EDISON

BerGenBio

Initiation of coverage

Bemcentinib leading the AXL charge

Pharma & biotech

BerGenBio (BGBIO) is a pioneer in AXL biology and the development of AXL inhibitors: selective tyrosine kinase inhibitor (TKI) bemcentinib and functional blocking monoclonal antibody tilvestamab. AXL expression is a negative prognostic marker in most cancers. Its upregulation drives aggressive disease including drug resistant, immune-evasive and metastatic cancers, as well as fibrosis and viral infection. AXL signalling is the essential mediator of EMT and immune suppression. Selective AXL inhibition can prevent and reverse acquired drug resistance and stop immune suppression, potentially augmenting the efficacy of other cancer drug classes. Bemcentinib, an oral once-a-day pill, has demonstrated efficacy in 2L AML and 2L NSCLC, and could be the first selective AXL inhibitor to market. We value BGBIO at NOK59.1 per share.

Year end	Revenue (NOKm)	PBT* (NOKm)	EPS* (NOK)	DPS (NOK)	P/E (x)	Yield (%)
12/18	2.3	(191.7)	(3.60)	0.0	N/A	N/A
12/19	8.0	(199.3)	(3.43)	0.0	N/A	N/A
12/20e	0.0	(249.1)	(3.09)	0.0	N/A	N/A
12/21e	0.0	(300.9)	(3.45)	0.0	N/A	N/A

Note: *PBT and EPS are normalised, excluding amortisation of acquired intangibles, exceptional items and share-based payments.

Multiple shots to AXL inhibition

Bemcentinib, a highly selectivity AXL inhibitor, works synergistically with other drug classes, with potentiating effects to address drug resistance and immune evasion. Its first approved indication (forecast in 2024) is likely to be in combination with low-dose cytarabine (LDAC) in relapsed AML (FDA fast track designation granted). The opportunity in NSCLC is in our view significant as bemcentinib in combination with Keytruda (CPI) could enable a treatment paradigm shift by addressing PD-1 resistance, an unmet need. Other indications are being explored by investigator sponsored trials, highlighting interest in this novel class. Inhibition of AXL has the potential to be a powerful therapeutic option. However, its success may be predicated on being able to identify tumours with sufficient AXL overexpression.

COVID-19 presents a potential opportunity

Positive headline data from both Pfizer's and Moderna's COVID-19 vaccines could lead to widespread vaccination, but it will take time on a global level and multiple treatment options will be required in the short term. Two Phase II trials are exploring bemcentinib efficacy (in combination with SOC) in hospitalised patients; data are expected by year end. We forecast $300m peak sales in this setting.

Valuation: NOK5.16bn or NOK59.1 per share

We value BerGenBio at NOK5.16bn or NOK59.1 per share based on a risk-adjusted NPV analysis. The key drivers are bemcentinib in 2L NSCLC (peak sales $1.2bn, NOK40.7/share) and AML (peak sales $588m, NOK13.3/share) plus COVID-19 opportunity (NOK5.9/share). We add net cash of NOK 777.9m at end-Q320 and net off payments due to Rigel (NOK9.8/share). We do not include tilvestamab in our valuation. Key R&D inflection points in 2020/21 will determine registration pathways.

17 November 2020

Price	**NOK29.1**
Market cap	**NOK2539m**
	NOK9.57/US$
Net cash (NOKm) at 30 September 2020	777.9
Shares in issue	87.3m
Free float	61%
Code	BGBIO
Primary exchange	Oslo
Secondary exchange	N/A

Share price performance

%	1m	3m	12m
Abs	(1.0)	(18.5)	79.1
Rel (local)	(6.4)	(21.5)	90.2
52-week high/low	NOK46.1		NOK11.69

Business description

BerGenBio is a clinical stage biopharmaceutical company developing innovative drugs for aggressive diseases, including immune evasive, drug resistant and metastatic cancers. It focuses on AXL inhibitors bemcentinib (small molecule) and tilvestamab (mAb).

Next events

Phase II AML and MDS bem + LDAC combo data at ASH	5–8 Dec 2020
NSCLC data bem plus Keytruda at WCLC	28–29 Jan 2021
ACCORD-2 data in COVID-19	Q420/Q121

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Edison profile page

BerGenBio is a research client of Edison Investment Research Limited

Investment summary

Company description: Leader of the pack

BerGenBio (BGBIO) is a clinical stage biopharmaceutical company headquartered in Bergen, Norway, with clinical development capabilities in Oxford UK (BGBIO employs 45 personnel across two locations). CSO and scientific founder Prof James Loren is a pioneer in the field of AXL biology, and CEO Richard Godfrey has been critical in leading the company's development since his appointment in 2008. BGBIO focuses on the development of inhibitors against AXL receptor tyrosine kinase, a protein that has been implicated in a myriad of tumour cellular processes and plays a critical role in mediating treatment resistance, tumour immune evasion and development of metastatic disease. AXL is notable for its overexpression in a range of cancers and is implicated in the pathophysiology of fibrotic and viral conditions. BGBIO has two assets in development: bemcentinib (an oral small molecule AXL inhibitor, in-licensed from Rigel) and wholly owned anti-AXL antibody tilvestamab. Bemcentinib is being evaluated across a multitude of cancer types, both in BGBIO sponsored trials (in second-line (2L) acute myeloid leukaemia (AML) and 2L non-small-cell lung cancer (NSCLC)) and investigator-sponsored trials (in mesothelioma, melanoma, glioblastoma and pancreatic cancer). Parallel development of biomarkers or a companion diagnostic test too could aid monetisation. BGBIO listed on the Oslo Stock Exchange in 2007.

Valuation: NOK5.16bn or NOK59.1 per share

We value BGBIO NOK5.16bn or NOK59.1 per share , based on a risk-adjusted NPV analysis, which includes cash and cash equivalents of NOK777.9m at 30 September 2020 (end-Q320) (BGBIO is debt free) and risk-adjusted contributions for bemcentinib in 2L NSCLC (NOK40.7/share) and 2L AML (NOK13.3/share) oncology indications plus NOK5.9/share for the COVID-19 opportunity. We use a 12.5% discount rate for assets in development. We assume a licensing deal for bemcentinib (in all oncology indications and use in the first-line (1L) setting) after proof-of-concept data in AML in 2023. We do not assign any value to tilvestamab due to its early stage of development and we will review the potential of this asset as the clinical strategy becomes evident.

Sensitivities: Near term rests on bemcentinib

BGBIO is subject to the usual risks associated with drug development including clinical development delays or failures, regulatory risks, competitor successes, partnering setbacks and financing risks. Value crystallisation is dependent on successful R&D progress and potential partnering activities. BGBIO is focused on AXL inhibition; there is a risk that AXL as a drug target could be futile, which would have major implications for its pipeline. The biggest near-term development sensitivity relates to lead asset bemcentinib (see Exhibit 17 for licensing deal terms).

Financials: Near-term cash burn depends on multiple factors

In 2021, BGBIO should initiate the potentially pivotal trials in AML and NSCLC. The rapidly developing COVID-19 programme is the main determinant in terms of BGBIO's cash flows in the near term (2021–22). If the readouts from the ongoing COVID-19 studies warrant bemcentinib's progress into a Phase III trial, this would further increase the R&D spending. $8m is payable to Rigel (BGBIO's in-licensing partner) on the Phase III trial initiation. Another $28m could become payable to Rigel in 2021/22 if bemcentinib is approved for COVID-19 (so $36m in total). Only the milestone on Phase III entry ($8m) is subject to Phase III risk. If this trial is successful and the drug is launched for the treatment of COVID-19, we believe the remainder of the R&D-related payments could be covered from multiple sources. We forecast the operating loss to increase to NOK260.7m in 2020 and to NOK305.2m in 2021, which is related to more intensive R&D. At 30 September 2020 (end-Q320), BGBIO had a comfortable cash position of NOK777.9m.

Outlook: Potential for paradigm shift in oncology

BGBIO is a global leader in the field of AXL biology, and is developing selective AXL inhibitors: bemcentinib, a first in class oral AXL TKI, originally in-licensed from Rigel Pharmaceuticals (BGBIO holds worldwide rights), and wholly owned functional monoclonal antibody tilvestamab. The biology is complex, and industry understanding of the far-reaching role AXL plays in tumour pathophysiology is evolving; AXL mediates aggressive disease and its overexpression is a negative prognostic marker in cancer. AXL is overexpressed as a response to a hostile tumour micro-environment and this drives a tumour survival programme including the epithelial–mesenchymal transition (EMT), a process in which epithelial cells acquire mesenchymal features and in cancer EMT is associated with tumour initiation, invasion, metastasis and drug resistance. AXL is indiscriminate on tumour type, upregulation occurs to various degrees and thus AXL inhibitors could have utility across many solid tumours and haematological malignancies. The commercial prospects for the AXL inhibitor class could be propelled from defined subsects in a handful of cancer types to broader use in AXL positive tumours (diagnosed by companion drug testing or biomarkers) and potentially in an earlier line of therapy in combination with chemotherapy, immunotherapy or targeted therapies (due to its treatment enhancing effects). BGBIO's strategy in oncology is to establish efficacy in proof-of-concept studies to identify opportunities for rapid regulatory approval (bemcentinib plus LDAC in relapsed AML) and concurrently develop line extensions (2L NSCLC in combination with CPI Keytruda (pembrolizumab), and other indications) and move higher up the treatment paradigm.

Bemcentinib peak sales could evolve significantly

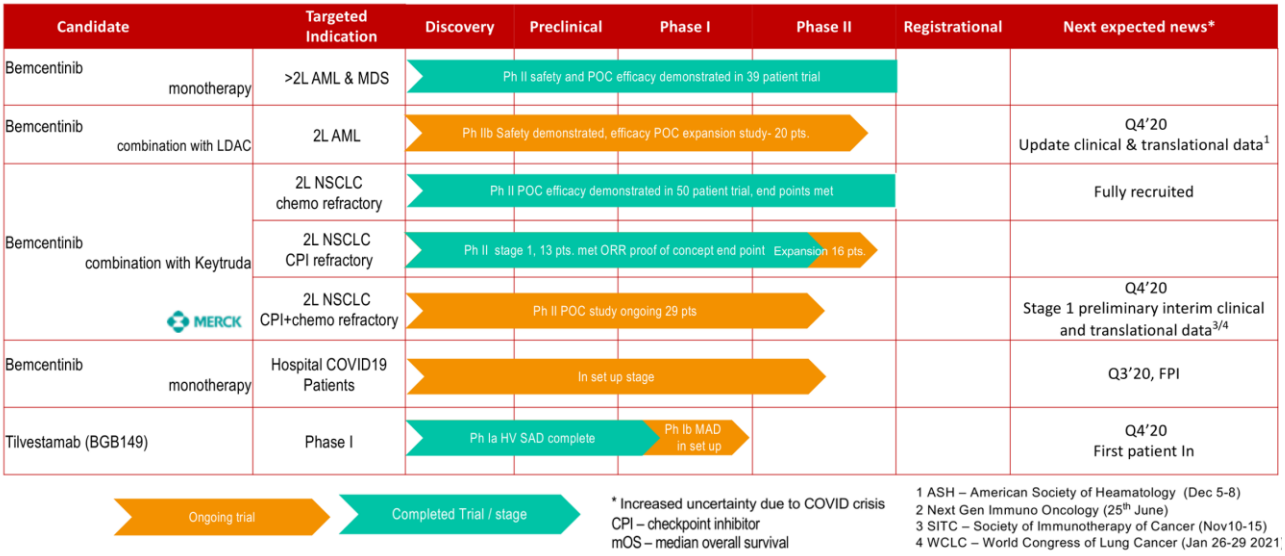
Our peak bemcentinib sales estimates of $1.2bn in 2L NSCLC and $588m in 2L AML could prove conservative as bemcentinib moves into earlier lines of treatment and/or proves efficacious in other cancer indications. Data in 2L AML (ASH December 2020) and 2L NSCLC (WCLC January 2021) will define the pivotal registrational clinical trials. We anticipate a Phase III study of bemcentinib plus LDAC (palliative care) in relapsed elderly AML patients to start in 2021, and if successful, an NDA submission in 2023 (FDA fast track has been granted in resistant AML in elderly patients), an unmet need. AML is the likely first indication to market (in 2024), followed by approval in 2L NSCLC in 2025. We expect the 2L NSCLC Phase III study to start later in 2021 in combination with Keytruda for PD-L1 refractory patients (an accelerated approval pathway is a possibility given the unmet need). We would expect filing for a companion diagnostic (to identify patients with high AXL expression and therefore most likely to respond to treatment) alongside and anticipate NDA submission in 2024. NSCLC, in our view, represents a significant opportunity; bemcentinib in combination with Keytruda could enable a treatment paradigm shift by addressing PD-1 resistance. Additionally, we highlight bemcentinib's utility in NSCLC could be further defined in earlier lines of treatment and in combination with other cancer drug classes, notably targeted therapies. Exploratory trials are warranted in our view, and the sooner the better given the relatively short timeframe to patent expiry and market exclusivity.

BGBIO is a forefront innovator in selective AXL inhibitors

BGBIO's multiple shots on goal development strategy includes development of small molecules and a functional humanised mAb tilvestamab (plus undisclosed preclinical inhibitors) targeting a range of conditions, Exhibit 1. Approved multi-kinase inhibitors that include AXL as a target (eg Pfizer's Xalkori and Exelixis's Cabometyx/Cometriq), but notably not prime target, have to an extent validated the role of AXL inhibition in tumour migration. However, in order to gain benefit in the magnitude required, research efforts have focused on selective AXL inhibitors, which could enable combinatorial approaches as well as monotherapy. We see combination therapy as the largest commercial opportunity in the near term and monotherapy as a potential bonus in the future. During

2019, BGBIO presented initial clinical proof-of-concept data that bemcentinib can increase the efficacy of immunotherapy, targeted and chemotherapy, particularly in patients whose tumours overexpress AXL. While oncology is the current focus, expansion into fibrotic conditions (eg idiopathic pulmonary fibrosis or non-alcoholic steatohepatitis (NASH)) is likely (the monoclonal antibody approach may be optimal) and COVID-19 has advanced its assessment in virology. Near-term inflection points include COVID-19 Phase II data by year end. In oncology indications we anticipate additional data from BGBIO sponsored trials to define the registration enabling studies in NSCLC and AML.

