



BerGenBio

Axl inhibitors for aggressive disease

SEB Annual Pharma & Biotech Seminar

22 January 2020

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BerGenBio corporate over view



World leaders in understanding AXL biology

AXL tyrosine kinase is a novel drug target that mediates immune evasion, therapy resistance & metastasis

AXL mediates EMT, stabilises M2 macrophages, immune suppressive dendritic cells and blocks T-cell & NK cell activity

AXL inhibitors – potential cornerstone of cancer therapy

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 clinical development focus
AML (monotherapy), **AML** (chemo-combo)
NSCLC (KEYTRUDA combo)



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 drives aggressive cancer

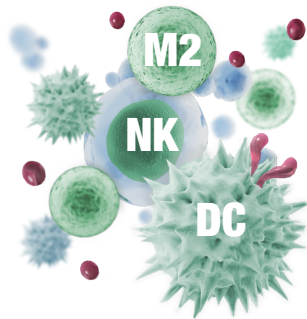


AXL is a key survival mechanism 'hijacked' by aggressive cancers and drives drug resistance, immune-suppression & metastasis

very low expression under healthy physiological conditions

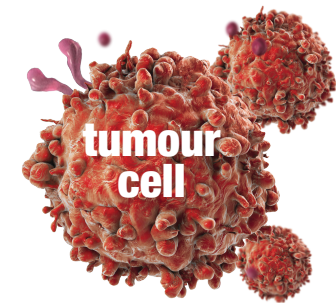
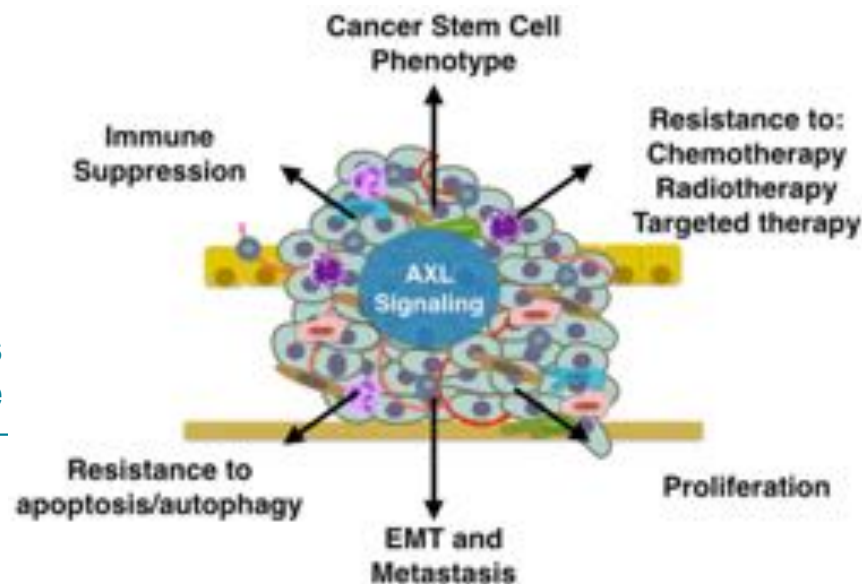
overexpressed in response to hypoxia, inflammation, cellular stress & drug treatment

overexpression correlates with worse prognosis in most cancers



AXL increases on immune cells and suppresses the innate immune response

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

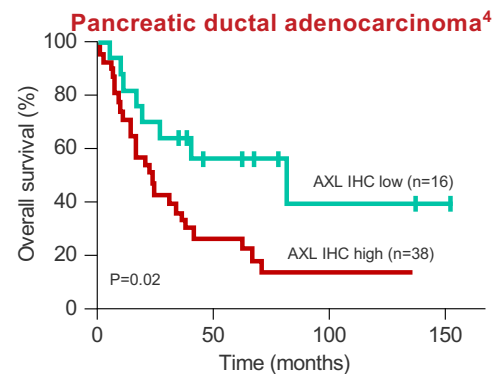
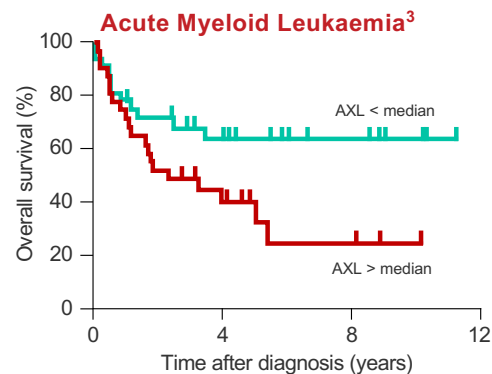
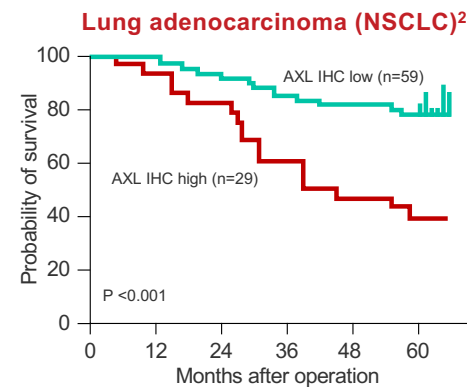
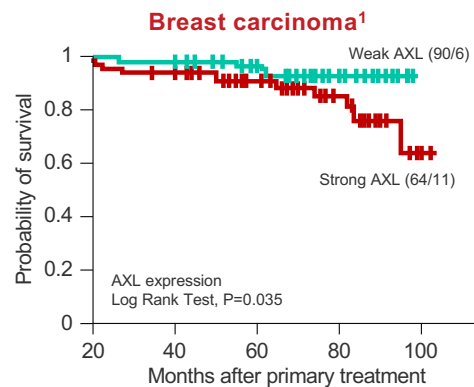


AXL increases on the tumor cell and causes cancer escape and survival

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

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

Strong AXL expression correlates with poor survival rate



Broad evidence of AXL linked with poor prognosis⁵

Astrocytic brain tumours

Breast cancer

Gallbladder cancer

GI

- Colon cancer

- Oesophageal cancer

- Gastric cancer

Gynaecological

- Ovarian cancer

- Uterine cancer

HCC

HNC

Haematological

- AML

- CLL

- CML

Melanoma

Mesothelioma

NSCLC

Pancreatic cancer

Sarcomas

- Ewing Sarcoma

- Kaposi sarcoma

- Liposarcoma

- Osteosarcoma

Skin SCC

Thyroid cancer

Urological

- Bladder cancer

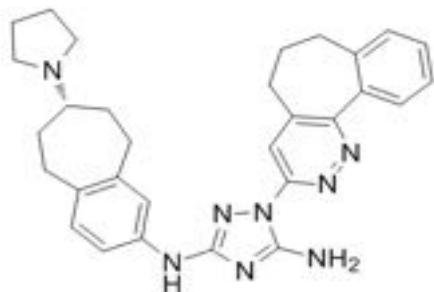
- Prostate cancer

- RCC

Bemcentinib



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



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





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

- ✓ Uniquely selective for AXL
- ✓ MOA is synergistic with other Immunotherapies enhancing response
- ✓ Favourable safety and tolerability profile supports broad use in lower risk first line as well as advance elderly fragile patients
- ✓ Once daily oral dosing
- ✓ Fast Track Designation by FDA for AML
- ✓ Safety and tolerability profile supports use in combination with chemo, targeted and IO drugs

