley from the U. S. Public Health Service who have requested a representative from the Virginia Health Dept., and myself. The purpose of the meeting is to acquaint us with their specific program and how it can best be accomplished without upsetting any of the employees and at the same time reduce any interference or plant interruptions to the minimum. They would like their survey to be independent of any of our medical department findings and conclusions; however, their findings would be checked with the findings of the Medical Department.

A part of the study is to determine if there is any increased causal relationship between those exposed to asbestos in their normal work and those subsequently developing cancer of the lung.

We should be able to work out a procedure which will be satisfactory to the U. S. Public Health Service and ourselves as it seems to be a worthy project. I will advise you later regarding my arrival.

L. H. Evans

/hm
cc: DKRennie
MBTerry
RBParker
CCBlackwell, M. D.

JWK007021

A66

October 17, 1968

Personal

Dr. C. C. Blackwell
Medical Director
Chicago

Dear Charles:

Attached hereto please find a copy of Johns-Manville Asbestos, Ltd.'s letter of October 1, 1968, pointing out that each bag of asbestos fibre will soon contain a caution label.

This could give us some repercussions at Winchester if our people working with asbestos get concerned about the hazard to their health.

Any comments you might give us in rebuttal to questions the employees might raise would be appreciated.

/mle
Encl.

D. K. Dennis

cc: M. B. Terry - Encl.
C. B. Mallory "
F. E. Taylor "
F. C. Milner "
J. R. Shepard "

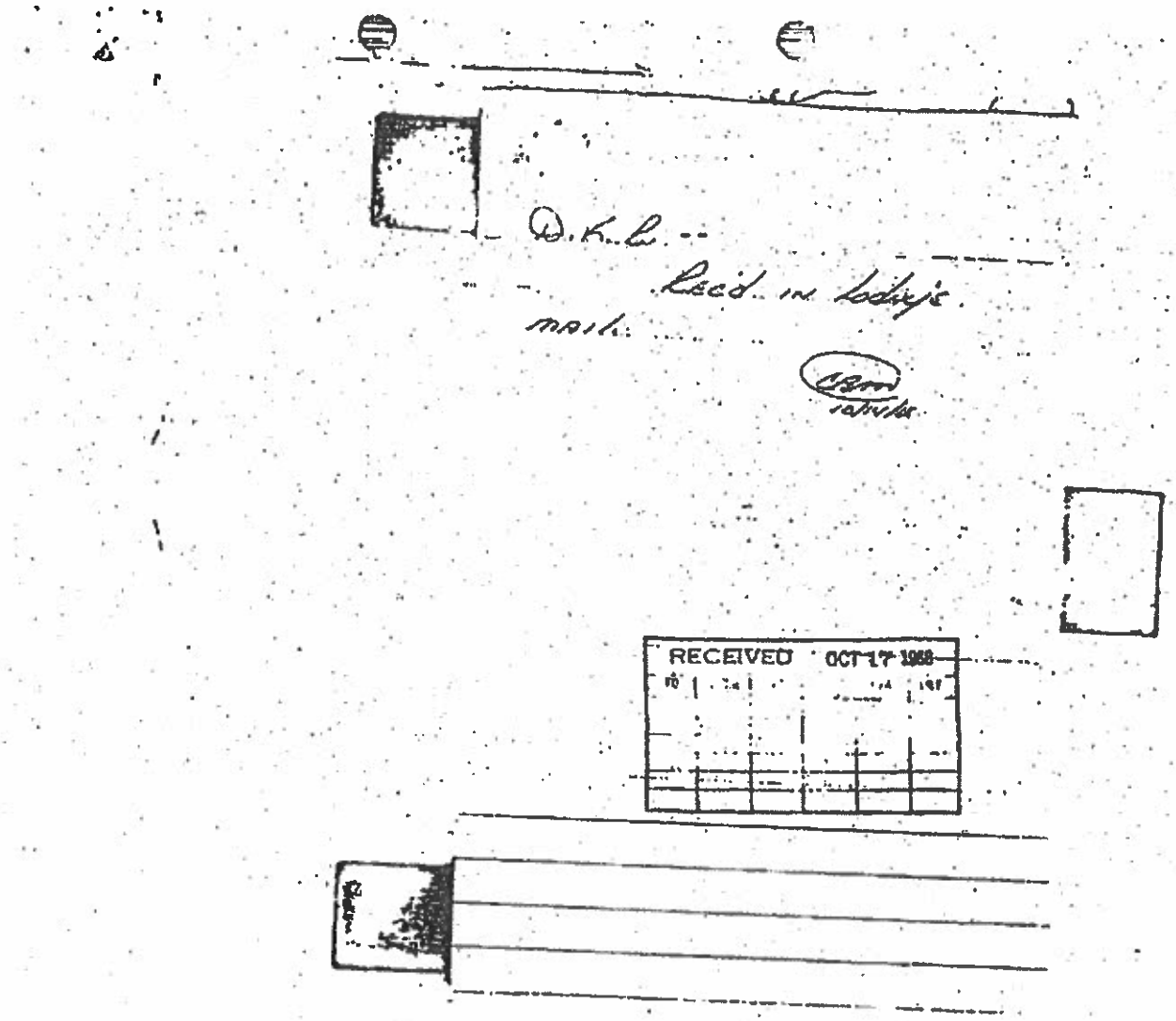
010767

PLAINTIFF EXHIBIT 187

JWK007710

CC6685

A67



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A68

CANADIAN Johns-Manville Asbestos LIMITED

ASBESTOS FIBRE DIVISION

P.O. BOX 1500 — ASBESTOS, QUEBEC — TELEPHONE: 879-5431

October 1st, 1968.

Gentlemen:

You will notice that beginning shortly each bag of chrysotile asbestos fibre shipped by this Company will carry a label reading as follows:

CAUTION

"This bag contains chrysotile asbestos fibre. Persons exposed to this material should use adequate protective devices as inhalation of this material over long periods may be harmful."

The label is intended to remind all industrial users of asbestos that proper handling will contribute to improved conditions in work areas.

Physical protection for employees is provided through the use of safety hats, shoes, glasses, and other devices when circumstances warrant. Health protection is just as important, and should include appropriate practices and equipment such as collectors, ventilators, masks, etc., to prevent inhalation of fumes and particulate matter.

As you know, in the past several years there has been increasing publicity and medical attention given to health effects of inhaling industrial dust and fumes of all kinds. Some studies have raised the question whether adequate control measures are being taken in certain industrial operations to prevent the inhalation of asbestos particles. Other studies have shown that where proper protective measures are taken, occupational health risks are minimized.

/ continued

010769

JWK007712

CC6687

A69

Medical research on health questions relating to asbestos is being sponsored by Johns-Manville, the Quebec Asbestos Mining Association, and several other organizations. Such research will lead to a better understanding and control of health hazards associated with inhalation of asbestos particles.

Until more concrete information is available from the above-mentioned research, we have concluded that it is in the best interest of all concerned that we place the above label on bags containing asbestos to encourage careful handling of the fibre.

If you have any questions, we would be pleased to hear from you.

Yours very truly,

N. W. HENDRY,
General Sales Manager.

NWH / oh

May 23, 1975

OSHA Plant Visit
May 20-22, 1975

On May 20, 1975 at approximately 9:00 a.m., 3 federal OSHA inspectors visited the plant and asked for Mr. C. B. Mallory. Their names were Mr. Pauley - Richmond, Virginia Office; Mr. Everett Stratton - Norfolk, Virginia Office and Mr. Kip Hartman - Roanoke, Virginia Office.

I went to C. B. Mallory's office and met the aforementioned group. Also in attendance were Mr. T. Ritter, Union President; Mr. D. Cooper, Union Chairman and Mr. Anthony Fedele, International Union Representative. R. G. Brown, C. B. Mallory, D. F. Johnson and C. L. Curtis represented Abex management.

The OSHA group outlined the purpose of the visit and briefly what they proposed to accomplish at this visit. They referred to the plant visit as a "STRATEGY (?) SESSION".

They stated that we (Abex) could permit or refuse the union to be present on the walk around tour and on subsequent test procedures. Abex expressed the opinion that the union representatives could at their option accompany the tour and witness the test procedures.

At approximately 1:30 p.m. the group met and a walk around tour of the plant facilities, including the boiler room and the Belt Iron Manufacturing Building, was made.

A short discussion then evolved about their (OSHA) plans to take asbestos level exposure samples and audio Dosimeter tests.

On May 21, 1975 at approximately 8:00 a.m. they again appeared at the lobby, and having brought their test equipment went out to the Book Press Preforming area and set up sampling of preform operators to determine their exposure to asbestos fibers. All filters will be sent to a Government lab in Colorado to get the dust count. They changed filters about every hour. As they changed filters they interviewed the employees concerning what management was doing to improve dust control, what was done in the past and company attitude towards healthy working conditions. They asked the employees about the procedure the company followed in giving physicals and x-rays. They questioned them about their feelings concerning working in a highly hazardous environment - asbestos. They also were probing into the history of illnesses and lost time from work to see what was attributable to hot or cold conditions, whether the employee thought dust or asbestos could have been the cause of illness or lost time, what did they think of the company's doctor and if their own physician had any opinions on working in an asbestos related industry.

I secured 5 blank forms of the questionairs they used in the interview with the employees.

JWK006304

C-2.

C06690

A71

Mr. Pauley and Mr. Hartman ran the studies in this department with Doug Cooper constantly being asked his opinions by Hartman. Hartman in my opinion is a very clever individual and at every opening he would mingle with Cooper in a corner. He required more following than did Pauley.

On May 22, 1975, they moved to Block Preforming and duplicated the previous days mode of operating.

Hartman would gather a group of employees, usually near control aisles or drinking fountains, and question them on their knowledge of asbestos hazards. He was told by most employees that, "One day they came to work and found mix boxes labeled, 'Asbestos Is Hazardous To Your Health, Do Not Breathe', and no one ever informed the employees why the labels were installed or what the hazard was".

He made quite a big thing of this and explained how bad an actor asbestos is.

On May 21, 1975, Everett Stratton ran audio Dosimeter tests in Strip Finishing and was accompanied by Tom Ritter and Don Johnson.

On May 22, 1975, Stratton took samples in the Block Finishing and Disc Brake Finishing Departments.

The results of the audio Dosimeter tests run on these two days is as follows:

May 21, 1975

Employee	Machine	Reading
Hamilton	SKA-395 Slitter	84
McConley	SKA-395 Slitter	88
Barrett	SKA-395 Slitter	85
Kinnie	SKA-421 O.D. Grinder	144
MacFarland	Auto-Chamfer	115
Ryan	I.D. Grinder Badger	291
Pinnell	#6, I.D. Grinder Badger	201
Baughman	SKA-200A	96

May 22, 1975

Woods	SKA-442	307
White	Besley	169
Link	SKA-442 #2 Finish Line	123
Shuvalter	SKA-539	200
Meadows	SKA-539	219
Gross	Disc Brake Belt Sander	81
Thacker	Disc Brake Drill	98
Berry	SKA-396	114
Sager	D.B. Printer	85

JWK006305

C06691

A72

The readings are those figures obtained by removing a loading cell from the Dosimeter and putting into a digital readout R100 DuPont unit. We are to be cited for violations where reading exceeds 100.

This group will return either the week of May 26th or June 2nd, in order to complete additional studies in Compounding and other departments.

C. L. Currie
Division Project Engineer

CLC/djt

FRICTION PRODUCTS GROUP
Winchester

(AIA)

December 15, 1978

Mr. E. F. Potts

Subject: Asbestos Warning Label

This will confirm our conversation regarding asbestos warning labels on all boxes and cartons shipped from Winchester.

You will determine which customers, if any, have requested we not label their product containers. These customers will be contacted through the Sales Department, who will inform them their request will be honored only if made by a member of their senior management in writing, specifically releasing Abex from this responsibility.

All such requests are to be directed to Mr. Iwarsson, who will determine whether each individual request will be honored. There will be no exceptions to the label rule unless authorized by Mr. Iwarsson.

L. F. Keenan

LFK:jt

cc: Messrs. B. Iwarsson
J. H. Barbazette

002173

attention

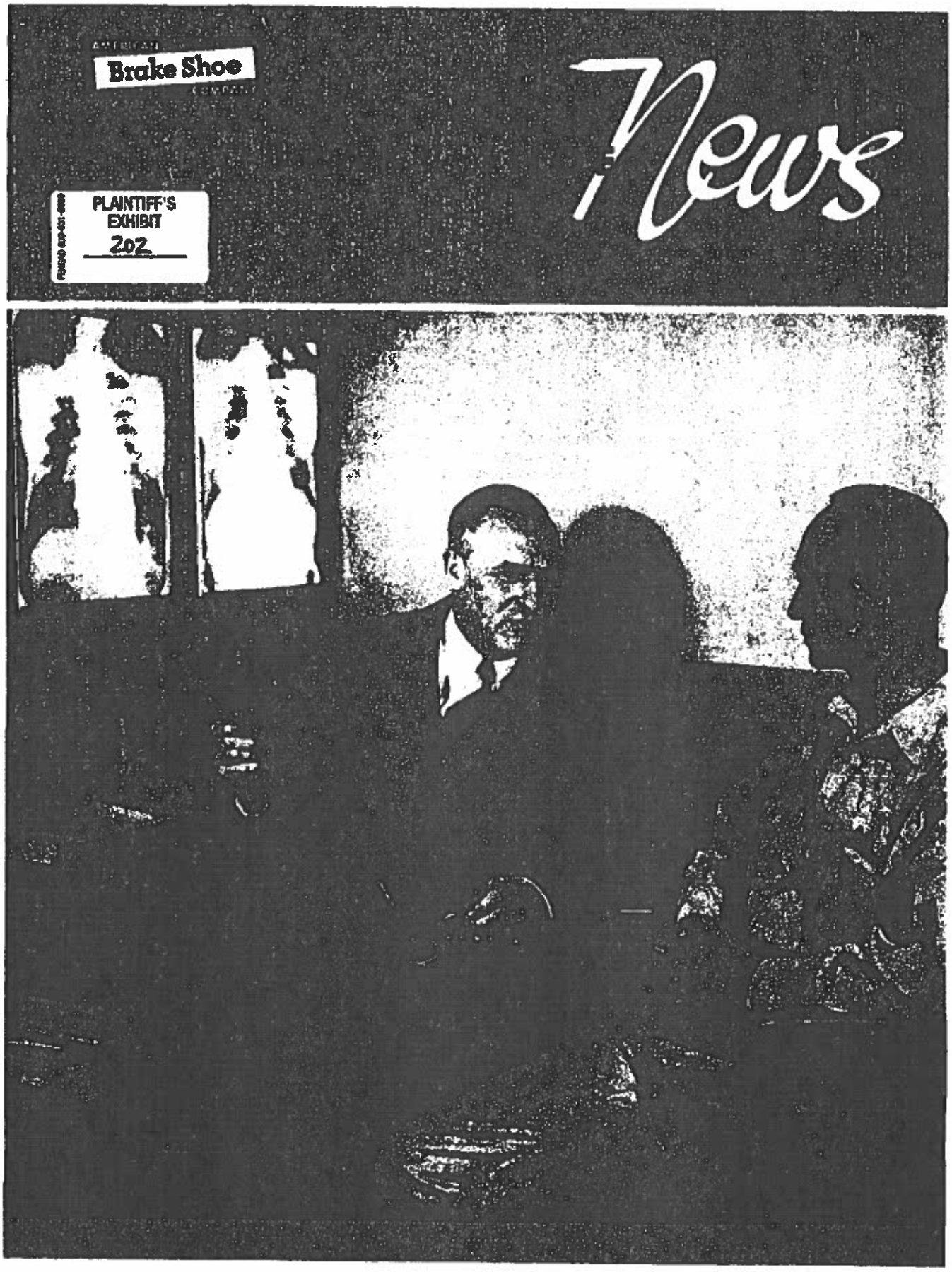
CONTAINS ASBESTOS

OBSERVE THE SAFETY RULES

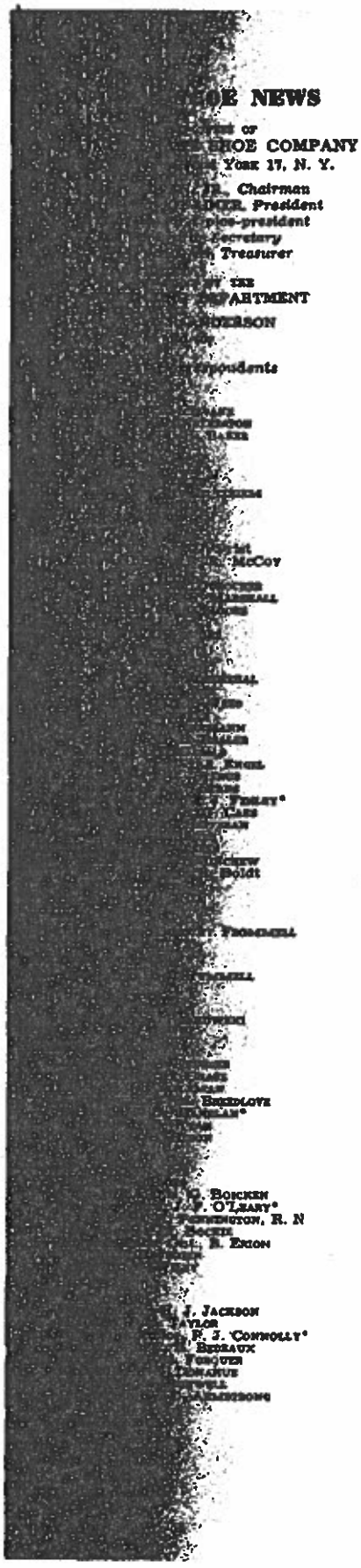
AVOID BREATHING DUST

002174

A75



A76



Editor's Corner

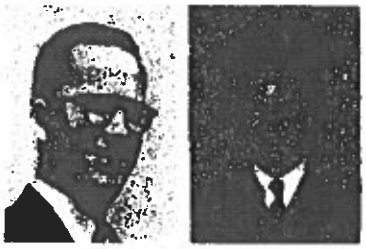
One of the questions most frequently asked us is, How do you find enough news to fill up a magazine every month?

Our answer to this question is easy: We have more news than we can ever hope to use. From 10,000 employees spread over 57 different plants and sales offices, there is enough going on all the time to fill a magazine twice the size of the News. Most of the news items are naturals. They are the events that go on all the time in a progressive, modern company like ours, and these merely have to be reported. They're the items about new equipment, products, plants, etc. Then there are the articles on the various benefits which the company provides for all employees. (There are more than 25 different ones, you know.) Reports on group insurance, retirement plans, safety programs, etc. These affect all employees in some way or other, and so we try to keep you up to date on any new development about them, or translate policy jargon into readable English.

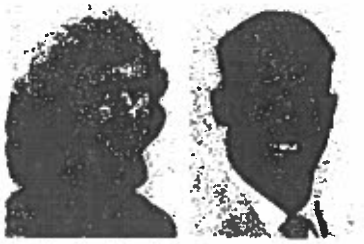
But perhaps the most interesting and most varied news we report every month is the stories of you, the people. Journalists all know that people make the best stories, or as they say, "people make news." Hardly a day goes by when our mail doesn't report news of an employee who has a unusual hobby, performed a useful community service, or just had a heck of a good time at a favorite pastime. All these make lively, readable features, and give some insight into the people of our company. These stories are usually reported by your plant correspondent upon whom we depend quite a lot for news of our readers.

So when it comes time to publish all the news about Brake Shoe each month we don't wonder *how* we're going to fill up 20 pages with news and features. We usually spend hours deciding *where* we can put all the news and pictures we have on hand. Yes, people make news, and at Brake Shoe they make it with a capital N. And as long as you people make the news, we'll continue to report it.

More June Graduates—



William Weimer — Billy Jones



Dolores Maleski — Nathan Redditt

Reports of five more 1953 graduates have been received by THE NEWS. Included at the end of the commencement procession are one Company employee and one newcomer to Brake Shoe. William Weimer, Purchasing New York, received his bachelor of science degree from New York University's School of Commerce, Accounts and Finance. Billy Jones, son of Homer Jones, BS&C Portland, graduated from Benson High School and is now working at the Portland plant. Other graduates are Nathan Redditt, son of Sammie Redditt, BS&C Portland, who graduated from Jefferson High School; Elizabeth Arias, daughter of Frank Arias, Wheel Houston, who graduated from Jefferson Davis High School; and Dolores Maleski, daughter of Ted Maleski, Amsco Chicago Heights, who graduated from Bloom Township High School.

On the Cover:

Dr. Lloyd E. Hamlin, medical director of American Brake Shoe Company discusses a chest x-ray with a Brake Shoe employee in Chicago as part of his regular medical treatment under the company's program of medical attention which is described on Pages 4 through 6 in this issue.

WORK: the master key

EVERY so often I come across in my readings something which seems to express so well the feeling we all share about a particular idea. Recently, while reading a memorial address about James Buchanan Duke, founder of the American Tobacco Company, who endowed Duke University with great funds, his explanation of his success stood out as particularly significant and worth repeating.

"I have succeeded in business," said Mr. Duke, "not because I have more natural ability than those who have not succeeded in business but because I have applied myself harder and stuck to it. I know plenty of people who have failed to succeed in anything who have more brains than I had but they lacked application and determination."

There you have the master key that opened the door of success for Mr. Duke as it has for so many successful men and women. The master word is work. And further on in this same speech there is a surpassing tribute to work written by Dr. Osler which is the key to most successes. He said to a graduating class:

"It seems my bounden duty on such an occasion to be honest and frank, so I propose to tell you the secret of life as I have seen the same game played, and as I have tried to play it myself. You remember in one of the Jungle Stories that when Mowgli wished to be avenged on the villagers he could only get the help of Hathi and his sons by sending them the master word. This I propose to give you in the hope, yea, in the full assurance, that some of you at least will lay hold upon it to your profit. Though a little one, the master word looms large in meaning. It is the open sesame to every portal, the great equalizer in the world, the true philospher's stone, which transmutes all the base metal of humanity into gold. The stupid man among you it will make bright, the bright man brilliant, and the brilliant student steady. With the magic word in your heart all things are possible, and without it all study is vanity and vexation. The miracles of life are with it; the blind see by touch, the deaf hear with eyes, the dumb speak with fingers. To the youth it brings hope, to the middle-aged confidence, to the aged repose. True balm of hurt minds, in its presence the heart of the sorrowful is lightened and consoled. . . . Not only has it been the touchstone of progress, but it is the measure of success in everyday life. Not a man before you (on the rostrum) but is beholden to it for his position here, while he who addresses you has that honor directly in consequence of having had it graven on his heart when he was as you are today. And the master word is *Work*, a little one, as I have said, but fraught with momentous sequences if you can but write it on the tablets of your heart and bind it upon your foreheads."

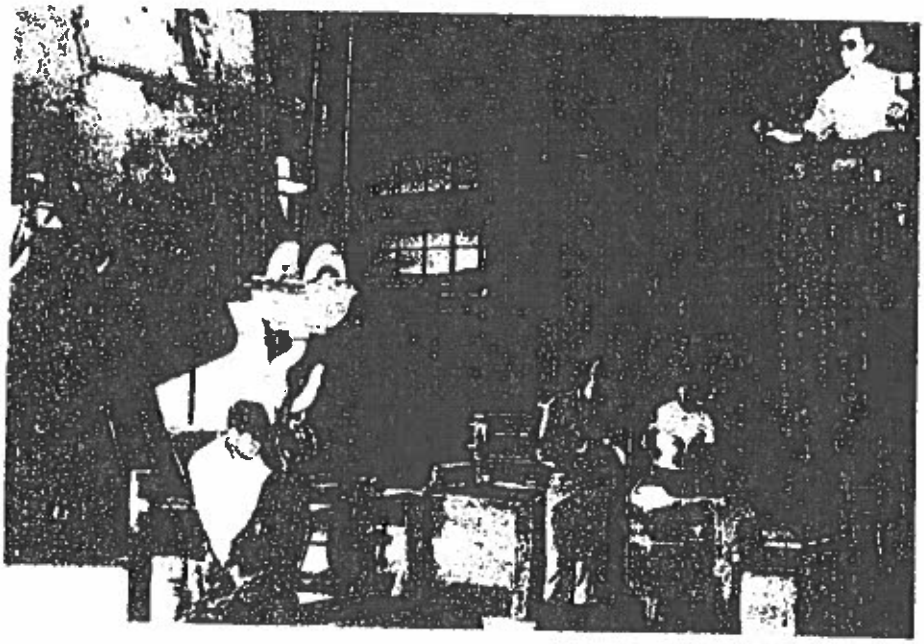
Wm. B. Given Jr.
Chairman

PREPLACEMENT physical examination assures Andy's physical fitness for his job and would uncover conditions which might later develop into more serious ailments. Here Dr. T. J. Bonick checks Andy's blood pressure.

SAFEGUARDING YOUR HEALTH

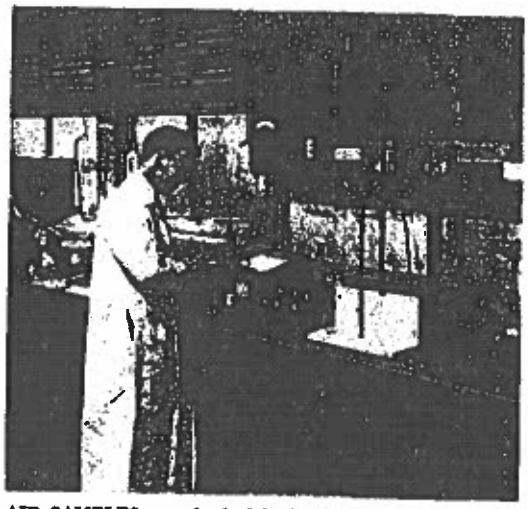
THE AVERAGE INDUSTRIAL worker loses less than seven days a year because of illness. On the whole, we are a healthy, energetic group of people and according to statistics by public health groups, we are growing healthier every year. Modern medicine and protective health measures have reduced the severity of most human ailments, and in recent years modern developments in drugs and biotics have cut down drastically the number of days of recuperation from illnesses that were considered serious years ago. Much of the credit for reducing the absence frequency of industrial workers goes to company medical programs.

Brake Shoe's Medical Department is constantly searching for new and better methods to safeguard your health as well as to provide the routine medical services. Under the direction of Dr. Lloyd E. Hamlin, our Medical Department, which is located in Chicago, includes an X-ray Department, a complete Industrial Hygiene Laboratory and a supervisory nursing section. Industrial nurses are employed at all but a few Brake Shoe plants. The Medical Department works in close conjunction with plant management, employees, Industrial Nurses and outside health, hygiene and medical organizations. To illustrate the extent of our medical program we followed Molder Andy Ross of Amsco Chicago Heights, through the complete medical services routine.

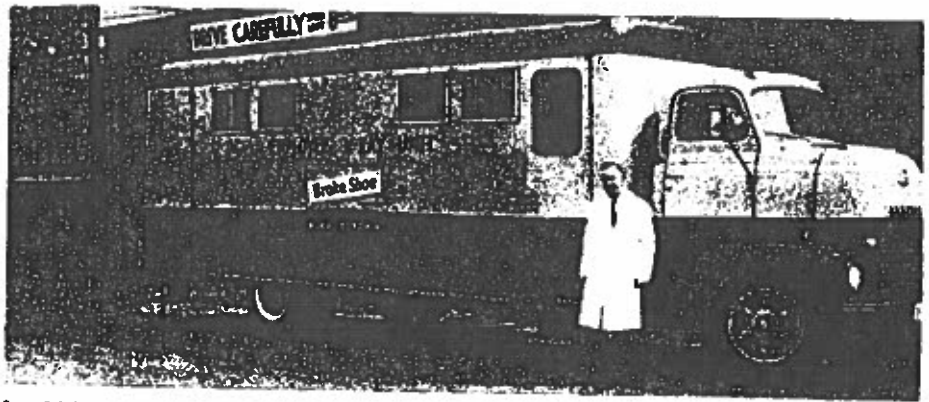


WORKING CONDITIONS are checked constantly by the Medical Department. Here Industrial Hygiene Technician Don Carlson (*center*) takes air samples from Andy's working area. Andy is shown in crane cab, upper right.

NURSES are able to handle routine medical problems like first aid as well as most emergencies in well-equipped first aid rooms, virtually miniature hospitals. Here Louise Matthews, R.N., attends a minor injury for Andy.



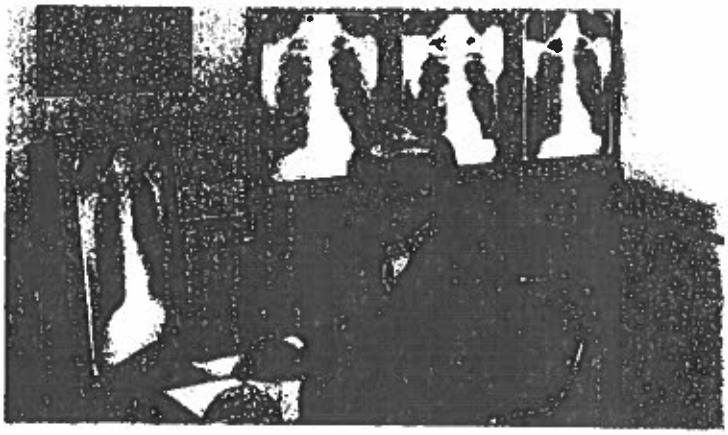
AIR SAMPLES are checked by Henry Getmer, assistant in Industrial Hygiene laboratory. Any harmful elements detected would mean immediate corrective attention.



A MOBILE X-RAY unit, operated by Herbert Johnson, x-ray technician, travels over 12,000 miles a year to x-ray the nearly 10,000 Brake Shoe employees as well as retired employees.

SAFEGUARDING YOUR HEALTH *(Cont.)*

X-RAYS from all locations from the mobile unit and preplacement examinations are checked and filed in the x-ray department by Richard Walters (*below*). If complications are found, the findings are reviewed with Dr. Hamlin and further medical studies are recommended. The Company doctor and plant nurses assist in arranging consultations with personal physicians, chest clinics and competent specialists. At right, Andy gets into position for his annual free x-ray.



PERIODIC PHYSICAL check-ups follow the preplacement examinations as part of the Company's continuous health program. Here Andy receives a check-up by Dr. Bonick while Nurse Louise Matthews takes down the data.

ASSURED of thorough medical check-ups available periodically, Andy, shown here with wife Mary Jean and son Andrew Jr., can work and live with confidence and peace of mind.

AMERICAN BRAKE SHOE COMPANY

AMERICAN BRAKE BLOK DIVISION

WINCHESTER, VIRGINIA

EVALUATION OF ENVIRONMENTAL HEALTH IN MANUFACTURE OF BRAKE LININGS

SUBMITTED BY

S. T. WAGNER
INDUSTRIAL HYGIENIST

AUGUST 7, 1959

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AMERICAN BRAKE SHOE COMPANY

AMERICAN BRAKE BLOK

WINCHESTER, VIRGINIA

EVALUATION OF ENVIRONMENTAL HEALTH

IN

MANUFACTURE OF BRAKE LININGS

On June 25 and 26, 1959, an environmental health study was conducted to evaluate the hazards associated with the manufacture of brake linings. An investigation was made of airborne lead, airborne asbestos, methyl ethyl ketone and noise exposures. The following general conclusions summarize the exposure in the order of importance.

GENERAL CONCLUSIONS

1. Airborne lead during dry compounding existed in sufficient quantities to be considered a health hazard. The concentration was far above the recognized safe limit of 0.2 milligrams per cubic meter.

2. A borderline health hazard from airborne asbestos exists in the dry compounding area. Improved capture velocities on the dust control equipment will reduce the exposure.

3. The exposure to methyl ethyl ketone is not significant except at the applicator machine in the PC Department.

4. The Ventilation Study indicates improvement is required in control velocities in such areas as Strip Finishing, Compounding and Brake Blok.

5. The Hearing Conservation Study indicates a moderate exposure exists in the Strip Finishing and Brake Blok areas.

The interest of management in the health of the workers is clearly demonstrated by the provision and maintenance of an excellent dust control program.

S. T. Wagner
Industrial Hygienist

STW/oq

GENERAL INFORMATION

Weather Conditions:

This latter part of June was extremely warm. The temperature during the first day was a little above 90°F. Later in the evening local showers raised the humidity to an uncomfortable level. On the second day the same warm conditions existed with very little natural draft. As a result, every available window and door was open to increase natural drafts. The weather at the time of the survey produced conditions to adequately evaluate the dust control equipment with the exception of make-up air.

Production Activity:

On the first shift, production was normal with all departments in operation. On the second shift, a make-up force was in operation.

Acknowledgements:

We would like to thank Mr. C. B. Mallory, Works Manager, for the courtesy and interest that was shown to us in this survey. In particular, we would like to thank Mr. W. H. Baker, Personnel Manager, for his assistance and co-operation in organizing the activities with the various department heads. The supervisory personnel also provided valuable assistance in co-ordinating the various operations required for an environmental study.

AIRBORNE LEAD

Air sampling was conducted in the Strip Finishing, Compounding, Rolling, Brakeblok and Book Press Departments. This phase of the study was conducted to evaluate the exposure of the workers to airborne lead during drilling, grinding, dusting, champering, cut-off, rolling, pre-forming and compounding. The results of this investigation are summarized as follows:

CONCLUSIONS

Compounding:

1. A serious health hazard exists during lead weighing on the dry line in compounding. The airborne lead concentration was 4.33 milligrams per cubic meter; this exceeded the threshold limit of 0.2 milligrams.

2. A borderline health hazard may exist at mixer No.1 when the batch material is discharged to the cart. An increase in the inward draft on the hood is indicated.

3. Airborne lead concentrations during dumping from blender to hammermill No.1 indicates a borderline health hazard may exist.

4. The exposure to airborne lead during lead handling on line No.2 and weighing is not considered a health hazard. Batch weighing and blender loading did not produce excessive airborne lead on the Dry line.

Rolling:

5. Minimal concentrations of airborne lead existed at the rolling machines except backing machine No.6. At the rolling position the airborne lead level was 0.16 milligrams per cubic meter.

Strip Finishing:

6. Airborne lead did not exceed 0.1 milligrams per cubic meter at the operations in Coil or Industrial Strip Finishing Department.

7. Close surveillance on the exhaust systems is required where the higher lead contamination existed (0.1 milligrams). This consists of Automatic Champer No.2, back grinder No.3 and the exhaust of the Torit Collector for the automatic strip grinder.

Brakeblok:

8. Airborne lead in the finishing and preforming operations did not exist in sufficient concentrations to be considered a health hazard.

General:

9. The exhaust of Torit Dust Collector in the ball joint area was connected to ducts that conveyed the lead particles out of the plant.

10. In the final analysis, an excellent dust control program is in operation except in specific areas where improvement is required.

CONTROLS FOR ATMOSPHERIC CONTAMINATION

1. An exhausted hood covering two-thirds of the periphery of the storage container will maintain airborne lead at a safe limit in dry compounding. A minimum inward draft of 100 feet per minute across the top of the container is required.

2. A bottom door or baffle on No.1 mixer discharge in dry compounding will increase the inward draft or a 3-sided enclosure may be considered.

3. Side baffles on the exhausted transfer from blender to hammer mill will increase the inward draft. Provision of the baffle would not interfere with material flow.

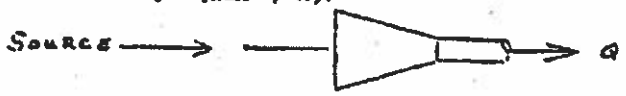
4. Hopper charging on No.6 backing machine in the Rolling Department requires a partial cover to reduce the airborne lead contamination. Moreover, consideration should be given to enclose the feed chute.

DISCUSSION

Provision of an engineered dust control system maintains airborne lead below the threshold limit except the dry line in the Compounding Department. Of course, there are areas where the threshold limit is approached and these are evaluated as borderline health hazards. However, on an overall basis, airborne lead is within reasonable limits and the health hazard is under control.

In the Compound Department dry line, lead for the batch is removed from the fiber drum container without the benefit of local exhaust. Moreover, the handling technique was poor and accounted for the high airborne lead contamination. Local exhaust at the storage drum is required to maintain the exposure at a minimum. This can be accomplished by providing an exhaust for at least two-thirds of the periphery of the fiber drum. Moreover, sufficient capacity is required to maintain an inward draft of at least 100 feet per minute at the source of contamination. The capacity required for the hood can be computed as follows:

$$Q = (10x^2 \not\propto A)V$$



Q = Required exhaust volume, cfm.

X = Distance from hood face to farthest point of contaminant release, feet.

A = Hood face area, square feet.

V = Capture velocity, fpm at distance x.

Application of this equation will provide the minimum capacity required to remove the airborne lead from the operator's breathing zone. However, the operator should be required to remove the material from the container without spillage or careless handling.

The blender, hammermill, mixer and discharging cycle in Dry Compounding did not produce exceedingly high airborne lead contamination. However, it is felt that improved inward draft can be achieved by addition of baffles. This is a semi-automatic transfer cycle and properly placed baffles should not interfere with the operating cycle.

In the Rolling Department, airborne lead approached the hygienic standard and a borderline health hazard may exist. It is felt that the primary source of airborne lead is associated with the hopper feed as raw material is introduced. A partial cover for the hopper and enclosure for the feed chute would reduce the airborne lead.

Airborne lead in Strip Finishing was not above the safe limit. However, in comparison, 0.1 milligrams may be considered relatively high with the exposure in other areas. Close surveillance on the Torit collector exhaust, automatic champer No.2 and back grinder No.3 is required because additional airborne lead may result in unsafe concentrations. Primarily, this is a problem of ventilation and will be covered in a separate section.

Hygienic Standard:

The threshold limit for airborne lead has been established as 0.2 milligrams per cubic meter.

Application of controls for atmospheric contamination will control the exposure associated with the use of lead.

DATA

Results of: Airborne Lead

Method of Collecting: Willson pump with filter paper cones and collected at a rate of 20 liters per minute.

Method of Analysis: Dithizone method and read on the Beckman Spectrophotometer.

Strip Finishing (Coil Side)

Sample No.	Time	Location	Results*	MAC*
1.	10:50AM 10 Min.	Breathing zone of operator hand drill No.1.	0.01	0.2
2.	11:01AM 10 Min.	Breathing zone of operator hand drill No.3.	0.02	0.2
3.	12:33PM 5 Min.	Breathing zone of operator multiple drill operator.	0.04	0.2
4.	12:50PM 5 Min.	Breathing zone of operator duster and printer No.2.	0.08	0.2
5.	12:27PM 5 Min.	Breathing zone of operator duster and printer No.1.	0.04	0.2
6.	1:02PM 5 Min.	Breathing zone of operator champer machine No.1.	0.07	0.2
7.	1:10PM 5 Min.	Breathing zone of operator automatic champer No.2.	0.10	0.2
8.	1:19PM 5 Min.	Breathing zone of operator strip grinder No.2.	0.08	0.2
9.	1:25PM 5 Min.	Breathing zone operator back grinder No.3.	0.10	0.2
10.	1:51PM 5 Min.	At exhaust of Torit Dust Collector automatic strip grinder.	0.10	0.2
11.	1:57PM 5 Min.	Breathing zone operator at automatic strip grinder.	0.09	0.2
12.	2:05PM 5 Min.	Breathing zone of operator at No.2 coil grinding.	0.04	0.2

Sample No.	Time	Location	Results*	MAC*
13.	2:11PM 5 Min.	Breathing zone of operator coil cut-off saw.	0.06	0.2
Strip Finishing (Industrial Side)				
14.	11:12AM 10 Min.	Breathing zone operator every cutting.	Trace	0.2
15.	11:23AM 10 Min.	Breathing zone strip grinder.	0.04	0.2
16.	12:27PM 5 Min.	Breathing zone internal grinding.	0.03	0.2
Compounding Room:				
17.	2:55PM 4 Min.	Breathing zone drawing material from storage line No.2.	0.09	0.2
18.	2:59PM 5 Min.	Breathing zone loading mixer line No.2.	0.09	0.2
19.	4:25PM 6 Min.	Breathing zone weighing material on dry line.	4.33	0.2
20.	4:32PM 5 Min.	General room dumping material from blender to hammer mill No.1 dry line.	0.16	0.2
21.	4:45PM 5 Min.	General room at transfer from hammer mill to mixer dry line.	0.09	0.2
22.	5:02PM 5 Min.	Breathing zone at mixer No.1 transfer to cart.	0.18	0.2
Rolling:				
23.	7:17PM 5 Min.	Breathing zone of operator SKA machine No.2.	0.02	0.2
24.	7:23PM 5 Min.	Breathing zone of operator SKA machine No.4.	0.06	0.2
25.	7:53PM 5 Min.	Breathing zone at rolling position backing machine No.6.	0.16	0.2

Brakeblok (Preforming):

Sample No.	Time	Location	Results*	MAC*
26.	8:04PM 5 Min.	Breathing zone at preform machine No.4.	0.09	0.2
27.	8:11PM 5 Min.	Breathing zone at preform machine No.3.	0.03	0.2
Brakeblok (Finishing):				
28.	9:45AM 5 Min.	Breathing zone at trimming machine No.BR-7.	0.03	0.2
29.	9:50AM 5 Min.	Breathing zone at automatic burr and champer machine.	0.06	0.2
30.	9:56AM 5 Min.	General room at radius grinder No.3.	0.09	0.2
31.	10:05AM 5 Min.	Breathing zone at automatic drilling machine No.3.	0.05	0.2
32.	10:11AM 5 Min.	Breathing zone at strip grinder No.3.	0.04	0.2
Book Press:				
33.	8:22PM 5 Min.	Breathing zone at No.7 preforming press.	0.03	0.2
34.	8:28PM 5 Min.	Breathing zone at No.3 preforming press.	0.04	0.2
35.	8:35PM 5 Min.	Breathing zone at champer preforming.	0.05	0.2

*Results and Maximum Allowable Concentrations (MAC) are expressed in milligrams per cubic meter.

AIRBORNE ASBESTOS

An investigation was conducted in the Compounding Department to evaluate the exposure to airborne asbestos. Air sampling was conducted in the Wet and Dry Compounding lines. The results are summarized as follows:

CONCLUSIONS

1. A borderline health hazard exists at the weigh and dumping stations in the wet line. Airborne abestos was slightly above and below the recognized safe limit of 5 million particles per cubic foot of air.

2. During weighing and blender charging on the Dry Line, airborne asbestos exceeded the recognized safe limit of 5 million particles.

3. The primary cause of airborne dust can be attributed to lack of sufficient inward drafts at the source of contamination. The velocities ranged from 0 to 100 feet per minute.

CONTROLS FOR AIRBORNE ASBESTOS

1. An inward draft of 100 feet per minute from the furthest point from the lateral exhaust at the weigh station in wet compounding will maintain the airborne dust at a minimum. The inward draft at the source of contamination was zero.

2. The inward draft during dumping into the digestor has insufficient capture velocity to remove the dust generated by the surge of material. A minimum inward draft of 200 feet per minute is required at the wet line.

3. A minimum inward draft of 100 feet per minute at the source of contamination is required at the weigh scale in dry compound. Baffles with increased capacity may provide the control velocity.

4. The lateral exhaust hoods on the blender are not evenly balanced. Balancing of the system is required to maintain a control velocity of 100 feet per minute at the source of contamination.

DISCUSSION

Adequate dust control equipment is provided but the inward draft does not have sufficient capture velocities. The velocities ranged from zero to 100 feet per minute on the various lateral exhaust installations. This is primarily caused by insufficient capacity and an unbalanced exhaust system. Since the design incorporates lateral exhaust, the control velocities should comply with the American Standards Association Code for Ventilation and Operation of Open Surface Tanks. This design criteria takes into consideration the tank width and length so that adequate control velocity is maintained at the center of the tank or the source of airborne dust.

In the wet compounding area the velocity at the weigh station was zero. The lateral hood had insufficient capacity to provide a control velocity of 100 feet per minute at the furthest point from the hood. This is also reflected in the results of air sampling as shown by Sample No.1. Dumping into the digester produced airborne dust that exceeded the threshold limit. A velocity of 25 feet per minute is maintained but it is ineffective when the surge from the hopper displaces the air from the digester. A minimum inward draft of 100 feet per minute is required.

In Dry Compounding, a similar condition exists at the weigh scale. A lateral exhaust is provided but a inward draft of only 50 feet per minute exists. Much of the dust generation is outside of the capture effect of the control velocity. The blender is also provided with lateral exhaust but the system is unbalanced. The inward draft on the left side is zero (this is opposite the hood) and increase to 100 feet per minute near the hood. Apparently, the system was designed to operate with a cover so that the capture velocity was reduced when the area was increased. A minimum inward draft of 100 feet per minute is required from the furthest point of the hood to remove airborne asbestos from the breathing zone of the worker.

Hygienic Standard:

The safe limit for airborne asbestos has been established as 5 million particles per cubic foot of air.

An evaluation of the capacities of the asbestos exhaust equipment will provide adequate inward draft to maintain the airborne dust at a minimum.

DATA

Results of: Airborne Asbestos

Method of Collecting: Willson pump with midget impingers and collected at a rate of 5 liters per minute in distilled water.

Method of Analysis: The samples were counted microscopically by the light field technique on a Spencer Bright-Line Haemacytometer Counting Cell.

Sample No.	Time	Location	Results*	MAC*
1.	3:08PM 10 Min.	Breathing zone operator dumping material at weigh station - Wet Compounding.	4.8	5.0
2.	3:08PM 10 Min.	Breathing zone while dumping material into digestor - Wet Compounding.	6.0	5.0
3.	3:42PM 10 Min.	General room at digestor No.1 in Wet Compounding.	13.1	5.0
4.	3:42PM 10 Min.	General room at digestor No.2 in Wet Compounding.	3.7	5.0
5.	3:58PM 5 Min.	Breathing zone opening and charging weigh scale in Dry Compounding.	8.5	5.0
6.	3:58PM 5 Min.	Breathing zone charging blender in Dry Compounding.	6.8	5.0

*Results and Maximim Allowable Concentration (MAC) are expressed in million particles per cubic foot of air.

Should the Worker with Silicosis Be Informed of His X-Ray Findings

L. E. HAMLIN, M.D., F.A.C.S.,
Medical Director,
American Brake Shoe Company

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Should the Worker with Silicosis Be Informed of His X-Ray Findings

L. E. HAMLIN, M.D., F.A.C.S.,
Medical Director,
American Brake Shoe Company

THE time-honored proverb "Honesty is the best policy" is as true today as it has always been, but unfortunately in some instances its practical application still leaves much to be desired. Progress brought about by modern industrial medicine and hygiene and the growing cooperation between management, labor, and public health departments in this field augur well for better mutual understanding in the future.

In past years, management, not without reason, has been reluctant to face realities concerning occupational disease. Vicious litigation and unfair administration of existing laws in some localities produced an understandable hysteria with regard to these conditions out of all proportion to the actual facts. Inadequate knowledge of fibrosis and the development of the ubiquitous silicosis rackets a few years ago added much to the concern of both workers and employers who responded each in his own tongue and to the discomfiture of all. The employer proceeded to divest himself of those workers who were potential liabilities. To screen out anticipated claims of disability he entered the field of mass radiography and sought to escape responsibility by the wholesale discharge of men, many of whom were old and loyal employees. The worker, abetted by unscrupulous legal advice, demanded his full pound of flesh for disability, actual or not, caused by silicosis.

The effects of this regrettable situation are still apparent today. Workers who have not forgotten that they lost their jobs through unreasonable attitudes of management are skeptical of plant health and x-ray surveys which executives now desire to institute. Recalling former unpleasant experiences, some employers are still apprehensive about their men being informed of results of their roentgenograms, because of possible claims for disability.

In the light of experience neither assumption seems warranted. Generally speaking, the employer has nothing to lose and everything to gain by having a competent physician discuss x-ray findings in a frank and honest manner with the party concerned. The word *competent* is emphasized because the success of the whole effort will be determined by the diplomacy of the doctor and the extent of his understanding of industrial ailments. He must know his subject and be able to explain findings to employees in an intelligible manner. Today too many physicians have not familiarized themselves with the more recent aspects of occupational disease. They have failed to recognize their responsibility to workers and management, and in many cases have caused unnecessary hardship to each by unconsidered judgment and snap diagnosis based largely on a history of exposure and incorrect interpretation of roentgenograms.

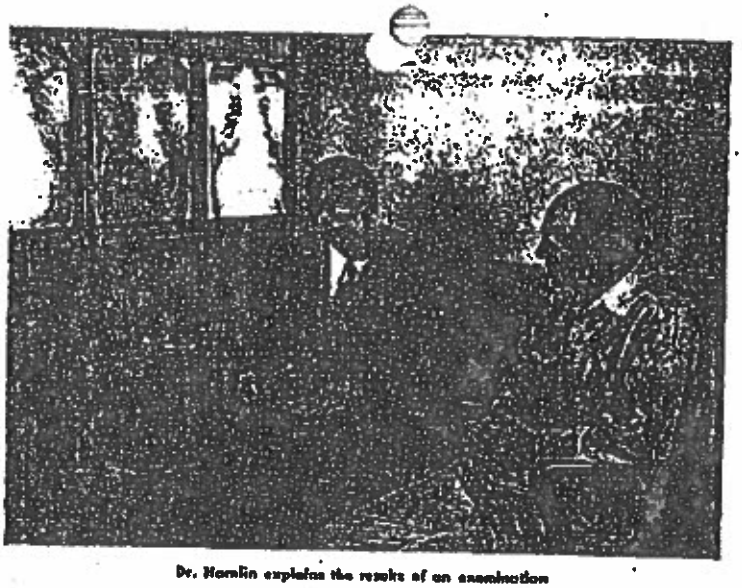
Some doctors, even those engaged entirely in industrial practice, have not taken the trouble to make trips through the plants they service in order to obtain first hand knowledge of actual working conditions and the physical requirements essential to efficient performance of the diverse operations. They do not hesitate, however, to advise the employee against continuing work in his present position because of an alleged exposure. They are unaware that a hygiene-conscious management may have made every effort to eliminate occupational risks through plant surveys and improved ventilation. One recognizes that the physician usually offers such advice with the best intentions in the world but it may also be a temptation for him to follow the line of least resistance and tell his patient something he would perhaps expect to hear and which would possibly offer a reasonable though not necessarily true explanation of his symptoms.

Frequently the doctor does not stop to consider that the employee may have spent his whole life at the job in question. Aside from the fact that he has become very proficient at it, the man knows no other occupation. Transfer to other work not only serves to discourage him but also very often disturbs his earning capacity. The plant superintendent loses the services of a valuable worker, and production suffers at a time when it is urgently needed. Such action upsets the mental outlook of the employee, stirs up unjust claims for compensation, and may transform a satisfied workman into a chronic invalid.

Consider the case of a grinder in a certain plant. His 33 years of experience in this location are an indication of loyalty and satisfaction with his job. A routine x-ray of his chest suggested a very fine nodulation which was interpreted as a mild, non-disabling siderosis or iron pigmentation of the lungs. Air samples in the area in which he worked showed dust concentrations to be well within recognized safe limits. Serial x-rays from the year 1938 indicated no change in the condition observed in his chest. The employee was interviewed personally and advised of the findings. He was entirely satisfied and unconcerned. Some weeks later he contracted a cold and consulted his personal physician, a very able practitioner. The doctor viewed the man's recent chest x-ray and informed him that he had a second degree silicosis. He advised him, in good faith, to discontinue working. The employee returned to the plant very much perturbed. The plant superintendent could ill-afford to lose an experienced grinder and feared that some of his other men might refuse to work in the plant after hearing of the case. Owing to the shortage of capable men and the demand for increased production, the situation could easily become serious. Further discussion failed to convince the grinder that his symptoms were not due to dust in his lungs. He was later allowed to return to his work by his doctor but was still dissatisfied and worried. Finally, he was tendered a physical examination by well recognized authorities at a university hospital. He agreed to abide by their conclusions. The company assumed the expense. After very thorough clinical tests and further x-rays, the original diagnosis of mild non-disabling siderosis was substantiated and the clinical symptoms ascribed to

early cardiovascular disease was fully explained to the emplo, at a later interview. He seemed to be reassured but did not wish to go back to grinding. In view of his loyal service and cooperative attitude, other employment was found for him but at a reduced rate of pay. Inasmuch as he was a single man and had saved his earnings, he was content to take the work offered.

The upshot of the whole episode was that the services of a valuable man were lost to a plant engaged in national defense at a very critical time, production was interfered with, an experienced grinder suffered financial loss and acquired additional unnecessary worry, the cost to the company in time, money and inconvenience was substantial --all this because of well-meaning but unconsidered medical care.



Dr. Hamlin explains the results of an examination

EXPERIENCE in discussing x-ray findings and occupational fibrosis in some 196 cases in the iron mining district and foundry industry during the past 12 years has convinced me that such a policy can be carried out with mutual satisfaction to management and its employees. In one mining district a recognized hazard caused a large percentage of workers to be affected with silicosis in all its various stages. In addition, the incidence of tuberculosis in the community was very high. The mining company, while aware of its responsibility and keenly desirous of eliminating the danger, was apprehensive about informing men of results of chest x-ray surveys. Their fear of litigation was justifiable to a large extent but not entirely so. For several years periodic examinations were made but the employees were not told of the outcome. As a consequence they became disinterested and uncooperative, and many refused to submit to further tests except under pressure. At the conclusion of the next survey each employee was contacted and the results of the examination were explained in language he could understand. The effect was dramatic. Employees expressed their appreciation and showed greater interest in protecting themselves. Many who had been afraid that their lungs were already affected exhibited evident relief on being told that the films were negative. Others whose roentgenograms showed evidence of disease became more cooperative and less apprehensive. A very decided lift in morale was apparent and not a single case of litigation resulted. Management was definitely relieved of a moral responsibility and the men realized that the company for which they worked was sincere in its effort to care for their health. In several instances those whose attitudes the company feared most proved to be exceptionally cooperative.

Employees working in cloudy atmospheres are not unmindful of the potential risks they face, but many, through long familiarity with such environments, show no undue anxiety when told that there is evidence of dust accumulation in their lungs. It is recognized as something to be expected. They worry, however, about the presence of infection, and manifest relief on being told that they do not have tuberculosis. Once a man has had his findings so explained, he is very willing to submit to subsequent x-ray and physical examinations. He recognizes their value and appreciates the fact that management has made them available.

If an employee is not informed of the results of examinations he frequently suspects something is wrong and seeks advice from his personal physician. An x-ray is usually made and he is told that he has silicosis. He immediately becomes resentful and returns to his place of employment to find out why this information has been kept from him, and wants to know what the company proposes to do about it. Management has no logical answer and is faced with an embarrassing situation which may involve legal proceedings and the loss of a good workman.

On the other hand, if the employee has already been made aware of the findings disclosed by the plant health survey he is usually satisfied and has no legitimate com-

THE AMERICAN BRAKE SHOE COMPANY
Medical Department
2501 Blue Island Avenue
Chicago 8, Illinois

Study No._____ Date_____

Mr._____

Address_____

B X-Ray Examination / Physical Examination extended to you by the

_____ Division of the American Brake Shoe Company

on (Date)_____ indicates the following_____

It is recommended that you

- ☐ See your Dentist
- ☐ Consult your family physician for advice and treatment.
- ☐ Have your eyes examined and fitted with glasses by a reputable eye doctor.
- ☐ Report for a re-check of your chest in_____ months.

Signed_____ Medical Director

(Note: Pay attention only to items checked.)

Confidential notice to employee indicating results of examination. These forms are printed in duplicating sets using different colors. The original is light green and the duplicate which is filed with the employee's medical record is yellow. The contrast in color makes for more rapid recognition of the various forms in the employee's folder

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plaint against the management as far as being informed is concerned.

THE American Brake Shoe Company firmly believes in reporting results of physical examinations and x-rays to its employees, and during the past four years the medical department has evolved the following procedure. After completion of a plant survey, those participating are informed of findings by means of printed slips sent out from the medical department. These notices are confidential and are distributed in sealed envelopes. The recipients are advised to seek medical advice for correction of defects. A duplicate is kept in the individual's record in the medical department. Significant occupational fibrosis is not recorded on the form, as the medical director will arrange for a personal interview with the affected worker to interpret the results to him. This arrangement has a definite beneficial psychological effect. Most people are greatly interested in seeing x-rays, particularly their own, but many have never had the opportunity of doing so. The doctor can easily explain the various shadows in the roentgenogram so that even the unintelligent are able to grasp the idea. If necessary, a normal chest film may be demonstrated for comparison. One has only to interview a few workers and display x-rays to sense the keen interest such a procedure evokes. Recently one of our old employees ventured the information that he was very glad to see his film because he had just been on the point of going to the hospital to have an x-ray taken, on his own volition, "just to see what it looked like."

In discussing silicosis with an employee, the man is reminded that he has worked in a dusty occupation for a considerable number of years and it is not surprising that his x-ray shows some evidence of reaction. He usually agrees to this and readily admits that it is to be expected. The effects of different dusts on the lungs are explained, and he is assured that his particular condition is no immediate cause for worry. His serial films are shown to demonstrate that the condition has not changed over the years, and he is told that there is no reason why he cannot continue his regular work. Steps taken to control the dust and make his working place safe are outlined to him, and he is asked to submit to subsequent x-rays as a matter of routine check-up. He is also informed that if his condition shows any progression he will be immediately consulted about it. The word "silicosis" is not used unless the man brings it into the conversation himself. Some men become unduly apprehensive at the mention of the word, whereas if told that their x-rays show evidence of accumulation of dust they are not nearly as concerned. This is in no way an attempt to cover up the true facts, but is simply a matter of good psychology. Most workmen are familiar with the term but do not have any real knowledge of the various stages of the condition or the degree of disability it may produce. If told they have "silicosis" their first reaction is one of shock, and subsequent attempts to correct the impression will be difficult. The same response might be expected from an apparently healthy person who is told simply that he has tuberculosis even though the shadow observed in the x-ray represents a healed fibrotic lesion of no clinical significance.

When the x-ray discloses complicated silicosis the disposition of the case is more of a problem. An effort is made to evaluate the degree of disability as accurately as possible. All the factors concerned, such as the man's age, length of service, symptoms, state of present health, ability to perform the job, mental reaction, and roentgenographic findings are given due consideration. The exposure to dangerous-sized particles of hazardous dust is re-checked by representative air samples taken at the working place. The actual conditions, general and local, are studied to determine if continued employment will offer further risk to the workman in the event that his physical condition will permit him to remain on the job, under observation. If his infection appears to be indeterminate or suggestive of activity, he is referred to a recognized chest clinic or sanatorium for further examination and study. In the event that the disease is found to be quiescent the employee is allowed to continue working but under strict observation and safe exposure. He is re-x-rayed at intervals of from three to six months and notified of the results. When there is evidence of progression of his infection, either by x-rays or clinical examination, he is advised to seek sanatorium care. If it is decided that he has a legitimate claim for compensation, the case is placed before the industrial commission for settlement. Financial aid is extended through plant welfare funds and company donations. Effort is made to secure the best medical care and effect rehabilitation.

Experience has proved the value of this policy. In the great majority of cases it has resulted in better understanding and respect for the company. In any industry some individuals will always be unreasonable, but they are in the minority. In such instances the men are informed that it is their prerogative to take their cases before the industrial commission if they are not satisfied. At the same time they are given to understand that their claims will be strongly contested if they do not have definite disability. The insurance carrier is not allowed to settle cases for their nuisance value and no attempt is made to evade responsibility for just demands.

The time has come when the human factor in industry must be fully recognized. Many organizations have long since realized this and in spite of numerous obstacles have worked out admirable policies and commendable industrial relationships with their employees. Some have gone to the extreme and adopted paternal attitudes that are neither appreciated nor desirable. Others, with traditional aloofness, still ignore the handwriting on the wall and dismiss workers' problems as something for the insurance carrier to handle. Somewhere along the middle road lies a path of mutual cooperation between labor and management, and one of the direct avenues of approach can well be via the plant health survey, sincerely conducted and honestly administered.