Exhibit 1: Company sponsored trials



Source: BGBIO corporate presentation

Oncology a growing market accelerated by new drug classes

The armament of cancer treatments has widened significantly in recent years to include targeted therapies, checkpoint inhibitors and the first cell therapies (CAR-T); these therapies can achieve unprecedented responses by identifying patient populations with specific genes and or proteins that are hallmarks of a particular cancer and selectively targeting them. Combinations of targeted therapies (eg TKIs, monoclonal antibodies and immunotherapies) and chemotherapy are increasingly becoming the best approach to treating the complex and constantly mutating disease that is cancer. According to IQVIA, sales of total oncology therapeutics (including supportive care drugs) reached ~$150bn in 2018. In the US spending on cancer drugs exceeded $56bn in 2018 (with $9bn in growth accounted for by growth in PD-1/PD-L1 inhibitors), while RoW oncology costs exceeded $66bn value. Over the next five years total oncology therapeutics are forecast to grow at a CAGR of 11–14% to exceed $200–230bn in sales.

PD-1 inhibitor class evolution a case study in new treatment paradigms

Immunotherapies particularly those that target PD-1 (eg Keytruda, Opdivo) or PD-L1 (eg Tecentriq, Imfinzi), so called checkpoint inhibitors, have changed the treatment paradigm for advanced cancers across many tumour types. This class of drugs work to enhance the function of T lymphocytes and have yielded exceptional results in the treatment of cancer. Merck launched Keytruda for advanced melanoma, the first FDA approved PD-1 inhibitor in 2014. It has quickly become the primary 1L treatment in the US for metastatic NSCLC (with no driver mutation) and metastatic melanoma (and is approved for gastric cancer and hepatocellular cancer), supported by Merck's foresight to invest in a broad range of clinical trials (KEYNOTE series). Merck reported worldwide sales in 2019 of $11bn and in 2026, worldwide sales are forecast to reach $26bn

(according to EvaluatePharma). Initially the FDA approved Keytruda monotherapy for 1L metastatic NSCLC patients whose tumours express PD-L1 > 50% (using the tumour proportion score (TPS)) and for whom it is the current standard of care. For patients with PD-L1<50%, Keytruda plus platinum doublet chemotherapy is now becoming the standard of care (following a recent ASCO guideline update). This means that all NSCLC patients without an actionable driver mutation receive Keytruda (either monotherapy or in combination) in the first line, irrespective of their PD-L1 status. Patients with PD-L1 TPS <1% show the lowest response rates. PD-L1 status measures the amount of the protein PD-L1 on cancer cells; some cancer cells have high PD-L1, which cloaks the cell from the immune system.

AXL inhibitors a potential for paradigm shift?

AXL receptor tyrosine kinase has recently emerged as an attractive target due to its role in an ever-expanding list of cellular processes associated with tumorigenesis (including survival programmes and EMT). AXL is notable for its overexpression in a range of solid tumours and haematological malignancies (caused by conditions of stress due to hypoxia, inflammation due to immune reaction and therapeutic treatment such as chemo-toxicity). Given this range and the heterogeneities within cancer, an important factor will be identification of patients who may benefit from AXL inhibitors regardless of tumour origin. If successful, AXL inhibitors as a class could lead to a paradigm shift in cancer treatment much like checkpoint inhibitors (eg Keytruda) and targeted therapies (eg Tagrisso) have demonstrated in recent years.

Biomarker strategy/companion diagnostic critical to approval pathway

BGBIO's strategy includes the development of companion diagnostic testing (IHC) and biomarker scores to identify and diagnose patients who show durable benefit. Biomarker tests under evaluation include soluble AXL (sAXL) in blood cancers and a composite AXL tumour-immune composite AXL (cAXL) score in solid tumours scores. Our model assumes that BGBIO will successfully validate a biomarker/companion diagnostic, which will be used to evaluate patients' AXL status. This strategy is beneficial as it can be used to enrich clinical trials with patients more likely to respond and, in general, is favoured by regulators and payors. Since the target patient population is smaller in this scenario, the economics of such a strategy can be balanced with more attractive pricing. These biomarkers and companion diagnostics are still in development and it is currently unclear whether they will be used in the potential registrational studies. Instead BGBIO may need to open the trial to all comers and then retrospectively analyse AXL status. Interim Phase II data generated thus far supports bemcentinib's utility as a monotherapy in ≥2L AML/myelodysplastic syndromes (MDS) (43% response rate in AXL biomarker positive patients) and in combination with LDAC in 2L relapsed AML/MDS (disease control rate (DCR) of 50%) and with Keytruda in NSCLC (33% overall response rate and 8.4 months median progression-free survival (mPFS) in cAXL positive patients).

COVID-19 Phase II, unique MOA and convenience oral dosing

Both Pfizer/BioNTech's and Moderna's ongoing COVID-19 vaccine programmes recently announced positive headline data; while we believe this may lead to widespread vaccination, this will take time globally and furthermore data on safety, durability and the impact of mutagenicity will increasingly become evident. In the meantime, for the next one to two years, there remains from a global public health perspective a need for effective treatments, at least until wide-scale vaccination has curbed the pandemic (assuming mutagenicity does not lead to further issues). COVID-19 represents an additional opportunity that could expediate bemcentinib's route to market in 2022. Ongoing Phase II trials will determine efficacy (suppression of viral entry and activation of the patient's immune system) in hospitalised patients. Bemcentinib could prevent viral intracellular entry and an augmentation of the type 1 interferon response (a key anti-viral defence mechanism) in the

treatment setting plus standard of care (including remdesivir and dexamethasone) in acute lung injury patients. We forecast peak COVID-19 sales of $300m.

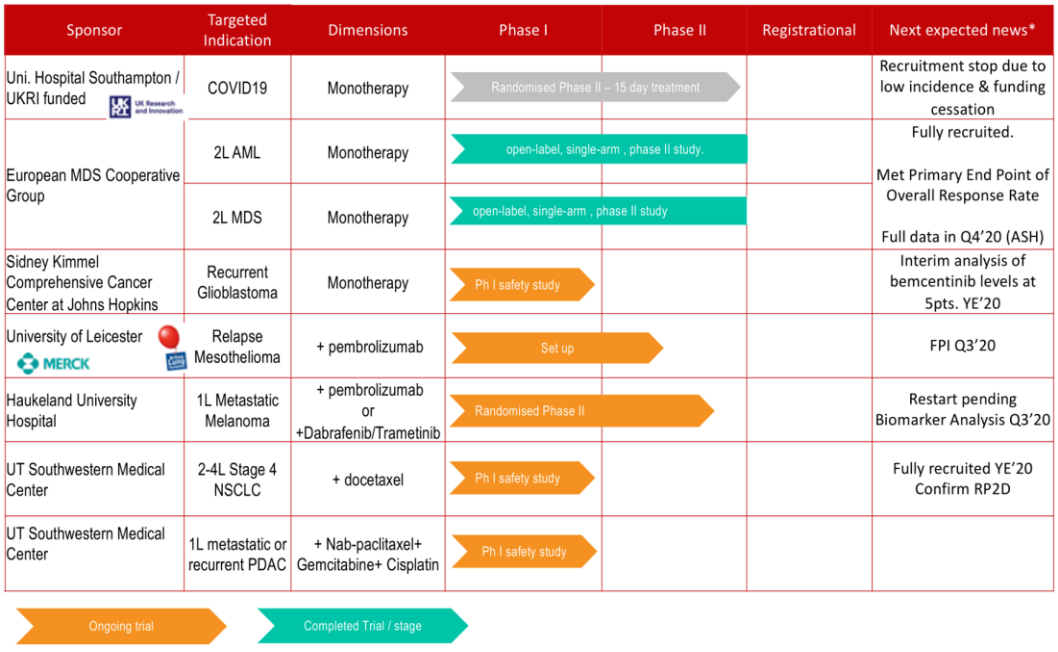
Global partnering deal economics will depend on data

Given the potential breadth of use for the AXL inhibitor class, we believe a global partnering deal to be the most value maximising proposition. As the data unfolds and bemcentinib progresses in Phase III, positive registrational intent data would validate the class and could command significant deal economics. A global partner would additionally have the resources to invest in a fuller and wider programme in oncology. BGBIO could focus efforts in fibrosis and virology. We also believe that a proof-of-concept in one indication could have a read-across effect to other indications with known overexpression of AXL, for a potential partner (one with an established PD-1, for example, would make sense). In our assumptions on a deal we reflect biotech licensing deals that included assets with potential in multiple indications as the benchmark. Based on deals that have occurred since 2015, we assume an upfront payment of c $250m and c $1.4bn in total milestones (one-third allocated to R&D related payments; the rest are commercial milestones). We assume tiered royalty rates of 15–18% on sales.

Bemcentinib: Multiple opportunities in oncology

Bemcentinib is an oral, first-in-class, highly selective AXL tyrosine kinase inhibitor in Phase II development in oncology and virology; Exhibit 1 highlights BGBIO led trials. A number of investigator-initiated trials (IIT) in a range of solid and haematological cancers are exploring additional indications (mesothelioma, melanoma, glioblastoma and pancreatic cancer) as well as label expansions for AML/MDS and NSCLC, Exhibit 2.

Exhibit 2: Bemcentinib investigator-initiated trials



Source: BGBIO corporate presentation

Bemcentinib has been granted US FDA orphan drug designation for AML as well as fast track designation in relapsed AML for elderly patients, which will likely be the first route to market. We expect US NDA and EMA MAA filings in 2023 with launch for this indication in 2024. While 2L will be the initial filing, ongoing clinical trials in AML/MDS will further define whether the monotherapy opportunities in later lines (≥2L) of therapy are worth investigating further. NSCLC represents a

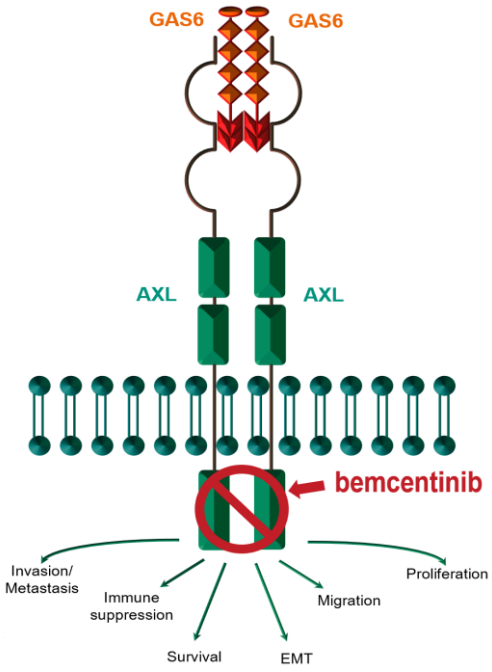
significant opportunity. We expect initial filing in combination with Keytruda for 2L NSCLC in checkpoint inhibitor refractory patients. However, given bemcentinib works synergistically to augment the effect of immunotherapies, targeted therapies and chemotherapies, wider adoption will depend on addressing the full clinical potential for bemcentinib, in particular defining the following:

- How early in tumorigenesis an AXL inhibitor shows benefit, and whether AXL inhibition can benefit in a 1L setting.
- Whether AXL inhibition is beneficial in all tumours expressing AXL irrespective of tumour origin, and whether a tissue agnostic approach is the most appropriate approach.
- The extent of synergies with combination therapy approaches, and whether AXL inhibitors should be added to CPIs regardless of molecular status (all comers) in resistant cancers such as NSCLC.

Convenient oral dosing profile supports combination therapy

Bemcentinib is well tolerated due to its high selectivity and has a clean safety profile (well tolerated up to maximum tolerated dose (MTD) of 1.5g per day. The recommended Phase II dose (RP2D) is a loading dose of 400mg per day for three days and then a 200mg per day maintenance dose) and is conveniently administered as a once daily oral pill. Oral formulations reduce the patient burden of receiving treatment as an in-patient. As a small molecule TKI bemcentinib has low cost of goods, rapidly scalable manufacturing and three-year shelf life, making it suitable for global distribution. Bemcentinib's selectivity for AXL coupled with its safety profile makes it a prime candidate for the treatment of a range of cancers as both a monotherapy and in combination (particularly with but not limited to immunotherapies) with current standard of care treatment. Bemcentinib blocks the intracellular domain of the transmembrane AXL protein receptor. Unlike with conventional cancer treatments, inhibition of AXL signalling does not induce apoptosis of tumour cells, rather inhibition enhances the body's immune response and through reversal of the EMT (see later) makes cancer cells more susceptible to attack by the body's immune system or a combination therapy, Exhibit 3. Inhibition of AXL has the potential to be a powerful therapeutic option. However, its success may be predicated on being able to identify tumours with sufficient AXL overexpression. We detail the complex biology later in Appendix 1.

Exhibit 3: AXL receptor biology



Source: BGBIO corporate presentation

Companion diagnostics key to patient identification

The use of a predictive biomarker and companion diagnostic strategy can significantly shorten the path to registration and greatly improve the chances of a drug reaching the market. Precision medicine approaches are becoming more common place in oncology drug development; biomarkers that stratify the patients most likely to respond to treatment are now included in ~39% of oncology clinical trials. Additionally, precision medicines can lead to wider reimbursement. AXL overexpression has been identified as a negative prognosis factor in a multitude of cancers, and BGBIO is currently developing a range of different biomarkers and diagnostics to identify patients with high AXL expression from tissue and blood samples. Due to the complexity and evolving understanding of this novel therapeutic class, several different biomarker approaches are being explored, including soluble AXL (sAXL) from blood samples for use in haematological cancers (eg AML) and a composite AXL (cAXL) score based on tissue biopsies for solid tumours (eg NSCLC). These biomarkers and companion diagnostics are still in development and it is currently unclear whether they will be used in the potential registrational studies. Instead, BGBIO may need to open the trial to all comers and then retrospectively analyse AXL status.

Bemcentinib, is a highly selective inhibitor of AXL (250-fold over nearest TAM (TYRO3-AXL-MER) kinase family member MER and >1,000-fold over TYRO3) and the beneficial disease response it has elicited in a range of Phase II studies so far is believed to be solely due to the inhibition of AXL (and subsequent downstream processes). This implies that patients with high AXL expression who will likely show the highest response rates and benefit most from treatment can be selected. However, the exact amount of AXL overexpression required for bemcentinib to elicit a statistically significant beneficial disease response, thus outlining the addressable patient population, is still being determined. Current cut offs used in the ongoing Phase II studies in AML/MDS and NSCLC suggest this could include >50% of patients.

BGBIO is developing a cAXL score that uses a solid biopsy to stratify patients in the current Phase II NSCLC study and a more convenient sAXL diagnostic that uses a liquid biopsy and is being validated in the Phase II AML/MDS study. Data generated thus far by retrospective analysis of patient populations has confirmed their ability to predict patient responses and supports their use in stratifying patients. BGBIO has also more recently reported the discovery of BGBM033, a serum biomarker candidate that appears to be predictive of responders to bemcentinib in both NSCLC and AML/MDS patients. Determination of the cAXL score requires a cumbersome and invasive solid tumour biopsy and the successful development of serum biomarker BGBM033 could enable stratification of patients with a simple and convenient liquid biopsy, although we note it is in a much earlier stage of development.