BerGenBio pipeline - 3 selective AXL inhibitors in clinical development

Multiple attractive opportunities in AML and NSCLC

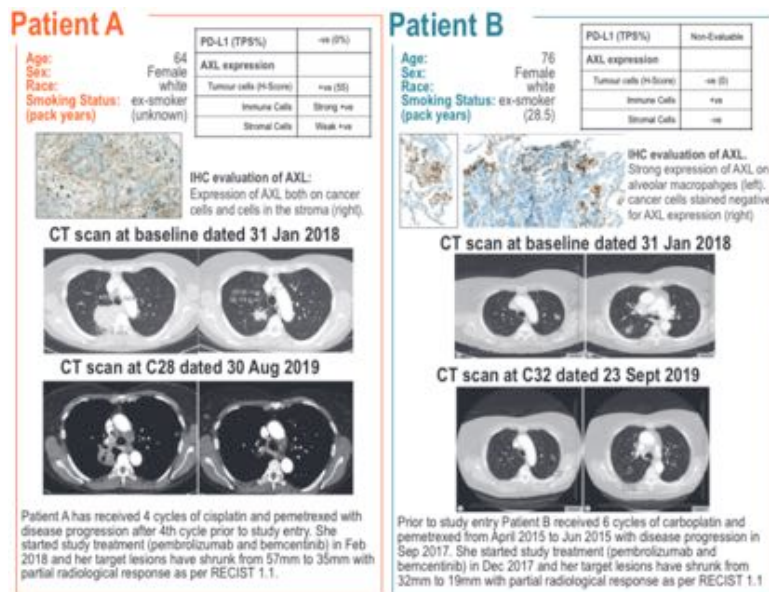
Candidate	Targeted Indication	Discovery	Preclinical	Phase I	Phase II	Phase III.
Bemcentinib	>2L AML	Ph II safety and POC efficacy demonstrated in 39 patient trial				
Bemcentinib (combination with LDAC)	2L AML	Ph Ib Safety demonstrated, efficacy POC expansion study- 28 pts.				
Bemcentinib (combination 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 on going 29 pts				
Tilvestamab (BGB149)	TBA	Ph I Healthy volunteer study ongoing				
BGB601 	Various solid tumors	Ph I safety study ongoing				

Potential label expansion with additional phase II studies with bemcentinib

		Clinical Proof-of-concept	Late stage Opportunities
Monotherapy Selected, biomarker directed patients	AML / MDS	Completed	
	Glioblastoma (IIT)	Ongoing	
	Ovarian (EMT signature selected)	Potential	
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	
	Melanoma, (IIT)	Ongoing	
	Mesothelioma (IIT)	In set-up	
	Bladder ++, CAR-T combos	Under consideration	
Targeted Therapy Combinations Target resistance, enlarge addressable patient population	NSCLC + EGFRi	Completed	
	Melanoma, (IIT)	Ongoing	
	PARPi combos ++	Under consideration	
Earlier Line Opportunities Radiotherapy and maintenance opportunities	Multitude of maintenance opportunities given very favourable safety profile		

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

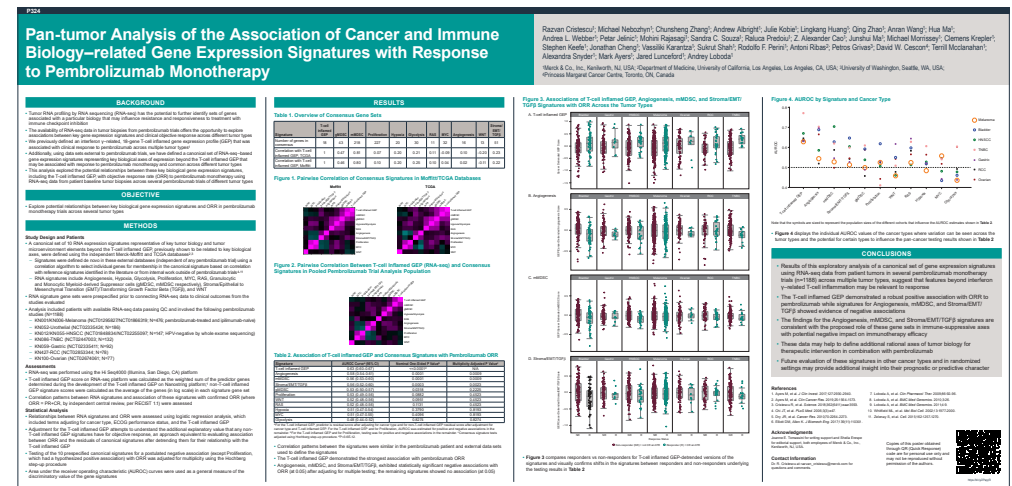
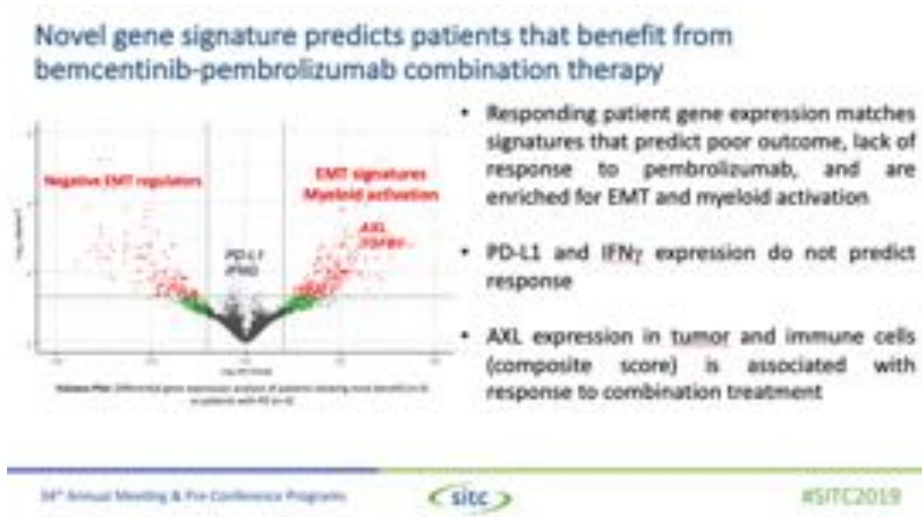
- AXL mediated EMT in tumour cells
- AXL+ve Mesenchymal tumour cells are drug resistant & immune evasive

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

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

AXL inhibitors – emerging competitive landscape



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

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

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



Acute Myeloid Leukaemia (AML)

Most common type of acute leukaemia in adults¹

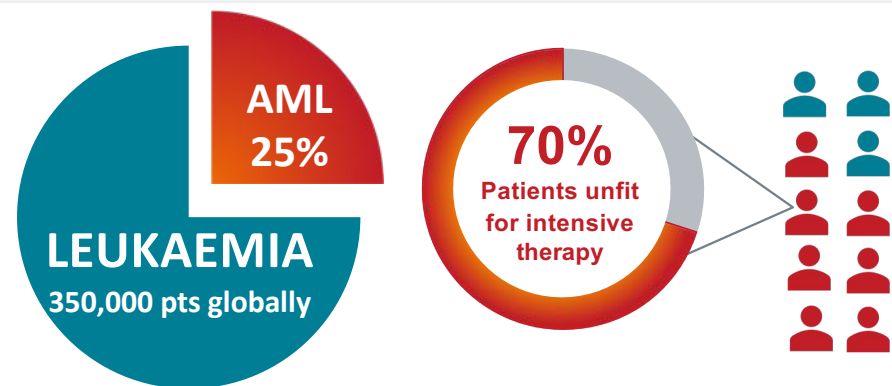
AML is a rare aggressive cancer of the blood and bone marrow characterised by difficult to treat malignancies

~ 21,000 new cases diagnosed and >11,000 deaths in the US in 2018²

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 years⁶

5 year survival rates of 3-8% in patients over 60 years old⁷



(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/>

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)
- Targeted agent for AML with mutation of FLT3, IDH1/2

