BerGenBio

Axl inhibitors for aggressive disease

Corporate Presentation

May 2020

Forward Looking statements

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BerGenBio corporate over view

World leaders in understanding AXL biology

AXL tyrosine kinase mediates aggressive disease:
immune evasion, therapy resistance & metastatic cancer, fibrosis and viral infection

Selective AXL inhibitors have the potential to treat many serious unmet medical needs

Pipeline opportunities in multiple cancers and fibrosis

3 selective AXL inhibitors in clinical development

Bemcentinib (oral once a day pill)
Tilvestamab (mAb), ADCT601* (ADC)

Phase II: Monotherapy and combos with, CPI, targeted & chemo

Biomarker correlation, parallel CDx development

Bemcentinib phase II trials:
AML (monotherapy), **AML** (chemo-combo)
NSCLC (KEYTRUDA combo) **COVID19** (mono)

Resourced to deliver milestones

Listed on Oslo Børs: BGBIO

Clinical trial collaborations with Merck and leading academic centres EU & USA

38 staff at two locations:
HQ & R&D in Bergen, Norway;
Clinical Development in Oxford, UK

*AXL antibody out licensed to ADC Therapeutics SA

Senior Management Team

Richard S. Godfrey, *Chief Executive Officer*

- Pharmacist / MBA – joined BerGenBio in 2008 as CEO
- Formerly CEO Aenova Inc., USA
- Previously Managing Director DCC Healthcare, earlier Eli Lilly, Reckitt Benckiser, Catalent
- 28 years industry experience, led and managed multiple international drug development and commercialization partnerships

Prof. James Lorens, *Founder and Chief Scientific Officer*

- Professor University of Bergen Medical School
- 30 years biotech research experience, academic biomedical research positions at Stanford University and University of Bergen
- Former Director Oncology R&D, Rigel Inc. (San Francisco, CA)
- The first to recognize that Axl kinase is an essential mediator of cancer development (EMT)

Prof. Hani Gabra MD, PhD, *Chief Medical Officer*

- MD Oncologist – joined BerGenBio in 2019
- Former VP Clinical Development Astra Zeneca UK.
- Professor of Medical Oncology at Imperial College London and Honorary Consultant in Medical Oncology at Imperial College Healthcare NHS Trust
- 20 years clinical / cancer biology research at Imperial College London.

Rune Skeie, *Chief Financial Officer*

- 20 years of financial management, corporate development, corporate governance and advisory experience across multiple industry sectors. – Joined BerGenBio in 2018
- Previously Executive Director at EY and CFO of REMA Franchise Norge AS, the multinational supermarket business.
- Registered Accountant and a State Authorized Public Auditors

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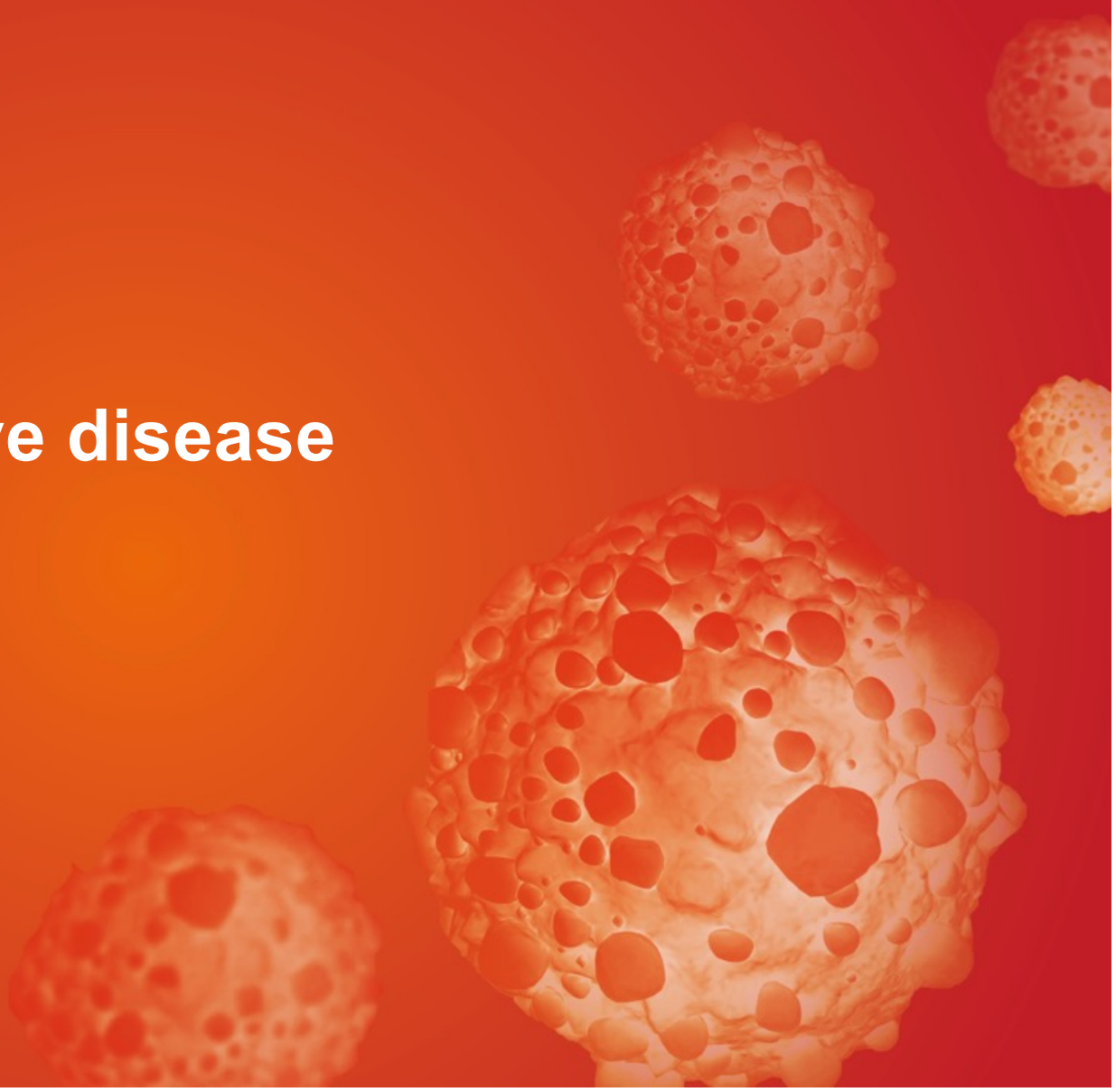
Recent highlights

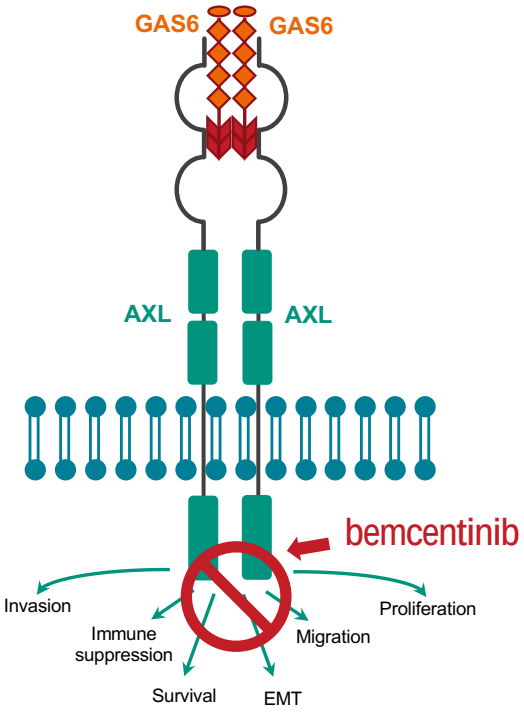
Oct 2019	FDA Fast Track designation received for bemcentinib in relapse AML
Nov 2019	Primary & Secondary endpoint of ORR met in Phase II 2L NSCLC (cohort A) in combination with KEYTRUDA® Four-fold improvement over Keytruda monotherapy**
Nov 2019	CDX: Proprietary composite AXL tumor-immune (cAXL) score developed to diagnose patients with clinical benefit Five-fold improvement in ORR and four-fold mPFS improvement for cAXL +ve patients
Dec 2020	Presented preliminary clinical data from Ph II combination trial of bemcentinib and LDAC in AML patients at ASH conference Durable responses reported with long duration
Jan 2020	Met Primary end point of ORR in phase II clinical trial in NSCLC (cohort B) in 2L IO refractory patients Bemcentinib in combination with KEYTRUDA® meets primary end point and progress to stage 2 of the study cohort
May 2020	FPI COVID19 rPhII ACCORD trial Uk Govt selected bemcentinib as first experimental compound to enter fully funded seamless platform trial for efficacy and safety

ORR – Overall Response rate OS – Overall Survival. mPFS – median Progression Free Surviva *cf. KN001; Garon *et al* NEJM 2015 ** cf. KN-010 Herbst *et al*, Lancet 2016

AXL drives aggressive disease



AXL Biology



- AXL a receptor tyrosine kinase that is important for regulating innate immune cells.[1]
- AXL levels are elevated by cellular stress and is strongly associated with inflammatory diseases including cancer and fibrosis.[2]
- It functions as a homeostatic regulator in adult tissues and organ systems that are subject to continuous challenge and renewal throughout life – immune, nervous, vascular and reproductive
- AXL drives cancer progression, immune evasion, and resistance to targeted therapies.[3]
- AXL is a key suppressor of the type I interferon response and is targeted by viruses to block the anti-viral immunity.[4]
- AXL is used by several different enveloped viruses (e.g. Ebola, Zika) to enter cells.[5]
- Bemcentinib potently inhibits SARS-CoV-2 infection of cells.[6]
- A lung cancer patient currently under treatment with bemcentinib who was high risk for COVID19 reported a mild Covid-19 infection.[7]

Very low expression under healthy physiological conditions	Elevated AXL signaling strongly associated with cancer progression, immune evasion, drug resistance and metastasis	AXL mediates viral entry to cells and dampening of viral immune response

[1]Lemke Cold Spring Harb Perspect Biol 2013; [2]Zagórska Nat Immunol 2014, Ludwig Cancer Res 2018, Espindola, Am J Respir Crit Care Med. 2018;[3]Gay, Br J Cancer 2013; [4]Chen Nat Microbiol 2018; [5]Moller-Tank Virology 2014; [6]W.Maury, unpublished;[7]BerGenBio, unpublished

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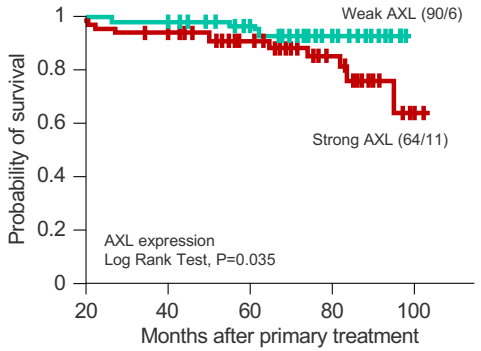
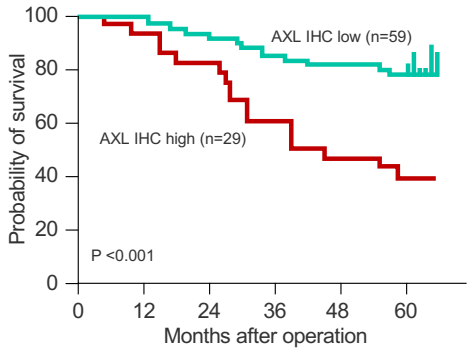
AXL is independent negative prognostic factor in a broad variety of cancers

Strong AXL expression correlates with poor survival rate

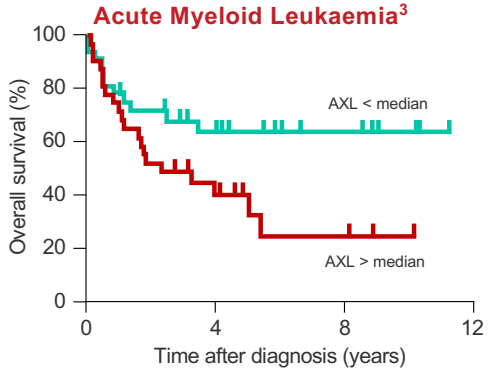
Breast carcinoma[1]



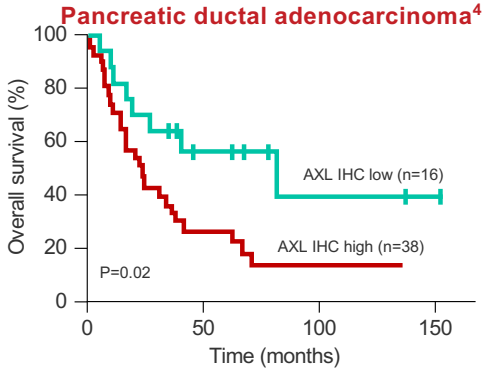
Lung adenocarcinoma (NSCLC)[2]



Acute Myeloid Leukaemia[3]



Pancreatic ductal adenocarcinoma[4]



Broad evidence of AXL linked with poor prognosis[5]