Bemcentinib 2L AML/MDS first route to market

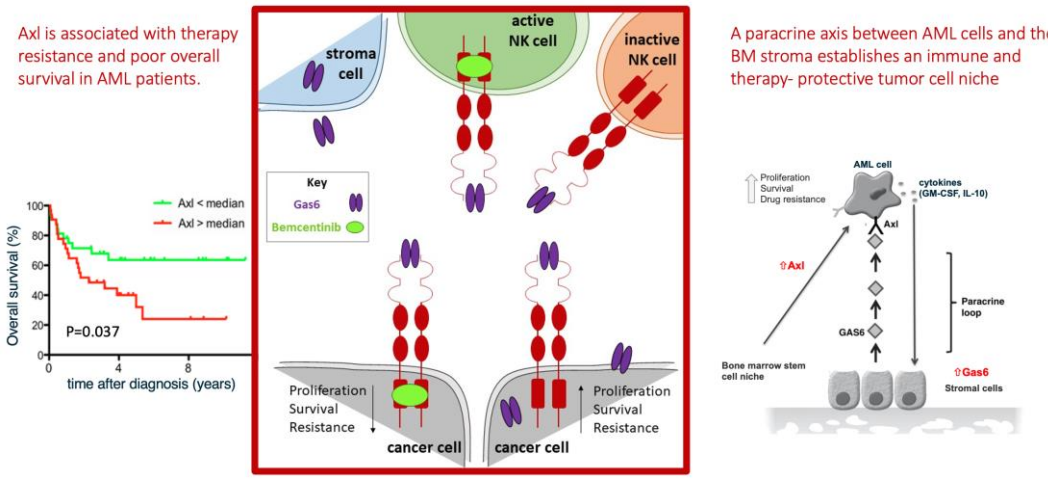
Bemcentinib's first NDA filing (expected in 2023) is likely to be in 2L AML in combination with LDAC therapy, a standard of care chemotherapy regimen. BGBIO is exploring multiple development options in AML for the asset with two Phase II studies underway in AML/MDS (including multicohort study BGBC003 and the investigator-sponsored BERGAMO trial); data readouts expected this year at ASH 2020 will define the registrational strategy. The **bemcentinib plus LDAC combination in relapsed AML** patients unsuitable for intensive chemotherapy will likely form the registration enabling Phase IIb/III study (we would expect it to enrol approximately 200 patients and test the combination vs LDAC alone with six-months follow up). This represents a currently unmet medical need and is a sizeable market. Additionally, the combination of bemcentinib plus LDAC could find utility as a 1L treatment for elderly (> 60 years old) patients unsuitable for hypomethylating agents (HMAs) plus venetoclax. Furthermore, due to the limited treatment options, bemcentinib could find

utility as a monotherapy treatment in the 2L and above for heavily pre-treated elderly patients. The multi-arm BGBC003 trial is evaluating these potential treatment scenarios.

Bemcentinib inhibits AML cell survival and enhances immunity

AXL overexpression has been widely established as a negative prognostic factor in AML and early clinical data of AXL inhibition with bemcentinib has shown promising anti-leukemic activity and immune activation. In addition to AXL's putative role (described later) in cancer growth and spread, AXL inhibition has been shown to block activation of FLT3 (caused by internal tandem duplication, the most prevalent type of FLT3 driver mutation present in c 25% of AML patients); FLT3 inhibitors are a recent class of drug approved for AML. Furthermore, leukemic cells induce expression of Gas6 (the ligand that activates AXL) in bone marrow stroma cells, which further amplifies their growth and therapy resistance, Exhibit 4.

Exhibit 4: Bemcentinib inhibits AML/MDS cell survival and enhances anti-leukemic immunity



Source: BGBIO corporate presentation

Relapsed AML an unmet need

AML is one of the most prevalent types of leukaemia in adults and affects the myeloid cells of the bone marrow. The American Cancer Society estimates 19,940 new cases of AML and ~11,180 deaths in 2020 (mostly in adults). AML is an aggressive disease with a five-year survival rate of 28.7% (this is lower for frail elderly patients specifically). It consists of a heterogenous group of haematological cancers that originate in the bone marrow and are caused by clonal expansion of malignant hematopoietic precursor cells. The proliferation of cancer cells in the bone marrow interferes with the normal production of mature blood cells leading to anaemia, thrombocytopenia and neutropenia. MDS can be considered a premalignant disease that affects myeloid cells in bone marrow and encompasses a range of haematological conditions that are characterised by chronic cytopenia due to abnormal haematopoiesis and cell maturation. Approximately 30% of MDS patients' disease progresses to AML. Studies suggest that 12,000 to 15,000 MDS cases are diagnosed annually in the US, and an estimated 50,000 to 75,000 people currently live with MDS.

AML is classified by subtype, which is relevant for treatment and prognosis (eg chromosomal aberrations, gene mutation including FLT3 gene, also mutations in the TP53, RUNX1 and ASXL1 genes are linked with a worse outlook). The primary aim of 1L treatment of AML is the induction of complete remission. Treatment options include aggressive dosing regimens (7+3) of chemotherapy (eg cytarabine plus an anthracycline drug), however disease relapse is observed. These patients will then be treated with HMA/LDAC (median overall survival (mOS) ~ six months) or targeted therapy. The approval of targeted therapies has added new treatment options for patients who test

positive for the specific gene mutation or overexpression, with varying response rates and median overall survival of six to nine months in relapsed/refractory disease, Exhibit 5. However, despite these advances AML remains a significant unmet medical need, particularly in the relapsed setting where treatment options are limited.

Exhibit 5: Examples of targeted AML drug treatment

Product	Manufacturer	MOA	Comment
Venclexta (venetoclax)	AbbVie/Roche	BCL-2 inhibitor	Recently approved in combination with decitabine, azacitidine or low dose cytarabine in newly diagnosed AML patients over the age of 75 or who cannot tolerate intensive induction chemotherapy. New SOC for these frail patients. BCL-2 is overexpressed in almost 85% of AML patients.
Rydapt (midostaurin)	Novartis	FLT3 inhibitor	First-line use in combination with induction chemotherapy (cytarabine and daunorubicin) for FLT3 mutation-positive patients. FLT3 mutations are observed in c 30% of AML patients. Not indicated as a monotherapy.
Mylotarg (gemtuzumab ozogamicin)	Pfizer	Anti-CD33 antibody drug conjugate	Approved for both first line and r/r AML in CD33-positive patients. 85-90% of AML patients express the CD33 antigen. IV dosed and black box warning for hepatotoxicity.
Daurismo (glasdegib)	Pfizer	Hedgehog pathway inhibitor	First-line treatment in combination with LDAC for patients over 75 years or with comorbidities that preclude the use of intensive induction chemotherapy. Black box warning for embryo-foetal toxicity.
Xospata (gilteritinib)	Astellas	FLT3 inhibitor	Second-line treatment for r/r AML patients harbouring FLT3 mutation (c 30% of AML patients). Black box warning for differentiation syndrome, which can be fatal if untreated.
Tibsovo (ivosidenib)	Agios Pharmaceuticals	IDH1 inhibitor	First-line treatment for patients unable to tolerate intensive induction chemotherapy and second-line treatment for any r/r AML patients. Patients must have IDH1 mutation, which is observed in less than 15% of patients. Black box warning for differentiation syndrome.

Source: Edison Investment Research

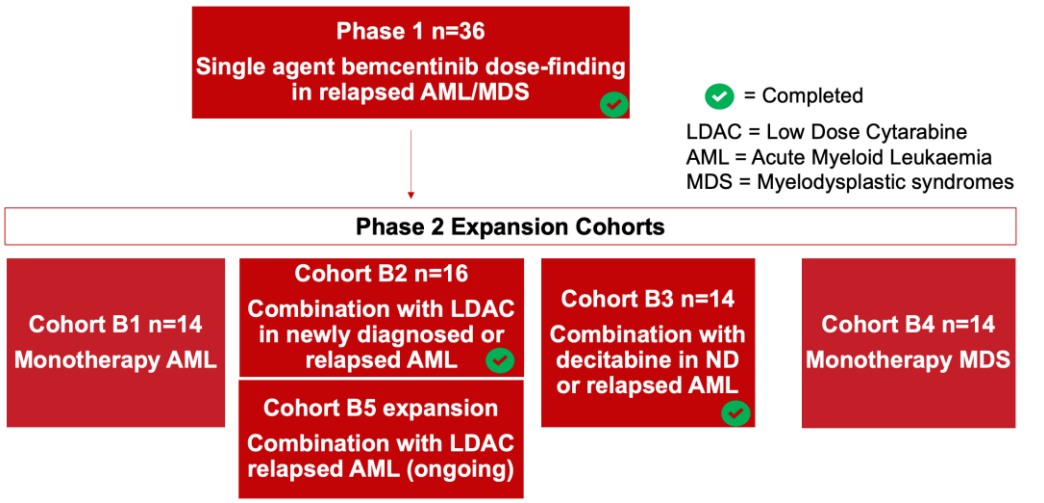
Bemcentinib's promising data in relapsed AML

Within the context of available treatment options for AML/MDS, BGBIO is evaluating bemcentinib across a multi cohort study, BGBC003, conducted in two parts: Phase 1 and Phase 2. This study targets patients with AML who are not suitable for intensive induction chemotherapy. The Phase I trial was expanded from dose finding single agent bemcentinib (Cohort A) into five Phase II expansion cohorts to evaluate bemcentinib monotherapy and in combination with chemotherapy agents (LDAC or decitabine) and biomarker correlations, Exhibit 6. The cohorts to focus on in the near term are Cohort B2: bemcentinib combination with LDAC in newly diagnosed (ND) or relapsed AML; and Cohort B5: further expansion of Cohort B2 LDAC combination in relapsed AML, as these will likely define Phase IIb/III clinical trial design and registration strategy, **interim data will be presented at ASH 2020** (5–8 December).

Cohorts B1 and B4 are currently recruiting AML patients unsuitable for intensive chemotherapy and MDS patients who are either intermediate/high risk or have previously received treatment respectively. These patients will receive bemcentinib monotherapy and **interim data is expected to be presented at a scientific conference in 2021**. This cohort will determine the monotherapy strategy. Interim data from Cohort B3 presented at ASCO 2019 suggest there is limited benefit of adding bemcentinib to decitabine.

The primary endpoints of BGBC003 are safety and tolerability, but key secondary endpoints measuring efficacy include overall response rate (ORR) (complete remission (CR) for AML), relapse free survival (RFS) and overall survival (OS).

Exhibit 6: Phase I/II multi cohort study (NCT02488408)



Source: BGBIO corporate presentation

Cohort A bemcentinib monotherapy dose finding in r/r AML/MDS

While Cohort A defined the RP2D for the expansion portion of BGBC003, early efficacy signals were demonstrated in a difficult to treat patient population (elderly patients with aggressive disease who had received multiple prior lines of therapy) with extremely poor prognosis. CR was achieved in 22% (six patients) of the efficacy evaluable patients (n=27) with bemcentinib monotherapy, with 30% (eight patients) maintaining stable disease (SD), Exhibit 7. An impressive 43% of patients with high AXL expression (sAXL low) achieved CR vs 0% of patients with low AXL expression (sAXL high). CR is the gold standard for 1L therapy in AML. However, this patient population had received multiple lines of prior treatment and thus the DCR of 52% may be a more appropriate measure of the promising anti-leukemic properties of bemcentinib.

Exhibit 7: Cohort A responses to bemcentinib and sAXL status

	Overall (n=27) % (n)	sAXL low (n=14) % (n)	sAXL high (n=11) % (n)
CR/CRi/CRp	22 (6)	43 (6)	0 (0)
SD	30 (8)	21 (3)	45 (5)
PD	48 (13)	36 (5)	55 (6)

Source: Edison Investment Research, BGBIO corporate presentation. Note: CR: complete remission; CRi: CR with incomplete haematological recovery; CRp: CR with incomplete platelet recovery; SD: stable disease, PD: progressive disease.

In terms of biomarkers to select patients that could benefit most from treatment, sAXL has been looked at most closely in the context of haematological malignancies. The patient population can be divided into sAXL low (which translates into high AXL expression in the malignant bone marrow) and sAXL high (which translates to low AXL expression in the malignant bone marrow), enabling identification of a patient's AXL status through a simple blood sample. Post hoc analysis of the Phase I study in relapsed/refractory (r/r) AML proved sAXL to be a predictive biomarker for response; this suggests that for patients with high AXL expression (low sAXL), inhibition of AXL signalling plays a vital role in achieving disease remission.

Cohort B2 and B5 aiming for 2L in AML

Promising interim data for the combination of bemcentinib with LDAC (Cohort B2) was presented at ASH 2019 in elderly AML patients with newly diagnosed or r/r AML. At the interim data cut off, 50% of newly diagnosed AML patients (n=6) had achieved a CR/CRi and the ORR (CR, CRi or PR) was 66% in this subpopulation (ORR is <3% for LDAC monotherapy). As with Cohort A, AXL status was found to be predictive of responses. The relapse-free survival (median duration of CR/CRi

response) for patients with newly diagnosed AML had not been reached at the data cut off, but was >9.9 months (mOS data was also not mature). Although in small patient numbers, this positions the bemcentinib plus LDAC combination favourably vs other 1L treatment combinations with LDAC, Exhibit 8. One possible registration pathway could be as a 1L treatment for elderly (> 60 years old) patients unsuitable for HMA plus venetoclax. A randomised Phase IIb/III study enrolling approximately 200 patients with 12 months follow up may be sufficient. However, in the near term BGBIO will focus on the combination with LDAC in 2L relapsed AML patients.

Exhibit 8: Comparison of responses to current LDAC combination therapies in newly diagnosed AML

Combination Therapy	Number of patients	CR/CRi	CR/CRi rate (%)	Median DOR (months)	Median OS (months)
LDAC + bemcentinib	6	3	50	>9.9	N/A*
LDAC + glasdegib	88	24	27	9.9	8.8
LDAC + venetoclax	82	44	54	8.1	10.1

Source: Edison Investment Research, BGBIO corporate presentation. Note: *data not mature.

The promising efficacy signal (DCR 50%) observed in relapsed (not refractory) AML patients (n=4) led BGBIO to expand this combination to an additional cohort B5, specifically focusing on patients with relapsed (not refractory) AML. **Interim data will be presented at ASH 2020**, and results disclosed in the abstract highlight an ORR of 50% (n=8) and impressive DCR of 75%. Patient AXL status was not reported and is eagerly awaited.