Opportunity for
Bemcentinib + LDAC

1st Relapse

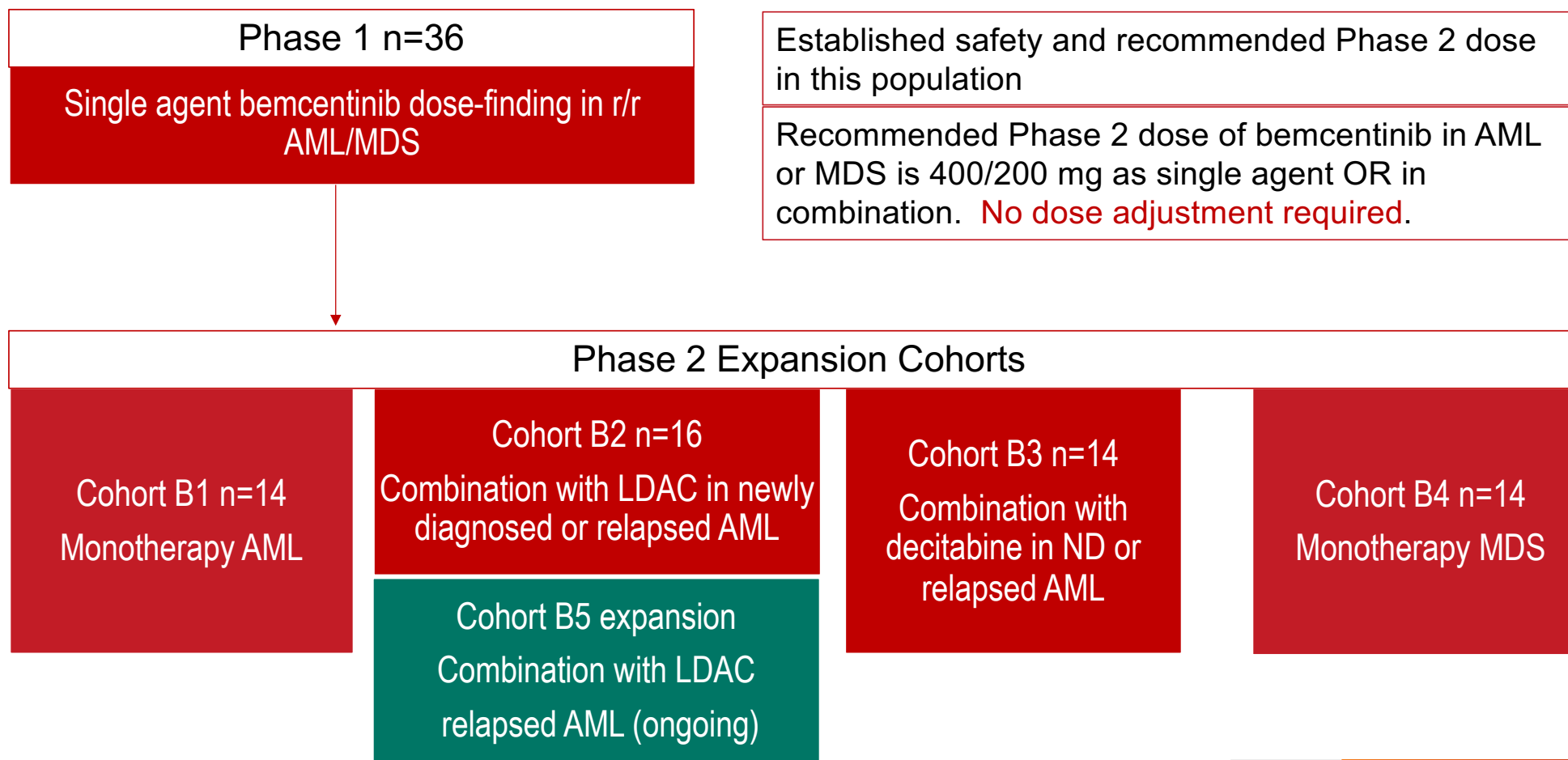
- Clinical trial
- No approved therapy, but options may include HMA, LDAC or single agent venetoclax
- Best supportive care (BSC) or palliative care

2nd Relapse

- Clinical trial
- BSC or palliative care

Opportunity for Single
Agent Bemcentinib

Bemcentinib clinical development in Acute Myeloid Leukemia, (BGBC003)



Results of the Phase 1 Bemcentinib monotherapy in relapsed/refractory AML

(Loges et al ASH 2018)

Bemcentinib monotherapy in $\geq 2L$ r/r AML patients >75 yrs.

	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%

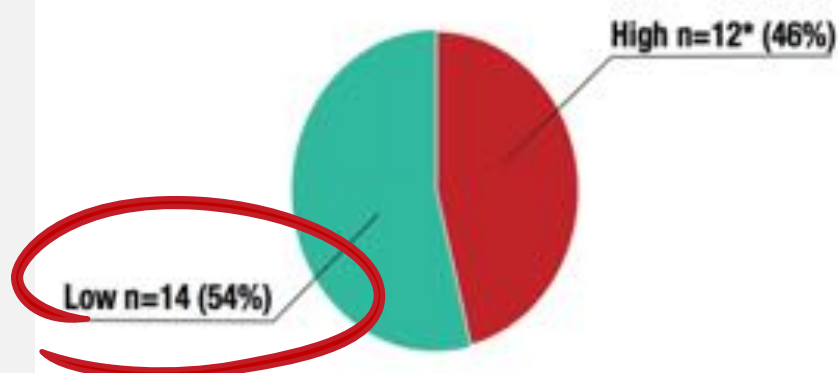
* 2 evaluable patients were not evaluable for sAXL status

* Monotherapy responses. One additional response was reported in combination with decitabine for a total of 7 responses in phase 1/2.

* 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



$\geq 2L$ Relapse patients >75yrs

No approved SoC

Bemcentinib Monotherapy

ASH 2018

AXL +ve* patients

14/27
54%

Stable Disease

3/14
21%

CR/Cri/CRp
6/14
43%

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

Results of the Phase IIa of LDAC+ Bemcentinib combination in newly diagnosed and relapsed/recurrent AML

(Loges et al ASH 2019)

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

Clinical Activity in Newly-Diagnosed Patients

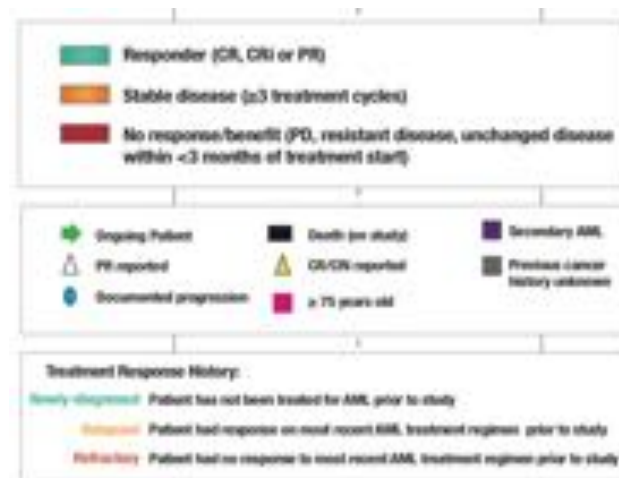
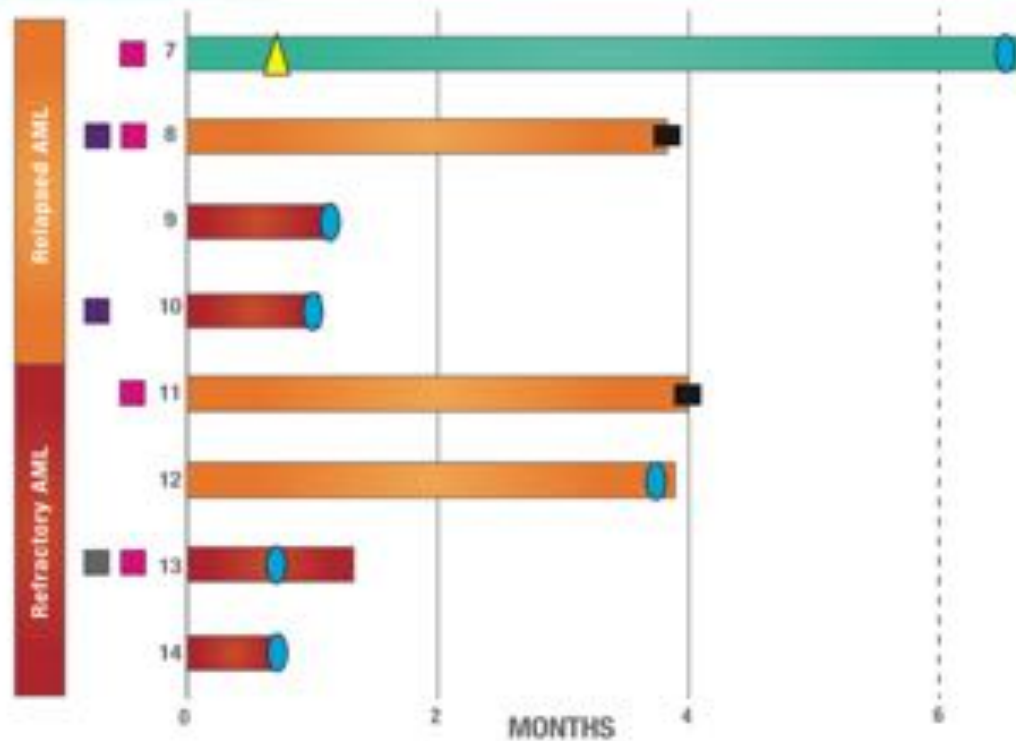
Time on treatment