Astrocytic brain tumours	Melanoma
Breast cancer	Mesothelioma
Gallbladder cancer	NSCLC
GI	Pancreatic cancer
• Colon cancer	Sarcomas
• Oesophageal cancer	• Ewing Sarcoma
• Gastric cancer	• Kaposis sarcoma
Gynaecological	• Liposarcoma
• Ovarian cancer	• Osteosarcoma
• Uterine cancer	Skin SCC
HCC	Thyroid cancer
HNC	Urological
Haematological	• Bladder cancer
• AML	• Prostate cancer
• CLL	• RCC
• CML	

1 Gjerdrum, 2010; 2 Ishikawa, 2012; 3 Ben-Battala, 2013; 4 Song, 2010, 5 supported by > 100 publications

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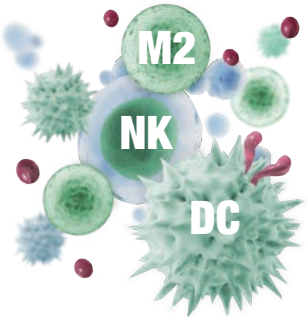
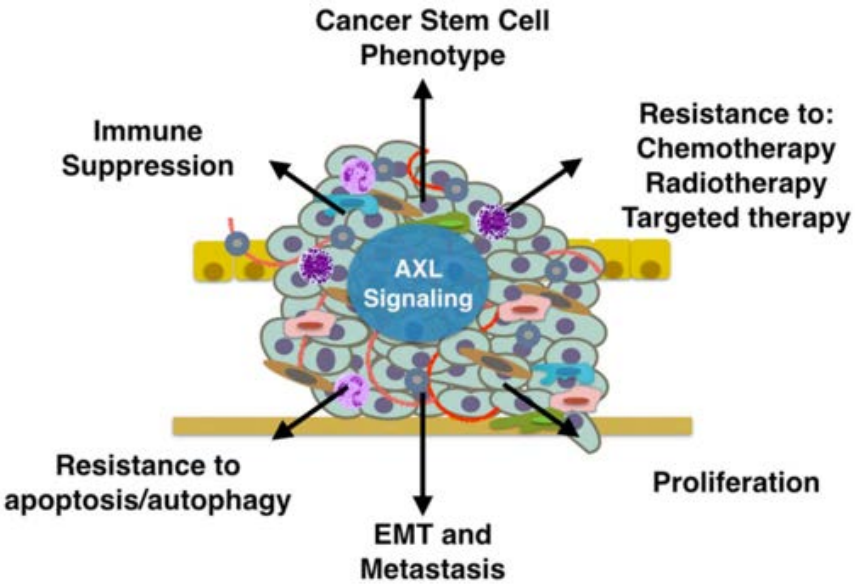
AXL is a key survival mechanism 'hijacked' by aggressive cancers and drives drug resistance, immune-suppression & metastasis

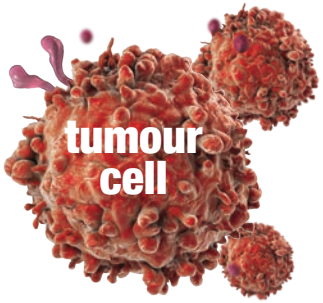
very low expression under healthy physiological conditions → overexpressed in response to hypoxia, inflammation, cellular stress & drug treatment → overexpression correlates with worse prognosis in most cancers



AXL increases on immune cells and suppresses the innate immune response

- M1 to M2 macrophage polarisation[1]
- Decreased antigen presentation by DCs[2]
- Prevent CD8+ T cell mediated cell death[3]
- Activates Treg cells





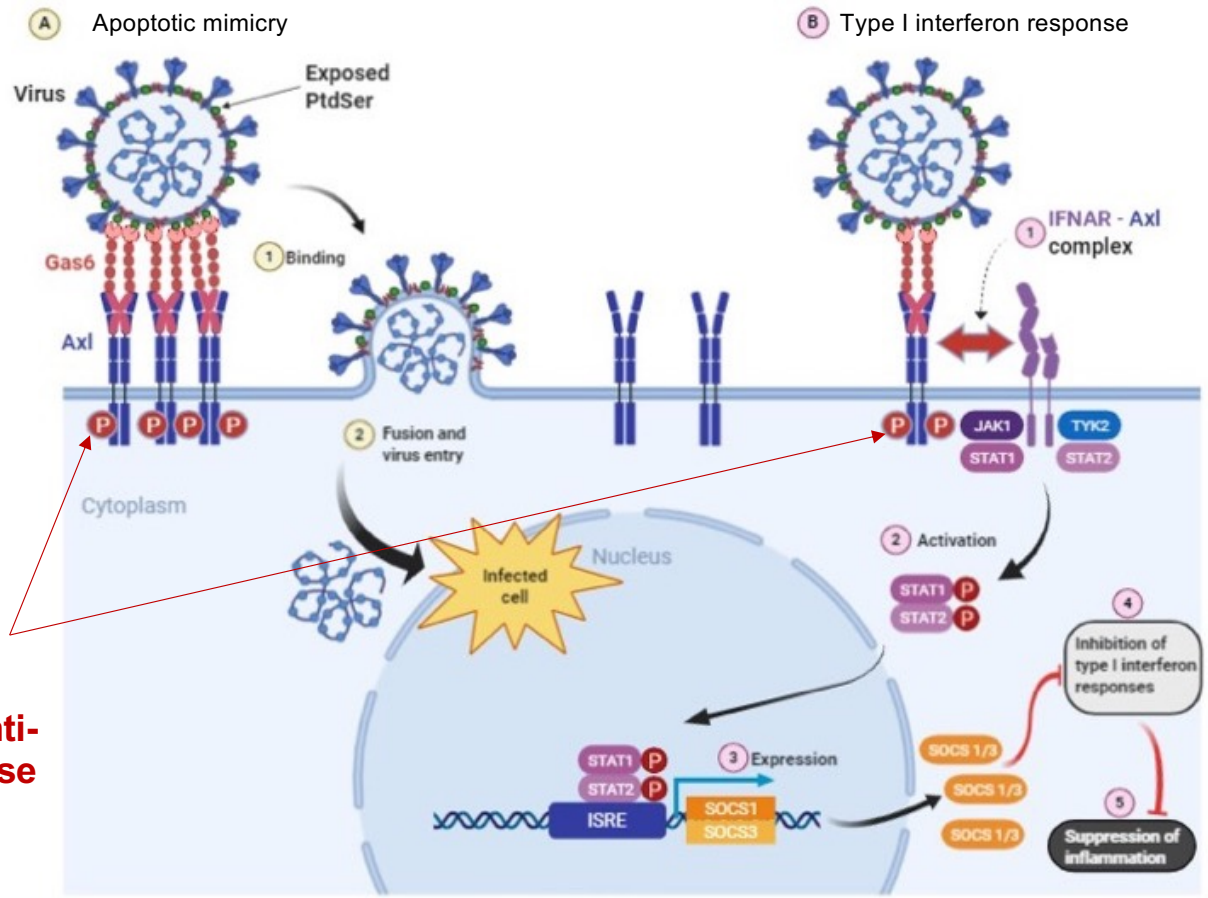
AXL increases on the tumor cell and causes cancer escape and survival

- AXL is a unique type I interferon (IFN) response checkpoint
- Acquired drug resistance
- Immune cell death resistant
- Metastasis

DC- dendritic cells Treg – Regulatory T Cell
1.Ludvig et al Can Res, 2018; Davidsen et al., submitted 2.Kurowska-Stolarska et al Nature Comm 2017; Rothlin et al Cell 2007 3.Ludvig et al Can Res, 2018; Davidsen et al., submitted

AXL is targeted by enveloped viruses to enter cells and dampen the viral immune response

Enveloped viruses display phosphatidylserine that is recognized by GAS6, the AXL receptor ligand, that mediates viral entry through “apoptotic mimicry”.



Viral-mediated AXL receptor activation dampens type I interferon responses, a key anti-viral defence mechanism for all cells

bemcentinib blocks AXL-dependent viral entry and enhances anti-viral interferon response

Muary IOWA unpublished / Meertens L *et al. Cell Host & Microbe* 2012, *12*:544 / Chen J *et al. Nat Microbiol* 2018 *3*:302

Bemcentinib

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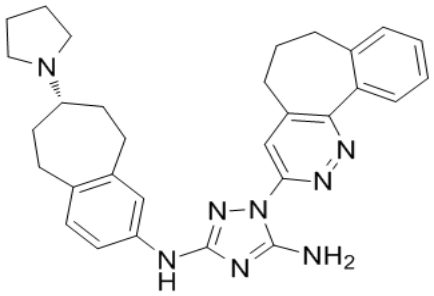
Bemcentinib, a first-in-class, potent, oral, highly selective AXL inhibitor



- ✓ IC_{50} = 14 nM
- ✓ Uniquely selective for AXL
  - ✓ 50-100 fold selective *cf.* TAM kinases

- ✓ CMC scaled for regulatory filing
- ✓ Size 0 100mg HPMC capsules
- ✓ 3 years stability confirmed

- ✓ Once daily oral dosing
- ✓ Already trailed in >300 patients
- ✓ Favourable safety profile supports use in first line, high risk fragile patients
- ✓ Safety and tolerability profile supports use in combination with other drugs
- ✓ MOA is synergistic with other therapies enhancing response
- ✓ Global regulatory exposure with Fast Track Designation by FDA
- ✓ IMP available in stock for immediate clinical trial use

BerGenBio pipeline - 3 selective AXL inhibitors in clinical development

Multiple attractive opportunities in cancer and viral infection

Candidate	Targeted Indication	Discovery / Preclinical / Phase I / Phase II / Phase III.
Bemcentinib	>2L AML	Ph II safety and POC efficacy demonstrated in 39 patient trial
Bemcentinib (combination with LDAC)	2L AML	Ph Ib Safety demonstrated, efficacy POC expansion study- 28 pts.
MERCK Bemcentinib (combination with Keytruda)	2L NSCLC. (chemo refractory)	Ph II safety and POC efficacy demonstrated in 50 patient trial, end points met
	2L NSCLC (CPI refractory)	Ph II POC study on going 29 pts – stage 1 met end point
	2L NSCLC (CPI+chemo refractory)	Ph II POC study ongoing 29 pts
Bemcentinib UK Research and Innovation	COVID19	Ph II Efficacy & Safety study ongoing 120 pts
Tilvestamab (BGB149)	TBA	Ph I Healthy volunteer study ongoing
BGB601 ADC THERAPEUTICS	Various solid tumors	Ph I safety study ongoing

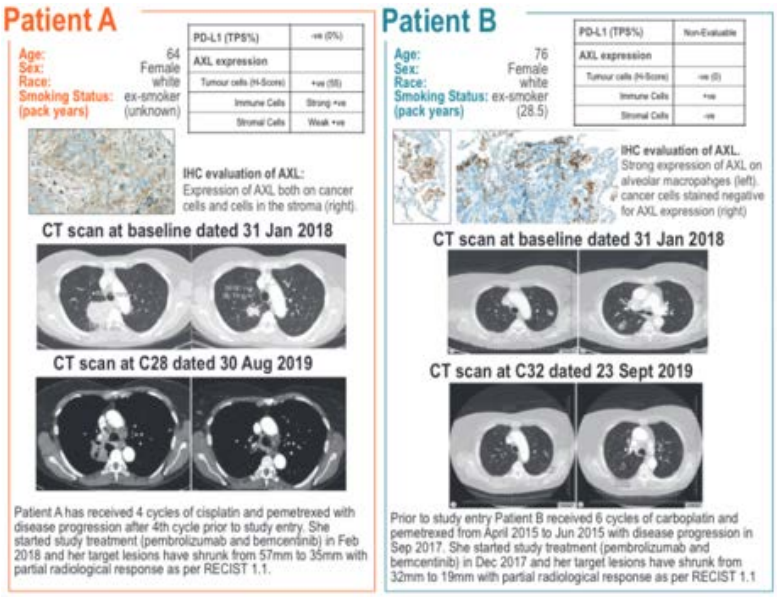
CPI – checkpoint inhibitor

Broad phase II clinical development plan with bemcentinib

		Clinical Proof-of-concept	Late stage Opportunities
Monotherapy Selected, biomarker directed patients	AML / MDS	Completed	
	Glioblastoma (IIT)	Ongoing	
	COVID19	Ongoing	
Chemotherapy Combinations Improve responses in hard to treat settings	AML + LDCT (LDAC)	Complete. -EXPANSION	
	Pancreatic, (IIT)	Ongoing	
	NSCLC (IIT)	Ongoing	
Immunotherapy Combinations Target resistance, enlarge addressable patient population	NSCLC (PD-L1 / AXL all comers)	Cohort A Complete Cohort B ongoing - EXPANSION Cohort C ongoing	
	Melanoma, (IIT)	Ongoing	
	Mesothelioma (IIT)	In set-up	
Targeted Therapy Combinations Target resistance, enlarge addressable patient population	NSCLC + EGFRi	Completed	
	Melanoma, (IIT)	Ongoing	

Companion Diagnostic (CDx)

- Developed a proprietary duplex IHC method with composite AXL tumor-immune Score (cAXL)
- A proprietary diagnostic algorithm using IHC scoring of AXL on tumor cells and on immune cells to identify solid tumour (NSCLC) patients that will respond / benefit from bemcentinib + CPI