C06714

A101

THE TRUDEAU FOUNDATION
FOR
THE CLINICAL AND EXPERIMENTAL STUDY OF PULMONARY DISEASE

AT SARANAC LAKE, N.Y.
THE SARANAC LABORATORY
THE TRUDEAU FOUNDATION
THE TRUDEAU SCHOOL

AT TRUDEAU, N.Y.
THE DEPARTMENT OF PHYSIOLOGY
THE DEPARTMENT OF BIOCHEMISTRY
THE DEPARTMENT OF RADIOLOGY
THE TRUDEAU LABORATORY

November 16, 1948

Mr. U. E. Boves
Owens-Illinois Glass Company
Toledo 1, Ohio

PLAINTIFF'S EXHIBIT 263

Dear Mr. Boves:

Enclosed you will find three copies of a report on the results of animal experiments with Kaylo dust. As is our custom, we have summarized briefly material previously presented, including our interim report dated October 30, 1947, and have given detailed discussion only of subsequent developments. When all experiments have been completed, we expect to prepare a final report which will include details of each phase so that all data will be available in one place. However, the experimental study of the effects of inhaled Kaylo dust on normal uninfected animals is now finished and conclusions expressed on that subject are final rather than tentative.

In the report issued one year ago, which describes the findings in animals that inhaled Kaylo dust for periods up to 30 months, the following tentative conclusion was made:

> "In consequence of the experimental studies with guinea pigs to determine the biological activity of Kaylo, it may be tentatively concluded that Kaylo alone fails to produce significant pulmonary damage when inhaled into the lung."

During the 30 to 36 months period, however, definite indication of tissue reaction appeared in the lungs of animals inhaling Kaylo dust and therefore, I regret to say, our tentative conclusion quoted above must be altered. In all animals sacrificed after more than 30 months of exposure to Kaylo dust unmistakable evidence of asbestosis has developed, showing that Kaylo on inhalation is capable of producing asbestosis and must be regarded as a potentially-hazardous material. It should be noted that since neither silicosis nor the diffuse pulmonary fibrosis caused by inhaled [illegible] apparently do not produce their typical lesions.

In order to present more information on the subject asbestosis, certain evidence derived from our experimental work with asbestos dust has been discussed. As these findings have not yet been released for publication, I request that, while using them as required in formulating a safety program, you regard them as confidential.

THE TRUDEAU FOUNDATION
FOR
THE CLINICAL AND EXPERIMENTAL STUDY OF PULMONARY DISEASE

AT SARANAC LAKE, N.Y.
THE SARANAC LABORATORY
THE TRUDEAU FOUNDATION
THE TRUDEAU SCHOOL

AT TRUDEAU, N.Y.
THE DEPARTMENT OF PHYSIOLOGY
THE DEPARTMENT OF BIOCHEMISTRY
THE DEPARTMENT OF RADIOLOGY
THE TRUDEAU LABORATORY

The new series of experiments with respect to the influence of Kaylo on tuberculous infection are well under way and are progressing satisfactorily. It is, of course, too early to expect significant results on which to base even tentative conclusions.

At this time may I review briefly the financial arrangement for conducting the investigation with Kaylo? The research program up to this year was carried on under a contract, initiated in 1945, by which the experiments would be subsidized with a grant of $5,000. per year. As pointed out in my letter of March 3, 1948, the contract terminated officially on February 15, 1948, but the investigation would be continued without charge until June because the original experiments were started late. Since a check was received which took care of the subsidy up to November 15, 1947, there is due on the old concract the sum of $1,250. for the final quarter of the contract (from November 15, 1947 to February 15, 1948). We have delayed sending an invoice for the final quarter until all work could be finished and a final report submitted. Following the termination of the experiment in June, it has required several months to do the histological work, study the tissue sections, collate the data and prepare the report which accompanies this letter.

We are including in the report a brief review of the new experiments in which the effect of inhaled Kaylo dust on tuberculous infection is being studied. Your purchase order AS-170 authorizing this experiment at $5,000. for one year is dated February 3, 1948, but owing to a shortage of animals and other unavoidable delays the actual experimental work did not get under way until May. Hence we have concluded that financial support for this new program should be dated from May 1, 1948. For reasons outlined in my letter of March 3, I suggested a two-year contract at $7,000. per year. Your letter of March 31 acknowledged this but failed to confirm the extra amount of $2,000. involved. Nevertheless, we have proceeded at the old rate and are endeavoring to absorb the increased cost from our Foundation reserve.

In a few days our accounting department will forward a statement of the payments now due in support of the research program, as follows:

Original experiment, begun February 15, 1945, at rate of $5,000. annually
For final quarter (November 15, 1947 to February 15, 1948) $1,250.

New experiment, begun May 1, 1948, at rate of $5,000. annually
For first quarter (May 1, 1948 to August 1, 1948) $1,250.
For second quarter (August 1, 1948 to November 1, 1948) 1,250.
$2,500.

CC7279

A103

THE TRUDEAU FOUNDATION
FOR
THE CLINICAL AND EXPERIMENTAL STUDY OF PULMONARY DISEASE

AT SARANAC LAKE, N.Y.
THE SARANAC LABORATORY
THE TRUDEAU FOUNDATION
THE TRUDEAU SCHOOL

AT TRUDEAU, N.Y.
THE DEPARTMENT OF PHYSIOLOGY
THE DEPARTMENT OF BIOCHEMISTRY
THE DEPARTMENT OF RADIOLOGY
THE TRUDEAU LABORATORY

Page 3.

Mr. U. E. Bowes
Owens-Illinois Glass Company

November 16, 1948

I realize that our findings regarding Keylo are less favorable than anticipated. However, since Keylo is capable of producing asbestosis, it is better to discover it now in animals rather than later in industrial workers. Thus the company, being forewarned, will be in a better position to institute adequate control measures for safeguarding exposed employees and protecting its own interests.

Sincerely yours,

Arthur J. Vorwald, M.D.
Director

AJV:LB
Encs. (3)

C07280

A104

The third point concerns the experimental investigations with Kaylo which are about completed. Only a few more animals remain. They will be sacrificed next month. Then we will be ready to prepare the final report. For your information, at this time, I believe the findings permit the following:

Kaylo dust on inhalation by experimental animals does not produce silicosis irrespective of the small amount of quartz present. It does produce the asbestotic type of reaction in the lungs and, therefore, we believe every precaution should be taken to minimize exposure of industrial employees.

Kaylo dust on inhalation by experimental animals infected with tubercle bacilli (H) produces only a very mild stimulation of the tuberculous infection, much less than that caused by the inhalation of pure quartz. This evidence leads us to believe that in industrial practice the minimal allowable concentration of Kaylo could be far in excess than that accepted for quartz before Kaylo would have an adverse stimulating effect upon a tuberculous infection in exposed employees.

This evidence would support the view that inhalation of Kaylo dust would not be hazardous from the standpoint of tuberculosis, especially provided that the dust hazard is controlled as cited above.

We are looking forward to having you and Doctor Shook with us the latter part of June.

Sincerely yours

Arthur J. Vorwald, M.D.
Director

AJV:bw

cc: Dr. Shook
Kaylo exp. file

C07281

A105

September 8, 1961

Mr. W. O. Hazard,
Owens-Illinois Glass Company,
Toledo, Ohio.

Dear Mr. Hazard:

Mr. Ames has asked me to return the following pamphlets:

"Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers"

and

"A Study of Dust Control Methods in an Asbestos Fabricating Plant"

We thank you for your courtesy in lending us these pamphlets.

Very truly yours,

OWENS-CORNING FIBERGLAS CORPORATION

Legal and Patent Department

C E Stuelin
emf

THE TRUDEAU FOUNDATION
FOR
THE CLINICAL AND EXPERIMENTAL STUDY OF PULMONARY DISEASE

AT SARANAC LAKE, N.Y.
THE SARANAC LABORATORY
THE TRUDEAU FOUNDATION
THE TRUDEAU SCHOOL

AT TRUDEAU, N.Y.
THE DEPARTMENT OF PHYSIOLOGY
THE DEPARTMENT OF BIOCHEMISTRY
THE DEPARTMENT OF RADIOLOGY
THE TRUDEAU LABORATORY

February 7, 1952

PLAINTIFF'S EXHIBIT 278

Mr. W. G. Hazard.
Industrial Relations Division
Owens-Illinois Glass Company
Toledo 1, Ohio

Dear Bill:

Herewith is the final report of our studies concerning The Capacity of Inhaled Kaylo Dust to Injure the Lung. We are enclosing four copies for distribution by you. One copy has been sent to Doctor Shook for his information and we are retaining a copy for our files.

The results of the investigations with animals show that Kaylo dust is capable of producing a peribronchiolar fibrosis typical of asbestosis. The dust also has a slightly unfavorable influence upon a tuberculous infection. Although extrapolation from animal to human experience is difficult, nevertheless the results of the study indicate that every precaution should be taken to protect workers against inhaling the dust. Therefore, control measures should be directed to reducing the amount of atmospheric dust, especially at those points of operation where dust is generated. Our report of May 29, 1951 concerning the industrial hygiene survey may be of help to you in this regard.

We hope to publish this study either separately or in combination with similar studies pertaining to other dust. In doing so, however, reference will be made only to hydrous calcium silicate and not to "Kaylo;" thus the interest of your Company will be safeguarded. Of course the final manuscript will be forwarded to you for review before being released to the publisher. Your comment in this regard would assist us greatly in preparing the manuscript.

In submitting this final report, may I express to you and others of the Company our sincere appreciation for having had the privilege of collaborating with the Owens-Illinois Glass Company in the study of Kaylo. The collaboration has always been most pleasant and stimulating.

My every best wish.

Sincerely yours,

Arthur J. Vorwald, M.D.
Director

AJV:LB
Encs. (4)
CC: C. F. Shook, M.D.

Greetings + Salutations
01 501 1347 ant

C07288

A107

An IC Industries Company

Abex Corporation
Friction Products Division
P.O. Box 3250
Winchester, VA 22601-2450
(703) 662-3871

September 2, 1987

To: Charles Borcherding

From: Vince Weiss

Subject: Inspection by Va. Division of Occupational Health

Per our conversation this morning, enclosed is an outline of the closing conference with VDOH hygenist, M. Lindsay, including a copy of the actual citations.

Vince

copy: R. Armer
A. Indelicato

JWK006848

A108

FRICTION PRODUCTS DIVISION
Winchester

August 21, 1987

To: A. D. Indelicato

From: R. L. Armer

Subject: Meeting with Virginia OSHA on Asbestos Fiber Results

On 8/20/87 a meeting was held by Mr. Mike Lindsay, Virginia OSHA representative, and Messrs. F. Hernandez, B. Tizio, M. Funk, R. Armer and Messrs. D. Cooper (UAW), M. Martin (UAW), R. Roddehaven (UAW) to review the proposed citation against Abex Corporation for exceeding the .2 fiber per cubic centimeter PEL (permissible exposure limit) standard for airborne asbestos fiber. The findings and subsequent citation resulted from sampling and testing conducted last Spring.

Results and Required Actions

1. One Block preform press was determined to have a fiber level of .24 f/cc. This is considered a "serious violation" and a fine of $490.00 will be imposed. Also, considerable abatement and corrective actions have been triggered due to this violation. Specific time periods have also been agreed to for compliance to required corrective action. Because of our cooperativeness with Virignia OSHA, our obvious efforts to maintain a safe work environment, and our strong corporate policy to withdraw from the use of asbestos by December 31, 1987, a very favorable ruling on the size of citation penalty was made.

2. Numerous "non-serious" violations were noted related to the now established position that Abex violates the fiber standard as follows:

   a. Abex did not monitor fiber levels in the new pelletizer area.

   b. Abex did not have a formal written program for compliance to fiber standards and must institute program within thirty (30) days.

JWK006849

A109

c. Protective clothing is required at the B-16 Block press.

d. A regulated work area must be established with adequate warning signs.

e. All employees working in regulated area must wear respirator, protective clothing, shower daily and refrain from any eating, drinking or smoking in area.

f. All asbestos mix containers require warning signs.

g. Official training program needs modifications to clearly define asbestos hazard and safe work methods.

h. Hazardous Communication Program required modification to explain correct "clean up" procedures for asbestos fiber and dust.

i. Medical Surveillance Program requires modification to utilize OSHA recommended questionnaire and to provide physician with data on employees working in regulated area. Record keeping program requires some modification per OSHA guidelines.

j. Deficiencies in MSDS program need correction.

Compliance

1. If it can be demonstrated that Engineering actions have reduced the asbestos fiber level below the PEL and verified by Virginia OSHA, then the citation will be considered in full compliance and rules governing the "regulated work area" will no longer apply.

2. Engineered solutions would be required by law for compliance after July 1988 in any case.

JWK006850

A110

Actions

1. F. Hernandez/B. Tizio/M. Funk/V. Weiss have been assigned various tasks and activities to fully meet the various compliance dates agreed to as part of the citation. A separate schedule of action items will be published by 8/27.

R. L. Armer

RLA:rf

cc: E. H. Feierabend
D. P. Johnson

JWK006851

A111

COMMONWEALTH of VIRGINIA

Department of Labor and Industry
Division of Occupational Health
934 N. Augusta Street
P. O. Drawer 2708
Staunton, VA 24401
(703) 332-9240
August 24, 1987

Mr. Vince Weiss, Plant Manager
Abex Corp.-Friction Products Div.
P. O. Box 3250
Winchester, Virginia 22601

Dear Mr. Weiss:

Enclosed you will find citations for violations of the Virginia Occupational Safety and Health Act which, in some instances, may have accompanying proposed penalties. As explained in the closing conference, you may request an informal conference with me during the 15-working-day notice of contest period. During the informal conference you may present any evidence or views which you believe would support an adjustment to the citation or penalty.

If you decide to request an informal conference, please complete the enclosed form and post it next to the citations immediately after determining the time, date and place of the informal conference.

If you have any questions about the enclosed citations and penalties, I would welcome further discussion at the informal conference, and, where warranted, I am authorized to enter into an informal settlement agreement which amicably resolves this matter without litigation or contest.

Should an informal settlement agreement not be reached, then payment of penalties or notice of contest should be sent to:

Commissioner
Department of Labor & Industry
205 North Fourth Street
P. O. Box 12064
Richmond, VA 23241-0064

All citations require a written notice of abatement on or before the latest abatement date on the citation. Progress reports are required for abatement exceeding sixty (60) days as specified in the citation.

JWK006852

A112

If you have any questions regarding this matter, please contact me at the address in this letterhead.

Sincerely,

Charles L. Clouse

Charles L. Clouse, Regional Supervisor
Division of Occupational Health
Northwest Region

CLC/dmb

Virginia Department of Labor and Industry

Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

3. Issuance Date	4. Inspection Number
08/24/87	115221674
5. Reporting ID	6. CSHO ID
355122	L3863
7. Optional Report No.	8. Page No.
006 87	1 of 2

1. Type of Violation(s)	2. Citation Number
Serious	1

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Sectic May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
1a 1910.1001(c)	Employer did not ensure that employees working with asbestos were not exposed to an airborne concentration in excess of .2 fibers/cc of air as an 8 hour time weighted average (TWA) as determined by the method prescribed in Appendix A of this section or an equivalent method: Location - Block Prefora Area. Operation - Block preforing. Condition - Employee working at block prefora machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Effective reduction of exposure levels to below .2 fiber/cc through modifications to existing ventilation system or through documented work practice changes.	September 15, 1987	$490.00
1b 1910.1001(e)(1)	Employer did not establish a regulated area wherever airborne concentrations of asbestos, trecolite, anthophyllite, actinolite, or a combination of these minerals were in excess of the permissible exposure limit prescribed in paragraph (C) of this section: Location - Block Prefora Area. Operation - Block prefora.	September 15, 1987	-0-

17. Enforcement Director: Operations Division Regional Supervisor

18. Page

Total Penalty for This Citatic

Make Check o Order Payt Virginia Dept Labor and In

Indica Inspec Numb on Remitt

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51. 2:1. Code of Virginia.

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006854

A114

Virginia Department of Labor and Industry
Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	152216?4
5. Reporting ID	6. CSHO ID
355177	?3843
7. Optional Report No.	8. Page No.
806 87	2 of 2

1. Type of Violation(s)	2. Citation Number
Serious	1

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp. Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet) This Sect May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
1	Condition - Employee working at block preform machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Establish a regulated area which meets minimum requirements as set forth in 1910.1001(e)(1)-(5).		

AUG 1987 RECEIVED

17. Enforcement Director: Charles L. Crews, Regional Supervisor

18. $[illegible]

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1, Code of Virginia.

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006855

A115

Virginia Department of Labor and Industry
Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
09/24/87	15271674
5. Reporting ID	6. CSHO ID
355122	13863
7. Optional Report No.	8. Page No.
096 87	1 of 8

1. Type of Violation(s)	2. Citation Number
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Sector May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
1 1910.1001(d)(2)(i):	Employer did not perform initial monitoring of employee airborne asbestos exposures as work operations covered by this standard where exposure might reasonably be expected to be at or above the action level: Location - Pellitizer Room. Operation - Pellitizer operation. Condition - Employee working at pellitizer had not been monitored to determine his 8 hour TWA airborne asbestos exposure.	IMMEDIATE	-0-
2 1910.1001(f)(2)(i):	The employer did not establish and implement a written program to reduce employee exposure to at or below the PEL by means of engineering and work practice controls as required by paragraph (f)(1) of this section: Location - Block Preform Area. Operation - Block preform. Condition - Employee working at block preform machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Establish a written compliance program.	September 25, 1987	-0-

1 AUG 1987 RECEIVED

17. Enforcement Director: Charles L. Brown, Regional Supervisor

18. Total: -0-

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia.

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

Total Penalty for This Citation

Make Check or Money Order Payable to "Virginia Department of Labor and Industry"

Indicate Inspection Number on Remittance

ORIGINAL

JWK006856

A116

Virginia Department of Labor and Industry

Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	152718474
5. Reporting ID	6. CSHO ID
355127	13843
7. Optional Report No.	8. Page No.
004 87	2 of 8

1a. Type of Violation(s)	2. Citation Number
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Section May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
3 1910.1001(h)(1):	Employer did not provide at no cost and ensure that the employee used appropriate protective work clothing and equipment where employees were exposed to asbestos, tremolite, anthophyllite, actinolite or a combination of these minerals above the PEL: Location - Block Prefora Area. Operation - Block prefora. Condition - Employee working at block prefora machine 416 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Provide appropriate equipment as defined in 1910.1001(h)(1)(i)-(iii).	IMMEDIATE	-0-
4 1910.1001(j)(2)(i):	Employer did not ensure that employees who work in areas where there airborne exposure is above the permissible exposure limit shower at the end of the work shift: Location - Block Prefora Area. Operation - Block prefora. Condition - Employee working at block prefora machine 416 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure.	IMMEDIATE	-0-

17. Enforcement Director

18. -0-

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006857

Virginia Department of Labor and Industry
Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	15221674
5. Reporting ID	6. CSHO ID
355122	L3863
7. Optional Report No.	8. Page No.
006 87	3 of 9

1a. Type of Violation(s)	2. Citation Number
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Paperalli Road, Winchester, VA 22601

8. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet) The Sector May Be Detached Before Posting

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
	ABATEMENT: Require showers at the end of workshift for all over exposed employes.		
5a 1910.1001(j)(1)(i):	Employer did not provide and display warning signs at each regulated area and additionally did not post warning signs at all approaches to regulated areas so that an employee may read the signs and take necessary protective steps before entering the area: Location - Block Prefore Area. Operation - Block prefore. Condition - Employee working at block prefore machine 116 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc. Sample period of 475 minutes with 5 minutes assumed zero exposure.	September 3, 1987	-0-
	ABATEMENT: Post signs in regulated areas and approaches which meet requirements established in 1910.1001(j)(1)(ii).		
5b 1910.1001(j)(2)(ii):	Labels affixed to raw materials, strictures, scrap, waste, debris, and other products containing asbestos, tremolite, anthophyllite, or actinolite fibers did not comply with the requirements of 29 CFR 1910.1200(f) of OSHA Hazard Communication Standard and did not contain the following: DANGER, CONTAINS ASBESTOS FIBERS, AVOID CREATING DUST, CANCER AND LUNG DISEASE HAZARD:		

AUG 1987 RECEIVED

17. Enforcement Director

18. -0-

Total Penalty for This Citation

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006858

A118

Virginia Department of Labor and Industry
Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	15221674
5. Reporting ID	6. CSHO ID
355122	L3863
7. Optional Report No.	8. Page No.
006 87	5 of 8

1. Type of Violation(s)	2. Citation Number
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

8. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Section May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.
An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.
You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated / 14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
3d 1910.1001(j)(5)(iii)(E): Employees exposed to asbestos at or above the action level were not informed of the specific procedures implemented to protect employees from exposure to asbestos, tremolite, anthophyllite, or actinolite, such as appropriate work practices, emergency and clean-up procedures, and personal protective equipment to be used: Location - Block Prefore Area. Operation - Block prefore. Condition - Employee working at block prefore machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc (Action level .1 fiber/cc). Sample period 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Include cited sections in asbestos training program.	September 25, 1987	-0-
3e 1910.1001(j)(5)(iii)(G): Employees exposed to asbestos at or above the action level were not informed of the purpose and description of the medical surveillance program required by paragraph (v) of this section: Location - Block Prefore Area. Operation - Block prefore.	September 25, 1987	-0-
17. Enforcement Director		18. -0-

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1, Code of Virginia

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006860

Virginia Department of Labor and Industry

Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	15221674
5. Reporting ID	**6. CSHO ID**
355122	L3863
7. Optional Report No.	**8. Page No.**
006 87	6 of 8

1. Type of Violation(s)	2. Citation Number
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp.-Friction Products Div. and its successors, P. O. Box 3250, Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Section May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
	Condition - Employee working at block preform machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc (Action level .1 fiber/cc). Sample period 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Include cited sections in asbestos training program.		
5f 1910.1001(j)(5)(iii)(H):	Employees exposed to asbestos at or above the action level were not given a review of the asbestos standard and its appendices: Location - Block Preform Area. Operation - Block preform. Condition - Employee working at block preform machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc (Action level .1 fiber/cc). Sample period 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Include cited section in training program.	September 25, 1987	-0-
6 1910.1001(l)(6):	Employer did not provide to the examining physician information required in 29 CFR 1910.1001(l)(6)(i)-(v):	IMMEDIATE	-0-

RECEIVED AUG 1987

17. Enforcement Director

18. -0-

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

JWK006861

Virginia Department of Labor and Industry

Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below.

3. Issuance Date	4. Inspection Number
08/24/87	15221674
5. Reporting ID	6. CSHO ID
355122	L3863
7. Optional Report No.	8. Page No.
006 87	7 of 8

1. Type of Violation(s)	2. Citation Number(s)
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

9. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested (See enclosed Booklet)

This Section May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

You are hereby notified that the Department of Labor and Industry has proposed penalty(ies) in the amount set forth below and in accordance with the Virginia Occupational Safety and Health Law as a result of the alleged cited violation(s). You have the right to contest any or all parts of either the citation(s), the abatement period(s) or the proposed penalty(ies) by notifying the Commissioner of Labor and Industry. If you do contest, you should submit a letter to the Commissioner at the address shown above within 15 working days after receipt of the certified mail notice. If you fail to contest within the 15-working-day-period, the citation(s), abatement period(s) and/or penalty(ies) shall be deemed to be a final order of the Commissioner and not subject to review by any court or agency.

12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date by Which Violation Must Be Abated	16. Penalty
	Location - Block Prefora Area. Operation - Block prefora. Condition - Employee working at block prefora machine #16 on 5/7/87 was exposed to an 8 hour TWA asbestos exposure of .24 fibers/cc (action level .1 fiber/cc). Sample period 475 minutes with 5 minutes assumed zero exposure. ABATEMENT: Furnish required information to examining physician when future physicals are performed.		
7 1910.1001(a)(1)(i)(i):	Employer did not keep an accurate record of all measurements taken to monitor employee exposure to asbestos, tremolite, anthophyllite, or actinolite as prescribed in paragraph (d) of this section: Location - Entire Plant. Operation - Asbestos containing materials. Condition - Employee personnel records did not contain the information required in section (a)(1)(i)(i)A-F. ABATEMENT: Include all monitoring results and pertinent information in personnel files.	September 15, 1987	-0-
8a 1910.1200(g)(2)(v):	Each material safety data sheet for hazardous chemicals did not include the	September 15, 1987	-0-

17. Enforcement Director

18.

RECEIVED 4 AUG 1987

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation.

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia

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CITATION AND NOTIFICATION OF PENALTY — ORIGINAL

JWK006862

A121

Virginia Department of Labor and Industry

Occupational Safety and Health Program

P.O. Box 12064
Richmond, VA 23241

Citation and Notification of Penalty

The violation(s) described in this Citation are alleged to have occurred on or about the day the inspection was made unless otherwise indicated within the description given below

3. Issuance Date	4. Inspection Number
09/24/87	152214474
5. Reporting ID	6. CSHO ID
355122	L3963
7. Optional Report No.	8. Page No.
006 87	8 of 8

1. Type of Violation(s)	2. Citation Number(s)
Other	2

10. Inspection Date(s): 02/26/87 - 08/20/87

11. Inspection Site: 2410 Papermill Road, Winchester, VA 22601

8. To: Abex Corp.-Friction Products Div.
and its successors
P. O. Box 3250
Winchester, Virginia 22601

Penalties Are Due Within 15 Working Days of Receipt of This Notification Unless Contested

(See enclosed Booklet)

This Sect May Be Detached Before Posting

THE LAW REQUIRES that a copy of this citation shall be prominently posted in a conspicuous place at or near each place the alleged violation referred to in the citation occurred. The citation must remain posted until all alleged violations cited therein are corrected, or for 3 working days*, whichever period is longer.

An inspection of a place of employment has revealed conditions which we believe do not comply with the provisions of the Virginia Occupational Safety and Health Law as set forth in Title 40.1, Code of Virginia. The nature of such alleged violation(s) is described below with references to applicable standards, rules, regulations and provisions of the said law. These conditions must be corrected on or before the date shown to the right of each alleged violation therein.

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12. Item Number / 13. Standard, Regulation or Section of the Law Violated	14. Description	15. Date By Which Violation Must Be Abated	16. Penalty
	Location - Plant Wide. Operation - Plant Wide. Condition - Engelhard calcium hydroxide ABD-10 and other MSDS did not include primary route of entry. ABATEMENT: MSDS should be updated to include primary route of entry.		
8b 1910.1200(g)(2)(xi)	Each material safety data sheet for hazardous chemicals did not include primary route of entry: Location - Plant Wide. Operation - Plant Wide. Condition - Engelhard calcium hydroxide ABD-10 and other MSDS did not include the date preparation of the MSDS or the date of last change to it. ABATEMENT: MSDS should be updated to include date of preparation or last change to it.	September 25, 1987	-0-

17. Enforcement Director: Charles L. Cleveland, Regional Supervisor

18. -0-

Total Penalty for This Citation

RIGHTS OF EMPLOYEES

Any employee or representative of the employees who believes that any period of time fixed in this citation for the correction of a violation is unreasonable has the right to contest such time for the correction by submitting a letter to the Commissioner of Labor and Industry at the Address shown above within 15 working days of the issuance of this citation

"No person shall discharge or in any way discriminate against an employee because the employee has filed a safety or health complaint or has testified or otherwise acted to exercise rights under the safety and health provisions of this title for themselves or others." Section 40.1-51.2:1 Code of Virginia

*The term "Working Day" means Monday through Fridays but does not include Saturdays, Sundays or Legal Holidays

ORIGINAL

JWK006863

A122

Mr. C. L. Clouse
Supervisor of Enforcement
Division of Occupational Health
Department of Labor and Industry
P. O. Box 2708
Staunton, Virginia 24401

Dear Mr. Clouse:

The purpose of this letter is to request that we be granted an Informal Conference in your office, 934 N. Augusta Avenue, Staunton, Virginia or other state office, on _____ at _____.
Date Time

A copy of this letter has been posted with the citations to notify employees of our Informal Conference request. Posting this letter affords an opportunity for employees to also request an Informal Conference.

Management Rep. _____

Company Name _____

Inspection Number _____

JWK006864

Excerpts from

ABEX'S ANSWERS TO CERTAIN INTERROGATORIES

* * *

15. Did the Defendant sponsor since 1930 for its employees or distributors any meetings, seminars, conferences, or conventions where the subject of occupational health and exposure to asbestos was discussed.

ANSWER: Yes.

16. If the answer to Interrogatory 15 is in the affirmative, state:

a) The date and place of such meeting, seminar, conference, or convention where the subject of occupational health and exposure to asbestos was discussed.

ANSWER: Meetings were held for employees at Winchester plant in approximately early 1978.

b) The name and address of the speaker or discussant.

ANSWER: Not applicable.

* * *

c) The method used to distribute the warning to persons who are likely to use the products.

ANSWER: By hand.

d) The date each such warning was issued.

ANSWER: Approximately early 1978.

* * *

Respectfully submitted,

Richard B. Scherrer

Richard B. Scherrer
611 Olive Street, Suite 1900
St. Louis, Missouri 63101
(314) 621-5070
Attorneys for separate defendant
ABEX CORPORATION

OF COUNSEL:

ARMSTRONG, TEASDALE, KRAMER & VAUGHAN

STATE OF ~~VIRGINIA~~ NEW YORK)
) SS.
COUNTY OF ~~FREDERICK~~ NEW YORK)

The undersigned, A. H. Casey, of lawful age, being first duly sworn upon his oath, states that he is corporate counsel of defendant, ABEX CORPORATION, and that as such he is authorized to execute this Affidavit; affiant further states that he has reviewed the foregoing Answers and Objections to Interrogatories which have been prepared based upon such information and documents as are available to said defendant and that said Answers and Objections are true and accurate to the best of affiant's knowledge, information and belief.

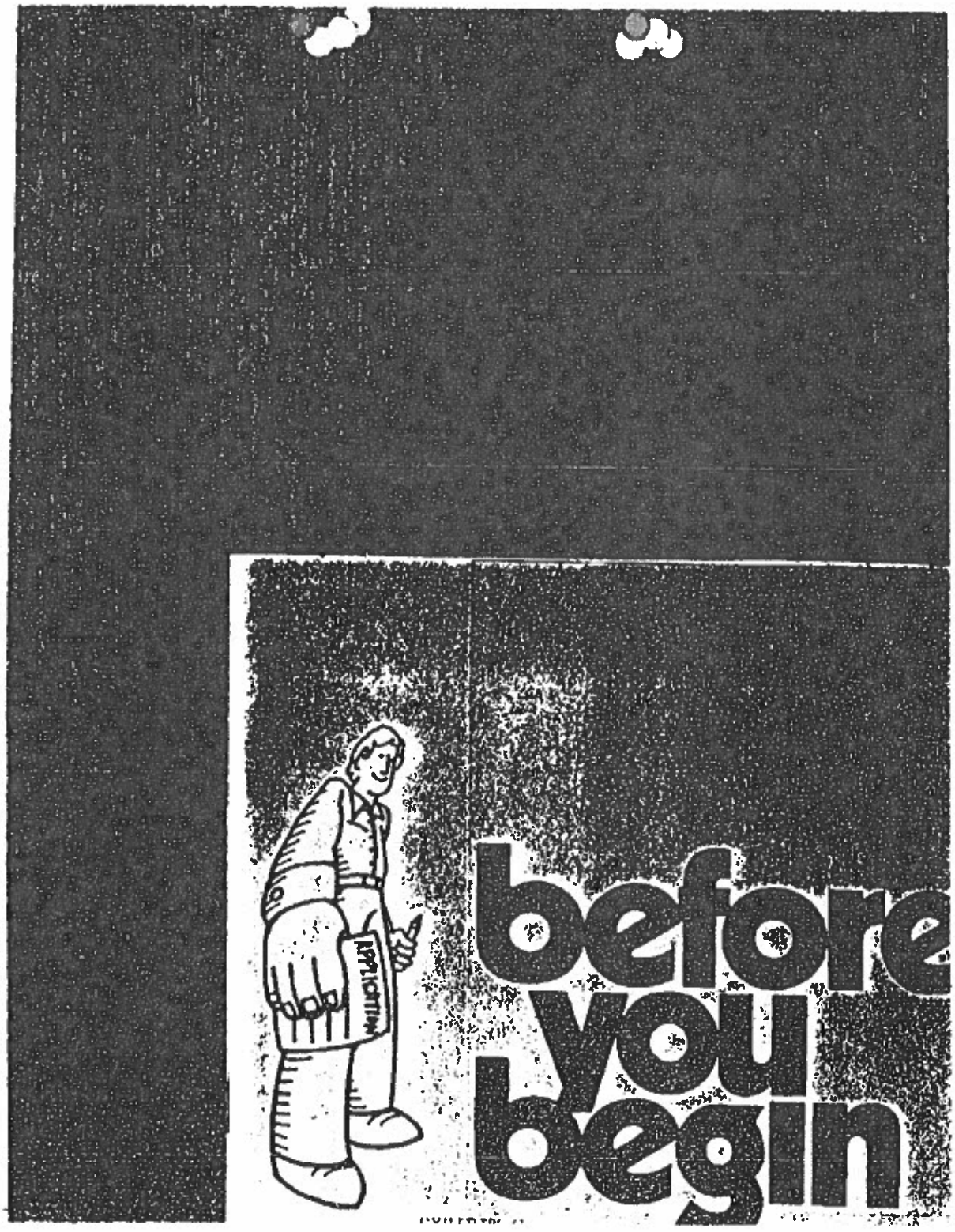
A. H. Casey

Subscribed and sworn to before me, a Notary Public, this 7th day of November, 1983.

Eileen M. Godin
Notary Public

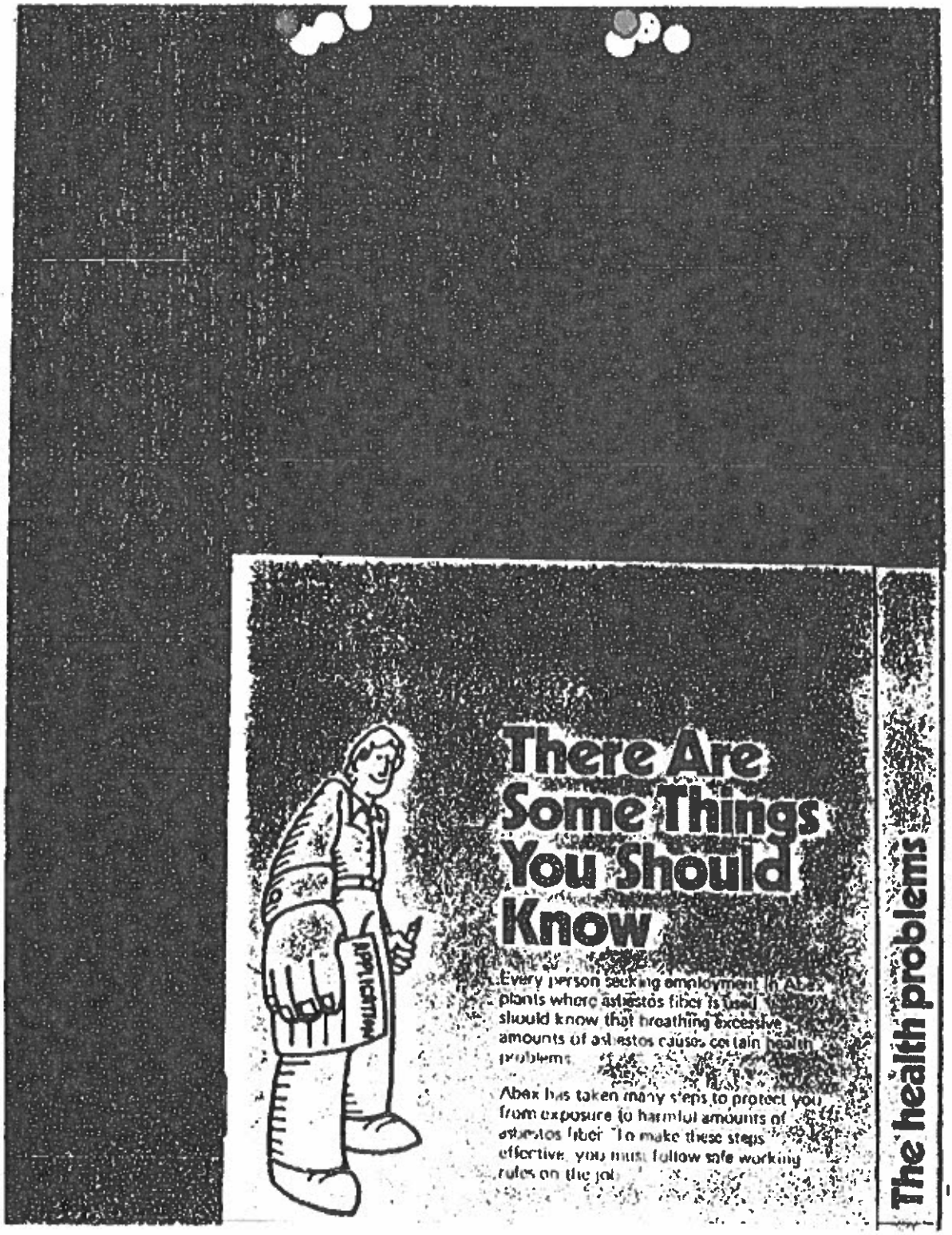
EILEEN M. GODIN
NOTARY PUBLIC, State of New York
No. 41 4711565
Qualified in Queens County
Cert. Filed in New York County
Commission Expires March 30, 1984

CC6720



CC6721

A126



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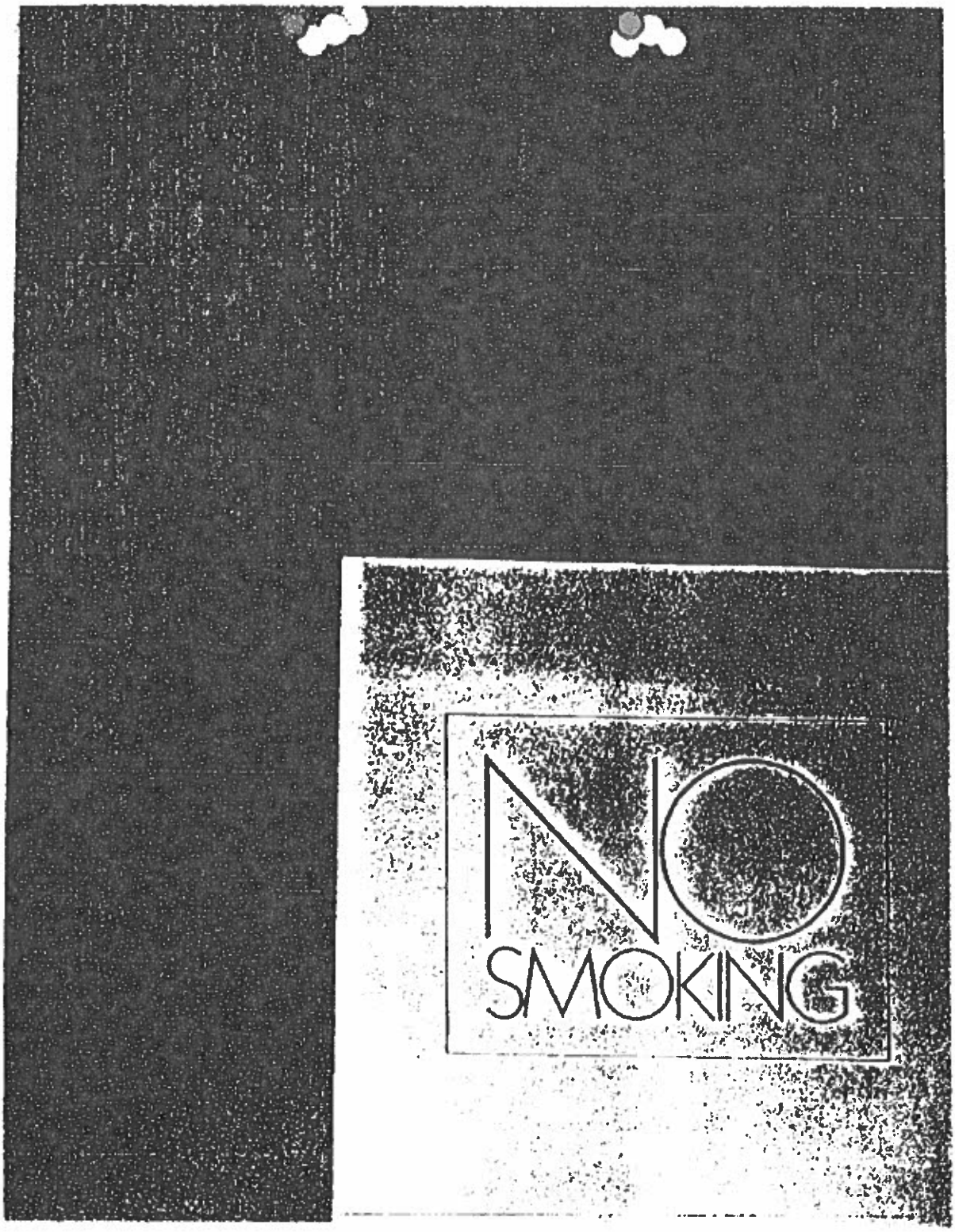

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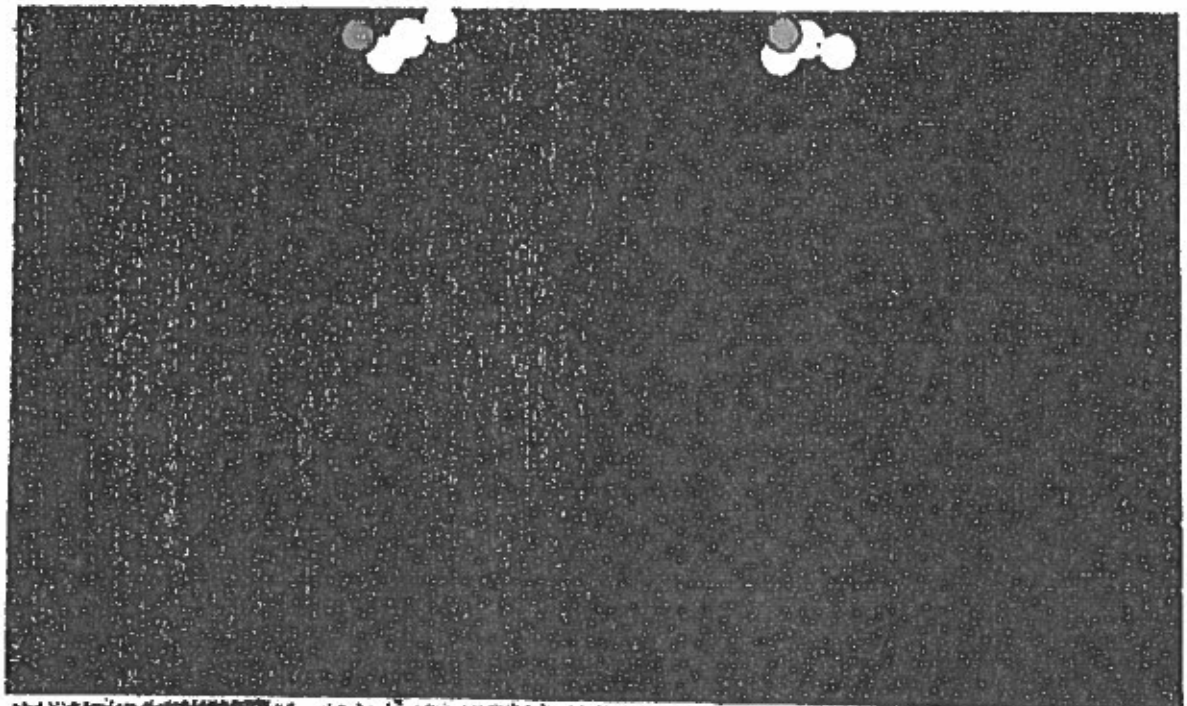

The health problems

The health problems that have been connected with exposure to asbestos are these:

- Breathing excessive amounts of asbestos fibers for many years might result in a disease called asbestosis. Asbestosis is a scarring of lung tissue which, when severe, makes it difficult to breathe properly.
- Persons who smoke cigarettes and inhale excessive amounts of asbestos dust are at a greater risk of developing lung cancer. Asbestos alone does not appear to cause this disease, but it can make people who smoke cigarettes more likely to develop this cancer. Studies show that if you don't smoke cigarettes, asbestos exposure does not increase your risk of getting lung cancer.
- Persons exposed to excessive amounts of asbestos have a greater risk of developing mesothelioma, which is a rare cancer of the lining of the chest or abdomen. There also is some indication from unconfirmed studies that there might be a greater incidence of cancer of the digestive tract in persons exposed to excessive amounts of airborne asbestos.

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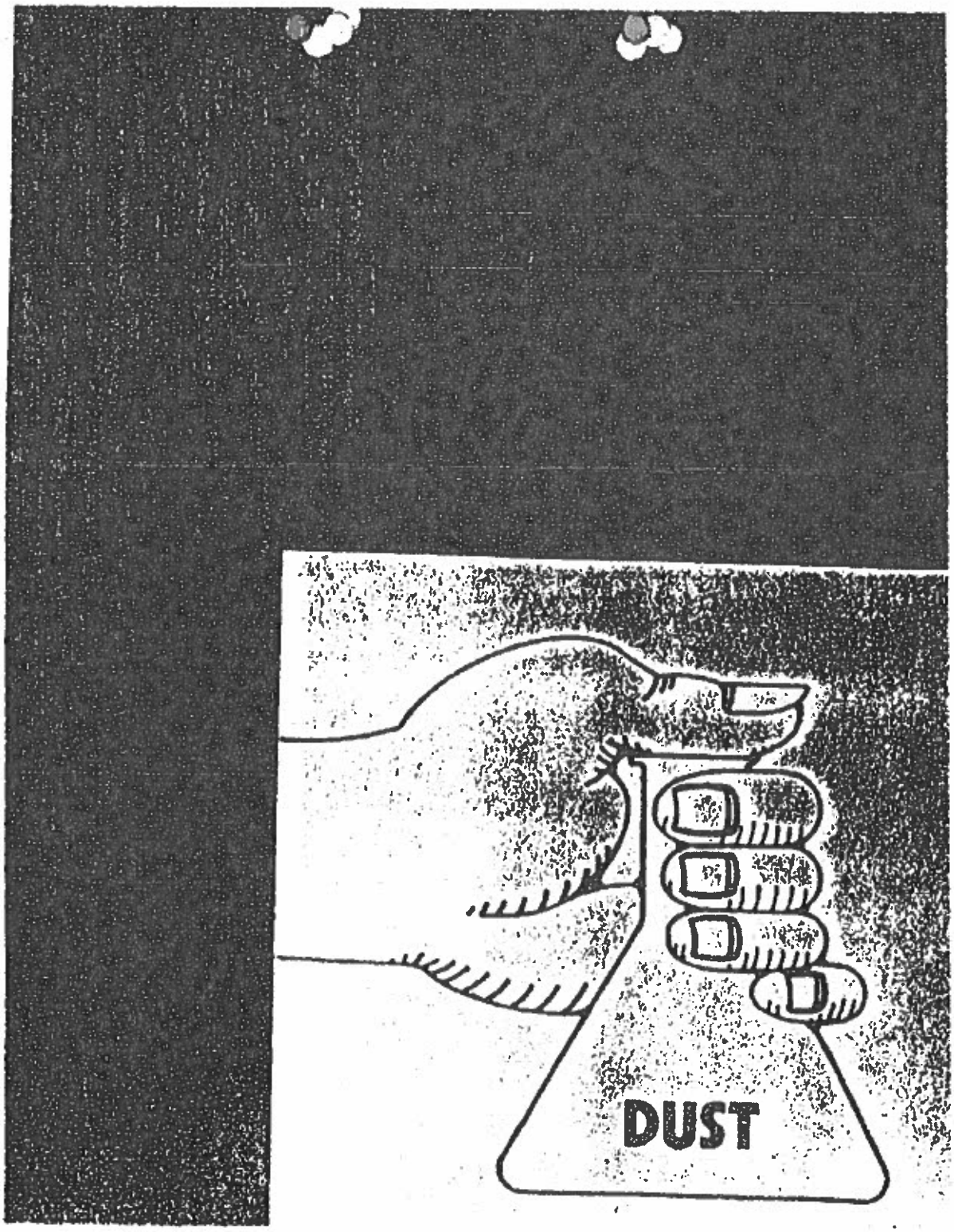

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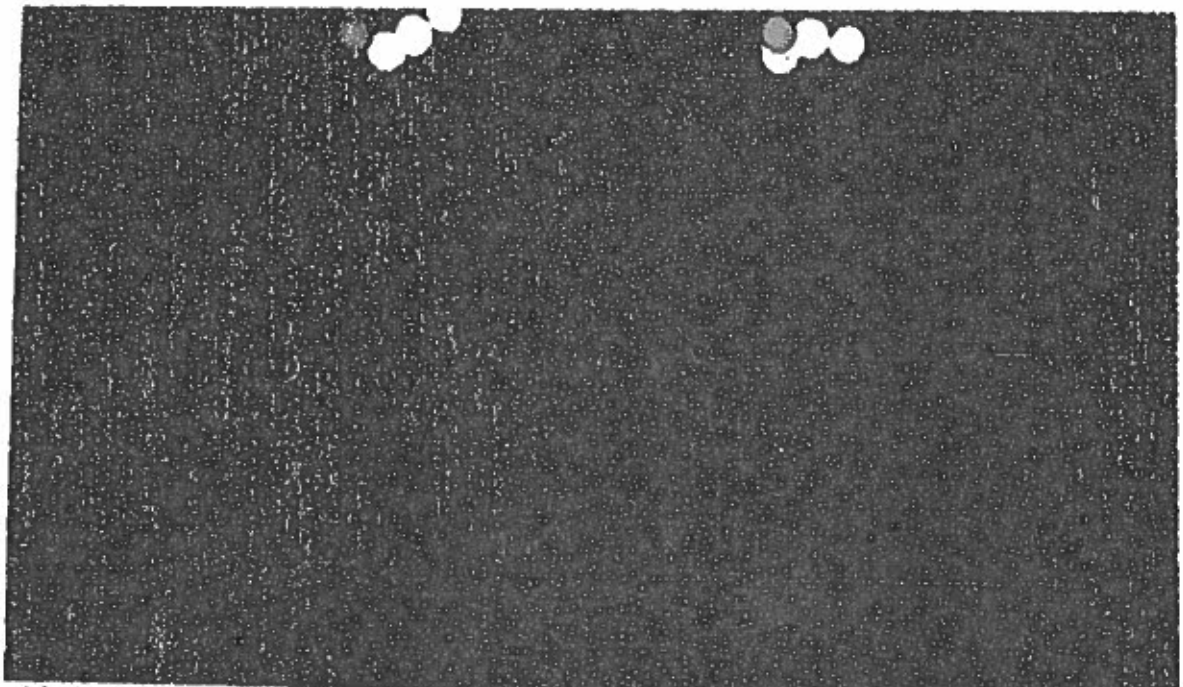

Be concerned, not alarmed

Be concerned, not alarmed about asbestos-related health problems in coming to work for Abex today. Present cases of the health problems we mentioned are related to working conditions that existed many years ago. As we became aware of the risks, Abex began spending millions of dollars to improve equipment and processes and to upgrade dust control systems to prevent excessive asbestos exposures. We are confident that as long as employees follow safe work rules and equipment is operated properly, no employee will be exposed to hazardous amounts of asbestos fiber in our operations.

To assure continued control of risk, the company will be regularly checking asbestos-using operations with trained industrial hygienists. You must cooperate with these hygienists when they are checking your work areas.

Government inspections also are made of our facilities to make sure you are not being exposed to hazardous levels of asbestos. The government has set standards that limit how much asbestos you can be exposed to, and Abex abides fully with those regulations.

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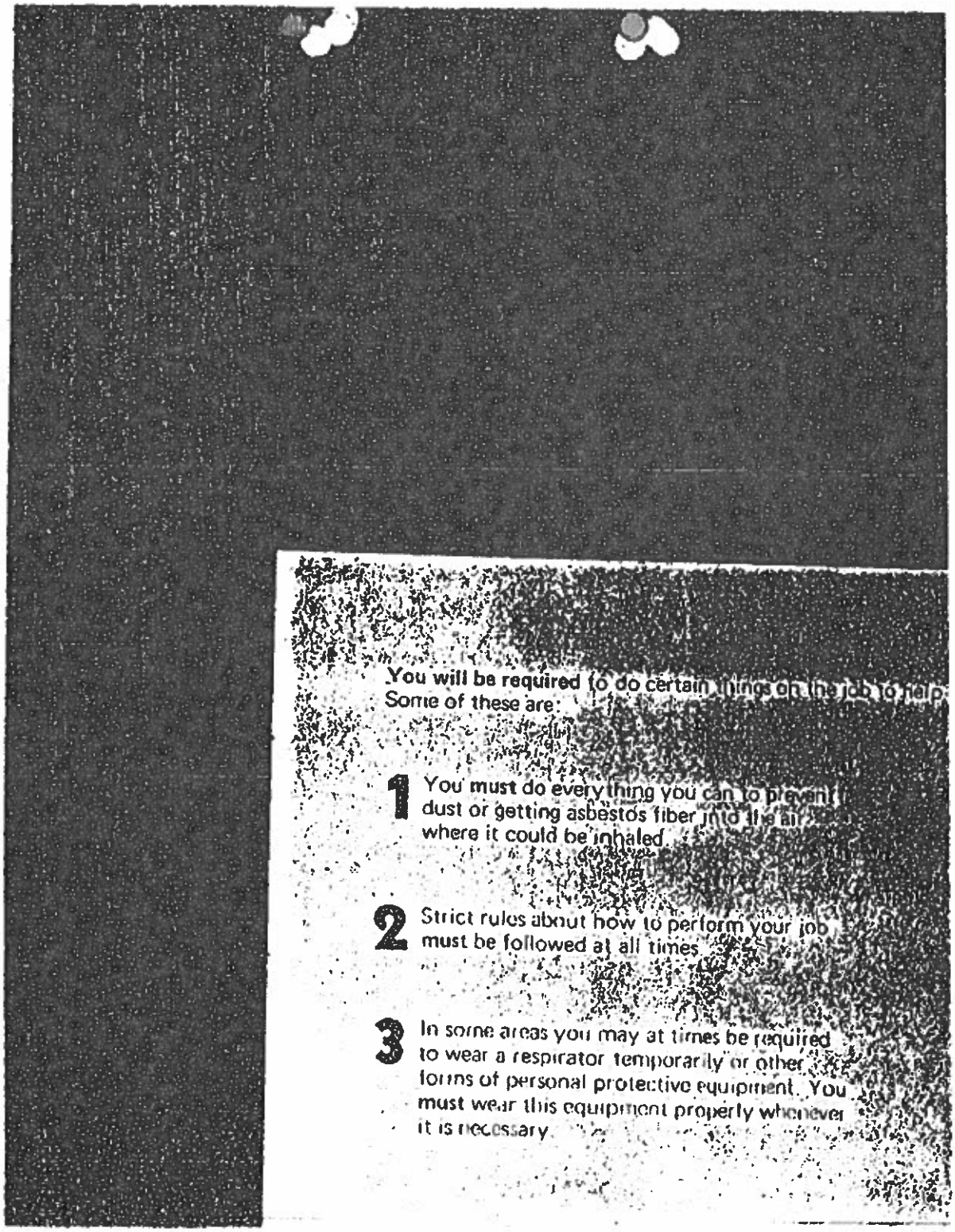

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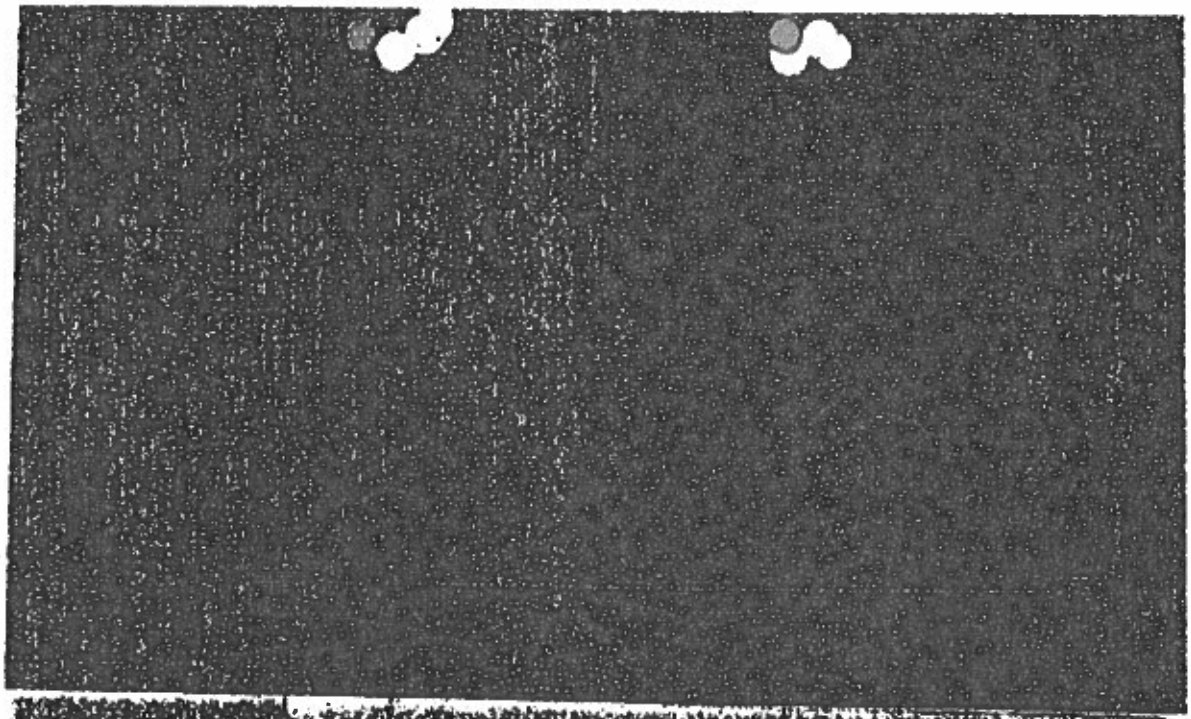

s on the job to help protect you

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whenever

4 You will be requested not to smoke on the job if you might be exposed to asbestos. You will be urged, for your health's sake, to quit smoking completely.