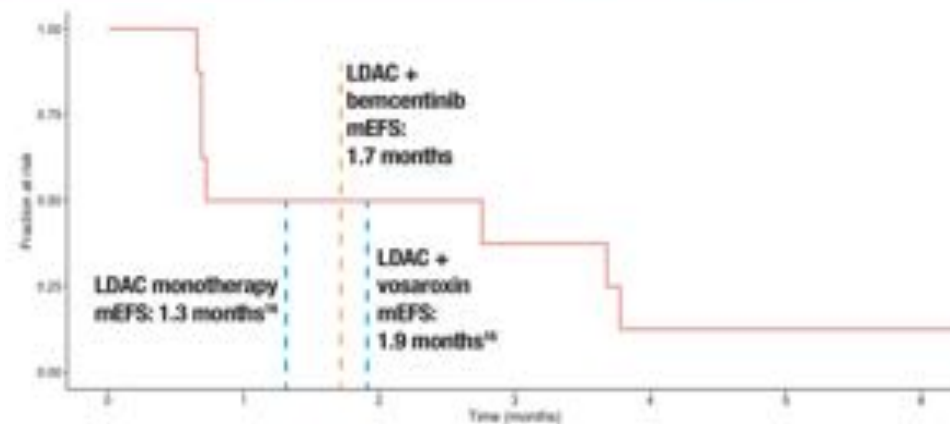
Bemcentinib + LDAC in r/r AML patients

Clinical Activity in Relapsed/Refractory Patients

Time on treatment



Event-free Survival (Relapsed/Refractory Patients)



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

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



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

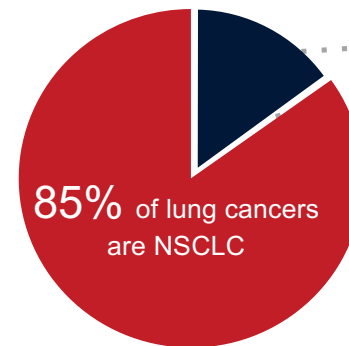
The largest cancer killer, most patients depend on drug therapy

The most common type of cancer

2.09 million new cases of lung cancer diagnosed/yr worldwide, making up 11.6% of all cancer cases¹

1.76 million lung cancer deaths/yr worldwide¹

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



Non- Small Cell Lung Cancer (NSCLC)

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

US market
(non mutation)

<1% PD-L1 expression 39%	1-49 % PD-L1 expression 38%	>50% PD-L1 23%	driver mutations*
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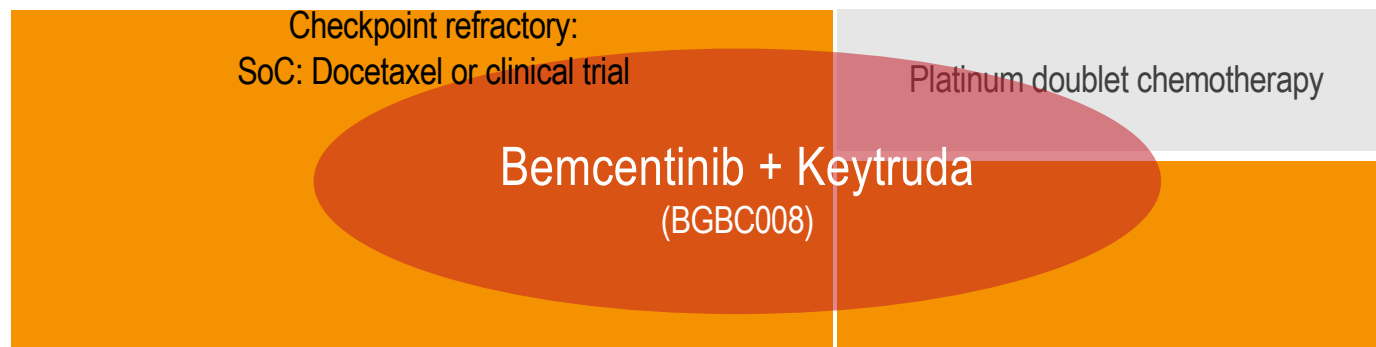
Bemcentinib Opportunities

1st Line
101,000 pts**
\$4,5bn

Platinum doublet chemotherapy +/- checkpoint inhibitor	Checkpoint inhibitor monotherapy	Targeted therapy
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Deepening 1L responses,
particularly PD-L1 negative/low