AML remains a significant unmet medical need, particularly in the relapsed setting where there are minimal treatment options, and bemcentinib has the potential to cause a shift in the treatment paradigm. BGBIO has indicated it plans to seek regulatory advice from the FDA and EMA to determine the optimal regulatory path for bemcentinib approval in relapsed AML. We expect BGBIO to initiate a registration Phase IIb/III trial in the next nine to 12 months.

BERGAMO monotherapy study hits primary endpoint

In August, the investigator-initiated Phase II BERGAMO study of bemcentinib monotherapy in AML and high-risk MDS patients (n=45) met its primary endpoint of ORR at week 17. This trial recruited patients who had failed (r/r) 1L treatment with HMAs (azacitidine or decitabine – mOS of approximately six months), which represents standard of care for patients unfit for intensive chemotherapy. **Top-line data and analysis will be presented at ASH 2020**. Results disclosed in the abstract highlight that bemcentinib showed meaningful clinical efficacy in the MDS population (n=21) with 19% of patients achieving CR/CRi (four patients) and a DCR of 33%. This population is of particular interest as these patients are less frail and have a higher likelihood of achieving an immune response and meaningful clinical benefit from treatment with bemcentinib. These results warrant further development. AXL status again proved to be predictive of responders.

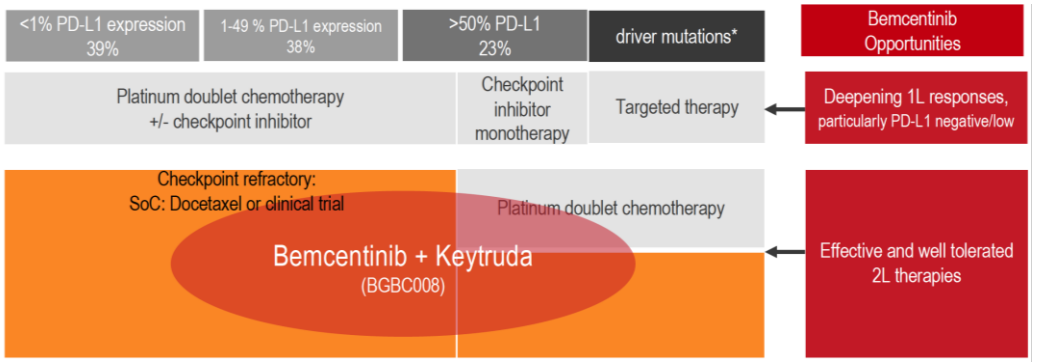
Bemcentinib in non-small cell lung cancer

NSCLC, in our view, represents a significant opportunity. Bemcentinib in combination with Keytruda (CPI) could enable a treatment paradigm shift by addressing PD-1 resistance, an unmet need. Bemcentinib is being evaluated in a broad Phase II study (BGBC008) with multiple cohorts including monotherapy and combination with standard of care Keytruda. The pivotal registration phase study is likely to focus on a bemcentinib plus Keytruda combination in PD-1 resistant NSCLC patients, an unmet need. Additionally, we highlight bemcentinib's utility in NSCLC could be further defined in earlier lines of treatment and in combination with targeted therapies.

NSCLC generally presents late and is frequently diagnosed at stage IV when metastatic. The American Cancer Society estimates 228,820 new lung cancer patients will be diagnosed in 2020, 84% of which will have NSCLC. There are ~1.76 million lung cancer deaths per year worldwide, and while smoking is a risk factor in a percentage, driver mutations (alteration in a gene that can

drive cancer growth) can lead to NSCLC even in non-smokers. Over the last decade the treatment paradigm for NSCLC has evolved significantly with the approval of targeted therapies and immunotherapies, Exhibit 9. It is now common practice to screen NCSLC patients for biomarkers to determine the most effective treatment approach: immunohistochemistry (IHC) is a standard method of testing for PD-L1, EGFR and ALK status in particular; and those with addressable driver mutations (approximately 25% of NSCLC patients) receive targeted therapies specific to the mutation as first line treatment.

Exhibit 9: Treatment options for non-small cell lung cancer



Source: BGBIO corporate presentation. Note: *Mutations/rearrangements with available targeted therapies such as EGFR and ALK.

CPIs are increasingly used as a 1L standard of care for patients not harbouring a specific driver mutation. Keytruda (Merck) is the market leading PD-1 checkpoint inhibitor. Patients with PD-L1 ≥50% are eligible for Keytruda monotherapy, while patients with PD-L1 <50% receive platinum doublet chemotherapy with or without Keytruda. The advancement of Keytruda to the 1L setting in metastatic NSCLC has left a vacuum in the 2L with limited treatment options (chemotherapy agents such as docetaxel or Taxol that achieve ORR<10% for limited durations). This has created a high unmet medical need and a large market potential for a well-tolerated therapy such as bemcentinib that could increase patient responses to Keytruda, Exhibit 9. Bemcentinib will be tested for 2L patients that have relapsed on Keytruda. As patients with PD-L1<1% are less responsive to Keytruda more of them relapse, so there is a higher portion of patients in the 2L with PD-L1<1%. Bemcentinib will target all patients in the 2L but those with a PD-L1<1% represent the highest unmet need.

AXL potentiates immunotherapy

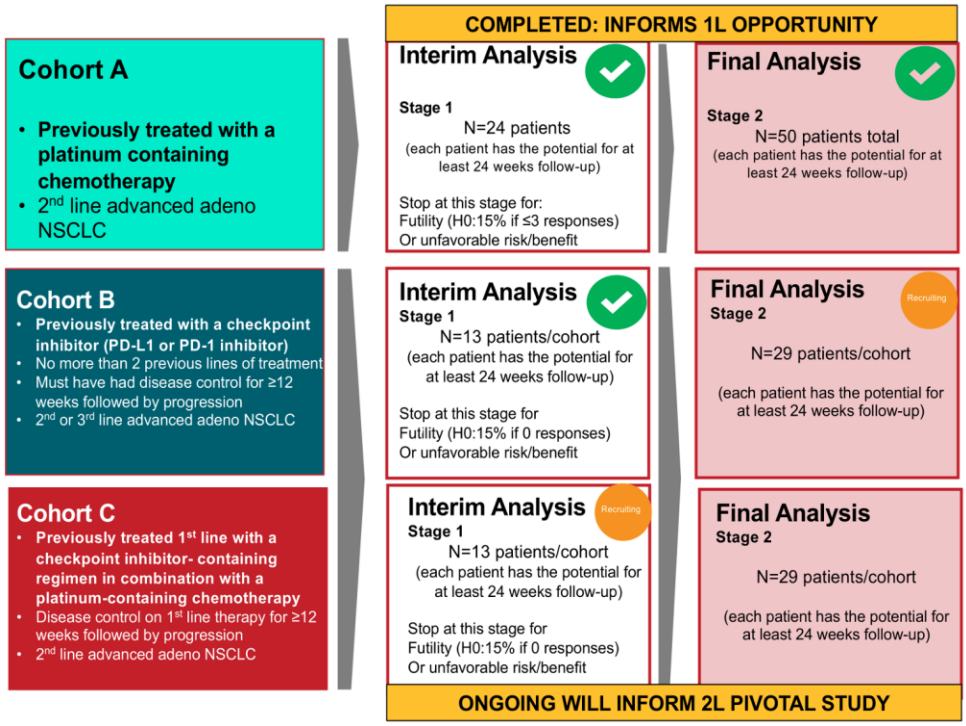
AXL is a recognised negative prognostic factor and resistance mechanism in NSCLC as it plays a key role in a plethora of cellular processes that are critical for the development, growth and spread of cancer including the EMT that allows cancer cells to evade the body's immune system. This is further compounded by the immunosuppressive effects of AXL overexpression on immune cells where it plays a critical role in inhibiting cytokine release and activation of tumour killing T cells. Bemcentinib's unique mechanism of action (selective AXL inhibition) reverses EMT and repolarizes tumour-associated macrophages. Bemcentinib's safety profile (well tolerated) makes it an ideal candidate for combination with an immunotherapy like Keytruda. The addition of bemcentinib to immunotherapy increases the tumour's immunogenicity (its ability to be recognised and targeted by the immune system) while reducing its immunosuppressive effects, thus enhancing the efficacy of the immunotherapy; this is supported by initial data from the ongoing Phase II study in combination with Keytruda. A key part of this trial is the use of biomarker analysis to identify patients who are most likely to benefit from treatment based on their AXL status; for NSCLC the cAXL score looks the most promising at present.

Phase II (BGBC008) multi cohort study including CPI naive patients

The multicohort Phase II (BGBC008) study is assessing bemcentinib utility in combination with current standard of care Keytruda, Exhibit 10; this enables BGBIO to efficiently explore bemcentinib's potential in a range of different treatment lines to determine the most beneficial target patient population. The trial's objectives are to determine the safety, objective response rate and overall survival benefit of the combination. Biomarker correlations will also be investigated. BGBC008 consists of three cohorts:

- Cohort A (n=50) has completed enrolment of checkpoint naive patients who had received a maximum of one prior line of platinum-containing chemotherapy. Positive top-line data for this cohort was reported at SITC 2019 and confirmed bemcentinib enhanced patient responses in combination with Keytruda. **This checkpoint naive cohort is of particular interest as the results highlight bemcentinib's 1L use potential**.
- Cohort B (n=29) is enrolling patients who have relapsed after achieving disease control for ≥12 weeks with a checkpoint inhibitor and have received a maximum of two prior lines of therapy. Positive interim data (n=13) was presented at the Next Gen Immuno-Oncology Congress in June 2020 and expansion and recruitment of patients into the second stage is ongoing.
- Cohort C (n=29) is enrolling patients who have relapsed after achieving disease control for ≥12 weeks with a checkpoint inhibitor (with or without chemotherapy) in the first line. **Cohort C will be critical to defining bemcentinib's utility in the second line in combination with Keytruda**.

Exhibit 10: NSCLC Phase II study design (BGBC008)



Source: BGBIO corporate presentation

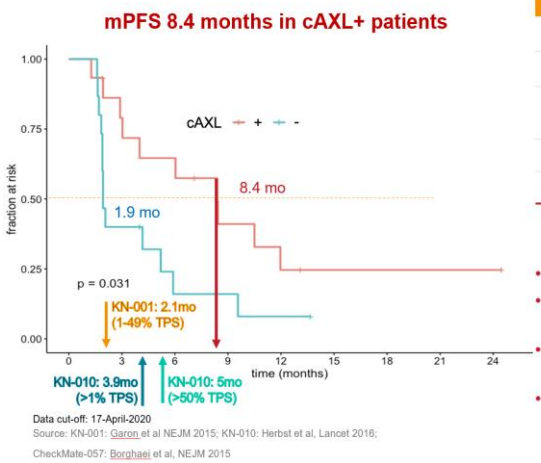
Cohort A evaluating 1L opportunity

Positive top-line data from Cohort A (checkpoint naive patients) was reported at SITC 2019. AXL status was measured using BGBIO's inhouse developed IHC diagnostic and patients were then classified based on their cAXL score as cAXL positive (50%) and expected to be most responsive

to treatment, or cAXL negative (50%). Cohort A met its primary endpoint of ORR with 33% of cAXL positive patients achieving a response, compared to only 7% of cAXL negative patients, underling a fivefold increase in ORR. This translated to a 73% DCR in cAXL positive patients (vs 40% in cAXL negative patients) with a mPFS of 8.4 months, a fourfold increase compared to cAXL negative patients (1.9 months) and significantly longer than that achieved with Keytruda monotherapy in the KEYNOTE-001 (Phase I) and KEYNOTE-010 (Phase III) trials, Exhibit 11. The safety profile of the combination was consistent with that observed for each individual drug. We note that the results are in comparably smaller patient numbers and that comparisons between trials can be challenging due to differing patient demographics (PD-L1 status and number of prior lines of therapy in particular).

A further update was given at Next Gen Immuno-Oncology Congress in June 2020 and four patients were still receiving treatment at the data cut off (7 April 2020), highlighting the durability of the response to treatment. Although mOS data was still maturing, a clear clinical benefit can be seen for cAXL positive patients (mOS >17.3 months) compared to both cAXL negative patients (mOS >12.4 months) and historical controls for checkpoint inhibitor monotherapy, Exhibit 11.

Exhibit 11: Cohort A mPFS Kaplan-Meier curve and median OS data



Cohort	mOS	12-mo OS
Cohort A – cAXL +ve pts**	**17.3 mo***	**79%**
Cohort A – cAXL -ve pts**	12.4 mo*	60%
BGB Cohort A – all pts**	12.6 mo*	64%* (up to 67%)
CheckMate-057 (Opdivo)	12.2 mo	51%
KEYNOTE-010 (Keytruda)	10.4 mo	43.2%

*OS data still maturing, current calculation (cut-off survival: 28-May-2020)
**pts who have been on study treatment for at least 1 cycle (n=42)

- 4-fold improvement in PFS in cAXL +ve vs. cAXL -ve patients.
- 12 mo OS in cAXL positive patients 79% vs 60% in cAXL negative patients
- Clinical benefit reflected in mOS of cAXL +ve patients vs. cAXL -ve
- cAXL -ve patient survival data is comparable to historic controls

Source: BGBIO corporate presentation

Due to the broad uptake of Keytruda (CPI) in the first line, the patient population Cohort A is targeting (2L CPI naive) is rapidly shrinking. However, although patients in Cohort A are relapsed and refractory, they are checkpoint inhibitor naive (like 1L patients) and we believe this data supports further investigation of bemcentinib's potential as a 1L treatment in combination with Keytruda.