5 You will be encouraged to use facilities provided to change work clothes and wash before going home.

6 If equipment is not working correctly, you must report it to your supervisor immediately.

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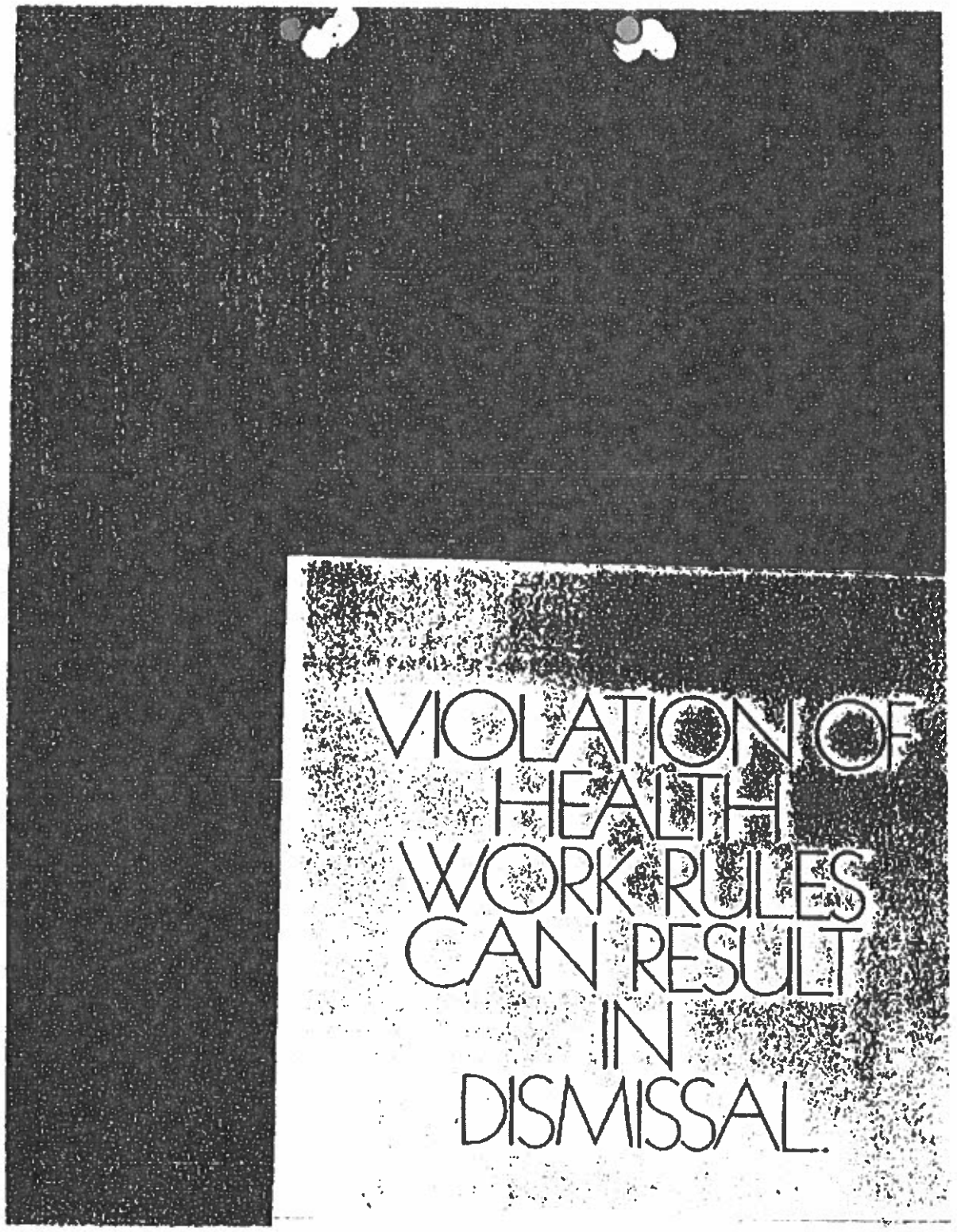

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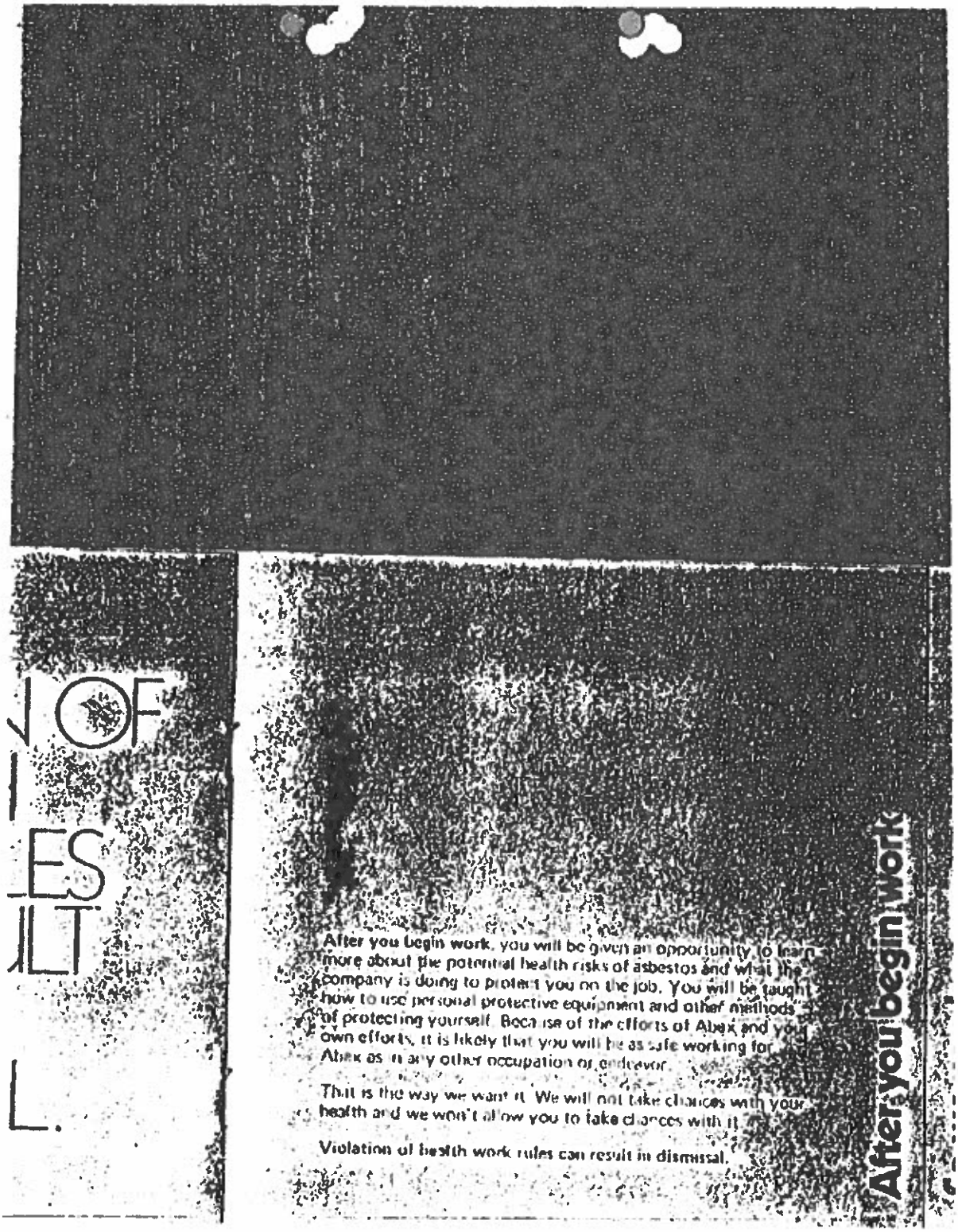

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After you begin work

After you begin work, you will be given an opportunity to learn more about the potential health risks of asbestos and what the company is doing to protect you on the job. You will be taught how to use personal protective equipment and other methods of protecting yourself. Because of the efforts of Abex and your own efforts, it is likely that you will be as safe working for Abex as in any other occupation or endeavor.

That is the way we want it. We will not take chances with your health and we won't allow you to take chances with it.

Violation of health work rules can result in dismissal.

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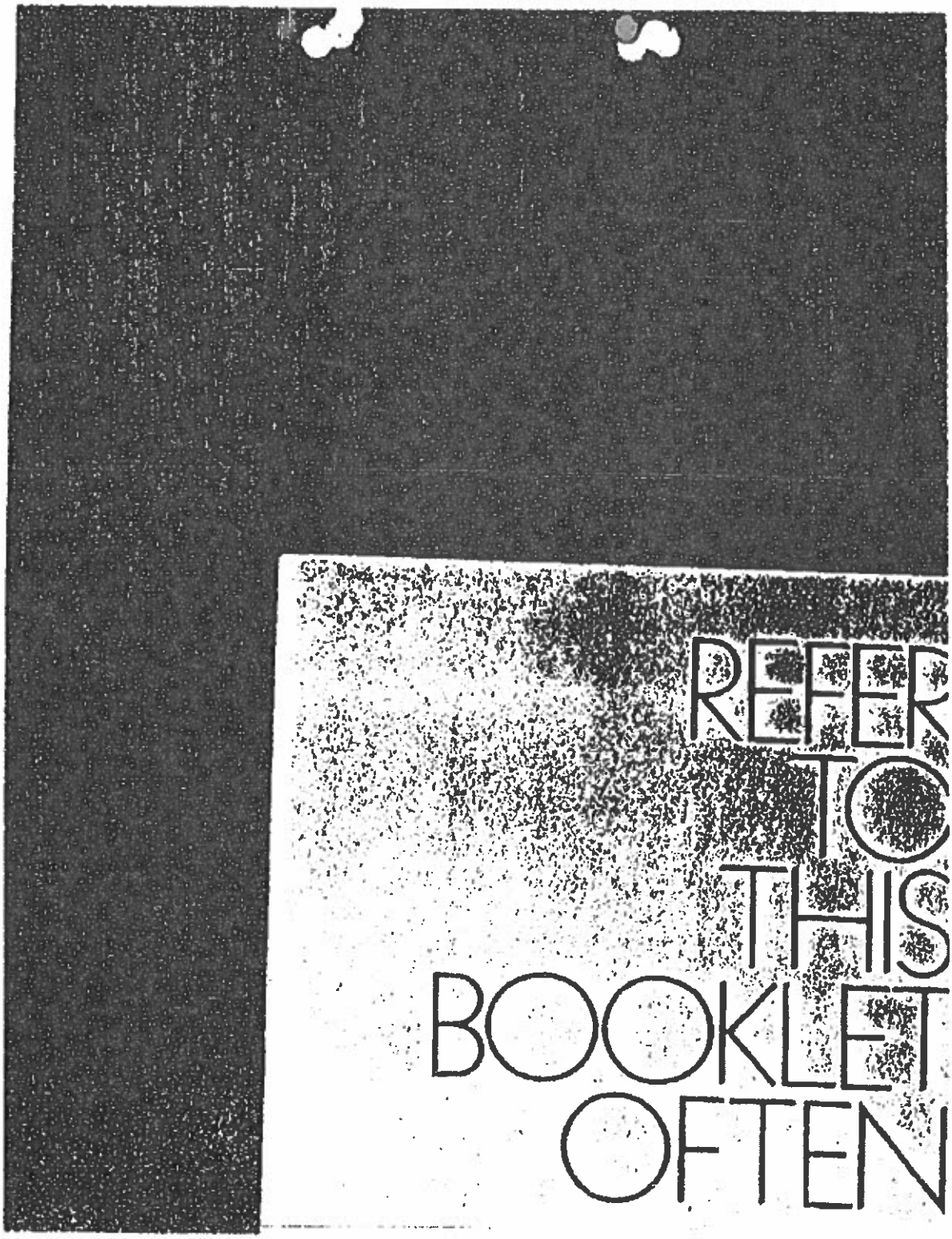

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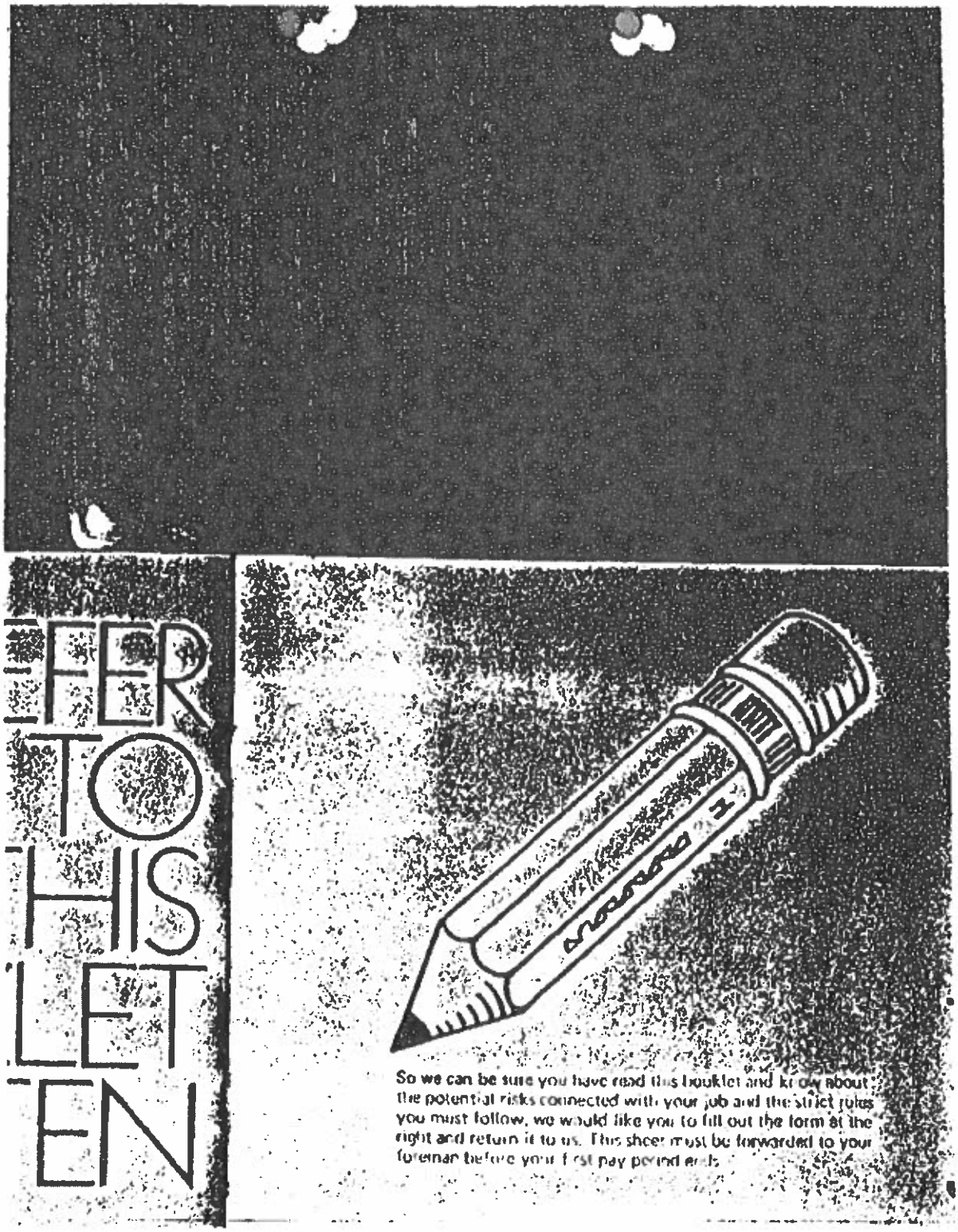

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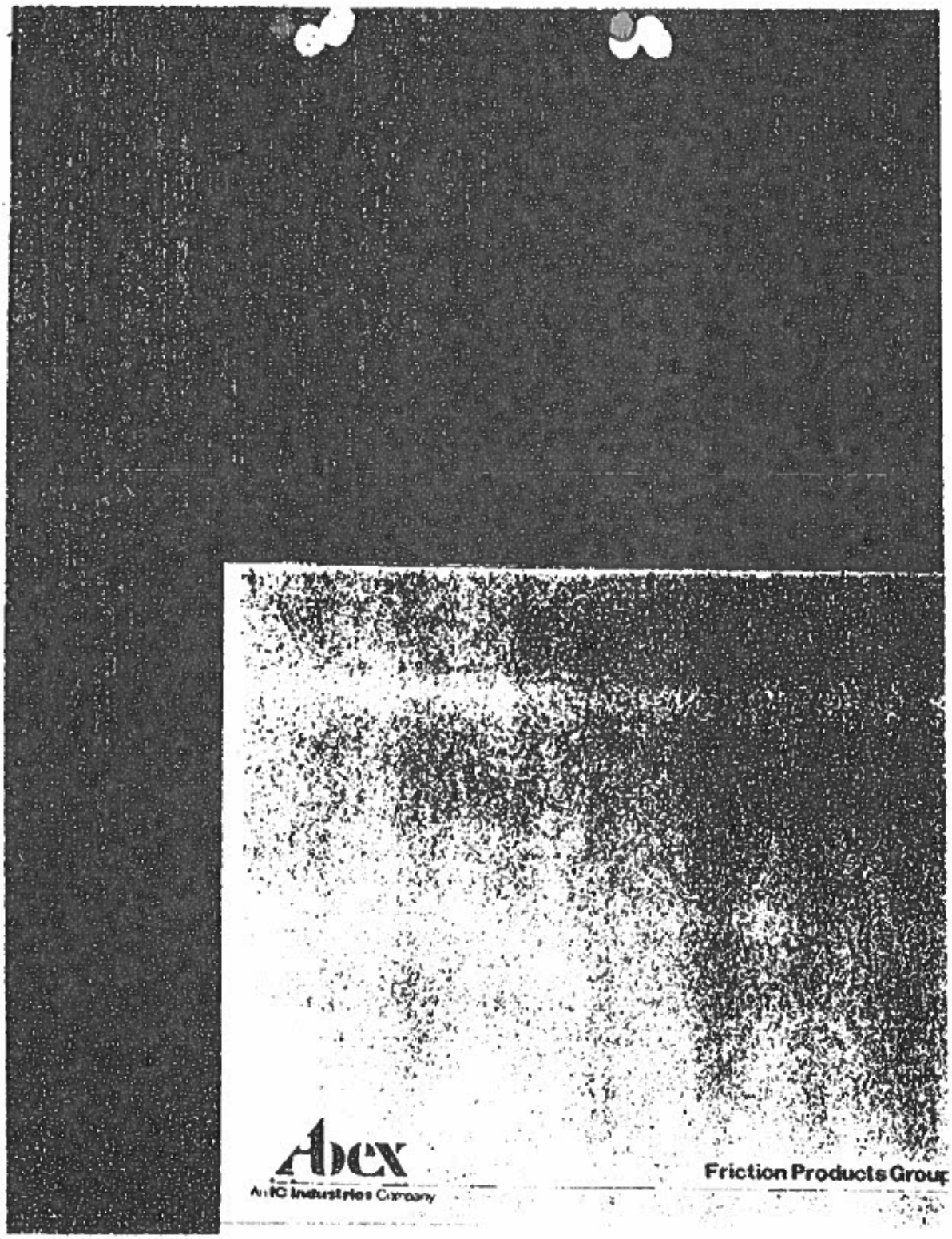

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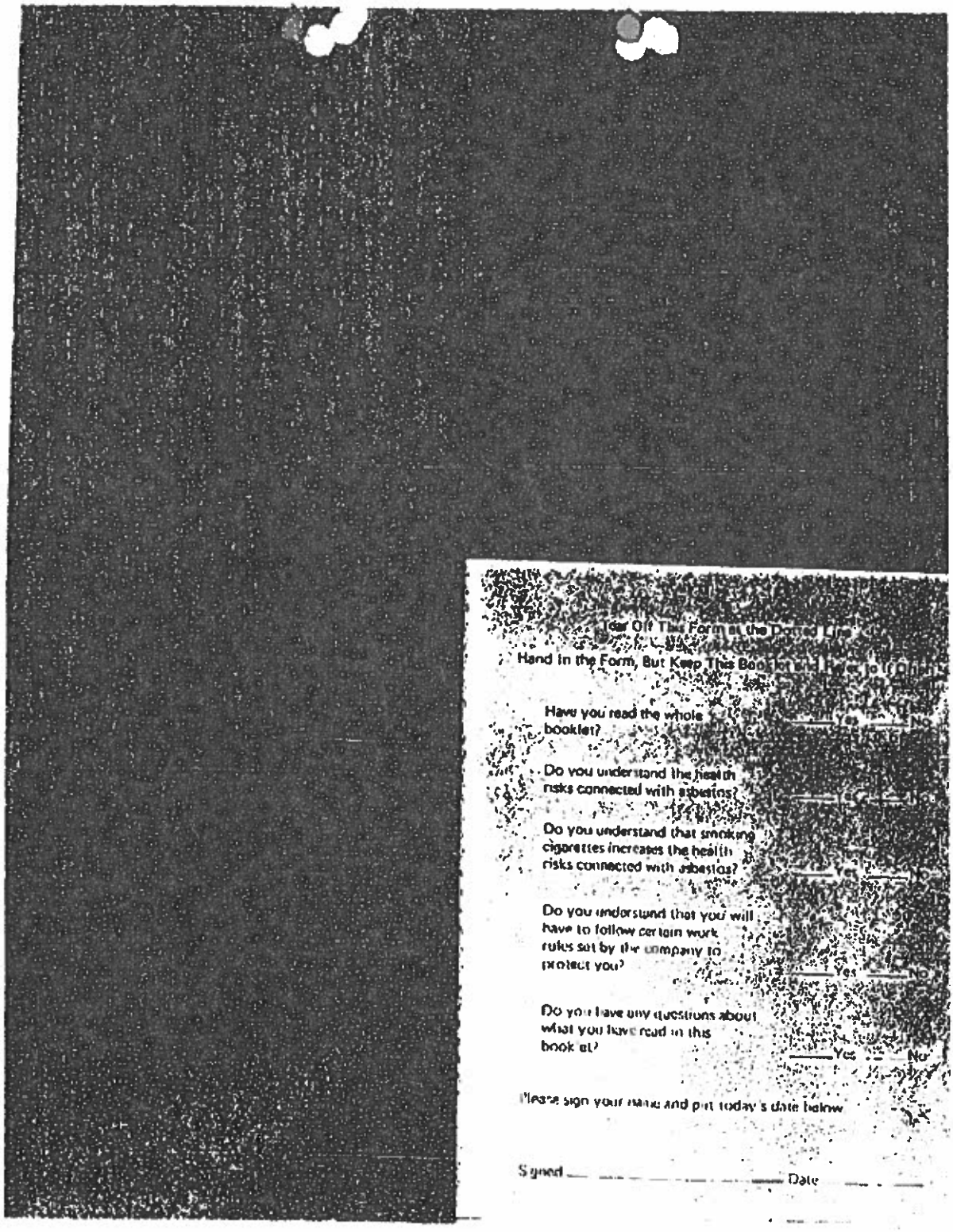

Tear Off This Form at the Dotted Line

Hand in the Form, But Keep This Booklet and Refer to It Often

Have you read the whole booklet? ____Yes ____No

Do you understand the health risks connected with asbestos? ____Yes ____No

Do you understand that smoking cigarettes increases the health risks connected with asbestos? ____Yes ____No

Do you understand that you will have to follow certain work rules set by the company to protect you? ____Yes ____No

Do you have any questions about what you have read in this book at? ____Yes ____No

Please sign your name and put today's date below

Signed _____ Date _____

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[redacted]

Bridgeport, Conn.
November 10, 1936

Mr. H. D. LaMont,
Asbestos Manufacturing Co.,
Huntington, Ind.

My dear Mr. LaMont:

The writer and Mr. Vandiver Brown, of Johns-Manville, had a conference with Dr. Leroy Gardner, of Saranac, New York, and Dr. Lanza and Dr. McConnell, of the Metropolitan Life Insurance Co., with reference to the Asbestosis situation in the Asbestos industry.

As you no doubt know, Johns-Manville and ourselves have been doing considerable work, in conjunction with the Metropolitan Life Insurance Co., in the way of eliminating Asbestos Dust in our factories, and we have made a very satisfactory job of it.

We have now reached the point where we can eliminate a large part of the Dust, but as yet have no definite information as to what Asbestosis will lead into. They claim it leads to Tuberculosis, but we do not find this to be the case. Still, we cannot go into court and state definitely and specifically that it will not do so. We do know that Asbestos Fibres can, and do, get into the lungs, and may set up a Fibrosis condition, which, for want of a better name, some doctors have called Asbestosis.

Dr. Gardner has a well equipped Laboratory, and a number of separate Dust Chambers in his Laboratory at Saranac, and he and his associates have been studying various types of Dust for a number of years, in connection with Tuberculosis, but practically nothing has been done with Asbestos.

In order to carry on a systematic Dust investigation, it takes from two to three years, and these investigations are generally paid for by industry, mostly working as a unit.

Dr. Gardner's Laboratory charge is $5,000.00 a year, and any study should be based on a three year period, in order to get results that will stand up and be accepted by the medical fraternity, and, I believe, by the courts.

I think the Compensation Laws in the various states will become more rigid in the next few years, and, no doubt, Asbestos, on account of the advertising it has had lately, will become one of the Compensation cases, and we should have all the information we can possibly get to submit to the Compensation Commissions of the various states when the question of Asbestosis comes up.

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Dr. Gardner now has a Dust Chamber open and could start work on experimenting with Asbestos at once, and has suggested to us

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that the Asbestos industry take over this Dust Chamber and employ him to carry on the experiments with guinea pigs and rabbits.

The idea of Mr. Brown and myself would be to have four or five (or even more if we could get them) Asbestos manufacturers take over this study by subscribing an equal amount per year, for three years, and then we could determine from time to time after the findings are made, whether we wish any publication or not. My own idea is that it would be a good thing to distribute the information among the medical fraternity, providing it is of the right type and would not injure our companies.

This would mean, if four of us went in to carry on this work, it would be $1,250.00 a year for each one of us for a period of three years.

If you are interested in this subject, I should like to have a meeting in New York, on Tuesday, November 17, at 12:30 o'clock, at The Biltmore, or if this is not satisfactory, can make it any place you care to have it.

I am asking the following to attend the meeting,

Mr. Vandiver Brown, Johns-Manville, Inc.
Mr. F. H. Schluter, Thermoid Rubber Co.
Mr. A. S. Blagden, Keasbey & Mattison Co.
Mr. H. D. LaMont, Asbestos Manufacturing Co.
Mr. G. M. Williams, Russell Manufacturing Co.
Mr. S. Simpson, Raybestos-Manhattan, Inc.

Is there any one else you would suggest?

Very truly yours,

SS-C.

President

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JOHNS-MANVILLE CORPORATION
TWENTY-TWO EAST FORTIETH STREET
NEW YORK, N.Y.

EXECUTIVE OFFICES

May 3, 1939.

Sumner Simpson, Esq.,
President,
Raybestos-Manhattan, Inc.,
Bridgeport, Conn.

Dear Mr. Simpson:

I wonder if you have seen the 1938 report of The Saranac Laboratory. If not, I suggest you obtain a copy from Dr. Gardner and note the references to Asbestosis on Page 9 of the "Report of the Director".

Included in the report are reprints of several articles or addresses delivered by various persons connected with the Laboratory. One of these by Dr. Gardner is a paper read before a meeting of the American Institute of Mining and Metallurgical Engineers in February, 1938. In this you will note also certain references to asbestos on Page 5.

There is also a reprint of an article by Dr. Gardner entitled "Etiology of Pneumoconiosis" reprinted from the November, 1938 issue of the Journal of the American Medical Association. In this article your attention is directed to a reference to Asbestosis on Pages 12, 13, and particularly at the top of Page 14.

The information covered by these references has presumably been derived from the experiments which Dr. Gardner is conducting for, and with funds provided by, the group of members of the Asbestos Textile Industry. The Progress Reports which we have received to date would seem to indicate as much. This raises a question upon which I would like to have your thoughts, namely, whether Dr. Gardner's use of this material is proper in view of the following paragraph from my letter of November 30, 1936, in which I outlined our proposal to Dr. Gardner:

> "It is our further understanding that the results obtained will be considered the property of those who are advancing the required funds,

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> who will determine whether, to what extent and in what manner they shall be made public. In the event it is deemed desirable that the results be made public, the manuscript of your study will be submitted to us for approval prior to publication."

The proposal contained in this letter was accepted by Dr. Gardner in a letter of November 23rd, from which I quote the following paragraph:

> "The Saranac Laboratory agrees that the results of these studies shall become the property of the contributors and that the manuscripts of any reports shall be submitted for approval of the contributors before publication."

Sincerely,

Vandiver Brown

Vandiver Brown,
General Attorney.

VB:y

t

Bridgeport, Conn.
May 4, 1939

Mr. Vernlver Brown, General Attorney
Johns-Manville Corporation
22 East Fortieth St.
New York, N. Y.

My dear Mr. Brown:

I have your letter of the 3rd, in reference to Dr. Gardner's papers giving some results of the experiments with the asbestos dust.

I have not seen any of Dr. Gardner's reports, the one of the Saranac Laboratory, the paper before the meeting of the American Institute of Mining and Metallurgical Engineers, or the one in the Journal of the American Medical Association, but I shall send for them at once.

I do not believe it is proper for Dr. Gardner to use any of the material regarding asbestosis without our consent, or without submitting the report to you for approval, and I am a little surprised that Dr. Gardner has done so. He is certainly not living up to his agreement of November, 1938.

The reports may be so favorable to us that they would cause us no trouble, but they might be just the opposite, which could be very embarrassing.

As soon as the articles are received, I will again take it up with you.

Very truly yours,

CHG

President

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CONFIDENTIAL

A S B E S T O S I S

Experimental Studies

by

THE SARANAC LABORATORY
SARANAC LAKE, NEW YORK

Report to the

JOHNS-MANVILLE CORPORATION
NEW YORK, NEW YORK

September 30, 1948

Personal copy
P.C. Platt

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ABSTRACT

Asbestosis is a pulmonary disease caused by the inhalation of asbestos dust. In animals it is characterized by a peribronchiolar fibrosis which seems to be the result of mechanical, rather than chemical, irritation of the tissue by asbestos fibers. Only the long fibers produce a typical reaction; short fibers are relatively inert. The filamented structure of the fibers is an essential factor in the mechanism of irritation. A characteristic tissue response can be produced by non-siliceous as well as siliceous fibrous minerals. Inhalation of asbestos dust apparently does not alter significantly the course of experimental tuberculosis in guinea pigs. The asbestosis body, which is a specific concomitant of asbestosis and forms soon after the entrance of the asbestos fiber into the lung, is believed to prevent further damage to the tissue by the fiber and thus to limit progression of the reaction when exposure ceases. Aluminum does not exert a protective action against the tissue irritation of asbestos fibers as it does against that of quartz particles.

EXPERIMENTAL STUDIES

OF

ASBESTOSIS

2. Introduction

Asbestosis is a form of pneumoconiosis resulting from prolonged inhalation of asbestos dust. The name asbestos, literally "unburnable," is not that of a particular mineral but is a term applied to a number of different minerals whose characteristic feature is a structure composed of long, parallel, flexible fibers. This structure is unique because the fibers are capable of repeated longitudinal subdivision to units of molecular proportions. In length the fibers vary from a few microns to six or more inches. Some varieties are stiffer than others but many are sufficiently flexible to be spun into yarn and woven on modified textile machinery.

3. Asbestos Minerals

The asbestos minerals are silicates of variable composition and belong to the serpentine and the amphibole groups. Listed below are the more common varieties.

Amphibole Group	
Anthophyllite	(Mg, Fe) silicate
Amosite	(Mg, Fe, Al) silicate
Amphibole	(Ca, Mg, Fe, Al, Na, K) silicate
Tremolite	(Ca, Mg) silicate
Actinolite	(Ca, Mg, Fe) silicate
Crocidolite	(Na, Fe) silicate + Fe silicate
Serpentine Group	
Chrysotile	Mg silicate (hydrous)

The bulk of the asbestos of commerce is chrysotile, $3MgO.2SiO_2.2H_2O$, which is mined in the Thetford region of the Province of Quebec. Crocidolite and amosite are also used commercially but in much smaller amounts. Chrysotile occurs as veins in serpentine, a mineral similar in chemical composition to chrysotile but which exists in massive form and is made up of microscopic fibers without the parallel orientation characteristic of chrysotile. The massive blue black serpentine, which is smooth and soapy to the touch, is traversed by veins of fibrous chrysotile varying in width from a barely perceptible line to six or more inches. The fibers run across the vein and not lengthwise with the formation.

Attention is directed to the mineral brucite, $MgO.H_2O$, which is often found in the same formations with serpentine and chrysotile and may be fibrous in structure. It has no commercial value at present because its fibers are not sufficiently flexible to be used in textiles but they are capable of repeated longitudinal subdivision. Unlike other asbestiform minerals, brucite is not a silicate and for this reason it has been a valuable tool in an experimental evaluation of the action of fibrous minerals upon lung tissue.

IV. EXPERIMENTAL ASBESTOSIS

For many years studies have been carried on by the Saranac Laboratory in an investigation of the cause, nature and development of asbestosis. The present report is devoted to Experimental Asbestosis. In it are described the animal experiments with various kinds of asbestos dust. Another report, to be prepared and issued later, will be concerned with Human Asbestosis and will cover the health aspects of workers who have been exposed to asbestos dust in an industrial environment.

Although asbestosis in man is a chronic disease which requires years to develop, it is possible to reproduce in one or more species of animal characteristic tissue changes which are similar to the lesions of human asbestosis. Since the life-span of the experimental animal is relatively short, it is not possible to develop the characteristic lesions in animals under the usual industrial conditions. Consequently, to obtain a complete evaluation of the tissue response to inhaled particulate and fibrous material, it is necessary to accelerate the reaction by employing higher concentrations of dust than would ordinarily be encountered in industry. While conditions of exposure are thus different, the information yielded by experiments with animals is invaluable in furnishing a better understanding of the reaction of the human organism to inhaled asbestos dust.

5. Experimental Methods

For investigating the biological reaction of the experimental animal to the various asbestos minerals, two types of technique have been employed, namely, the inhalation method and the injection method. In inhalation experiments, groups of animals - up to 100 or more guinea pigs and sometimes smaller numbers of rabbits, cats, dogs, rats or mice - are kept for eight hours a day in a cubical dust room, eight feet in dimension, in which a cloud of asbestos dust is maintained. At intervals during the experiment, a few animals are sacrificed and the tissue examined to determine the nature and extent of the dust reaction. Some animals are exposed for periods up to three years. The injection experiments, in which the dust, either dry or suspended in fluid, is introduced into the animal by the intravenous, intraperitoneal or intratracheal procedure, are used to determine whether or not a particular dust has a potential capacity to produce tissue reaction.

Long-term inhalation experiments furnish information upon which great reliance is placed when estimating the degree to which a dust might be hazardous to industrial workers. Whether or not atmospheric dust, even though potentially dangerous, can be inhaled, pass the natural defense barriers and reach the pulmonary tissue in quantities sufficient to cause damage can be determined only by inhalation procedures. Injection experiments are useful because in them contact between the dust particles and tissues is assured and the potential capacity of the dust to produce reaction can be estimated accurately. When dealing with fibrous minerals like asbestos, the intratracheal method is valuable since it permits observing the effect of the fibers on pulmonary tissue.

6. Species Susceptibility

Unlike free silica, asbestos does not exert its specific effect in all organs of all species of animal (Table 1). Injection of fine quartz into various organs of the guinea pig, rabbit, rat, cat, dog, chicken and even tadpole will produce silicotic nodules. However, similar injections of long or short fiber asbestos have resulted in a fibrous reaction in the lung and, to a lesser extent, in the peritoneum but not in other organs.

7. Peculiar Characteristics of Asbestos

Experience has demonstrated that most of the particulate matter inhaled into the lungs of man and animal is 10 microns or less in maximum diameter. Larger particles apparently are excluded by the protective mechanism of the upper respiratory tract. In the case of fibrous materials, however, this restriction does not apply and fibers 100 and even 200 microns in length have been found in the terminal air spaces of human lungs. In small laboratory animals exposed to asbestos dust the maximum length of fiber found in the lung rarely exceeds 60 microns. Not every kind of fibrous material is inhaled with equal readiness; for example,

the synthetic fibers of glass wool apparently are too inflexible to pass easily through the nose, pharynx, trachea and bronchi and seldom reach the terminal bronchioles and alveoli.

Inhaled particulate matter comes to rest throughout the terminal air spaces (alveolar duct, atria, alveoli) in all parts of the lungs; inhaled asbestos fibers are first retained in the respiratory bronchioles. These very small tubes are immediately distal to bronchioles lined by ciliated epithelium. Their own essential lining is a low cuboidal type of epithelium but, as their name implies, they actually function in respiration through lateral alveoli given off as pouches along their walls. Either these pouches, or the abrupt change in the character of the lining epithelium, or the decrease in diameter of the tube, or perhaps the combination of all three factors is responsible for local retention of the inhaled fiber. Only after asbestosis is well established are appreciable numbers of fibers carried into the more peripheral air spaces.

8. Rate of Tissue Reaction to Asbestos Fibers

The rate of tissue reaction to asbestos is much more rapid than to an active dust like quartz. Evidences of tissue response appear as soon as fibers have localized in sufficient concentration in specific areas. In rats receiving asbestos fibers by intratracheal injection this evidence is visible as early as two weeks after injection; for quartz dust the latent period might be two months or more.

The behavior of the tissue reaction to inhaled dust after the termination of exposure is not the same in silicosis as in asbestosis. In silicosis, the young nodules become larger; in asbestosis, young scar tissue, that may have formed, contracts and becomes more dense but the area of involvement decreases in size. If exposure to asbestos dust is terminated after a brief period, the recently-inhaled

fibers in the lung may cause the fibrous tissue response to continue for a short time, until the fibers have been coated. This progression is of only a slight degree and of little significance.

9. Asbestosis Bodies

The peculiar structure known as the asbestosis body or curious body is a specific concomitant of asbestosis. The typical body is a golden-yellow, beaded or haustrated rod which may be either straight or curved. Often one or both ends are bulbous like a dumb-bell. The bodies vary considerably in length, and dimensions up to 250 microns have been recorded.

It is believed that asbestosis bodies are due to a deposit of protein and iron pigment upon the surface of inhaled fibers. In guinea pigs they form after about 60 days of contact with the tissue. They are abundant in man and the guinea pig (see Table 1) but are much larger in the former, probably because the larger-sized air tubes admit fibers of greater dimension. In cats, rabbits and mice there is an atypical coating of a few of the fibers after much longer residence in the lungs. In rats and dogs no bodies could be discovered. Although the evidence is incomplete, it appears that the formation of the asbestosis body prevents damage to the tissue by the fiber.

X. INHALATION EXPERIMENTS

Four comprehensive inhalation experiments have been conducted at the Saranac Laboratory with various forms of asbestos dust. In each of these investigations more than 160 animals were used and the experiments were carried on for periods ranging from 2 to more than 5 years. The four kinds of asbestos dust employed are identified as King's floats, short fiber, 100 per cent ball-milled, and long-fiber asbestos dust.

11. Inhalation Experiment with "King's Floats" Asbestos Dust

The first inhalation experiment conducted at the Saranac Laboratory with asbestos dust was begun in 1928. Animals inhaled the dust for periods up to nearly three years and some guinea pigs lived for about four years after their first exposure to dust. A preliminary report giving observations after 29 months of exposure appeared in the February, 1931 issue of THE JOURNAL OF INDUSTRIAL HYGIENE.* At that time observations covered a period of only 2-1/4 years and the conclusions as to the ultimate effects of inhaled asbestos dust were provisional. Results of the completed study show that most of the conclusions drawn in the preliminary report were substantiated. A complete review of this experiment follows,

12. The dusting material was a commercial variety of asbestos dust known as King's floats and was composed of short fibers and particles of variable size. It was obtained from the Thetford, Quebec plant of the Asbestos Corporation of America.

13. The dust composition (Table 2) reveals that the amount of fibrous chrysotile was only 14 per cent, a rather low value. However, there was sufficient fibrous material to produce a characteristic fibrosis.

14. The dust concentration at first was quite low and for impinger samples taken soon after the experiment was started, the average light field count by the standard technique was only 6.0 million particles per cubic foot of air. An appreciable number of large particles (or fibers) also were present, as

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*STUDIES ON EXPERIMENTAL PNEUMOCONIOSIS. VI. Inhalation of Asbestos Dust., Gardner, L.U., and Cummings, D.E. J. Ind. Hyg., 13: 65-81, 97-114, 1931.

shown by an average count of 0.8 million for particles greater than 10 microns. After the inhalation experiment had been under way for about two years, the speed of the rotating paddle in the dusting machine was increased and for the remaining 9 or 10 months of the experiment considerably more dust was dispersed into the atmosphere. Average dust counts for impinger samples collected after this change were 53.7 million for the usual light-field method, and 1.6 million for particles larger than 10 microns.

15. Reaction in Animals to Inhaled "King's Floats" Asbestos Dust. Results of the investigation, briefly summarized in Table 3, show that inhalation of King's floats asbestos dust produced a typical peribronchiolar fibrosis in guinea pigs but not in rabbits or rats.

16. Guinea Pigs. Seven groups of guinea pigs were used. In three groups the effect of a continuous and of an interrupted dust exposure was studied; in two other groups the relationship between infection and dust exposure was investigated. The remaining two groups were infection controls.

17. Rate and Type of Reaction. Guinea pigs inhaling this dust for periods up to 33 months developed a characteristic fibrosis occurring in conical patches about the respiratory bronchioles. During this exposure the peripheral alveoli were not involved. The particulate elements in the dust were transported to the lymphatic system where they caused no significant reaction; the fibrous elements remained fixed at the site of original localization and were seldom detected in the lymphoid tissue. Pleurisy and fibrosis in the septa were observed only when infection complicated the process.

After exposure of approximately a year, a small amount of cellular reaction had been produced about many respiratory bronchioles. As more dust was inhaled, it continued to accumulate in the same location and later stages of the disease

consisted of extensions of the original lesions. New areas were not involved.

Apparently, the inhaled fibers were caught in the pocket-like alveoli that are given off from the lateral walls of the respiratory bronchioles. There they were phagocytized and many of them were carried into the wall by migratory cells. Mononuclear leucocytes attracted to the area caused an appreciable thickening of the bronchial wall. After 15 months a delicate fibrosis made its appearance. The process evolved so gradually that mitotic division of fibroblasts could rarely be discovered; nevertheless, the number of fine intercellular collagenous fibers (fibrosis) steadily increased. As this fibrosis contracted, it partially closed the alveoli, and with this atelectasis the lining epithelium assumed its embryonic cuboidal form. The result was the adenoma-like appearance that Willis described in guinea pigs inhaling silicon carbide. The longer exposures resulted only in more thickening of the walls of the air spaces, largely due to an increase in the amount of fibrosis. The fibrous tissue always remained cellular and never showed the hyalinization characteristic of silicosis.

18. Progression. The reaction produced in exposed guinea pigs did not progress significantly during a subsequent period of 37 months when the animals lived in a normal atmosphere. Between 8 and 11 months after exposure ceased, the cellular reaction had been completely replaced by thin strands of fibrous tissue. Observed still longer, the scar tissue decreased in amount but in the last animal sacrificed, 37 months after discontinuing dust exposure, some fibrosis was still visible.

19. Infection Coincident with Dust Inhalation. Of the group of 40 guinea pigs infected with attenuated tubercle bacilli (R_1 strain) 31 died or were sacrificed before two years of dust exposure and were reported in the paper by Gardner and Cummings mentioned

above. Seventeen of these died from intercurrent pneumonia. Briefly, the results were as follows: 10 revealed some evidence of spread of the tuberculous process; in 6 of these it was confined to the lungs and in the other 4 the abdominal viscera also were involved. Usually a slight local extension of the tuberculous infection had occurred but subsequent healing had resulted in fibrosis of both the pulmonary lesions and the secondary lesions in other organs. The healed pulmonary lesions showed more fibrosis than is characteristic of either tuberculosis or asbestosis alone.

The 9 animals which were still alive after two years of dust exposure were sacrificed at intervals during the following year. In 4 of them the primary foci of infection had healed with fibrosis and even calcification and there was no evidence of progression. In the other 5 the tuberculous foci showed evidence of having previously spread locally: in 4 of them it had healed, by the time of autopsy, with excessive fibrosis; in the other animal there was a generalized chronic tuberculous pneumonia in one lobe and isolated primary tubercles, which were still active but had not spread in the other lobes.

Evidence of extension of the infection was first seen after 7 months of dust inhalation; during the next 20 months more than half of the animals showed an actively spreading tuberculosis and in 3 of them small cavities had developed. During the last 8 months no animals exhibited any evidence of active infection although in half of them the healed fibrous scars of previous extensions were obvious. Sixty per cent of the guinea pigs with spreading pulmonary tuberculosis showed tuberculosis of the spleen and liver.

20. Infection Superimposed Upon an Established Asbestosis. Twelve guinea pigs, after inhaling asbestos dust for nearly 26 months, were infected with tubercle bacilli and then removed to normal air. The subpleural tubercles in the dusted animals were

no more numerous than in non-dusted controls, but a considerable number were found in the depths of the lung about foci of asbestosis. The reaction to infection showed only slight local extension about the original sites in the lungs and tracheobronchial lymph nodes. The abdominal viscera were involved in only one animal. Caseation was found in tubercles 1-1/2 months old but by 5-1/2 months it had completely disappeared, leaving only scar tissue. The latter still persisted in the last animal, which was killed 14 months after infection.

21. Asbestosis Bodies. Moderate numbers of asbestosis bodies occurred in the lungs of the guinea pigs, becoming more numerous and more distinctly segmented in later months.

22. Rabbits. Rabbits exposed to the asbestos dust for periods up to 19 months developed a low-grade foreign-body type of reaction but no fibrosis. Although their lungs contained particulate elements of the dust, fibers were not present, indicating that the upper respiratory mechanism of the rabbit is adequate to exclude fibrous foreign bodies. Two rabbits, after inhaling dust for 6 and 18 months, lived in normal air for more than two years. At autopsy neither animal showed any evidence of cellular reaction or fibrosis in the terminal bronchioles nor were there any asbestosis bodies.

23. Rats. All the white rats had acquired an infection, resulting in the formation of pulmonary abscesses, before they came to autopsy. Apparently, so much heavy mucus obstructed their bronchi that very few fibers could have entered their lungs. In a few of the rats, an occasional asbestosis body was discovered but there was no fibrosis.

24. Summary and Interpretation of Inhalation Experiment with King's Floats Dus

The findings in the experiment with King's floats dust can be summarized under three headings.

A. Effect of the inhaled dust on normal animals. The King's floats dust caused a characteristic peribronchiolar fibrosis in guinea pigs but not in rabbits or rats. The fibrosis did not progress after the dust exposure was discontinued and the guinea pigs transferred to normal air.

B. Effect of the inhaled dust on tuberculosis in guinea pigs. In guinea pigs infected with attenuated tubercle bacilli and then placed in the dust room, the results were more variable than is usual in an experiment of this type. A few animals showed no sign of progression; in most of them there was evidence of temporary progression with subsequent healing; in one animal there was continuous progression to death. In contrast, when guinea pigs, after being infected, are exposed to quartz instead of asbestos dust, the infectious process continues to progress and eventually causes the death of the animals. On the other hand, exposure of infected animals to a harmless dust like calcite or gypsum does not lead to any progression of the infection. Guinea pigs infected with attenuated tubercle bacilli following the termination of about two years' exposure to asbestos dust did not develop progressive disease. The only modification of the infection was in its localization, a few bacilli being retained in the fibrous terminal bronchioles and forming tubercles there in addition to the usual foci beneath the pleura.

In view of this variability, the unusual nature of the response and the high proportion of deaths from intercurrent pneumonia it is felt that definite conclusions as to the influence of this dust on the course of tuberculous infection are not justified.

C. Effect of tuberculous infection on the reaction to inhaled dust.

Infection complicating asbestosis accentuated the tendency of the dust to produce fibrous tissue. This change occurred whether or not the bacterial lesion was in contact with the area of dust reaction.

25. Inhalation Experiment with Short-fiber Asbestos Dust.

Since hazardous dusts like quartz are most effective in producing fibrosis when the particles are 3 microns and less in size, an inhalation experiment was carried on to determine whether this condition is true also for asbestos dust. It was thought that by using a short-fiber asbestos dust consisting almost entirely of fibers and particles smaller than 3 microns an accelerated tissue response might be initiated and an advanced reaction obtained in a short time. The previous inhalation experiment with King's floats asbestos, which contained fibers from 1 cm. to 1 micron or less in length as well as a great deal of particulate matter and which produced a typical peribronchiolar fibrosis in exposed guinea pigs, served as a basis of comparison.

26. The dusting material for this experiment was forwarded from the Manville plant of the Johns-Manville Corporation. It was the remains of fibers collected in dust bins after a carding operation and screened to pass 200 mesh. Since the material as received contained many long fibers, it was ground in a steel ball mill to reduce practically all the particles to 3 microns or less in size. When used alone in a standard dusting machine, this finely-ground asbestos tended to pack in the hopper and it became necessary to mix one volume of the unground material with three volumes of the ground to generate a satisfactory dust cloud. The addition of the small quantity of unground asbestos was unfortunate because it confused the interpretation of results. Probably the minor amount of reaction that developed was due to the long fibers in the mixture although the

data of this experiment do not prove the point.

27. The composition of the short-fiber asbestos as received is disclosed by the chemical and petrographic analyses given in Table 4. Samples taken before and after grinding yielded about the same values on analysis, indicating that there was no contamination from the mill or loss of water content.

28. The dust concentration varied somewhat during the experiment and light field counts for atmospheric samples collected inside the animal cages with the impinger apparatus ranged from 83 million to 182 million. The average of counts was 130 million for the first year of the experiment, 124 million for the second year and 140 million for the third year.

29. Size-frequency measurements of air-floated dust from inside the cages at a magnification of 1300X revealed a great preponderance of fine particles (Table 5). Nearly 90 per cent of the particles seen were smaller than 3 microns.

30. Reaction in Animals to Inhaled Short-Fiber Asbestos Dust. Four species of animals - guinea pigs, white rats, cats and rabbits - were used in this experiment. The results of the dust exposure, which are summarized in Table 6, will be considered more in detail below.

31. Guinea Pigs. Eighty guinea pigs were originally placed in the dust room but 21 of them were later eliminated from the experiment and killed because of enlarged lymph nodes. Of the other 59 animals, 46 remained in the dust room until they were sacrificed or died from natural causes and 13, after being exposed to dust for 20 months, were transferred to a normal atmosphere.

32. Rate and Type of Reaction. The type of tissue reaction to the inhaled

short-fiber asbestos was essentially the same as that already observed in the experiment with King's floats asbestos. The rate of reaction also was approximately the same but the extent of involvement with the short-fiber dust was very much less and after 16 to 24 months of exposure only a very few small foci of reaction, which generally required microscopic examination for detection, were produced in the guinea pigs.

Until exposures had continued for approximately one year, there was little tendency for dust-containing phagocytes to collect into clumps. By 16 months phagocytes had begun to collect about the walls of a few of the respiratory bronchioles with a little proliferation or infiltration of mononuclear cells in these walls. There were also some multinucleated cells but they were always of the inert foreign-body type. At 20 to 24 months the cellular clumps were sometimes quite marked and sometimes changes in the epithelium resulted in the adenoma-like or "adenomatoid" appearance previously described in Section 17. In most of the subsequent members of the series, the reaction remained cellular in type. In a few, however, fibrous elements dominated the picture. In the latter case, the collagen was pale in color and tenuous with no heavy swollen hyalinization. As in the rats described below, the alveolar walls might be made up of a band of collagen supporting a layer of epithelium, but with no contained capillaries. In the tracheobronchial lymph nodes the reaction was more pronounced in this experiment than in the previous one with King's floats asbestos, probably because of the transportation of an excess of fine particles to the nodes in animals inhaling short-fiber asbestos. The reaction was essentially an increase in reticulum, rather than a fibrosis, with preservation of the original cells between the thickened reticular fibers. Diffuse chronic pleurisy without evidence of pulmonary infection was present in a few animals.

33. Progression. In the 13-3/4 months following the cessation of 20 months' exposure to dust, progression of disease was not definitely demonstrated but neither could it be absolutely disproved, owing to the variability of the response in different animals. At the end of the dust exposure of 20 months two pigs were read as + and one as 2+. Among the 13 removed from dust the findings were variable: in 2 the reaction was ±; in 4 it was +; in 3 it was 3+; in 3 it was 4+; and in one animal it was 5+. It is quite possible that those with the most marked changes had already developed more reaction than the remainder by the time exposure ceased. Since the more severe reactions occurred sporadically and bore no relationship to the length of time after cessation of exposure the differences were attributed to variation in individual susceptibility. This view received support from the chemical analyses (Table 7), which often revealed comparable amounts of ash and silica in lungs with widely different amounts of tissue change. For example, the ash and silica values were quite similar for three animals in dust 20 months and then in normal air 13-3/4 months, yet the tissue reaction for one animal was 4+; for another, +; and for the third, only ±.

34. Asbestosis Bodies. The formation of asbestosis bodies was at first extremely limited. After 5 months' exposure only a very rare short body could be found, usually inside of cells. Around the finest intracellular particles there were yellow deposits having the same color as the asbestosis body. Exposure of one year had permitted an accumulation of many longer fibers about which the asbestosis-body coating developed. Most of these were still short enough to be partially or entirely within phagocytic cells. By the 20th month and thereafter, they were comparatively numerous although still rare in comparison with the findings in the King's Floats experiment.

35. White Rats. Seventy-three white rats were exposed to atmospheric short-fiber asbestos dust for periods up to 32 months. Sacrificings during the first 10 months were made bimonthly and for the remainder of the experiment at less frequent intervals.

36. Rate and Type of Reaction. The dust cells until 8 months were widely scattered and existed in foci only sporadically. Reaction was limited to occasional slight thickenings of the septa about small accumulations of dust cells. In a few rats at 10 months, there was a suggestion of early fibrosis but the change was so slight that it would probably be overlooked without the clump of dust cells to attract attention to the area. Only 10 animals were exposed from 12 to 32 months. In each of them the lungs showed minute patches of well-defined fibrosis distributed like that of asbestosis but without asbestosis bodies. The lesions, visible only at a magnification of 150 diameters or more, consisted of patches along alveolar ducts in which the walls of the air spaces were very thick, due to swollen collagen framework. Connective tissue and Foot-Bielschowski silver preparations revealed complete loss of capillary bed locally. Outside the collagen was a thin layer of epithelial cells. This did not resemble the "adenomatoid" change characteristic of guinea pig asbestosis. No pleurisy was present. Near the lesions the air spaces were filled with phagocytes containing gray to yellow particulate dust and a rare long naked asbestos fiber. Careful search failed to reveal even a suggestion of an asbestosis body. The tracheobronchial nodes showed compact focal collections of monocytic cells at 12 months and, at 20 months, some diffuse thickening of the reticulum. In a few rats there was definite fibrosis along the margins of the node and extending into the mediastinal areolar tissue. Compared with the response to active dusts like quartz and chert the reaction to short-fiber asbestos was negligible.

Results of chemical analyses made on the white rats are given in Table 8 and the average values have been tabulated in Table 9 for comparison with similar values for rats inhaling other dusts. The concentration of atmospheric particles to which the animals were exposed was approximately the same for asbestos and quartz; for the gypsum-quartz mixture, it was about twice as high and for chert five times as high. It will be noted that the percentages for asbestos are lower than those for quartz or chert but are similar to those for the gypsum-quartz mixture, in which atmospheric agglutination tended to reduce the amount of dust inhaled. It might be inferred that the total quantity of asbestos dust inhaled was low or that it had been eliminated from or dissolved within the lungs. In the present state of our knowledge evaluation of these hypotheses is not possible.

37. Cats. Twenty cats were used in this inhalation experiment with the short-fiber asbestos. Eighteen were kept in the dust room until death, the exposure period ranging from one month to nearly 4-1/2 years, and two, after a dust exposure of 31-1/2 months, were removed to normal air. One of these was sacrificed 5 months, and the other 24 months, later.

38. Rate and Type of Reaction. The reaction was essentially that to an inert dust, even after more than 4 years of exposure. The tissue response in this species was confined to microscopic foci of fibrosis in the walls of groups of subpleural alveoli, rather than in the peribronchiolar areas. In one animal the change was extensive enough to be visualized on gross inspection of the section.

39. X-Ray Changes. Only in the animal with the longest exposure did the X-ray reveal definitely abnormal shadows. After 29-3/4 months the picture was negative; after 45 months a faint mottling could be detected throughout both lungs. At autopsy, 8 months later, there was only microscopic

fibrosis in the subpleural zone plus heavy lymphocytic infiltration about small bronchioles.

40. Asbestosis Bodies. On prolonged search a few yellow atypical asbestosis bodies, smooth and without haustrations, were found in two animals exposed for more than a year.

41. Rabbits. Eight rabbits were exposed to dust for periods extending from one to more than five years. The last animal was removed from the dust room and left in normal air 6 months before being sacrificed.

42. Rate and Type of Reaction. There was never enough fibrosis to be detected grossly and there was no chronic adhesive pleurisy. Microscopic evidence of alveolar wall thickening was first detected after about 3 years of exposure and was seen in all five animals examined thereafter. In one animal that died of paralysis after nearly four years of exposure the reaction was extensive enough to be visible on gross inspection of tissue sections. The possibility of pulmonary infection in this animal could not be excluded. However, in another animal dying two years later the focal fibrosis was not nearly as obvious or as advanced. Areas of involvement, which were largely visualized because of phagocytic reaction within the air spaces, tended microscopically to become more fibrous with the passage of time but there was never much encroachment upon the lumen of air spaces and the architecture of the lung was preserved.

43. Asbestosis Bodies. Asbestos bodies were not detected in rabbits that died early in the experiment but were seen in all animals that had been exposed to the dust for more than three years.

44. Summary and Interpretation.

The original purpose of the experiment was to evaluate the chemical theory of the pathogenesis of asbestosis. It was felt that if the tissue reaction to asbestos were chemical in origin an accelerated or accentuated response would result from exposure to finely-divided asbestos, as is the case with quartz. This experiment, in which the reaction was slower and less extensive than with King's floats, indicates that the reaction probably is not primarily chemical in nature.

Of the four species exposed in this experiment only the guinea pig and rat reacted with characteristic peribronchiolar fibrosis. The cat reacted with atypical sub-pleural fibrosis and in the rabbit the fibrosis which occurred could not be positively attributed to the dust because of a strong possibility of pulmonary infection.

45. Inhalation Experiment with 100 Per Cent Ball-Milled Asbestos Dust.

In the inhalation experiment with short-fiber asbestos dust a small quantity of unground asbestos was mixed with ground material in order to produce a suitable dust cloud. When evidence of a dust reaction appeared in the guinea pigs during the experiment, it was not clear whether this was a tissue response to the small number of long fibers in the unground asbestos or was a delayed effect of the more abundant fine dust. Consequently, another inhalation experiment was started in which no unground material was used.

46. The dusting material was the ground short-fiber asbestos used in the previous inhalation experiment but no unground material was mixed with it. To obtain a sufficient amount of atmospheric dust, the design of the dusting apparatus was changed to an open type of hopper and fresh dust was added daily.

Owing to the tendency of the material to form small spherules which prevented much of the fibrous portion from floating out of the hopper, the dispersal of the dust was not entirely satisfactory and after 7 months of operation, the dusting machine was reconverted to its original design. To prevent "piling" or the formation of spherules of asbestos, steel wire brushes were attached to the inside surface of the hopper and to the rotating paddle. This arrangement gave satisfactory results and was used for the remaining 17 months of the experiment.

47. The composition of the raw material (short-fiber asbestos) and of atmospheric dust liberated from the ball-milled product in the dusting machine is given in Table 10. These values are based upon petrographic study and X-ray diffraction analysis. The atmospheric sample was collected with an electrostatic precipitator after wire brushes had been installed in the dusting machine. Previous to this, the chrysotile content of the air-suspended material was undoubtedly less than the 15 per cent value given in Table 10. In an interim report, it was stated that the air-borne dust contained about 5 per cent of chrysotile before the wire brushes were used and up to 8 per cent afterwards, but these values were probably low. Quantitative estimates on ball-milled asbestos dust can be somewhat inaccurate because it is difficult to determine how much of a dust sample is fibrous chrysotile and how much is non-fibrous serpentine.

48. The dust concentration for the first 7 months of the experiment was about 100 million particles per cubic foot of air. After the wire brushes had been installed, the dust counts were a little higher and the overall average for the first year was 106 million. The average of counts for the second year was 163 million and for the third year 145 million.

49. The size-frequency of the components of atmospheric dust collected inside the animal cages with the electrostatic apparatus is reported in Table 11. Two samples were taken, one before the wire brushes were installed and one after. It will be noted that after the wire brushes were in use a greater proportion of very fine particles and also of longer fibers was released into the air.

50. Reaction in Animals to Inhaled 100 Per Cent Ball-Milled Asbestos Dust. Guinea pigs, rats and mice were used in the inhalation experiment with the 100 per cent ball-milled asbestos dust. The results are summarized in Table 12.

51. Guinea Pigs. The experiment was started with 100 guinea pigs. As the dust exposure proceeded, there were 39 accidental deaths, 32 of pneumonia in an epidemic. After 28 months of dusting the 16 surviving guinea pigs were transferred to normal air.

52. Rate and Type of Reaction. For the first year of exposure practically the only reaction to the dust was the presence of scattered phagocytes and an occasional minute asbestosis body. At 16 and 20 months no gross response was visible on the tissue section but microscopically peribronchiolar foci of inflammatory cells could be seen. At 24 months there was still no change large enough to be seen with a hand lens although microscopic examination revealed cellular accumulations about terminal bronchioles and many more asbestosis bodies, chiefly within cells.

Chemical analyses of the lungs (Table 13) reveal that in spite of the limited tissue reaction considerable dust had been retained in the lung.

53. Progression. The lungs of animals exposed for the full dusting period (28 months) and then living in normal air for 2 months revealed the changes described above and also very slight peribronchiolar fibrosis. After 8 months in normal air the findings were similar but at 12 months 3 of 4 animals showed grossly-visible characteristic peribronchiolar fibrosis with adenomatoid change.

54. Lymph Node Involvement. The tracheobronchial nodes were essentially negative until exposure had been continued for more than a year and a half. Animals sacrificed at 12 months and 16 months revealed a few minute collections of phagocytes containing particles but practically no fibers large enough to be recognized as such. After 20 months of exposure many monocytes filled with yellow granules were present. At 30 months there had been a slight increase in reticulum but no fibrosis. No further changes occurred in the nodes. Asbestosis bodies were not seen in the nodes of any of the guinea pigs.

55. Asbestosis Bodies. Minute asbestosis bodies were observed as early as 3 months after exposure began, but they did not become numerous until 16 months had elapsed. The bodies were short and practically all were intracellular, although at 20 months some were long enough to project beyond the cell borders.