Patient A: RESPONDER
- AXL stained +ve on tumor cells
- 61% tumor shrinkage

Patient B: RESPONDER
- AXL stained -ve on tumor cells
- AXL stained +ve on alveola macrophages
- 59% tumor shrinkage

AXL mediates aggressive cancer traits through EMT and Immune suppression in the tumour microenvironment:

Patient A: AXL +ve staining on lung tumour cells

- AXL mediated EMT in tumour cells
- AXL+ve Mesenchymal tumour cells are drug resistant & immune evasive

Patient B: AXL +ve staining on lung macrophages

- AXL is required to stabilize M2 macrophages
- M2 microphases are immune suppressive
- Bemcentinib inhibits AXL and macrophages switch to M1
- M1 macrophages are immune promoting

AXL inhibitors – emerging competitive landscape

Bemcentinib clinical development in COVID19

ACCORD-2 trial

To evaluate the efficacy and safety in hospitalized COVID19 patients

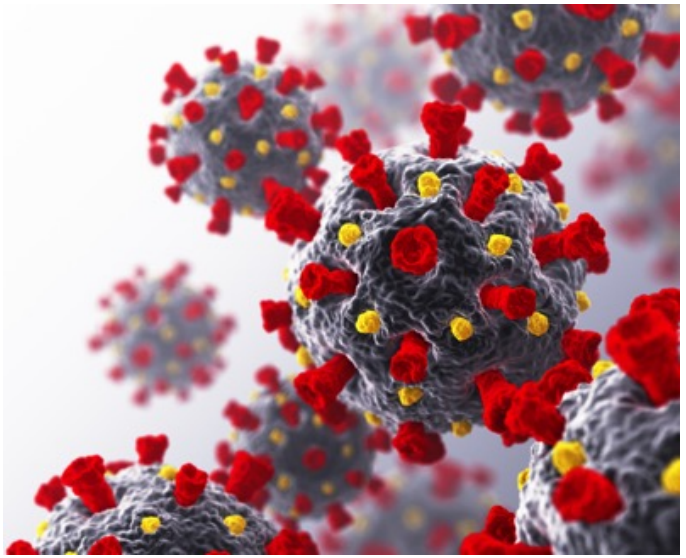
First compound selected by UK Govt. COVID19 Therapeutic Task Force

Trial funded by UK Govt.

A multicentre, randomised Phase II (120 patients) seamless Phase III transition option

BerGenBio's bemcentinib selected to be fast-tracked as a potential treatment for COVID-19

- Preclinical data suggest that bemcentinib is potentially useful for the treatment of early SARS-CoV-2 infection, as it selectively inhibits AXL kinase activity
- Bemcentinib selected as the first candidate to be fast-tracked in a new UK national multi-centre randomised Phase II clinical trial initiative to investigate potential treatments for hospitalised COVID-19 patients
- ACCORD (**AC**celerating **C**OVID-19 **R**esearch & **D**evelopment platform) study is funded by the UK Department of Health and Social Care and UK Research and Innovation
- Study is a collaboration between the UK Government Scientific Office, the NIHR's Biomedical Research centres and clinical research company IQVIA
- Professor Tom Wilkinson is the academic lead of ACCORD-2, based at the National Institute for Health Research (NIHR) Southampton Biomedical Research Centre
- The study will test 120 patients across 6 UK NHS hospital trusts, with the first patients due to be treated imminently



Protocol title: A Multicentre, Seamless, Phase 2 Adaptive Randomisation Study to Assess the Efficacy and Safety of Multiple Candidate Agents for the Treatment of COVID-19 in Hospitalised Patients

Rationale:

There are currently no approved therapeutic agents available to treat coronaviruses such as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative agent of COVID-19 disease, and there is an urgent public health need for rapid development of such interventions. This adaptive platform study is designed to rapidly assess multiple candidate agents as treatments for COVID-19. Candidate drugs that are initially assessed as being efficacious will be moved from an evaluation (pilot) stage to a confirmatory stage, with candidate agents being added to and removed from the study on an ongoing basis, depending on the results of their evaluation. Patients to be included in the study will be hospitalised and may require either supplemental oxygen, noninvasive ventilation or high flow oxygen devices, or invasive mechanical ventilation or extracorporeal membrane oxygenation (ECMO).

Objectives:

Stage 1: To evaluate the efficacy of candidate agents as add-on therapies to standard of care (SoC) in patients hospitalised with COVID-19 in a screening stage.

Stage 2: To confirm the efficacy of identified efficacious candidate agents in patients hospitalised with COVID-19 in an expansion stage.

Endpoints:

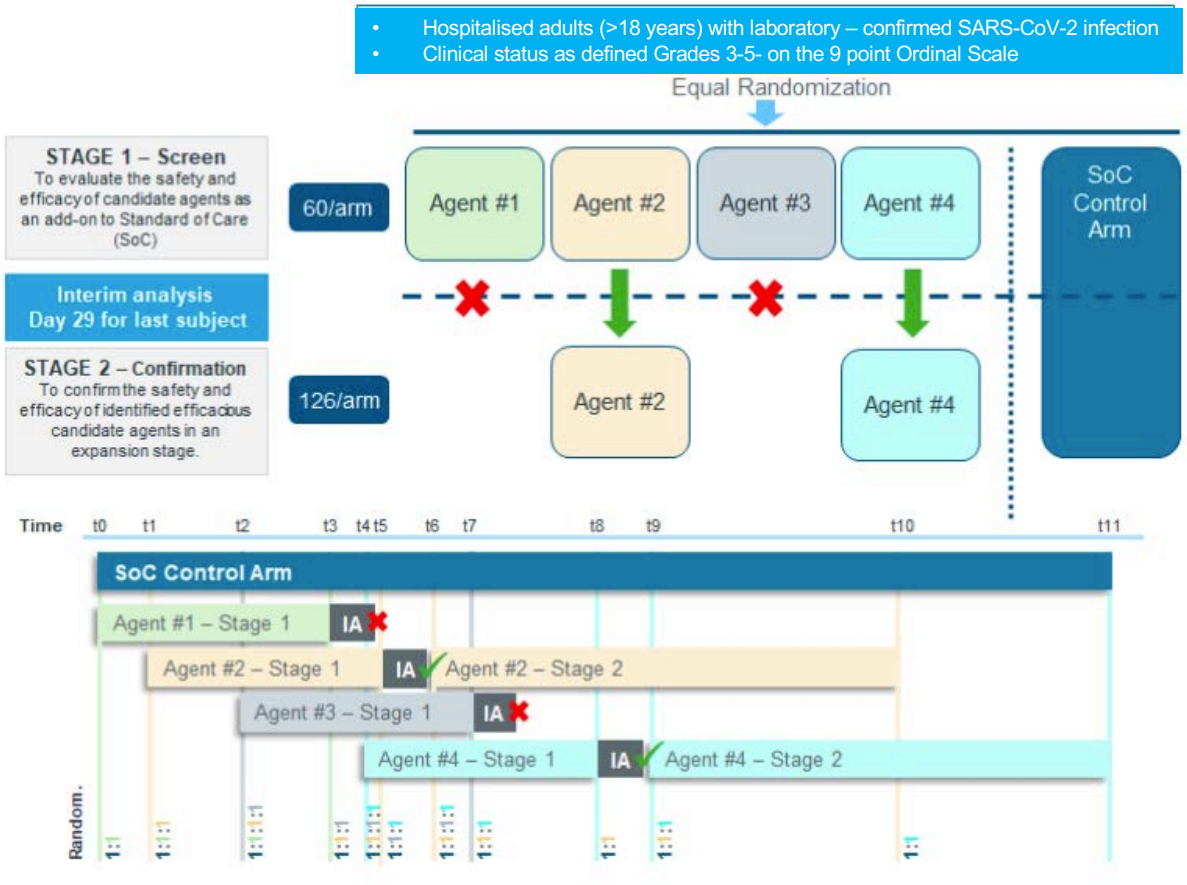
- Time to clinical improvement of at least 2 points (from randomisation) of patients stage 3, 4 or 5 on a 9-point category ordinal scale, or live discharge from the hospital, whichever comes first (this will also define the “responder” for the response rate analyses).

9-Point Category Ordinal Scale:

0. Uninfected, no clinical or virological evidence of infection
1. Ambulatory, no limitation of activities
2. Ambulatory, limitation of activities
3. Hospitalised – mild disease, no oxygen therapy
4. Hospitalised – mild disease, oxygen by mask or nasal prongs
5. Hospitalised – severe disease, noninvasive ventilation or high flow oxygen
6. Hospitalised – severe disease, intubation and mechanical ventilation
7. Hospitalised – severe disease, ventilation and additional organ support – pressors, renal replacement therapy (RRT), extracorporeal membrane oxygenation (ECMO)
8. Death

ACCORD Study overview

Figure 1 Study Schema



IA=interim analysis; SARS-CoV-2=severe acute respiratory syndrome coronavirus 2; SoC=standard of care.
Note: This figure shows a hypothetical situation, where in Stage 1 of the study there are 4 candidate agents being compared with the SoC, of which 2 candidate agents progress to Stage 2.

Study dimensions:

- 6 NHS sites across UK
- Randomized Phase II
- 120 patients (60 receive bemcentinib, 60 in SoC control group)
- IQVIA are the CRO
- Standard bemcentinib dosing
- 15 day dosing schedule
- Independent Data monitoring Committee
- Seamless transition to stage 2 (phase III)

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Ref. BGBC003 / NCT02488408

Bemcentinib clinical development in Acute Myeloid Leukemia (AML) and Myelodysplastic syndromes (MDS)

Objective: to evaluate the safety and efficacy of bemcentinib in AML and MDS

Bemcentinib monotherapy in patients relapsed AML or MDS

Bembentinib in combination with low-dose cytarabine (LDAC) in 1L newly diagnosed or relapsed patients with AML

Bembentinib in combination with LDAC in 2L relapsed patients with AML

Acute Myeloid Leukaemia (AML)

Most common type of acute leukaemia in adults[1]

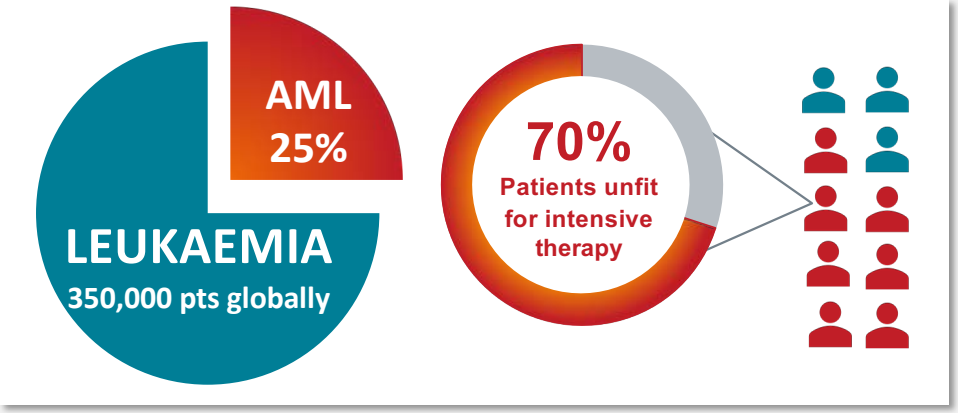
AML is a rare aggressive cancer of the blood and bone marrow characterised by difficult to treat malignancies

~ 21,000 new cases diagnosed and >11,000 deaths in the US in 2018[2]

AML makes up 32% of all adult leukaemia cases

Occurs in a predominantly elderly, frail patient population; 68% of patients diagnosed with AML were aged >60 years [6]

5 year survival rates of 3-8% in patients over 60 years old [7]



(1) Cancer.gov; (2) SEER; (3) https://www.who.int/selection_medicines/committees/expert/20/applications/AML_APL.pdf?ua=1ble
(4) https://www.cancer.net/cancer-types/leukemia-acute-myeloid-aml/statistics (5) https://www.businesswire.com/news/home/20190319005442/en/ (6) http://asheducationbook.hematologylibrary.org/content/2010/1/62.long, (7) https://www.ncbi.nlm.nih.gov/books/NBK65996/