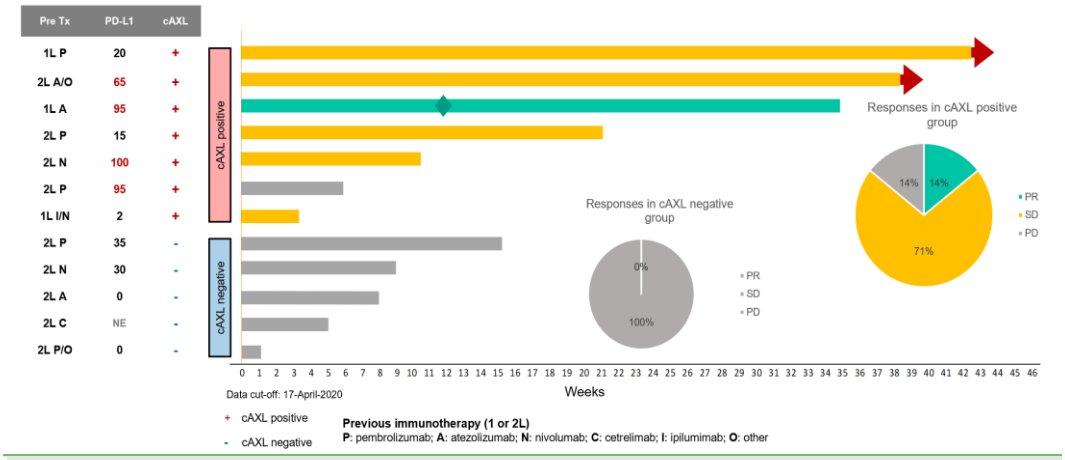
Cohort B progresses to stage 2 following interim analysis

Cohort B is assessing the 2L/3L treatment opportunity in patients who have relapsed after checkpoint inhibitor therapy. In June 2020, positive interim results from stage one were reported at the Next Gen Immuno-Oncology Congress and three patients were still receiving treatment at the data cut off. This checkpoint refractory cohort had a higher portion of cAXL positive patients than Cohort A, with 58% of patients cAXL positive and 42% cAXL negative (n=12 biomarker evaluable patients). However, only 25% of patients had a PD-L1 TPS of <1% (vs 55% in Cohort A) and 42% had a PD-L1 TPS score of 1–49%. The higher level of PD-L1 expression in this cohort is expected as patients with a higher PD-L1 TPS are more likely to respond to checkpoint inhibitors and are therefore more likely to have received them in a prior line of therapy.

Of the biomarker evaluable patients (n=12), six of the seven cAXL positive patients achieved disease control (DCR of 86%), Exhibit 12. This is even more impressive when compared to the cAXL negative patients who all exhibited disease progression. This resulted in a significantly higher

mPFS of 4.73 months for cAXL positive patients in comparison to 1.87 months for cAXL negative patients, underlining a real clinical benefit. Although in small patient numbers, these results are meaningful and suggest that bemcentinib has the potential to reverse acquired resistance to CPIs in previously treated AXL positive NSCLC patients and extend the efficacy of these immunotherapies (data presented at SITC 2020). These results further validate BGBIO's cAXL score and hypothesis that AXL status is predictive of treatment response.

Exhibit 12: Cohort B1 response and duration of treatment for cAXL evaluable patients



Source: BGBIO corporate presentation

Cohort C key to defining 2L opportunity

Cohort C (n=13) is enrolling patients into stage one who have relapsed after achieving disease control for ≥12 weeks with a checkpoint inhibitor in combination with platinum-containing chemotherapy in the first line. This cohort is particularly important for informing the opportunity of bemcentinib plus Keytruda in the second line due to the increasing uptake of Keytruda plus platinum doublet chemotherapy (cisplatin with Eli Lilly's pemetrexed (Alima)) in the first line for patients with a PD-L1 TPS <50% (and not harbouring a driver mutation), and thus ultimately a growing population of r/r patients. Early data is eagerly expected at WCLC 2021 (January 26–29).

Registrational NSCLC study expected to start during 2021

Data so far from the ongoing Phase II study (particularly Cohorts B and C) has been encouraging and we expect BGBIO to initiate a Phase IIb/III registrational study in the next nine to 12 months targeting patients who have relapsed after receiving a checkpoint inhibitor in combination with platinum-containing chemotherapy in the first line. BGBIO is currently in discussions with regulatory authorities regarding the trial design but we expect it will likely compare the combination of bemcentinib and Keytruda vs current standard of care docetaxel (chemotherapy). However, this is contingent on the continued positive signals from Cohorts B and C of the ongoing Phase II study. Data from these cohorts will also be used to further validate the cAXL score, which will form the basis of a companion diagnostic (CDx) for NSCLC patients. We expect the trial will be open to all comers and there will be a retrospective analysis of cAXL status.

Deepening of response in combination with Tarceva

For NSCLC patients harbouring driver mutations, targeted therapy is the 1L treatment. EGFR mutations are the most common among NSCLC patients (others commonly tested include ALK and ROS1) representing approximately 20% of patients. These patients are treated with EGFR inhibitors, although all patients eventually relapse due to bypass mechanisms such as the most common T790M mutation (up to 50% of cases) and MET amplification (c 20% of cases). Following disease progression, these patients receive platinum doublet chemotherapy unless diagnosed with

another targetable mutation. In preclinical in vivo studies, treatment with EGFR inhibitor Tarceva (erlotinib) induced AXL expression and activation, with resistance to Tarceva emerging over time. BGBIO found that the addition of bemcentinib to Tarceva prevented treatment resistance, suggesting AXL signalling is key to resistance to EGFR inhibitors.

Bemcentinib was investigated in a multi-arm Phase II (BGBC004) study in combination with EGFR inhibitor Tarceva in EGFRm+ NSCLC. The study enrolled 1L and 2L patients who were either currently receiving Tarceva without disease progression (Arm C) or had experienced disease progression having previously received an EGFR inhibitor (Arm B) into three treatment arms (Arm A tested safety and MTD). Interim data analysis presented at WCLC 2018 showed the combination led to a deepening of disease response in 1L patients (Arm C) receiving Tarceva whose disease had not progressed prior to receiving the combination. Additional tumour shrinkage was observed in six out of nine patients (67%) and while PFS data was not mature it exceeded 10 months and surpassed Tarceva monotherapy. This highlights the role of AXL (and subsequently EMT) in driving acquired treatment resistance and the utility of bemcentinib in preventing it. Despite the promising results, in the near term BGBIO will focus on the combination with Keytruda, which provides a faster route to market; resource permitting, in the longer term the bemcentinib plus EGFR combination warrants exploration.

Bemcentinib has multiple opportunities in COVID-19

Bemcentinib has presented a unique dual mechanism of action (MOA) in the potential treatment of COVID-19; drug treatments are currently limited to remdesivir and dexamethasone. With the recent announcements from Pfizer/BioNTech and Moderna on their vaccine candidates achieving >90% efficacy in Phase III trials, it is looking increasingly likely that these vaccines will be available globally late 2020/early 2021. Success of these vaccines bodes well for other vaccines targeting the coronavirus spike protein and while we believe this will lead to widespread vaccination, this will take time on a global level and furthermore, data on durability and the impact of mutagenicity will increasingly become evident. In the meantime, for the next one to two years, there remains from a global public health perspective a need for effective treatment options, at least until wide-scale vaccination has curbed the pandemic, assuming mutagenicity does not have an impact on vaccine efficacy. Noting bemcentinib's MOA, two Phase II clinical trials (ACCORD-2 in the UK and BGBC020 in South Africa and India) have been initiated to evaluate this asset's potential in hospitalised COVID-19 patients.

Recently both Pfizer/BioNTech and Moderna reported positive, preliminary top-line Phase III data from their respective vaccine candidates, BNT162b2 and mRNA-1273 respectively, at the first interim analysis. Based on the 94 confirmed cases of COVID-19 in the BNT162b2 study, initial data indicate vaccine effectiveness was >90% for the two-dose regimen (after 28 days), above the 50% threshold set by the FDA. Full peer reviewed data are yet to be published and we note the 90% effectiveness will need to be confirmed once the trial completes. The study will continue to enrol until final analysis once 164 cases have occurred. No significant safety concerns were noted and an Emergency Use Authorization (EUA) application is planned to be submitted to the FDA after requisite safety data are available (expected third week of November). For mRNA-1273, Moderna has reported point estimate efficacy of 94% ($p<0.0001$), this first interim analysis was based on 95 cases, of which 90 cases of COVID-19 were observed in the placebo group vs five cases observed in the mRNA-1273 group; again, no significant safety concerns were noted and Moderna is also planning to submit the EUA to the FDA in the coming weeks. In our view this is a positive signal for all the vaccines in development as these initial data go some way to validating the approach taken by most (immunisation against the coronavirus spike protein). Pfizer/BioNTech have guided that they will have c 1bn doses available during 2021, but we note downstream challenges in fill-finish, cold-chain storage, as well as the logistical hurdle of vaccinating the public en masse could present

bottlenecks for mass vaccination. We note that Moderna aims to manufacture 500m to 1bn doses globally in 2021 and that its mRNA vaccine technology means that mRNA-1273 is able to be stored in standard refrigeration units for up to 30 days (and six months in normal freezers), which is an improvement on the Pfizer/BioNTech vaccine cold-chain storage requirements.

Unique dual MOA with convenience of once a day tablet

We highlight that depending on its efficacy and given its convenience as a once a day tablet, it is possible that future clinical trials could focus on bemcentinib's use in the community setting or even as a post-exposure prophylactic; however, the impact of vaccination would define the need in this setting. We note that approximately 30% of people with SARS or Middle East Respiratory Syndrome (MERS) had persisting lung abnormalities after their acute illness. The NHS recently published guidance that lays out the expected aftercare needs of patients recovering from COVID-19 infections and identifies potential longer-term respiratory problems including chronic cough, fibrotic lung disease, bronchiectasis and pulmonary vascular disease. We believe BGBIO's fully humanised antibody tilvestamab (administered intravenously) that is highly selective for AXL could have potential to be developed for chronic COVID-19 related lung fibrosis.

Preclinical data promising; all eyes rest on the Phase II clinical data

SARS-CoV-2, the virus causing COVID-19 disease, is a membrane-bound or enveloped virus and like SARS-CoV, the SARS-CoV-2 spike glycoprotein primarily utilises the ACE2 cellular receptor to enter and infect cells. Enveloped viruses express phosphatidylserine (apoptotic mimicry) on their membranes that binds to Gas6 (the AXL receptor ligand) and mediates AXL dependent cell entry (through fusion with endosomes). Preclinical work suggests that AXL plays a key role in viral infections via two mechanisms (Exhibit 13):

1. it is a key receptor used by enveloped viruses (SARS-CoV-2, Ebola and Zika viruses) to gain entry into cells, facilitating viral replication and spread.
2. it has been implicated in the suppression of the type 1 interferon response, a key anti-viral defence mechanism of the innate immune system.

Selective AXL inhibition with bemcentinib has been shown to inhibit both these mechanisms (viral entry and enhanced interferon response) in animal models, as well as potentially turning on the adaptive immune response.

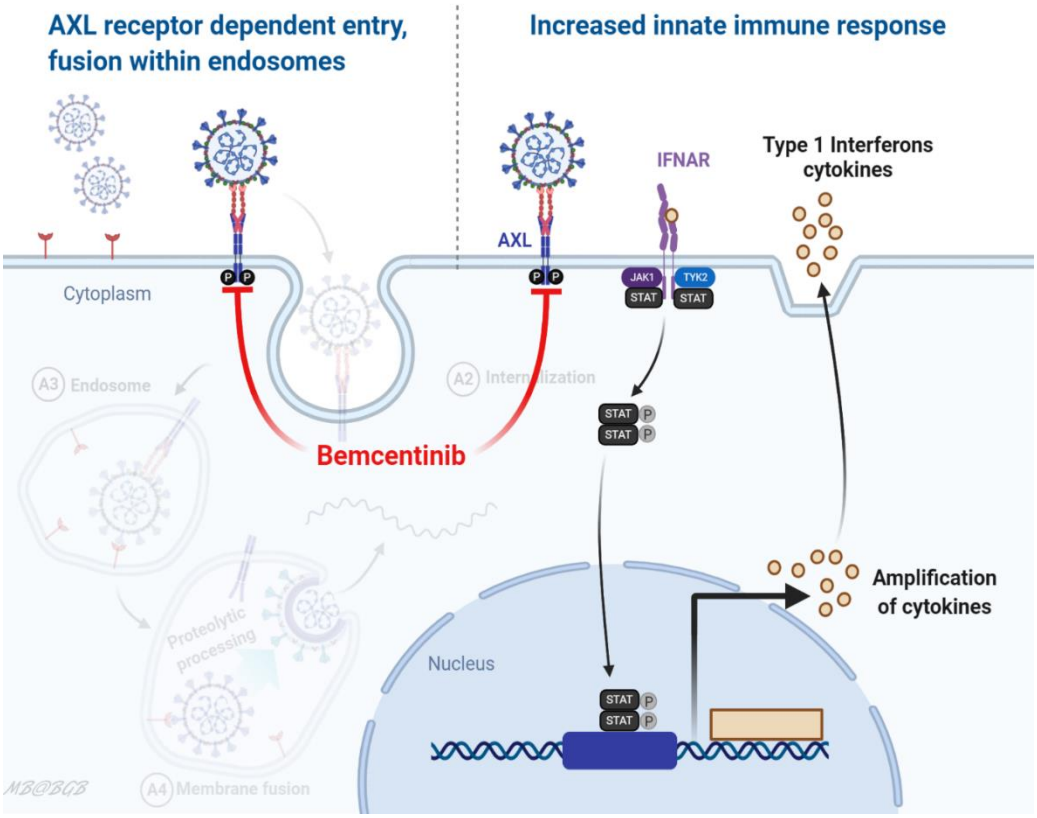
First treatment selected for the UK's ACCORD-2 trial

In April this year, bemcentinib was selected as the first treatment to be included in the ACCORD-2 clinical trial, which is being primarily funded by the Department of Health and Social Care (DHSC) and UK Research and Innovation (UKRI), with BGBIO making a modest financial contribution and providing drug material (bemcentinib). The first patient was dosed in early June, however, following a substantial decline in the incidence of COVID-19, the UKRI withdrew funding for the trial in late July and recruitment ceased. In late September, following a rise in the number of COVID-19 cases in the UK, the UKRI reinstated funding for the ACCORD-2 study. The first cut of data is now expected in H220 and data gathered before the trial was halted in July will be included in the analysis. It means that if positive, bemcentinib could progress into a larger-scale Phase IIb/III study before year end. Furthermore, as the primary mechanism for SARS-CoV-2 cell entry is thought to be the spike protein-ACE2 interactions, bemcentinib could find use in combination with a treatment that targets the spike protein-ACE2 pathway. Bemcentinib's tolerability makes it an ideal candidate for combination approaches.

ACCORD-2 (n=120) is a multicentre Phase II adaptive platform trial to assess the safety and efficacy of multiple candidates as add-on therapies to standard of care (SOC) treatments that include remdesivir and dexamethasone in hospitalised patients at stages 3–5 on the World Health

Organization's nine-point ordinal scale. The primary endpoint of the study will be time to clinical improvement of at least two points on the ordinal scale or live discharge from the hospital, whichever comes first. Secondary endpoints such as viral load (quantified by a polymerase chain reaction (PCR) test) will aid the planning of a potential Phase III study. This will enable bemcentinib's potential to be evaluated rapidly and feed into the UK's large-scale Phase III COVID-19 studies (such as RECOVERY).