It is important to note that in the later months of exposure there was a distinct increase in the number of long fibers (up to 70 microns in length) in the lungs and that after exposure ceased characteristic long asbestosis bodies were seen.

56. White Rats and Mice. In this experiment 40 rats were exposed for periods up to 20 months and 24 mice for periods up to 12

months. Neither species developed even a suggestion of asbestosis and reaction was limited to phagocytosis of inhaled particles by widely-scattered dust cells which remained free in air spaces or were transported to the tracheobronchial lymph nodes. No asbestosis bodies were found in the rats but in the mice there were a very few small non-haustrated forms within phagocytes.

In 21 mouse lungs sectioned there were 3 instances of pulmonary adenoma (14%).

57. Summary and Interpretation.

The tissue reactions observed in this experiment were much less extensive and slower in development than in the previous investigation with short-fiber asbestos. Since presumably there were fewer fibers longer than 3 microns in the material used in this experiment, the results tend to confirm the interpretation made in Section 44 of the short-fiber experiment that the reaction probably is not primarily chemical in nature.

The finding of long asbestosis bodies in animals inhaling the ball-milled material is an example of the difficulty of completely eliminating long fibers from an asbestos preparation.

In regard to progression of reaction after removal from dust, which was observed in this experiment but not in the others, the following interpretation is offered: When the reaction is well-developed at the termination of exposure, the contraction of the fibrous tissue would obscure any possible progression. In this experiment, however, since only the earliest stage of reaction was present at the time of removal from dust, its subsequent progress was apparent. It should be noted that the degree of progression was so slight that it can have little, if any, practical significance.

59. Inhalation Experiment with Long-fiber Asbestos Dust.

After animals inhaling short-fiber asbestos dust for more than a year had failed to develop significant reaction, the hypothesis that asbestosis is produced by the mechanical irritation of long fibers was given added support. Since the King's Floats asbestos used in the first inhalation experiment had a rather low content of fibrous chrysotile and contained considerable serpentine and other impurities, it was decided to conduct a new inhalation experiment with a purer form of chrysotile which would be richer in long fibers.

58. The dusting material employed in this investigation was obtained from the Manville plant of the Johns-Manville Corporation. Samples of several varieties of asbestos dust were first submitted to the Saranac Laboratory for examination and one kind, identified as lot D, which was low in magnetite and chromite and had a fibrous content estimated to be about 75 per cent, was selected as most suitable. Steel wire brushes were fastened to the inside surface of the hopper and to the rotating paddle in order to open up the bundles of asbestos and liberate more fibers into the atmosphere.

60. The composition of the long-fiber asbestos used in this experiment is indicated by the chemical and petrographic analyses given in Table 14. It appears that this material was a much purer form of asbestos than the short-fiber dust used in other experiments. This is borne out by comparing the approximate analyses of the long-fiber and short-fiber dust in Table 15.

61. The dust concentration as revealed by impinger samples taken inside the animal cages was much lower than the concentration for the experiments with short-fiber or ball-milled dust. For the first year of the experiment with long-fiber asbestos the average of the light field counts was 32 million; for

the second year, 48 million; for the third year, 39 million; and for the fourth year, 43 million. Examination of the impinger samples with dark field illumination disclosed that many fine particles less than one micron in size accompanied the larger particles and dark field counts were, on the average, about 5 or 6 times larger than the light field counts.

62. The size-frequency of atmospheric samples of the long-fiber asbestos dust and of the ball-milled dust is shown in Table 16. Both samples were collected with the electrostatic precipitator. It will be noted that there was far more fibrous material in the long-fiber dust.

63. Reaction in Animals to Inhaled Long-Fiber Asbestos Dust. Guinea pigs, cats, rats and mice were employed in the inhalation experiment with long-fiber asbestos. Results of the experiment, summarized in Table 17, are described in greater detail below.

64. Guinea Pigs. The experiment was started with 100 guinea pigs. After exposure had been carried on for a year, a severe epidemic of pneumonia arose in the dust room and about one-third of the animals died or were killed. To replace them, 38 more guinea pigs were added to the surviving group in the dust room.

65. Rate and Type of Reaction. Histological examination revealed grossly visible lesions in the lungs after 8 months of exposure to dust, consisting of cellular infiltration about the terminal bronchioles. At 12 months, there were adenomatoid changes in the air spaces and by the 16th month a definite fibrosis was present in these areas in half the animals. The fibrous lesion could be seen macroscopically at 20 months. From this time on the reaction increased in extent and in the amount of collagen and

by the 24th month, it had fanned out into the parenchyma. The lesions were rather sharply localized and the extensions from different bronchioles showed no tendency to fuse, even in animals exposed for the maximum period (3 years). Although the intra-pulmonary reaction sometimes reached the pleura, there was no involvement of that membrane. No emphysema was visible at any point. Some thickening of the larger bronchi with a chronic inflammatory infiltration was revealed, but it probably was no more than would be produced by a similar exposure to any dust. For the first 5 months the phagocytes consisted of monocytes or very small giant cells; later, giant cell formation was more prominent. After 16 months the giant cells were large, filled with yellowish-brown pigment and sometimes vacuolated. An occasional animal showed an admixture of polymorphonuclear leukocytes and, in guinea pigs exposed for a considerable period, eosinophiles. The reaction was at first entirely cellular but by 16 months fibrous tissue formation was definite. However, it never attained a stage of hyalinization suggestive of silicosis.

A moderate individual variation occurred among the exposed animals, both in the rate of developing lesions and in the stage of development attained at the end of exposure.

Analyses of the lungs (Table 18) disclosed that although the tissue response was much greater in these guinea pigs than in those exposed to either short-fiber or ball-milled asbestos, the amount of mineral matter in the lung ash was less.

66. Progression. In guinea pigs exposed to the dust for 20 months and then removed to normal air, there was a marked tendency for cellular inflammatory reaction to clear. This effect, accompanied by contraction of the fibrous tissue, resulted in a diminishing size of the focal lesions. None

of these animals, killed at various periods up to 14 months after exposure, revealed lesions as large as those in the group sacrificed at the end of the 20-month exposure period or those in animals which remained in the dust room for more than 20 months. Fourteen months after dust exposure ceased, the foci in four of the six remaining guinea pigs were so small that they were visible only with a hand lens.

Reaction in the group exposed for 27 months and then transferred to a normal atmosphere was quite similar to the response in the 20-month exposure animals mentioned above. However, small foci were always visible on gross inspection of sections of all guinea pigs of the 27-month series but in no instance was there evidence of extension of the reaction.

67. Lymph Node Involvement. Reaction in the tracheobronchial lymph nodes was first visible at the third month of exposure. At the 8th month patches of cellular connective tissue began to appear in the medulla and by the 14th month most of the node had been replaced by cellular connective tissue. This picture, which resembled that in early silicosis, persisted to the end of the experiment. Some animals, as a variant, showed heavy sheets of diffuse monocytes and large active giant cells but there was never any necrosis or hyaline formation. The spindle-shaped new cells were yellowish in color from fine pigment granules that stained for iron. No fibers nor asbestosis bodies were seen.

68. Asbestosis Bodies. Although asbestosis bodies were seen as early as one month after exposure began, they were rare and hard to find. At 5 months more were visible, chiefly coiled inside giant cells, and at 8 months many bodies were free in connective tissue. They became fairly abundant as exposure progressed although in some later animals the asbestosis bodies were only moderately numerous.

69. Cats. Four cats inhaled the long-fiber asbestos dust for periods of 14, 25, 33 and 42 months, respectively, and were immediately sacrificed. Two other cats, after being exposed to dust for 18 months, lived in a normal atmosphere for an additional 24 months.

70. Rate and Type of Reaction. Exposure for 14 months was sufficient to produce cellular accumulations of phagocytes around terminal bronchioles and peripheral arterioles together with compact collections of similar cells in the tracheobronchial lymph nodes. At that time there were no typical asbestosis bodies, but smooth pointed yellow fibers were seen very rarely. With continued exposure, up to 42 months, reaction in the locations noted progressed to the formation of cellular connective tissue which made well-defined sheaths about the respiratory bronchioles and arterioles, marked lymphoid hyperplasia and lymphoid infiltration of bronchiolar walls. The bronchiolar epithelium was low and flattened, giving the tubes a smooth contour. Typical asbestosis bodies were not formed although there was an occasional yellow, smooth, pointed fiber. No pleurisy was present. The reaction was similar in location to that in the guinea pigs, but fibrosis was much slower in development and had not reached the same degree of maturity.

71. X-ray Changes. Roentgenograms of three cats were made after exposure periods of 25, 33 and 42 months, but tissue changes were not dense enough to be seen on an X-ray film.

72. Rats. Although 20 rats were placed in the dust room, many died from pneumonia and were not suitable for study. Five animals, of which one was exposed for 19 months and four for 25 months, were free from pulmonary infection and offer a basis for conclusions.

73. Rate and Type of Reaction. All four animals sacrificed at 25 months showed a well-marked peribronchiolar fibrosis. In the 19-month animal, reaction was just beginning. Asbestosis bodies were practically absent at both 19 and 25 months although two small smooth bodies were found in the 19-month animal after a long search. Thus, these animals exhibited fibrosis without asbestosis bodies.

74. Mice. Out of 20 white mice used in this experiment, 11 lived a year or more in dust and died or were killed without showing an appreciable degree of pulmonary infection.

75. Rate and Type of Reaction. Reaction was limited to phagocytosis by mononuclear cells. Usually these were widely scattered through the air spaces; a limited number were grouped about the terminal bronchioles producing some thickening of their walls. There was no suggestion of fibrosis. The striking feature of the experiment was that 9 out of the 11 mice (82 per cent) exposed to dust for a year or more showed pulmonary tumors, usually adenomatous in type. These lesions did not contain dust or asbestosis bodies.

Numerous asbestosis bodies were observed in animals killed late in the experiment. Thus, these animals exhibited asbestosis bodies without fibrosis.

76. Summary and Interpretation.

The purpose of this experiment was to evaluate the importance of long fibers in the tissue response to inhaled asbestos. The results indicate strongly that long fibers are chiefly responsible for the reaction. Thus, in guinea pigs reaction developed earlier and became more extensive than in previous experiments, in spite of a smaller concentration of atmospheric dust and a lower mineral content in the lungs. Furthermore, a typical peribronchiolar fibrosis was produced

in cats although in a previous experiment with short-fiber dust it did not develop in this species.

The cause of the cellular fibrosis in the lymph nodes of the guinea pigs is not clear. It did not occur in other inhalation experiments with asbestos.

LXXVII. INJECTION EXPERIMENTS

In order to determine to what extent the various fibrous minerals possess the capacity to produce tissue damage, numerous injection experiments were performed. In these experiments guinea pigs and rabbits were used and the mineral dust was injected by the intratracheal, intraperitoneal and intravenous techniques. For the purpose of simplification the findings in each series of tests have been condensed and reported in tables, to which reference will be made later.

78. Experiments Using Intratracheal Technique.

Since the asbestos minerals do not cause a typical advanced fibrosis in extra-pulmonary tissue, the intratracheal technique is the perferred way of introducing fibrous dust into the experimental animal. In this method the dust suspension is injected by means of a special needle or catheter deep into the trachea, from which it flows into the lungs.

79. Comparison of Fibrous and Non-Fibrous Dusts. To demonstrate that the ability of asbestos to produce fibrosis resides in its fibrous character, the series of injection experiments reported in Table 19 were performed. The tests were made with unheated long-fiber chrysotile and with chrysotile that had been ignited to destroy its

flexible structure or ball-milled to reduce the length of fiber to 5 microns and less. At the same time control tests were made with serpentine, which has the same chemical composition as chrysotile but is non-fibrous. A review of the findings reveals that only the unheated long-fiber chrysotile produced fibrosis. Fibers subjected to ignition or shortened by ball-milling had lost their capacity to cause serious tissue damage. Ignition produced important changes in the chrysotile fibers, among them being loss of water, an alteration from a flexible to a brittle structure and possibly other changes.

80. Comparison of Various Long-Fiber Dusts. Some very interesting findings are disclosed by the results of the experiments included in Table 20. First, all the long-fiber asbestos minerals tested, with the exception of anthophyllite, produced a typical fibrosis. It is not entirely clear why anthophyllite behaved differently from the other asbestos minerals. Unfortunately, 5 of 8 animals died of pneumonia within the first two weeks of the experiment and the remaining animals were sacrificed at 1, 8 and 12 months; thus observations were not made at the optimum periods of 2 and 4 months.

Second, with the mineral brucite, which is not a silicate but is a fibrous form of magnesium hydroxide, a characteristic fibrosis like that of the asbestos minerals was obtained. Since the brucite used contained only 0.90 per cent silica (as an impurity), it is obvious that a siliceous component is not an essential factor in the development of asbestosis.

Third, no fibrosis resulted from the injection of glass wool fibers, even though glass wool resembles asbestos in some ways. There are fundamental differences, however. A glass wool fiber 3 microns in diameter is a solid rod and, in short lengths, is fairly rigid, while an asbestos fiber of the same diameter is a bundle of extremely fine filaments which impart to the fiber a high degree of flexibility. It would seem that this structure and the associated flexibility are

important factors governing the capacity of a mineral to produce peribronchiolar fibrosis.

81. Comparison of Long-Fiber and Short-Fiber Dusts. With quartz dust it has been demonstrated that the smaller the particles, the more intense is the tissue reaction, and that there is little reaction to particles larger than 3 microns in diameter. In the case of asbestos, however, the reverse is true and apparently only long fibers have any specific effect. This is confirmed by the data of Table 21, in which a series of tests with fibrous minerals is reported. When the injected dust consisted of fibers 20 to 50 microns long, all the minerals tested (except anthophyllite, as noted in Section 80) produced a fibrosis; when the material was prepared by first grinding the fibrous dust until the length of fibers was reduced to 20 microns and less (or, in some cases, 3 microns and less), none of the injected mineral dusts caused fibrosis.

82. Experiments Using Intravenous Technique.

The experiments, described in Table 22, in which the intravenous method of injection was employed, show that the asbestos minerals are far different from quartz in their action on tissue. It has been repeatedly demonstrated that intravenous injection of quartz particles 3 microns and less in diameter will cause a typical tissue reaction with the development of fibrosis in extrapulmonary sites, such as the liver and spleen. Asbestos minerals, however, on intravenous injection generally produce only an inert type of reaction, as is revealed by the results given in the table. The reason for the early deaths in the experiment with chrysotile particles is not clear; it may have been caused by silicic acid liberated by the finely-ground mineral.

83. Experiments Using Intraperitoneal Technique.

The results of injection experiments with the intraperitoneal technique are given in Table 23. It will be noted that the long-fiber dusts produced a fibrous reaction while dusts composed of particles 3 microns and less in size caused only an inert type of response. These experiments indicate also that the fibrosis initiated by the irritation of asbestos fibers is not restricted to the lungs, as was formerly assumed, but can be produced in the peritoneum as well.

LXXXIV. OTHER EXPERIMENTS WITH ASBESTOS MINERALS

A number of additional experiments were conducted to throw more light on specific phases of the asbestosis problem.

85. Protective Action of Aluminum Compounds.

Intratracheal injection of a suspension of long-fiber chrysotile to which colloidal aluminum hydroxide had been added revealed that the addition of the aluminum compound did not prevent the tissue irritation produced by chrysotile. If anything, the acute inflammatory response to the injected fibrous mineral was accelerated. One month after the last injection of the dust suspension the bronchiolitis was becoming fibrous.

86. Formation of Asbestosis Bodies.

The iron in the coating of the asbestosis body appears to be derived from blood or tissue elements and not, as has been suggested, from the mineral fiber. Following subcutaneous injection of two kinds of chrysotile into the groin of guinea pigs - one kind containing 2 per cent and the other 0.2 per cent Fe_2O_3 - the asbestosis bodies were equally numerous at both sites of injection.

An attempt to produce asbestosis bodies in guinea pigs by implantation of three silk bags containing fibrous chrysotile was unsuccessful. One bag planted subcutaneously in the abdominal wall disappeared; the other two bags, placed in the peritoneal cavity, produced a little foreign body reaction but no asbestosis bodies in a year.

Intratracheal injection into guinea pigs of asbestosis bodies recovered from human lung tissue failed to produce the typical tissue reaction to asbestos fibers. The injected material was obtained by digesting with sodium hypochlorite solution lung tissue removed at autopsy from an asbestos worker. The asbestosis bodies could be seen in the guinea pigs for at least a year after injection. This experiment shows that the asbestosis body has a rather resistant coating which is not destroyed by moderate hypochlorite treatment and may be maintained in vivo for a year or longer.

LXXXVII. THEORY OF IRRITANT ACTION OF ASBESTOS MINERALS

Two hypotheses have been proposed to explain the tissue irritation and reaction caused by asbestos fibers: the chemical and the mechanical. In the chemical theory, which is based upon experience with quartz, it is assumed that the asbestos minerals dissolve in the body fluids and that in this process their bases are leached away to leave silica in a form capable of irritating tissues. According to this hypothesis, asbestosis would be merely an indirect silicosis. Several facts make the chemical theory untenable: (1) intratracheal injections of brucite fibers, which had a silica content of only 0.90 per cent, caused a typical fibrosis like that produced by the asbestos minerals; (2) free-silica particles increase in potency as the particle size becomes less, but asbestos fibers shorter than about 10 to 20 microns are relatively innocuous; (3) aluminum

hydroxide neutralizes the irritating effect of quartz but not of asbestos; (4) serpentine has the same chemical composition as long-fiber chrysotile but it does not produce the same kind of tissue reaction; (5) there is a wide range in the chemical composition of the minerals which do cause asbestosis (see Table 24). In view of this evidence it seems more likely that asbestosis is caused by an unusual mechanical irritation from long asbestos fibers. Probably this irritation is related to the peculiar filamented structure of the fiber and the associated flexibility, which are possessed by no other foreign body. For example, ignition of chrysotile fibers changed their structure and made them inert while the same fibers, before being heated, would produce fibrosis (see Table 19). Further support for the theory of mechanical irritation is that asbestosis occurs in an organ of high mobility - the lung - and that a fibrous reaction can be produced by injection of asbestos fibers into the peritoneum, where there is also a degree of mobility, but not in other extrapulmonary organs.

LXXVIII. COMPLICATIONS

The experimental investigation with asbestos minerals was concerned primarily with the effect of the dust on normal tissue but some attention was given to other phases, such as susceptibility to infection and occurrence of malignancy.

89. Infection.

The only experiment in which the effect of inhaled asbestos dust on a pulmonary infection was studied was the first inhalation experiment, carried on with "King's floats" dust. It is, perhaps, unfortunate that infection studies were not made in the other inhalation experiments also.

90. Susceptibility to Tuberculous Infection. The development of a tuberculous process initiated at the beginning of exposure to asbestos dust, and also of an infection superimposed upon an established asbestosis, was described in Sections 19 and 20 of this report. It will be noted that asbestos, when classified according to the effect of a dust on tuberculous infection, would be placed below an active dust like quartz but above inert dusts, such as calcite and gypsum. In animals infected with attenuated tubercle bacilli, quartz will cause the infectious process to progress until the animal dies of tuberculosis. Inert dusts will have no effect on the infection and the lesions will usually heal and the disease disappear. Asbestos dust is in a different category. When the fibrous dust was being inhaled during the evolution of the infection, there was a spreading of the tuberculous process for a time but usually the stimulus for continued proliferation of the tubercle bacilli was not sustained, the progression was arrested and healing followed. In guinea pigs infected with attenuated tubercle bacilli following the completion of nearly three years of exposure to asbestos dust, progressive disease did not develop. The only modification of the infection was one of localization, a few bacilli being retained in the fibrous terminal bronchioles and forming tubercles there in addition to the usual foci beneath the pleura. Such tubercles healed in a few months and there was nothing to suggest any influence on the course of the disease.

91. Susceptibility to Non-Tuberculous Infection. There was no pointed experiment concerning the effect of inhaled asbestos dust on non-tuberculous infection. Intercurrent pneumonia among animals exposed to asbestos dust was rather common, the frequency in guinea pigs exposed in the four inhalation experiments ranging from 16 to 39 per

cent. This incidental evidence suggests the possibility of an effect of asbestos dust on non-tuberculous infection. Nevertheless, since such epidemics are not uncommon in inhalation experiments with other dusts and even in the colony of normal animals, it is felt that the inhalation of asbestos dust does not exert a significant effect on the susceptibility to non-tuberculous pulmonary infection.

92. Neoplasm.

No specific experiment was conducted to determine whether the inhalation of asbestos favors the development of neoplastic disease but certain observations on this subject were recorded in the outline of the proposed monograph on asbestosis submitted by the late Dr. L. U. Gardner in February 1943. In it he called attention to the high incidence of lung cancer among mice inhaling long-fiber asbestos. In his experimental notes, however, he referred to these lesions as adenomas.

There is an important distinction between adenoma and cancer which should be made clear. A cancer is a tumor, or neoplasm, capable of local invasion and destruction of tissue, which can distribute cells through the lymphatics or blood stream to produce isolated foci, from which new tumors develop. This phenomenon of dissemination is known as metastasis and any tumor which exhibits it is a malignant growth, of which cancer is one type. An adenoma, on the other hand, is a so-called benign or non-malignant tumor (neoplasm) which may or may not be capable of local invasion but which does not metastasize.

In order to clarify the exact nature of these lesions the pathological material is being carefully examined. Since it is felt desirable to have the benefit of Doctor Vorwald's judgment, a review of the data on this subject is being postponed until after his return from Europe. Rather than delay the entire report, further discussion will be reserved for a supplement to be issued later.

XCIII. CONCLUSIONS

Owing to the vast amount of data included in this report it seems most convenient to state the conclusions derived from the investigation and, when necessary, follow each one with a brief resume of the evidence.

A. Various forms of asbestos fibers produce a peribronchiolar fibrosis of the lungs of guinea pigs, rats, cats and rabbits but not of mice and dogs.

Both inhalation and injection experiments provide ample support for this conclusion. Figures 5 and 6 show the reaction to two different kinds of asbestos mineral.

B. The mode of action appears to be primarily mechanical rather than chemical in nature.

The evidence is given in section LXXXVII. Figures 1, 2, 3, 4 and 7 illustrate the important points. The fibrous filamented structure of asbestos appears to play an essential part in the irritating action, since the solid fibers of glass wool do not produce fibrosis (see Figure 8).

C. Short asbestos fibers do not produce fibrosis.

The conclusion is implied in the evidence mentioned in paragraph B above. Experiments which further support this finding are reported in Tables 21 and 23.

D. Typical fibrosis can be produced by an atmospheric suspension of asbestos dust containing only an extremely small proportion of long fibers.

In the inhalation experiment with 100 per cent ball-milled asbestos dust a typical, though delayed, fibrosis was

obtained (see Table 12), although less than 1 per cent of the atmospheric dust consisted of fibers longer than 10 microns, as is shown in Table 11. In contrast, the intratracheal injection experiment with fine asbestos dust containing no long fibers failed to produce fibrosis, (see Table 21).

E. Inhalation of asbestos dust apparently does not alter significantly the course of experimental tuberculosis in guinea pigs.

This conclusion is tentative since the evidence on which it is based does not conform with our usual experience. Reference to Table 3 will show that when infection was coincident with onset of dust exposure there was temporary progression of the disease with subsequent healing; when infection was initiated after 25 3/4 months of dust exposure the course of the tuberculous disease was not appreciably altered. In contrast, it has been observed in experiments with mixed dusts containing quartz that if there is a slight progression of the tuberculosis when infection and dust exposure are coincident, this effect is more marked (instead of less, as with asbestos) when infection is initiated after a period of dust exposure.

This conclusion concerning the effect of inhaled asbestos dust on tuberculosis seems justified because in the more sensitive test (infection initiated after a period of dust exposure) there was no appreciable increase in susceptibility to the tuberculous infection. However, since the findings in the asbestos study do not conform with previous experience, and

since the dusting material used in the single experiment dealing with infection contained only a relatively small amount of fibrous asbestos, it is recommended that a further investigation of this phase of asbestosis be considered.

7. The formation of asbestosis bodies seems to represent a coating of the fibers and results in loss of the ability to produce asbestosis. Intratracheal injection of asbestosis bodies alone does not produce the typical tissue reaction (see section 36). The cessation of progressive reaction to inhaled asbestos dust seen after exposure terminates may be due to the formation of asbestosis bodies.

PLAINTIFF'S EXHIBIT 326

March 3, 1949

American Brakeblok Div. of A.B.S. & F.
Celite Corporation
Keasbey & Mattison Company
Raybestos-Manhattan, Inc.
The Russell Mfg. Co.
Thermoid Company (Southern Asbestos)
Union Asbestos & Rubber Co.
United States Gypsum Co.

Saranac Laboratory
Asbestos Dust Experiments

We have now received from Saranac Laboratory a revised report (copy of which is enclosed) entitled "ASBESTOSIS PNEUMOCONIOSIS". I have examined this carefully and I believe it adopts the substance of all of the suggestions that were made by representatives of the underwriting group with respect to the report of September 30, 1948. You will recall that the earlier draft was reviewed at a meeting in New York on November 11, 1948. Minutes of this meeting, including a list of the recommended changes, were sent you by Mr. J. P. Woodard under date of November 30, 1948.

As the findings are generally favorable, we have renewed our request that Saranac Laboratory arrange for the publication of the report as promptly as possible, preferably in the Journal of Industrial Hygiene, and with an appropriate introduction by Dr. A. J. Lanza.

Very truly yours,

Vanderveer Brown,
Secretary and General
Attorney.

VBvy

Encl.

cc A. R. Fisher
L. C. Hart
J. P. Woodard

[redacted]

October 27, 1948

No. 1- American Brake Blok Div. of A.B.S.AF.
No.2- Carbo Corporation x
Keasbey & Mattison Company x
Raybestos-Manhattan, Inc.
No.5- The Russell Mfg. Co. x
No.6- Thermoid Company (Southern Asbestos) x
No.7- Union Asbestos & Rubber Co.
No.8- United States Gypsum Co. x

Saranac Laboratory
Asbestos Dust Experiments

With the request that you treat it with the utmost confidence and make it available to no one outside your organization, I am enclosing what purports to be "Part I" of a report by the Saranac Laboratory entitled "Asbestos Pneumoconiosis".

My own comments after a preliminary study of the report are contained in the attached memorandum, which will likewise refresh your recollection as to the origin of the experiments covered thereby.

Saranac will undoubtedly wish to publish the report either independently or in conjunction with the proposed report on "Human Asbestosis", and it would likewise appear desirable from the point of view of the industry that the report be published provided some of the speculative comments are omitted.

As a preliminary to a discussion with representatives of Saranac, a meeting of representatives of the companies which financed the experiments is indicated. I am therefore inviting the companies to which this memorandum is addressed to be represented at a luncheon in the Johns-Manville Corporation Board Room on Thursday, November 11, at 12:30 p.m. During and following the luncheon the report can be reviewed. If you are unable to have a representative attend, it would be desirable for you to designate some representative of another company to act for you in connection with decisions that will have to be made.

Please advise me as promptly as possible whether you will be able to attend and give me the name of your representative.

[redacted]

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In any event, please see that the draft of the report is returned to me on or before November 11 either by mail or by your representative. It is obviously undesirable that the report in its present form receive any distribution or publicity outside a limited number of people in our respective organizations.

Very truly yours,

Vandiver Brown,
Secretary and
General Attorney

VB:s
Enc.

bl cc A. R. Fisher
L. C. Hart
J. P. Woodard

C06784

(November 1948)

American Brakeblok Division of
American Brake Shoe Company
230 Park Avenue
New York 17, N. Y.

W. T. Kelly, Jr.
Executive V-P

Gatke Corporation
228 N. La Salle St.
Chicago 1, Ill.

Thomas L.Gatke
President

Keasbey & Mattison Company
Ambler, Pennsylvania

Ernest Muehleck
President

Raybestos-Manhattan, Inc.
Passaic, New Jersey

J. F.D. Rohrbach
Gen. V-P

The Russell Manufacturing Co.
Middletown, Conn.

T. M. Russell, Jr.
Vice-President

*Thermoid Company
Trenton, New Jersey

W. D. Perdue
Vice-President

Union Asbestos & Rubber Company
4th Ave. & E. 11th St.
Paterson, New Jersey

Edward Shuman

(Send copy to: Lewis J. Silverman, Executive V-P
Union Asbestos & Rubber Company
332 South Michigan Avenue
Chicago 4, Illinois

United States Gypsum Co.
300 West Adams Street
Chicago, Illinois

J. W. Butler

*Note: Southern Asbestos Company, Charlotte, N. C., now owned by Thermoid Company

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AMERICAN BRAKEBLOK

AMERICAN BRAKE SHOE COMPANY

November 8th, 1948.

From
230 PARK Avenue
New York 17, N.Y.

Mr. Vandiver Brown
Secretary and General Attorney
Johns-Manville Corporation
22 East 40th Street
New York 16, N.Y.

Dear Mr. Brown:

Thank you for your letter of October 27th, which has been reviewed by our Medical Department and while Dr. Hamlin would like very much to be in attendance at the meeting that you have scheduled for November 11th it is impossible for him to do so. We would, therefore, like to ask that you act for us in connection with any decisions that have to be made. We will, of course, be interested in receiving a copy of the minutes of the meeting recording any actions taken.

We would like very much to keep the report on permanent file in the Medical Department headquarters in Chicago and you may rest assured that the information contained therein will be treated as confidential.

I thought you would be interested in the remarks of our Medical Director, Dr. L. E. Hamlin, in connection with this report and I am therefore attaching a copy.

Very truly yours,

Executive Vice President
American Brakeblok Division.

WTK:EB
Enc.

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PLAINTIFF'S EXHIBIT 360 A

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AMERICAN BRAKE SHOE COMPANY

253

MEDICAL DEPARTMENT
2501 Blue Island Ave.
Chicago 8, Ill.

[redacted]

C
O
P
Y

November 3, 1948.

I have read carefully the report you forwarded and am returning it with my comments.

Mr. Brown's remarks on the "qualifying adjectives" in the abstract and conclusions.

While admitting the advisability of reconciling the two, it must be remembered that any animal experimentation cannot be absolutely conclusive. Observations on the tissue reactions to various substances can mean nothing more than reasonably accurate supportive evidence that the effect noted is what is likely to occur in man after similar exposure. The report notes that the same result is not always obtained in each species of experimental animal used. It is therefore only possible to employ such phraseology as, "the dust apparently does not alter significantly the course of experimental tuberculosis in guinea pigs," etc. Possibly a similar experiment carried over a longer period of time could produce an entirely different result. There are too many intangibles to allow dogmatic or more definite statements. This is true of any experimental work in animals or humans. The value of the experimental work lies in the fact that it forms a reasonable basis for study of characteristic tissue changes which are similar to those seen in humans.

I gain the impression from Mr. Brown's letter that he is concerned with possible repercussions from the legal point of view but I must confess I do not see anything in the report in its present form which need cause undue concern. Similar reports are frequent in the literature not only in this country but also from aboard.

Certain implications have been made in the report such as that referring to the marked incidence of pneumonia among the experimental animals and the suggestion that asbestos dust might have some degree of responsibility for such a development, but these are explained and discounted in the ensuing text. Perhaps these implications might be modified somewhat pending further factual data.

I feel that since most of the basic facts with the exception of the more detailed studies mentioned in the report are already known and have been published in other studies on asbestos, no unfavorable reaction need be anticipated. I think the idea of reviewing the manuscript prior to publication is a good one in order to achieve mutual understanding with Saranac, but I feel that this can be accomplished quite satisfactorily without my presence.

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Mr. Brown has stated that it would be advisable for us to designate a representative of another company to act for us in connection with the decisions to be made. I think this would be a satisfactory alternative and would like to suggest that we request him (Mr. Brown) to act for us. It would be most difficult for me to attend personally at this time because of the meetings I mentioned to you over the phone. I am sure our interest in the matter would be adequately protected by Mr. Brown.

/s/ L. E. Hamlin

Medical Director

PLAINTIFF'S EXHIBIT 360 D

AMERICAN BRAKE SHOE COMPANY
AMERICAN BRAKEBLOK DIVISION
230 PARK AVENUE
NEW YORK N.Y.

March 8th, 1960.

Mr. Vandiver Brown
Secretary & General Attorney
Johns-Manville Corporation
22 East 40th Street
New York 16, N. Y.

NOTED V. B.

Dear Mr. Brown:

This is to acknowledge receipt of and thank you for the revised report on Saranac Laboratory's Asbestos Dust Experiments, which I found on my desk when I returned this morning.

I am looking forward to the opportunity of reading it and will then pass it on to our Medical Department.

Very truly yours,

Executive Vice President
American Brakeblok Division

WTK:ls

November 12, 1948

Mr. W. T. Kelly, Jr.,
Executive Vice-President,
American BrakeBlok Division
of American Brake Shoe Company,
230 Park Avenue,
New York 17, N.Y.

Asbestos Dust Experiments

Dear Mr. Kelly:

The meeting to consider the Saranac report was held yesterday as scheduled, with all the interested companies represented except The Russell Manufacturing Co. I read to the meeting Dr. Marlin's memorandum of November 3rd and it was the consensus that his judgment was correct concerning the references to pneumonia among the experimental animals. Accordingly, we will not request that this be deleted but merely that it be modified somewhat with the view of placing more emphasis on the factors which made it doubtful whether the disease developed as a result of dust exposure.

It was the unanimous opinion, however, that the reference to cancer and tumors should be deleted and this is a point we will insist upon for the following reasons:

1. The experiments were not directed towards determining the incidence, if any, of cancer as a result of asbestos dust exposure.

2. Dr. Gardner indicated prior to his death that he believed this aspect should be made the subject of a separate study, which would take from two to three years.

3. Dr. Gardner also indicated that he believed the question of cancer susceptibility should be omitted from the report. This statement is contained in his letter to me of February 24, 1943 with which he enclosed his outline of a proposed monograph on asbestosis.

4. It also appears from Dr. Gardner's outline that certain strains of white mice develop tumors without apparent cause and that "such a strain of white mice was unintentionally used in three inhalation experiments with asbestos".

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A considerable number of other changes in the report will be suggested to Geranac, but they relate to form and to emphasis rather than to substance, and I believe there is no necessity of detailing them in this report to you.

We have retrieved all of the copies of this tentative and confidential report except the one we sent you, which I note Dr. Hamlin would like to keep. I wish, however, you would prevail upon him to return it to us. Everyone felt it would be most unwise to have any copies of the draft report outstanding if the final report is to be different in any substantial respect. The feeling of the representatives of the various companies was very emphatic on this point.

I am enclosing an extra copy of this letter in case you wish to send it to Dr. Hamlin.

Sincerely,

Vandiver Brown,
Secretary

VBr:y

Encl.

cc J.P.Woodard

AMERICAN BRAKEBLOK

AMERICAN BRAKE SHOE COMPANY

November 16th, 1948.

230 PARK AVENUE
New York 17, N.Y.

Mr. Vandiver Brown
Secretary
Johns-Manville Corporation
22 East 40th Street
New York 16, N. Y.

NOTED
V. B

Dear Mr. Brown:

Thank you very much for your letter of the 12th concerning the meeting held to discuss the Sarnmas Report.

Since we have the only carry copy of the tentative report I am asking Dr. Hamlin to return it directly to you.

Very truly yours,

Executive Vice President
American Brakeblok Division

ATK:LR

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THE SARANAC LABORATORY
FOR THE STUDY OF TUBERCULOSIS
OF THE EDWARD L. TRUDEAU FOUNDATION
SARANAC LAKE, N.Y.

February 24, 1943

Mr. Vandiver Brown
Johns-Manville Corporation
22 East 40th Street
New York, New York

Dear Mr. Brown:

I have at last succeeded in analyzing most of our voluminous experimental data and assessing the results. I realize that this should have been completed before this, but the emergency has left me short-handed in the Laboratory and also necessitated my doing a good deal of extra traveling. I hope that the sponsors of our study of asbestosis will be charitable and realize that the work has far exceeded its original scope.

We have done over 40 different experiments, many of them divided into several parts, which involved exposure of animals for 1 to 3 years to various dusts. The business of preparing microscopic sections and chemically analyzing the tissues on more than 600 animals has been a job in itself. My time for studying sections and analyzing data has been so limited that we are behind our schedule. I have still not had time to write a full report of this work which will of necessity be monographic. However, for the benefit of the contributors, I am submitting a table of contents and an annotated outline to indicate conclusions and the line of argument that will be developed. The latter itself occupies 15 pages, but I hope they will find it sufficiently interesting to read. I shall work on the final manuscript as I have opportunity.

There are a few experiments still in progress which should be completed by the time we are ready for them. The work on methods of dust determination, I consider important enough to include in the study. The question of cancer susceptibility now seems more significant than I had previously imagined. I believe I can obtain support for repeating it from the cancer research group. As it will take two or three years to complete such a study, I believe it would better be omitted from the present report. If it should become possible to make this study, I hope that I may count on some of your members to supply me with enough pure, long fibre asbestos for the purpose.

PLAINTIFF'S EXHIBIT 400 A

EX. H

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Naturally, I shall welcome any criticism that you or any of the other contributors would care to offer. Should any of them want to discuss details, I would be pleased to meet with them. May I take this opportunity to thank all the sponsors through you for the support that we have had.

Sincerely yours,

Leroy U. Gardner, M. D.
Director

LUG:mH

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Ross

OUTLINE OF PROPOSED MONOGRAPH ON ASBESTOSIS

Saranac Laboratory Study under Grant from Asbestos Association

PART I HUMAN ASBESTOSIS

I Human Pathology - a study of 25 autopsy cases, Illustrated

2 X-ray Patterns in Asbestosis, Illustrated

3 Ash and Mineral Values in Human Asbestosis

4 Complications of Asbestosis

(a) Susceptibility to infection

i Tuberculous
ii Non-Tuberculous
iii Cancer of the lung

5 Disability, causes and comparison with silicosis

6 Diagnosis

(a) History of adequate exposure
(b) X-ray film pattern
(c) Physical examination
(d) Asbestosis bodies in sputum, their significance

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PART II Experimental Asbestosis

I Methods

(a) Inhalation exposure to plant dusts.
(b) Injection into lungs through trachea of pure minerals.
(c) Injection of pure minerals into other organs.

2 Species Susceptibility

Man, guinea pigs, rabbits, cats, white mice and rats, dogs.

3 Peculiar Characteristics of Asbestosis

(a) Unusual localizaion of chrysotile fibre in lungs.
(b) Rate of resultant tissue reaction more rapid than to quartz.
(c) Reaction to chrysotile not progressive after exposure ceases; again the reverse of the situation in silicosis.
(d) Asbestosis Bodies

i Composition and methods of formation.
ii Occurence in different species.
iii Formation does not parallel development of fibrosis.
iv Gradual disappearance after exposure ceases.

4 Comparative Effects of Different Asbestiform minerals.

(a) Canadian Chrysotile.
(b) Arizona Chrysotile, low iron.
(c) Crocidolite
i Bolivian specimen, stiff and elastic.
ii South African, soft and flexible.
(d) Anthophyllite
(e) Amosite.
(f) Tremolite.

5 Effects of Control Minerals

(a) Granular Serpentine, same chemical composition as chrysotile.
(b) Glass Wool, a synthetic silicate fibre.
(c) Brucite, a fibrous magnesium hydroxide almost free of silica

6 Chemical Composition of Asbestiform minerals in Relation to Irritation.

(a) Nothing in composition correlated with relative irritatin capacity.
(b) Preliminary acid treatment

i Hydrochloric acid
ii Carbonic acid

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(c) Effects of Aluminum

7 Physical Properties in Relation to Irritation

(a) Length of fibre
(b) Effect of Crushing
(c) Heat treatment

8 Nature and Significance of Asbestosis Body

(a) Formation
(b) Protective effect preventing further irritation
(c) Ultimate solubility in tissue

9 Theories of Action of Asbestiform Minerals

(a) Chemical - reasons for considering invalid
(b) Mechanical - experimental demonstration of.

10 Complications

(a) Infection, tuberculosis and other varieties
(b) Cancer of lung - experimental data suggestive but not proven

11 Disability

12 Essential Features of Hazardous Exposure

(a) Nature of dust - fibrous component and size factors
(b) Atmospheric Concentrations - probably lower than for quartz

i Inadequacy of standard impinger sampling method which does not collect the dangerous fibres
ii Electrostatic precipitator sampling preferable but method must be modified

(c) Duration of Exposure

13 Recommendations for a New Standard of Safe Atmospheric Concentrations of Asbestos Dust

(a) The Quasi-official standard of 4 to 5 million particles per cu. ft.
(b) Work upon better method of sampling

(c) Necessity for comparison of results with X-ray findings in employees

14 Prevention

(a) Chemical means not practical

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i Acidosis necessary to dissolve fibre in lungs worse than Asbestosis
ii Aluminum Therapy inapplicable
iii Chief reliance still upon dust prevention with special emphasis upon the fibrous components

CC6865

ASBESTOSIS - ANNOTED OUTLINE INDICATING RESULTS

PART I - HUMAN ASBESTOSIS

I Human Pathology and X-ray Patterns - Description

Based upon 23 human autopsies

2 Ash and Mineral Values in Human Lungs.

3 Complication of Asbestosis

(a) Susceptibility to Infection

i Tuberculous- High incidence in English experience not duplicated in surveys of American Plants. Available autopsy statistics deceiving because of selection of material.

ii Non-Tuberculous- The same reason probably applies should be checked by analysis of absenteeism among asbestos workers.

iii Cancer of Lung Ditto, but there are now on record 10 cases of lung cancer in asbestos workers. Compared to the total number of autopsies on asbestosis, this incidence is excessive. No such frequency has been discovered in silicosis or other forms of pneumoconiosis except the Schneeberg mines of radioactive ores. The evidence is suggestive but not conclusive that asbestosis may precipitate the development of cancer in susceptible individuals.

4 Disability

Clinical experience suggests that truly disabling asbestosis is manifested by less striking X-ray changes than a corresponding degree of silicosis. Such disability in asbestosis is due to disease within the lungs and not to secondary heart disease. As in silicosis, associated pulmonary infection increases the amount of severity of the dust fibrosis with resultant accentuation of disability. There is urgent need for a careful physiological study of pulmonary function in asbestosis of varying severity. Undoubtedly, there are many diagnosable cases with no significant disability.

5 Diagnosis depends upon three factors.

(a) History of adequate exposure, usually 6 to 8 years, sometimes longer, at work where both the concentration and character of the "asbestos" dust are hazardous.

P 426 - 66 (1)

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(b) Evidence of disease in a characteristic X-ray pattern.

(c) A physical examination which reveals certain characteristics signs and may reveal evidences of disability if present.

(d) Asbestosis bodies in the sputum are confirmatory when the previous factors are all positive. However, the bodies may be absent, particularly in the absence of bronchitis infection.
Asbestosis bodies unsupported by other evidence do not make a diagnosis. Occasional ones have been found in about 15 autopsy specimens of persons with no known exposure and no fibrosis, probably of non-occupational origin.

****

PART II Experimental Asbestosis

I. Methods

(a) Inhalation Exposures to Plant Dust - Specific fibrosis in lungs, but no changes elsewhere.

(b) Injections of Suspensions of Pure Minerals into Trachea - Similar results.

(c) Injection of Suspensions of Pure Mineral[8] into other organs - Fibres dissolved; no asbestosis bodies; tissue reaction confined to phygocytosis and encapsulation. Compare the asbestos cases of human subjects

II Species Susceptibility

Unlike free silica, asbestos does not produce its specific effec in any organ of any species of animal. It causes fibrosis only the lungs of man and those of a few of the species tested.

Species	Fibrosis	Asbestos Bodies	Species	Fibrosis	Asbestos Bo
Man	4+	4+	Cat	+	±
Guinea Pig	2+	3+	White Mouse	0	±
Rabbit	+	±*	White Rat	0	0
			Dog	0	0

(* ± = very small atypical asbestos bodies)

III Peculiar Characteristics of Asbestosis

(a) Localization of fibrous minerals in lungs differs from that of granular dust particles.

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i Fibres like chrysotile having a certain degree of flexibility and elasticity accumulate within the finest air tubes; granular dust is carried further on and is widely scattered through the terminal air spaces.

(b) Rate of tissue reaction to asbestos is much more rapid than to an active dust like quartz. Evidences of formation appear as soon as sufficient concentration of fibres has localized in specific areas; with quartz, there is a latent period of months.

(c) Reaction to asbestos does not progress on cessation of exposure. Young scar tissue that may have formed, contracts and becomes more dense but the area of involvement decreases in size. In silicosis, the young nodules become larger after exposure ceases.

(d) Asbestosis Bodies are a specific concommitant of this form of pneumoconiosis. They are due to a deposit of protein and iron upon the surface of inhaled fibres. In guinea pigs they form after about 60 days of contact with the tissue. They are abundant in man and guinea pigs, (See paragraph 2 above) but much larger in the former probably because the larger sized air tubes admit larger fibres. In cats, rabbits and mice, there is an atypical coating of a few of the fibres after much longer residence in the lungs; in rats and dogs no bodies could be discovered. From paragraph 2 it is apparent that their occurence does not parallel development of fibrosis. In lung injection experiment, the bodies have not developed until after fibrosis ~~is well~~ advanced. The number of bodies seems to decrease several years after exposure ceases.

V Comparative Effects of Different Asbestosis Minerals

(a) Canadian Chrysotile - highly irritating.

(b) Arizona Chrysotile (low in iron) equally irritating and produces just as many asbestosis bodies as the Canadian product with over 11 times as much iron.

(c) Crocidolite - The South African blue asbestos is known to cause asbestosis. Only a limited supply of this material in pure form was available, most of it was used earlier in the work in non-productive experiments. For the later critical injection tests into the lungs, a Bolivian variety was substituted because of its high purity. Its fibres were much straighter, stiffer and more elastic than the cottony South African variety. Perhaps because of these peculiarities, it has not given reactions comparable to chrysotile. It produced asbestosis bodies but did not localize in the terminal air tubes nor cause any fibrosis. Tests now being repeated with a typical South African crocidolite with physical characteristics simulating chrysotile.

(d) Anthophyllite - stiff, straight fibres - atypical asbestosis bodies forms slowly but no localization or fibrosis in lungs.

(e) Amosite - Ditto.

(f) Amphibole - Ditto.

(g) Tremolite - Ditto but very few asbestosis bodies - Observation being continued.

V Control Mineral

(a) Granular Serpentine of same chemical composition as chrysotile is inert causing no fibrosis in lungs or other organs. It attracts iron from the lungs but of course, no "bodies" develop.

(b) Glass Wool(a synthetic silicate) fibres are not inhalable from air-borne suspensions, apparently because of their stiffness (the diameter is not responsible as some used were less than 1 micron thick) On injection into the lungs, they do not localize in the air tubes but are widely scattered. They cause no fibrosis. After 3 or 4 months in contact with lung fluid, a few glass fibres take up iron but remain smooth. They never show the swollen ends and lateral projections of the true asbestosis bodies.

(c) Brucite of interest because it is a fibrous mineral practically free of silica (0.9%). Crystallographically the arrangement of its Mg and OH groups in each unit cell is similar to that in chrysotile. The sample used also contained about 16% iron, probably from contaminating magnetite. The fibres are stiff and needle like. Observations not yet completed but after 2 months in the lungs, typical asbestosis bodies develop but there is as yet no fibrosis. The fibre are scattered through the lung instead of being localized inside the terminal air tubes and tissue reaction occurs around, instead of within the tubes.

VI Chemical Composition in Relation to Irritation

(a) Nothing in the following chemical compositions can be correlated with variations in capacity to provoke tissue reaction.

(See Table Next Page)

	Chrysotile	Crocidolite	Anthophyllite	Amosite	Amphibole	Tremolite	Brucite
SiO_2	37.43%	54.99%	57.43%	46.23%	55.04%	56.20%	0.90%
Fe_2O_3	2.35%	16.27%	0.51%	4.06%	3.06%	7.21%	6.78%
FeO	0	4.29%	0 ?	33.83%	0	0	9.36%
Al_2O_3	0.36%	1.02%	0.25%	1.09%	1.89%	0.56%	0.45%
CaO	0.05%	0.79%	0.44%	2.01%	12.22%	4.45%	0.40%
MgO	38.77%	12.25%	29.64%	8.29%	23.20%	20.82%	53.99%
Na_2O	0.29	6.92	0.52	0.33	0.40	6.78	0.91
K_2O	0.08	0.57	0.15	0.10	0.12	0.99	0.16
Loss <105°	4.30	0.02	0.62	0.68	0.32	0.34	0.53
>105°	14.92	2.55	2.42	1.51	3.59	2.78	25.66

(b) Acid treatment of chrysotile fibres

i One hour in dilute or concentrated HCL does not alter appearance of fibre but after such treatment it rapidly dissolves and disappears on injecting into living tissues.

ii Treatment with CO_2 bubbled through water or lung juice suspensions of chrysotile fibres causes partial solution with liberation of silica and magnesia.

(c) Colloidal allumina does not neutralize the effects of asbestos as it does quartz. On injecting chrysotile suspended in aluminum hydrate solution, fibrosis and asbestos bodies develop at the usual rate. It remains to demonstrate whether the fibres become coated with a layer of aluminum monohydrate as has been proved in the case of quartz. If they have, the coati does not affect their capacity to irritate tissue.

VII Physical Properties in Relation to Irritation of Lung Tissue

(a) Length of Fibre

Short chrysotile fibres under 3 microns are practically inert. 20 to 50 micron fibres cause typical fibrosis. The effects of longer ones could not be tested for technical reasons.

(b) Crushing completely destroys the fibrous structure of chrysotile. With loss of structure, all capacity to irritate disappears.

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(c) Heat At ignition temperatures, water is driven off but the fibre-structure is preserved. Fibres become extremely brittle. The physical change alters typical localization within the lungs, no fibrosis develops, reaction limited to phyagocytosis. No asbestosis bodies formed.

VIII Nature and Significance of Asbestosis Bodies

(a) Bodies probably result from deposition of iron and organic matter on a slowly dissolving fibre of asbestos. The source of the iron is more likely to be the lung tissue than the mineral itself as the coating is just as heavy on fibrous minerals of low iron content as upon those high in iron.

(b) The effect of asbestosis body formation is assumed to be protective although it has not been possible to isolate a large enough quantity of these structures in unaltered form for purposes of test. The smooth rounded ends presented by the bodies would not be mechanically irritating.

(c) If they were capable of causing irritation, the fibrosis in the lungs should progress after exposure ceases which it does not.

(d) The gradual disappearance of bodies (and fibres) long after exposure points to ultimate solubility in tissue fluids.

(e) The bodies are probably a fortuitous concomitant rather than a cause of fibrosis.

IX Theories of Irritant Action of Asbestiform Minerals

(a) Chemical These experiments do not confirm the theory that asbestosis is merely a form of silicosis resulting from free silica liberated in the solution of a silicate molecule. If this were true asbestos like quartz should cause fibrosis in any organ of any species for the experiments have shown that injected asbestos dissolves in these locations. However, it does not cause such reaction in any location but the lungs of a ~~few~~ species.

(b) Mechanical Irritation

We are proposing the theory of mechanical irritation which is manifested only in the lungs because this organ is the only one whose normal physiological functions involve a high degree of mobility. Experiments designed to prove the necessity for motion in this tissue have failed for technical reasons.
For this theory to be applicable, it is necessary that the fibres be concentrated in the fine terminal air tubes. It has been shown that only chrysotile of the asbestiform minerals thus far has the proper physical characteristics to insure such localization. It is also essential that the fibre shall persist long enough before it dissolves in the lungs of species like mice, rats, dogs. It apparently dissolves so rapidly that it exerts no irritation. Similarly rapid solution may explain in part, the lack of fibrosis in organs other than the lungs.

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The fibres must be long enough so that they cannot be completely surrounded by phagocytic cells which would prevent contact of these round, broken ends with the delicate cells supporting the air tubes. Observation has demonstrated that these conditions are realized in guinea pigs treated with long fibre chrysotile and fibrosis results. With the other asbestiform minerals that have been tested, some or all of the prerequisits were lacking and no fibrosis developed.

(c) If the irritation were chemical, fine serpentine which has the same chemical composition as chrysotile should have also caused fibrosis. The crushed chrysotile should have been more active than intact fibres, because of the greater surface areas exposed to body fluids. On the assumption that chrysotile, like quartz, becomes coated with a very thin layer of aluminum on treatment with colloidal aluminum hydroxide, capacity to cause fibrosis should be destroyed if the action were chemical but such is not the case.

(d) The heavy coating resulting from asbestosis body formation apparently does stop tissue reaction but here the effects are probably mechanical for reasons cited.

(e) Heat sufficient to alter chemical structure destroys power to irritate but it also alters essential physical characteristics that effect localization of the fibres in the lungs.

X Complications

(a) Susceptibility to Infection

i Tuberculous - Asbestos dust behaves like most other minerals in this respect and not like quartz which specifically increases native susceptibility to the tubercle bacillus. This infection may spread for a time but then heals. The resultant fibrosis accentuates that caused by the mineral fibre.

ii Non-Tuberculous - of no greater frequency than in animals inhaling dusts of other kinds. Occasional epidemics of pneumonia occur in our dust rooms, but these are due to methods of housing rather than to dust; they can also occur in unexposed animals.

iii Cancer of Lungs

No experiments were designed to elucidate this point but certain evidence suggests that asbestosis may actually favor development of tumors in susceptible species.

1 In guinea pigs, rabbits, rats, cats and dogs lung tumors are rare.

2 When these species were subjected to 2 to 3 years inhalation of asbestos dust, the incidence of lung tumor was not increased.

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3 Some strains of white mice do develop tumors without apparent cause.

4 Such a strain of white mice was unintentionally used in three inhalation experiments with asbestos.

5 Of 11 mice inhaling long fibre asbestos for 15 to 24 months 8 developed malignant tumors in their lungs and 6 of them had tumors in other organs. The incidence rate 81.8% is excessive.

6 Of 22 mice inhaling short fibre asbestos for not longer than 12 months only 3 developed lung tumors. Rate 13.6%

7 As controls, we have only the experience with mice in other dust experiments.
For short periods, there were 51 mice exposed to 4 other kinds of dust for 10 to 12 months. Incidence of lung tumor 1.9%.
For long periods, there were 143 mice exposed to 4 different kinds of dust, including pure quartz, 23 to 31 months. For all this group of mice the average incidence of lung tumor was 13.8%; the highest rate (28%) was in a subgroup exposed to flint dust.

Thus the incidence of lung cancer in the long fibre asbestos mice was over 15 times the average for mice inhaling other dusts for comparable periods and over 3 times the maximum for any other group. Mice exposed to the practically inert short fibre asbestos showed fewer lung tumors although 7 times more than those in short exposures to other dusts.

These observations are suggestive but not conclusive evidence of a cancer stimulating action by asbestos dust. They are open to several criticisms. The strain of mice was not the same in the asbestos experiment as in many of the others cited; apparently the former were unusually susceptible. Not enough animals survived in the dust for longer than the 15 months apparently necessary to produce many tumors. There were no unexposed controls of the same strain and age and no similar controls exposed to other dusts. It is hoped that this experiment can be repeated under properly controlled conditions to determine whether asbestos actually favors cancer of the lung.

XI Disability

Cannot be determined in animals. The accidental deaths were from the same causes met with in all our dust inhalation experiments.

XII Essential Factors of Hazardous Exposure

(a) Nature of Dust

i The hazrds increases with the proportion of intact fibres in the dust. Granular material and crushed fibre are inert diluents.

ii The long fibres must be thin enough (1-3 Microns) and short enough (under 50 Microns?) to be inhaled.

iii Very short fibres (under 3 Microns) are practically inert.

(b) Atmospheric Concentration Still under study.

Apparently this factor is lower than in the case of dusts composed of granular minerals but methods of estimation are misleading.

i The average standard Public Health Service impinger count in the long fibre asbestos dust room was 40 million particles per cu. ft. of air. This concentration caused fibrosis visible to the naked eye in 20 to 24 months. For comparison the average impinger counts in an experiment with pure quartz was 120 million particles per cu. ft. and fibrosis developed at about the same rate.

ii However, impinger counts are deceptive because by this method of sampling very few fibres, which are the significant elements in the dust are collected.

iii Sampling with an electrostatic precipitator is a much more efficient means of collecting fibres from air-borne suspensions. Samples from our long fibre asbestos room showed that the dust in the air contained 32.5% of fibres few of which had been collected or counted in the impinger sample. The latter sampled largely the inert granular particles.

iv As ordinarily employed precipitator samples are weighed and the results expressed in mg. per cubic foot of air. There is no means of converting such values into numbers of particles particularly when these vary in size, shape and specific gravity.

v Simultaneous sampling with precipitator and standard impinger yielded the respective values of 0.65 mg and 60 million particles per cubic foot of air.

vi Theoretically the best index of hazard would be either the number or weight of fibrous elements in the dust.

26-66(9)

(c) Duration of Exposure

i Fibrosis visible to the naked eye after 18 months exposure increases in extent in subsequent 18 months.

ii The span of life of our most susceptible laboratory animal, the guinea pig has prevented continuing exposure longer than three years. In this period only the comparatively early stages of asbestosis have been produced. With the knowledge that we have gained, it is probable that more extensive disease could have been produced with a purer long fibre chrysotile. The long lived species, like cats, dogs are unfortunately not susceptible.

iii For these reasons X-ray changes have been minimal and we were not able to fulfill one of the objectives of this program.

XIII Recommendation for a New Standard of Safe Atmospheric Concentration of Asbestos Dust.

(a) While there is no official standard, the tentative one of 4 or 5 million particles per cubic foot of air is frequently quoted.

(b) This is probably unreliable because it is based upon sampling with a standard impinger which we havershown does not collect most of the fibres that are the source of hazard.

(c) We now think that a standard should be based upon samples collected with an electrostatic precipitator if it is feasible to determine readily the relative proportion of fibres in such material.

(d) Work is still in progress upon the latter point.

(e) To be of value the new standard would have to be correlated with the X-ray findings upon employees exposed to different concentrations of dust.

XIV Prevention of Asbestosis

The experiments have failed to develop any practical chemical means of neutralizing the action of fibrous asbestos.

(a) To alter the human lung so that it would dissolve asbestos fibres rapidly like a rat would necessitate [redacted], creating an acidosis which would be worse than the effects of the fibrosis.

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(b) Apparently aluminum, which is so effective against free silica, has little influence upon fibrous chrysotile.

(c) However, it would be of great theoretical interest to treat a few cases with real dyspnoea from asbestosis by aluminum inhalation. We are by no means certain whether the favorable results upon advanced silicosis are due to a chemical neutralization of quartz in the lungs. Aluminum might have some more general pharmacological effect which influences dyspnoea.

(d) Chief reliance must still be based upon control of dust in the air and, in view of these experiments, particularly upon the fibrous components of such dust.

C06876

(5)

THE SARANAC LABORATORY
OF THE EDWARD L. TRUDEAU FOUNDATION
SARANAC LAKE, N.Y.

LEROY U. GARDNER, M.D.
DIRECTOR

March 18, 1947

Mr. Vandiver Brown, Secretary
Johns-Manville Corporation
22 East 40th Street
New York 16, N. Y.

NOTED
V.B.

Dear Mr. Brown:

Thank you for your letter of March 17th. I am very glad to comply with your suggestion and am doing so by sending to Dr. Lynch copies of your letter and enclosure, and of this reply.

The distinction which Dr. Gardner used to discuss with me was mainly that between fibrous and particulate asbestos dust, whereas Dr.King and his colleagues refer to long and short fibres. Just what they mean by these designations is not clear from the abstract and I find that we do not have the original paper. King was here last summer for an extended stay and I am sure that this was one of the many subjects on which he and Dr. Gardner exchanged notes, though the paper was probably submitted for publication before King left England. I am taking steps to secure a copy of the paper.

Dr. Gardner, in the notes which have been transcribed and sent to Dr. Lynch, makes the following points:

1. The English experience with regard to tuberculosis among workers exposed to asbestos dusts has not been duplicated in our American plants.

2. Fibres under 3 μ are practically inert, those between 20 and 50 μ caused typical fibrosis, while material crushed to destroy the fibrous structure is wholly inert.

3. To cause damage, the fibres must be long enough so that they cannot be completely surrounded by phagocytic cells.

4. 81.8% of mice inhaling long fibre asbestos develop lung cancer, a figure sixteen times that of the average for other dusts. 13.6% of mice inhaling short fibre asbestos develop lung cancer, a figure seven times the average for other dusts. These results are suggestive, rather than conclusive.