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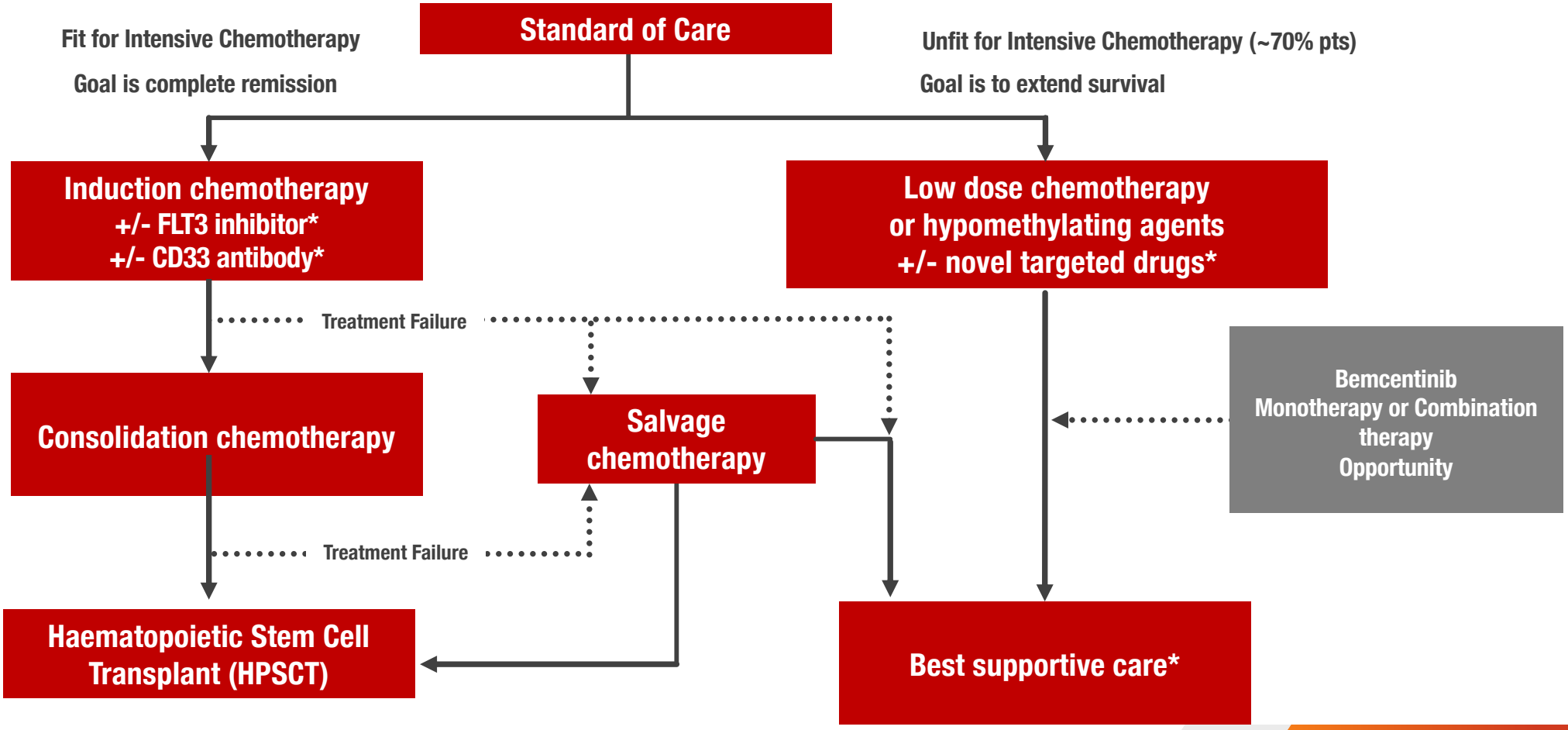
Acute Myeloid Leukaemia: Standard of Care & Bemcentinib Positioning



*A small number of patients may be eligible for targeted therapy

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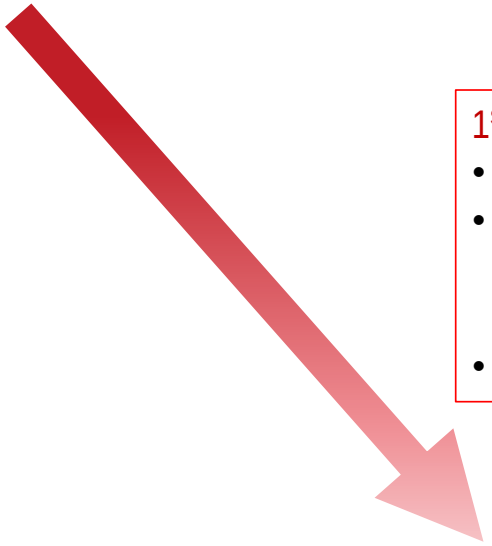
Current Approach to AML in Elderly Patients Unfit for Intensive Chemotherapy

Newly Diagnosed AML: Choice of Low Intensity Induction Therapy:

- Hypomethylating agent (HMA) +/- venetoclax (approved in US only)
- LDAC alone or in combination with venetoclax or glasdegib (approved in US only)
- Future direction: AML with mutation of FLT3, IDH1/2

Opportunity for Bemcentinib + LDAC



1st Relapse

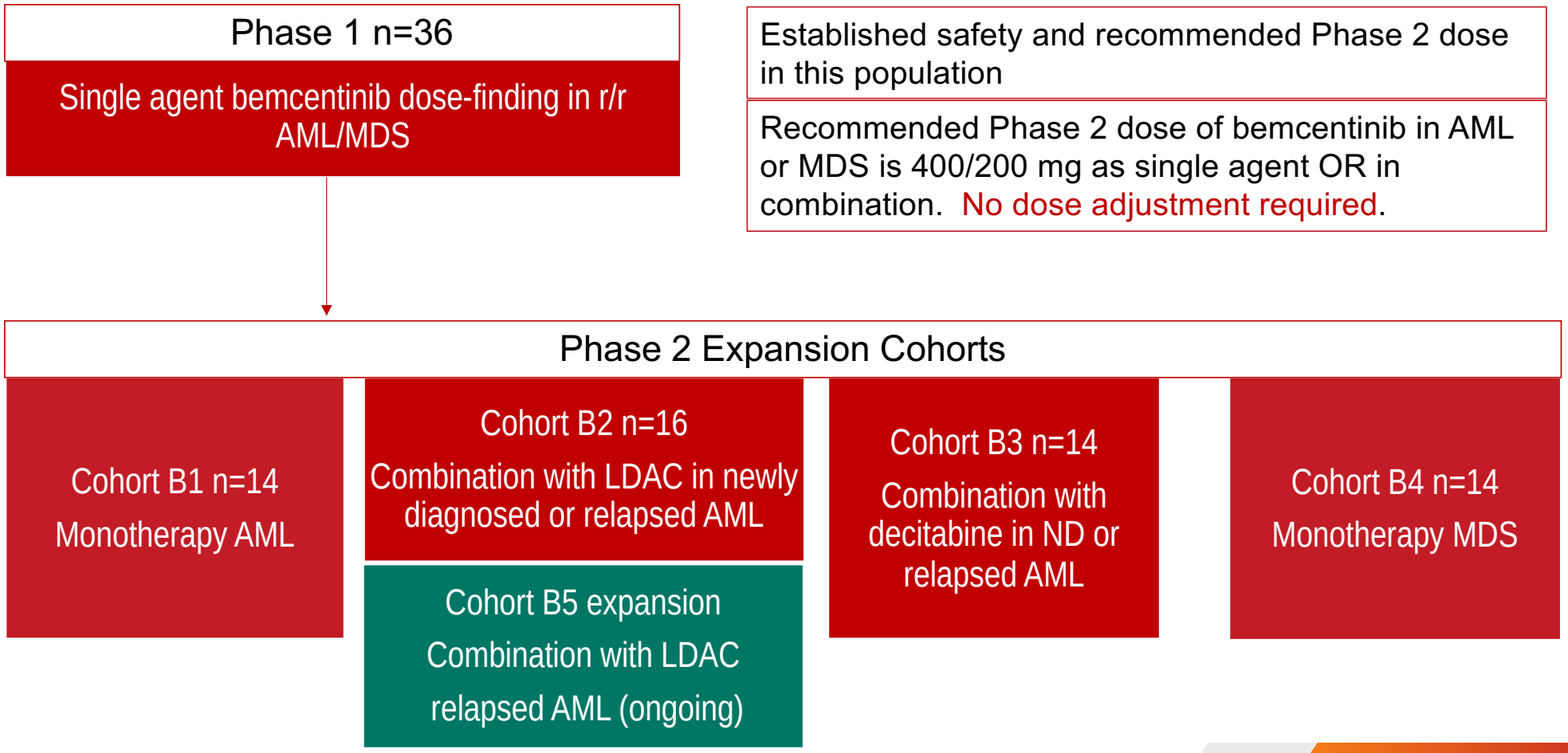
- Clinical trial
- No approved therapy, but options may include HMA, LDAC or single agent venetoclax dependent on funding
- Best supportive care (BSC) or palliative care

2nd Relapse

- Clinical trial
- BSC or palliative care

Opportunity for Single Agent Bemcentinib

Bemcentinib clinical development in Acute Myeloid Leukemia, (BGBC003)



LDAC = Low Dose Cytarabine

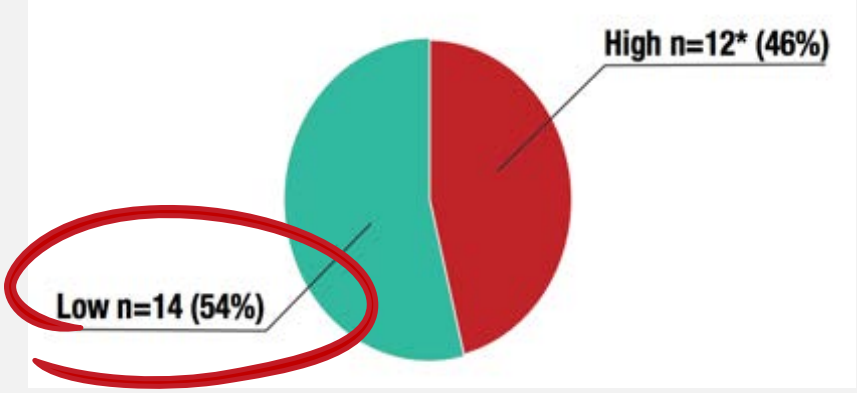
Results Bemcentinib monotherapy in relapsed/refractory ≥2L r/r AML.

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

- 2 evaluable patients were not evaluable for sAXL status
- Monotherapy responses. One additional response was reported in combination with decitabine for a total of 7 responses in phase I/II.
- 1 CR, 4 CRi, 1 CRp

* PD includes patients who progressed or came off study before having completed 3 cycles of treatment.

Biomarker: Soluble AXL (sAXL) at screen:
Inversely correlated with AXL receptor activity



≥2L Relapse patients >75yrs
No approved SoC
Bemcentinib Monotherapy

AXL +ve patients*
14/27
54%

CR/Cri/CRp
6/14
43%

Stable Disease
3/14
21%

mDOR **3.1mo. (5.5* mo.)**

Safety profile was well tolerated

* including 2 patients with low dose decitabine, one remains in CR after 20 months