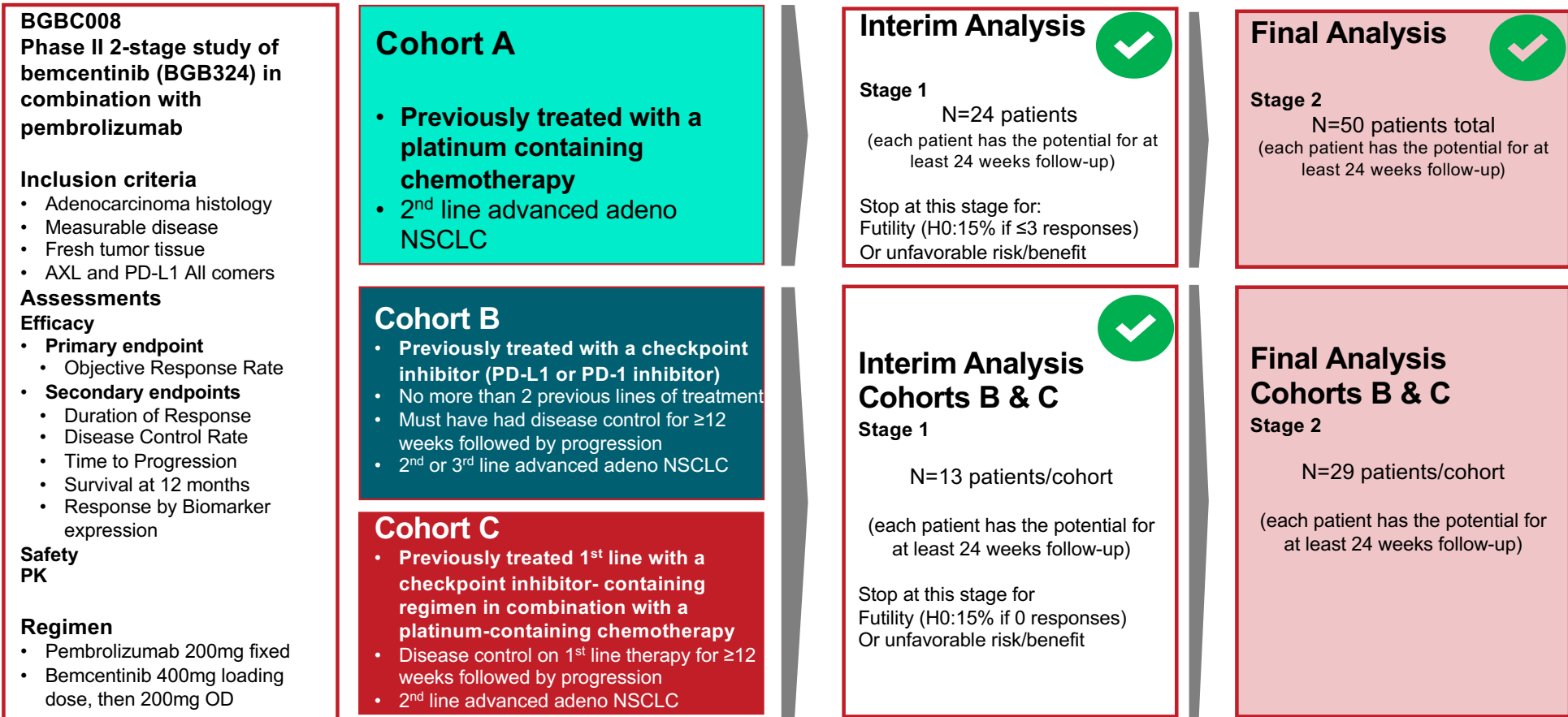
2nd Line
61,000pts**
\$3bn



Effective and well tolerated
2L therapies

Bemcentinib + KEYTRUDA in refractory/relapsed NSCLC

Phase II Study Design



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

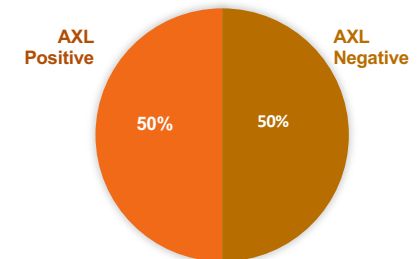
Treatment related adverse events were generally mild and reversible

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

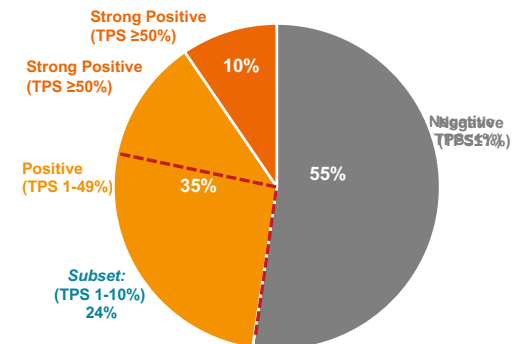
Most frequent TRAEs (≥10% dosed pts)				
Event Terms	All Grades		Grade ≥3	
	n	%	n	%
Transaminase increased*	19	38 %	7	14%
Asthenia / Fatigue	15	30 %	4	8%
Diarrhoea	12	24 %	0	0%
Nausea	7	14 %	0	0%
Anaemia	6	12 %	1	2%
Blood creatinine increased	6	12 %	0	0%
Decreased appetite	6	12 %	0	0%
Pruritus	5	10 %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