The above quotations from Dr. Gardner's notes are given merely for the purposes of this correspondence and should not be regarded as anything more. To what extent they may be modified, in the light of other findings, in the final report remains to be seen.

Sincerely yours,

Manfred Bowditch
Field Director

MB:AD

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A220

[redacted]

March 21, 1947

Mr. Manfred Bowditch,
Saranac Laboratories,
Saranac Lake, N. Y.

Dear Mr. Bowditch:

Thank you for your letter of March 18th, commenting upon the summary of Dr. King's article which I sent you on March 12th.

I am very much concerned by paragraph numbered 4, indicated by you as one of Dr. Gardner's notes. None of his interim reports, so far as I recall, had ever indicated any such abnormal incidence of lung cancer in the experimental animals.

Sincerely yours,

Vandiver Brown,
Secretary.

VB:y

bld cc: J. P. Woodard

JPW:- The finding referred to looks like dynamite.
VB

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A221

March 12, 1943

Mr. U. E. Bowes
Director of Research
Owens-Illinois Glass Company
Toledo, Ohio

Dear Mr. Bowes:

Thank you very much for your further information on the composition of your synthetic insulating material.

I am disappointed to hear that what we thought to be synthetic asbestos proved to be chrysotile which had been added as a reenforcing agent.

The fact that you are starting with a mixture of quartz and asbestos would certainly suggest that you have all the ingredients for a first class hazard. However, the particle size of the former will, of course be determined.

The asbestos may or may not be in such form as to be inhalable. We ourselves, will be able to get rid of the matrix I think so that we can determine the particle size of the quartz.

I would estimate that the cost of making the preliminary tests and particle size determination and so on would not exceed $300. With this information in hand, you can then decide whether you care to go into an experiment of a larger scale for which our charge is $5,000 for the first year and less in the succeeding ones as the number of animals to be kept under observation decreases.

I trust that this suggestion may meet with your approval.

Very truly yours,

Leroy U. Gardner, M. D.
Director

LUG:RBE

C07275

June 1, 1950

Mr. W. G. Hazard
Industrial Relations Division
Owens-Illinois Glass Company
Toledo 1, Ohio

Dear Bill:

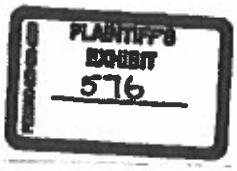
The Trudeau School currently in session prevented a more prompt answer to your letter of May 18. It finished today and I am now ready to attack the accumulated correspondence.

In reviewing your letter the first order of business is to urge you and Doctor Shook to visit Saranac Lake. My calendar is full up to the middle of June. Select any time after that date which is most convenient for both of you.

The second point which you raise relates to the X-Ray Department and the service by the Foundation for the reading and interpretation of chest roentgenograms of your employees. In commenting upon this matter as requested in your letter of April 3, there is certainly no objection on our part should you prefer that the matter be formalized on a contract basis. In many respects, some formal agreement would ease our administrative duties. I rather expect also that it would satisfy your purchasing department.

The agreement of last year was based on a flat rate of $4.00 per chest case. In explanation, per chest case refers to the roentgenograms of each man at a given date. Thus, the case might consist of only a single film, or it might include a stereo pair, a lateral, and an oblique. The fee of $4.00 would cover the entire lot. If on the other hand two dates were involved, we would consider that as two cases.

We see no reason to change that fee, unless there is some objection on the part of the company. It is indeed a relatively low figure and we keep it there only because of our continued interest in problems relating to the inhalation of dust. The opportunity to study clinical material of this sort is of tremendous help in our research program.



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The third point concerns the experimental investigations with Kaylo which are about completed. Only a few more animals remain. These will be sacrificed next month. Then we will be ready to prepare the final report. For your information, at this time, I believe the findings permit the following:

Kaylo dust on inhalation by experimental animals does not produce silicosis irrespective of the small amount of quartz present. It does produce the asbestatic type of reaction in the lungs and, therefore, we believe every precaution should be taken to minimize exposure of industrial employees.

Kaylo dust on inhalation by experimental animals infected with tubercle bacilli (R) produces only a very mild stimulation of the tuberculous infection, much less than that caused by the inhalation of pure quartz. This evidence leads us to believe that in industrial practice the minimal allowable concentration of Kaylo could be far in excess than that accepted for quartz before Kaylo would have an adverse stimulating effect upon a tuberculous infection in exposed employees.

This evidence would support the view that inhalation of Kaylo dust would not be hazardous from the standpoint of tuberculosis, especially provided that the dust hazard is controlled as cited above.

We are looking forward to having you and Doctor Shook with us the latter part of June.

Sincerely yours

Arthur J. Vorwald, M.D.
Director

AJV:bw

cc: Dr. Shook
Kaylo exp. file

C07285

A224

16158-004

AMERICAN ARBITRATION ASSOCIATION

OWENS-CORNING FIBERGLAS CORP.,

Claimant,

-against-

OWENS-ILLINOIS, INC.,

Respondent.

13 199 00953 93

REPLY OF OWENS-CORNING FIBERGLAS CORP. TO THE ANSWER AND COUNTERCLAIMS OF OWENS-ILLINOIS, INC.

Preliminary Statement

Claimant Owens-Corning Fiberglas Corp. ("OCF") submits this Reply to the Answer and Counterclaims of Owens-Illinois, Inc. ("O-I").[1]

This arbitration presents first and foremost an issue of contract interpretation: the plain meaning of an agreement made May 9, 1958 between O-I and OCF (the "1958 Agreement"). Section 6 of the 1958 Agreement requires that "O-I will save OCF harmless" from "any and all claims" for the breach of "all warranties" relating to Kaylo delivered prior to May 1, 1958.

1. Owens-Illinois Glass Company, the predecessor of O-I, is also referred to as O-I. O-I's Answer and Counterclaims is cited as "Ans."

The language of the indemnity comports with the manifest purpose of the Agreement and Section 6. For $6.9 million, OCP purchased the assets of the Kaylo division as of the close of business on April 30, 1958. In Section 6, OCP assumed obligations for Kaylo sales after that date. O-I retained the accounts receivable from and responsibility for Kaylo supplied prior to May 1, 1958. O-I's obligation to save OCP harmless reinforced this division of responsibility.

Decades after the parties signed the 1958 Agreement, individuals who worked with Kaylo delivered prior to May 1, 1958 began to get sick from exposure to asbestos-containing insulation. At first gradually, and then in unprecedented numbers, these individuals filed and continue to file personal injury lawsuits against manufacturers and sellers of asbestos-containing products. Those lawsuits assert claims for breach of warranty and its substantive equivalent, strict liability in tort. Since "O-I will save OCP harmless" from "all claims" for the breach of "all warranties" relating to Kaylo delivered prior to May 1, 1958, O-I must save OCP harmless from such personal injury claims.

O-I attacks this reading of the 1958 Agreement as "tortured," "contorted" and "unquestionably erroneous."

(Ans. at 8, 9) Such hyperbole is no substitute for analysis. It is telling that O-I barely addresses the language of the 1958 Agreement, focusing instead on a 1953 Sales Agreement between the parties (the "1953 Contract"), the history of warranty law, punitive damage awards levied against OCP in a handful of cases, and other diversions. None of these arguments can withstand scrutiny or diminish the clarity of the language chosen by the parties.

Finally, O-I asserts several counterclaims. Although the parties agreed in Section 11 to arbitrate disputes arising out of the 1958 Agreement, OCP and O-I never agreed to arbitrate O-I's purported claims arising under the 1953 Contract, under federal or state statutes, or at common law. These counterclaims should be dismissed.

I. THE 1958 AGREEMENT OBLIGATES O-I TO SAVE OCP HARMLESS

A. The Language of the 1958 Agreement is Plain

Under Ohio law:

> The nature of an indemnity relationship is determined by the intent of the parties as expressed by the language used. All words used must be taken in their ordinary and popular sense, and '[w]hen a . . . [writing] is worded in clear and precise terms; when its meaning is evident, and tends to no absurd conclusion, there can be

D5

> no reason for refusing to admit the meaning which . . . [it] naturally presents.'

Worth v. Aetna Cas. & Sur. Co., 513 N.E.2d 253, 256 (Ohio 1987) (citations omitted, ellipses in original).

The language of Section 6 of the 1958 Agreement is as sweeping as it is straightforward. The words of the 1958 Agreement, taken in their ordinary and popular sense, establish O-I's obligation to save OCP harmless. Section 6 states in relevant part:

> O-I will save OCP harmless from any and all claims . . . for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958.

OCP has been and continues to be sued by asbestos personal injury claimants for breach of warranties relating to Kaylo delivered prior to May 1, 1958. O-I is thus contractually obligated to save OCP harmless from such claims.

B. O-I Presents No Reason to Disregard the Plain Meaning of the 1958 Agreement

O-I asserts five reasons why this straightforward contractual provision does not mean what it says. Those reasons are based on the 1953 Contract, the history of warranty law, and an alleged course of performance -- in short, on everything but the 1958 Agreement.

i. The 1953 Contract

O-I's first three reasons for disregarding the plain language of the 1958 Agreement all depend on the applicability of the 1953 Contract. The 1958 Agreement and the 1953 Contract, negotiated five years apart, create distinct rights and obligations. O-I nevertheless asserts that the 1953 Contract "controls the relationship between the parties" (Ans. at 10) and thereby alters the 1958 Agreement's meaning. O-I is wrong for several reasons.

First, in matters of contract interpretation, Ohio law forbids reference to writings or statements extrinsic to an unambiguous written agreement. Section 6 of the 1958 Agreement is clear, so the parties' intent does not turn on any other writing.

Second, O-I's justification for looking to the 1953 Contract is premised on a logical fallacy. O-I assumes that the "warranties" mentioned in the 1958 Agreement must be defined by a written contract predating the 1958 Agreement: O-I reasons that "the 1958 Agreement itself creates no warranties. . . . Therefore, paragraph 6 [of the 1958 Agreement] only refers to preexisting contracts." (Ans. at 11) (emphasis added)

This is a non sequitur. Section 6 of the 1958 Agreement creates an indemnity not only for breaches of

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"agreements" or preexisting contracts, but also for breaches of "all warranties." The warranties relevant to this arbitration -- those invoked by asbestos personal injury plaintiffs against OCP -- were not created by "preexisting contracts" between OCP and O-I. Rather, they were created either by statements about Kaylo amounting to express warranties or, as to implied warranties, by operation of law. The 1958 Agreement created an indemnity for all claims for breach of "all warranties," regardless of whether they are embodied in a preexisting contract. Thus, the reference to "all warranties" in the 1958 Agreement is not limited to the single warranty in the 1953 Contract.

Indeed, O-I attempts to limit the 1958 indemnity to warranties in preexisting executory contracts. (Ans. at 10-11) However, while the 1958 Agreement provides indemnity for prior breaches of executory contracts assigned under that agreement, it provides an additional indemnity for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958:

> O-I will save OCP harmless from any and all claims for any breach, prior to assignment thereof, of any agreement so assigned, and for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958.

1958 Agreement § 6 (emphasis added). Since the initial clause applies to the executory contracts, the underscored

clause would serve no purpose if it, too, were limited to the executory contracts. Ohio law forbids O-I's attempt to render a clause of a contract meaningless.

Third, the 1958 Agreement -- not the 1953 Contract -- controls the relationship of the parties. To begin with, Section 6 of the 1958 Agreement contains an assignment by O-I to OCP of "all of the executory contracts . . . of the Kaylo division, including those for the purchase or sale of goods." Since the 1953 Contract was an "executory" contract for the purchase and sale of goods (Ans. at 11), O-I assigned the 1953 Contract to OCP in 1958. Moreover, the 1953 Contract had obligated O-I to sell millions of dollars worth of Kaylo to OCP (1953 Contract ¶ 1), but after May 1, 1958, O-I no longer had the right or capacity to manufacture Kaylo. Similarly, the 1953 Contract required that all sales and advertisements of Kaylo "shall be under [O-I's] trade name" and that OCP indicate that the Kaylo sold or advertised was "manufactured by" O-I. (1953 Contract ¶ 9) But under the 1958 Agreement, Kaylo was not in fact manufactured by O-I after May 1, 1958. In short, from the time of its execution, the 1958 Agreement has controlled the relationship of the parties.

Fourth, even if reference to an earlier contract were appropriate, the 1953 Contract does not limit the

claims subject to the 1958 indemnification. The 1958 Agreement and the 1953 Contract address entirely different types of claims. The 1958 Agreement, by its terms, requires O-I to "save OCP harmless from" breach of warranty claims. Thus it creates an indemnity for claims asserted against OCP. In contrast, the 1953 Contract addresses claims by OCP for breaches of that contract.

Moreover, the 1953 Contract deals only with Kaylo sold by O-I to OCP, while the 1958 Agreement applies to "all claims" for breach of warranty "relating to goods delivered prior to May 1, 1958." Prior to May 1, 1958, O-I "sold Kaylo to customers and distributors other than OCP." (Ans. at 11 n.4) The 1953 Contract does not even purport to deal with these Kaylo deliveries and thus cannot limit O-I's obligation in the 1958 Agreement to save OCP harmless.

O-I therefore misses the point in quoting out of context a provision of the 1953 Contract limiting OCP's remedies for breach of that contract and requiring those limitations in its contracts of resale. (Ans. at 12-13 & n.5; see 1953 Contract ¶ 5(a)) OCP is not suing O-I for breach of the 1953 Contract, nor are the claims for which OCP seeks indemnity brought by parties to contracts of resale. The 1958 Agreement squarely addresses the responsibility for warranty claims by third parties against

OCF for Kaylo delivered prior to May 1, 1958; the 1953 Contract does not. In a similar vein, the provision in the 1953 Contract requiring written notice within 90 days of shipment (Ans. at 14; 1953 Contract ¶ 5(e)) applies only to a claim for breach of the 1953 Contract. It has no relevance to an indemnification claim under the 1958 Agreement, which does not impose any notice requirement. Nor would such a requirement make sense since OCF's right to be held harmless does not arise until third parties bring claims triggering indemnification.

Thus, the unambiguous 1958 Agreement alone controls the obligation of O-I to save OCF harmless. The 1953 Contract could not and does not address that obligation. O-I's thesis -- that "the 1953 Sales Contract absolutely precludes OCF's claim for indemnification" (Ans. at 12) -- is untenable.

ii. Warranty Law Circa 1958

O-I's fourth challenge to the 1958 Agreement is that Ohio law in 1958 provided that claims for breach of warranty could be made only by persons "in privity" with a defendant. O-I does not dispute that asbestos personal injury claimants sue OCF for breach of warranty, even though they were not in privity with O-I or OCF. O-I instead asserts that the 1958 Agreement "was never intended to

encompass" asbestos personal injury claims by industrial workers who used Kaylo but who had not purchased it. (Ans. at 14)

Questions of the parties' intent are best answered by the language of the 1958 Agreement. That Agreement expressly provides indemnity for "all claims" for breach of "all warranties," without regard to whether the claimant is in privity or not. Additionally, the 1958 Agreement refers not to "warranties in agreements," but to "warranties and agreements." The language thereby contemplates claims based on extracontractual warranties.

Even if the history of warranty law were relevant, O-I is simply wrong in asserting that the 1958 Agreement cannot "reasonably be interpreted to encompass" personal injury claims. (Ans. at 14) In 1958, Ohio and other jurisdictions recognized the doctrines of express and implied warranty. In addition, the doctrine of privity was eroding, as Judge Cardozo had observed as early as 1931. By 1951, an Ohio court had upheld a warranty claim by the ultimate user of an industrial product against a manufacturer who was not in privity with the plaintiff.[2]

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2. The case involved a grinding wheel. O-I is thus wrong in attempting to limit the erosion of privity to "the sale of food or medicine" (Ans. at 14), although cases involving food, medicine, cosmetics and other goods had already discarded the concept of privity.

In 1953, an Ohio court observed that there was "hapless confusion" over the continued existence of the privity rule.[3] In light of the trend in Ohio and elsewhere toward rejection of the archaic privity rule, sophisticated parties represented by counsel could not reasonably have assumed that only those in contractual privity with a manufacturer would have standing to assert warranty claims.

iii. Course of Performance

Finally, O-I attempts to allege a "course of performance" as evidence of the parties' intent. (Ans. at 18) Courts sometimes look to a course of performance to explain ambiguous terms in contracts, but O-I has not identified any ambiguous word or phrase that it seeks to clarify by looking to a course of performance. Instead, it seeks to negate the plain language of the 1958 Agreement's indemnity provision. No extrinsic evidence, including evidence of a course of performance, is admissible for that purpose.

Additionally, OCF's waiting for an appropriate time to seek performance is not a "course of performance" as

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3. In addition to mischaracterizing Ohio law, O-I errs in asserting that the indemnity for "all claims" for the breach of "all warranties" is controlled by Ohio warranty law. (Ans. at 14) Kaylo was sold nationwide, so the parties would have necessarily considered the possibility of warranty claims under a panoply of state laws.

that term is used under settled law. In the past, OCF has sought indemnity from its insurers. OCF thus did not seek performance by O-I of its indemnity obligation, let alone engage in a course of performance. Now that OCF faces hundreds of millions of dollars of uninsured costs of asbestos personal injury claims, it is seeking performance under the 1958 Agreement.

O-I also distorts history in referring to a purported course of performance "since 1958." (Ans. at 15) Before the late 1970s, there had been only a handful of asbestos personal injury claims against OCF. O-I itself asserts that "the asbestos litigation began in the late 1970's." (Ans. at 4) (emphasis added) Thus, it is meaningless to talk of a "course of performance" for the first twenty or so years after the contract was executed.

The 1958 Agreement means what it says -- that O-I must save OCF harmless from all breach of warranty claims relating to Kaylo delivered prior to May 1, 1958.

C. O-I's Obligation to Save OCF Harmless Applies to the Six Specific Claims in OCF's Demand

The 1958 Agreement obligates O-I to save OCF harmless from "all claims" asserting breach of "all warranties" relating to Kaylo delivered prior to May 1, 1958. In addition to seeking a declaration of its rights

under the 1958 Agreement, OCP's demand for arbitration identifies six specific claims to illustrate types of asbestos personal injury claims made against it. The following chart shows the name of the claimant, the federal or state court in which the claim was brought, whether the claim is pending or settled, the claimant's dates of alleged exposure to Kaylo, and the type of warranty claim made.

Plaintiff	Court	Pending/ Settled	Kaylo Exposure	Claim
Hayes	U.S.D.C.	Pending	1950s	Implied Warranty
Haggart	Michigan	Pending	1953-72	Express/ Implied Warranties
Lindsey	Texas	Pending	1948-56	Implied Warranty/ Strict Liability
Barnett	Florida	Pending	1952, 1957, 1960	Implied Warranty/ Strict Liability
Tighe	Mass.	Settled	1953	Express/ Implied Warranties
Cardile	D. Mass.	Settled	1953-74	Express/ Implied Warranty

O-I contends that because these and other claimants allege various theories of liability in addition to breach of warranty, O-I is somehow absolved of any obligation to hold OCP harmless for breach of warranty claims.[4] (Ans. at 30) O-I's argument attempts to defeat

4. Count Two of Mr. Lindsey's complaint, denominated "Strict Liability," alleges that the defendants manufactured and distributed asbestos products for use as insulation materials, that installation of the (continued...)

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the plain intent of the parties to provide an indemnity and, in any event, is contrary to governing law. Under Ohio law, where allegations in a complaint indicate that indemnification is possible, the indemnitor has a duty to defend the indemnitee even though some of the asserted theories of liability are not covered by the indemnity. Nor is the indemnitor relieved of its obligation ultimately to indemnify as long as a settlement or judgment is based at least in part on causes of action for which the indemnitor is liable.

II. O-I'S OBLIGATION TO SAVE OCF HARMLESS APPLIES TO WARRANTY CLAIMS RELATING EXCLUSIVELY TO KAYLO DELIVERED BEFORE MAY 1, 1958

Many asbestos warranty claims -- and three of the illustrative cases (Hayes, Lindsey, Tighe) -- relate

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4.(...continued)
materials was part of their intended use or purpose, that the asbestos products "were not reasonably fit for the purposes for which they were intended," and that by placing the asbestos products on the market the defendants "represented that they would safely do the job for which they were intended." Count II of Mr. Bartnett's complaint is essentially the same as Mr. Lindsey's. As Ohio courts have observed, "strict liability in tort and implied warranty are 'virtually indistinguishable.'" Anderson v. Olmsted Utility Equip., Inc., 573 N.E.2d 626, 629 n.3 (Ohio 1991) (quoting Temple v. Wean United, Inc., 364 N.E.2d 267, 270 (Ohio 1977)). The reference in the 1958 Agreement to "all claims" for the breach of "all warranties" encompasses "strict liability" claims that are in substance breach of warranty claims.

exclusively to Kaylo delivered before May 1, 1958.[5] These claims fall squarely in the grip of the 1958 Agreement. O-I nonetheless argues that OCF's alleged misconduct precludes any indemnity. (Ans. at 16-29)

As a preliminary matter, O-I's smear campaign has doubtful relevance. O-I assembles a hodgepodge of allegations that "tort plaintiffs' lawyers" have made in support of levying punitive damages against OCF. (Ans. at 18) O-I does not, however, actually believe these allegations -- to the contrary, "Owens-Illinois does not here endorse the arguments and conclusions plaintiffs draw from OCF's history of asbestos-related products." (Ans. at 27) Consequently, O-I apparently argues that any indemnity is barred because punitive damages have wrongly been imposed on OCF in a handful of cases. This position has no legal or logical support.

Punitive damages are at most a side issue in this arbitration. OCF has been found liable for punitive damages in only a tiny fraction of asbestos cases, and juries and

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5. Some of the Kaylo O-I delivered prior to May 1, 1958 sat unused in the hands of distributors, other middlemen and other purchasers as of May 1, 1958. It may have been months or years before workers were first exposed to such Kaylo. Thus, in existing asbestos cases, O-I has traditionally settled claims against it alleging exposure to Kaylo before January 1, 1960. That same pragmatic rule should apply here.

courts have repeatedly rejected punitive awards against OCF. As of June 30, 1994, OCF had paid or settled punitive damage judgments in just fourteen cases, for a total of approximately $15 million.[6] This amount pales in comparison to well over $1 billion that OCF and its insurers had paid to resolve 131,000 asbestos personal injury claims as of June 30, 1994. In short, the vast majority of OCF's asbestos liability has nothing to do with punitive damages.

In any event, for claims relating to Kaylo delivered exclusively before May 1, 1958, there is no credible evidence of any OCF misconduct that could conceivably defeat the contractual indemnity. During this period, Kaylo was the only asbestos-containing insulation that OCF sold. O-I appears to argue that OCF engaged in "willful or wanton" misconduct by selling a product that O-I invented, manufactured and labelled.

O-I bases its farfetched argument about OCF's pre-1958 activities on tort plaintiffs' allegations regarding

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6. Jury awards in additional cases are under judicial review. Post-verdict proceedings often reduce or eliminate punitive awards. For example, a recent jury award in a New York case of $54.6 million, which O-I mentions (Ans. at 26), has been remitted by the trial court to $3 million. Similarly, in a case O-I repeatedly cites, Dunn v. Hovic, 1 F.3d 1371, 1391 (3d Cir.) (en banc), cert. denied, 114 S. Ct. 650 (1993), the district court reduced the jury's $25 million punitive damage award to $2 million, and the court of appeals further reduced the award to $1 million.

OCF's "actual independent knowledge" of health hazards attributable to asbestos. (Ans. at 19) This "actual independent knowledge" consists of publicly available reports -- equally available to O-I, and to the rest of the world for that matter. Those reports discussed the dangers of substantial exposure to concentrated asbestos dust in the asbestos textile industry but concluded that there was a safe level of exposure below which there would be no adverse health effects. This hardly supports O-I's assertion that OCF had "special knowledge." (Ans. at 7)

By contrast, O-I began comprehensive research and development of asbestos-containing Kaylo in 1938, started to produce limited quantities in 1943, and engaged in the full-scale manufacture of Kaylo starting in 1948. As plaintiffs in countless cases have pointed out, O-I's knowledge went far beyond the information available to OCF and the public. For example:

- In February 1943, O-I sent Dr. Leroy U. Gardner of the Saranac Laboratory samples of Kaylo and asked him to conduct tests to see if Kaylo was dangerous when "considered from the standpoint of employees working in the plant where the material is made or where it may be sawed to desired dimensions, and also considered from the standpoint of applicators or erectors at the point of use."

- One month later, Dr. Gardner informed O-I in a letter that the asbestos and quartz composition of Kaylo provided "all the ingredients for a first class hazard."

- Starting in 1943, Dr. Gardner and others at the Saranac Laboratory tested Kaylo on animals. By spring 1944, Dr. Gardner reported to O-I that injecting Kaylo into animals' lungs caused a condition similar to asbestosis.

- In November 1948, the Saranac Laboratory issued to O-I an Interim Report Regarding the Biological Activity of Kaylo Dust, which concluded that Kaylo, when inhaled, "is capable of producing asbestosis and should be handled as a hazardous industrial dust." The Report also noted that "very small numbers of fibers are capable of producing asbestosis," and recommended a safety program.

- The 1948 Report was forwarded with a cover memorandum from Dr. Arthur J. Vorwald, which stated: "In all animals sacrificed after more than 30 months of exposure to Kaylo dust unmistakable evidence of asbestosis has developed, showing that Kaylo on inhalation is capable of producing asbestosis and must be regarded as a potentially-hazardous material." Dr. Vorwald further observed: "since Kaylo is capable of producing asbestosis, it is better to discover it now in animals rather than later in industrial workers." (emphasis added)

- In June 1950, the Saranac Laboratory stated in a letter to O-I that dust from Kaylo produced "asbestotic type of reaction in the lungs and, therefore, we believe every precaution should be taken to minimize exposure of industrial employees."

- In early 1952, the Saranac Laboratory sent a final report to O-I entitled The Capacity of Inhaled Kaylo Dust to Injure the Lung. The Report informed O-I that Kaylo, inhaled for a prolonged period, could produce fibrosis typical of asbestosis in lungs of animals.

- The 1952 Report was accompanied by a cover letter stating that: "The results of the investigation with animals show that Kaylo dust is capable of producing a peribronchiolar fibrosis typical of asbestosis . . . . the results of the study indicate that every precaution should be taken to protect workers against inhaling the dust." The letter also informed O-I that the study would not be published without first being shown to

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O-I, and that it would not use the names "Kaylo" or "Owens-Illinois" in order to avoid harm to O-I.

Despite such knowledge, O-I reassured OCF prior to the sale of the Kaylo division that Kaylo was non-toxic and was safe to use. Indeed, part of the Kaylo business that OCF bought was O-I's copyrighted descriptions of Kaylo reflecting its safe and non-toxic qualities. (Even after O-I sold the Kaylo business, O-I continued to manufacture packaging for Kaylo for OCF into the late 1960s.) Not only did O-I fail to warn OCF of the health risks of Kaylo, it promoted Kaylo in publications as a safe product. For example, in a 1952 article, O-I's director of research for Kaylo stated that "Applicators appreciate the fact that hydrous calcium silicate is non-toxic and 'easy on the hands.'" An advertisement for Kaylo in the same magazine described it as "non-toxic."

A centerpiece of the asbestos plaintiffs' punitive damages argument against OCF is evidence of O-I's knowledge, which they wrongly impute to OCF because OCF bought the Kaylo division. On the strength of such evidence, O-I itself has been held liable for punitive damages to such an extent that, in 1991, a New York state court barred tort plaintiffs in the New York City Asbestos Litigation from obtaining further punitive damage awards against O-I because it had already suffered "repeated punitive damages awards."

Similarly, O-I has argued to appellate courts that the multiple punitive damage awards against it violate due process.

III. O-I'S OBLIGATION TO SAVE OCF HARMLESS APPLIES TO CLAIMS RELATING TO KAYLO DELIVERED BOTH BEFORE AND AFTER MAY 1, 1958

Many asbestos warranty claims — and three of the illustrative cases (Haggart, Bartnett, Cardile) — relate to Kaylo delivered both before and after May 1, 1958 (the "mixed claims"). On a strictly literal reading, the 1958 Agreement compels O-I to indemnify OCF completely against these mixed claims as well. Ohio courts have read the term "relating to" broadly, and the mixed claims indisputably "relat[e] to" Kaylo delivered prior to May 1, 1958, even if they also relate to Kaylo delivered later. See 1958 Agreement § 6.

However, OCF is not urging this most expansive reading of the 1958 Agreement. The language and structure of Section 6 manifest an intention of OCF and O-I to divide responsibility for Kaylo deliveries based on the date the Kaylo division changed hands, and OCF believes it should be interpreted accordingly. Consequently, liability for the mixed claims should be allocated between OCF and O-I based on some reasonable approximation of the harm attributable to Kaylo delivered prior to May 1, 1958, as compared with the

harm attributable to Kaylo delivered later. (As to pending and future claims, this result could readily be accomplished by requiring O-I to include OCF in the releases O-I obtains in resolving claims against it for Kaylo exposure prior to January 1, 1960.)

The existence of mixed claims cannot absolve O-I of responsibility for such claims, as O-I suggests (Ans. at 30-31). It would be inappropriate and inequitable to interpret the 1958 Agreement to bar any indemnity, even though a claim in fact relates to Kaylo delivered prior to May 1, 1958, simply because the claim also involves some Kaylo delivered later. Barring an indemnity for mixed claims would contravene the plain intent of the parties to divide responsibility for Kaylo deliveries based on the date the Kaylo division changed hands.

Nonetheless, O-I relies heavily on "plaintiffs' allegations" of misconduct following OCF's purchase of the Kaylo division in an attempt to nullify its obligation to save OCF harmless. (Ans. at 21-27) OCF categorically denies that it has engaged in misconduct; O-I's allegations are based on misstatements or misunderstandings of the facts. For example, O-I alleges that OCF refused to market "an asbestos-free Kaylo substitute, Multitemp." (Ans. at 24) However, Multitemp was no substitute for Kaylo.

Multitemp was effective at temperatures up to about 900°F, while Kaylo was effective up to 1800°. Specifications for naval and power plant use typically required insulation at over 1000°. If Multitemp became wet, it would shrink, leading to potentially dangerous gaps in insulation. By contrast, Kaylo was resilient even after being soaked. Multitemp, a clay product, was far heavier and more difficult to use than Kaylo, a calcium silicate.

O-I's allegations of OCF's "fraudulent concealment" in asbestos litigation (Ans. at 27-29) are similarly groundless. For example, O-I repeatedly quotes from an order of a Texas trial court in Bodine v. Owens-Corning Fiberglas. (Ans. at 2, 17, 28-29 & n.10) The Texas Court of Appeals has already issued seven writs of mandamus against the trial judge in Bodine for his inappropriate rulings against OCF, has threatened the judge with an order to show cause why he should not be held in contempt, and has already expressed its tentative opinion that OCF is entitled to relief from the order O-I quotes.

But even if O-I could prove the allegations of OCF misconduct that "plaintiffs" have made but that O-I disbelieves, this alleged misconduct would bear only on the apportionment of responsibility for individual claims between OCF and O-I. OCF seeks to be indemnified only as to

pre-May 1, 1958 Kaylo deliveries; it does not seek an indemnity for any of its own alleged misconduct. The alleged misconduct does not change the fact that Kaylo for which O-I is responsible also contributed to a tort plaintiff's injury in breach of a warranty, and that O-I is contractually obligated to save OCF harmless from that degree of injury.

IV. O-I'S OTHER DEFENSES ARE MERITLESS

A. The Statute of Limitations Bars, At Most, Only A Handful of Claims

Under Ohio law, each of OCF's claims against O-I for indemnification accrues upon the filing of a specific personal injury claim against OCF. From the time each indemnification claim accrues, OCF has fifteen years in which to bring its indemnification claim, a matter of Ohio law on which the parties agree (Ans. at 30). OCF served its demand for arbitration based on O-I's obligation to save OCF harmless in October 1993. Thus, as to asbestos personal injury suits filed against OCF since October 1978 (fifteen years prior to OCF's service of its demand), OCF's claims for indemnification are timely.

The fifteen-year period represents the Ohio Legislature's considered judgment as to length of time an indemnitee should have to enforce a contractual indemnity

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after a claim is filed against the indemnitee. Since virtually all the asbestos personal injury suits against OCF were brought in the last fifteen years, O-I's time-based defenses have almost no merit.

B. Laches and Estoppel

By asserting claims within the statute of limitations, OCF has not unreasonably delayed in seeking indemnification from O-I, and O-I has neither changed its position as a result of OCF's actions nor suffered any prejudice. O-I thus cannot satisfy any of the elements of a laches or estoppel defense.[7]

OCF seeks indemnification from O-I for six specific asbestos personal injury claims and a declaration with respect to claims brought against OCF for breach of warranty relating to Kaylo delivered prior to May 1, 1958. Under Ohio law, these claims did not accrue until the filing of a lawsuit against OCF. OCF cannot be charged with unreasonably delaying its assertion of its indemnification rights as to recently accrued claims against OCF for breach of warranty, including the six claims described in OCF's demand for arbitration. Moreover, OCF's ability to seek a

7. Estoppel is used only to stem activity that is wrongful and deceitful; the doctrine is disfavored under Ohio law.

declaration as to indemnification for future asbestos claims is clearly unimpaired since the law does not require a party to seek a declaration of its rights before they accrue.

As for earlier asbestos personal injury claims, they took decades to materialize. In 1975, fewer than 100 claims were pending against OCF. As O-I acknowledges, asbestos litigation did not begin in earnest until "the late 1970's." (Ans. at 4) From that time forward, OCF sought indemnity from its insurers. Now that OCF has begun to exhaust its insurance, it seeks indemnification from O-I under the plain terms of the 1958 Agreement. Thus, OCF has not unreasonably delayed in asserting its rights against O-I.

The doctrines of laches and estoppel do not apply in any event because O-I cannot make a clear showing that it suffered material prejudice as a result of any delay or as a result of OCF's conduct generally.[8] O-I contends that it has been prejudiced because it "would have handled the OCF asbestos litigation defense differently and minimized the amounts OCF now demands in indemnity." (Ans. at 29) O-I gives no clue as to how it would have engineered such a

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8. Under Ohio law, mere delay without prejudice is not sufficient to support the application of either the doctrines of laches or estoppel.

feat. Indeed, since the beginning of the flood of asbestos litigation, OCF has retained experienced counsel at substantial cost -- approximately $61 million in 1993 alone -- to aid in an effective defense. In some cases, O-I and OCF were represented by the same counsel.

Moreover, O-I could not have been materially prejudiced or misled by any "delay" in OCF's assertion of its indemnification claims, because O-I was fully aware of the existence of the litigation for which OCF seeks indemnification.[9] Indeed, O-I has been OCF's co-defendant in many of these cases. O-I therefore had knowledge that OCF had been sued on grounds for which O-I had agreed to indemnify OCF. For these reasons, O-I cannot meet its burden of establishing the requirements for application of the doctrines of laches or estoppel.

V. MOST OF O-I'S COUNTERCLAIMS ARE NOT SUBJECT TO ARBITRATION

Section 11 of the 1958 Agreement provides that "[a]ny controversy or dispute arising out of this Agreement shall be settled by arbitration conducted in accordance with the rules, in effect at the time the controversy or dispute arises, of the American Arbitration Association." Disputes

9. There is no requirement in the 1958 Agreement for OCF to provide formal notice to O-I of its liability in a specific case.

that do not arise out of the 1958 Agreement are not subject to arbitration. Nearly all of O-I's counterclaims attempt to arbitrate issues that do not arise out of the 1958 Agreement. Because the Panel lacks jurisdiction over such counterclaims, they should be dismissed.

In its first counterclaim, O-I seeks a declaration of its rights under the 1958 Agreement, the 1953 Contract, federal and state statutes, and the common law. This Panel has jurisdiction to issue such a declaration only with respect to O-I's rights under the 1958 Agreement. The 1953 Contract does not contain an arbitration clause applicable here,[10] and nothing in the 1958 Agreement obligates OCF to arbitrate disputes arising out of the 1953 Contract, much less out of federal and state statutes or the common law. For the same reason, O-I's second and third counterclaims are not arbitrable. They arise not out of the 1958 Agreement, but out of the 1953 Contract and the common law. No agreement obligates OCF to arbitrate these claims here, and OCF does not consent to do so.

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10. The 1953 Contract does contain a clause mandating arbitration of claims under paragraph 5(e) of the contract in Lucas County, Ohio, in accordance with the Ohio Arbitration Act, before a panel including two party-appointed arbitrators. See 1953 Contract ¶ 5(f). OCF has made no claim under paragraph 5(e). However, in light of paragraph 5(f), it is inconceivable that the parties intended to have an AAA arbitration in New York as to the meaning of the 1953 Contract.

Conclusion

The 1958 Agreement is clear: O-I is contractually obligated to save OCF harmless from all breach of warranty claims relating to Kaylo delivered prior to May 1, 1958. The obligation to save OCF harmless applies to all six of the illustrative cases and entitles OCF to the declaration it seeks.

Dated: September 22, 1994

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PLAINTIFF'S EXHIBIT 710

COMMON OFFICERS AND DIRECTORS OF OCF AND OR ACCORDING TO ANNUAL REPORTS

	OWENS CORNING FIBERGLAS, INC.		OWENS-ILLINOIS, INC.	
YEAR	DIRECTORS	OFFICERS	DIRECTORS	OFFICERS
1938	C.B. Belknap		C.B. Belknap	C.B. Belknap (Exec. VP)
	Harold Boeschenstein	Harold Boeschenstein (P)	Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis (__ & Pres)
1939	C. B. Belknap		C.B. Belknap	C. B. Belknap (Exec. VP)
	Harold Boeschenstein	Harold Boeschenstein (P)	Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis
1940	C.B. Belknap		C.B. Belknap	C.B. Belknap (Chairman of the Board)
	Harold Boeschenstein		Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis
1941	C. B. Belknap		C.B. Belknap	C.B. Belknap (Vice Chairman of the Board)
	Harold Boeschenstein		Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	William E. Levis (COB)
	J. P. Levis		J.P. Levis	J.P. Levis (P)
1942	C. B. Belknap		C. B. Belknap	C. B. Belknap (Vice Chairman of the Board)
	Harold Boeschenstein		Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis (COB)

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1943	C. B. Belknap		C.B. Belknap	C.B. Belknap (Exec. VP)
	Harold Boeschenstein		Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis (COB)
	J.P. Levis		J.P. Levis	J.P. Levis (P)
1944	C.B. Belknap		C.B. Belknap	C.B. Belknap (Exec. VP)
	Harold Boeschenstein		Harold Boeschenstein	
	Wm. E. Levis		Wm. E. Levis	Wm. E. Levis (COB)
	J.P. Levis		J.P. Levis	J.P. Levis (P)
1945	C.B. Belknap		C.B. Belknap	C.B. Belknap (E. VP)
	Harold Boeschenstein	Harold Boeschenstein (P/GM)	Harold Boeschenstein	
	William E. Levis		William E. Levis	William E. Levis (COB)
	J.P. Levis		J.P. Levis	J.P. Levis (P)
1946	Harold Boeschenstein	Harold Boeschenstein (P/GM)	Harold Boeschenstein	
	J.P. Levis		J.P. Levis	J.P. Levis (P)
	William E. Levis		William E. Levis	William E. Levis (COB)
1947	Harold Boeschenstein	Harold Boeschenstein (P/GM)	Harold Boeschenstein	
	William E. Levis		William E. Levis	William E. Levis (COB)
1948	Harold Boeschenstein	Harold Boeschenstein (P)	Harold Boeschenstein	
	William E. Levis		William E. Levis	William E. Levis (COB)

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AMERICAN ARBITRATION ASSOCIATION

Owens-Corning Fiberglas Corporation :

v. : No. 13 Y 199 00953 93

Owens-Illinois, Inc. :

STIPULATION OF FACTS

The parties to this proceeding, Owens-Corning Fiberglas Corp. ("OCF") and Owens-Illinois, Inc. ("Owens-Illinois"), hereby stipulate and agree to the following facts for purposes of the hearing in this proceeding.

1. OCF has never indicated in any asbestos personal injury settlement agreement that it has entered into with plaintiffs' counsel that it possesses a claim for contractual indemnification against Owens-Illinois.

2. Commencing in or about May 1958, OCF sold asbestos-containing pipe and block insulation that was branded "Kaylo" which OCF manufactured.

3. At various times subsequent to May 1958, OCF sold asbestos-containing pipe and block insulation that was branded "Kaylo" which was manufactured by other entities pursuant to rebranding agreements with OCF.

4. In settling asbestos personal injury lawsuits, OCF obtains a single release from the plaintiff discharging it from liability for all causes of action or theories of liability advanced by the plaintiff, without allocating specific portions of the total settlement payment to the plaintiff to particular causes of action or theories of liability.

PLAINTIFF'S EXHIBIT 105

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Produced pursuant to Protective Order

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AMERICAN ARBITRATION ASSOCIATION

Owens-Corning Fiberglas Corporation :

v. : No. 13 Y 199 00953 93

Owens-Illinois, Inc. :

STIPULATION OF FACTS

The parties to this proceeding, Owens-Corning Fiberglas Corp. ("OCF") and Owens-Illinois, Inc. ("Owens-Illinois"), hereby stipulate and agree to the following facts for purposes of the hearing in this proceeding.

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In some cases, the release permits a plaintiff who has settled on the basis of a non-malignant condition to seek additional compensation (either under the settlement agreement or in the tort system) in the event the plaintiff subsequently develops certain types of asbestos-related cancer.

5. In settling asbestos personal injury lawsuits, Owens-Illinois obtains a single release from the plaintiff discharging it from liability for all causes of action or theories of liability advanced by the plaintiff, without allocating specific portions of the total settlement payment to the plaintiff to particular causes of action or theories of liability. In some cases, the release permits a plaintiff who has settled on the basis of a non-malignant condition to seek additional compensation (either under the settlement agreement or in the tort system) in the event the plaintiff subsequently develops certain types of asbestos-related cancer.

6. OCF is not able to locate documents showing the Heat Insulating Products' "performance specifications" as that term is used in the 1953 Sales Agreement.

7. Owens-Illinois is not able to locate documents showing the Heat Insulating Products' "performance specifications" as that term is used in the 1953 Sales Agreement.

8. OCF is not able to locate documents showing whether or not it complied with its obligation to include in its resale contracts the limitation on Owens-Illinois's liability referred to in paragraph 5(e) of the 1953 Sales Agreement.

9. Owens-Illinois is not able to locate documents showing whether or not OCF complied with its obligation to include in its resale contracts the limitation on Owens-Illinois's liability referred to in paragraph 5(e) of the 1953 Sales Agreement.

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10. To the best of its available information, prior to May 1, 1958, OCF sold no Kaylo products other than the Heat Insulating Products identified in the 1953 Sales Agreement and the amendments and schedules thereto.

11. Between April 1953 and May 1958, Owens-Illinois sold Heat Insulating Products, door core material, roof tile, laminated panels and other specialty products, all under the brand name Kaylo, to parties other than OCF.

12. Except for invoices, OCF is unable to locate copies of any sales contract documents pursuant to which it sold Kaylo pipe and block insulation prior to May 1, 1958.

13. Except for the 1953 Sales Agreement and the amendments and schedules thereto, OCF is unable to locate any sales contract documents pursuant to which it purchased Kaylo pipe and block insulation prior to May 1, 1958.

14. Except for the 1953 Sales Agreement and the amendments and schedules thereto, records relating to government renegotiation contracts, and records relating to sales commissions, Owens-Illinois is unable to locate copies of sales contracts documents pursuant to which it sold Kaylo pipe and block insulation prior to May 1, 1958.

15. OCF has never informed its banks or other lenders of the indemnity claim being asserted in this proceeding.

16. Prior to late 1992, OCF had not informed its accountants or auditors of the indemnity claim being asserted in this proceeding.

17. OCF has, on a monthly or otherwise fairly regular basis, submitted a list of cases to its insurance carriers for reimbursement.

18. OCF's producer share under the Wellington Agreement was initially approximately 21.4% and after January 1, 1987 was reduced to approximately 19.4%.

19. Owens-Illinois's producer share under the Wellington Agreement was initially 5.6% and after January 1, 1987 was reduced to 5.1%.

20. The Asbestos Claims Facility established pursuant to the Wellington Agreement began operations in the fall of 1985 and was dissolved effective October 3, 1988.

21. During the negotiation of the Wellington Agreement, OCF never indicated to any Owens-Illinois representative that OCF had a contractual indemnification right against Owens-Illinois.

22. During the negotiation of the Wellington Agreement, Owens-Illinois never indicated to any OCF representative that Owens-Illinois had a contractual indemnification right against OCF.

23. During the period that Owens-Illinois manufactured asbestos-containing pipe or block insulation that was branded "Kaylo", Owens-Illinois's annual sales of such Kaylo pipe and block were as follows:

Year	Sales (Dollars)
1943	5,125.14
1944	54,983.16
1945	61,068.62
1946	101,485.57
1947	137,747.26
1948	360,555.64
1949	840,016.19
1950	1,435,335.79
1951	2,692,643.37
1952	3,335,841.65
1953	3,455,375.77
1954	3,134,158.59
1955	3,224,764.16
1956	4,400,777.58
1957	4,596,591.42
1958	1,731,765.77

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24. During the period that OCF manufactured asbestos-containing pipe or block insulation that was branded "Kaylo", OCF's annual sales of such Kaylo pipe and block were as follows:

Year	Sales (Dollars)
1953	2,849
1954	3,798
1955	3,868
1956	5,637
1957	5,558
1958	6,732
1959	5,990
1960	7,167
1961	6,812
1962	7,062
1963	7,864
1964	7,859
1965	9,138
1966	9,193
1967	10,054
1968	9,683
1969	9,045
1970	8,844
1971	8,436
1972	9,430
1973	4,715

25. Owens-Illinois has never indicated in any asbestos personal injury settlement agreement that it has entered into with plaintiffs' counsel that it possesses a claim for contractual indemnification from OCF under the terms of either the 1953 agreement or the 1958 agreement.

26. In connection with its settlement of asbestos personal injury cases or claims, Owens-Illinois customarily requires proof that the plaintiff or claimant was exposed to asbestos-containing Kaylo manufactured or sold by Owens-Illinois.

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27. In connection with its settlement of asbestos personal injury cases or claims, Owens-Illinois customarily requires proof that the plaintiff was exposed prior to January 1, 1959 to asbestos-containing Kaylo manufactured or sold by Owens-Illinois.

28. In connection with its settlements of asbestos personal injury cases or claims, Owens-Illinois customarily refuses to compensate plaintiffs for exposure to asbestos-containing Kaylo subsequent to January 1, 1959 (one exception to this policy occurred in connection with cases in Mississippi where a date of January 1, 1960 was used).

29. The releases which Owens-Illinois obtains from asbestos personal injury plaintiffs or claimants do not include a release or discharge of OCF from liabilities arising out of OCF's sale or distribution of asbestos-containing Kaylo delivered prior to May 1, 1958.

Owens-Corning Fiberglas Corp.

By its attorneys,

DEBEVOISE & PLIMPTON
875 Third Avenue
New York, New York 10022

Owens-Illinois, Inc.

By its attorneys,

DAVIS POLK & WARDWELL
450 Lexington Avenue
New York, New York 10017

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PLAINTIFF'S EXHIBIT 708

6/24/94 T-0158-OCF

AMERICAN ARBITRATION ASSOCIATION -- # 13 199 00953 93

ANSWER AND COUNTERCLAIMS OF
OWENS-ILLINOIS, INC.
TO THE ARBITRATION DEMAND MADE BY
OWENS-CORNING FIBERGLAS CORPORATION

On October 27, 1993, Owens-Corning Fiberglas Corporation ("OCF"), an Ohio company, commenced this arbitration proceeding against Owens-Illinois, Inc., its Toledo-based neighbor. OCF demands indemnity from Owens-Illinois for the $188,750 (plus costs) OCF paid to settle two lawsuits brought in Massachusetts by individuals who alleged personal injury from OCF's sale of an asbestos-containing insulation product whose trademark name was Kaylo. OCF supplemented its demand on April 20, 1993, seeking its costs in defending four pending (and as yet unresolved) lawsuits alleging injuries from Kaylo, and seeking a declaratory judgment that Owens-Illinois is obligated to indemnify OCF for all the monies it previously paid in settling tens of thousands of similar lawsuits alleging disease from exposures to Kaylo prior to May 1, 1958. Although OCF quantifies neither the amounts it demands in indemnity for the four pending lawsuits nor the value of its declaratory judgment demand, the fact is that hundreds of millions of dollars are at stake.[1]

OCF's demand for indemnity is predicated on an agreement dated May 9, 1958 between Owens-Illinois, as seller of the assets of a business, and OCF, as the buyer (the "1958

[1] A copy of OCF's October 27, 1993 demand for indemnification is attached as Exhibit 1 and a copy of its April 20, 1994 supplemental demand is attached as Exhibit 2.

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Protective Orders

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AMERICAN ARBITRATION ASSOCIATION

ANSWER AND COUNTERCLAIMS OF
OWENS-ILLINOIS, INC.
TO THE ARBITRATION DEMAND MADE BY
OWENS-CORNING FIBERGLAS CORPORATION

On October 27, 1993, Owens-Corning Fiberglas Corporation ("OCF"), an Ohio company, commenced this arbitration proceeding against Owens-Illinois, Inc., its Toledo-based neighbor. OCF demands indemnity from Owens-Illinois for the $158,750 (plus costs) OCF paid to settle two lawsuits brought in Massachusetts by individuals who alleged personal injury from OCF's sale of an asbestos-containing insulation product whose trademark name was Kaylo.[1] OCF supplemented its demand on April 20, 1993, seeking its costs in defending four pending (and as yet unresolved) lawsuits alleging injuries from Kaylo, and seeking a declaratory judgment that Owens-Illinois is obligated to indemnify OCF for all the monies it previously paid in settling tens of thousands of similar lawsuits alleging disease from exposures to Kaylo prior to May 1, 1958. Although OCF quantifies neither the amounts it demands in indemnity for the four pending lawsuits nor the value of its declaratory judgment demand, the fact is that hundreds of millions of dollars are at stake.

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Agreement"; copy attached as Exhibit 3). The 1958 Agreement unquestionably does not provide indemnity for the personal injury claims for which OCF is now seeking indemnification. Moreover, the 1958 Agreement could not under any circumstances be a proper vehicle by which to shift OCF's liability for misconduct that tort plaintiffs have been able to establish was deliberate, knowing, fraudulent, and, as one court wrote, "if not criminal, borders on being criminally culpable conduct". The only party entitled to relief in this proceeding is Owens-Illinois, not OCF.

I.

BACKGROUND

Owens-Illinois began the manufacture and sale of Kaylo in commercial quantities in 1948. Kaylo was a thermal insulation product line that encompassed premolded pipe covering, insulation block, roof tiles, door core material, and other specialty products. Kaylo contained asbestos.

Not mentioned in OCF's demand is a contract between Owens-Illinois and OCF dated March 20, 1953, pursuant to which OCF became the primary purchaser of the Kaylo product made by Owens-Illinois and, in turn, marketed this product through OCF's own organization (such contract is hereinafter referred to as the "1953 Sales Contract"; copy attached as Exhibit 4). The 1953 Sales Contract expressly limited Owens-Illinois' liability to OCF for any damages that OCF might incur to third

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parties as a result of OCF's resale of Kaylo. The 1953 Sales Contract's limitations preclude OCF's indemnity claims here.

Five years later, pursuant to the 1958 Agreement, OCF purchased the Kaylo division from Owens-Illinois. From May 1, 1958 onwards, it was OCF, not Owens-Illinois, that manufactured, marketed, and sold Kaylo. With the 1958 sale of the Kaylo division to OCF, Owens-Illinois left the business of manufacturing asbestos-containing insulation products. OCF continued to manufacture and sell Kaylo at least until 1972, and possibly for years thereafter.

A. The Nature of Asbestos Litigation

The Panel is undoubtedly aware of the deluge of asbestos claims that have been filed over the past two decades in courts across the country. Over 200 companies have been named as defendants in the litigation. Owens-Illinois and OCF are among those defendants. Owens-Illinois is generally alleged to have tort liability to those who have an asbestos-related disease resulting from exposure to Kaylo manufactured and sold during the period from 1948 to 1958. OCF is alleged by asbestos tort claimants to have independent tort liability for selling asbestos-containing Kaylo from 1953 to 1958 and for manufacturing and selling Kaylo from 1958 to at least 1972. OCF is also alleged to have manufactured and sold other asbestos-containing products, including asbestos cement, from the 1940's through the 1970's. OCF's arbitration demand represents an effort by OCF to shift its tort liability to

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Owens-Illinois for every claim that involved any alleged exposure to Kaylo before 1958.

Asbestos, a naturally occurring mineral, is fibrous and friable. That means when asbestos is crushed or cut, fibers are released. The fibers in this asbestos "dust", if inhaled, may lodge in the lungs. Exposure to asbestos fibers does not necessarily result in physical impairment or disease. There has to be sufficient exposure over sufficient time before a risk of pulmonary disease arises, although there is medical debate as to what constitutes a "sufficient" dose or period of exposure. Even then, asbestos-related disease may or may not result.

An important aspect of asbestos-related disease is its latency period: disease or impairment may not manifest itself until decades after exposure to asbestos dust ceased. Thus, when the asbestos litigation began in the late 1970's, the individuals claiming asbestos-related disease most often were workers such as World War II ship insulators or building tradesmen who installed insulation from the 1940's to the 1960's. In recent years, the exposures alleged and the occupations involved have shifted to those individuals more peripherally exposed, but long latency periods continue to characterize the claims.

The tort plaintiffs contend that, once asbestos fibers are inhaled, the resulting disease, if any, is cumulative, indivisible, and progressive. The range of injuries alleged runs from non-impairing x-ray changes of the

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lung to fatal malignancies. Experience shows that the occurrence and severity of asbestos-related disease turns on such factors as intensity and duration of exposure, latency, other potentially contributory factors (e.g., smoking), and individual susceptibility. As a general proposition, the likelihood of harm increases with cumulative dose.

Many complaints allege that the plaintiff was exposed to numerous types of asbestos-containing products at many different jobsites during the worker's career. The complaints often name many (up to thirty or more) defendants, and allege that the defendants knew or should have known that their products were dangerous to users of the products and that the defendants failed to warn of that danger.

Asbestos plaintiffs typically pursue claims based on a number of tort legal theories, including negligence, strict liability, and claims under particular states' products liability laws.[2] The gist of these theories is a culpable failure to warn of the health hazards possibly associated with exposure to asbestos. Punitive damages are often requested (see, e.g., Exhibit 6 ¶¶ 19-31).

A critical issue in asbestos cases under current tort law is when manufacturers and sellers of asbestos-

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[2] See, e.g., Exhibit 5 ¶¶ 9-35; Exhibit 6 ¶¶ 6-28. Exhibits 5 and 6 are the complaints in the Tighe and Cardile cases, two lawsuits OCF settled and for which OCF's October 27, 1993 letter (Exhibit 1) demands indemnification. While these complaints do not name as many defendants as do most asbestos-related complaints, they are otherwise typical of the hundreds of thousands of asbestos-related complaints.

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containing products knew, or should have known, of the potential health risks to end-users. As a general matter, although the health risks of prolonged exposure to massive levels of pure asbestos dust in textile mills and mines were known as long ago as the 1930's, the use of finished insulation products containing asbestos was considered safe for many years thereafter. In 1946, the American Conference of Governmental Industrial Hygienists recommended a standard "threshold limit value" or "TLV" concerning asbestos dust which represented the recognized safe level of occupational exposure over a worker's lifetime. Thereafter, many states, including Ohio whose laws govern this dispute and New Jersey where the Kaylo manufacturing facilities were located, passed laws establishing TLV standards for occupational dust exposure, including asbestos. In that same year, 1946, an epidemiological study of shipyard insulators (the Fleischer-Drinker report) established that use of finished insulation products did not generate exposures above the TLV and that pipecovering was not considered a dangerous occupation.

In 1965 Dr. Irving J. Selikoff and his colleagues at New York's Mount Sinai Hospital published a landmark study reporting that exposures to asbestos-containing insulation products within the TLVs previously thought to be safe were in fact causing disease in insulation workers. Follow-up studies confirmed this finding and broadened its scope. Asbestos was banned by the EPA from most commercial uses in 1972.

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Despite the general state of medical knowledge prior to Dr. Selikoff's study, plaintiffs sometimes persuade juries that the defendants that manufactured or sold products containing asbestos prior to 1965 are nonetheless liable. The plaintiffs' liability case is often centered on the special knowledge of certain defendant sellers or manufacturers concerning the health risks to users of asbestos-containing products prior to 1965, or on those companies' failure to take appropriate steps to warn users of those risks even after Dr. Selikoff's 1965 report. As is discussed below, OCF was a seller and manufacturer who is alleged both to have had special knowledge concerning the health risks to users of asbestos-containing products and to have failed to take appropriate steps to warn users after publication of the Selikoff study.

B. OCF's Asbestos Litigation History

According to OCF's most recent Form 10-K filed with the Securities and Exchange Commission, OCF faced 97,800 pending asbestos personal injury claims as of December 31, 1993, having previously resolved more than 120,000 such claims. To date, OCF has paid or agreed to pay over $1.2 billion to satisfy judgments and to settle asbestos claims. In 1992, OCF established a balance sheet reserve of $950 million for its anticipated uninsured asbestos liabilities through 1999.

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OCF's litigation history includes significant punitive damage awards. In 1993 and 1994 alone, evidence of OCF's special knowledge of the risks to users of asbestos-containing products, its failure to warn users, and its efforts to cover up its culpable conduct has led juries to impose punitive damages aggregating more than $116,000,000.[3] Because the same body of evidence is available to every plaintiff, punitive damages have been a mounting threat to OCF. Recently, that threat has become the primary focus of OCF's defense. In a recent consolidated trial of the claims of 11,000 plaintiffs (the largest such trial to date), OCF, choosing to contest only punitive damages, conceded in its opening statement that it would not contest its liability for compensatory damages: "When you go back to the jury room, you can check Plaintiffs win on that, or the Judge will instruct you so." In re Asbestos, Civil Action No. 92-C-8888, April 12, 1994 Tr. 218 (W. Va. Cir. Ct. Kanawha Co.).

Having conceded in the public record its own independent tort liability to thousands of tort plaintiffs, OCF now seeks, for the first time, reimbursement from Owens-Illinois for the damages it has paid to personal injury claimants. It bases its claim on a tortured construction of a commercial agreement written more than thirty-six years ago.

[3] During the same period, Owens-Illinois incurred verdicts imposing punitive damages aggregating $60,000.

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C. OCF's Demand for Indemnity

OCF's demand for indemnity is, in effect, a demand that OCF is entitled to be indemnified for all amounts it has paid or will pay in settlement of Kaylo-related claims where the complaint alleges that the plaintiff was exposed to Kaylo for some period prior to May 1, 1958. In addition, OCF seeks a declaratory judgment from this Panel that amounts to a "blank check" on Owens-Illinois' funds to pay OCF settlements and judgments in the future (See Exhibit 2 ¶ (2)).

Although OCF has been defending asbestos-related products liability personal injury suits since the 1960's, and has paid many hundreds of millions of dollars to settle claims and pay judgments, it is only now -- more than thirty-five years after the execution of the 1958 Agreement by which OCF purchased the Kaylo division -- that OCF demands indemnification from Owens-Illinois. This belated action appears to stem from the fact that OCF is running out of insurance money with which to defend or settle asbestos claims. Since 1990 OCF has gone through over $1 billion in confirmed insurance coverage and whatever insurance it has remaining will soon be exhausted. Thus, the Panel should recognize from the outset that OCF's effort to bring a claim based upon a contorted and unquestionably erroneous interpretation of a thirty-six year old agreement is just a desperate ploy by OCF to avoid its own responsibility for resolve third party personal injury claims and to transfer that liability to Owens-Illinois.

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II.

OWENS-ILLINOIS' ANSWER TO OCF'S DEMAND

OCF's demand for indemnification is utterly without merit. OCF is attempting to rewrite the 1958 Agreement and totally ignores the 1953 Sales Contract which, in fact, controls the relationship between the parties.