Loges ASH 2018

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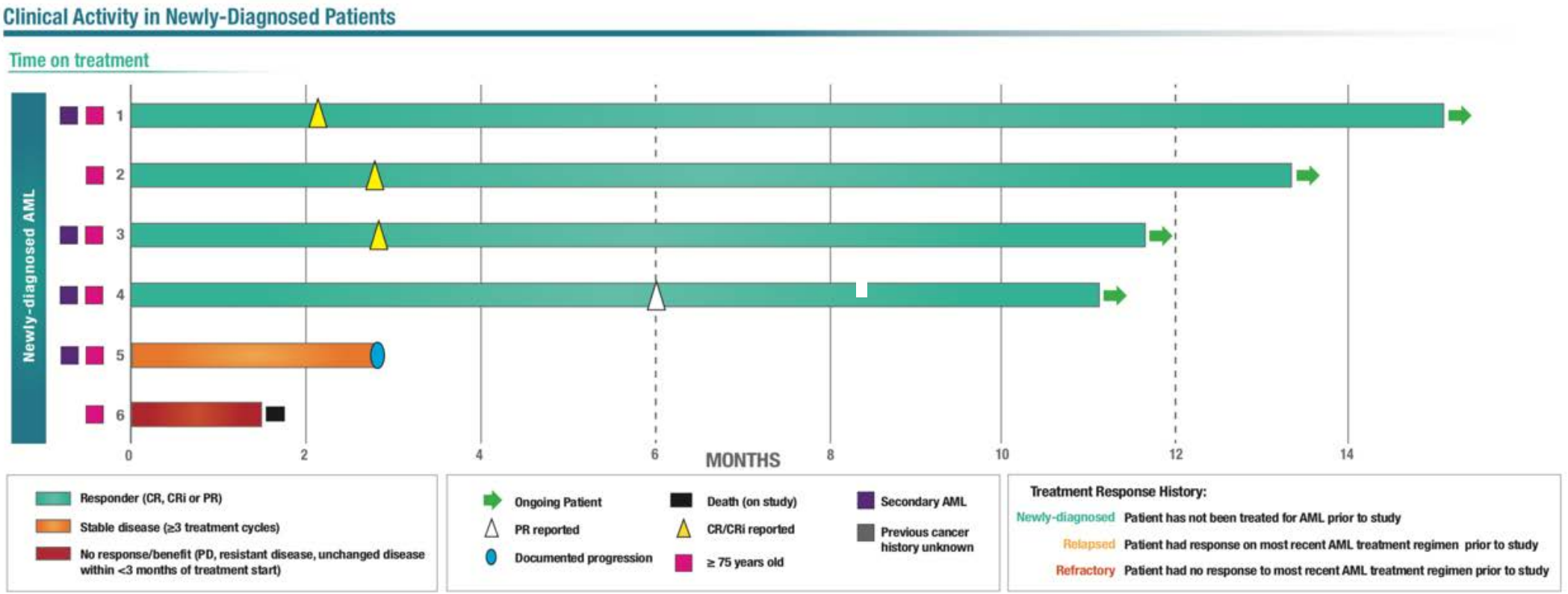
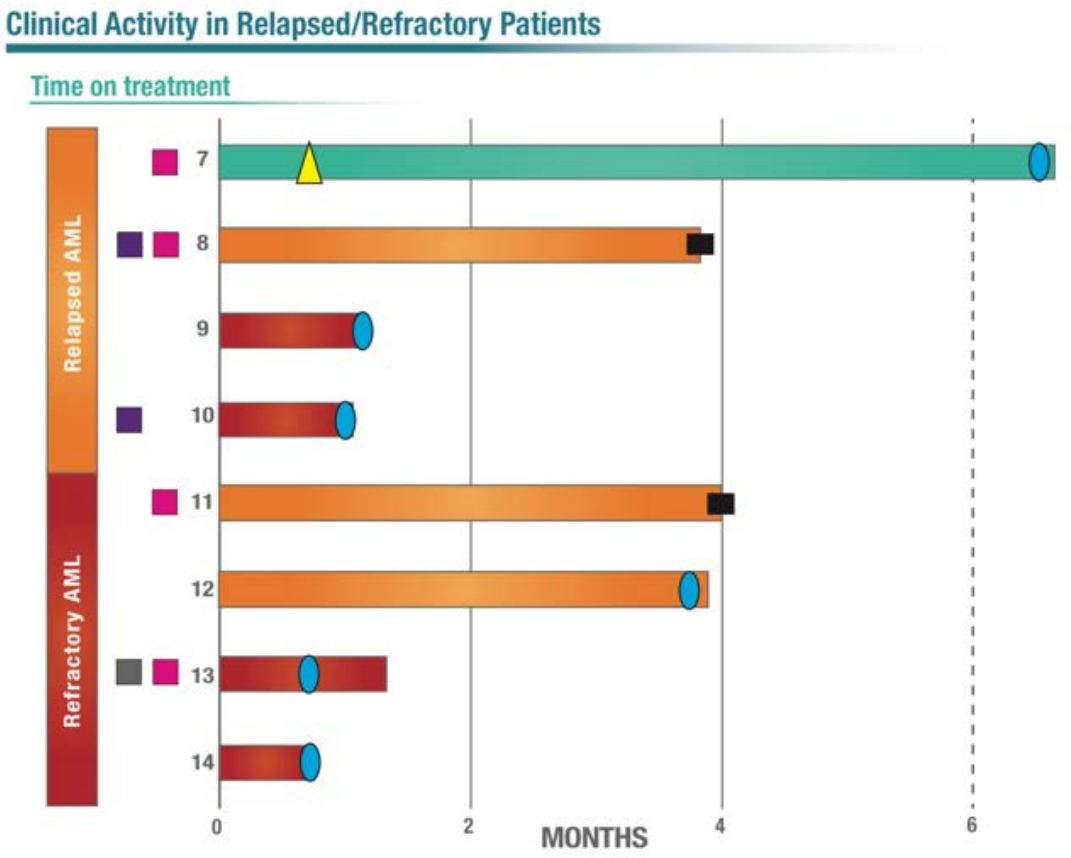
Bemcentinib + LDAC combination is active and effective in 1L newly diagnoses unfit/elderly AML patients

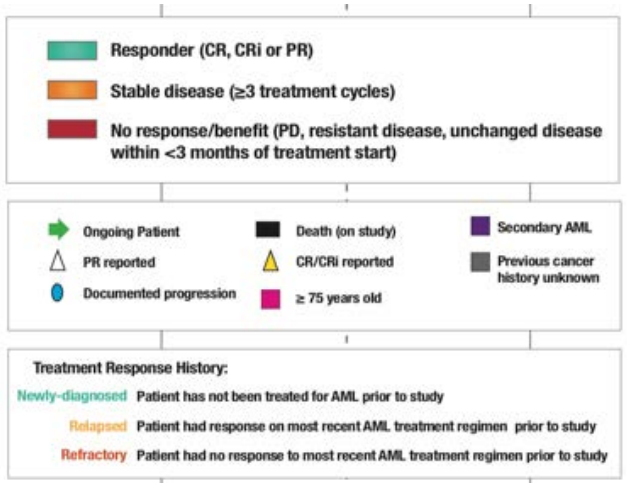
- 4/6 patients with ORR
- mDoR immature >12months and all 4 responding patients ongoing
- Responding patients have poor risk factors

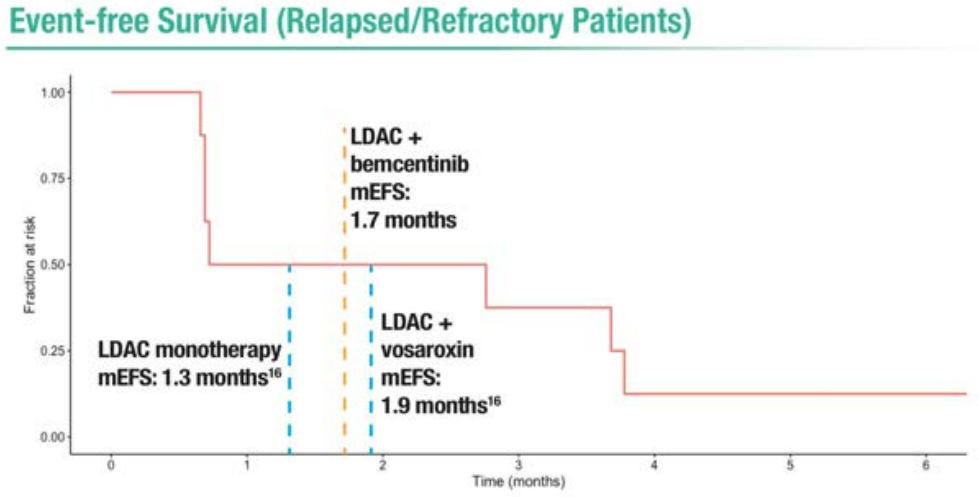


Loges et al ASH 2019

Bemcentinib + LDAC in r/r AML patients







2L r/r AML LDAC combo expansion cohort 28pts ongoing

BerGenBio

Registration strategies for bemcentinib in AML under consideration

Bemcentinib has FAST TRACK DESIGNATION by FDA in AML.

3 possible registration paths are apparent, in slightly different patient populations

Scientific advice will be sort early 2020, route to registration to be discussed

1. 2L Bemcentinib + LDAC combination

- relapse patients >60 years, patients having failed HMA or HMA+Venetoclax
- rPh II / III, to receive bem+LDAC or LDAC alone
- End points: ORR and DoR
- Anticipated sample size 200 with 6 month f/u

2. ≥2L bemcentinib mono therapy

- Heavily pre-treated, ≥2L relapse patients >75yrs, with low sAXL
- sAXL assay is a CLSI validate Clinical Trial Assay method performed at a CLIA lab.
- Possible single arm or comparator being best supportive care (BSC) or palliative care
- End points: ORR and DoR
- Anticipated sample size 100 with 6 month f/u

3. 1L Bemcentinib + LDAC combination

- 1L patients >60 yrs, unsuitable for HMA+Venetoclax
- rPh II / III
- End points: ORR and DoR/OS
- Anticipated sample size 200 with 12 month f/u

Ref. BGBC008 / NCT03184571

Bemcentinib clinical development in Non Small Cell Lung Cancer (NSCLC)

Objective: to improve the effectiveness of immune check point inhibitor (CPI) (pembrolizumab/Keytruda) refractory NSCLC patients, with a well tolerated, effective, and convenient drug

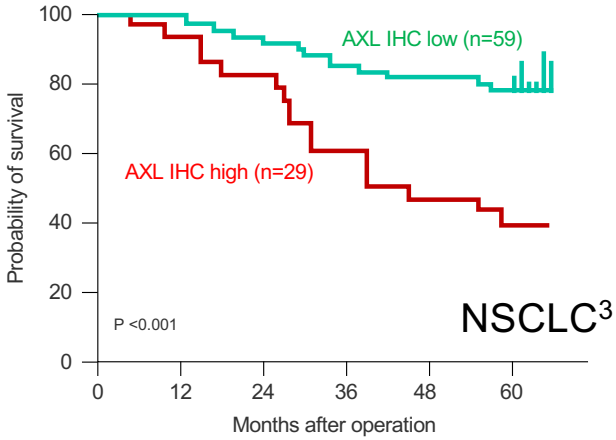
Chemotherapy refractory patients

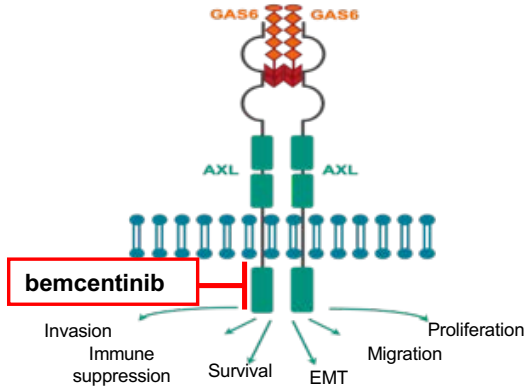
CPI +/- chemotherapy refractory patients

CPI+Chemotherapy refractory patients

Rationale for AXL inhibitor bemcentinib as an immuno-oncology agent in combination with check point inhibitor (CPI)

- AXL drives tumor EMT and resistance to cytotoxic lymphocyte-mediated cell killing[1]
- AXL receptor tyrosine kinase is a negative prognostic factor for many cancers including NSCLC[2]
- AXL expression is associated with anti-PD-1 therapy failure in melanoma patients[3]
- AXL is expressed by suppressive tumor-associated M2 macrophages and dendritic cells[4]
- Bemcentinib is a first-in-class highly selective, potent, and orally bioavailable, small molecule AXL kinase inhibitor
- Bemcentinib reverses EMT, repolarizes TAMs and potentiates efficacy of immunotherapy in murine cancer models[4]





[1]Terry, 2019; [2]Hugo, 2016; [3]Ishikawa, 2012, Davidsen,2017; [4]Ludwig, 2018, Davidsen, submitted

NSCLC causes more cancer related deaths than breast, colon, pancreas and prostate combined

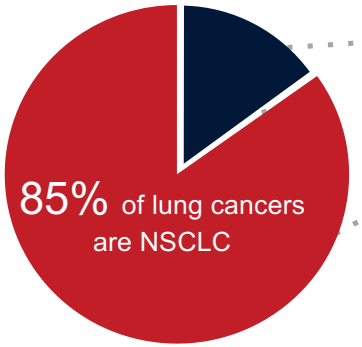
The largest cancer killer, most patients depend on drug therapy

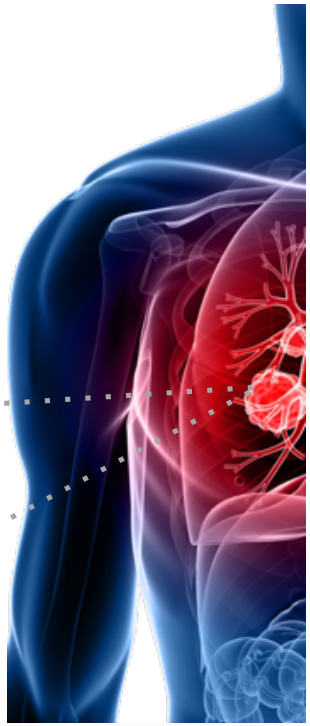
The most common type of cancer

2.09 million new cases of lung cancer diagnosed/yr worldwide, making up 11.6% of all cancer cases[1]

1.76 million lung cancer deaths/yr worldwide[1]

5-year survival rate is 3.5% in patients with PD-L1 <1%, and **12.6%** in patients PD-L1 1-49%

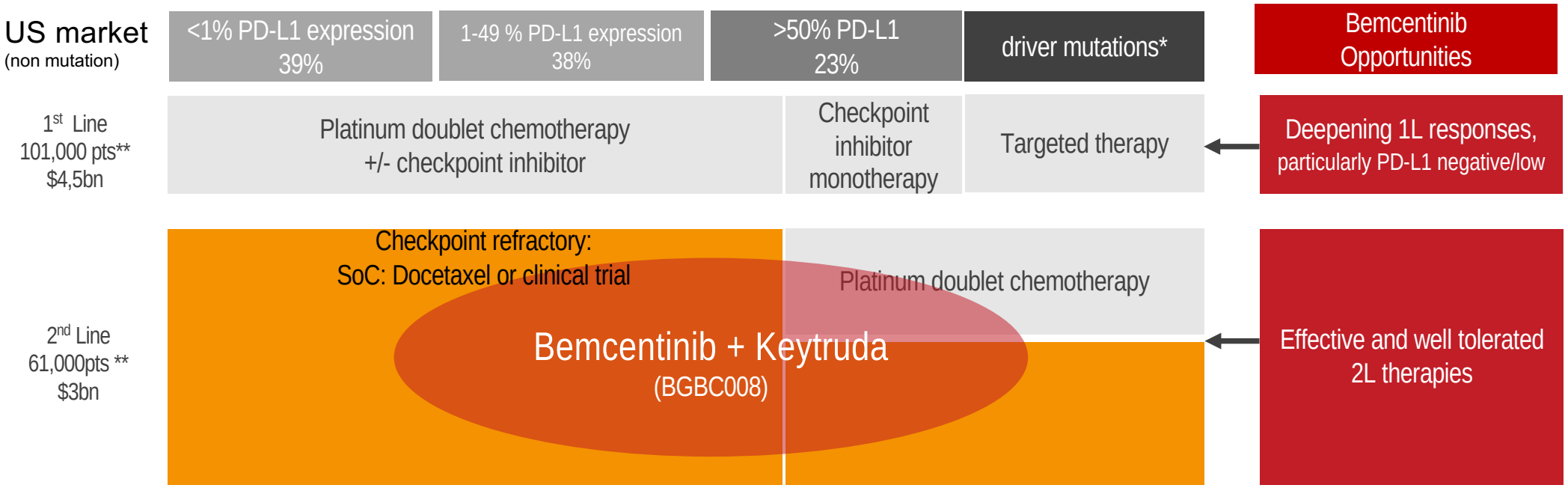




(1) fortunebusinessinsights.com/industry-reports/non-small-cell-lung-cancer-therapeutics-market-100484