Biomarker

cAXL status
n = 30



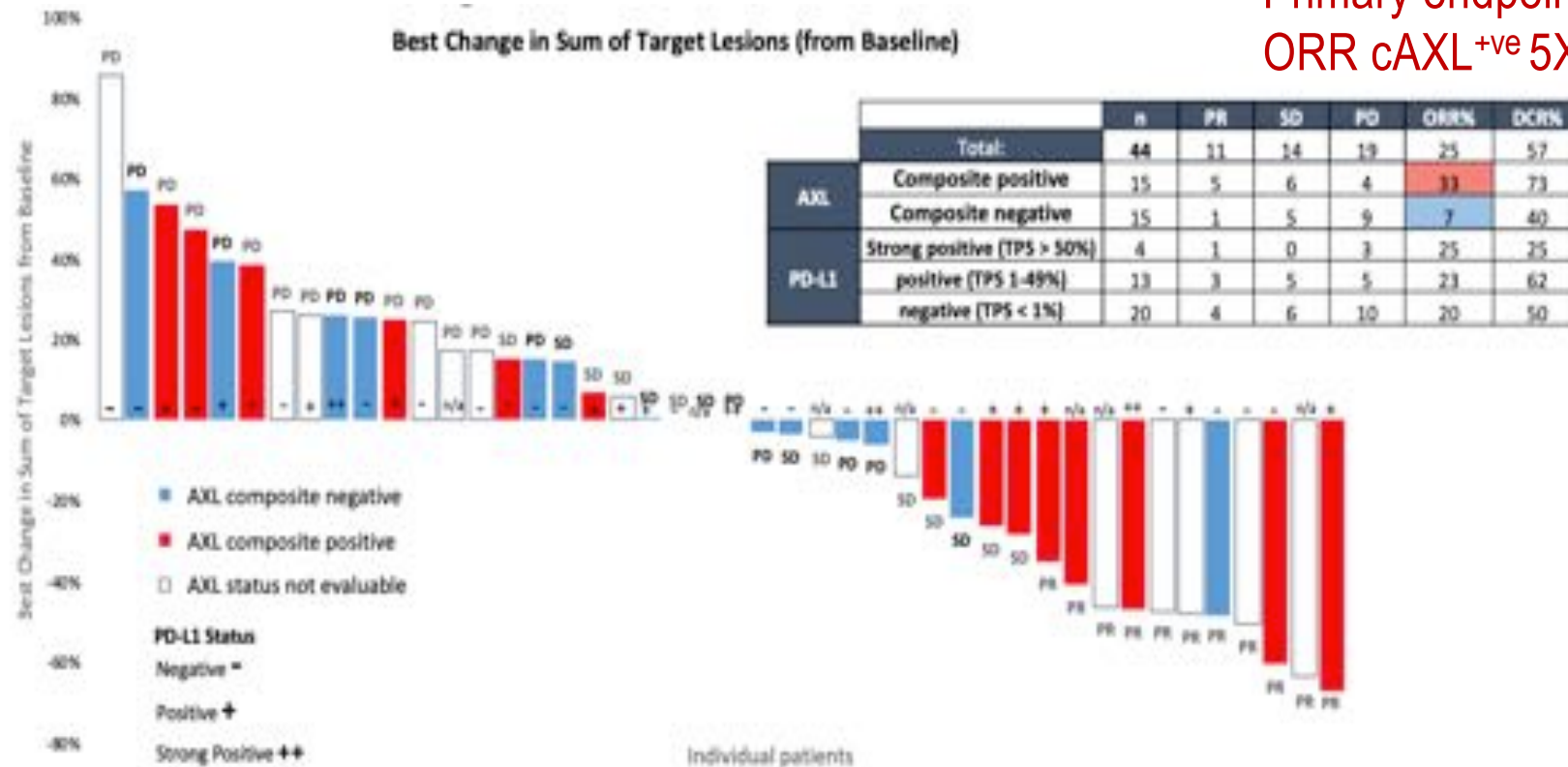
PD-L1 status
n = 37



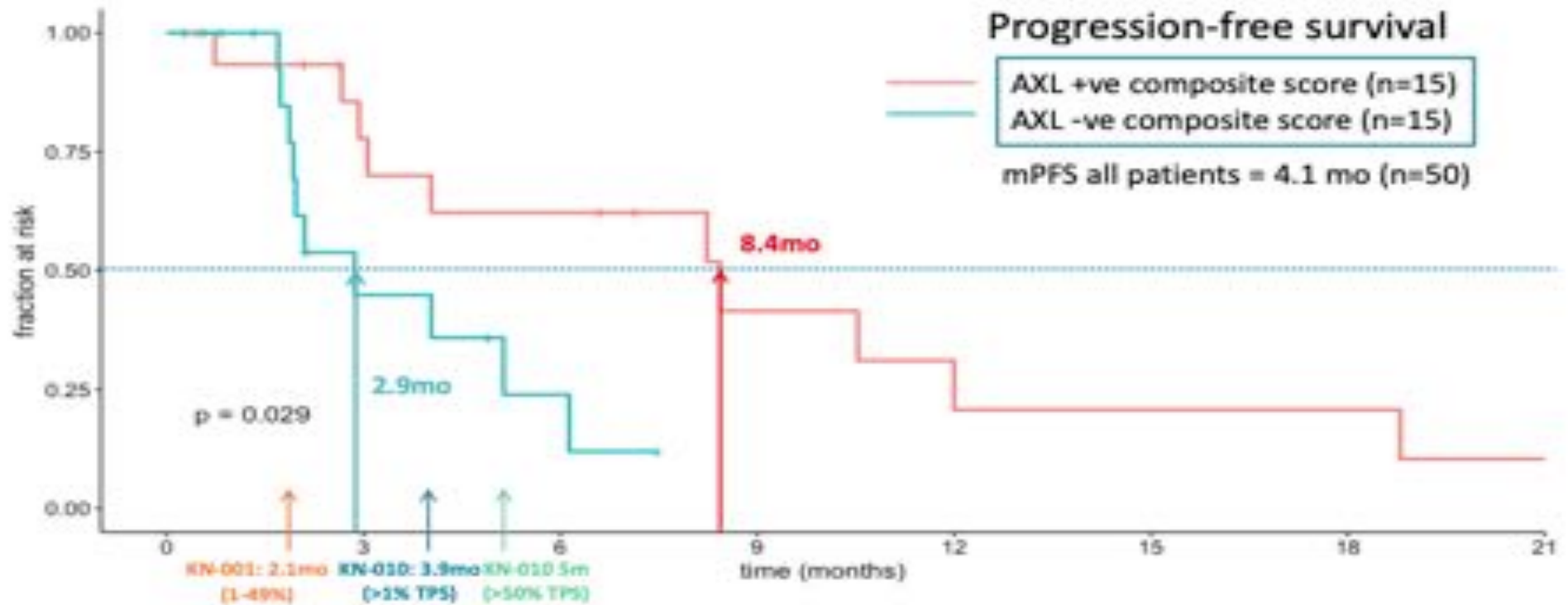
*Data cutoff (30 Sep 2019)

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

Primary endpoint met:
ORR cAXL⁺ve 5X > cAXL⁻ve



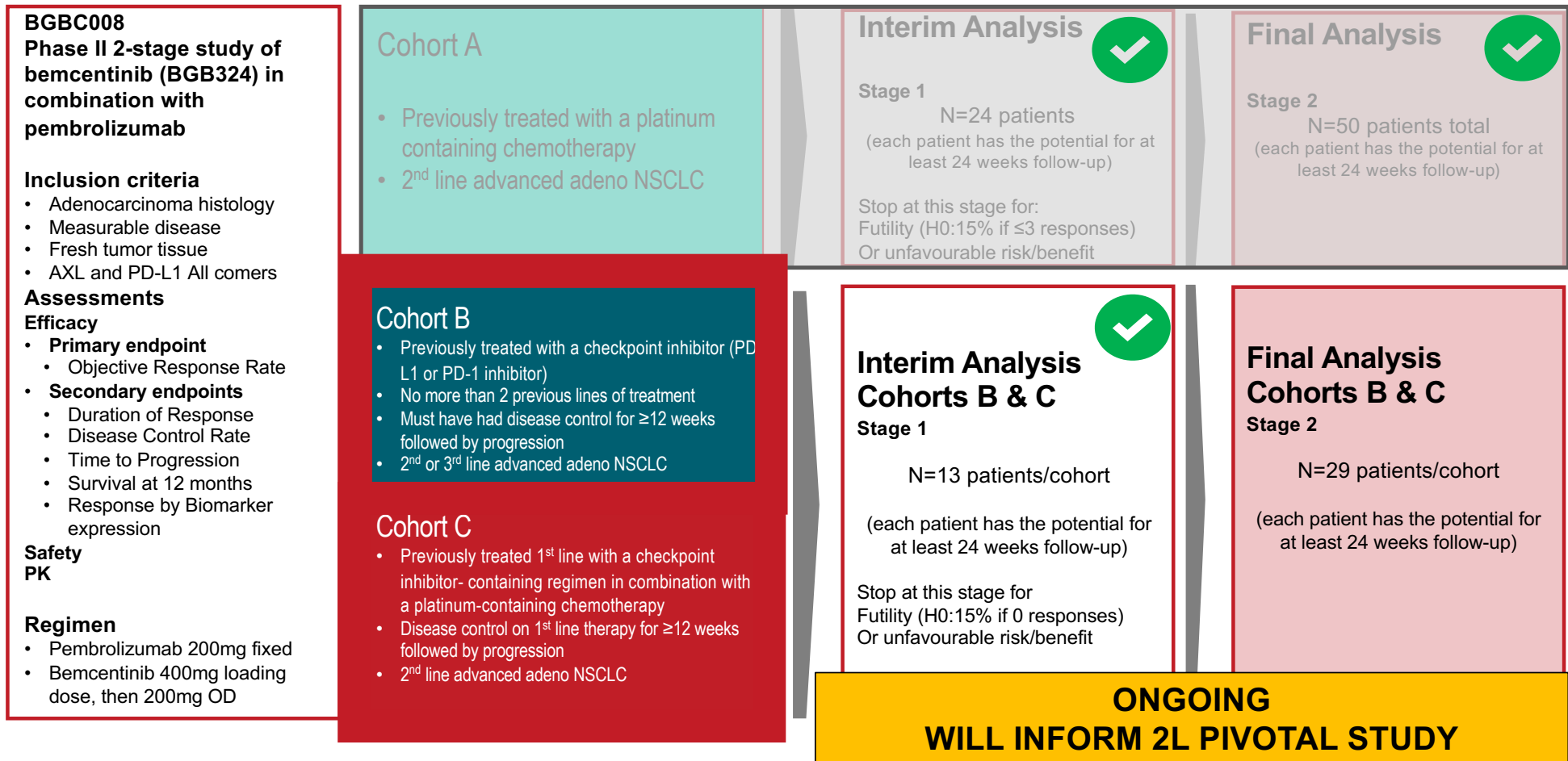
Significant mPFS improvement in cAXL +ve patients:
AXL is an adverse prognostic biomarker – therefore cAXL score is predictive



- ✓ 3-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

Bemcentinib + KEYTRUDA in refractory/relapsed NSCLC

Phase II Study Design



Bemcentinib + KEYTRUDA in refractory/relapsed NSCLC – cohort B & C

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

Interim Analysis Cohort B 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 unfavourable risk/benefit



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 1° endpoints: Interim mPFS, (for C/AA) 6 & 12mn OS, OS (for full approval) 2° endpoints: ORR, DoR, Safety, tolerability.	<ol style="list-style-type: none"> 1. Pending BGBC008 cohort B + C 2. SA advice from FDA & EMA 3. cAXL assay validation in BGBC008 B&C
1L	TBA		