A. The 1953 and 1958 Contracts

In 1958, Owens-Illinois sold its division that manufactured Kaylo to OCF. The contract of sale, the 1958 Agreement, contained a paragraph addressing the Kaylo division's then-existing executory contracts, contracts that Owens-Illinois had entered into but had only partially performed prior to the May 1, 1958 sale of the Kaylo business. The paragraph reads as follows:

> "6. O-I hereby assigns to OCF all of the executory contracts as of May 1, 1958, of the Kaylo Division, including those for the purchase or sale of goods, materials, equipment, supplies and capital assets, agreements with labor unions, consultant agreements and all other contracts having to do with the conduct of its business (excepting, however, accounts receivable arising from goods supplied, services rendered or other transactions prior to May 1, 1958) and OCF agrees to perform and discharge all executory obligations under such contracts (excepting, however, any obligation for goods supplied and services rendered prior to that date, these obligations remaining the responsibility of O-I and excepting the obligation, if any, of O-I to pay compensation to any salaried employee of its Kaylo Division by reason of the termination of his employment by O-I), and will save O-I harmless from any and all claims of any third person or persons for any breach, after assignment thereof, of any agreement so assigned. O-I will save OCF harmless from any and all claims for any breach,

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prior to assignment thereof, of any agreement so assigned, and for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958." (Exhibit 3 ¶ 6.)

The last clause of this paragraph has been seized upon by OCF to claim improperly that Owens-Illinois owes it an indemnification for all liabilities arising from OCF's pre-1958 sales of Kaylo.

OCF is wrong for at least five reasons. First, the 1958 Agreement itself creates no warranties. The contracts assigned by the 1958 Agreement were transferred "as is", the parties having agreed that no additional representations or warranties were created by the contract (See Exhibit 3 ¶ 10). Therefore, paragraph 6 only refers to preexisting contracts. Unless a warranty in those contracts was breached, OCF could not assert any claim against Owens-Illinois. With respect to Kaylo, the preexisting contract was the 1953 Sales Contract, which governed all sales of Kaylo by Owens-Illinois to OCF and any obligations arising thereunder. Under both Ohio law (which expressly governs both the 1953 and 1958 contracts) and the terms of the 1953 Sales Contract itself, the 1953 Sales Contract extended until January 1, 1959 (Exhibit 4 ¶ 10). Thus, the 1953 Sales Contract was executory and the 1958 Agreement did not abrogate or amend the 1953 Sales Contract.[4]

[4] Owens-Illinois sold Kaylo to customers and distributors other than OCF. An indemnification claim by OCF arising from those sales would be barred for the reasons set forth beginning at page 14, below.

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OCF has neither attached, nor incorporated, nor referred the Panel to the 1953 Sales Contract. There is a reason for this: the 1953 Sales Contract absolutely precludes OCF's claim for indemnification.

The 1953 Sales Contract provided only one warranty in favor of OCF:

> "(d) Seller warrants that all Kaylo Heat Insulating Products sold to Buyer pursuant to this Agreement will meet Seller's performance specifications in effect at the time of sale." (Exhibit 4 ¶ 5(d).)

The 1953 Sales Contract further provided unequivocally that there was:

> "no warranty, agreement, or understanding, express, statutory or implied, either in fact or in law, with reference to or a part of this Agreement, except such as is set forth herein." (Exhibit 4 ¶ 17.)

In short, Owens-Illinois only warranted Kaylo's "performance specifications". OCF does not and cannot contend that any asbestos claims for which it is seeking indemnification alleged breach of Kaylo's "performance specifications". By virtue of the 1953 Sales Contract's plain language, therefore, OCF's claim for indemnification under the 1958 Agreement for Owens-Illinois' purported breach of "warranty" must fail.

Second, the 1953 Sales Contract expressly provided that, with respect to the Kaylo sold by OCF:

> "Seller shall not be liable in any event for special or consequential damages" (Exhibit 4 ¶ 5(e)).

In 1953, the term "consequential damages" meant "damage, loss, or injury as does not flow directly or immediately from the

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act of the party, but only from some of the consequences or results of such act" or "damage which, though actionable, does not follow immediately, in point of time upon the doing of the act complained of." Black's Law Dictionary (4th ed. 1951). In other words, in the 1953 Sales Contract OCF expressly agreed that Owens-Illinois would not "in any event" be liable for indirect or late-arising damages resulting from the Kaylo sold by Owens-Illinois to OCF and then by OCF to third parties whose use of the product allegedly resulted in injury to individuals exposed to asbestos fibers. It is precisely for damages to those individuals who inhaled the asbestos fibers that OCF is now demanding indemnification. Since OCF agreed in the 1953 Sales Contact that Owens-Illinois would not be liable for such damages, OCF's claim for indemnity is barred.[5]

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[5] Where Owens-Illinois breached a provision of the 1953 Sales Contract, the amount of damages OCF could claim from Owens-Illinois was expressly limited to the amount OCF paid for the Kaylo:

> "Seller shall not be liable for any breach of this Agreement in any amount in excess of the agreement price of the products with respect to which such breach occurs." (Exhibit 4 ¶ 5(e).)

Thus, should the Kaylo sold by Owens-Illinois to OCF have breached the performance specifications Owens-Illinois warranted, the most OCF could recover from Owens-Illinois is the amount OCF paid Owens-Illinois for that Kaylo.

In paragraph 5 OCF also agreed that the limitation on Owens-Illinois' damages to OCF and the provision that Owens-Illinois would not be liable for "special or consequential damages" were to be included in OCF's resale contracts. OCF agreed to indemnify Owens-Illinois for any liabilities arising from OCF's failure to include these limitations:

> "Buyer shall include this same limitation upon the amount of Seller's liabilities in contracts effecting all resales by Buyer to third persons and Buyer shall

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Third, the 1953 Sales Contract required OCF to give Owens-Illinois written notice of any "errors, shortages, imperfections, deficiencies or any failure of the products to conform with the terms of this Agreement" within 90 days after shipment (Exhibit 4 ¶ 5(e), (f)). If OCF wished to preserve the right under the 1953 Sales Contract to be recompensed for a claim, OCF had to have given notice to Owens-Illinois within 90 days after shipment. OCF did not meet this deadline, which expired by the middle of 1958, at the latest.

Fourth, the hold harmless language in the 1958 Agreement was never intended to encompass, nor can it reasonably be interpreted to encompass, an obligation by Owens-Illinois to indemnify OCF for the cost of personal injury tort claims. Such claims are based on a tort theory of products liability that was not even recognized in 1958 by the State of Ohio, the law of which governs the 1958 Agreement and the 1953 Sales Contract. The Uniform Sales Act was the law of Ohio in 1958, and it provided that claims for breach of warranty could be made only by persons "in privity" with the defendant, that is, persons (or their immediate families) who were parties to a contract with the seller or manufacturer respecting the goods alleged to be defective. Except in limited circumstances such as the sale of food or medicine, remote consumers who were injured by a defective product could

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indemnify and save Seller harmless from any liabilities arising from Buyer's failure so to contract in making resales." (Exhibit 4 ¶ 5(e).)

not recover on an implied "warranty" theory for personal injuries until legal decisions and statutes first began to create such rights years after the 1958 Agreement was entered into. Since the 1958 Agreement must, as a matter of law, be construed and applied in conformity with the law as it existed in 1958, a vastly expanded notion of "warranty" liability for personal injuries cannot be, and should not be, retroactively read into the 1958 Agreement.[6]

Finally, the parties' course of performance since 1958 proves that OCF has no indemnity right for third party personal injury tort claims under the 1958 Agreement. Within a few years after OCF negotiated and signed the 1958 Agreement, OCF began to be sued in asbestos personal injury lawsuits. At that time, presumably, the drafters, negotiators, and signatories of the 1958 Agreement were living and employed by or available to OCF. Their collective recollections of the meanings attributed to the language of

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[6] The Panel should recognize that although a contract is interpreted in light of the law as it existed at the time of its execution, tort liability is determined as of the time a plaintiff commences a lawsuit. Tort law began to change in the 1960's, culminating in theories of liability that today impose "warranty" liability on manufacturers and sellers of products that cause personal injury to those who use the product. Because of the latency aspect of asbestos-related disease, asbestos-related injuries did not begin to manifest themselves in large numbers until after this change in tort law. Courts generally have held that statutes of limitations applicable to tort claims do not begin to run until an asbestos-related disease manifested itself, in other words until a plaintiff discovers the injury. The terms of an underlying contract, however, do not change even if the tort law develops. Rather, the question remains what did the parties intend and what was the law of contracts, at the time the contract was executed?

paragraph 6 by the parties would have been fresh. Their collective experience in applying the provisions of paragraph 6 would have given them a great advantage in determining its reach. It strains credulity to believe that, if anyone at OCF had thought that an indemnity for personal injury claims existed in paragraph 6, they would not have asserted it within a reasonable time after OCF began to be sued. Instead, OCF did not commence this proceeding until October 1993, after OCF had already resolved more than 120,000 such lawsuits at a cost of approximately 1.2 billion dollars. OCF's course of conduct thus confirms the objective truth as well as the subjective belief and acknowledgement of OCF officials and counsel for many prior years and decades -- that OCF has no indemnity right against Owens-Illinois for OCF's Kaylo liabilities.

OCF's rights concerning pre-May 1, 1958 Kaylo sales were created and defined by the 1953 Sales Contract, and under the plain terms of that contract OCF retained the liability for which it now seeks indemnification. The 1958 Agreement did not magically expand -- retroactively -- Owens-Illinois' contractual obligations or OCF's contractual rights. OCF is therefore entitled to no relief in these proceedings.

B. <u>OCF's Own Conduct Bars Its Claim for Indemnification</u>

OCF's liability to tort plaintiffs has been and continues to be based upon evidence of (1) OCF's <u>own</u> pre-1958 knowledge and conduct concerning products other than Kaylo; (2) OCF's <u>own</u> alleged knowledge and misconduct after 1958,

when OCF assumed complete control over the manufacture and sale of Kaylo; and (3) OCF's own attempt since the early 1930's to conceal its culpable conduct, an effort recently characterized by one court as a "deliberate and intentional fraud upon the Courts," Pickering v. Owens-Corning Fiberglas Corp., No. 90-L-1546, Order dated April 7, 1992 (Ill. 3d Jtr. Cir.), aff'd No. 5-92-0691 (Ill. App. Ct., 5th Dist. May 3, 1994), and by another as conduct that "if not criminal, borders on being criminally culpable." Bodine v. Owens-Corning Fiberglas, No. 92-C-2440, slip op. (June 11, 1993 23rd Jud. Dist., Brazoria Co., Texas), petition for writ of mandamus pending, No. 01-93-00532-CV (Ct. App. 1st Sup. Jud. Dist. Houston). The fact that OCF's liability to tort plaintiffs arises from pre-1958 knowledge and conduct concerning products other than Kaylo and from acts that courts have found to be fraudulent bars OCF's claim in several further respects.

As to the period prior to May 1, 1958, under no reading of the 1958 Agreement did Owens-Illinois agree to indemnify OCF for OCF's actions concerning products other than Kaylo. As to the period following May 1, 1958, because OCF's liability has been based on acts evidencing misconduct and illegality, by its conscious, willful actions, OCF voided any indemnity right it may have had from Owens-Illinois. It is well-established as a matter of law that any act on the part of an indemnitee (here, OCF) which materially increases the risk, or prejudices the rights of the indemnitor (here, Owens-

Illinois), will discharge the indemnitor under a contract of indemnification.

Further, OCF's actions have both inflated the amounts that OCF has had to pay to settle claims and the amounts of jury verdicts rendered against OCF. It will be impossible for OCF to demonstrate what part, if any, of the amounts for which it is seeking indemnity were not attributable to its post-1958 misconduct.[7]

Finally, as a matter of well-established public policy, Ohio law forbids the use of an indemnification agreement to escape damages caused by willful or grossly reckless conduct of the indemnitee. To permit OCF to receive a "breach of warranty" indemnity for third party personal injury claims in light of its activities that courts have found to constitute "willful or wanton" misconduct would contravene sound public policy.

To give content to the extent of OCF's conduct, we set forth below a summary of the evidence against OCF as developed by the tort plaintiffs' lawyers.

1. OCF's Pre-1958 Activities

In numerous lawsuits, OCF's independent conduct prior to May 1, 1958 has been crucial to establishing the fact and amount of OCF's liability by verdict or in settlement.

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[7] Similarly, OCF will not be able to demonstrate what part, if any, of its liability arises from its pre-1958 conduct concerning asbestos-containing products other than Kaylo.

That misconduct is premised upon OCF's actual independent knowledge and actions. Plaintiffs have successfully argued that, even before its 1958 purchase of the Kaylo division from Owens-Illinois, OCF was aware of the health hazards caused by the inhalation of asbestos and the resulting risks to workers installing asbestos-containing products. This knowledge is alleged to have predated the publication of the seminal 1965 study by Dr. Selikoff, and to have been possessed by OCF at a time when the connection between asbestos exposure and cancer was not generally recognized in the medical literature.[8]

The "willful or wanton" conduct and "reckless indifference" courts have found respecting OCF is most often traced back to its efforts in the early 1940's to take commercial advantage of its actual knowledge of asbestos-related health hazards. The tort plaintiffs argue that OCF was concerned about complaints by workers that OCF's fiberglass products irritated their skin and a resulting

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[8] As discussed above, prior to Dr. Selikoff's work, the authoritative study of asbestos-containing insulation products had been the 1946 Fleischer-Drinker report which suggested that, so long as asbestos dust was kept within threshold limit values, applying asbestos-containing insulation was a safe process. Whether the information OCF possessed during the 1940's and 1950's was sufficient to contradict the then-known state of medical literature is not an issue this Panel will have to decide. (Owens-Illinois believes the information OCF possessed was not.) Rather, the point here is that plaintiffs have obtained settlements and judgments against OCF on the basis of OCF's own individual conduct and alleged knowledge respecting this information. OCF's liability for pre-1958 sales is thus founded upon conduct unrelated to the conduct that (accepting arguendo OCF's "interpretation") would be within the indemnity provided for in the 1958 Agreement.

demand for wage premiums to handle those products. According to the tort plaintiffs, OCF believed that the makers of competing asbestos-based products might be behind the workers' demands and decided to convince the insulators that their alternative to handling fiberglass -- handling asbestos-containing insulation -- posed a greater danger to their health. Plaintiffs point to a series of OCF documents, written between 1941 and 1946, to show juries that OCF had well-developed actual knowledge concerning the health hazards of asbestos-containing insulation.

Additionally, plaintiffs allege that during the 1950's OCF continued to learn more about the health risks associated with asbestos, particularly the harms it posed to those who installed asbestos-containing insulation in the field. OCF continued to acquire and use this knowledge to protect its sales of fiberglass, its principal product. The tort plaintiffs stress that OCF's willingness to use its actual knowledge of asbestos-related hazards for commercial advantage -- placing dollars over people -- transforms OCF's tortious conduct into willful and wanton misconduct. Plaintiffs support these contentions by pointing to numerous OCF documents dated 1955, 1956, and 1957 -- years before Dr. Selikoff's study published in 1965.[9]

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[9] Throughout the time Owens-Illinois was producing Kaylo, Owens-Illinois believed, based on information actually available to it, that end-users of Kaylo were not at risk. The grounds for Owens-Illinois' belief were (1) evidence derived from its extensive factory health program where no occupational illnesses had been found relating to asbestos;

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Recently, the Virginia Supreme Court upheld a punitive damage award against OCF because the evidence established "positive acts" committed by OCF "which the jury could have concluded constituted willful or wanton evidence evincing a conscious disregard of the rights of others." Owens-Corning Fiberglas Corp. v. Watson, 243 Va. 128, 146, 413 S.E.2d 630, 641 (1992); see also Dunn v. Hovic, 1 F.3d 1371, 1374 (3d Cir. 1993) (en banc), cert. denied, 114 S. Ct. 650 (1993) (sustaining punitive damages based upon a jury's finding that OCF's failure to warn constituted "reckless indifference"). These acts and omissions by OCF and the liability arising therefrom are wholly unrelated to the 1958 Agreement's indemnity provision even as erroneously construed by OCF and OCF consequently has no right to indemnification for them.

2. OCF's Activities Following Its Purchase of the Kaylo Division from Owens-Illinois in 1958

After OCF purchased the Kaylo division in 1958, OCF embarked on a course of action respecting Kaylo that factually, legally, and equitably bars OCF, from obtaining an indemnity from Owens-Illinois.

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(2) the fact that Owens-Illinois had never received a claim for injury resulting from Kaylo's use; (3) its actual review of and reliance upon the Fleischer-Drinker study; (4) its actual knowledge and implementation of the asbestos TLV in its Kaylo manufacturing plants; and (5) the Saranac Laboratory study concerning Kaylo. Thus, Owens-Illinois believed that the use of Kaylo insulation in the field would not generate asbestos exposures in excess of the threshold limit values and would not pose a health hazard to end-users.

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(a) Rebranding Other Manufacturers' Products. Immediately upon its purchase of the Kaylo division from Owens-Illinois, OCF began entering into rebranding agreements whereby other companies' asbestos-containing insulation products (such as Johns-Manville's Thermobestos) were rebranded and sold under the Kaylo brand name. As OCF documents recognize, some of these rebranded products, particularly Thermobestos, had a higher asbestos content than the original Kaylo. Such rebranding by OCF of asbestos-containing insulation products posing a potentially greater health hazard than Kaylo vitiates OCF's claim for indemnity because it is possible that plaintiffs who claim they were exposed to "Kaylo" were in fact exposed to other insulation products.

(b) OCF Made Kaylo Dustier. OCF changed the composition of Kaylo in such a way as to make it more "dusty". OCF unquestionably knew that such a change would make its Kaylo potentially more hazardous. After the change, a 1963 OCF memorandum summarized the "associated problems involved in the category of Kaylo dust":

> "1. The amount of dust which occurs on the surface of the material which becomes bothersome when handling the product.
>
> 2. The dust becomes airborne when the product is cut or fabricated.
>
> 3. The health hazard of the dust with respect to asbestosis."

In 1963, OCF's recognition of the potential health hazards associated with asbestos was memorialized by a member

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of OCF's product development group who wrote, "Asbestos [as found in Kaylo] when breathed into the lungs causes asbestosis which often leads to lung cancer." This admission is directly contrary to OCF's litigation position (in interrogatory answers, briefs filed in court, and arguments to juries) that OCF could not have known of the connection between lung cancer and asbestos-containing insulation products prior to the 1965 publication of Dr. Selikoff's seminal study.

(c) OCF's Failure to Remedy Dustiness. Having made Kaylo more dusty and hence a greater health hazard, OCF then refused to implement some simple steps that it discovered would have cut down on Kaylo's dustiness. In 1962, OCF's research and development department was charged with reducing Kaylo's dustiness. That department discovered that, by coating Kaylo with Ludox, a sodium silicate, it could practically or even completely eliminate Kaylo's dustiness. Consistent with a record during the 1960's of failing to invest in equipment or processes at Kaylo plants that would reduce the dust unless they contributed to bottom line profitability, OCF elected not to use Ludox on Kaylo. The cost of Ludox, as OCF's documents demonstrate, would have been only one penny per square foot for the complete elimination of Kaylo dust, and "less complete elimination of dustiness could be achieved with correspondingly lower figures." Yet, no Ludox was ever used.

(d) OCF's Refusal to Market an Asbestos-Free Product. Even more remarkably, in the early 1960's OCF's

recognition of asbestos-related health hazards led its research and development department to develop an asbestos-free Kaylo substitute, Multitemp. This product was not, in the words of a contemporaneous OCF memorandum, "as detrimental to the health as is Kaylo." When OCF field-tested Multitemp the reaction was "quite favorable" and Multitemp was even preferred by some pipe fitters to Kaylo. With improvements, OCF's research department concluded, Multitemp could be "superior to any product presently on the market." However, as the head of OCF's Kaylo research department emphasized in a memorandum, "Do we attempt to improve Multitemp? My answer would be no . . . . It does not lend itself to adequate profit margins."

Consequently, OCF did not continue trying either to improve or to sell Multitemp. A later OCF memorandum confessed that, despite the company's official position that Multitemp had been discontinued because distributors did not want to handle both Multitemp and Kaylo, "the real reason for dropping Multi Temp was low gross margins."

Even after the publication of Dr. Selikoff's study in 1965 created an awareness of the potential health hazards associated with asbestos-containing products, OCF placed a low priority on replacing the asbestos in Kaylo. Instead, OCF's research and development efforts respecting Kaylo emphasized a program for reducing stress corrosion in order to enable Kaylo to gain an economic advantage in the emerging nuclear industry. As summarized in a 1967 memorandum by the head of

OCF's Kaylo research laboratory, OCF's marketing department kept the Kaylo asbestos-replacement research in "low gear" because "[m]ost of our effort is being directed toward stress corrosion." In fact, OCF's immediate reaction to Dr. Selikoff's study was to try to impugn his reputation and to try "to find some way of preventing Dr. Selikoff from creating problems and affecting sales."

(e) OCF's Inadequate Warning Labels. Plaintiffs also successfully argue that, even with the release of Dr. Selikoff's study in 1965, it was several years before OCF grudgingly placed warning labels on Kaylo. OCF delayed despite the fact that other manufacturers, such as Johns-Manville, had labelled their products immediately upon learning of Dr. Selikoff's results. Indeed, according to plaintiffs, OCF was the last insulation manufacturer to do so.

Moreover, the label that OCF ultimately utilized is alleged to have been inadequate. Plaintiffs contend that the OCF label did not include words of warning such as "caution," "hazardous," or "dangerous", and it was located on Kaylo boxes so as to be unreadable when the boxes were opened; it was smaller than originally proposed; and one of OCF's own vice presidents testified that OCF's label was so "vague" that "somebody with a Ph.D would have difficulty understanding" what it was supposed to mean.

The foregoing recitation of the facts developed in the tort litigation is only illustrative. Plaintiffs also point to a number of other actions by OCF that demonstrate

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willful and wanton misconduct. These actions show both a disregard for the health of those who manufactured or used asbestos-containing Kaylo and a desire to cover-up knowledge respecting the potential health hazards associated with asbestos. For example:

- In 1967, OCF forced a college student who had spent a summer working at OCF to delete from the paper he wrote about his experience references to asbestos as causing lung cancer because OCF claimed that this was "extremely proprietary" business information.

- Even after OCF had developed an asbestos substitute for Kaylo in 1971, it continued to fill orders for Kaylo products with both asbestos-containing Kaylo and asbestos-free Kaylo to avoid throwing out its inventory of asbestos-containing Kaylo and yet also to be able to claim that it was selling an asbestos-free product.

- A decade after OCF started manufacturing asbestos-free Kaylo, tests showed that this allegedly asbestos-free Kaylo actually contained small percentages of asbestos, a fact (among others) that was recently used by one asbestos claimant to obtain a $15 million punitive damage verdict against OCF.

- Even more recently, OCF's Chairman and CEO stated at the 1992 annual OCF shareholders meeting that "We put the asbestos issue behind us. . . [OCF is] not shedding tears about the past." At a recent trial, plaintiffs utilized this statement in obtaining a $54.6 million punitive damage verdict.

Reviewing only some of the foregoing evidence, the United States Court of Appeals for the Third Circuit recently rejected OCF's claim that punitive damages against it were not warranted. The court, sitting en banc, sustained the jury's finding that OCF had acted with "'reckless indifference' in failing to place adequate warnings on its Kaylo product." Dunn v. Hovic, 1 F.3d 1371, 1374 (3d Cir. 1993) (en banc), cert. denied, 114 S. Ct. 650 (1993).

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While Owens-Illinois does not here endorse the arguments and conclusions plaintiffs draw from OCF's history of asbestos-related products, it is beyond dispute that juries and courts across the country have accepted this proof as the basis for compensatory and punitive verdicts against OCF. The amounts that OCF has paid and will continue to pay to resolve cases are predicated upon these allegations of actual knowledge and independent corporate misconduct, not the mere resale of a product made by Owens-Illinois. At a minimum, OCF's tort liability -- even in cases involving some pre-1958 Kaylo exposure -- is inextricably intertwined with OCF's independent acts and omissions in the post-1958 period. OCF's conduct after it purchased the Kaylo Division in 1958 bars its present claim for indemnity as a matter of law, equity, and public policy.

C. OCF's Fraudulent Concealment Of Its Asbestos History

According to the tort plaintiffs' proof, OCF's attempts to hide the health hazards associated with asbestos did not end when OCF stopped putting asbestos in Kaylo in 1972. With the explosion in asbestos litigation, OCF embarked on what courts have concluded was an effort to conceal fraudulently what it historically knew respecting the hazards of asbestos. For example:

- In 1980, an OCF attorney found records of workers' compensation claims involving OCF that dated back to the 1950's, contradicting interrogatory answers that OCF had been certifying as true. The attorney concluded that OCF had to correct its interrogatory

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answers. OCF did not do so and, indeed, continued denying the existence of such claims, even up to the United States Supreme Court. This misconduct was only discovered in 1989. One court recently characterized OCF's cover-up of the relevant documents as a "deliberate and intentional fraud upon the courts." Pickering v. Owens-Corning Fiberglas Corp., No. 90-L-1546, Order dated April 7, 1992 (Ill. 3rd Jur. Cir.), aff'd No. 5-92-0691 (Ill. App. Ct. 5th Dist. May 4, 1994). See also Owens-Corning Fiberglas Corp. v. Watson, 243 Va. 128, 142, 413 S.E.2d 630, 639 (1992) (concluding that by virtue of an interrogatory answer that was "patently false" OCF had "committed a fraud" upon a federal court).

- Another judge also recently ruled that two OCF documents (one related to the OCF discovery of the workers' compensation records just mentioned) represented "clear evidence of [OCF's] continuing conspiracy to defraud claimants as well as courts with respect to the knowledge of the defendant concerning the effects of asbestos, asbestos-related diseases and facts specifically relating thereto." Hanna v. Owens-Corning Fiberglas Corp., No. L-05154-90, slip op. (N.J. Super. Ct., Camden Co. Dec. 3. 1993).

- OCF's failure to produce four corporate witnesses for examination at trial was recently sanctioned and characterized by an appellate court as "deliberate, contumacious and an unwarranted disregard of the court's authority." Pickering v. Owens-Corning Fiberglas Corp., No. 5-92-0691, slip op. at 22 (Ill App. Ct. 5th Dist. May 3, 1994).

- Yet another court recently concluded that "OCF, with assistance of counsel, knowingly, intentionally and in bad faith filed false and evasive answers"; that OCF and its counsel "have knowingly, willfully, intentionally, in flagrant bad faith and callous disregard disobeyed" a court discovery order; that OCF's conduct and that of its counsel are part of OCF's "pattern of obstructing justice in the asbestos cases"; that OCF has a "history of committing acts of fraud in connection with discovery proceedings"; that OCF has "a national network of lawyers to obstruct justice in asbestos cases"; and that "OCF has routinely used counsel for the purpose of carrying out conduct that, which if not criminal, borders on being criminally culpable". Bodine v. Owens-Corning Fiberglas, No. 92-C-2440, slip op. (June 11, 1993 23rd Jud. Dist., Brazoria

Cp. (mand), petition for writ of mandamus pending, No. 01-98-00512-CV [ct. App. 1st Sup. Jud. Dist. Houston].[10]

In light of the foregoing history of OCF's misconduct concerning Kaylo and the very litigation which gives rise to the present indemnity claim, OCF is legally and equitably barred from receiving the relief it seeks. The foregoing history also provides the basis for several of the counterclaims set forth in Point III below.

C. Other Separate and Independent Bars to OCF's Claim

There are still other separate and independent reasons why OCF's demand should be rejected.

First, OCF's claim is barred by the doctrines of laches and estoppel. OCF unfairly delayed bringing this proceeding and this delay has prejudiced Owens-Illinois. Even assuming OCF had a valid claim for indemnification for personal injury lawsuits under the 1958 Agreement, had OCF not delayed in demanding the indemnity until now -- when OCF has paid over a billion dollars in settlements, when OCF has repeatedly been held liable for punitive damages, and when OCF has repeatedly been sanctioned for litigation abuse -- Owens-Illinois would have handled the OCF asbestos litigation defense differently and minimized the amounts OCF now demands in indemnity from Owens-Illinois.

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[10] In words equally applicable to the torturous logic by which OCF is attempting to press the present indemnity claim the court also found that "OCF and its lawyers have attempted to engage in semantic gymnastics." Id.

Second, the various claims for which OCF is demanding indemnification allege not only breach of warranty but also negligence; malicious, willful, wanton and reckless conduct or gross negligence; strict liability; and violation of state trade practice acts (see, e.g., Exhibits 5 and 6 (Tighe and Cardile complaints)). The amounts paid by OCF in settlement or judgment resolved all claims under all of these theories of liability and represent payment of one indivisible amount to discharge all claims. OCF will not be able to provide the Panel with reliable evidence as to the actual portion of the settlements, if any, that is solely attributable to the alleged breach of warranty claims.

Third, OCF cannot obtain -- as it seeks -- a declaratory judgment giving it a blanket indemnity for "all obligations" that arise "in connection with any asbestos personal injury claim" in which "the injury is alleged to result from the injured person's exposure to Kaylo delivered prior to May 1, 1958." (Exhibit 2 ¶ (2)). As a matter of applicable Ohio law, the statute of limitations for asserting such a blanket indemnity lapsed no later than 15 years after the first assertion of such a personal injury claim against OCF.

Finally, and in all events, the indemnity provision in the 1958 Agreement upon which OCF bases the present claim does not provide any indemnity for Kaylo that OCF delivered after May 1, 1958. Yet OCF is making just such a claim. Mr. Cardile who "was exposed to Kaylo during the period from 1953

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to 1974" (Exhibit 3). Mr. Baggart was "exposed to Kaylo from 1953 to 1972" (Exhibit 2 ¶ 1(b)). Post-May 1, 1958 Kaylo exposures are also alleged in innumerable other claims encompassed by OCF's demand for a declaratory judgment (Exhibit 2 ¶ 2). OCF will not be able to provide the Panel with reliable evidence as to the actual portion of these claims, if any, that is solely attributable to pre-May 1, 1958 deliveries as opposed to post-May 1, 1958 deliveries of Kaylo.

III.

OWENS-ILLINOIS' COUNTERCLAIMS

As demonstrated above, not only is OCF's claim for indemnity from Owens-Illinois without legal or equitable merit, but if any party should be entitled to relief in this proceeding it is Owens-Illinois and not OCF. Accordingly, Owens-Illinois asserts the following counterclaims against OCF:

1. For a declaratory judgment that Owens-Illinois owes no obligation to indemnify OCF for injuries to third persons claimed to result from the use of Kaylo, either under the 1958 Agreement transferring the assets of the Kaylo division, under the 1953 Sales Contract, under any federal or state statute, or under the common law principles of any state, including under theories of common law or statutory contribution.

2. For the amounts it has had to pay in legal fees and by judgment or in settlement for claims against it

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asserting injury from exposure to Kaylo between 1963 and 1958, an obligation owed by OCF to Owens-Illinois under ¶ 5(e) of the 1953 Sales Contract in which OCF agreed that Owens-Illinois "shall not be liable for any breach of this Agreement in any amount in excess of the agreement price for the products with respect to which such breach occurs," that Owens-Illinois "shall not be liable in any event for special or consequential damages", and that OCF "shall include this same limitation upon the amount of [Owens-Illinois'] liabilities in contracts effecting all resales by [OCF] to third parties and [OCF] shall indemnify and save [Owens-Illinois] harmless from any liabilities arising from [OCF's] failure so to contract in making resales."

3. For common law indemnity, or in the alternative for contribution, for the amounts Owens-Illinois has had to pay in legal fees and by judgment or in settlement of asbestos-related personal injury claims against it by virtue of OCF's wrongful acts following its acquisition of the Kaylo division in 1958, including, without limitation, (a) changing the composition of Kaylo so as to make it dustier and hence more harmful; (b) not utilizing means it had to reduce the dustiness of Kaylo because the means would have diminished OCF's profits from the manufacture and sale of Kaylo; (c) not replacing Kaylo with the alternative asbestos-free product OCF had developed, again because the product would have been less

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profitable; (d) not placing warning labels on Kaylo for several years after other asbestos-producers had placed labels on their products, and even then placing a warning label on Kaylo that was, by OCF's own admission, "vague" and would have required a graduate degree to understand; (d) by committing frauds upon courts in the 1960's as part of its defense to asbestos-related personal injury claims, and (e) the use of rebranding agreements that caused other companies' more harmful asbestos products to be sold as Kaylo. These acts and frauds, particularly in the light of Owens-Illinois' entirely passive conduct respecting Kaylo after 1958, have increased Owens-Illinois' litigation exposure and costs in ways such as by (i) increasing -- rather than minimizing -- Kaylo users' exposure to asbestos-containing dust thereby either increasing the likelihood that such users would develop an asbestos-related impairment or disease or increasing the severity of the asbestos-related impairment or disease suffered by those users, (ii) shifting to Owens-Illinois liabilities which, in actuality, should be borne by OCF, (iii) making Kaylo a more prominent target for plaintiffs' lawyers in the underlying asbestos personal injury actions, and (iv) causing Owens-Illinois to be sued in cases alleging harms to Kaylo users after 1958 even though Owens-Illinois is not legally responsible for those harms, thereby

increasing the number of claims against, and the amounts of damages demanded from, Owens-Illinois.

4. For the costs, including attorneys' fees, Owens-Illinois has incurred in defending against OCF's meritless claim for indemnity in this proceeding.

The value of the counterclaims set forth above is well in excess of one million dollars, and in an amount to be determined in this proceeding.

Date: June 24, 1994

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AMERICAN ARBITRATION ASSOCIATION

In the Matter of the Arbitration

Between

OWENS-CORNING FIBERGLAS CORPORATION

Claimant,

-and-

OWENS-ILLINOIS, INC.

Respondent.

No. 13Y 1990095393

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875 Third Avenue
New York, New York
May 11, 1995
11:00 a.m.

B E F O R E:

EUGENE D. McGAHREN, JR., ESQ., Chairman

J. WARREN WOOD, III, ESQ.,

RICHARD K. JEYDEL, ESQ.,

Panel

JACK FINZ, C.S.R., Hearing Reporter

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A P P E A R A N C E S (Continued):

ALSO PRESENT:

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Owens-Illinois, Inc.

THOMAS YOUNG,
Owens-Illinois, Inc.

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CHAIRMAN McGAHREN: For purposes of the court record, this is the hearing set for May 11, 1995. We have had a number of preliminary hearings. I guess we could qualify this as the beginning of the hearings. And this would be the first hearing after the preliminary hearings that we have held.

The hearing is starting at 11 o'clock, and the claimant's case will be presented.

We have received a motion here that indicates it would be presented for entry of a partial award at the panel hearing today, and I don't know how the parties want to handle that, the opening statements of the claimant, or do you want to start addressing the motion of Owens-Illinois?

MR. LIMAN: Can I say something about it in the opening statement?

CHAIRMAN McGAHREN: In the opening?

MR. LIMAN: Right.

CHAIRMAN McGAHREN: Fine. Then why don't we commence the hearing, the opening statements by claimant, and Mr. Arthur Liman

would be, I suppose, giving the opening statement, from what I'm hearing.

MR. LIMAN: I will begin the opening statement. My colleague will have something to add.

CHAIRMAN McGAHREN: Just one more minute. One of the arbitrators has a disclosure note to make.

MR. JEYDEL: Which I think has previously been submitted to everyone through the AAA, but seeing such prominent mention made in one of the briefs of Andrew Barry's name, I thought it best to repeat that I was now nearly 20 years ago an associate of McCarter & English. I knew Andrew Barry. I didn't work for him then, bumped into him in court numerous times since then.

And I was able to determine that at one point my present employer, I guess 15, 16 years ago, did have a project in the Soviet Un: with Owens-Illinois, on which I did work.

I felt it incumbent upon me to rep-: these disclosures. I don't think it will influence me one way or the other, but you

certainly have the right, and perhaps before we start investing a lot of time in this, perhaps a duty with respect to your client to consider that and decide whether you want to have the panel reconstituted.

MR. LIMAN: We have no problem with that whatsoever.

MR. KING: We have no problem.

CHAIRMAN McGAHREN: It seems to me that the parties have no problem with the disclosure. And in view of the time lapse, and the circumstances, it is clear that the parties feel this will not affect your decision.

Why don't we commence? Any other housekeeping details?

MR. LIMAN: No. And let me say that, you know, we really are appreciative of your willingness to serve. Far from wanting you not to serve, we really welcome the fact that you took on this responsibility, which, if you look at the exhibit books behind you, you get a sense that it is not necessarily the easiest of assignments that you have undertaken.

Our case, which I will outline -- I

the allegations of the plaintiff were not safe, and caused a variety of asbestos-related diseases, including cancer.

And that warranty allegation, that allegation that you have represented as a matter of law that your product is safe, is the cornerstone of all of these actions, whether they are denominated strict liability or, as in most of the cases, implied warranty.

And the third and final part of our proof is that OCF is about to suffer, in a material sense, the harm from which it was to be saved by reason of the O-I shipments during this '53 to '58 period. Until recently, most, though not all of the costs that OCF had to bear, were covered by and reimbursed by insurance. The insurance is running out, and we are now in a situation in which the client has no choice but to go to where the problem was caused, because there is nobody else who will bear these costs, and they are going to fall entirely on OCF.

So that's the three elements of the case. The background of the case, the relationship between the parties and how the

contract came into being, I think are not a matter of dispute. I can take only a few minutes just to refresh you on them and to highlight it.

OCF was founded in the 1930s as a joint venture between Owens-Illinois and Corning Glass. Its purpose was to manufacture and sell fiberglass. It continued in that joint ownership situation until the 1950s, when the Antitrust Division obtained a consent decree that required the two parents to divest themselves of their interest. In fact, Owens-Illinois continued with nonvoting interest of some size, 20 percent, into the 1970s, but it was no longer able to have control after the antitrust decree.

The antitrust decree, of course, could preclude Owens-Illinois from having voting stock or exercising control. It could not eliminate the filial feelings, is the only way I could describe them, that OCF had toward its former parent, O-I, and that is important in understanding the actions here. O-I is the dominant company in Toledo, and that's where OCF is also located. The management, first management of OCF, many of the employees and

executives, even during the period that this agreement was negotiated, executed, came from O-I. They mixed socially, and in professional organizations. And the relationship was a close one, and you will see through various witnesses that O-I was always looked up to as a role model in the community.

And I think it was as difficult for OCF to launch into this type of arbitration, as long as there was insurance against its former parent, as it would be for one member of a family to sue another member of a family. It's just not the type of thing that is done in Toledo. And this arbitration was undertaken with some reluctance. And we will have something to say about that, and the proof involved.

The product involved here is Kaylo. It was developed in the 1940s by O-I from a foreign patent that it acquired. It is an asbestos-based insulating material that is particularly suited for high temperature applications. In 1953 O-I entered into two sales agreements with OCF, under which OCF served as a nonexclusive distributor of Kaylo for O-I. That

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AMERICAN ARBITRATION ASSOCIATION

Owens-Corning Fiberglas Corporation :

v. :

Owens-Illinois, Inc. :

No. 13 Y 199 00953 93

OWENS-ILLINOIS' PRE-HEARING MEMORANDUM

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INTRODUCTION

Owens-Illinois, Inc. ("Owens-Illinois") submits this Pre-Hearing Memorandum setting forth the facts it intends to prove at the hearing, the construction and interpretation of the contracts at issue, and the legal authorities applicable to the claims asserted by Owens-Corning Fiberglas Corporation ("OCF") and by the defenses and counterclaims asserted by Owens-Illinois.[1]

SUMMARY OF ARGUMENT

This arbitration is a belated attempt by OCF to reallocate retroactively to Owens-Illinois some undefined portion of hundreds of millions of dollars of tort liability arising from OCF's forty-year involvement with asbestos-containing products. The alleged right to indemnity relates to Kaylo products manufactured by Owens-Illinois and sold by OCF from 1953 to 1958.

OCF does not purport to find this indemnity right in the terms of the two 1953 contracts pursuant to which those Kaylo products were sold (hereinafter referred to as "the 1953 Master Product Sale Contract"), and for good reason -- the 1953 Master Product Sale Contract disclaims any such indemnity right. Rather, OCF relies upon a single clause in a single sentence in the 1958 Asset Sale Agreement whereby OCF acquired the assets necessary to become both the manufacturer and seller of Kaylo. As explained below, there is no legal or factual basis for this claim, and it should be denied by the Panel.

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[1] This Pre-Hearing Memorandum reflects the results of discovery and analysis to date and, to the extent necessary, supplements, amends, or supersedes prior submissions by Owens-Illinois.

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From 1953 to 1958, OCF purchased and resold to commercial customers asbestos-containing Kaylo pipe and block insulation products manufactured by Owens-Illinois. Owens-Illinois provided these products to OCF under the general terms of the 1953 Master Product Sale Contract which established the parameters and legal responsibilities of the parties with respect to sales to OCF under individual purchase orders placed by OCF.

The 1953 Master Product Sale Contract specifically provided that Owens-Illinois would not be responsible for "special" or "consequential" loss arising from OCF's resale of these products, limited Owens-Illinois' liability to the price of the goods sold, and imposed a one-year time limitation on claims. OCF agreed in the 1953 Master Product Sale Contract that it had no recourse to Owens-Illinois for claims of third party end users and bystanders arising out of OCF's resales to commercial customers.

In 1958, OCF purchased the assets of the Kaylo operation from Owens-Illinois. The 1958 Asset Sale Agreement did not make any reference to the 1953 Master Product Sale Contract. It did not modify, alter or expand the limitations and rights carefully set out in detail in 1953. It did not retroactively reverse the risk allocation established by the 1953 Master Product Sale Contract. Nor for the next three decades did OCF ever assert that it did. Indeed, from the 1960's when OCF received its first asbestos claims, until the 1990's and the commencement of this arbitration, OCF never asserted that the 1958 Asset Sale Agreement modified the contractual obligations and limitations set forth in the 1953 Master Product Sale Contract.

Now, for reasons that it has not yet adequately explained, OCF has reversed course, seeking a declaratory judgment entitling it to indemnification for its

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liability to asbestos claimants who allege exposure to Kaylo products sold by OCF prior to May 1, 1958. After receiving more than 250,000 bodily injury claims from asbestos claimants, after resolving more than 140,000 of such claims, after sustaining more than $400 million in compensatory and punitive damage findings and verdicts, and after spending more than $2.0 billion in defending and resolving such claims, OCF is asking this Panel retroactively to transfer its massive tort liability to Owens-Illinois.

OCF contends that the retroactive reversal of risk was accomplished by one clause in paragraph 6 of the 1958 Asset Sale Agreement, the purpose of which was to assign executory contracts of the Kaylo Division to OCF. Paragraph 6 had nothing to do with sales to OCF, which are governed by the 1953 Master Product Sale Contract. Had the parties in 1958 intended to modify the 1953 Master Product Sale Contract which governed from 1953 to 1958, they had ample opportunity to do so, but did not. Had the parties in 1958 intended to include the concept of negligence or tort liability for personal injury within the warranty language of paragraph 6, they could have done so, but did not. That they did not is particularity compelling here since the law in Ohio at the time did not recognize personal injury tort warranty claims in situations like this. Careful lawyers, and there were such on both sides of the transaction at the time, would have been explicit if there were any intention to include anything beyond pre-existing commercial warranties that Owens-Illinois had made relating to performance specifications.

OCF's conduct, in numerous ways that will be proved at trial and which is summarized below, throughout the entire period -- 35-40 years -- was entirely consistent with the proper interpretation of the 1953 and 1958 contracts. It would defy both

contract law and common sense to permit OCF to shift enormous liabilities in a manner never contemplated in 1958.

OCF's failure to provide timely notice of its purported indemnity claim and its affirmative representations that it had no such claim have irreparably prejudiced Owens-Illinois and waived any such indemnity right even if it ever existed. OCF represented to Owens-Illinois in 1975 that each company should take care of its own lawsuits. OCF acknowledged to Owens-Illinois in 1979 that OCF had no claim under the 1958 Asset Sale Agreement. OCF has attempted in other litigations to take advantage of positions inconsistent with its present claim. These acts, together with OCF's own culpable conduct both before and after it purchased the Kaylo division, discharge any obligation of Owens-Illinois and bar and estop OCF under the law of indemnity, laches, equitable estoppel, judicial estoppel, and waiver.

Finally, OCF cannot prove any of the preconditions to reimbursement under the alleged indemnity. OCF cannot prove that it paid, let alone what it paid, plaintiffs due to Kaylo exposures within the asserted scope of the indemnity, or that OCF was "actually liable" to such plaintiffs. From the 1940's through 1970's, OCF made, sold and/or installed (through its insulation contracting business) non-Kaylo asbestos-containing products. It is a fact of the asbestos litigation that the plaintiffs' alleged Kaylo exposure almost invariably extends beyond 1958. Plaintiffs allege exposure to dozens of products other than Kaylo manufactured by at least twenty other companies, and plaintiffs' claims are based on theories of liability other than breach of "warranty". OCF's liability is based upon evidence of OCF's independent misconduct from 1941 into the 1980's, not its supposed role as a passive distributor of Owens-Illinois' Kaylo from

[redacted] OCF cannot prove the actual damages amount received by [redacted] plaintiff or the portion of any such amount received by any plaintiff attributable to a breach of warranty cause of action, as distinct from the damages attributable to other causes of action.

ISSUES PRESENTED

This Arbitration presents the Panel with the following fundamental questions:

1. Can the plain language of the 1953 Master Product Sale Contract -- pursuant to which all of the sales that are the subject of the arbitration took place and which expressly disclaimed the indemnity rights now asserted by OCF -- be retroactively reversed by the terms of the 1958 asset sale when the terms of the 1958 Asset Sale Agreement and the consistent conduct of the parties show that neither party contemplated or intended the massive shift of tort liability now sought by OCF?

2. Will the Panel engage in a detailed retrospective analysis of decades old commercial contracts for the purpose of reallocating the respective tort liabilities of two companies that over twenty years consciously had decided to resolve tens of thousands of those tort claims independently and without recourse to the indemnity rights asserted here, or, has the right to seek such a retroactive reallocation been waived?

3. Is OCF barred from asserting this claim on grounds of estoppel, laches, and its own egregious misconduct?

[redacted] Is there any practical or feasible method [redacted] to allocate hundreds of millions of dollars in tort liability arising from tens of thousands of individual claims where it is indisputable that OCF's tort liability is also (if not exclusively) based upon exposure to other OCF asbestos products (including Kaylo manufactured after 1958) and evidence of its own independent misconduct, and arises under tort theories (such as failure to warn and negligence) unrelated to its alleged right to indemnity for "breach of warranty"?

If any one of these four issues is resolved in Owens-Illinois' favor, then OCF is not entitled to the relief it seeks. Under the facts and law Owens-Illinois will present at this hearing, however, it is plain that all issues should be answered in its favor.

STATEMENT OF FACTS

Owens-Illinois developed Kaylo insulation from a German patent that used the same basic materials as did glass manufacture -- lime and silica. Owens-Illinois purchased the patent, refined it, and began selling insulation products on a commercial basis in 1948. Prior to commercially marketing Kaylo, Owens-Illinois had the health effects of Kaylo tested by a leading laboratory, the Saranac Laboratory in Saranac Lake, New York. The Saranac Laboratory research led Owens-Illinois to conclude, in the words of its industrial hygienist, that in expected use "[t]he product was safe and the dust from the product was safe."[1] As OCF itself has stated, "[t]he Saranac industrial hygiene study represents the state of the art for industrial health through the 1950's."

[1] The testimony and court papers quoted herein can be provided to the Panel upon request. A few particularly significant documents and cited legal authorities ("Ex.") are included in the accompanying exhibit volume. We can also provide copies of any or all other cited legal authorities that the Panel might want.

A. Background

In 1948, Owens-Illinois began the commercial manufacture and sale of a number of Kaylo products -- premolded heat insulation pipe covering, heat insulation block, firedoor core material, roof tile, laminated panels, and other specialty products. These products contained small amounts of asbestos and were produced at Owens-Illinois' plants in Berlin and Sayreville, New Jersey. In 1948, as today, Owens-Illinois was primarily a glass container company; Owens-Illinois had no experience in the sale of insulation or other construction materials. By 1952 it had become apparent to Owens-Illinois that the Kaylo product line was not profitable. In 1953, Owens-Illinois turned to OCF, which had substantial experience in the business of manufacturing and installing heat insulation products, to sell some Kaylo products. In 1958, Owens-Illinois sold the entire Kaylo business to OCF.

OCF's own manufacture and sale of asbestos-containing insulation products, however, predates the development of Kaylo. Beginning in 1938, the very year in which it was formed, OCF sold asbestos paper facing and asbestos tie yarn, which were used in conjunction with insulation blankets. In 1940, OCF began manufacturing and selling asbestos-containing insulating cement. In that same year it began selling an asbestos-containing mastic used as a protective finish for pipe and boiler insulation. In the early 1950's OCF sold Unibestos-brand pipe covering and, at the same time, expanded its role as a manufacturer and/or seller of asbestos-containing insulation products by acquiring an insulation contracting unit. From the 1950's onward, OCF's insulator employees applied a full range of asbestos-containing products including, but not limited to, Kaylo.

Almost immediately upon acquiring the capability to manufacture Kaylo in 1958, OCF entered into rebranding agreements with other manufacturers pursuant to which OCF bought the asbestos-containing products of others (e.g., Johns-Manville) and resold them under the OCF Kaylo label. While OCF claims to have removed the asbestos from Kaylo by 1973, its own former employee has testified that asbestos was included in Kaylo into the 1980's.

B. The 1953 Master Product Sale Contract

On March 20, 1953 the parties entered into the 1953 Master Product Sale Contract. OCF agreed to purchase annually during the term of the contract aggregate dollar amounts of Kaylo pipe and block insulation products. The 1953 Master Product Sale Contract listed the various types of Kaylo pipe and block insulation products which OCF could purchase in order to satisfy its annual dollar aggregate. Ex. 1 ¶ 2 & Ex. A. It referred to them as the "Heat Insulating Products". Ex. 1 ¶ 2. Owens-Illinois' Kaylo products other than pipe and block insulation, and associated accessories, were specifically excluded from the 1953 Master Product Sale Contract. Ex. 1 ¶¶ 2, 8.

The 1953 Master Product Sale Contract contained all the general terms that would be applicable to the long-term business relationship established by the parties and general contract terms that would be part of every OCF purchase order for every Heat Insulating Product. It dealt with subjects ranging from the responsibility for shipping costs to restrictions on OCF's use of Owens-Illinois' Kaylo trade name and trademark in advertising and sales. The 1953 Master Product Sale Contract contemplated that purchase orders from OCF for specific pipe and block products would be placed with,

and the acceptance of such orders would be given by Owens-Illinois. The parties' rights and liabilities with respect to particular goods sold would be established in these specific sales contracts made in the usual commercial manner by exchange of purchase orders and acceptances. Ex. 1 ¶ 5 ("[a]ll orders for Kaylo Heat Insulating Products placed by Buyer and accepted by Seller will be at the prices in effect at the time of shipment by Seller and will be subject to the terms set forth in Exhibit A and the following terms and conditions ...."). These individual sales contracts would set forth the type, amount, and price of the particular Kaylo product sold (e.g., 1-1/2" pipe), the delivery dates and any other terms. The individual sales contracts would contain or refer to the warranties made by Owens-Illinois for the particular product being sold (i.e., the "performance specifications"), which the parties understood would change from time to time and from product to product. Ex. 1 ¶ 5(d).

The 1953 Master Product Sale Contract provided that Owens-Illinois only warranted that the Kaylo Heat Insulating Products sold to OCF would meet performance specifications (e.g., thermal conductivity "K" factors and dimensional stability). Ex. 1 ¶ 5(d). All other warranties, express or implied, were specifically disclaimed (¶ 17), a disclaimer then-valid under Ohio law. See Ohio Gen. C. § 1315.72. Should the Kaylo not comply with performance specifications, Owens-Illinois' liability to OCF was expressly limited to the price OCF paid for the Kaylo. Ex. 1 ¶ 5(d).

The 1953 Master Product Sale Contract provided that "in any event" Owens-Illinois would not be liable for "special or consequential damages". Id. In 1953, "consequential damages" meant "damage, loss, or injury as does not flow directly or immediately from the act of the party, but only from some of the consequences or results

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of contract" or "damage which does not follow immediately, in point of time, upon the doing of the act complained of." Black's Law Dictionary 467 (4th ed. 1951). In other words, Owens-Illinois was not to be liable to OCF for any indirect economic loss or harm such as from personal injury.[3]

In a clear indication of the parties' intention to allocate the risk of liabilities for Heat Insulating Products to OCF, as the ultimate marketer and seller of them, the 1953 Master Product Sale Contract obligated OCF to include a damage limitation and a disclaimer of special or consequential damages in favor of Owens-Illinois in OCF's resale contracts and also provided an indemnity for OCF's failure to do so:

> "[OCF] shall include this same limitation upon the amount of [Owens-Illinois'] liabilities in contracts effecting all resales by OCF to third persons and OCF shall indemnify and save [Owens-Illinois] harmless from any liabilities arising from [OCF's] failure so to contract in making resales." Ex. 1 ¶ 5(e).

Thus, the 1953 Master Product Sale Contract specifically addressed third-party claims and exempted Owens-Illinois from any liability to OCF for any such damages that might arise from the Kaylo products sold by OCF.[4]

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[3] A decision rendered that same year, Maryland Cas. Co. v. Owens-Illinois Glass Co., 116 F. Supp. 122 (S.D. W. Va. 1953), involving identical language from an Owens-Illinois sales contract, demonstrates the operation of this contractual limitation. Owens-Illinois manufactured and sold glass soft drink containers to a bottler under a contract that provided Owens-Illinois would not "be liable in any event for ... special or consequential damages of any kind." After the bottler filled a bottle with soft drink and sold it, the bottle broke, injuring an individual. The bottler's liability insurer paid the individual for his injuries, and then the insurer, as subrogee of the bottler's rights under the liability insurance policy, sued Owens-Illinois. The court dismissed, ruling that the above-quoted language precluded the bottler (and its insurer as subrogee) from recovering amounts paid to the individual for personal injuries.

[4] The 1953 Master Product Sale Contract contains other provisions limiting Owens-Illinois' liability. It required OCF to give Owens-Illinois written notice of any "errors, shortages, imperfections, deficiencies or any failure of the products to

c. Owens-Illinois Sales: 1953-1958

As required by the 1958 Asset Sale Agreement (Ex. 3 ¶ 7), Owens-Illinois turned over to OCF documents relating to the business of the Kaylo division. The sales contracts, including purchase orders, acceptances and invoices, were included in the documents given to OCF. All those documents were later destroyed by OCF. Because OCF destroyed the sales contracts relating to individual sales, OCF cannot establish what performance specification warranties were in fact made for a particular Kaylo Heat Insulating Product. For the same reason it cannot be proven today whether OCF complied with its obligation to include in its resale contracts the protections of Owens-Illinois that the 1953 Master Product Sale Contract required. Nevertheless, the protective provisions disclaiming warranties and limiting damages apply between the parties with the same effect as if proven from individual sales contracts which OCF destroyed. Obviously, virtually all of the Heat Insulating Product sales from 1953 to 1958 under the 1953 Master Product Sale Contract were fully completed before May 1958 -- the goods had been delivered, payment had been made, and any claims by OCF relating to the goods had been resolved.

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conform with the terms of this Agreement" within 90 days after shipment. Ex 1. ¶ 5(e). The failure of OCF to give timely notice to Owens-Illinois "constitute[d] a waiver by [OCF] of all claims with respect thereto." Id. (emphasis supplied). OCF has no proof that it gave such notice concerning the products Owens-Illinois sold to it.

Finally, the 1953 Master Product Sale Contract provides that "[a]ny civil action against [Owens-Illinois] ... by reason of any sale hereunder ... shall be commenced within one (1) year from the date such cause of action arises." Ex. 1 ¶ 15. If, therefore, the 1953 Master Product Sale Contract had provided an indemnity to OCF covering third-party claims (but not for special or consequential damages), OCF would have had to bring an indemnity claim within one year.

[redacted] Owens-Illinois sold OCF [redacted] approximately $1 million worth of Kaylo Heat Insulating Products. By 1958 OCF had become the primary, if not only, purchaser of Kaylo Heat Insulating Products. However, from 1953 to 1958 Owens-Illinois continued to sell other Kaylo products directly to other companies. These products included firedoor cores, roof tile, laminated panels, and other specialty products. As of May 1, 1958, the Kaylo division had outstanding direct supply commitments to Western Electric, Chrysler Corporation, and Owens-Illinois Plywood Company. Even with OCF's expertise in handling the marketing and selling of Kaylo Heat Insulating Products, Owens-Illinois did not make a profit on Kaylo until 1955. From 1953 onwards, OCF had desired to purchase the entire Kaylo business. The sale was accomplished by the 1958 Asset Sale Agreement.

D. The 1958 Asset Sale Agreement

The Kaylo operation had been conducted through a division of Owens-Illinois, not through a separate subsidiary. Thus, the sale was structured as a sale of the assets of the Kaylo operation as of the close of business on April 30. Paragraph 6, like paragraphs 2, 3, 4, and 5, is primarily a conveyancing provision. Its primary purpose is to convey the executory contracts that will be of benefit to OCF. The only contracts of benefit to OCF are those contracts Owens-Illinois, as assignor, had with third parties. The 1958 Asset Sale Agreement set forth general, practical mechanisms so that when OCF finally took full possession and control of the facilities, the responsibilities and liabilities of the parties would be clear. As we shall show below in Point I, paragraph 6

was designed to accomplish this purpose and cannot be interpreted to transfer, convey, or vest a contract between Owens-Illinois and OCF.

B. OCF's Three-Decade Course of Conduct Inconsistent with an Indemnity for Asbestos Claims

During the entire time that Owens-Illinois manufactured Kaylo, Owens-Illinois never received a personal injury claim of any sort involving the health effects of the asbestos in Kaylo. Beginning in the 1960's, OCF began receiving asbestos personal injury claims and recognized that more claims could follow.[5] The individuals who negotiated and signed the 1958 Asset Sale Agreement for OCF were still alive then, but neither they nor anyone else at OCF notified Owens-Illinois of those claims or requested indemnification from Owens-Illinois.

Even as asbestos claims against OCF began to accumulate, OCF did not notify Owens-Illinois or assert any right to indemnification under the 1958 Asset Sale Agreement. Reasonable company officers who believed their company was entitled to an indemnity had a fiduciary obligation to shareholders to assert that right rather than incur any loss, even insurance deductibles or self-insured retentions. Still OCF stood silent.

Approximately 100 such claims were pending against OCF in 1975. OCF recognized that such claims "resulted in extraordinarily high compensation payment[s]." The OCF drafters of the 1958 Asset Sale Agreement were still alive, and the "tort revolution" liberalizing the theories under which tort damages could be recovered had

[5] This fact, as with many others concerning OCF, did not come to Owens-Illinois' attention until many years later.

already occurred. OCF never gave notice to Owens-Illinois of demand for indemnity upon Owens-Illinois.

In 1975, Owens-Illinois received its first asbestos-related personal injury lawsuit. Owens-Illinois' General Counsel and another Owens-Illinois attorney, David Ward, met with Carl Staelin, OCF's general counsel who had been an OCF signatory to both the 1953 Master Product Sale Contract and the 1958 Asset Sale Agreement. They asked OCF to take over Owens-Illinois' defense of the lawsuit. As justification for their request they explained that OCF had access to the relevant witnesses and documents, and OCF was defending similar asbestos cases at the time. Staelin considered the matter and returned the file he had been given, taking the view that each company should deal with its own cases. He made no mention of a purported right of OCF to be indemnified by Owens-Illinois.[6] Since it is logically inescapable that the earliest claims against OCF came from individuals who were exposed, at least in part, prior to May 1, 1958, the Panel could, and should, draw the rational inference from OCF's silence: those at OCF with actual knowledge of and involvement with the 1958 Asset Sale Agreement knew that the agreement gave OCF no contractual indemnity right from Owens-Illinois for asbestos personal injury tort claims.

Approximately four years later in 1979, as asbestos claims began to mount against OCF, OCF brought claims against Owens-Illinois for indemnity and contribution. These claims were in the form of third-party complaints in lawsuits where the plaintiffs had sued OCF, but not Owens-Illinois. Because OCF's claims in these third-party

[6] That 1975 case was settled by Owens-Illinois for $250. Owens-Illinois received no cases in 1976 and thus justifiably was not particularly concerned with the litigation until several years later.

Complaints were identical specifically referred to "acquisition documents" Owens-Illinois' lawyer in charge of asbestos litigation, Philip McWeeny, asked OCF to explain why it was suing Owens-Illinois and to inform him of the precise basis for OCF's claims. On September 12, 1979, OCF counsel Duane Callender assured him that OCF was not suing Owens-Illinois under the 1958 Asset Sale Agreement. Instead, Callender said, OCF was suing Owens-Illinois on the basis of common-law manufacturer/distributor liability arising out of the fact that prior to 1958 Owens-Illinois manufactured Kaylo and OCF distributed it.