Non- Small Cell Lung Cancer (NSCLC)

Rapidly evolving SoC creates opportunities for novel effective, chemo free well tolerated regimens



* Mutations / rearrangements with available targeted therapies such as EGFR and ALK
** Evaluate Pharma.USA only

BerGenBio

Bemcentinib + KEYTRUDA in refractory/relapsed NSCLC

Phase II Study Design

BGBC008
Phase II 2-stage study of bemcentinib (BGB324) in combination with pembrolizumab

Inclusion criteria
- Adenocarcinoma histology
- Measurable disease
- Fresh tumor tissue
- AXL and PD-L1 All comers

Assessments
Efficacy
- **Primary endpoint**
  - Objective Response Rate
- **Secondary endpoints**
  - Duration of Response
  - Disease Control Rate
  - Time to Progression
  - Survival at 12 months
  - Response by Biomarker expression

Safety
PK

Regimen
- Pembrolizumab 200mg fixed
- Bemcentinib 400mg loading dose, then 200mg OD

COMPLETED: INFORMS 1L OPPORTUNITY

Cohort A
- **Previously treated with a platinum containing chemotherapy**
- 2nd line advanced adeno NSCLC

Interim Analysis ✓
Stage 1
N=24 patients
(each patient has the potential for at least 24 weeks follow-up)

Stop at this stage for:
Futility (H0:15% if ≤3 responses)
Or unfavorable risk/benefit

Final Analysis ✓
Stage 2
N=50 patients total
(each patient has the potential for at least 24 weeks follow-up)

Cohort B
- **Previously treated with a checkpoint inhibitor (PD-L1 or PD-1 inhibitor)**
- No more than 2 previous lines of treatment
- Must have had disease control for ≥12 weeks followed by progression
- 2nd or 3rd line advanced adeno NSCLC

Interim Analysis ✓
Stage 1
N=13 patients/cohort
(each patient has the potential for at least 24 weeks follow-up)

Stop at this stage for
Futility (H0:15% if 0 responses)
Or unfavorable risk/benefit

Final Analysis Recruiting
Stage 2
N=29 patients/cohort
(each patient has the potential for at least 24 weeks follow-up)

Cohort C
- **Previously treated 1st line with a checkpoint inhibitor- containing regimen in combination with a platinum-containing chemotherapy**
- Disease control on 1st line therapy for ≥12 weeks followed by progression
- 2nd line advanced adeno NSCLC

Interim Analysis Recruiting
Stage 1
N=13 patients/cohort
(each patient has the potential for at least 24 weeks follow-up)

Stop at this stage for
Futility (H0:15% if 0 responses)
Or unfavorable risk/benefit

Final Analysis
Stage 2
N=29 patients/cohort
(each patient has the potential for at least 24 weeks follow-up)

ONGOING WILL INFORM 2L PIVOTAL STUDY

Ref. BGBC008 / NCT03184571 – clinical trial collaboration with Merck & Co., Inc.

Cohort A Patient Disposition and Demographics*

Patient disposition	N
Screened	74
Enrolled	50
Evaluable	44
Ongoing	9

Patient demographics		N (%)
Age	Median	65
	Range	39-82
ECOG at screen	0	22 (44%)
	1	28 (56%)
Sex	Female	20 (40%)
Smoking Status	Smoker	10 (20%)
	Ex-smoker	29 (58%)
	Never smoked	10 (20%)
	Unknown	1 (2%)

Disease mutations	N (=50)
None	36 (72)
KRAS	7 (14)
TP53	2 (4)
EGFR	3 (6)
Other	4 (8)

Safety Summary

The safety profile of combination treatment is consistent with that of each individual drug

Treatment related adverse events were generally mild and reversible

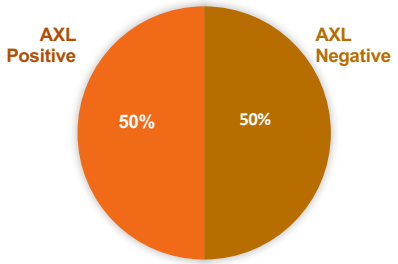
Treatment related adverse events were considered to be less severe and better tolerated than for other TKIs or CPI combinations used in NSCLC

Most frequent TRAEs (≥10% dosed pts)

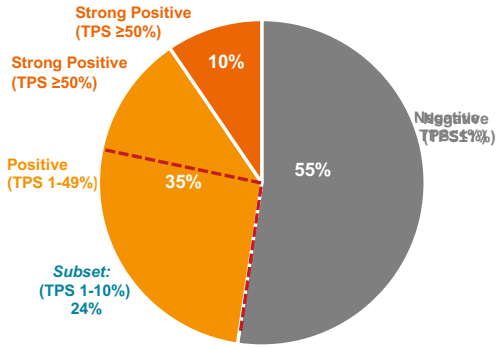
Event Terms	All Grades		Grade≥3	
	n	%	n	%
Transaminase increased*	19	38 %	7	14%
Asthenia / Fatigue	15	30 %	4	8%
Diarrhoea	12	24 %	0	0%
Nausea	7	14 %	0	0%
Anaemia	6	12 %	1	2%
Blood creatinine increased	6	12 %	0	0%
Decreased appetite	6	12 %	0	0%
Pruritus	5	10 %	0	0%

Biomarker

cAXL status
n = 30



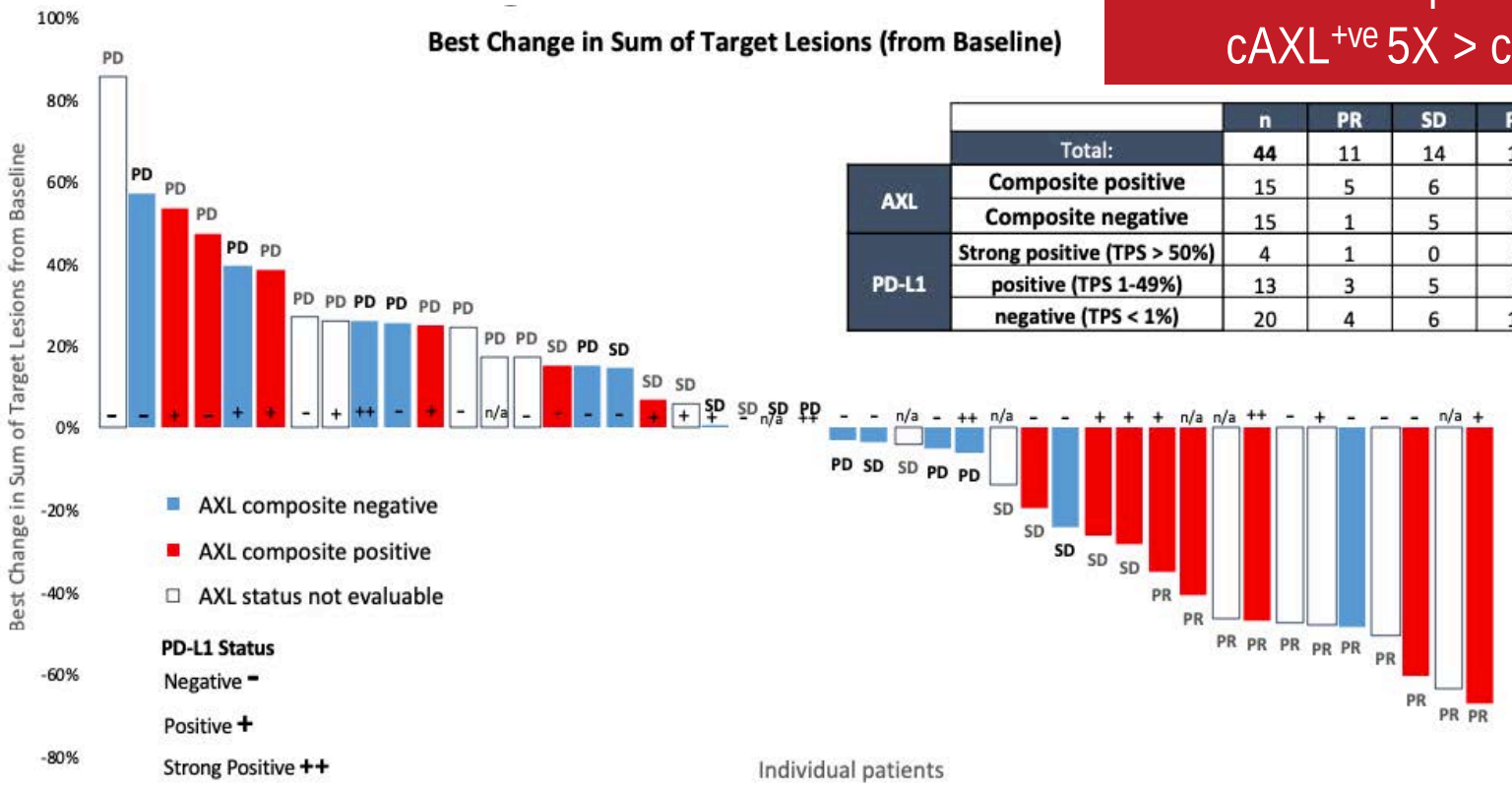
PD-L1 status
n = 37



*Data cutoff (30 Sep 2019)

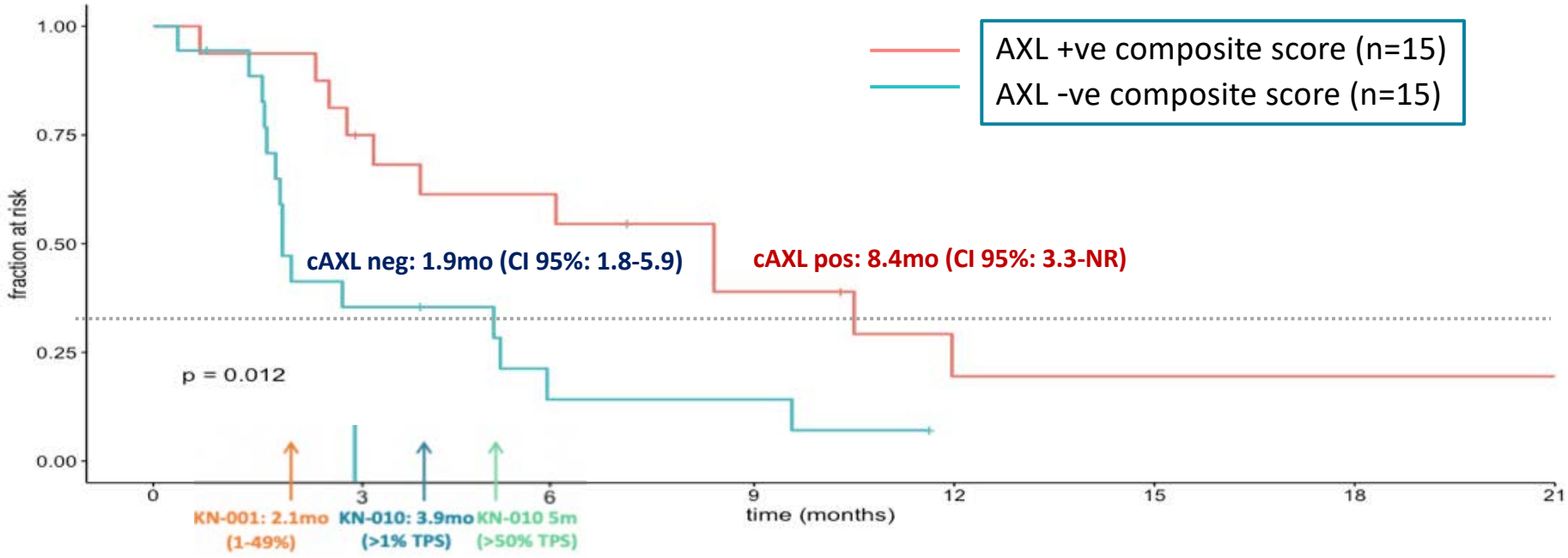
Cohort A: Anti-tumor activity of bemcentinib in combination with pembrolizumab
Change in tumour size from baseline by RECIST 1.1