BGB149

anti-AXL monoclonal antibody



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

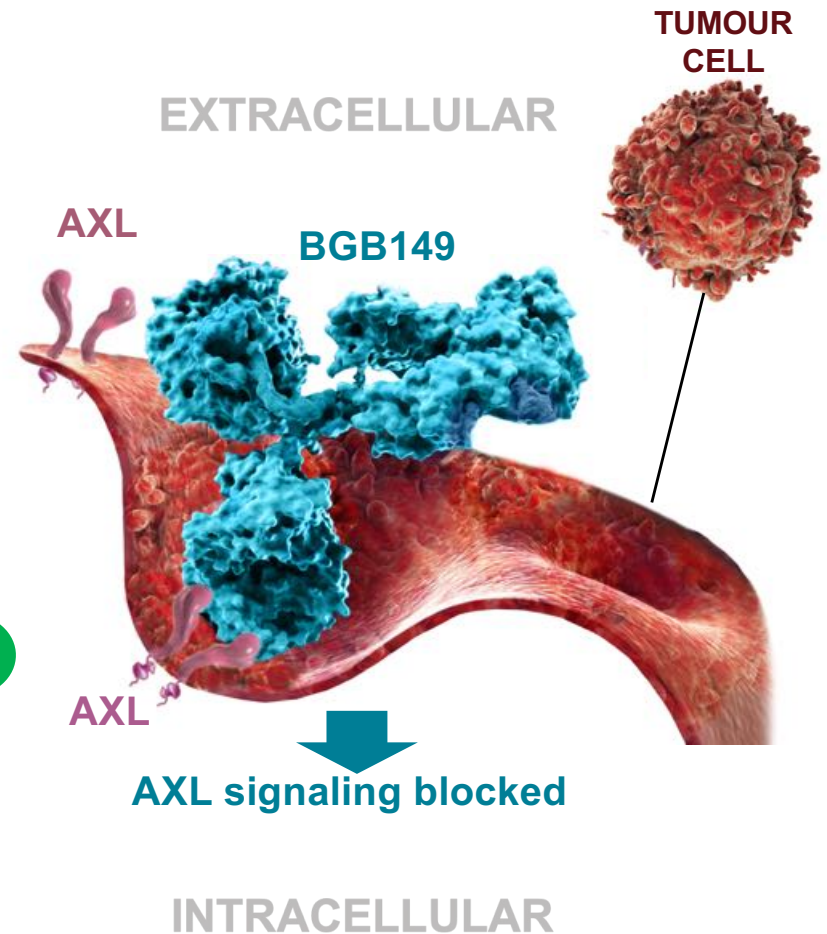
Phase Ia healthy volunteer SAD study complete

Safety – no dose limiting toxicity seen up to 3mg/kg dose

Pharmacokinetics - exposure predictable with dose proportional C_{max} increase

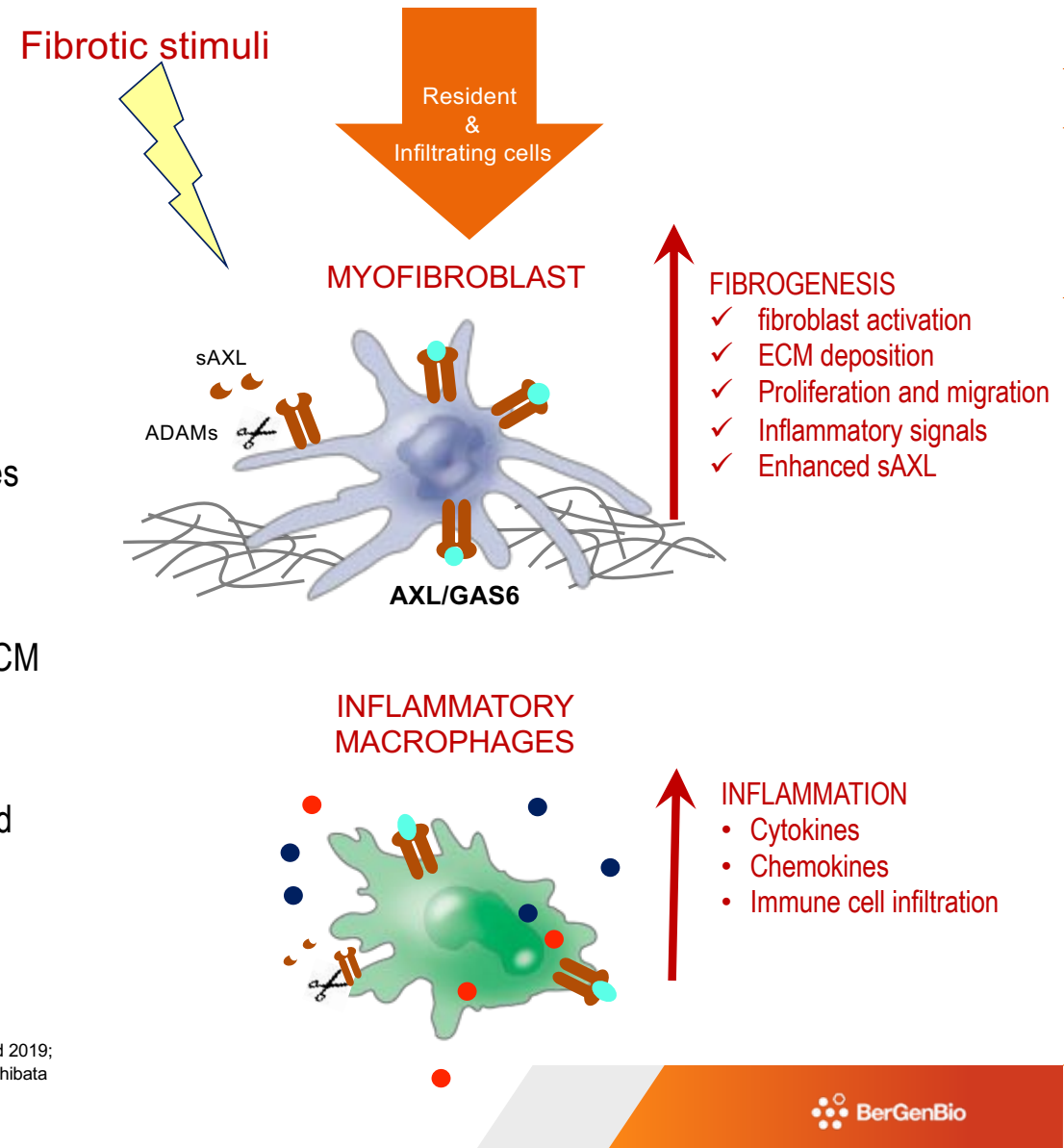
Confirmatory evidence of *in vivo* target engagement with sAXL -- stabilisation in circulation

First-in-patient trial expected in H2 2019



The role of AXL in fibrosis

- AXL Regulates and modulates key fibrogenic pathways
 - TGF β signaling^{1,2}
 - Mechanosensing Hippo pathway³
 - Peroxisome proliferator-activated receptor⁴
- Axl regulates cellular plasticity implicated in fibrotic pathologies e.g. EMT, EndMT, Macrophage polarity
- AXL is a negative regulator of epithelial cell barrier integrity⁵
- Axl is required for hepatic stellate cell (HSC) activation and ECM deposition⁶
- Pharmacological modulation of Axl inhibits pre-clinical development of Liver (CCl₄₆/HighFatDiet₇), Renal (UUO₈) and Pulmonary (Asthma⁹, Bleo¹⁰, IPF¹⁰) fibrosis