OCF confirmed in writing what had been stated to McWeeny. In the September 24, 1979 Phillips/Callender letter (Ex. 4), OCF expressly stated that its lawsuits against Owens-Illinois were based on the parties' prior distributor/manufacturer relationship, continuing:

> "At the present time, based on the information that we have, this is the only theory under which these Third Party Complaints are being filed. At this time, the information that we have presently reviewed will support this theory and no other. We are in the process of continuing to review our files and if there is any material change in our position we will advise you." Ex. 4 (emphasis supplied).

At the very time that OCF was making these assurances to Owens-Illinois, OCF was recognizing the magnitude of the asbestos litigation it would ultimately face. As an Ohio court has recently found, in 1979 OCF received a consultant's report evaluating the asbestos products liability exposure for certain companies, including OCF. This report -- of which Owens-Illinois did not know until the Ohio decision this February -- concluded that there existed "a realistic potential" of in excess of 200,000 asbestos-related lawsuits over the next several years. In a "conservative" estimate, the report

suggested that companies in OCF's position faced $3 million to $4 billion in potential liability for asbestosis victims alone. Shortly thereafter, OCF was compelled to release its primary and umbrella insurance carrier, Aetna, from asbestos coverage, because Aetna's reinsurers, fearing the escalating number of asbestos claims, were threatening to cancel coverage. See Ex. 5 at 14-15.

Despite being advised of such potential liability, OCF did not assert any express contractual indemnity claim against Owens-Illinois. Rather, OCF rapidly began spending hard cash to buy contractual rights to indemnity from insurers. In 1978 OCF disclosed that it had established reserves to pay asbestos claims. For a period beginning in 1979, OCF paid more than $16 million to buy insurance to cover asbestos claims.[7] Moreover, during this same period OCF's product liability insurers were not paying anything like the full cost of asbestos cases. In addition to having to pay deductibles and self-insured retentions, in 1982 and 1983 OCF disclosed that it was paying part of the average for every asbestos claim settlement. And in 1984 OCF took a charge to income of $9,700,000 for asbestos costs. Despite a mounting caseload and the direct payment of millions of dollars out of OCF's own pocket, OCF did not assert a contractual indemnity claim and did not keep records to permit OCF to assert or quantify any such claim.

As of year-end 1986, OCF had approximately 33,000 asbestos personal injury claims pending. Still, never once did OCF mention the supposed Owens-Illinois contractual indemnity obligation to insurance companies, to its accountants or banks, or to its shareholders. Nor, of course, did OCF mention it to Owens-Illinois. Moreover,

[7] Indeed, it appears that a large portion of the $16 million was spent on insurance which was for "claims handling" rather than real "risk transfer" insurance.

[redacted] paying millions [redacted] reserve [redacted] claims against Owens-Illinois. OCF did so solely on the "straight" equitable indemnity theory laid out in the Phillips/Cathcart letter. OCF has never pursued such a claim beyond the pleading stage, and Owens-Illinois has never paid OCF even one cent to settle such a claim.

In numerous other ways OCF failed to act like a company which believed it possessed even an arguable contractual indemnity right. For example, throughout the 1980's, Owens-Illinois and OCF jointly negotiated settlement agreements with plaintiffs. Both belonged to an asbestos defense group in San Francisco and negotiated settlements designed not only to settle with plaintiffs but to avoid claims between defendants as well. As the Owens-Illinois lawyer who negotiated these settlements will testify, he certainly would have expected OCF to have informed him that Owens-Illinois' payments toward settlement would not achieve an end to such claims, that instead OCF would one day turn around and sue Owens-Illinois for OCF's settlement payments.

Likewise, in 1985, an insurance settlement agreement was reached among 33 asbestos defendants and their insurance carriers which encompassed an indemnity sharing agreement. That agreement designated the Asbestos Claims Facility (the "ACF") to handle, by trial or settlement, the companies' bodily injury asbestos claims. OCF's 22% share was the highest among the signatory companies. Owens-Illinois' share was 5.5%. At the time OCF entered into the ACF insurance sharing agreement, it never mentioned to Owens-Illinois the existence of its supposed contractual indemnification right under the 1958 Asset Sale Agreement. It is reasonable to expect that OCF would

[redacted] such part of its share to Owens-Illinois if it had believed it had a right to indemnification under the 1958 Asset Sale Agreement.

The ACF dissolved in 1988, asbestos claims continued to roll in against OCF, and OCF's out-of-pocket costs rose. In 1989 OCF took a charge to income of $50,000,000 for the uninsured costs of asbestos claims. OCF never mentioned the supposed indemnity right to Owens-Illinois. In late 1989, with 73,500 asbestos cases pending against OCF, OCF attorney Bill Sowinski invited Owens-Illinois to join OCF's "outreach program": a program in which OCF was attempting to minimize its liability by identifying and joining as many defendants as possible in asbestos lawsuits. (Owens-Illinois declined to participate in the program.) Sowinski never mentioned to Owens-Illinois an indemnity claim under the 1958 Asset Sale Agreement.

There have been numerous other instances where OCF should have put forward the claim it now asserts. There were other defense sharing arrangements, there were negotiations concerning legislative proposals, and there were discussions concerning sharing of lawyers. As Andrew Berry of McCarter & English will testify, his firm had been representing Owens-Illinois when OCF approached the firm in the late 1980's to represent OCF as well. The firm accepted the assignment and represented both Owens-Illinois and OCF, something the firm would not and could not have done had OCF disclosed the conflict of interest posed by OCF's present claim for indemnification. Nor would Owens-Illinois have consented to such sharing of counsel (as it did) had OCF disclosed such a claim to McCarter & English.

OCF's conduct for three decades has been utterly inconsistent with that of a company that possessed, or even thought it might possess, a substantial indemnity right

against another company. No reasonable company with a good faith belief in an indemnity right would wait to assert that claim until the critical witnesses who might be expected to argue for such a claim had all died.[8] No reasonable company would incur tens of millions of dollars of additional uninsured costs, draw on a valuable insurance asset and spend millions of dollars to purchase additional insurance without at least also asserting a contractual indemnity right against another company. No reasonable company would fail to keep records to establish the amount of the claim and the cases to which it related. No reasonable company would take an $800 million charge against 1991 earnings for asbestos liabilities and not assert at the same time an indemnity claim it believed it possessed. And no reasonable company would conceal such claim for year after year and face the probability that it would eventually have that claim barred by laches or estoppel (which is precisely what should occur here). The only possible explanation for OCF's conduct is that OCF's "claim" is a reversal of course dictated by circumstances unrelated to the merits of the claim.

F. OCF's Inconsistent Testimony: 1993 to 1995

OCF's own conduct has led a large number of asbestos claimants to demand punitive damages from OCF. Between 1989 and 1994, juries awarded punitive damages of over $200 million against OCF. A list of the 142 verdicts as of last

[8] OCF has recently designated Robert Knight, Esq., to testify in OCF's case in chief as someone who participated in the 1958 Asset Sale Agreement. Even if he somehow "participated" and will testify as to what he supposedly "remembers" occurred in 1958, all other witnesses, including all of the Owens-Illinois' participants and all the signatories, are now dead. Carl Staelin, John Marshall Briley, and H. R. Winkle who negotiated and signed the 1958 Asset Sale Agreement on behalf of OCF, and Charles Babbs, Hugh Laughlin, and C. R. Megowen who represented Owens-Illinois in connection with and signed that agreement, are dead.

December in which punitive damages were assessed against OCF (and there have been more since then) is included as Exhibit 6. (The list does not include more than 400 other cases in which OCF has been found liable for compensatory damages.) In an effort to avoid punitive damages, OCF has taken a position that is directly contrary to OCF's position here.

Whether in affidavits, testimony, or argument, the OCF defense to further punitive damages is consistent: after its insurance is gone, OCF -- and OCF alone -- must pay every nickel of asbestos liability by itself. For example, Louis Rodewig, Director of Litigation for OCF, testified in 1994 to the jury how much OCF was going to "have to pay out of [OCF's] own resources" to settle asbestos claims. He described in detail OCF's remaining insurance, including OCF's estimate of the amount of indemnity coverage OCF expected to obtain in a pending arbitration with one insurer. He testified that OCF was going to pay the full amount, net of that insurance, of its liability to asbestos claimants out of OCF's own pocket: "Where we [OCF] took [a reserve of] $900 million, we have got to earn every penny of that $900 million." He repeated this numerous times: "[The reserve is] an estimate of our [OCF's] uninsured cost, those costs we would have to pay out of the company's own resources." In summation to the jury, OCF's counsel calculated for the jury how much OCF will have to pay in after-tax dollars on a daily basis to dispose of asbestos claims through 1999: "Owens-Corning will pay $269,733.33 every single day from April 1st to the end of this century out of its pocket for asbestos litigation. That's how much. It doesn't stop." In direct contradiction to these sworn factual representations to juries and courts, OCF is asserting here that Owens-Illinois is obligated to pay indemnity to OCF.

This testimony has been repeatedly given, by Rodewig and Price Waterhouse partners retained to do so, through February and March of this year. Never has Rodewig or any other OCF witness disclosed that OCF is seeking to have Owens-Illinois pay part of OCF's obligation. Rather, Rodewig and other OCF witnesses testify that, other than insurance, OCF must pay every cent out of OCF's pocket; and OCF's trial counsel then argues that juries should not penalize OCF.

ARGUMENT

In the points which follow we demonstrate (1) why OCF's interpretation of the 1958 Asset Sale Agreement is implausible and unreasonable and is contradicted by the contemporaneous evidence, and (2) the other separate and independent reasons why OCF's claim is barred. We also address Owens-Illinois counterclaims against OCF for OCF's wrongful, culpable acts after it purchased the Kaylo division in 1958.

POINT I

THE 1958 ASSET SALE AGREEMENT DID NOT REVERSE THE ALLOCATION OF RISK ESTABLISHED BY THE 1953 MASTER PRODUCT SALE CONTRACT

OCF's claim, in essence, is that Owens-Illinois agreed in 1958 to indemnify OCF for OCF's tort liabilities to third parties. The basis of the "agreement," OCF asserts, is an "all warranties" clause at the end of a provision in the 1958 Asset Sale Agreement transferring to OCF third party executory contracts. From this 37-year old acorn grows overnight the huge oak described by OCF, under which Owens-Illinois must indemnify OCF even for strict liability and statutory tort claims brought against OCF by asbestos claimants harmed by Kaylo manufactured and sold by OCF after 1958.

Under the allocation of risk established with respect to the Kaylo Products covered by the 1953 Master Product Sale Contract, Owens-Illinois' liability was expressly limited, in time and in amount, to claims by OCF for nonconformance with certain technical specifications. The risk of liability for any special or consequential damages was expressly allocated to OCF. Under the 1953 Master Product Sale Contract, Owens-Illinois manufactured the Kaylo block and pipe insulation and OCF employed the sales force, prepared the advertisements, and sold these Kaylo products to the public. In light of this relationship, the allocation of risk under the 1953 Master Product Sale Contract reflected sound business judgment since OCF was the party actually making warranties to customers. Nothing in the 1958 Asset Sale Agreement changed this business judgment. Owens-Illinois did not suddenly agree to assume warranty liabilities in 1958 that it had explicitly disclaimed in 1953 and did not issue a blank check to indemnify OCF for breaches of all the warranties OCF's salesmen may have made to third parties over the previous five years.

To the contrary, the 1958 Asset Sale Agreement provided only a discrete, limited indemnity. As set forth below, the most reasonable and compelling construction of paragraph 6, and its "all warranties" clause, is that it applies only to third party executory contracts.[9] The "all warranties" clause of paragraph 6 certainly was never

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[9] OCF proposed two different constructions of the "all warranties" clause in its Reply. Owens-Illinois believes, as summarized above, that paragraph 6 does not cover sales to OCF, but even if it were so construed, it would cover only a very small number of sales which were partly completed as of May 1, 1958. See page 40, below. As we explained responding to OCF's motion to dismiss Owens-Illinois' counterclaims, and is set forth below, one Owens-Illinois counterclaim is asserted in the alternative, based on the theory that paragraph 6 applies to OCF purchases and that the 1953 Master Product Sale Contract is one of the contracts "assigned" by paragraph 6.

intended to apply to sales contracts that had been completed by the date Illinois by April 30, 1958. The foregoing is compelled by analysis of the context, structure, and language of the 1958 Asset Sale Agreement. Moreover, the language and context affirmatively show that the actual drafters -- practical Ohio businessmen -- did not intend to indemnify for any tort liability, a conclusion confirmed by OCF's subsequent conduct and supported by the allocation of risk as to completed sales transactions established by the 1953 Master Product Sale Contract.

It is highly significant that the 1958 Asset Sale Agreement did not refer in any way to the 1953 Master Product Sale Contract. There was no attempt to modify, alter, or expand on the specific terms of the earlier agreement. That agreement remained in full force.

Ohio law is in accord with the common sense notion that businessmen and lawyers do not upset existing contractual rights and obligations lightly. Under Ohio law, the "substitution of a new contract, debt, or obligation for an existing one, between the same or different parties" is a novation.[10] Ohio law makes clear that "a novation is never to be presumed."[11] Rather, the party alleging the novation must establish by

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[10] Gross v. Cassidy, No. E-88-16, 1989 WL 5419, at *5 (Ohio App. Jan. 27, 1989) (citation omitted); see also Black's Law Dictionary 1212 (4th ed. 1951) (defining a "novation" as the "[s]ubstitution of a new contract between same or different parties").

[11] Astrup Co. v. Genie Copy Serv., No. 57548, 1991 WL 34868, at *2 (Ohio App. March 14, 1991) (citations omitted); accord Thompson v. Anderson, No. 93 APE08-1155, 1994 WL 14791, at *2 (Ohio App. Jan. 20, 1994); Grant-Holub Co. v. Goodman, 23 Ohio App. 540, 156 N.E. 151, 153 (1926).

[redacted] evidencing [redacted] was modified by the parties.

In light of this evidentiary standard, OCF's contentions are particularly implausible. OCF is not alleging that the 1958 Asset Sale Agreement merely modified the 1953 Master Product Sale Contract's long-existing allocation of risk. Rather, OCF, after a delay of more than 30 years during which it received 250,000 bodily injury claims, sustained more than $400 million in damage findings and verdicts, and spent more than $2.0 billion to resolve claims, is contending that a single phrase torn out of context simultaneously (i) extinguished the 1953 Master Product Sale Contract's careful delineation of risks and liabilities assumed and not assumed and (ii) retroactively replaced these specific provisions with a complete reversal of the terms of $19 million worth of prior sales contracts. There is, however, no evidence in the 1958 Asset Sale Agreement or elsewhere that the parties intended paragraph 6 to address -- much less reverse -- the allocation of risk established in the 1953 Master Product Sale Contract, which is not referred to once in the entire 1958 Asset Sale Agreement.

A. Paragraph 6 Only Addresses Executory Contracts with Third Parties for the Sale of Products Not Governed by the 1953 Master Product Sale Contract

Paragraph 6 of the 1958 Asset Sale Agreement cannot fairly be read to address -- much less supersede -- the terms of the 1953 Master Product Sale Contract.

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[13] See Astrup, 1991 WL 34868, at *2; Bolling v. Clevepak Corp., 20 Ohio App. 3d 113, 125, 484 N.E.2d 1367, 1379 (1984); Grant-Holub, 23 Ohio App. at 546, 156 N.E. at 153.

The 1958 Asset Sale Agreement provides that Ohio law governs the contract's interpretation. Ex. 3 ¶ 12. Under Ohio law, "[w]hile each part of the contract should be considered by itself,.. the several parts should be construed together and the intent gathered from a consideration of the contract as a whole."[13]

OCF's 1958 acquisition of the Kaylo division was an asset purchase and not a purchase of stock or merger. The 1958 Asset Sale Agreement consists of thirteen paragraphs, the first six of which directly address the transfer of assets to OCF and the last seven of which primarily concern miscellaneous procedural matters and contingencies.

After the first paragraph announces the sale of the Division, paragraphs 2 through 6 each transfer a different class of assets to OCF. Although most assets could be simply transferred, contract rights of Owens-Illinois posed a unique problem: because the parties wanted the transfer "as of" April 30, the parties had to allocate between themselves responsibility for uncompleted contractual commitments. Paragraph 6's function was to assign to OCF that remaining, otherwise unaddressed class of assets -- all of the Kaylo's Division's third-party executory contracts -- and allocate responsibilities with respect to any unperformed obligations under them. Paragraph 6 provides that:

> "6. O-I hereby assigns to OCF all of the executory contracts as of May 1, 1958, of the Kaylo Division, including those for the purchase or sale of goods, materials, equipment, supplies and capital assets, agreements with labor unions, consultant agreements and all other contracts having to do with the conduct of its business (excepting, however, accounts receivable arising from goods supplied, services rendered or other transactions prior to May 1,

[13] 18 Ohio Jur. 3d, Contracts § 150.

1958) and OCF [illegible] and discharge all [illegible] obligations [illegible] for goods supplied or services rendered [illegible] obligations including the responsibility of O-I and accepting the obligation, if any, of O-I to pay compensation to any salaried employee of the Kaylo Division by reason of the termination of his employment by O-I, and will save O-I harmless from any and all claims of any third person or persons for any breach, after assignment thereof, of any agreement so assigned. O-I will save OCF harmless from any and all claims for any breach, prior to assignment thereof, of any agreement so assigned, and for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958." Ex. 3 ¶ 6 (emphasis supplied).

Assigning the executory contracts -- and not the completed contracts -- of the Kaylo division made business sense. OCF wanted those assets of future value in running the Kaylo business -- labor contracts, consulting agreements, supply agreements, and outstanding sales contracts. It made no sense to deal with fully completed contracts since they were not "assets" of the business and could not be "used" by OCF in the future operations of the business.

When read as a whole and in the context of the entire agreement the purpose of the parties is clear. Paragraph 6 deals exclusively with executory contracts. That is the plain -- and the only -- subject of the entire paragraph.

2. Paragraph 6 Addresses Executory Contracts with Third Parties

The language and the context of paragraph 6 demonstrate that the most reasonable construction is that it only addresses executory contracts between Owens-Illinois and parties other than OCF. There were a number of Owens-Illinois third-party (non-OCF) executory sale-of-goods contracts in existence at the time. While OCF was virtually the exclusive purchaser of Kaylo Heat Insulating Products under the 1953

Master Product Sale Contract, Owens-Illinois continued to sell other Kaylo products -- such as door core and roof tile -- directly to third-parties. As of May 1958, Owens-Illinois had outstanding executory contracts for the direct sale of Kaylo products other than Heat Insulating Products with at least Western Electric, Chrysler, and Owens-Illinois Plywood Company. It was these third-party executory sale-of-goods contracts that were assigned to OCF in paragraph 6.

The Kaylo products Owens-Illinois sold to third parties other than OCF were outside the scope of the 1953 Master Product Sale Contract which covered only sales to OCF. Since OCF was acquiring the entire business, there was good reason to deal with the executory contracts relating to these other sales. It was reasonable, as well, for Owens-Illinois expressly to agree in paragraph 6 to indemnify OCF for any breaches of agreement with respect to the products already delivered in those direct sales by Owens-Illinois to non-OCF third-parties under contracts that were executory as of May 1, 1958, since Owens-Illinois was retaining the right to receivables on these deliveries and since Owens-Illinois and OCF had no agreement between themselves defining their responsibilities with respect to them.

These executory sales contracts were in all respects similar to the other six categories of executory contracts dealt with by paragraph 6, those between Owens-Illinois and various suppliers, labor unions and consultants. The executory contracts for direct sales of goods to third parties were assets of the Kaylo business, OCF was not a party to them and needed to be, and Owens-Illinois had unperformed obligations outstanding with respect to them, which OCF had to assume. The division of obligations with respect to them and the common sense rationale for Owens-Illinois' indemnity of OCF for some of

them applies in a manner parallel to the handling of the other executory contracts. This interpretation of paragraph 6 is the only interpretation which does not pull the phrase "executory contracts ... for the purchase or sale of goods" out of the context established by the rest of the paragraph and does not disrupt the parties' other well-defined and well-established contractual rights and obligations.

- 3. The Use of Assignment Proves an Intention To Address Only Third-Party Contracts

The operative legal conveyance chosen by the parties proves that paragraph 6 addresses only contracts between Owens-Illinois and third-parties, not between Owens-Illinois and OCF. Paragraph 6's first sentence provides that:

> "O-I hereby assigns to OCF all of the executory contracts as of May 1, 1958 of the Kaylo Division ...." Ex. 3 ¶ 6 (emphasis supplied).

The contracts intended to be addressed by the paragraph must have been of a character or kind "assignable" to OCF. Owens-Illinois' contracts for the sale of Kaylo to OCF cannot meet this test. As a matter of assignment law and business practice, Owens-Illinois' contracts with OCF were not subject to assignment to OCF, the other contracting party.

Owens-Illinois and OCF were the only two parties to the sales contracts entered into under the 1953 Master Product Sale Contract. Assignment law concerns the transfer of contract rights to persons who are not parties to the contract creating the rights. Businessmen do not assign one party's rights under a two party contract to the contract's only other party. Such an assignment would leave the only remaining party to the contract simultaneously with a duty to perform an obligation to itself and the right to

receive performance from itself. No business purpose would be accomplished by such an arrangement.

4. The Last Clause of Paragraph 6 Addresses Only Third-Party Executory Contracts as to Which Post-Delivery Executory Obligations Existed as of May 1, 1958

OCF bases its entire case on the last clause of paragraph 6 which provides that Owens-Illinois would indemnify OCF for breach of "all warranties and agreements" relating to goods delivered prior to May 1, 1958. However that clause only dealt with a small number of contracts -- those third-party executory contracts assigned to OCF as to which Owens-Illinois had outstanding "executory obligations" as of May 1, 1958. It has nothing to do with the Heat Insulating Products that had been sold by Owens-Illinois to OCF from 1953 to 1958. This is demonstrated by the structure and drafting history of paragraph 6.

The third-party executory sales contracts assigned to OCF under the 1958 Agreement fall into two categories: contracts where Owens-Illinois had not yet made delivery and contracts where Owens-Illinois had delivered some or all of the goods but still other obligations were to be performed. With respect to the latter category, since clause 1 of paragraph 6 transferred all open third-party "contracts" to OCF, OCF would, without more, obtain the right to receive payments for deliveries made prior to May 1 even though Owens-Illinois had delivered those goods. To avoid this anomaly, Owens-Illinois explicitly retained the rights to accounts receivable arising from goods delivered by Owens-Illinois to third-parties prior to May 1, 1958:

> "O-I hereby assigns to OCF all of the executory contracts as of May 1, 1958, of the Kaylo Division ... (excepting, however, accounts receivable arising from goods supplied, services rendered

(emphasis supplied)

Correspondingly, Owens-Illinois retained all executory obligations with respect to those delivered goods (e.g., the obligation to accept the return of unused goods for a credit, the obligation to grant a discount). Although OCF in general assumed Owens-Illinois' performance obligations under the assigned executory contracts with third-parties, there was an exception for those contracts where Owens-Illinois had partly performed by delivering, prior to May 1, some of the goods called for under the executory contract, and as to which deliveries Owens-Illinois retained the economic benefits (i.e., the accounts receivable):

> "... and OCF agrees to perform and discharge all executory obligations under such contracts (excepting, however, any obligation for goods supplied or services rendered prior to [May 1, 1958] ...." Id.

Thus, for executory contracts involving goods already delivered to third-parties, the right to receivables and any executory obligations remained with Owens-Illinois as to that part of the "executory contract" Owens-Illinois had performed prior to the May 1, 1958 assignment.

The original draft of paragraph 6 did not contain either the "accounts receivable exception", the "assumption exception", or the "all warranties and agreements" clause. Owens-Illinois added the right to retain accounts receivable and the exception to the assumption of all executory obligations — the exception reserving to Owens-Illinois the liability and cost of performing post-May 1 obligations with respect to the goods Owens-Illinois delivered under the assigned contracts prior to May 1. OCF agreed, but, as one would expect, wanted to be sure the warranty and agreement risk on these goods

would say with Owens-Illinois. The "all warranties and agreements" clause was therefore added to paragraph 6 in response to the accounts receivables exception:

> "O-I will save OCF harmless from any and all claims ... for the breach of all warranties and agreements relating to goods delivered prior to May 1, 1958." Id.

Since Owens-Illinois was getting the money for these delivered goods, Owens-Illinois explicitly took the warranty and agreement risks. This is what the final warranty clause refers to, and it has nothing whatsoever to do with the subject of this arbitration, the sale by Owens-Illinois of Heat Insulating Products to OCF.

This allocation of financial responsibilities was an important part of the parties' understanding. After May 1, Owens-Illinois was to operate the business "at the risk, and for the account of OCF" until OCF took possession and an accounting for receipts and disbursements could be made. The parties clearly intended that post-May 1 receipts relating to pre-May 1 deliveries would be for the account of Owens-Illinois and that post-May 1 costs of, and liabilities associated with, performing obligations incurred in connection with pre-May 1 deliveries would also be "for the account of Owens-Illinois" and not chargeable to OCF.

The structure of the indemnity provisions at the end of paragraph 6 confirms that the last clause refers to the performed portion of the assigned executory contracts -- the duties and responsibilities not assigned to and assumed by OCF by paragraph 6. In other words, the last clause refers to the duties and responsibilities that remained on Owens-Illinois after the assignment by virtue of the exception to OCF's assumption of "executory obligations". In the first two indemnification provisions, the parties divided responsibility for breach of contract claims arising from the assigned

executory contracts based on when the breach occurred. If the breach occurred after assignment of the contract (when OCF had the rights and obligations under the contract), OCF would indemnify Owens-Illinois for claims; if the breach occurred prior to assignment of the contract (when Owens-Illinois had the rights and obligations under the contract), Owens-Illinois would indemnify OCF for claims. Accordingly, it is stated:

> "... and [OCF] will save O-I harmless from any and all claims of any third person or persons for any breach, after assignment thereof, of any agreement so assigned. O-I will save OCF harmless from any and all claims for any breach, prior to assignment thereof, of any agreement so assigned ...." Id. (emphasis supplied).

This division of responsibility for breach of contract claims based on the timing of the breach made good sense as to most of the assigned executory contracts. Each party assumed responsibility for breaches which occurred when that party had the rights and obligations under the contracts. This exchange of indemnities based on time of breach, however, produced an anomalous result in one particular situation: where Owens-Illinois had delivered Kaylo to a third-party purchaser and Owens-Illinois breached after May 1 an executory obligation owed the third party with respect to goods delivered prior to May 1. Owens-Illinois was entitled to the account receivable on this sale. Yet, if a dispute arose with a customer based on a claimed breach of agreement or warranty arising after the assignment of the contract to OCF on May 1, 1958, Owens-Illinois' indemnity of OCF would not require Owens-Illinois to indemnify OCF for breach of the warranty or agreement because the breach would have occurred "after assignment," not "prior to assignment thereof." Thus, in this one situation Owens-Illinois would have obtained both payment for the goods and OCF would have no indemnity for the claim. This structure would have created an anomaly, because in all other situations

covered by paragraph 6 the party who was to receive payment for the goods would bear the risk of claims. This anomaly was remedied by the last clause of paragraph 6, which required Owens-Illinois to indemnify OCF for any post-assignment breaches of agreements or warranties relating to goods which Owens-Illinois had delivered prior to May 1, 1958 under the "executory contracts" which were the subject of the assignment.

B. Even if Paragraph 6 Were Construed to Apply to OCF Sales, It Provides No Recovery

Even if paragraph 6 were construed to include executory contracts concerning sales of Kaylo to OCF, OCF's claim is without merit. First, at most, the warranties referred to in the last clause are Owens-Illinois' pre-existing warranties under the assigned contracts. Second, in 1958 Ohio law did not recognize the types of "warranty" claims for which OCF is now, over thirty years later, seeking to be indemnified. Third, in any event, paragraph 6 only applies to a narrow class of contracts, i.e., the assigned executory contracts, and, hence, does not apply to all of Owens-Illinois' sales to OCF over the previous five years.

1. The Warranties Clause Did Not Create Any New Warranties

The warranties clause only addressed existing warranties and did not create any new warranties, express or implied. Were paragraph 6 construed to include sales to OCF, the indemnity's reference to "all warranties ... relating to goods delivered prior to May 1, 1958" would refer to nothing more than Owens-Illinois' existing warranties under

the 1953 Master Product Sale Contract, Owens-Illinois and OCF carefully defined the exact nature and scope of Owens-Illinois' warranty obligations in the 1953 Master Product Sale Contract. In 1958, any businessmen who negotiated the sale of the Kaylo division would have had no need to specify these obligations, and instead employed a general term, "all warranties," as a convenient and efficient means of referring to Owens-Illinois' pre-existing warranty obligations under its prior sales contracts. If these parties had instead intended this indemnity to extend beyond the scope of Owens-Illinois' pre-existing warranties, they would have expressed their intent in a clear and detailed provision (as they did in a detailed side-letter concerning an indemnity for taxes).

Paragraph 5(e) of the 1953 Master Product Sale contract limits Owens-Illinois' liability as a seller of Kaylo products to OCF to the contract price of the products sold and excludes any liability for special or consequential damages. The 1958 Asset Sale Agreement goes even further in its allocation of Kaylo risks to OCF. The $633,000 worth of Kaylo inventory and raw materials that are sold to OCF pursuant to paragraph 2 of the 1958 Asset Sale Agreement are sold as is -- with no "representation or warranty whatsoever, except as to title ...." Ex. 3 ¶ 9. These are clear and strong provisions. They show that a dominant, consistent theme of the parties' agreements was to allocate most Kaylo risks to OCF. It makes no sense, and would surely be contrary to the parties' intent, to interpret language such as that contained in paragraph 6 as contradicting, or warring with, this pattern of risk allocation to OCF.

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[14] The parties carefully preserved Owens-Illinois' other pre-existing rights (i.e., Owens-Illinois' accounts receivable) and obligations under the contracts assigned by paragraph 6. The warranties clause was likewise intended as a means of preserving, rather than replacing, Owens-Illinois' pre-existing warranty obligations.

Ohio law recognizes that business negotiations take place against a backdrop of pre-existing contractual relationships and that, therefore, a contract may not be entirely divorced from its environment."[15] Accordingly, "a contract should be construed in the light of a previous contract which is evidently designed to control the relations of the parties for a period covered by the latter contract, unless the later contract is manifestly an abrogation the earlier contract."[16] Since the 1953 Master Product Sale Contract was clearly designed to control the relations of the parties as to Kaylo sales prior to May 1, 1958, and there is no evidence in the 1958 Asset Sale Agreement or elsewhere that the parties intended to reverse this allocation of risk, the term "all warranties ... relating to goods delivered prior to May 1, 1958" must be construed as a general reference to the pre-existing commercial warranties specified in the 1953 Master Product Sale Contract (and in Owens-Illinois' other third party contracts for the sale of Kaylo).

2. The Last Clause of Paragraph 6 Addresses Only Breach of Commercial "Warranties"

Even aside from the foregoing, the term "breach of all warranties" in the last clause of paragraph 6 only refers to breach of commercial warranties (such as the product's dimensional stability or insulating capability). To sustain its position, OCF must persuade the Panel that the phrase "breach of all warranties" in the 1958 Asset Sale Agreement includes, and that the parties intended in 1958 for it to include, third party personal injury tort claims. This is not credible.

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[15] Starr Co. v. Columbia Broadcasting Sys., 68 Ohio App. 352, 356, 36 N.E.2d 861, 863 (1941).

[16] Id., 68 Ohio App. at 356, 36 N.E.2d at 864.

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First, a reading of the entire paragraph makes it manifestly clear that it addresses the rights and obligations of the parties concerning ordinary commercial contracts incidental to running a manufacturing operation -- "contracts ... for the purchase or sale of goods, materials, equipment, supplies and capital assets, agreements with labor unions, consultant agreements and all other contracts having to do with the conduct of [the Kaylo division's] business." Ex. 3 ¶ 6.

Second, and more importantly, the parties could not have intended the word "warranties" to have encompassed torts which Ohio law did not recognize in such circumstances at the time the contract was executed. As a matter of law, the contract must be interpreted in light of the law existing at the time the contract was executed.[17] Personal injury tort claims for breach of "warranty" where there was no privity of contract between the manufacturer and the claimant were not recognized in Ohio in 1958. At that time, the Uniform Sales Act governed warranty claims and placed significant restrictions on such claims. Aside from limited exceptions recognized by Ohio and some

[17] "It is well settled that the law in existence when a contract is formed enters into the contract and becomes a part of the agreement, thereby requiring the contract to be construed in light of the law." Vannoy v. Capital Lincoln-Mercury Sales, Inc., 88 Ohio App. 3d 138, 144, 623 N.E.2d 177, 181 (1993). This principle is based on the presumption that the language employed by the contracting parties in their agreement is adopted in reliance on the then-existing law. See City of Middletown v. Ferguson, 25 Ohio St. 3d 71, 78, 495 N.E.2d 380, 386 (1986), cert. denied, 479 U.S. 1034 (1987). Statutes and legal concepts developed after the execution of the contract are not read into and do not become a part of the contract. See City of Columbus v. Public Utils. Comm'n, 103 Ohio St. 79, 116-17, 133 N.E. 800, 813 (1921); Case Mfg. Co. v. Garven, 45 Ohio St. 289, 298-99, 13 N.E. 493, 496 (1887); Drane v. Lawton Co., 1 Ohio Op. 2d 426, 141 N.E.2d 253, 255, aff'd, 1 Ohio Op. 2d 431, 141 N.E.2d 259 (1956).

objects for profit used in or on the body (e.g., foods, medicines),[16] the law of warranty was well established by the Ohio Supreme Court in 1958. Only persons in contractual privity with the defendant could maintain an action for breach of an implied warranty. Thus, remote consumers who were injured by a product could not bring a claim for personal injury against the manufacturer or a seller on an implied warranty theory (which does not require a showing of fault). Rather, such claims could be brought only if the plaintiff could prove the manufacturer's negligence.[19]

It was not until 1966 that the Ohio Supreme Court recognized a personal injury claim for breach of implied warranty by persons not in privity with the defendant

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[16] By 1958, some Ohio cases had recognized implied warranty claims in the absence of privity for injuries from defective foodstuffs and medicines, see Rogers v. Toni Home Permanent Co., 167 Ohio St. 244, 246, 147 N.E.2d 612, 614 (1958) (discussing cases); Mahoney v. Shaker Square Beverage, Inc., 46 Ohio Op. 250, 102 N.E.2d 281 (C.P. Cayahoga 1951), and products applied to the body, see Krupar v. Proctor & Gamble Co., 113 N.E.2d 605, 608 (Ct. App. 1953) (soap), reversed on other grounds, 160 Ohio St. 489, 117 N.E.2d 7 (1954); Markovich v. McKesson & Robbins, Inc. 106 Ohio App. 265, 276, 149 N.E.2d 181, 188 (1958) (hair product). But see Kennedy v. General Beauty Prods., Inc., 112 Ohio App. 505, 507-08, 167 N.E.2d 116, 119 (1960) (hair product); Rogers v. Toni Home Permanent Co., 105 Ohio App. 53, 76, 139 N.E.2d 871, 886-87 (1957) (hair product), aff'd on other grounds, 167 Ohio St. 244, 147 N.E.2d 612 (1958).

[19] See Welsh v. Ledyard, 167 Ohio St. 57, 146 N.E.2d 299 (1957); Wood v. General Elec. Co., 159 Ohio St. 273, 278-79, 112 N.E.2d 8, 11-12 (1953) ("To support an implied warranty there must be contractual privity between the seller and buyer."); Steele v. Westinghouse Elec. Corp., 107 Ohio App. 379, 380, 159 N.E.2d 469, 471 (1958) ("There is no privity of contract between the defendant manufacturer and the plaintiff or her decedent, so that the cause of action must rest upon the alleged negligence of the defendant manufacturer"). The status of the privity requirement in the nation generally in 1958 mirrored that in Ohio: there was no implied warranty claim for personal injuries available to persons not in contractual privity with the defendant. See, e.g., Kennedy v. Brockelmen Bros., Inc., 334 Mass. 225, 227, 134 N.E.2d 747, 748 (1956); Wyatt v. North Carolina Equip. Co., 253 N.C. 355, 359-60, 117 S.E.2d 21, 24-25 (1960); Brown v. Howard, 285 S.W.2d 752, 754 (Tex. Civ. App. 1955); 46 Am. Jur. Sales §§ 306, 307, 816 (1943, reprint 1960).

in Lonzrick v. Republic Steel Corp., 6 Ohio St. 2d 227, 239-40, 218 N.E.2d 185, 194 (1966).[20]

In the 37 years since the 1958 Asset Sale Agreement was executed there has been a change — indeed a revolution — in tort law. OCF will call a professor to testify, with the benefit of 37 years of 20-20 hindsight, perhaps as to "indications," "developments," "trends," or "policies" in other states, in lower courts, and in academic circles. OCF's counsel will undoubtedly vacuum the reported and unreported decisions of lower Ohio courts in an attempt to find a case or two discussing "warranties."[21] The

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[20] As Lonzrick explains, the change in Ohio's law occurred in several stages: (1) the 1958 Toni decision allowed a personal injury claim by the ultimate consumer of a hair product based on express and relied upon warranties in the manufacturer's advertising, but explicitly distinguished such claims from implied warranty actions; (2) the 1962 enactment of the Ohio Uniform Commercial Code, which extended a seller's warranty in some circumstances to family or household members of the buyer; and (3) Inglis v. American Motors Corp., 3 Ohio St. 2d 132, 140, 209 N.E.2d 583, 588 (1965), which permitted an express warranty claim to proceed in the absence of privity for property damage.

Moreover, asbestos personal injury complaints against OCF are premised on a failure to warn theory, and, occasionally, on a design defect theory. While implied warranty claims based on a manufacturing defect theory were recognized in 1966 by Lonzrick, such claims based on failure to warn or design defect theories were not recognized in Ohio until years later. The Ohio Supreme Court did not recognize implied warranty/strict liability for failure to warn until 1977, see Temple v. Wean United, Inc., 50 Ohio St. 2d 317, 321-22, 364 N.E.2d 267, 270-71 (1977), and did not recognize implied warranty/strict liability for design defect until 1981, see Leichtamer v. American Motors Corp., 67 Ohio St. 2d 456, 464, 424 N.E.2d 568, 575 (1981).

[21] OCF previously referred to a 1951 Ohio case, Di Vello v. Gardner Mach. Co., 46 Ohio Op. 161, 102 N.E.2d 289 (C.P. Cayahoga 1951), which permitted a warranty claim by an ultimate user of a grinding wheel. Not only does the opinion use the term "negligence," and thus it is not clear that the decision is based on warranty alone, but it represents the unappealed decision of a single trial judge. At the same time, throughout the 1950's the Ohio Supreme Court repeatedly rejected third-party implied warranty tort claims.

fact remains, however, that by whatever exercise will OCF establish that the parties intended in 1958 that the term "warranties" have a meaning not commonly recognized in the law at that time, indeed, a meaning that was repeatedly rejected by the Ohio State Supreme Court until 1966, eight years after the contract was signed.

And in all events, even were there any doubt whatsoever that the parties did not intend the last clause of paragraph 6 to provide an indemnity for third-party personal injury claims, the absence of words like "negligence" or "claims for personal injury" must eliminate that doubt. In 1958, as today, personal injury suits in Ohio (and in other states) almost invariably were based on, or at least included, a negligence claim.[22] Had the parties intended the 1958 Asset Sale Agreement to indemnify OCF for third party personal injury claims they undoubtedly would have provided an indemnity for negligence, the one claim that was clearly available and routinely used in third-party personal injury cases in Ohio in 1958. It would have made no sense whatsoever for them not to have included an indemnity for negligence and OCF surely cannot explain that inexplicable failure. As the Ohio Supreme Court stated, "[t]he liability of such an indemnitee is regarded to be so hazardous, and the character of the indemnity so unusual and extraordinary, that there can be no presumption that the indemnitor intended to assume the liability unless the contract puts it beyond doubt by express stipulation".[23]

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[22] See, e.g., Krupar v. Proctor & Gamble Co., 160 Ohio St. 489, 490, 117 N.E.2d 7,8 (1954); Steele, 107 Ohio App. at 380, 159 N.E.2d at 471; Tennebaum v. Pendergast, 57 Ohio L. Abs. 195, 90 N.E.2d 451, 452 (C.P. Franklin 1947).

[23] George H. Dingledy Lumber Co. v. Erie R. R. Co., 102 Ohio St. 236, 242, 131 N.E. 723, 725 (1921); accord Kay v. Pennsylvania R. R. Co., 156 Ohio St. 503, 504-05, 103 N.E.2d 751, 752-53 (1952).

3. The Last Clause of Paragraph 6 Would Only Apply to OCF Sales Which Were Executory

As explained above, the only sales contracts addressed in paragraph 6 are those with third-parties, not OCF, and, even as to these sales, the warranties clause refers only to pre-existing commercial warranties and did not create any new warranties, express or implied. Even assuming arguendo, that paragraph 6 applied to Owens-Illinois' sales to OCF (as OCF contends), the only sales to OCF to which paragraph 6 would apply would be those sales orders which were executory as of May 1, 1958. Paragraph 6 is an assignment of executory contracts. As such, the paragraph does not concern completed contracts, and would not apply to completed sales of Kaylo to OCF. OCF has not identified any sales of Kaylo which fall within this narrow category and, if there were any, they were immaterial. Thus, OCF's claim must fail even assuming OCF erroneous premise that paragraph 6 relates to Owens-Illinois/OCF 1953 to 1958 sales.

C. OCF's Actions Since 1958 Prove That the 1958 Asset Sale Agreement Does Not Provide OCF with the Indemnification It Now Claims

In a leading case a century ago, the Supreme Court wrote that the "practical interpretation of an agreement by a party to it is always a consideration of great weight. The construction of a contract is as much a part of it as any thing else." In words directly applicable here, the Court explained that there "is no surer way to find out what parties meant, than to see what they have done." Insurance Co. v. Dutcher, 95 U.S. 269, 273 (1877). The Restatement (Second) of Contracts agrees: "The parties to

an agreement knowledge what they meant, and their action under it often the strongest evidence of their meaning."[24]

As set forth above, OCF's actions in the three decades since the 1958 Asset Sale Agreement was signed demonstrate conclusively that the 1958 Asset Sale Agreement did not provide OCF with an indemnity for asbestos claims.

From the first asbestos personal injury claims in the 1960's through the 1970's when asbestos tort claims against OCF began accelerating, OCF was silent. The OCF businessmen and lawyers who negotiated the 1953 Master Product Sale Contract and the 1958 Asset Sale Agreement -- those who knew OCF's actual intent -- did not assert any contractual indemnity claim. Nor did OCF or those individuals ever keep records to support such a claim "later." In 1975, Carl Staelin, who signed the 1958 Asset Sale Agreement on OCF's behalf, spoke to Owens-Illinois' counsel about the companies' responsibilities for handling asbestos claims but did not mention "indemnity," much less inform Owens-Illinois that OCF had a contractual indemnity right against Owens-Illinois. In direct contradiction to OCF's present claim, Staelin said that each company should go its own way, and each should handle its own claims.

Throughout the 1970's and 1980's, numerous instances arose when a reasonable company, advised by competent counsel, would have asserted the purported indemnity right. OCF paid millions of dollars in deductibles and self-insured retentions,

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[24] Restatement (Second) of Contracts § 202(4) cmt. g (1979). Such evidence is admissible whether or not the contract at issue is "ambiguous". Accord O'Connor v. United States, 479 U.S. 27, 33 (1986) ("the course of conduct of parties to any contract[] is evidence of its meaning"; statement made in context of determining meaning of a treaty); Langer v. Monarch Life Ins. Co., 879 F.2d 75, 81 (3d Cir. 1989) (even if a contract is "not patently ambiguous" course of performance can used to interpret its meaning) (insurance case).

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[redacted] Owens-Illinois recovers from insurers. Beginning at least in [redacted] established [redacted] annual quarterly [redacted] charges to income for asbestos bodily injury claims: [redacted] $9,700,000, then $50,000,000 in 1989, then $6 million a quarter, and then $800 million against 1991 earnings. OCF entered into the ACF Agreement and accepted a share of liability four times that of Owens-Illinois without trying to use the (supposed) indemnity right to negotiate a smaller share. OCF sued other defendants in an "outreach" program to reduce OCF's costs. All the while OCF did not mention the supposed contractual indemnity right. Moreover, OCF never had any procedure or system in place to segregate the cases it believed were Owens-Illinois' ultimate financial responsibility. Nor, in settling with plaintiffs, did OCF attempt to specify which claims being settled were for breach of warranty claims arising from pre-May 1, 1958 exposure to Kaylo.

OCF's actions and inactions, external and internal, show that for over three decades OCF did not construe the 1958 Asset Sale Agreement as an indemnity for asbestos claims. The present claim is a lawyer's contrivance, asserted after virtually all the Owens-Illinois and OCF participants have died and documents destroyed.

OCF will undoubtedly proffer some "explanation" or "excuse" for keeping OCF's present "interpretation" of the 1958 Asset Sale Agreement secret from Owens-Illinois, and the world, for three decades. Earlier in this proceeding, OCF asserted that there was no economic reason to assert the contractual indemnity because OCF had not yet exhausted its insurance. But this ignores the tens of millions in deductibles, self-insured retentions and other uninsured costs that OCF has paid out of its own pocket. Moreover, the falsity of that assertion was recently exposed by a court decision regarding OCF's receipt of a consultant's report in early 1979 projecting huge exposure to asbestos

[redacted] in 1979 OCF had to allay the concerns of its insurers who were increasingly concerned at mounting claims -- a concern manifested in mid-1979 in a substantial cessation of normal insurance coverage. This was the very time that OCF informed Owens-Illinois in the Phillips/Callender letter that OCF had no indemnity claim based on the 1958 Asset Sale Agreement.

The actual conduct of the parties shows that the 1958 Asset Sale Agreement was not intended to, was not construed to, and does not in fact, provide for indemnification of OCF for asbestos claims.

POINT II

OCF'S FAILURE TO GIVE TIMELY NOTICE OF ITS CLAIM BARS RECOVERY

OCF's three decade failure to provide Owens-Illinois with notice of its indemnification claim has seriously prejudiced Owens-Illinois and bars OCF for two independent reasons. First, under basic principles of indemnity law, this failure precludes a putative indemnitee from asserting the claim. Second, under the equitable doctrine of laches, the failure to bring a timely action bars that action.

A. The Prejudice from OCF's Delay

Owens-Illinois cannot now amass evidence in support of the proper construction of the 1958 Asset Sale Agreement: individuals who negotiated and drafted the contract are not alive, memories have faded, document files are no longer intact, and documents have been destroyed. The documents that could have demonstrated the specific commercial warranties that were made concerning Kaylo products throughout the

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1953-1958 period are gone. We cannot tell with certainty today the content of the "performance specifications" which were incorporated by reference into the 1953 Master Product Sale Contract, the content and duration of the warranties Owens-Illinois made on the third party contracts that were executory as of May 1, 1958 are unknown, and we do not know what warranties OCF gave in its contracts reselling Kaylo pipe and block.

Further, OCF's delay has prevented Owens-Illinois from minimizing the financial consequences of the supposed indemnity obligation. Had OCF notified Owens-Illinois of a purported contractual indemnity claim even as late as the mid-1980's, Owens-Illinois could have (and undoubtedly would have) taken a number of other steps to protect itself financially.

Had OCF not hid its claim from Owens-Illinois, as the litigation began over a decade ago, Owens-Illinois could have developed more fully the evidence demonstrating that Owens-Illinois is not liable for any "warranty" claims for which OCF demands reimbursement. Not only did OCF fail to give timely notice of its purported right, it actually has admitted, in thousands of cases and in public forums, its liability for the compensatory damages for which it is now seeking indemnity from Owens-Illinois. Furthermore, OCF's culpable acts increased materially Owens-Illinois' risk concerning the purported indemnity. As the Panel will also hear, those acts began long before OCF purchased the Kaylo division and continued thereafter. They include, as is outlined below, OCF's 1940's "weapon in reserve" concerning the hazards of asbestos, and, after OCF purchased the Kaylo division in 1958, OCF's making Kaylo dustier, changing Kaylo's composition, failing to warn after Dr. Selikoff's landmark 1964 study, and failing to utilize asbestos-free or reduced-asbestos alternatives when OCF knew the

alternatives posed less of a health risk. Today, however, large numbers of witnesses have died and memories are faded. Further, for a decade and a half, OCF has engaged in litigation misconduct by concealing its historic knowledge concerning the hazards of asbestos. OCF has been repeatedly sanctioned for this misconduct. OCF's egregious actions have defined the "OCF" liability in the asbestos litigation. Owens-Illinois would be irreparably prejudiced if it had to assume any part of the OCF liability.

B. Indemnity Law Bars OCF's Claim

"[I]t is well established that any act on the part of an indemnitee which materially increases the risk, or prejudices the rights of the indemnitor, will discharge the indemnitor under a contract of indemnification."[25] Where, as here, an indemnitor is prejudiced by an indemnitee's failure to provide timely notice of its claim or by misrepresentations made by the indemnitee concerning the indemnitor's obligation, the indemnitor's obligation is extinguished.[26]

OCF's claim is extinguished for both of these reasons. OCF failed to make a timely demand and it made misrepresentations concerning Owens-Illinois' purported obligation. Despite receiving its first asbestos claims in the 1960's, OCF

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[25] Unisys Corp. v. Legal Counsel, Inc., 768 F. Supp. 6, 8 (D.D.C. 1991); see also Rochelle Bail Agency, Inc. v. Maryland Nat'l Ins. Co., 484 F.2d 877, 878-79 (7th Cir. 1973); American Casualty Co. v. Idaho First Nat'l Bank, 328 F.2d 138, 142-43 (9th Cir. 1964); Hiern v. St. Paul-Mercury Indem. Co., 262 F.2d 526, 529 (5th Cir. 1959); American Export Isbrandtsen Lines, Inc. v. United States, 390 F. Supp. 63, 68-70 (S.D.N.Y. 1975).

[26] Unisys Corp., 768 F. Supp. at 6-8; Hiern, 262 F.2d at 529.

maintained a three decade silence concerning its purported right to indemnity.[23] The 1979 Rumlins/Callender letter affirmatively represented that this claim did not exist. As a result of OCF's delay and misrepresentations, Owens-Illinois has suffered prejudice, and any indemnity obligation that Owens-Illinois owed OCF (even if one existed) would be discharged as a matter of law.

C. OCF's Claim Is Barred by Laches

The established equitable doctrine of laches also bars OCF's claim. Under Ohio law, "[l]aches is an omission to assert a right for an unreasonable and unexplained length of time, under circumstances prejudicial to the adverse party."[24]

Two months ago OCF successfully defended a claim on precisely these grounds in a dispute with one of its insurance carriers. Owens-Corning Fiberglas Corp. v. American Centennial Ins. Co., Case No. 90-2521, Slip Op. (Ohio Ct. C.P., Lucas Cty. Feb. 22, 1995). The carrier had disclaimed coverage on the ground that OCF had

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[23] A claim under the 1958 Asset Sale Agreement was first asserted by an OCF regional counsel in a 1990 litigation in Rhode Island. During trial of the underlying asbestos claim Owens-Illinois settled; OCF moved to add a claim based on the 1958 Asset Sale Agreement; the court did not permit OCF to amend but said on the record that such claims would be severed so the underlying trial could proceed. OCF did not pursue that claim or similar cross-claims in other cases before commencing this arbitration. The 1990 date does not affect any part of the analysis contained in this memorandum.

[24] State ex rel. Wean United v. Industrial Comm'n of Ohio, 66 Ohio St. 3d 272, 275, 611 N.E.2d 828, 830 (1993) (citation omitted). OCF has previously argued that its indemnification claim could not be barred by laches because OCF has brought the claim within the applicable statute of limitations. OCF 9/22/94 Reply at 24. As a matter of Ohio law, this is wrong – merely asserting a claim within the limitations period will not bar the operation of laches where, as here, there has been an unreasonable delay and prejudice. Thirty-Four Corp. v. Sixty-Seven Corp., 15 Ohio St. 3d 350, 352-53, 474 N.E.2d 295, 297-98 (1984).

fraudulently misrepresented in 1979 what OCF knew about its potential liabilities from asbestos. The court noted that the insurer had first learned about OCF's alleged misrepresentation in 1979, but did not assert its counterclaim for reformation or rescission until 1992. Id. at 21. This was held to constitute unreasonable delay. Id. at 22. In words that could have been written to describe the present case, the court described the prejudice suffered by OCF as a result of the insurer's failure to raise the claim in a timely fashion:

> "OCF could have done a number of things: (a) it could have immediately litigated the matter, while witnesses were alive, memories were fresh, and document files were intact; (b) alternatively, if Associated had threatened to rescind the policy or add an absolute asbestos exclusion, OCF could have immediately sought out another carrier to replace Associated on the risk, during a time when excess coverage without asbestos exclusions was easier to obtain ...." Id. at 22-23.

The OCF v. American Centennial Ins. decision confirms the relevant benchmarks for what constitutes unreasonable delay and prejudice under Ohio law: a thirteen year failure to assert the claim which prevented immediate litigation of the matter "while witnesses were alive, memories were fresh, and document files were intact" and which prevented an opportunity to obtain insurance "during a time when excess coverage without asbestos exclusions was easier to obtain". The prejudice to Owens-Illinois from OCF's three decade failure to assert its present claim is starker and more extreme than that arising from the insurance company's thirteen year delay in OCF v. American Centennial Ins. Laches therefore bars OCF's claim.

POINT III

OCF IS EQUITABLY ESTOPPED BY OCF'S REPRESENTATIONS

In the Phillips/Callender letter in 1979 OCF assured Owens-Illinois in writing that, after reviewing the matter, the only indemnity claim OCF possessed against Owens-Illinois was one for equitable indemnity arising out of Owens-Illinois and OCF's pre-1958 manufacturer/distributor relationship. In the intervening years, not only did OCF not advise Owens-Illinois of "any material change in [OCF's] position," OCF acted consistently with the representations in the Phillips/Callender letter. In a 1980 letter, OCF articulated the same position in the Kolezar litigation which was pending in California. OCF did not advise Owens-Illinois of "any material change" in OCF's position when, for example, Owens-Illinois and OCF negotiated sharing agreements in the early 1980's, when the ACF Agreement was negotiated from 1983 to 1985, or when releases were negotiated in 1989 after its demise.

The Phillips/Callender representations and OCF's subsequent conduct consistent with these representations equitably estop OCF from pursuing its indemnity claim.[29] Under Ohio law, equitable estoppel is applicable when a party makes a factual

[29] The Phillips/Callender representations also constitute a waiver of OCF's claim. When these assurances were made, OCF was fully aware of any rights it possessed -- it had recently reviewed the 1958 Asset Sale Agreement in connection with bringing the Philadelphia third-party claims and, according to OCF's privilege log, at various times in the 1970's OCF lawyers reviewed the 1953 and 1958 contracts and compiled documents concerning them. The representations also were made with full knowledge of the value of a right to indemnity for asbestos liability. As discussed, OCF had received a consultant's report dated January 18, 1979 which evaluated the asbestos products liability exposure for certain companies, including OCF, and estimated both the number of lawsuits which would be brought over the next several years (perhaps more than 100,000) and estimated that potential liability for mesothelioma cases alone for a company such as OCF could be nearly $1 billion.

representations on which another party reasonably relies to that party's prejudice or [illegible] circumstances.[30] There is no requirement that the party make the representation with an intent to deceive.[31] However, deliberateness and calculation by the first party increases both the justification and the need for equitable estoppel.[32]

The Phillips/Callender assurances grew out of specific requests by Owens-Illinois for an explanation as to why OCF was suing Owens-Illinois. Owens-Illinois particularly asked OCF whether its claims were based on the 1958 Asset Sale Agreement. Both orally and in the Phillips/Callender letter, OCF assured Owens-Illinois that equitable indemnity was "the only theory under which these Third Party Complaints [were] being filed" and that it would advise Owens-Illinois of "any material change in [OCF's] position."

Mr. McWeeny will testify that for the next 14 years Owens-Illinois relied on these assurances and conduct. Such reliance was manifestly reasonable since OCF thereafter acted consistently with its representations. OCF continued to bring indemnity claims against Owens-Illinois but only based upon principles of equitable indemnity. Numerous occasions arose when OCF would have been expected to inform Owens-Illinois of a contractual indemnity claim under the 1958 Asset Sale Agreement had it thought such a claim existed — such as during the negotiation of defense sharing agreements,

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[30] First Fed. Sav. & Loan Assoc. v. Perry's Landing, Inc., 11 Ohio App. 3d 135, 145, 463 N.E.2d 636, 647-48 (1983); Columbus Trade Exch., Inc. v. AMCA Int'l Corp., 763 F. Supp. 946, 957 (S.D. Ohio 1991).

[31] In re Ohio Knife Corp., Nos. C-910482, C-910488, 1992 WL 308365, at *7 (Ohio App. Oct. 21, 1992)

[32] E.g., Archdeacon v. Cincinnati Gas & Elec. Co., 76 Ohio St. 97, 104-05, 81 N.E. 152, 153 (1907).

particularly the ACF Agreement, nor when OCF asked Owens-Illinois to execute conflict waivers which allowed OCF to retain Owens-Illinois' counsel. OCF never did so. The prejudice Owens-Illinois will suffer if OCF is now allowed to bring this claim is indisputable. The doctrine of equitable estoppel bars OCF from now, over a decade later, asserting a claim for contractual indemnity under the 1958 Asset Sale Agreement.

POINT IV

OCF'S CULPABLE MISCONDUCT BARS INDEMNITY

OCF's asbestos tort liabilities to third parties do not arise merely from its passive resale of Kaylo manufactured by Owens-Illinois. Rather, OCF's own consistent, repeated, and often intentional misconduct over the past five decades has created the factual record upon which asbestos claims are based. Asbestos plaintiffs have sued and will continue to sue OCF because of OCF's own pre-1958 knowledge and conduct, OCF's own knowledge and conduct after 1958, and OCF's own efforts to hide its culpable conduct from plaintiffs, courts, and juries. OCF's egregious record of active misconduct underlies its numerous punitive damage verdicts, including 45 in 1994 alone. Some of these punitive damage awards have resulted in cases in which the plaintiff's only exposure to Kaylo was prior to 1958, thereby demonstrating that OCF's liability to plaintiffs is not simply a consequence of the fact that it distributed Kaylo but due to OCF's other bad acts, both prior to 1958 and since then.

Two principles of the law of indemnity bar any recovery by OCF. First, as set forth above, "[i]t is well settled that any act on the part of an indemnitee [OCF] which materially increases the risk, or prejudices the rights of the indemnitor [Owens-

Illinois] will discharge the indemnitor under a contract of indemnification." There can be (and we expect will be) no dispute that the record of OCF's misconduct has vastly increased the financial risk of any putative indemnitor. Therefore, as a matter of indemnity law, any supposed contractual obligation of Owens-Illinois to indemnify OCF for asbestos claims is discharged.

Second, under Ohio law as it existed in 1958, an intention to indemnify an indemnitee for his own conduct must be expressed in clear and unequivocal terms.[34] The contractual language upon which OCF relies in the written agreement an indemnity for "all warranties ... relating to goods delivered prior to May 1, 1958" -- cannot begin to satisfy the "clear and unequivocal" standard of Ohio law to evince the express intention that OCF be indemnified for liability based on OCF's own independent conduct.

The following is a summary of but some of the evidence of OCF's culpable misconduct which forms the basis of OCF's liability to asbestos claimants.[35]

•OCF's Early Knowledge of the Health Risks of Asbestos. Virtually every case against OCF begins with 1941. In that year, OCF started gathering information on the health risks of asbestos-containing insulation as "a weapon in reserve" in order to convince insulation workers that OCF's most important product, fiberglass, was a preferable product to other insulation alternatives even though

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[33] Unisys Corp. v. Legal Counsel, Inc., 768 F. Supp. 6, 8 (D.D.C. 1991); Rochelle Bail Agency, Inc. v. Maryland Nat'l Ins. Co., 484 F.2d 877, 878 (7th Cir. 1973). Furthermore, public policy in Ohio and elsewhere forbids a party to escape liability by indemnity for harm caused by such person's willful or reckless conduct. See Cain v. Cleveland Parachute Training Center, 9 Ohio App. 3d 27, 28, 457 N.E.2d 1185, 1187 (1983); French v. Special Servs., Inc., 107 Ohio App. 435, 437, 159 N.E.2d 785, 787 (1958); Restatement (Second) of Contracts § 195, cmt. a (1979).