Exhibit 13: Impact of bemcentinib on SARS-CoV-2 infection of cells



Source: BGBIO corporate presentation

Company sponsored COVID-19 study, BGBC020

Due to the uncertainty of progress in the UK ACCORD-2 study, BGBIO initiated a company sponsored Phase II trial, BGBC020 (n=120) in South Africa and India (countries with significantly higher rates of COVID-19) using a trial protocol similar to ACCORD-2 with patients randomised to receive either bemcentinib plus SOC or SOC only. As with the ACCORD-2 study, the primary endpoint will be time to clinical improvement of at least two points on the ordinal scale or live discharge from the hospital, whichever comes first. The first patient was enrolled in South Africa in mid-October and initial data are expected in Q121. We expect the COVID-19 trials to progress following an accelerated timeline due to the urgent medical need so bemcentinib could receive emergency use authorisation earlier than expected.

Forecast peak COVID-19 sales of $300m

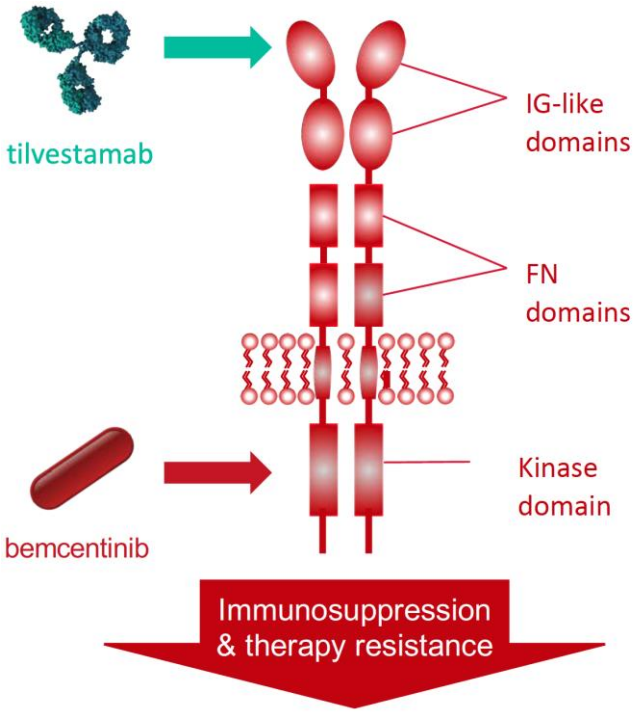
We note that there are many potential therapies in late stages of development for the treatment of COVID-19 and to reflect this likely fragmentation of the market, we forecast peak sales of $300m for bemcentinib. Another critical question is whether vaccines will be able to eradicate – or at least control – the infection rates; the durability of effect and frequency of administration in the context of evolving mutations will be key. Even in the best-case scenario, if effective vaccines are approved in late 2020/21 (US and Europe), it is likely that it will take time, likely months, to manufacture,

distribute and vaccinate the majority of the population and there could be a long tail until a sufficient proportion of the population is vaccinated to eradicate or control the virus. On the other end of the spectrum, there is a scenario where the vaccines either do not provide a reliable protection (less likely given the headline data on BNT162b2 and mRNA-1273) or there is an issue with protection durability and there will be a need to revaccinate, which could affect compliance rates. So, from the therapeutic drug perspective, near-term (probably the next several years) demand for effective treatment options is likely, albeit for smaller population numbers and if bemcentinib is efficacious. COVID-19 could represent a huge global opportunity, particularly as the scale of manufacturing is not an issue for a small molecule pill with a low cost of production. Medium- to near-term prospects are less visible and will depend on the efficacy of preventative measures, but also how other therapeutic drugs will perform in ongoing clinical trials.

AXL antibody tilvestamab

Inhibition of AXL activity can be achieved by targeting either the intracellular or extracellular binding domain. Bemcentinib is a highly selective small molecule inhibitor of the intracellular kinase domain, and tilvestamab (BGB149) is a fully humanised IgG1 monoclonal antibody that exhibits high affinity and selectivity for the extracellular domain and blocks AXL signalling by competitively inhibiting the binding of the Gas6 ligand, Exhibit 14. This 'multiple shots on goal' strategy increases the chance of success by using complementary but orthogonal treatment modalities. There are a number of key differences between these approaches. Therapeutic antibodies like tilvestamab are administered intravenously, unlike small molecule bemcentinib which can be taken orally. However, this is balanced by the long half-life of antibodies, which allows weeks between dosing instead of the daily dosing of small molecules. This can prove advantageous for treating chronic diseases (like fibrosis) as it increases treatment adherence, although is less convenient. Due to the complexity of manufacturing biologics (bioreactors), monoclonal antibodies tend to be more expensive but are more selective and have a higher affinity for their target receptor.

Exhibit 14: Schematic of the AXL transmembrane receptor. Tilvestamab targets the extracellular domain and bemcentinib targets the intracellular kinase domain



Source: BGBIO corporate presentation

BGBIO's second clinical asset tilvestamab is a first-in-class AXL-targeting antibody that was developed in-house and is wholly owned by BGBIO. It has shown promising preclinical anti-tumour activity and there are currently no other AXL-targeting monoclonal antibodies in clinical development. Tilvestamab is currently in a Phase I first in human single ascending dose study in healthy volunteers (n=24) to establish safety and tolerability as well as pharmacokinetics. If the final read out is positive and safety is maintained, expansion to the Phase Ib multiple ascending dose study is expected in Q420.

BGBIO has not yet disclosed its development plans for tilvestamab, which has the potential to be used in both oncology and fibrotic indications. We expect to receive more clarity from BGBIO once the Phase I trial has completed. We believe that the development of tilvestamab will most likely be pursued primarily in fibrosis due to the potential for pricing negotiations without affecting other indications, as fibrotic treatments in general reimburse at a lower rate than oncology treatments. Tilvestamab has already been shown to reduce the transcription of fibrotic genes in preclinical IPF models and alveolar epithelial recovery. We also note that tilvestamab could be developed for the treatment of COVID-19 and through its potent inhibition of AXL will benefit from the same dual mechanism of action as bemcentinib, inhibiting AXL-mediated viral entry and enhancing the type 1 interferon response (a key anti-viral defence mechanism of the innate immune system).

Competitive landscape for AXL inhibition

There are currently several FDA-approved multi-kinase inhibitors that unintentionally exhibit activity against AXL in addition to their primary targets. These nonselective inhibitors notably include Pfizer's Bosulif (bosutinib) an ABL/SRC kinase inhibitor approved for r/r chronic myelogenous leukaemia and Xalkori (crizotinib) an ALK/ROS1/MET inhibitor approved for NSCLC patients harbouring these specific driver mutations. Other approved inhibitors that have shown activity against AXL include Cabometyx/Cometriq (cabozantinib) and Sutent (sunitinib). Interestingly, in some cases, preclinical studies have suggested that their inadvertent inhibition of AXL may in part be responsible for their antitumour activity.

The unique biology of AXL, and growing evidence that it plays a critical role in mediating treatment resistance, tumour immune evasion and the development of metastatic disease, has led to an increasing interest in it as a standalone target in recent years. Bemcentinib is currently the most advanced AXL selective oral small molecule inhibitor in clinical development. However, BGBIO is facing growing competition as AXL's value proposition becomes better understood. Exhibit 15 summarises the wider competitive landscape; this includes selective AXL inhibitors, multi-kinase inhibitors and antibody-drug conjugates.

Bemcentinib's key advantage lies in its potent and highly selective inhibition of AXL. It therefore exhibits a clean safety profile and its broad tolerability is ideal for use in combination with other treatments. This is highlighted by the fact that no dose reductions were required when used in combination with checkpoint inhibitors, targeted therapies or chemotherapy in the ongoing Phase II studies. We see this as the largest commercial opportunity for AXL inhibitors due to their unique MOA, which makes tumours more responsive to treatment.

Exhibit 15: Competitive landscape – examples of drugs in development that are targeting AXL

Product	Company	MOA and Target	Status	Indication	Notes
AB-329 (Previously DS-1205c)	AnHeart Therapeutics	Small molecule AXL inhibitor	Phase I	EGFRm+ NSCLC plus Tagrisso	In-licensed from Daiichi Sankyo in September 2020. Potent and highly selective inhibitor of AXL that is orally dosed. AnHeart plans to pursue development in combination with CPIs or Tagrisso in NSCLC, as well as other solid tumours and haematological malignancies. The estimated primary completion date of the Phase I study in Taiwan is April 2021.
Dubermatinib (TP-0903)	Sumitomo Dainippon Pharma	Small molecule AXL inhibitor	Phase I	Advanced solid tumours (EGFRm+ NSCLC, plus TKI or immunotherapy	Potent and selective inhibitor of AXL that is dosed orally. Preliminary data from the ongoing Phase Ib expansion (n=132) is promising. Efficacy data are still being analysed and the estimated primary completion date of the Phase I study is December 2020.
			Phase Ib/II	r/r AML (FLT3+) plus Vidaza	Phase I/II study in r/r FLT3 mutation-positive AML patients as a monotherapy and in combination with SOC HMA Vidaza (azacitidine). The estimated primary completion date of Phase I/II study is December 2022.
Xospata (gilteritinib)	Astellas Pharma	Small molecule inhibitor of FLT3 and AXL	Marketed	r/r AML (FLT3+)	Rational of combined inhibition of FLT3 and AXL is that AXL overexpression has been shown to drive resistance to FLT3 inhibitors. Approved by the FDA in 2018 for the treatment of patients with relapsed or refractory FLT3-mutated AML. The Phase III ADMIRAL trial (n=371) reported an OS of 9.3 months and CR in 14% of patients with a mDOR of 14.8 months. Ongoing Phase I/II studies in AML in combination with checkpoint inhibitor Tecentriq and venetoclax.
Sitravatinib	Mirati Therapeutics	Multi-targeted small molecule inhibitor	Phase III	2/3L NSCLC plus Opdivo	Orally available small molecule inhibitor of multiple receptor tyrosine kinases including TAM (TYRO3, AXL and MER), VEGFR2 and KIT. Interim data from the Phase III SAPPHIRE study in 2/3L NSCLC in combination with Opdivo is expected in H221. Encouraging preliminary OS data (18.1 months) from the Phase II MRTX-500 trial (n=73) in combination with Opdivo in NSCLC patients who have progressed on/after receiving a checkpoint inhibitor. Other ongoing Phase II studies include urothelial cancer and breast cancer.
Enapotamab vedotin (HuMax-AXL-ADC)	Genmab	Antibody drug conjugate (ADC) targeted to AXL	Phase I/II	Solid tumours	First in human Phase I/II study in solid tumours, notably NSCLC, melanoma and ovarian cancer. Expansion cohort in r/r NSCLC patients without EGFR/ALK driver mutations (n=26) achieved ORR of 19%, DCR of 50% and 75% of evaluable patients were AXL positive by tumour cell staining. The estimated primary completion date of the Phase I/II study is February 2021.

Source: Edison Investment Research. Note: +: mutation positive; HMA: hypomethylating agent; r/r: relapsed or refractory; DCR: disease control rate.

Multi-kinase inhibitors are seen as less competitive due to their inferior safety profiles as a result of off-target engagement that is inherent to non-selective inhibitors. This reduces their potential for use in combination with other therapies. We believe bemcentinib's nearest competitor is Sumitomo Dainippon Pharma Oncology's (previously Tolero Pharmaceuticals) orally dosed selective AXL inhibitor dubermatinib (TP-0903). This asset is currently in a Phase I/II study in r/r AML as a monotherapy and in combination with standard of care HMA azacitidine, as well as a broad Phase I study in solid tumours that includes NSCLC and melanoma in combination with Keytruda or Tagrisso. Bemcentinib's key advantage is it is currently poised to be the first to market and its biomarker-driven strategy will expedite this process. BGBIO's AXL-targeting monoclonal antibody tilvestamab is currently in a Phase I first in human study in healthy volunteers (n=24). This offers an alternative way of targeting AXL by inhibiting the extracellular domain and takes advantage of the high selectivity of antibodies, although dosed by IV. Tilvestamab has shown promising preclinical anti-tumour activity and there are currently no other AXL-targeting monoclonal antibodies in clinical development. BGBIO has out-licensed an AXL-targeting antibody for use in ADC Therapeutics antibody drug conjugate ADCT-601 (previously BGB601). The rationale here is to utilise the high selectivity of an AXL-targeting antibody to deliver a non-selective cytotoxin to tumour cells with high AXL expression, although IV dosing is required. Data from the Phase I study in advanced solid tumours were underwhelming and the trial was terminated. We note that the licence has not been returned to BGBIO and ADC Therapeutics may be pursuing further development with a combination strategy. Genmab's ADC enapotamab vedotin (HuMax-AXL-ADC), is in a more advanced stage of development; Phase I/II trials in multiple types of solid tumours are ongoing.

Sensitivities

BGBIO is subject to the usual risks associated with drug development including clinical development delays or failures, regulatory risks, competitor successes, partnering setbacks and financing risks. BGBIO has two clinical stage assets in development and value crystallisation in the future will depend on successful R&D progress and potential partnering activities. BGBIO is focused on the inhibition of AXL and if this was proven to be a futile target then it would have major implications for the company's pipeline. Additionally, treatments are targeted at patients with high AXL expression. Therefore, successfully developing a suitable diagnostic to facilitate the biomarker-driven strategy of developing precision medicines is paramount. Furthermore, uncertainty remains around the proportion of patients that exhibit high AXL expression. This is likely to vary by disease and a lower prevalence of patients with higher AXL expression than expected could compromise treatment efficacy and the probability of success, as observed in the Phase II study in breast cancer.

The biggest near-term development sensitivity relates to BGBIO's lead asset bemcentinib. Given this highly selective AXL inhibitor could be first-in-class, timely development is necessary to maintain its potential first-to-market position. The largest commercial opportunity for AXL inhibitors is in combination with other therapeutic treatments. Bemcentinib's composition of matter patent expires in 2027, but it is likely this could be extended by five years. Additionally, the FDA grants new chemical entities such as bemcentinib five years' market exclusivity upon approval.

BGBIO will continue to be cash consumptive and operate as a non-revenue-generating biotech for the foreseeable future. However, any increases in clinical trial costs or head count could reduce our forecast cash runway.

Valuation

We value BGBIO at NOK5.16bn or NOK59.1 per share based on a risk-adjusted NPV analysis, which includes cash and cash equivalents of NOK777.9m at end-Q320 (BGBIO is debt free). Our risk-adjusted valuation includes bemcentinib in 2L NSCLC (peak sales $1.2bn, NOK40.7/share) and 2L AML (peak sales $588m, NOK13.3/share) oncology indications plus the COVID-19 opportunity ($300m peak sales, NOK5.9/share), Exhibit 16. We use a 12.5% discount rate for assets in development. We assume a licensing deal for bemcentinib (in all oncology) after registrational intent data in AML and include a pay away to Rigel based on the original in-licensing deal (Exhibit 17).