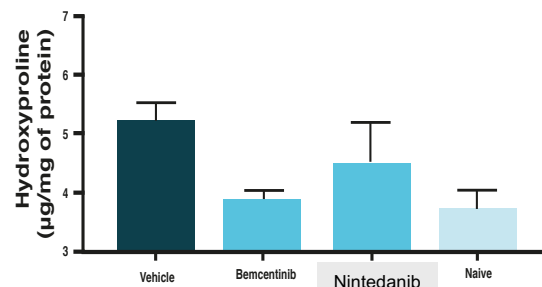


1 Gilbane ART 2015; 2 Reichl Hep. 2015; 3 Gibault ChemMed 2017; 4 Zhu AJTR 2016; Fujino Lab invest 2017, J Exp Med 2019; 6 Barcena J. Hep 2015; 7 Tutusaus A. Cell Mol Gastroenterol 2019 Hepatol. 2019; 8 Landolt L. Physiol Reports 2019; 9 Shibata J Immunology 2014; 10 BerGenBio ASA, unpublished; 11 Espindola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

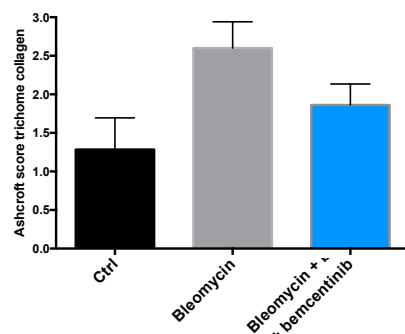
AXL inhibition prevents fibrosis in a panel of pre-clinical models

Lung

Bemcentinib reduces fibrosis in a human xenograft model of IPF¹

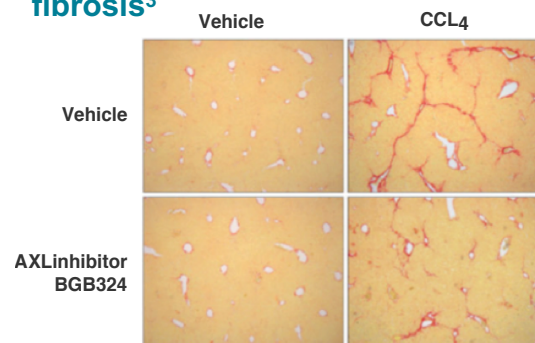


Bemcentinib reduces bleomycin induced fibrosis²

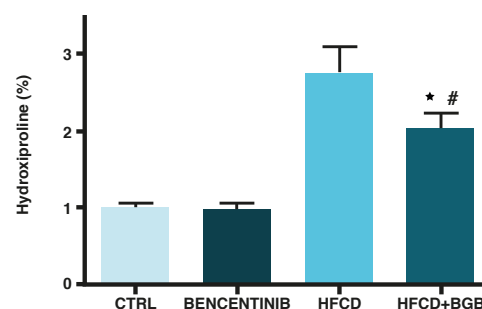


Liver

Bemcentinib reduces fibrosis in the CCL₄-induced model of liver fibrosis³



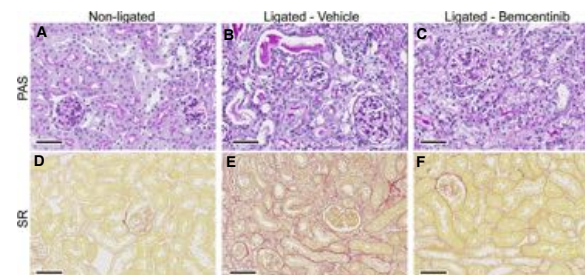
Bemcentinib reduces fibrosis in a diet induced model of NASH⁴



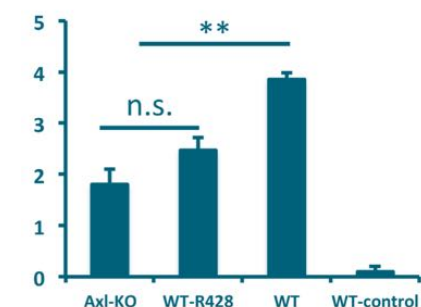
HFCD = high-fat, choline deficient diet
Leads to NASH in animal models

Kidney

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



Bemcentinib ameliorates anti-GBM induced lupus like nephritis and improved kidney function⁶



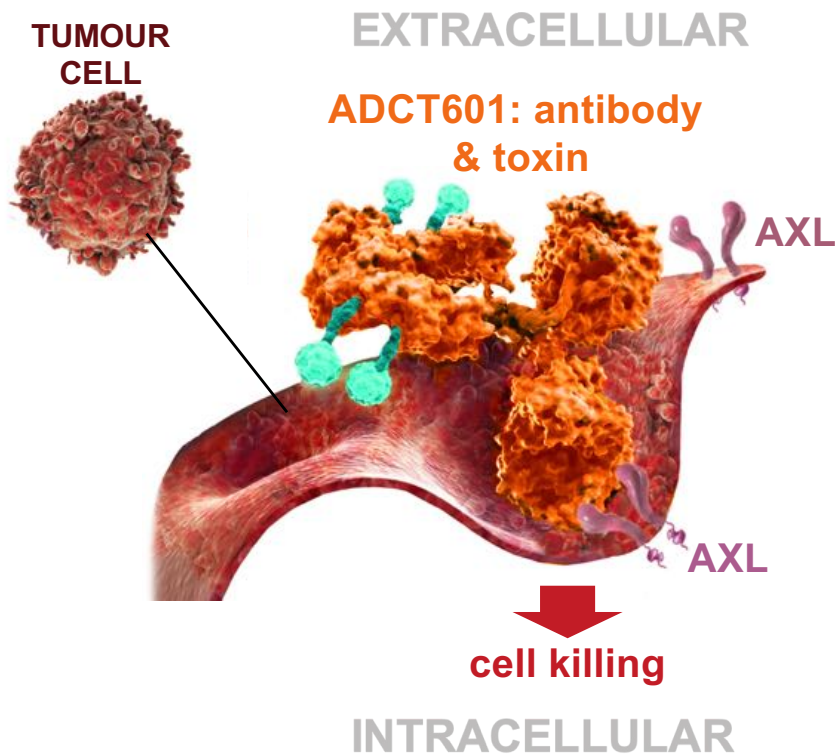


ADCT-601 – AXL ADC

BGB601/ADCT-601: Anti-AXL ADC

Phase 1 in solid tumours ongoing

Out-licensed to ADC Therapeutics (ADCT)



Antibody Drug Conjugate (ADC)

Targets human tumour AXL, induces cell death when internalised

Potent and specific anti-tumour activity demonstrated preclinically¹

First-in-human Phase I study initiated in Jan 2019

- Solid tumours
- Up to 75 patients
- Safety, PK/PD, preliminary efficacy

Based on anti-AXL antibody BGB601 licensed from BerGenBio

Corporate.



Expected Milestones Through 2020

2H19		1H20		2H20				
ASH		AACR	ASCO	EHA	WCLC	ESMO	SITC	ASH
bemcentinib	AML: Expand 2L r/r efficacy & durability combination with LDAC (BGBC003/B5)		AML: Expand 2L r/r interim efficacy & durability combination with LDAC					
	NSCLC: 2L IO refractory headline efficacy combination with pembrolizumab (BGBC008/B1)	NSCLC: Expand 2L Phase 2 IO refractory in combination with pembrolizumab (BGBC008/B2)		NSCLC: Expand 2L IO refractory interim efficacy & mPFS combination with pembrolizumab				
	NSCLC: Initiate 2L Phase 2 IO + CHEMO refractory in combination with pembrolizumab (BGBC008/C1)			NSCLC: Expand 2L IO refractory interim efficacy & mPFS combination with pembrolizumab				
tilvestamab	Healthy volunteers Phase Ia SAD study			Phase 1b/2a patient study initiate				

Select Company Financials

Oslo Børs	BGBIO
Cash (Q3'19)	\$32m
Shares Outstanding	61,1m

Analyst coverage



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Link to reports from Trinity Delta:

<https://www.bergenbio.com/investors/analyst-coverage/>

Thank you