Primary endpoint met:
Overal Response Rate
cAXL^{+ve} 5X > cAXL^{-ve}



		n	PR	SD	PD	ORR%	DCR%
	Total:	44	11	14	19	25	57
AXL	Composite positive	15	5	6	4	33	73
	Composite negative	15	1	5	9	7	40
PD-L1	Strong positive (TPS > 50%)	4	1	0	3	25	25
	positive (TPS 1-49%)	13	3	5	5	23	62
	negative (TPS < 1%)	20	4	6	10	20	50

Cohort A: >4 X improvement in mPFS* incomposite AXL positive patients

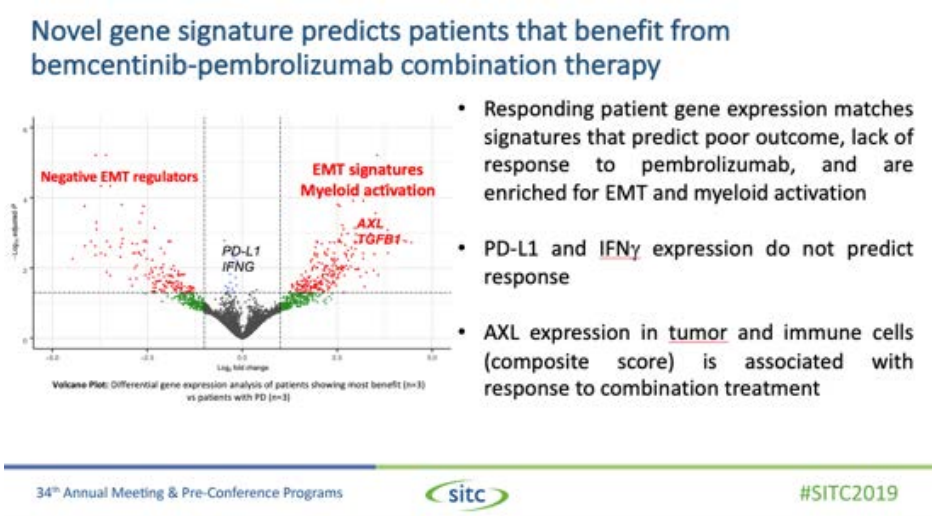


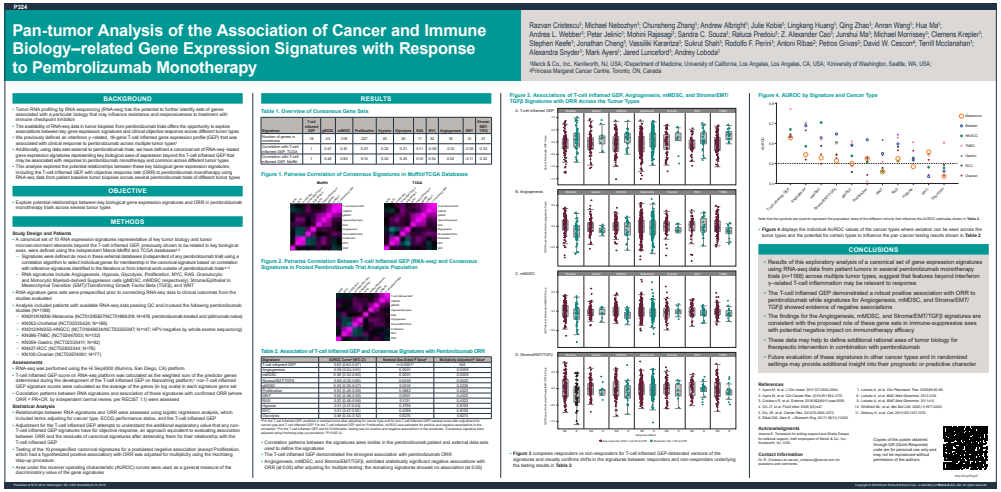
- ✓ 4-fold improvement in cAXL +ve vs. cAXL –ve patients.
- ✓ 4-fold improvement in what might be expected in the same patient population with Keytruda monotherapy

Cohort A results update : January 20, 2020
Source: KN001; Garon *et al* NEJM 2015; KN-010 Herbst *et al*, Lancet 2016 *Progression-free survival

BerGenBio's proprietary novel gene signature predicts patients that benefit from bemcentinib - pembrolizumab combination therapy

SITC 2019: BerGenBio & Merck independently published related gene signatures that predict response or resistance to pembrolizumab





Merck reported a gene signature from patients that did not respond to Keytruda monotherapy in many cancers, this was similar to the BerGenBio gene signature EXCEPT these patients did respond to Keytruda + bemcentinib

Cohort B: Bemcentinib + KEYTRUDA in CPI refractory patients

CHECK POINT INHIBITOR REFRACTORY PATIENTS: precise and specific definition

Patients must have reported an initial clinical benefit (CR, PR or SD) for at least 12 weeks and subsequently progressed on treatment with an anti-PD1/L1 monoclonal antibody (mAb) administered either as monotherapy, or in combination with other checkpoint inhibitors or other therapies. PD-1 treatment progression is defined by meeting all of the following criteria:

a) Has received at least 2 doses of an approved anti-PD-1/L1 mAb.

b) Has demonstrated disease progression after PD-1/L1 as defined by RECIST v1.1. The initial evidence of disease progression (PD) is to be confirmed by a second assessment no less than four weeks from the date of the first documented PD, in the absence of rapid clinical progression.

c) Progressive disease has been documented within 12 weeks from the last dose of anti-PD-1/L1 mAb. Seymour et al; iRECIST: Guidelines for response criteria for use in trials testing immunotherapeutics. Lancet Oncol 18: e143-52

This determination is made by the investigator. Once PD is confirmed, the initial date of PD documentation will be considered the date of disease progression.

a) Other therapies not to be administered between last dose of anti PD-1/L1 mAb and commence of clinical trial agent

Jan 2020

Interim Analysis Cohort B
Stage 1 (N=13 patients)
CONFIRM PROGRESSION TO STAGE 2

- Recruitment continues to enroll additional 16 patients
- Protocol amended to limit to 2L patients

Development strategy for Bemcentinib in NSCLC (ad. & Sc.)

Clinical Position	Patient Population	Concept	Development Plan – target conditional approval / BT
2L IO(+chemo) refractory	Stage III/IV Ad. PD-L1 all comer cAXL +ve.	Randomised Phase IIb / III Bemcentinib + CPI vs. docetaxol 1º endpoints: Interim mPFS, (for C/A A) 6 & 12mn OS, OS (for full approval) 2º endpoints: ORR, DoR, Safety, tolerability.	1. Pending BGBC008 cohort B + C 2. SA advice from FDA & EMA 3. cAXL assay validation in BGBC008 B&C
1L	TBA		

BerGenBio

BGB149

anti-AXL monoclonal antibody

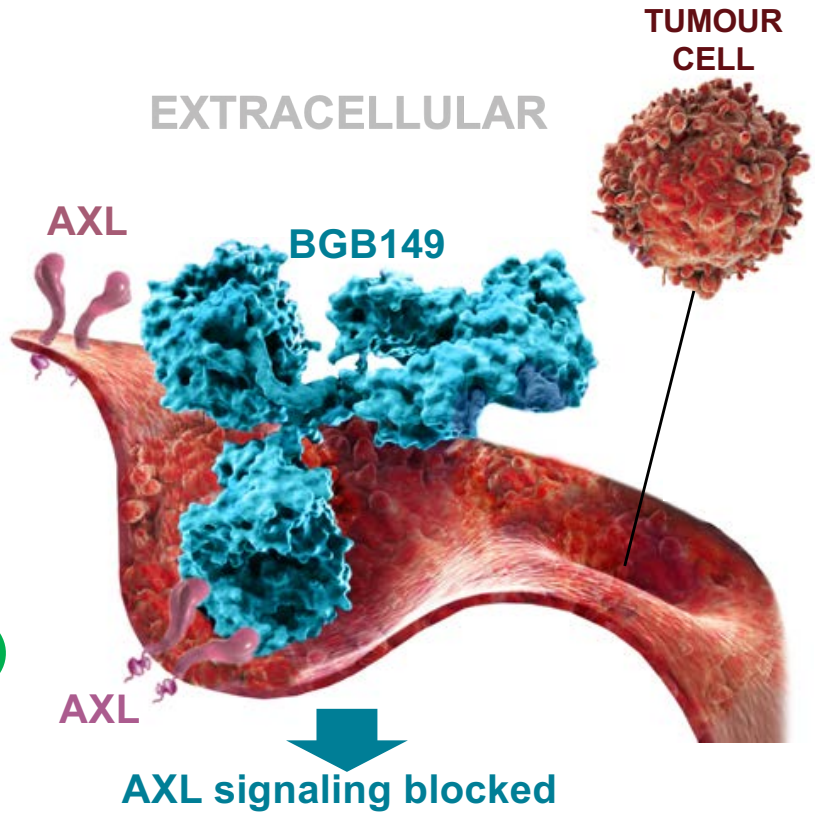
Ref. BGB149-101 / NCT03795142

BGB149: Anti-AXL monoclonal antibody

Phase I clinical trial ongoing

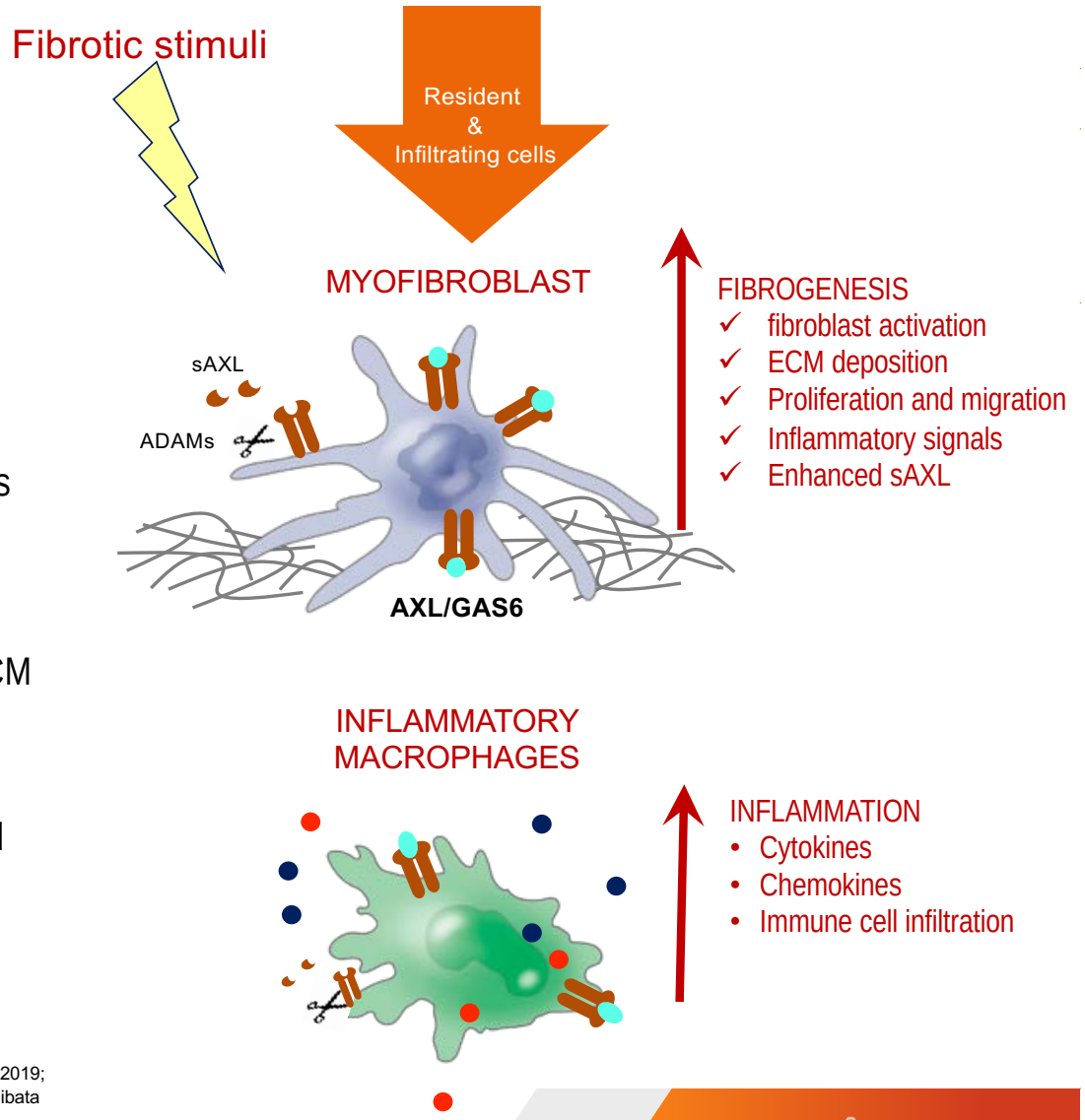
- Functional blocking fully-humanised IgG1 monoclonal antibody
- Binds human AXL, blocks AXL signalling
- High affinity (KD: 500pM), Anti-tumour efficacy demonstrated *in vivo*
- Robust manufacturing process established, 18 months stability
- Phase Ia healthy volunteer SAD study complete

  Safety – no dose limiting toxicity seen up to 3mg/kg dose
  Pharmacokinetics - exposure predictable with dose proportional Cmax increase
  Confirmatory evidence of *in* vivo target engagement with sAXL -- stabilisation in circulation
- Phase I safety trial ongoing