Exhibit 16: Sum-of-the-parts BerGenBio valuation

Product	Indication	Launch	Peak sales ($m)	NPV (NOKm)	Probability of success	rNPV (NOKm)	NPV/share (NOK)
Bemcentinib	2L AML	2024	588	3,260.3	35%	1,161.7	13.31
Bemcentinib	2L NSCLC	2025	1,165	8,107.9	35%	3,554.4	40.73
Bemcentinib	COVID-19	2022	300	3,684.7	15%	517.1	5.93
Payments to Rigel						(850.8)	(9.75)
Net cash, last reported				777.9	100%	777.9	8.91
Valuation				**15,830.7**		**5,160.1**	**59.1**

Source: Edison Investment Research. Note: WACC = 12.5%.

Currently we include only bemcentinib 2L in AML and NSCLC peak sales in our model and potential upside includes utility in the 1L setting plus other oncology indications (eg indications under investigator-led trial); value for the latter is captured implicitly through our deal valuation assumptions. We do not assign any value to tilvestamab, as BGBIO progresses this asset and we have clarity on the prioritised indications for development we will review the potential of this asset. We highlight that as a wholly owned asset, high economic value potentially resides in this asset.

Exhibit 17: Assumptions for valuation

Asset/indication	Comments
Bemcentinib 2L AML/MDS	■ Target population*: is around 15k in the US and 18k in Europe, which is newly relapsed/refractory AML patients. AXL expression assumed in 50% of patients. Assumed 30% peak penetration, due to specific target population. ■ Pricing: $100k per patient per year in the US, 50% discount in Europe. Peak sales in five years. ■ Trial timelines and R&D cost: $30m to conduct a Phase III study (2021-2023); then out-licensed; launched in 2024.
Bemcentinib 2L NSCLC	■ Target population*: is around 25k in the US and 27k in Europe. Assumed 30% peak penetration, due to specific target population. – Calculated as follows. Total lung cancer expected incidence is 223k in the US and 242k in Europe in 2020 (see notes for countries included in the model), with 85% being NSCLC. Stage IIIb/IV approximately 53% of NSCLC cases; 76% are EGFR wild-type, and no ALK rearrangements and we estimate most (90%) are treated 1L and 2L with a checkpoint inhibitor and chemotherapy either individually or in combination (Keytruda + platinum doublet chemotherapy). Many of these patients (we estimate 50%) move on to 2L/3L. Of these patients, those who have high AXL expression (c 50%) are addressable with bemcentinib. ■ Pricing: $100k per patient per year in the US, 50% discount in Europe. Peak sales in five years. ■ Trial timelines and R&D cost: Assumed BGBIO will sponsor $33m out of the total trial cost of $50m to conduct a Phase III study (2021–23); out-licensing assumed in 2023, which is when the partner takes over the development. Regulatory approval around one year and launch assumed in 2025.
Bemcentinib COVID-19	■ Top-down valuation approach: Oseltamivir (Tamiflu, Roche) and remdesivir (Gilead Sciences) used as benchmark drugs. Tamiflu achieved sales of $3.0bn during the H5N1 bird flu epidemic in 2009. Gilead's remdesivir (Veklury), which received FDA approval in October 2020, is now forecast to reach sales of $2.4bn in 2020 and will peak at around $3bn. Both drugs had no real competition, while there are many potential therapies in late stages of development for the treatment of COVID-19. To reflect this likely fragmentation of the market, we therefore use peak sales of $300m for bemcentinib, roughly one tenth of the peak sales achieved by oseltamivir (and forecast for remdesivir). ■ Pricing: $100k per patient per year in the US, 50% discount in Europe. Peak sales in two years. ■ Trial timelines and R&D cost: $10m for the registrational Phase III trial. Launch in 2022. ■ Commercial strategy: in the rNPV project we apply COGS and S&M margins that add up to 25%, which is a conservative assumption of profitably of an anti-infective drug. BGBIO might as well partner with a pharma company that has commercial capability in place. ■ Product life cycle is unique. We assume peak sales in 2022/23, then decline to 0 by 2026 (see text).
Licensing deal assumptions	■ We assume a deal in 2023 and use the median values of benchmark deals in Exhibit 18. Upfront payment of $250m, $1.4bn in total milestones (one-third allocated to R&D-related payments; the rest are commercial milestones). Tiered 15–18% royalty rates used. The deal values are split proportionally (using peak sales) and allocated to AML and NSCLC projects to get true rNPV per share value.
IP status and agreement with Rigel	■ Bemcentinib. In-licensed. Composition of matter patent expires in 2027, but there is likelihood this could be extended by five years. In addition, new chemical entities are granted a market exclusivity period in certain cases. ■ In 2011, BGBIO in-licensed the intellectual property associated with bemcentinib from Rigel (exclusive worldwide licence). R&D milestones to Rigel are payable: – After the initiation of the first Phase III trial ($8m) – After the submission of the NDA to the FDA ($12m) – After the approval of the first drug ($16m) ■ Rigel will be eligible for tiered royalties of 5% (net sales <$500m), 7% ($500m-$1bn) or 9% (>$1bn). If BGBIO sub-licenses bemcentinib, then its revenues will be shared with Rigel: – 35% after completion of first Phase II trial with 60 or more patients; – 30% after completion of a Phase III study. ■ IP relating to all biomarkers and companion diagnostics is wholly owned by BGBIO and falls outside the scope of the licensing deal with Rigel, thus there in no pay away. BGBIO's second clinical asset tilvestamab was developed in-house and is wholly owned by BGBIO.

Source: Edison Investment Research. Note: *Target countries used in the model are the US, and top 14 European countries (EU4 + the UK, Netherlands, Belgium, Luxembourg, Denmark, Finland, Norway, Sweden, Austria and Switzerland).

Bemcentinib in 2L AML and 2L NSCLC

Currently, we value bemcentinib only in 2L AML and 2L NSCLC. These two settings likely represent the fastest route to market, given the huge unmet need; however, AXL overexpression has been demonstrated in many other cancer types. Bemcentinib's potential could extend well beyond these two indications. To capture the potential value across a wider oncology setting, we have reflected a global out-licensing deal that covers bemcentinib in oncology. We also believe that a proof-of-concept in one indication could have a read-across effect for a potential partner to other indications with a known overexpression of AXL (one with an established PD-1 for example would make sense).

We use a bottom-up approach to calculate the market sizes in oncology, utilise industry average data for the basis of our other assumptions (eg probability of success, eligible patient population, pricing), Exhibit 17. The partnering strategy is a key element in our rNPV valuation of BGBIO. We note that partnering deals can vary widely from co-development and co-commercialisation to full out-licensing globally or for specific territories. A partnering deal could also centre on specific indications or the entire asset could be out-licensed. BGBIO is likely to go with an optimal strategy depending on the strength of the data. The timing of any deal is uncertain, but in our model we

assume this happens in 2023. This means there is potential for BGBIO to rapidly expand the R&D pipeline (which we would reflect in our valuation model accordingly as clinical trials are initiated by the company). Thus, the investigator-led trials are particularly useful as BGBIO could fast-track the indications if the data look attractive.

In our assumptions on a deal we reflect biotech licensing deals that included assets with potential in multiple indications, Exhibit 18. Based on deals that have occurred since 2015, we assume an upfront payment of c $250m and c $1.4bn in total milestones (one-third allocated to R&D-related payments such as completion of the Phase III trial and NDA approval; the rest are commercial milestones). We assume tiered royalty rates of 15–18% on sales. The deal values are split proportionally (using peak sales) and allocated to AML and NSCLC projects to get a true rNPV per share value.

Exhibit 18: Phase II oncology deals used as a benchmark

Date	Licensor	Licensee	Product	Pharmacological class/target	Upfront ($m)	Milestones ($m)
04/09/2020	AbbVie	I-Mab	lemzoparlimab (TCJ4)	anti-CD47 mAb	200	1,740
27/05/2020	Gilead	Arcus Biosciences	zimberelimab (AB122) domvanalimab (AB154)	anti-PD-1 mAb anti-TIGIT mAb*	175	1,225
05/02/2019	GSK	Merck KGaA	bintrafusp alfa (M7824)	TGF-βxPD-L1 bsAb	354	4,012
05/07/2017	Celgene	BeiGene	tislelizumab (BGB-A317)	anti-PD-1 mAb	263	980
10/02/2017	Seattle Genetics	Immunomedics	sacituzumab govitecan (Trodelvy)	TROP2 ADC	250	1,700
15/10/2015	BMS	Five Prime	Cabiralizumab (FPA008)	CSF-1R mAb	350	1,390
24/04/2015	AstraZeneca	Innate	Monalizumab (IPH2201)	anti-NKG2A mAb	250	1,025
Median					**c 250**	**c 1,390**

Source: Edison Investment Research, EvaluatePharma. Note: *Gilead/Arcus deal includes options for additional assets not listed; we had excluded the licensing deals signed between BMS/Nektar and AstraZeneca/Daiichi Sankyo as outliers.

Bemcentinib for treatment of COVID-19

Our valuation of BGBIO includes the COVID-19 opportunity for bemcentinib; for this we use a top-down approach due to the inherent uncertainties about the epidemiology and thus the future market size. Challenges in modelling the potential of a drug treatment for COVID-19 relates to the rapidly evolving infection levels across different regions. The 'go-stop-go' scenario that played out since April this year and the associated share price volatility only exemplifies that. Nevertheless, the scientific rationale behind AXL inhibition in viral infections and existing preclinical data seem sound. In addition, this is an opportunity to reach the market much quicker than in cancer indications.

As such, bottom-up modelling of sales (ie calculating the target patient population and growth rates years in advance) is not appropriate given the rapidly changing epidemiology. Of note is that the achievable pricing in infectious diseases and oncology will obviously be different, although it is too early for BGBIO to guide on any potential commercial details. Using a top-down approach, we looked at the performance of two anti-viral drugs during a pandemic. Roche's oseltamivir (Tamiflu) achieved sales of $3bn during the H5N1 bird flu epidemic in 2009. Gilead's remdesivir (Veklury), which received FDA approval in October 2020 (even though efficacy data were limited), is now forecast to reach peak sales at 2021 of ~$3bn (coincidentally, but likely that consensus is basing assumptions on Tamiflu as the comparator). We forecast peak bemcentinib, COVID-19-related sales of $300m which is roughly one-tenth of the peak sales achieved by oseltamivir (and forecast for remdesivir). Our rNPV project for this indication is relatively short and extends to 2025 only from the assumed launch in 2021/22, so three to four years with peak sales in the first two years, then rapid decline. We believe that current consensus forecasts of remdesivir sales support our approach, ie sales peak in the near term, then decline. The caveat is that it is still early days and the remdesivir consensus will inevitably be revised, as the new preventative and therapeutic drugs

currently in Phase III trials will start reporting data in coming months. Furthermore, the approval and potential roll-out of multiple vaccines will be of prime importance. We will revise our assumptions as new information appears. Of note is the fact that even though bemcentinib is being investigated in the in-hospital setting, like remdesivir, due to the convenient use (one-a-day pill) there is potential to expand the label for use in the community setting, which is a far larger patient pool.

Financials

BGBIO's operating loss in 2019 was NOK204.4m, up by 5.1% from the previous year. So, the cash burn rate has been fairly stable over the past three years while the company has run its Phase II programme. The operating loss in 9M20 was NOK189.3m versus NOK145.3m in 9M19. We forecast the operating loss will increase to NOK260.7m in 2020 and to NOK305.2m in 2021 as BGBIO enters the Phase IIb/III stage of development. At 30 September 2020 (end-Q320), BGBIO had a comfortable cash position of NOK777.9m. If the COVID-19 programme does not proceed into Phase IIb/III, then the cash reach likely extends into 2023, but if the COVID-19 programme proceeds, then existing cash would be sufficient until 2022.

In 2021, BGBIO should start working on the potentially pivotal trials in AML and NSCLC. The rapidly developing COVID-19 programme is the main focus area for us in terms of BGBIO's cash flows in the near term (2021/22). If the readouts from the ongoing studies warrant bemcentinib's progress into the Phase III trial, this will further increase R&D spending. In addition, a total of $36m could become payable already in 2021/22, but only if all goes well with the COVID-19 programme. $8m is due on Phase IIb/III trial initiation, but if the Phase IIb/III trial in COVID-19 is successful, the remainder of the R&D-related payments could be covered from multiple sources, in our view. The funding of the AML and NSCLC programmes will depend on the interplay of whether the COVID-19 programme moves into a Phase III trial, what payments to Rigel will be made and how successful the initial launch of bemcentinib for the treatment of COVID-19 will be. As discussed above, we have modelled the peak sales immediately after the launch. So, even though the new COVID-19 programme decreases the visibility of cash burn in the next two years, the potentially rapid uptake of the drugs is a significant prize. Our current financial projections do not include the milestones to Rigel, nor the potential sales already in 2022.

Appendix 1: Understanding the role of AXL in oncology

Cancer is recognised as an incredibly heterogenous disease. In recent years the armament for cancer treatment has widened with the advent of targeted therapies, immune modulators (PD-1 inhibitors) and even CAR-T cell therapies for subsets of haematological cancers. Critical to individual patient treatment paradigms (which increasingly include combination drug strategies) is understanding the individual tumour pathway, activated genes and consequent aberration etc. Understanding the tumour microenvironment and why even with initial positive response to treatment classes, cancers often exhibit tumour proliferation, metastasis and treatment resistance is becoming an ever-more critical focus area. The role of AXL is becoming increasingly defined in tumorigenesis, propagation and treatment resistance.

AXL (derived from anexelekto, meaning uncontrolled) is a member of the TAM family of receptor tyrosine kinases that includes TYRO3, AXL and MER. These transmembrane receptors are characterised by an extracellular domain that is activated in part by the binding of the vitamin K-dependent ligand growth arrest-specific protein 6 (Gas6) and an intracellular tyrosine kinase domain. The canonical activation of AXL by Gas6 requires an additional interaction between Gas6 and phosphatidylserine (PS), a phospholipid ubiquitously expressed in cell membranes but specifically found on the surface of apoptotic cells and enveloped viruses (such as SARS-CoV-2).

This triggers AXL dimerisation, phosphorylation of the intracellular kinase domains and downstream signalling that has been implicated in a myriad of tumour cellular processes (Exhibit 19) and plays a critical role in immune suppression, EMT, tumour cell proliferation, migration and treatment resistance.