[34] See page 39 & n.23, above, citing George H. Dingledy Lumber Co. and Kay v. Pennsylvania R. R.

[35] Owens-Illinois did not become aware of most of these facts until the 1980's.

[illegible] workers' [illegible] medical [illegible] OCF concluded [illegible] containing insulation products [illegible] of disease. Throughout the [illegible] employed insulators in its contracting units, OCF carefully [illegible] insulation workers' unions as those unions began to worry that their workers were becoming ill from installing asbestos-containing products. By 1962 OCF conceded that: "Asbestos (as found in Kaylo) when breathed into the lungs causes asbestosis which often leads to lung cancer."

•<u>OCF's Failure to Warn.</u> Critical evidence in innumerable trials against OCF is OCF's failure to warn for years after Dr. Selikoff's 1964 study demonstrating that asbestos-containing insulation materials caused asbestos-related diseases in insulators. Other manufacturers did so at once. And, as one of OCF's own vice presidents testified, the label OCF eventually used was so "vague" that "somebody with a Ph.D would have difficulty understanding" what it was supposed to mean.

•<u>OCF's Failure to Remedy Kaylo's Dustiness.</u> By the early 1960's OCF knew that asbestos dust from its products could lead to disease. Not only did OCF change the composition of Kaylo to make it dustier but OCF discovered that it could reduce or completely eliminate Kaylo's dustiness by coating Kaylo with Ludox, a sodium silicate. Despite the fact that the cost of Ludox would have been only one penny per square foot for the complete elimination of Kaylo dust, there is no record evidence showing that OCF used Ludox.

•<u>OCF's Refusal to Market an Asbestos-Free Product.</u> In the early 1960's, OCF developed an asbestos-free Kaylo substitute, Multitemp, which was not, in the words of an 1963 OCF memorandum, "as detrimental to the health as is Kaylo." Despite successful field experience and positive customer response, OCF terminated Multitemp. In 1966 documents analyzing the termination of Multitemp, OCF officials reported that the decision was made because "most of the sales of Multitemp and High Temperature Block were made by substituting them for Kaylo" and because OCF forecast that Multitemp would not produce desired profits.

Asbestos plaintiffs point to other OCF actions to demonstrate that OCF's misconduct was willful and wanton.

•In 1967, OCF forced a college student who had spent a summer working at OCF to delete from the paper he wrote about his summer job all references to asbestos as causing lung cancer because, OCF claimed at the time, this was "extremely proprietary" business information.

•A decade after OCF started manufacturing asbestos-free Kaylo, tests showed that this allegedly asbestos-free Kaylo actually contained asbestos, a fact (among

others) that was recently used by one asbestos claimant to obtain a $15 million punitive damage verdict against OCF.

•Even more recently, OCF's Chairman and CEO stated at the 1992 annual OCF shareholders meeting that "We put the asbestos issue behind us ... [OCF is] not shedding tears about the past." At a recent trial, plaintiffs utilized this statement in obtaining a $54.6 million punitive damage verdict.

Finally, numerous courts have concluded that OCF has engaged in a variety of litigation abuses and frauds in an effort to conceal what it historically knew about the health hazards of asbestos. For example:

•In 1980, an OCF attorney found records of workers' compensation claims involving OCF that dated back to the 1950's, contradicting interrogatory answers that OCF had been certifying as true. The attorney concluded that OCF had to correct its interrogatory answers. OCF did not do so and, indeed, continued denying the existence of such claims, even up to the United States Supreme Court. This misconduct was only discovered in 1989. One court recently characterized OCF's cover-up of the relevant documents as a "deliberate and intentional fraud upon the Courts." Pickering v. Owens-Corning Fiberglas Corp., No. 90-L-1546, Order dated April 7, 1992 (Ill. 3rd Jur. Cir.), aff'd, 265 Ill. App. 3d 806, 638 N.E. 2d 1127, appeal denied, 158 Ill. 2d 564, 645 N.E.2d 1367 (1994), petition for writ of certiorari filed, 63 U.S.L.W. 3672 (U.S. March 6, 1995). See also Owens-Corning Fiberglas Corp. v. Watson, 243 Va. 128, 142, 413 S.E.2d 630, 639 (1992) (concluding that by virtue of an interrogatory answer that was "patently false" OCF had "committed a fraud" upon a federal court).

•OCF's failure to produce four corporate witnesses for examination at trial was recently sanctioned and determined by one appellate court as presenting "no question" that OCF's actions had constituted a "deliberate, contumacious and an unwarranted disregard of the court's authority." Pickering v. Owens-Corning Fiberglas Corp., 265 Ill. App. 3d at 821, 638 N.E.2d at 1137.

•Another court recently concluded that "OCF, with assistance of counsel, knowingly, intentionally and in bad faith filed false and evasive answers"; that OCF and its counsel "have knowingly, willfully, intentionally, in flagrant bad faith and callous disregard disobeyed" a court discovery order; that OCF's conduct and that of its counsel are part of OCF's "pattern of obstructing justice in the asbestos cases"; that OCF has a "history of committing acts of fraud in connection with discovery proceedings"; and that OCF engages in "conduct that, which if not criminal, borders on being criminally culpable". Bodine v. Owens-Corning Fiberglas, No. 92-C-2440, slip op. (Tex., Brazoria Cty., June 11, 1993),

petition for writ of mandamus pending, No. 01-93-00582-CV (Ct. App. 1st Sup. Jud. Dist. Houston).

OCF's liability to asbestos plaintiffs is based on the totality of OCF's own acts and omissions both prior to and after 1958. That liability is based on, and inextricably intertwined with, OCF's decades of misconduct. Exhibit 7 contains excerpts from a court decision in which punitive damages against OCF were sustained on appeal. This decision demonstrates how it is the totality of OCF's misconduct that has led to the imposition of punitive damages. As a matter of law, OCF's claim for indemnification is therefore barred.

POINT V

OCF IS JUDICIALLY ESTOPPED FROM PURSUING THIS CLAIM

OCF has repeatedly argued to courts and juries that, aside from specifically identified and quantified insurance policies, OCF will not be indemnified for its asbestos liabilities but rather must pay every claim out of OCF's own pocket. OCF has taken this position after the filing of this demand for arbitration, and in fact continues to do so up to this very moment. Now OCF attempts to assert the contrary position -- that OCF is entitled to be indemnified by Owens-Illinois under the 1958 Asset Sale Agreement. Under the doctrine of judicial estoppel, OCF is precluded "from asserting a position in one legal proceeding which is contrary to a position it has already asserted in another."[36]

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[36] Patriot Cinemas, v. General Cinema Corp., 834 F.2d 208, 212 (1st Cir. 1987); accord Martin v. C.A. Prods. Co., 8 N.Y.2d 226, 231, 203 N.Y.S.2d 845, 849, 168 N.E.2d 666, 668 (1960); Anonymous v. Anonymous, 137 A.D.2d 739, 741, 524 N.Y.S. 2d 823, 824 (2d Dep't 1988); Sullivan v. Consolidated Rail Corp., 9 Ohio St. 3d 105 459 N.E.2d 513, cert. denied, 467 U.S. 1222 (1984).

As shown below, this doctrine has two independent bases. Either basis alone requires dismissal of any OCF claim for any indemnity from Owens-Illinois. This, however, is one of those rare situations where both bases are applicable.

A. OCF Is Precluded from Playing "Fast and Loose" with the Courts

To permit a party to assert one position before one tribunal and later to assert a contrary position before another is to permit a litigant to "play[] fast and loose with the" judicial system.[37] Judicial estoppel is utilized because "intentional self-contradiction is being used as a means of obtaining unfair advantage in a forum provided for suitors seeking justice."[38] Therefore, "the function of judicial estoppel is to protect the integrity of the" judicial system since:

> "An effective legal system depends upon norms of candor and responsibility. ... If parties feel free to select contradictory positions before different tribunals to suit their ends, the integrity and efficacy of the courts will suffer."[39]

Numerous courts have acknowledged and expanded upon this fundamental justification for this necessary rule.[40]

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[37] Scarano v. Central Rail Co., 203 F.2d 510, 513 (3d Cir. 1953).

[38] Id.

[39] Patriot Cinemas, 834 F.2d at 214.

[40] E.g., Fleck v. KDI Sylvan Pools, Inc., 981 F.2d 107, 121-22 (3d Cir. 1992); cert. denied, 113 S. Ct. 1645 (1993); Teledyne Indus., Inc., 911 F.2d 1214, 1217-18 (6th Cir. 1990); Bates v. Long Island R.R., 997 F.2d 1028, 1038 (2d Cir. 1993) cert. denied, 114 S. Ct. 550 (1993); Allen v. Zurich Ins. Co., 667 F.2d 1162, 1166-67 (4th Cir. 1982).

Arbitration proceedings are integral to and essential parts of the judicial system. Their integrity and fairness must likewise be protected from this threat. Courts have not hesitated to apply the doctrine of judicial estoppel where an arbitration proceeding was involved,[41] and have clearly stated that arbitration panels should apply it where, as here, a litigant might assert a position in a subsequent arbitration proceeding that was contrary to a position asserted in a prior court litigation.[42]

There is no question here that OCF has obtained (and continues to obtain) a benefit from asserting in courts that, aside from identified insurance in specified amounts, OCF is not indemnified. By causing its officers and agents so to testify, OCF repeatedly argues to juries that punitive damages should not be awarded or should be lower than the juries would otherwise have assessed. The juries have, with OCF's evidence and arguments, rendered their decisions; OCF has received its benefits from the judicial system.[43] Accordingly, OCF is judicially estopped in this proceeding.

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[41] E.g., Thompson v. Anderson, 632 A.2d 1349 (Pa. Super. 1993); Chemical Futures & Options, Inc. v. Resolution Trust Corp., No. 93-C2873, 1993 WL496696 (N.D. Ill. Nov. 29, 1993). (Judicial estoppel applicable where first position asserted in arbitration and contrary position later in court; not warranted on facts). Owens-Illinois is confident (although citation to private decisions is impossible) that arbitral panels, like the courts, have dismissed attempts to manipulate arbitration proceedings on grounds of judicial estoppel.

[42] E.g., Chemical Futures, 1993 WL 496696 at *3.

[43] See Lewandowski v. Amtrak, 882 F.2d 815 (3d Cir. 1989); cf. Patriot Cinemas, 834 F.2d at 213. OCF obviously believes it benefits from contending before juries that it should not be penalized because, after the insurance, OCF has no indemnity but must itself earn every nickel of the $800-900 million to pay asbestos claims. See Hazel-Atlas Glass Co. v. Hartford Empire Co., 322 U.S. 238, 246-47 (1944).

B. Judicial Estoppel Used Sparingly To Preclude Opportunistic Litigants From Contrary Positions

A small, limited number of courts have utilized judicial estoppel to preclude a party, who had taken an oath to tell the truth and testified, to testify to a contrary position in a later proceeding. "To countenance such a situation would be to regard with complacency a violation of the sanctity of the oath, and bring our system into disrepute."[44] This rule has been described as "a very valuable restraint on reckless or perjured litigants."[45] In Dapelo v. Banco Nacional De Mexico, No. 91 Civ. 0093 (JSM), 1993 WL 159943, at * 1 (S.D.N.Y. May 11, 1993), the court began its opinion as follows:

> "It has often been said that litigation has at its heart the search for the truth. For that reason, witnesses are sworn to tell the truth and may be prosecuted for perjury if they do not. This case raises the question of what consequences should follow when it is determined that a plaintiff relies on a version of the 'truth' to support a claim in one litigation which is irreconcilable with a version of the truth to which she swore in a contemporaneous, but unrelated proceeding."

The plaintiff had testified in an arbitration proceeding that she had limited education and financial acumen, and then attempted to testify in an employment discrimination suit in court that she was a college educated financial consultant. The court granted the defendant summary judgment:

> "The question presented here is whether plaintiff should now be [] allowed to attempt to persuade a jury that, despite her prior testimony in the arbitration, she is, in fact, a college educated financial accountant. The answer to that question is 'no.'" Id.

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[44] Melton v. Anderson, 32 Tenn. App. 335, 343, 222 S.W.2d 666, 669 (1948).

[45] Sartain v. Dixie Coal & Iron Co., 150 Tenn. 633, 649, 266 S.W. 313, 317 (1924).

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The same result must obtain here. OCF has repeatedly called its agents and officers -- including the person in charge of OCF's asbestos litigation who has been named as a witness in this arbitration -- to take an oath and to testify to the truth of the fact that, aside from quantified indemnities from identified insurers, OCF itself -- and itself alone -- has to earn all the money for and pay all its asbestos liabilities. In this proceeding, OCF will attempt to cause other of its agents and officers to testify under oath to the truth of the fact that Owens-Illinois has contractually agreed to indemnify OCF for a substantial portion of OCF's asbestos liability. These contrary claims are being made simultaneously. These diametrically opposite sworn statements of the "truth" are contemporaneous. This Panel cannot and should not permit OCF to proceed at all with its claim.

POINT VI

OCF CANNOT OBTAIN THE RELIEF SOUGHT

OCF's claim blithely assumes that OCF is entitled to an indemnity merely by showing that asbestos claims were settled. OCF ignores fundamental principles of indemnity law.

A. OCF Cannot Seek Indemnity for Claims Based on Tort Theories Other than "Warranty"

OCF urges the Panel to construe the "breach of all warranties" clause in paragraph 6 as an indemnity for third-party "implied warranty" bodily injury tort claims asserted by end users or bystanders. However, OCF knows full well that suits are not based just on "breach of warranty." Rather, the lawsuits against OCF (and all other

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asbestos [redacted] are premised mainly on an alleged failure to warn and invariably assert multiple causes of action -- strict liability, negligence, gross negligence, willful, wanton, and reckless misconduct, and even violations of trade practice statutes. In other words, OCF seeks to be indemnified under the "breach of all warranties" clause for tort liability not premised on warranty. Indemnity law precludes this effort to broaden the word "warranties."[46]

OCF argues that it can proceed because an indemnitor must indemnify if the "settlement or judgment is based at least in part on causes of action for which the indemnitor is liable." OCF 9/22/94 Reply at 14. This may be the rule in the context of insurance contracts -- but the 1958 Asset Sale Agreement is an asset purchase agreement, not an insurance contract. In this context, an indemnitor is only required to indemnify an indemnitee for that, and only for that, which the indemnitor has specifically contracted to

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[46] OCF speciously contends that implied warranty claims are "similar" to, or have been superseded by, strict liability tort and argues that therefore the last clause of paragraph 6 must also cover indemnity claims for OCF's liability for strict liability claims. This argument has multiple flaws. First, as explained above, the implied warranty claims which are similar to strict liability claims did not exist in Ohio in 1958 but rather were created in the 1966 Lonzrick decision. Second, the 1958 Asset Sale Agreement only indemnified OCF for pre-existing "warranties." The elements of an implied warranty tort claim differ from those of a strict liability claim, and Owens-Illinois did not indemnify OCF for any claims that OCF might assert resemble or are analogous to a warranty claim. See Altman v. Strong's Marineland, 180 A.D.2d 777, 778, 580 N.Y.S.2d 425, 426 (2d Dept. 1992) (stating that an indemnity which promised to "hold harmless ... Buyer from any claim ... arising out of ... any warranty of Seller" "did not impose upon [Seller] the obligation to indemnify [Buyer] with respect to negligence and strict products liability claims."). In all events, even if Owens-Illinois agreed to indemnify OCF for "strict liability" (which is not what the contract says), OCF still cannot explain how Owens-Illinois agreed to indemnify OCF for negligence claims or statutory claims, etc. OCF also contends that Owens-Illinois has a duty to defend OCF, but that, like the duty to indemnify, is limited by the scope of the indemnity. Since there is no duty to defend described in the indemnity provision, Owens-Illinois has no such duty. See Linkowski v. General Tire & Rubber Co., 53 Ohio App. 2d 56, 62, 371 N.E.2d 553, 557 (1977).

provide. Black letter law requires that OCF must prove that the asbestos claims for which OCF seeks to be indemnified were "warranty" claims, as the language of that clause specifically requires.[47] Failure to establish that the claim for which indemnification is sought is within the scope of the indemnity agreement results in denial of the indemnity claim.[48]

OCF cannot demonstrate that settled claims were only "breach of warranty" tort claims. Nor can OCF demonstrate that portion of any settled cases that was for the plaintiff's pre-1958 exposure as compared to post-1958 exposure or that portion of the settlement OCF paid to resolve a compensatory claim as opposed to a punitive damage claim. To the contrary, OCF has in case after case emphasized to the courts that some unknown portion of every settlement is for punitive damages.

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[47] "In an action to recover indemnity under an express contract, the burden is on the indemnitee to prove that the liability for which he has been charged is within the scope of the agreement." 41 Am Jur 2d § 43 at 732 (1968); see also Becker v. Central Tel. & Utils. Corp., 393 F. Supp. 1357, 1358-59 (D.S.D. 1975) ("In seeking to prevail on its indemnity claim, [the indemnitee] must of course meet the usual burden of proof imposed on any civil claimant ... [the indemnitee] must prove that its liability to the plaintiffs is based on conduct falling within the scope of the indemnity clause of the contract"); accord Illinois C. G. R. Co. v. International Paper Co., 889 F.2d 536, 543 (5th Cir. 1989); Pennsylvania R. R. Co. v. Indiana H. B. R. Co., 159 F. Supp. 19, 25 (N.D. Ill.), aff'd, 261 F.2d 939 (7th Cir. 1958). This requirement applies equally to those cases where the indemnitee settles the claim. See Illinois C. G. R. Co., 889 F.2d at 539; Peter Culley & Assocs. v. Superior C., 13 Cal. Rptr. 2d 624, 630 (1992). It is only where companies are in the business of selling indemnification (i.e., insurers) that the rule is different.

[48] See Ruddy v. New York Cent. R. R. Co., 224 F.2d 96, 99-100 (2d Cir. 1955) (L. Hand, J.), cert. denied, 350 U.S. 884 (1955); Becker v. Central Tel. & Utils. Corp., 393 F. Supp. 1357, 1358-59 (D.S.D. 1975); Pennsylvania R. R. Co. v. Indiana H. B. R. Co., 159 F. Supp. 19, 25 (N.D. Ill.), aff'd, 261 F.2d 939 (7th Cir. 1958).

B. OCF Cannot Demonstrate Actual Liability Even for Warranty Claims

From the 1960's, when OCF received its first asbestos lawsuits and workers compensation claims, to the 1990's, when OCF had settled over 100,000 asbestos claims, OCF never gave Owens-Illinois any notice of the purported indemnity claim. Where an indemnitee settles a claim without first providing notice or tendering defense to the putative indemnitor, the putative indemnitor does not have any obligation unless and until the putative indemnitee proves its "actual liability" to the claimants paid.[49] This common sense rule is designed to protect the putative indemnitor. Without it, the indemnitee has no incentive to act in the indemnitor's interests because financial responsibility ultimately falls on the indemnitor.[50] Accordingly, where notice is not given, to obtain indemnity the putative indemnitee must (1) produce evidence supporting each element of the claimant's cause of action against the indemnitee and (2) show that

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[49] This rule applies even where the contract at issue does not specifically require notice and a tender of the defense. 42 C.J.S. § 24 (1991) ("Where the indemnity clause in question is silent on certain issues, such as the issue of tender and notification before settlement ... then equitable principles of indemnity apply. Under equitable principles of indemnity, in order for a settling indemnitee to support his claim, he must prove actual liability to the original plaintiff and that the amount paid in settlement was reasonable."); see also GAB Business Servs., Inc. v. Syndicate 627, 809 F.2d 755, 760 (11th Cir. 1987) ("when an indemnitee has not given the indemnitor an opportunity to review, pass upon, or participate in the settlement, due process and 'equitable indemnity principles' compel a demonstration to actual as opposed to potential liability") (citations omitted); The Toledo, 122 F.2d 255, 257 (2d Cir.) ("A claim for indemnity, however, requires that an actual liability be sustained by the indemnitee, and if he settles a claim without a determination of the rights in question, he bears the risk of proving an actual liability in the action over for indemnity."), cert. denied, 314 U.S. 689 (1941); accord, e.g., L. B. Kaye Assocs., Ltd. v. Libov, 139 A.D.2d 440, 440, 527 N.Y.S.2d 216, 217 (1988); Crystal River Enters., Inc. v. Nasi, Inc., 399 So. 2d 77, 79 (Fla. Dist. Ct. App. 1981).

[50] See Feuer v. Menkes Feuer, Inc., 8 A.D.2d 294, 300, 187 N.Y.S.2d 116, 122-23. (1st Dep't. 1959).

[redacted] to the claim. In other words, the indemnitee would have to establish its case against the indemnitor on the same way that the underlying claimant would have been obligated (but for the settlement) to establish its case against the indemnitee.[51]

OCF cannot meet this standard. OCF will be able to show few, if any, settled cases in which it was actually liable to any plaintiffs for a "breach of warranty" for Kaylo delivered prior to May 1, 1958. The Panel will hear about OCF's settlement practices, such as paying a sum of money to settle hundreds of cases despite the fact that OCF has no precise idea how much each of the individual plaintiffs actually receives. The Panel will also hear that OCF settles cases without requiring claimants to establish the specific dates of their exposure, that they were actually exposed to and harmed by an asbestos-containing product manufactured by Owens-Illinois, or sometimes even whether the claimant really suffers from an asbestos-related disease. These settlement practices prove that OCF certainly cannot demonstrate "actual liability" to claimants.

OCF's claimed right to indemnity is not a hypothetical construct. If the right exists, it does so only in the context of individual personal injury claims filed against, and resolved by, OCF. Unless and until a specific plaintiff has suffered an injury caused by a "breach of warranty" concerning Kaylo manufactured by Owens-Illinois and sold by OCF, and OCF has paid the plaintiff a sum of money linked to that

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[51] See GAB Business Servs., Inc. v. Syndicate 627, 809 F.2d 755, 760 (11th Cir. 1987).

[52] Feuer, 8 A.D.2d at 299-300, 187 N.Y.S.2d at 122-23.

distinct portion of the plaintiff's claim, OCF has not established a right to the alleged indemnity.

OCF has the burden in this proceeding of proving the facts that give rise to a right to indemnification on a case-by-case basis. OCF cannot do so. Asbestos-related personal injury claims are not sub-divisible in that fashion. In almost none of the tens of thousands of claims resolved by OCF was 1953-58 Kaylo the only OCF asbestos-containing product to which the plaintiff claimed exposure. The norm in this litigation is alleged exposure to dozens of products, including Kaylo, throughout a working career that extends well beyond 1953-58. OCF can virtually never prove with the requisite certainty that it has paid an identifiable sum of money to any plaintiff due to an alleged breach of warranty concerning 1953-58 Kaylo.

Further, even if asbestos plaintiffs pursued breach of warranty claims, OCF cannot obtain indemnity because OCF would have valid defenses to "breach of warranty" claims in most jurisdictions. Warranty claims are, and have been for many years, time-barred. Most courts rule that the statute of limitations for breach of warranty claims is four years from tender or delivery of the goods, as set forth in the Uniform Commercial Code.[53] Therefore, breach of warranty claims brought more than four years after "tender or delivery" of the Kaylo to which the claimant was exposed are

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[53] U.C.C. §§ 2-725(1), (2). See, e.g., Rosenberg v. Celotex Corp., 767 F.2d 197, 198 n.4 (5th Cir. 1985) (New York law); Iida v. Allied Signal (In re Hawaii Fed. Asbestos cases), 854 F. Supp. 702, 708-09 (D. Haw. 1994); Johnson v. Hockessin Tractor, Inc., 420 A.2d 154, 158 (Del. 1980); Berry v. G.D. Searle & Co., 56 Ill. 2d 548, 554, 309 N.E.2d 550, 554 (1974). Since "strict liability" claims do not face similar statutes of limitations, this is yet another reason why OCF's contention that Owens-Illinois agreed to indemnify it not only for warranty claims but for other claims, such as strict liability claims, must fail.

barred. Accordingly, OCF could not settle even warranty claims and obtain indemnity because OCF would have no valid defense to such claims.

POINT VII

OWENS-ILLINOIS IS ENTITLED TO AWARDS OF DAMAGES AND DECLARATORY RELIEF

Owens-Illinois acknowledges that, but for OCF's filing of this arbitration, Owens-Illinois likely would have been satisfied to continue its litigation of asbestos personal injury actions on the basis originally suggested by Carl Stiefin in 1975 -- each company assuming responsibility for its own cases. Owens-Illinois believes, that the record of the past 30 years -- including OCF's unjustifiable failure to provide notice of its current contractual indemnity claim, its representations and conduct inconsistent with such a claim, and the resulting prejudice to Owens-Illinois -- forever bars OCF's claim. Nonetheless, if this Panel credits that claim despite the immutable record of OCF's actions and inaction, then, in addition to declaratory relief, Owens-Illinois should receive an award on its counterclaim for damages. We show below why, if OCF's claim is not barred, Owens-Illinois is itself entitled to the relief sought.

A. Owens-Illinois Is Entitled to an Award of Damages for OCF's Wrongful Conduct

A partial outline of the evidence concerning the numerous wrongful acts by OCF has been set forth above. OCF's acts and misconduct substantially increased OCF's liability to asbestos tort plaintiffs, and therefore OCF is precluded from obtaining any indemnity claim from Owens-Illinois. However, OCF's acts and misconduct have also substantially increased Owens-Illinois' liability. Owens-Illinois' third counterclaim seeks

damages for the harm OCF has inflicted on Owens-Illinois as a result of OCF's wrongful conduct in derogation of OCF's obligation as an indemnitee under the 1958 Asset Sale Agreement.

That OCF's actions harmed Owens-Illinois is unquestionable. First, OCF's conduct in such matters as changing the composition of Kaylo increased the exposure to asbestos dust of those persons who used Kaylo. There is a direct relationship between the duration and intensity of exposure to asbestos fibers and the incidence and severity of asbestos-related disease or impairment. OCF's conduct increased both the chance that Kaylo users would develop an asbestos-related injury and the severity of the asbestos-related diseases and impairments that users developed. In short, OCF's conduct increased both the number of claims and the severity of the injuries, thereby increasing the costs of the claims to Owens-Illinois because claimants routinely sue both OCF and Owens-Illinois.

Second, by failing adequately to warn of the health risks associated with asbestos that were discovered in the 1960's, by failing thereafter to use dust suppressants such as Ludox, and by failing to market asbestos-free substitutes to Kaylo such as Multitemp, OCF deliberately did not reduce the harms.

These deliberate acts and omissions by OCF increased the exposure not just to asbestos dust from OCF Kaylo but to dust from other asbestos products as well. As the Panel will hear, the announcement of Dr. Selikoff's results of his study in 1964 caused wide-spread apprehension to those then in the insulation business about the health effects of asbestos-containing products. OCF was aware of Dr. Selikoff's work,

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OCF had the engineering and technical ability to offer the market an asbestos-free insulation product in the mid-1960's. It had already developed and successfully marketed such an alternative (Multitemp) and had the means and knowledge to introduce an asbestos-free Kaylo. OCF did not pursue either option. Instead, OCF decided Multitemp's profit levels were insufficient and terminated the product, and failed to use its available resources and knowledge to introduce an asbestos-free Kaylo. OCF's conscious decision to abandon Multitemp in 1966 was made with knowledge of the health risks of asbestos. As a 1966 memorandum from OCF's Product Development Laboratory warned, "[t]his health hazards thing is closing in on us."[54] Had OCF, a dominant force in this insulation market, offered an asbestos-free product in the years after Dr. Selikoff's 1964 study, the market would have embraced it, just as the market did when such a product was finally marketed in the early 1970's. That would have meant less exposure to asbestos for thousands of people, thereby decreasing both the number of asbestos claimants which have sued Owens-Illinois and the severity of their diseases.

According to OCF, it is an indemnitee under the 1958 Asset Sale Agreement.[55] It is well-established that an "indemnitee has a duty to act reasonably

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[54] Significantly, Kaylo's profitability to OCF was reaching record levels during the years that OCF refused to market or develop asbestos-free substitutes.

[55] As Owens-Illinois argued to the Panel, the Panel must first construe the terms of the 1958 Asset Sale Agreement before addressing OCF's position that there is no jurisdiction over Owens-Illinois' counterclaims.

under all the circumstances, to provide the indemnitee against liability. OCF had a legal obligation to Owens-Illinois, not to engage in conduct which would harm Owens-Illinois. OCF's obligation not to harm Owens-Illinois after OCF purchased the Kaylo division was especially important here because, pursuant to the 1958 Asset Sale Agreement, OCF took control of a product, Kaylo, whose name had previously become linked with Owens-Illinois' name. OCF's conduct did in fact harm Owens-Illinois, in violation of OCF's obligations to Owens-Illinois. Accordingly, Owens-Illinois is entitled to the damages it has suffered caused by those violations.

B. Owens-Illinois Is Entitled to Declaratory Relief

Owens-Illinois first seeks a declaration that the 1958 Asset Sale Agreement does not give OCF any right to be indemnified for asbestos-related tort claims, including those claims previously asserted against and resolved by OCF (by settlement or judgment), those claims now pending against OCF, and those claims to be brought against OCF in future years. As set forth above, the 1958 Asset Sale Agreement never provided for an indemnification for these claims and, in any event, OCF's conduct since 1958 precludes any claim for such indemnification (even assuming such a right ever existed).

Owens-Illinois is also entitled to declaratory relief that OCF is not entitled to recover from Owens-Illinois under any common law or other theory, such as common

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[54] American Export Isbrandtsen Lines Inc. v. United States, 390 F. Supp. 63, 68 (S.D.N.Y. 1975) (Weinfeld, J.); see also Rochelle Bail Agency, Inc. v. Maryland Nat'l Ins. Co., 484 F.2d 877, 878-79 (7th Cir. 1973); American Casualty Co. v. Idaho First Nat'l Bank, 328 F.2d 138, 142-43 (9th Cir. 1964); Hiern v. St. Paul-Mercury Indem. Co., 262 F.2d 526, 529 (5th Cir. 1959); Unisys Corp. v. Legal Counsel, Inc., 768 F. Supp. 6, 8 (D.D.C. 1991).

law or statutory contribution or common law indemnity, any of the amounts OCF has paid or will pay to asbestos claimants. Owens-Illinois is entitled to this relief because where parties, like Owens-Illinois and OCF here, have contractually agreed between themselves to allocate the economic risks of their activity, that contractual allocation of risk overrides (as between the two parties) otherwise applicable rights of indemnity or contribution.[37] This declaratory relief is appropriate because the 1958 Asset Sale Agreement, and in particular the last clause of paragraph 6 upon which OCF bases its claim, will be interpreted by the Panel here and should not be reinterpreted in subsequent cases; because the rights and responsibilities of the parties were set forth in the two contracts which will be put before the Panel; and because such declaratory relief is merely the embodiment of the equitable equivalent for the future to the monetary relief for the past to which Owens-Illinois is entitled under the second and third counterclaims.

C. Owens-Illinois Is Entitled to Damages for OCF's Breaches of the 1953 Master Product Sale Contract

As discussed above, the 1953 Master Product Sale Contract contained detailed contractual provisions designed to protect Owens-Illinois from "special or consequential damages" arising out of its sale of Heat Insulating products to OCF. OCF's contractual obligation not to do anything to make Owens-Illinois liable "in any event for special or consequential damages" is strengthened by the 1958 Master Product Sale Contract's requirement that OCF include the same limitation on Owens-Illinois'

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[37] E.g., INA Ins. Co. v. Valley Forge Ins. Co., 722 P.2d 975, 979, 150 Ariz. 248, 252 (Ct. App. 1986) (citing authorities); Wyoming Johnson, Inc. v. Stag Indus., Inc., 662 P.2d 96, 101-02 (Wyo. 1983) (citing numerous authorities); Liberty Mut. Ins. Co. v. Paris, No. 66719, 1994 WL716545, at *17-19 (Ohio App. Dec. 22, 1994) (Ohio law).

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liability in OCF's resale contracts and to indemnify Owens-Illinois for OCF's failure to do so. Thus, in addition to agreeing that Owens-Illinois' shall not "in any event" have liability for "special or consequential damages," the 1953 Master Product Sale Contract required OCF to take affirmative measures to ensure that Owens-Illinois was not subject to such "special or consequential damages."

After OCF assumed this contract,[58] OCF breached the specific contractual obligations set forth above at numerous times. These breaches began in the 1960's when such OCF actions as changing the composition of Kaylo and failing to use a substitute like Multitemp increased the likelihood that Owens-Illinois would get sued. Similarly, in 1979, OCF began filing third-party actions against Owens-Illinois in the Philadelphia asbestos cases in which OCF, but not Owens-Illinois, was named as a defendant. These third-party complaints sought to impose some or all of OCF's actual or potential liability for special or consequential damages to the asbestos claimants on Owens-Illinois, because Owens-Illinois manufactured and sold the Kaylo to OCF -- under the 1953 Product Sale Master Contract! Such conduct directly violated OCF's agreement that Owens-Illinois shall not "in any event" be liable for "special or consequential damages." But OCF did not stop there. As the Panel will hear, OCF actually contacted asbestos personal injury lawyers and successfully encouraged them to sue Owens-Illinois. In other words, rather than take affirmative steps to limit Owens-Illinois' exposure, OCF did the opposite -- in a bad faith violation of OCF's agreements in the 1953 Master Product Sale Contract.

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[58] As noted previously, Owens-Illinois argued that this Panel must first construe the 1958 Asset Sale Agreement and its relationship with the 1953 Master Product Sale Contract.

Sale Contract was one of the contracts assigned by the 1958 Asset Sale Agreement.[40] As we have shown above, this position does not make practical sense. However, if the Panel so construes the 1958 Asset Sale Agreement, then OCF has to indemnify Owens-Illinois for its breaches of the (assigned) 1953 Master Product Sale Contract which have led to third-party claims against Owens-Illinois. As discussed, under paragraph 6 of the 1958 Asset Sale Agreement, Owens-Illinois assigned and OCF assumed certain executory contracts of the Kaylo division. Under paragraph 6, in addition to the indemnities running to Owens-Illinois, OCF itself owed Owens-Illinois an indemnity: to "save Owens-Illinois harmless from any and all claims of any third person or persons for any breach, after assignment thereof, of any agreement so assigned." In other words, if OCF assumed an executory contract and thereafter failed to abide by any obligations, OCF agreed to indemnify Owens-Illinois for claims of any and all third persons. Thus, if the 1953 Master Product Sale Contract were (under OCF's construction) assigned under paragraph 6, then OCF would be liable under the 1958 Asset Sale Agreement to Owens-Illinois for OCF's breaches of the 1953 Master Product Sale Contract after May 1, 1958 that lead to third-party claims against Owens-Illinois.

Consequently, based on the assumption set forth above, Owens-Illinois' counterclaim seeks the damages it has incurred, including defense costs and amounts paid in settlements or judgments, for OCF's breaches of the 1953 Master Product Sale Contract either (1) as an offset to OCF's claim here or (2), assuming the Panel accepts

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[40] Owens-Illinois argued in opposition to OCF's motion to dismiss the counterclaims for lack of jurisdiction that the 1958 Asset Sale Agreement must first be construed. Were the Panel to construe paragraph 6 as OCF urged and as Owens-Illinois demonstrated in the alternative, this result would obtain.

ANNUAL REPORT

FOR THE YEAR ENDED DECEMBER 31

1938

OWENS-ILLINOIS

GLASS COMPANY

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OWENS-ILLINOIS GLASS COMPANY

PATENTS

Our Company has been the pioneer in the development of automatic bottle blowing machinery and related equipment and in the manufacture and commercial adaptation of glass building block, glass wool insulation, and other Fiberglas products. Improvements in our bottle blowing machinery have been or are being patented and constitute a valuable asset, as do our basic and improvement patents, which cover important contributions of our engineers and inventors to the fields of glass building block and glass wool, fibers, plastic molding machines, etc. We are continuing our policy of encouraging inventions and improvements in the above and closely allied fields and obtaining patent protection thereon commensurate with the value of the improvements.

OWENS-CORNING FIBERGLAS CORPORATION

On November 1, 1938, the Company sold to Owens-Corning Fiberglas Corporation its entire assets formerly used in the development and production of glass fiber products, including its manufacturing plant in Newark, Ohio. Corning Glass Works likewise sold its Fiberglas assets and its Fiberglas plant in Corning, New York, to the same company.

Owens-Illinois Glass Company and Corning Glass Works each received 49.77% of the common stock of Owens-Corning Fiberglas Corporation for the assets so transferred and, in addition, Owens-Illinois Glass Company received 17,856 shares of the non-voting, cumulative preferred stock of the new corporation of the par value of $100 per share. This investment is included in the balance sheet in "Other security investments."

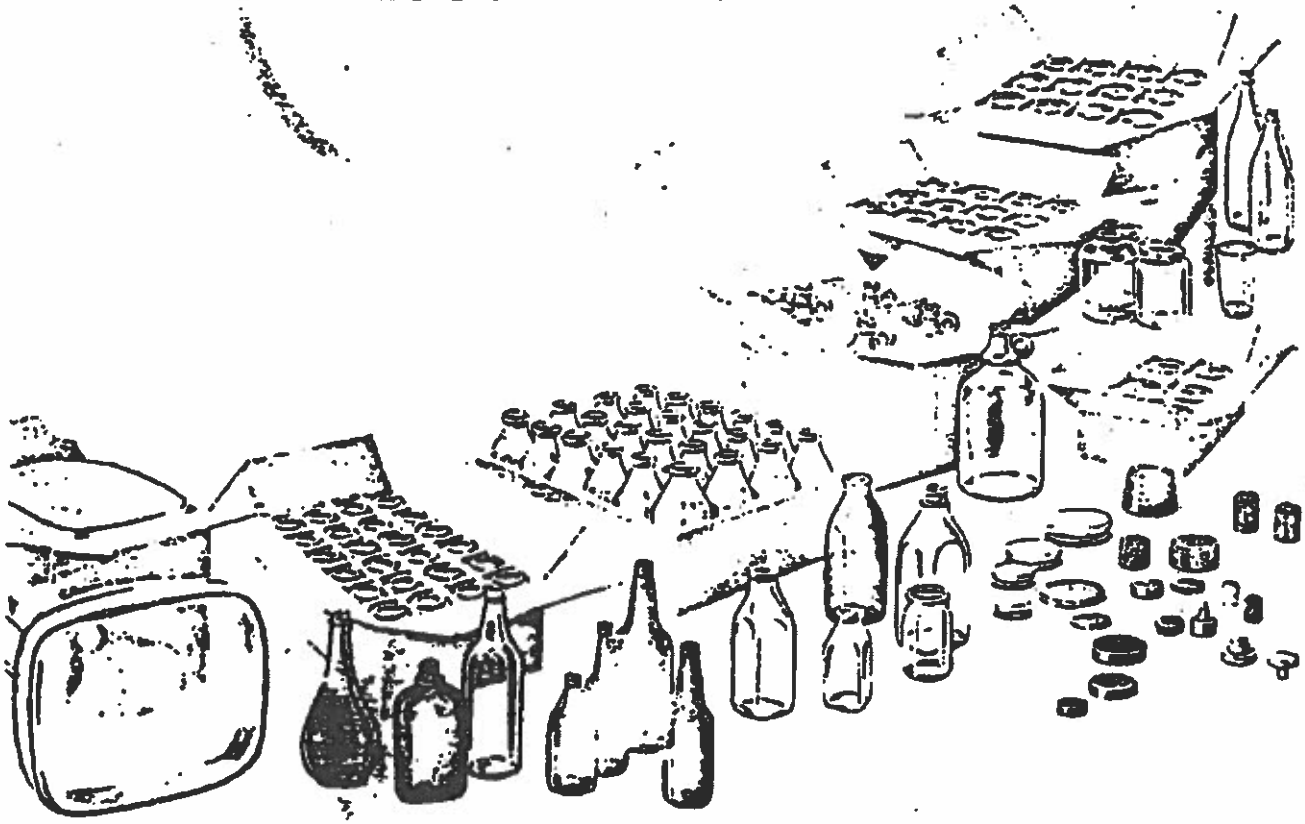
The general office of the new corporation is in Toledo, and sales offices are maintained in New York, Chicago, and other principal cities. While the Company is jointly owned and jointly financed by Owens-Illinois and Corning, it will not operate as a subsidiary of either.

The operations of the glass fiber division of Owens-Illinois Glass Company's business for the first ten months of 1938 are reflected in the consolidated figures given in this report. Beginning with November 1, 1938, Fiberglas operations were no longer included in the Company's consolidated figures, having been transferred to the new corporation. The operations of Owens-Corning Fiberglas Corporation will be reflected in our Company's earnings only to the extent of such dividends as may be received from this stock investment.

INSULUX PRODUCTS DIVISION

As a result of the sale of the glass fiber assets, the division formerly known as the Industrial & Structural Products Division has been reorganized and is now designated as the Insulux Products Division. It will comprise the Company's business in the development, manufacture, and sale of Insulux glass blocks and Hemingray insulators. Glass blocks and insulators will continue to be produced in the Muncie, Indiana, plant of Owens-Illinois Glass Company.

OWENS-ILLINOIS



OWENS-ILLINOIS GLASS COMPANY

1956 Annual Report

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Notes to Financial Statements (Continued)

2. SECURITIES:

The listed securities owned at December 31, 1956 consisted of the following:

	Common Shares	Cost	Quoted Market Value
Owens-Corning Fiberglas Corporation	1,235,859	$ 1,437,507	84,347,377
Continental Can Company	417,886	10,274,321	19,901,821
Container Corporation of America	270,000	467,227	5,163,750
Pennsylvania Glass Sand Corporation	37,214	328,200	2,232,840
		$12,507,253	$111,645,788

The shares of Continental Can Company are represented by a voting trust certificate for 522,358 common shares of Robert Gair Company, Inc. (See page 6).

The common stock of Owens-Corning Fiberglas Corporation, shown in the investment section of the balance sheet at a cost of $891,997, consisted of 864,141 shares having a market value of $58,977,623 at December 31, 1956. This stock is reserved for exchange of preferred shares as explained in Note 6.

The investment in Plax Corporation is represented by 250,000 shares of Class B common stock which is convertible into Class A common stock under certain conditions. This represents 50% of its outstanding capital stock. The Company's equity in the net assets of Plax amounted to $6,049,430 at December 31, 1956, and its equity in earnings for 1956 was $226,098.

3. FOREIGN SUBSIDIARIES:

The accounts of five newly organized foreign subsidiaries have not been consolidated. At December 31, 1956, the Company's equity in the net assets of these subsidiaries was approximately equal to its investments and advances. The subsidiaries had no significant operations in 1956.

4. LONG-TERM DEBT:

The indenture relating to the 4¼% and 4¼% debentures provides for a fixed sinking fund payment of $2,000,000 annually in the years 1957 and 1958, increasing gradually to $2,400,000 in 1965, with smaller amounts through 1969, and for a contingent sinking fund payment annually commencing April 20, 1958, in an amount equal to 25% of consolidated net earnings for the preceding fiscal year less certain credits as defined in the indenture.

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Notes to Financial Statements (Continued)

5. DEFERRED INCOME TAXES:

The Valdosta mill of National Container Corporation was constructed under a certificate of necessity. The certified portion of the cost of the mill is being amortized for income tax purposes over a period of sixty months beginning March 1, 1954. Normal depreciation has been recorded in the accounts and, concurrently, deferred income taxes have been provided in amounts equal to the reduction in income taxes currently payable because of the excess of amortization over book depreciation.

6. PREFERRED SHARES:

The preferred shares are redeemable after March 31, 1959 at $105 per share if redeemed during the year commencing April 1, 1959, the redemption price reducing 25¢ per share each subsequent year to $100 per share if redeemed after March 31, 1979.

On and after October 1, 1958, but prior to October 1, 1968, the preferred shares may be surrendered for retirement at the option of the holders in exchange for common stock of Owens-Corning Fiberglas Corporation held by the Company in the ratio of 1.05 shares of Fiberglas common for one preferred share if surrendered prior to October 1, 1963, and share for share if surrendered thereafter. The Company has segregated and reserved 864,141 shares of common stock of Fiberglas for exchange at the initial rate.

Commencing on May 1, 1962, the Company will be obligated, to the extent the net consolidated earnings of the next preceding fiscal year are sufficient, to set aside as a purchase fund the sum of $2,000,000 annually. The purchase fund is to be used for the purchase of preferred shares on the open market at not to exceed $100 per share, plus commissions, taxes and other incidental costs of acquisition. If any purchase fund installment is not so expended during the year following the date on which the installment was set apart, the balance shall be available for general corporate purposes of the Company.

7. STOCK OPTIONS:

199,200 common shares are reserved for issuance under a restricted stock option plan which is described on page 18.

8. SURPLUS RESTRICTIONS:

The merger agreement contains certain restrictions on the payment of dividends on common shares and the purchase of the Company's common shares while any of the 4% preferred shares are outstanding. At December 31, 1956, the amount of earned surplus so restricted was $77,914,407. The agreements relating to the long-term debt contain similar provisions which were less restrictive at December 31, 1956.

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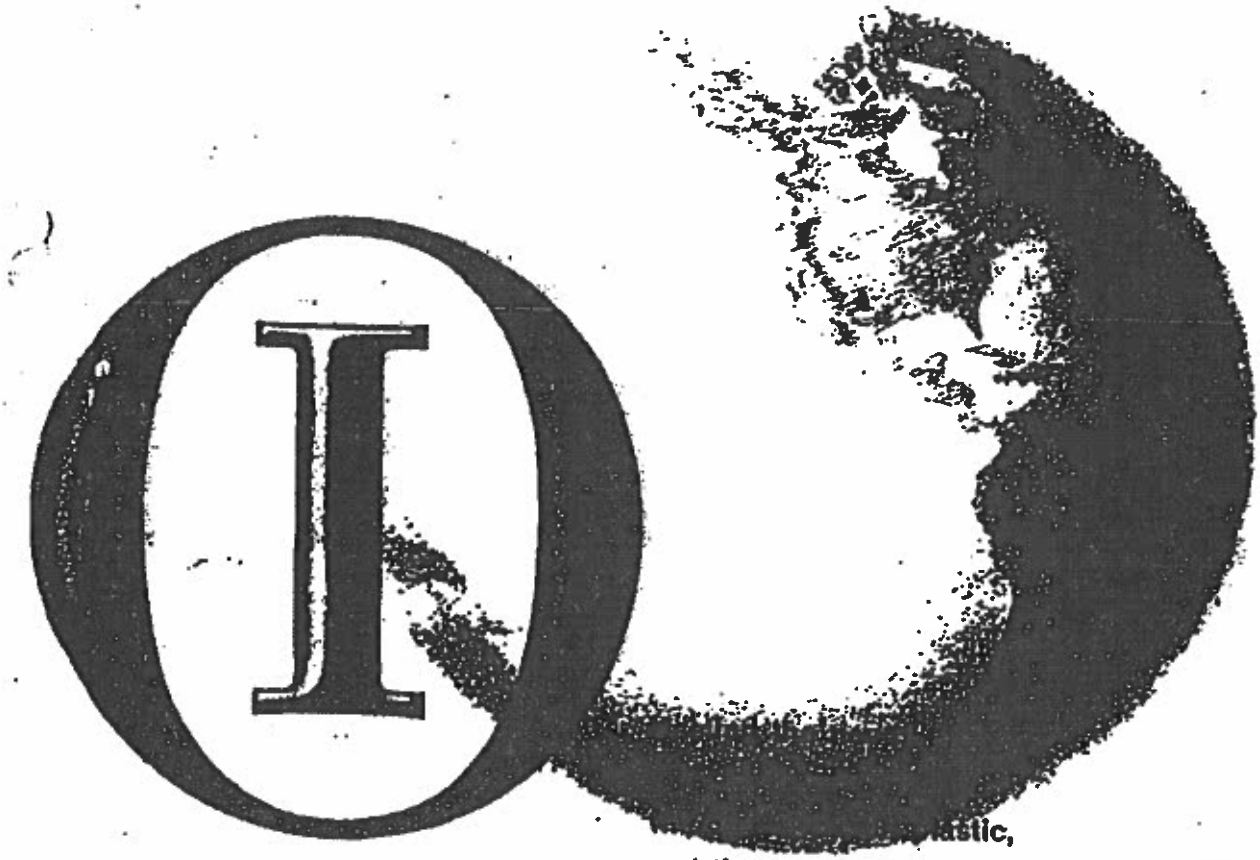
OWENS-ILLINOIS

ANNUAL REPORT 1968



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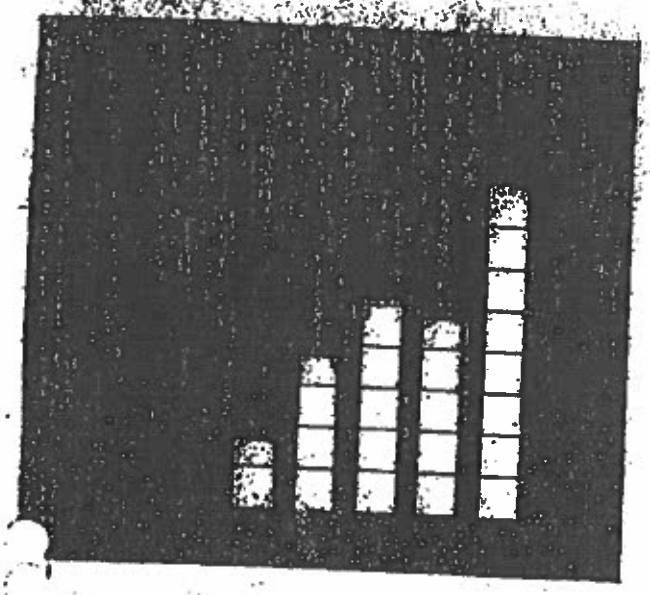

Income Taxes, Including Deferred Taxes

The 10% Federal surtax penalized earnings by $3.7 million or 23 cents per share.

The investment tax credit totaled $4.1 million in 1968, a substantial decline from the $7.1 million of 1967 which included the credit on the containerboard mill at Orange, Texas. Such credits have reduced provisions for taxes currently due.

Deferred tax accounting is employed in all major applicable areas. The deferred income taxes shown in the balance sheet are summarized as follows:

	1968	1967
	(Thousands of Dollars)	
Tax effect of costs deducted for tax purposes but not in the financial statements (principally fixed assets and depreciation)	$53,569	$43,324
Tax effect of provisions for costs and losses not yet deductible for tax purposes (principally provision for rebuilding furnaces)	(10,555)	(9,464)
Provision for future taxes, principally U. S. taxes on undistributed earnings of certain foreign subsidiaries	2,273	2,283
	$45,287	$36,143

In addition to the above, a minor net amount of deferred taxes is included in working capital. The net charge to earnings for current deferred taxes was $9.0 million in 1968 and $7.0 million in 1967.

Domestic Investments

Investments at the end of the year in securities of the following companies listed on the New York Stock Exchange were:

	Shares	Cost	Quoted Market Value
		(Thousands of Dollars)	
Owens-Corning Fiberglas Corporation, common	1,814,484	$2,040	$143,344
International Telephone and Telegraph Corp.:			
Common	46,741	179	2,723
Preferred Series I	20,096	149	2,211
Marcor Inc., preferred	2,000	17	112
		$2,385	$148,390

The 1,814,484 shares of Owens-Corning Fiberglas Corporation represent approximately 25.1% of the common stock of that company. During 1968, 285,443 shares of Owens-Corning were exchanged for 4% preferred shares of Owens-Illinois (see Preferred Shares). Based on shares outstanding at December 31, 1968, the Company's equity in the net assets of Owens-Corning at that date amounted to $56.7 million and the equity in 1968 earnings was $4.9 million.

The shares of International Telephone and Telegraph Corporation and Marcor Inc. were received in 1968 as the result of business combinations, in exchange for shares of Pennsylvania Glass Sand Corporation and Container Corporation of America previously held. Of 20,000 preferred shares of Marcor Inc. received, the Company later disposed of 18,000 shares.

Cash dividends of $3.0 million were received on listed stocks (principally Owens-Corning Fiberglas Corporation) in 1968 and were equivalent to 17 cents a share after taxes.

The Company also holds an investment in Alton Box Board Company, an unlisted company, represented by 77,000 common shares (about 9% of the total), warrants to subscribe for 73,000 additional shares at $36 per share, exercisable until 1975, and a 5½% subordinated note for $7 million due April 16, 1980. The stock and warrants have been deposited with St. Louis Union Trust Company as voting trustee. The Company is obligated to sell the deposited shares by 1975 and shares acquired upon exercise of the warrants within ten years after acquisition.

Domestic investments are carried at cost except National Petro Chemicals Corporation and Owens, Libbey-Owens Gas Department, which are 50% owned, and Red River Paper Mill, Inc. which is 75% owned but not controlled, each of which is carried at equity in net assets. Investments amounted to $4.0 million for National Petro Chemicals Corporation, $1.3 million for the Gas Department, and $3.8 million for Red River Paper Mill, Inc., at December 31, 1968. The equity in earnings of these associates was $1.8 million in 1968.

Foreign Subsidiaries and Investments

In 1968, as in previous years, the financing of foreign operations has been developed in close coordination with the U. S. Government's balance-of-payments program. To the fullest extent feasible, the funds for overseas investment and the operating needs of foreign affiliates have been provided from the cash flow of the foreign operations themselves, by local borrowing, and through direct Eurodollar borrowing by the parent company.

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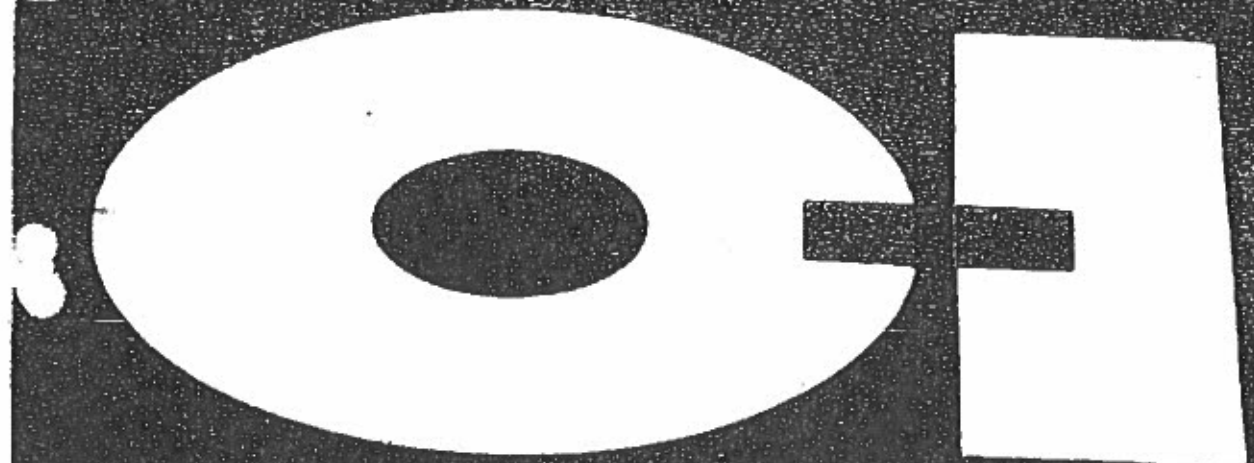
The Owens-Illinois 1978 Annual Report



Cash and Short-Term Investments. Cash and short-term investments at December 31, 1978 and 1977, were as follows:

	Thousands of Dollars	
	1978	1977
Cash	$ 26,903	$29,819
Time deposits	31,594	15,425
Short-term investments, at cost (approximately market)	49,140	7,476
	$107,637	$52,720

Inventories. Major classes of inventory in current assets at December 31, 1978 and 1977, were as follows:

	Thousands of Dollars	
	1978	1977
Finished goods and work in process	$299,231	$257,366
Raw materials and operating supplies	147,673	138,810
	$446,904	$396,176

If inventories valued on the LIFO method had been valued at standard or average costs, which approximate current costs, consolidated inventories would be higher than reported by approximately $102.2 million and $83.6 million at December 31, 1978 and 1977, respectively.

During 1977, certain LIFO inventory quantities were reduced. The reductions resulted in a liquidation of inventories carried at lower costs prevailing in prior years as compared with 1977 costs. The effect of these liquidations decreased costs of goods sold by approximately $7.9 million and increased net earnings by approximately $4.1 million or $.14 per share ($.13 per share, fully diluted). No significant liquidations occurred in 1978.

Investments. Summarized information for consolidated foreign operations is as follows:

	Thousands of Dollars	
	1978	1977
At year-end:		
Assets:		
Current assets	$286,593	$237,386
Property, plant and equipment (net)	291,949	267,022
Other assets	38,451	29,799
	616,993	534,207
Liabilities, except amounts due Owens-Illinois:		
Current liabilities	156,995	139,316
Long-term debt	87,273	57,413
Reserves and other credits	98,522	86,839
	342,790	283,568
Net assets	274,203	250,639
Minority shareholders' interests	43,929	40,118
Owens-Illinois' equity in net assets	$230,274	$210,521
For the year:		
Net sales	$710,752	$591,447
Earnings before extraordinary item and minority shareholders' interests	$ 22,675	$ 18,137
Minority shareholders' interests	6,026	4,379
Net earnings before extraordinary item	16,649	13,758
Dividends received by Owens-Illinois	10,792	12,734
Excess of net earnings before extraordinary item over dividends received	$ 5,857	$ 1,024

Exchange gains and losses for 1978 and 1977 principally resulted from the translation of the accounts of foreign subsidiaries into dollars. Although precise quantification is impractical, it is estimated that these exchange gains and losses were substantially offset during such years as a result of the continuing effects of rate changes and of minority shareholders' interests. The offsets occur as a result of the effects of these rate changes on gross margins due to translation of inventories and depreciation at historical rates, the continuing effect of translation of certain items in the statement of earnings at the current rates and the effect of the minority shareholders' interests in all of such items. Aggregate net exchange losses, before minority shareholders' interests and before the other offsetting effects of rate changes referred to above, were $20.6 million in 1978 and $8.0 million in 1977.

Domestic investments accounted for by the equity method are National Petro Chemicals Corporation (50% owned) and several minor corporate joint ventures. Investments in these companies, which equal equity in underlying net assets, amounted to $14.9 million at December 31, 1978 and $12.5 million at December 31, 1977, including equity in undistributed earnings of $11.0 million at December 31, 1978 and $8.8 million at December 31, 1977. Dividends received amounted to $4.5 million in 1978 and $6.8 million in 1977. Equity in earnings of these companies amounted to $6.9 million in 1978 and $8.4 million in 1977.

Domestic investments at December 31, 1978 and 1977 also includes 752,886 shares and 917,014 shares, respectively, of common stock (restated to reflect the effects of a 1978 two-for-one stock split) of Owens-Corning Fiberglas Corporation at a cost of $230,000 and $278,000, respectively, (market value, 1978—$19,951,000; 1977—$30,376,000) reserved for exchange for Owens-Illinois, Inc. 4½% exchangeable subordinated debentures (see Long-Term Debt).

No. 123895 & No. 124002
(Consolidated)

IN THE SUPREME COURT OF ILLINOIS

JOHN JONES and DEBORAH JONES, Plaintiffs-Appellees, v. PNEUMO ABEX LLC and OWENS-ILLINOIS, INC., Defendants-Appellants.	On Petition for Leave to Appeal from the Appellate Court of Illinois, Fifth Judicial District, No. 5-16-0239. There on Appeal from the Circuit Court of the Second Judicial Circuit, Richland County, Illinois, No. 13-L-21, Hon. William C. Hudson, Judge Presiding.

CERTIFICATE OF SERVICE

I hereby certify that on April 16, 2019, by electronic filing through File & Serve Illinois, I filed the *Brief of Plaintiffs-Appellees as to Owens-Illinois, Inc.* upon the Clerk of the Illinois Supreme Court and further served a copy of same, by electronic filing through File & Serve Illinois, upon counsel for Defendant-Appellants as follows:

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Attorney for Pneumo Abex

I further certify that I shall provide thirteen (13) duplicate paper copies bearing the Clerk's electronic file stamp to the Clerk of the Supreme Court in Springfield, Illinois, within five (5) days of the acceptance of the e-filed document.

Under penalties as provided by law pursuant to Section 1-109 of the Code of Civil Procedure, the undersigned certifies that the statements set forth in this instrument are true and correct.

By: _____

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James Wylder, ARDC #3122244
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