The role of AXL in fibrosis

- AXL Regulates and modulates key fibrogenic pathways
  - TGFb signaling[1,2]
  - Mechanosensing Hippo pathway[3]
  - Peroxisome proliferator-activated receptor[4]
- Axl regulates cellular plasticity implicated in fibrotic pathologies e.g. EMT, EndMT, Macrophage polarity
- AXL is a negative regulator of epithelial cell barrier integrity[5]
- Axl is required for hepatic stellate cell (HSC) activation and ECM deposition[6]
- Pharmacological modulation of Axl inhibits pre-clinical development of Liver ($CCl4_6$/$HighFatDiet_7$), Renal (UUO_8) and Pulmonary (Asthma[9], Bleo[10], IPF[10]) fibrosis

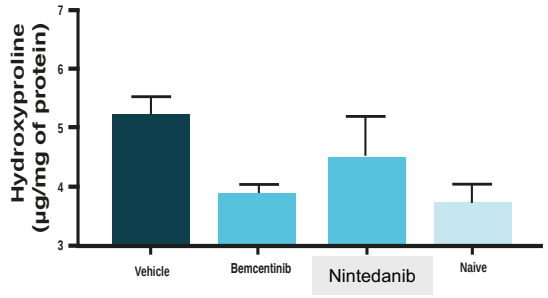


1 Gilbane ART 2015; 2 Reichl Hep. 2015; 3 Gibault ChemMed 2017; 4 Zhu AJTR 2016; Fuijno Lab invest 2017, J Exp Med 2019; 6 Barcena J. Hep 2015; 7 Tutusaus A. Cell Mol Gastroenterol 2019 Hepatol. 2019; 8 Landolt L. Physiol Reports 2019; 9 Shibata J Immunology 2014; 10 BerGenBio ASA, unpublilshed; 11 Espindola MS. Am J Respir Crit Care Med 2018)

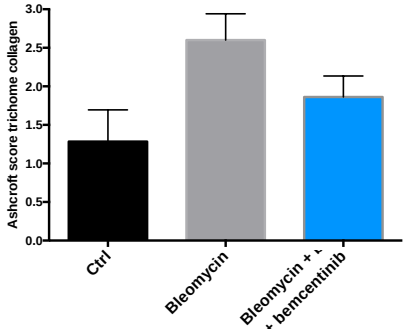
AXL inhibition prevents fibrosis in a panel of pre-clincial models

Lung

Bemcentinib reduces fibrosis in a human xenograft model of IPF [1]

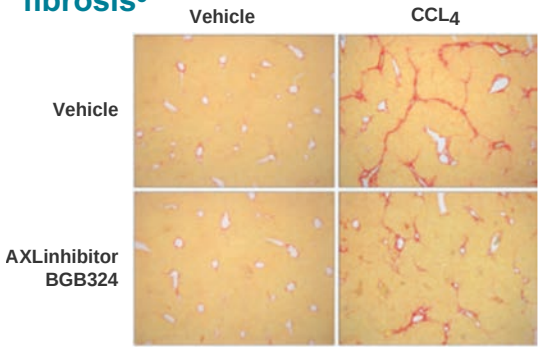


Bemcentinib reduces bleomycin induced fibrosis[2]

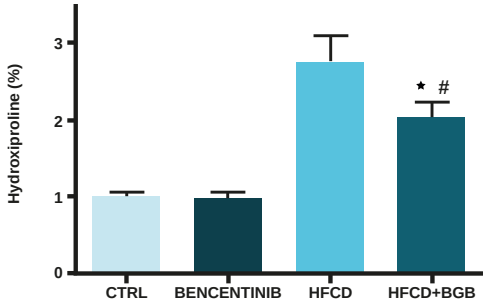


Liver

Bemcentinib reduces fibrosis in the CCL_4-induced model of liver fibrosis[3]



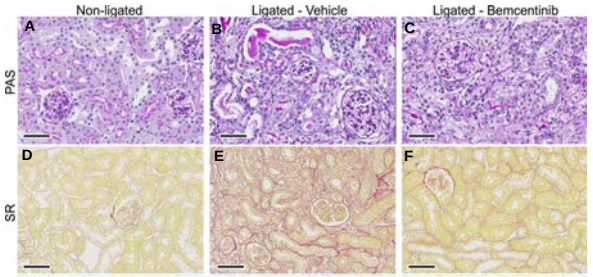
Bemcentinib reduces fibrosis in a diet induced model of NASH[4]



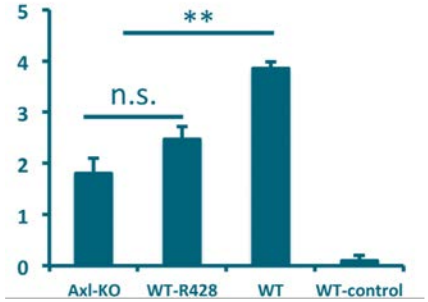
HFCD = high-fat, choline deficient diet
Leads to NASH in animal models

Kidney

Bemcentinib reduces kidney fibrosis following Unilateral Ureteral Obstruction (UUO) [5]



Bemcentinib ameliorates anti-GBM induced lupus like nephritis and improved kidney function [6]



1 Espindola et al., 2018; 2 BerGenBio ASA; 3 Barcena et al., 2015; 4 Tutsaus et al., unpublished; 5 Landolt et al., 2019 6 Zhen et al., 2018

Corporate

BerGenBio pipeline – near term news flow

Candidate	Targeted Indication	Discovery / Preclinical / Phase I / Phase II	Next expected news
Bemcentinib	>2L AML	Ph II safety and POC efficacy demonstrated in 39 patient trial	
Bemcentinib (combination with LDAC)	2L AML	Ph Ib Safety demonstrated, efficacy POC expansion study- 28 pts.	ASH 2020 Update clinical & translational data
Bemcentinib (combination with Keytruda)	2L NSCLC. (chemo refractory)	Ph II POC efficacy demonstrated in 50 patient trial, end points met	SITC 2020 Updated mOS
	2L NSCLC (CPI refractory)	Ph II POC study ongoing 29 pts – stage 1 met end point	SITC 2020 Stage 1 interim clinical and translational data
	2L NSCLC (CPI+chemo refractory)	Ph II POC study ongoing 29 pts	SITC 2020 Stage 1 interim clinical and translational data / WCLCL 2021
Bemcentinib	COVID19	Ph II Efficacy & Safety study ongoing 120 pts	Q2/3 2020 Top line clinical data
Tilvestamab (BGB149)	TBA	Ph I Healthy volunteer study ongoing	Q4 2020 Top line clinical data
BGB601	Various solid tumors	Ph I safety study Terminated (change in clinical plan and drug supply)	*Update by collaborators*

CPI – checkpoint inhibitor
mOS – median overall survival

ASH – American Society of Heamatology (Dec 5-8)
SITC – Society of Immunotherapy of Cancer (Nov10-15)
WCLC – World Congress of Lung Cancer (Jan 26-29 2021)

Select Company Financials

Oslo Børs	BGBIO
Cash (YE'19 + Q1'20 - PIPE)	$45m
Shares Outstanding	73,3m

Board of Directors

Sveinung Hole, Chairman of the board

- Non-Executive director of BerGenBio since 2010, chairman from 2019.
- Master of International Management.
- Representative of lead shareholder.

Prof. Stener Kvinnsland, MD.PhD Non-Executive Director

- Non-Executive director of BerGenBio since 2015
- More than 30 years of experience in oncology, Chair Oslo University Hospital, CEO of the Bergen Hospital Trust, Head of the Department of Oncology and Professor of Medicine (Oncology) at the University of Bergen and Director Clinical R&D, Oncology for Pharmacia & Upjohn in Milan.

Dr. Debra Barker MD, Non-Executive Director

- Non-Executive director of BerGenBio since 2019.
- Diploma in Pharmaceutical Medicine and MSc in immunology.
- Executive experience with Novartis, Roche, Smithkline Beecham and Knoll and served until recently as the Chief Medical and Development Officer at Polyphor Ltd.

Grunde Eriksen, Non-Executive Director

- Non-Executive director of BerGenBio since 2019.
- Experienced capital markets advisor and investor.
- 18 years international experience in corporate finance and equity sales with SEB & Arctic Securities

Dr. Pamela Trail, Noe-Executive Director

- Non executive director of BerGenBio since 2019.
- PhD from the University of Connecticut.
- Strategic oncology leadership roles at Regeneron, MedImmune, Bayer Healthcare and BMS and served as CSO at Seattle Genetics

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Link to reports from Trinity Delta:
https://www.bergenbio.com/investors/analyst-coverage/

Background published data on AXL role in viral infection

AXL mediates viral entry through “apoptotic mimicry” and suppresses the anti-viral type I interferon (IFN) response

AXL promotes the infection of a wide range of enveloped viruses including pox-, retro-, flavi-, arena-, filo-, and alpha-viruses (Shimojima 2006, Brindley 2008, Meertens 2012, Dowall 2016, Meertens 2017).

Viral particle binding via GAS6-AXL potently activates signal transduction through its tyrosine kinase domain to suppress type I interferon (IFN) signaling and facilitate viral replication (Bhattacharyya 2013, Meertens 2017).

AXL increases viral infection through two mechanisms:

1) enhanced viral entry through “apoptotic mimicry”; and
2) suppression of anti-viral type I interferon (IFN) responses

AXL mediates viral entry through “apoptotic mimicry” and suppresses the anti-viral type I interferon (IFN) response

AXL signaling suppresses viral-induced IFN responses via SOCS1/3, leading to increased viral replication in infected cells and decreased anti-viral defenses of neighboring cells (Huang 2015, Chen 2018, Strange 2019).

Therapeutic AXL receptor inhibition ameliorated pulmonary pathology resulting from primary viral infection in experimental models, indicating an important role for AXL within the lung (Shibata 2014).

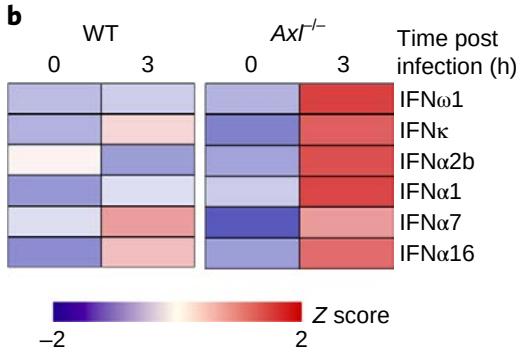
> During primary respiratory syncytial virus (RSV) infection, AXL inhibition increased the number of IFNg–producing T cells and NK cells, suppressed RSV replication and whole lung levels of IL-4 and IL-13.
>
> The lethal effect of intrapulmonary H1N1 infection inflammation was reduced by AXL inhibition. AXL inhibition in infected mice increased the number of IFN-b–producing macrophages and dendritic cells and suppressed neutrophil infiltration.

Axl-null mice are resistant to ZIKA pathogenesis likely due to a combination of reduced virus entry and enhanced IFN responses (Hastings 2019), **indicating a potential role for AXL inhibitors as therapeutics during viral infection.**

AXL is a unique type I interferon (IFN) response checkpoint

- IFNR signaling induces *AXL* expression[4,5]
- AXL is a critical negative feedback regulatory mechanism for TLR-induced type I interferon (IFN) responses in myeloid (dendritic cells, macrophage), NK and tumor cells[1-4].
- AXL is an IFN checkpoint: AXL signaling blocks IFNR signaling via SOCS1/3 and TBK1[5,6].
- AXL on dendritic cells is targeted by viruses (e.g. Zika) to abrogate IFN responses and inhibit anti-viral immunity[7].

AXL inhibition enhances type I interferon gene response to viral infection



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Bemcentinib showed effect against lethal EBOV infection in animal models conducted by PHE

viruses

MDPI

Article

Antiviral Screening of Multiple Compounds against Ebola Virus

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Abstract: In light of the recent outbreak of Ebola virus (EBOV) disease in West Africa, there have been renewed efforts to search for effective antiviral countermeasures. A range of compounds currently available with broad antimicrobial activity have been tested for activity against EBOV. Using live EBOV, eighteen candidate compounds were screened for antiviral activity in vitro. The compounds were selected on a rational basis because their mechanisms of action suggested that they had the potential to disrupt EBOV entry, replication or exit from cells or because they had displayed some antiviral activity against EBOV in previous tests. Nine compounds caused no reduction in viral replication despite cells remaining healthy, so they were excluded from further analysis (zidovudine; didanosine; stavudine; abacavir sulphate; entecavir; JB1a; Aimspro; celgosivir; and castanospermine). A second screen of the remaining compounds and the feasibility of appropriateness for in vivo testing removed six further compounds (ouabain; omeprazole; esomeprazole; Gleevec; D-LANA-14; and Tasigna). The three most promising compounds (17-DMAG; BGB324; and NCK-8) were further screened for in vivo activity in the guinea pig model of EBOV disease. Two of the compounds, BGB324 and NCK-8, showed some effect against lethal infection in vivo at the concentrations tested, which warrants further investigation. Further, these data add to the body of knowledge on the antiviral activities of multiple compounds against EBOV and indicate that the scientific community should invest more effort into the development of novel and specific antiviral compounds to treat Ebola virus disease.

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