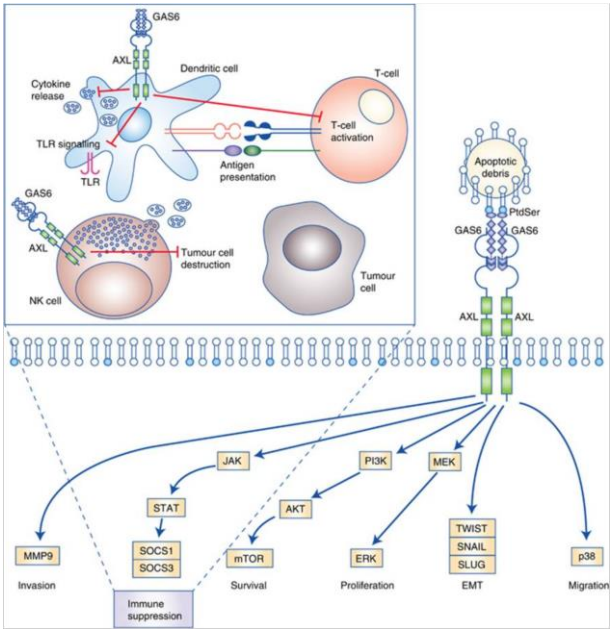
AXL inhibition as a therapeutic target

Unlike with conventional cancer treatments, the inhibition of AXL signalling does not directly induce apoptosis of tumour cells, instead it enhances the body's immune response and through reversal of the EMT, makes cancer cells more susceptible to attack by the body's immune system or a combination therapy.

AXL is unique among the tyrosine kinases in the sense that it is not a traditional oncogenic driver; genetic aberrations (mutations, fusions and amplifications) are very rare (AXL is altered in 1.79% of all cancers). AXL overexpression is instead primarily driven by epigenetic upregulation through post-translational mechanisms. Additionally, multiple transcription factors that are upregulated in the tumour microenvironment (HIF-1α) have also been implicated in AXL upregulation and overexpression.

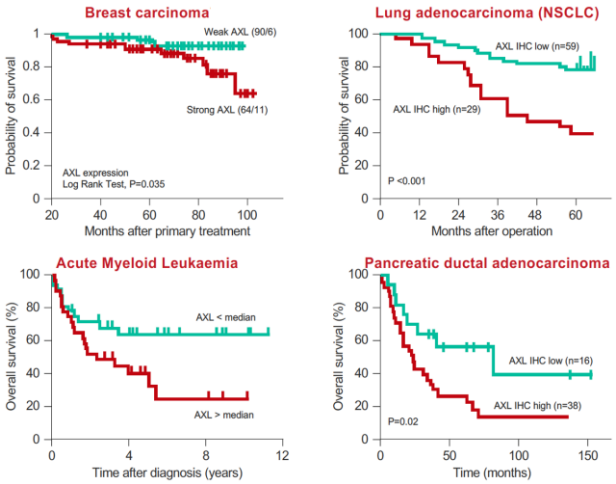
Cells exhibit low levels of AXL expression under healthy physiological conditions and its function in normal tissues include the clearance of apoptotic material and the dampening of innate immune-mediated inflammatory responses and NK cell activity. AXL is overexpressed under pathological conditions of stress such as hypoxia (lack of oxygen in the tumour microenvironment), inflammation (due to immune reaction) and therapeutic treatment (chemo-toxicity). This is a desirable characteristic for a therapeutic target as it allows cancerous cells to be selectively targeted. AXL overexpression has been identified as a negative prognosis factor in a multitude of cancers as well as other diseases, Exhibit 20. The success of this therapeutic target is predicated on being able to identify patients with sufficient AXL overexpression to benefit from its inhibition. The development of a diagnostic is therefore imperative.

Exhibit 19: AXL signalling is involved in a plethora of cellular processes in tumour cells



Source: www.ncbi.nlm.nih.gov/pmc/articles/PMC5318970/

Exhibit 20: AXL expression correlation with overall survival in a range of cancers



Source: BGBIO corporate presentation

AXL-erating tumourigenesis

The activation of AXL gives rise to a vast array of downstream signalling pathways that elicit a variety of cellular responses. Notably it promotes tumour cell survival and proliferation via the well-characterised PI3K/AKT pathway and MEK/ERK pathway respectively. Furthermore, it is also known to promote angiogenesis (the formation of new blood vessels from pre-existing vasculature), which is critical to tumour growth and migration. The two processes that have attracted the most attention from drug developers are its crucial roles in driving EMT and immune suppression.

AXL affects EMT

AXL is an essential mediator of EMT, a cellular process that enables cancer cells to migrate to other organs (metastasise), evade the immune system, and exhibit treatment-resistant properties. EMT is a reversible process in which cells undergo a transition from an epithelial phenotype, characterised by uniform cells in which cell-cell adhesion and interactions with basement membrane maintain tissue integrity, to a mesenchymal phenotype. Mesenchymal cells are unspecialised (have stem cell-like properties), have a more irregular fibroblast-like morphology with minimal cell-cell interactions, and exhibit increased migratory capacity and invasive properties. This plasticity allows tumour cells to seed secondary tumours in distant organs. AXL activation drives EMT and enables cells to retain a mesenchymal phenotype. However, EMT is a reversible process and cells can regain their epithelial properties in the absence of AXL.

AXL causes immune suppression

Mesenchymal cells are astute at evading the body's immune system as the immune cells (cytolytic T cells and NK cells) are unable to form a tight immune synapse with them due to their irregular morphology. This is further compounded by the immunosuppressive effects of AXL overexpression on immune cells (dendritic cells, macrophages and NK cells) where it plays a critical role in inhibiting cytokine release and decreasing antigen presentation by dendritic cells which is key to activating tumour killing T cells. Furthermore, AXL upregulates PD-L1 on tumour and dendritic cells, which further supresses T-cell activity. In the tumour microenvironment, AXL signalling has been reported to induce macrophage polarisation from the tumour cell killing M1-type, to protumour and anti-inflammatory M2 macrophages. AXL expression has also been linked to innate and acquired resistance to chemotherapy, targeted therapy, immunotherapy and radiation therapy.

Companion diagnostics and biomarkers for AXL

Identifying AXL expression in solid tumours – cAXL

The current industry standard and most widely utilised cancer diagnostics use antibody based IHC methods to determine the optimal course of treatment for most patients. BGBIO has developed a robust AXL IHC diagnostic that measures the AXL staining on both cancer cells and cells of the immune system in the tumour microenvironment to generate a proprietary cAXL score that can be used to stratify patients. The hope here is that this diagnostic assay can be seamlessly included in the standard biomarker screening panel for relevant cancers.

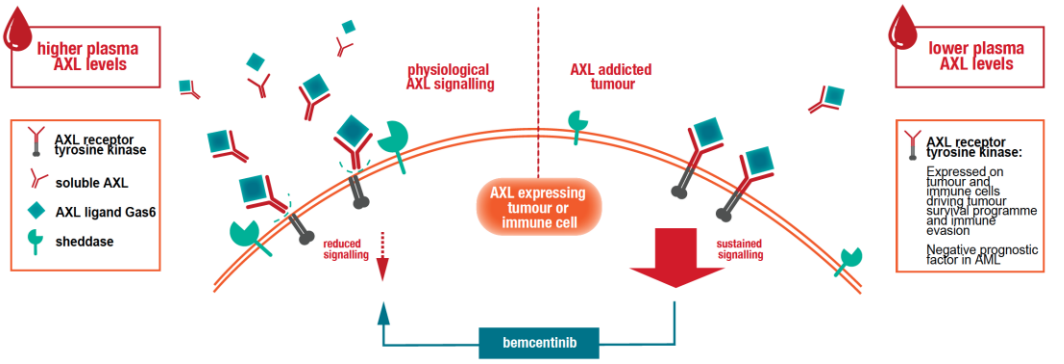
The cAXL score will form the basis of a CDx for NSCLC patients and data from Cohorts B and C of the ongoing Phase II study will be critical to further validating its utility at predicting patient responses. It is currently unclear whether this biomarker and diagnostic will be used to stratify patients in the potential registrational study for NSCLC. Instead, BGBIO may need to open the trial to all comers and then retrospectively analyse AXL status. This would allow the diagnostic to be refined further and a potentially more accurate cut-off between cAXL positive and negative patients to be determined from the larger data set. Once successfully validated, this diagnostic could be

used to prospectively stratify patients in future clinical trials and to select patients most likely to benefit from bemcentinib treatment.

sAXL status identifies responders to monotherapy in AML

BGBIO identified that the level of soluble inactive AXL (sAXL) in blood plasma is inversely correlated with the cellular level of AXL expression and is predictive of patient responses in AML. The basal patient population can be divided into sAXL low (high AXL expression on cells) and sAXL high (low AXL expression on cells), enabling easy identification of a patient's AXL status through a simple blood sample. Homeostatic downregulation of receptor tyrosine kinase signalling is particularly prominent in the case of AXL, Exhibit 21. In the absence of AXL signalling, proteases known as sheddases (ADAM10 and 17) have been shown to cleave the extracellular domain of AXL, generating sAXL, which can be detected in the blood plasma. Conversely, reduced shedding and lower levels of sAXL is observed in the presence of AXL activation and signalling associated with therapy resistance and poorer prognosis. Data to date in AML suggest that for patients with high AXL expression, inhibition of AXL signalling plays a vital role in achieving disease remission.

Exhibit 21: Expression of AXL receptor tyrosine kinase is regulated by receptor shedding



Source: BGBIO corporate presentation

Exhibit 22: Financial summary

NOK'000s	2018	2019	2020e	2021e
Year end 31 December	IFRS	IFRS	IFRS	IFRS
PROFIT & LOSS				
Operating revenues	2,335	8,900	0	0
Licensing revenues	2,335	8,900	0	0
Other revenues	0	0	0	0
Total operating expenses	(196,874)	(213,274)	(260,665)	(305,224)
Other operating expenses (R&D)	(133,699)	(141,630)	(164,325)	(205,406)
EBITDA (reported)	(194,335)	(203,589)	(259,720)	(304,844)
Depreciation and amortisation	(204)	(785)	(945)	(379)
Reported Operating Income	(194,539)	(204,374)	(260,665)	(305,224)
Operating Margin %	n/a	n/a	n/a	n/a
Finance income/(expense) excl lease expense	4,858	11,530	22,015	7,344
Exceptionals and adjustments	0	0	0	0
Reported PBT	(191,746)	(199,278)	(249,052)	(300,930)
Income tax expense	0	0	0	0
Reported net income	(191,746)	(199,278)	(249,052)	(300,930)
Average Number of Shares Outstanding (m)	53.3	58.0	80.6	87.3
EPS - normalised (NOK)	(3.6)	(3.4)	(3.1)	(3.5)
Dividend per share (NOK)	0.0	0.0	0.0	0.0
BALANCE SHEET				
Property, plant and equipment	581	974	429	450
Intangible assets	0	0	0	0
Total non-current assets	581	974	429	450
Cash and equivalents	360,413	253,586	716,292	418,494
Other current assets	17,831	15,818	16,825	16,321
Total current assets	378,244	269,404	733,117	434,815
Total non-current liabilities	0	0	0	0
Trade and other payables	23,939	26,746	38,814	40,863
Other current liabilities	12,875	21,803	22,393	22,993
Provisions	4,732	2,074	2,074	2,074
Total current liabilities	41,546	50,623	63,281	65,930
Equity attributable to company	337,280	219,754	670,264	369,334
CASH FLOW				
Operating Profit/(loss)	(191,746)	(199,278)	(249,052)	(300,930)
Depreciation and amortisation	204	785	945	379
Share based payments	1,678	3,842	0	0
Other adjustments	0	0	0	0
Movements in working capital	1,446	13,164	12,244	3,745
Interest paid / received	5,847	11,151	13,251	3,242
Income taxes paid	0	0	0	0
Cash from operations (CFO)	(186,706)	(184,145)	(235,863)	(296,805)
Capex	(228)	0	-400	-400
Acquisitions & disposals net	0	0	0	0
Other investing activities	0	0	0	0
Cash used in investing activities (CFIA)	(228)	0	(400)	(400)
Net proceeds from issue of shares	176,998	77,910	699,562	0
Movements in debt	0	0	0	0
Other financing activities	0	(593)	(593)	(593)
Cash from financing activities (CFF)	176,998	77,317	698,969	(593)
Cash and equivalents at beginning of period	370,350	360,414	253,586	716,292
Increase/(decrease) in cash and equivalents	(9,936)	(106,828)	462,706	(297,798)
Net (debt)/cash	360,413	253,586	716,292	418,494

Source: BerGenBio accounts, Edison Investment Research

Contact details	Revenue by geography
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Management team

CEO: Richard Godfrey	CFO: Rune Skeie
Richard Godfrey joined BerGenBio as CEO in 2008. He has more than 25 years' industry experience leading many international drug development and commercialisation partnerships. Formerly he served as CEO of Aenova, a specialist biopharmaceutical company. Prior to this he was the managing director of DCC Healthcare. He has also held R&D and commercial roles at Catalant, Eli Lilly and Reckitt Benckiser. Mr Godfrey qualified as a pharmacist from Liverpool University and received his MBA from Bath University.	Rune Skeie joined BerGenBio in 2018 as CFO. He has over 20 years of financial management, corporate development, corporate governance and advisory experience with public and private companies across multiple industry sectors. The majority of his career was spent at EY (formerly Ernst & Young), where he was executive director, before joining REMA Franchise Norge, the multinational supermarket business. Mr Skeie is a registered accountant and a state authorised public accountant.
CMO: Hani Gabra	**CSO: James Lorens**
Professor Hani Gabra joined BerGenBio in September 2019 as chief medical officer, based in Oxford, UK. He has extensive experience of preclinical cancer biology and clinical drug development, having previously been vice president in early clinical development at AstraZeneca in Cambridge, UK. He was previously head of medical oncology, director of the Ovarian Cancer Action Research Centre and head of the Imperial College Cancer Clinical Trials Unit, as well as chief of service of the West London Gynaecological Cancer Centre at Imperial College London. Prof Gabra's research interests include tumour suppressor genes that regulate receptor tyrosine kinase networks (including AXL), the molecular basis of clinical platinum resistance, and all phases of ovarian cancer clinical research.	Professor James Lorens is the co-founder of BerGenBio and serves as the company's senior scientific advisor and is also a professor at the department of biomedicine at the University of Bergen. On completing his postdoctoral research studies at Stanford University, he joined Rigel, a San Francisco-based biotechnology company, as a founding scientist and research director. Professor Lorens has managed several large scientific collaborations in cancer research and development with major pharmaceutical and biotechnology companies.

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