



Q4 and Full Year 2019

HIGHLIGHTS REPORT AND FINANCIALS

11th February 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

Cash: Q4'19 + Jan'20 PIPE NOK470m (\$53m)

Q4 2019 and post-period 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®
Three-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

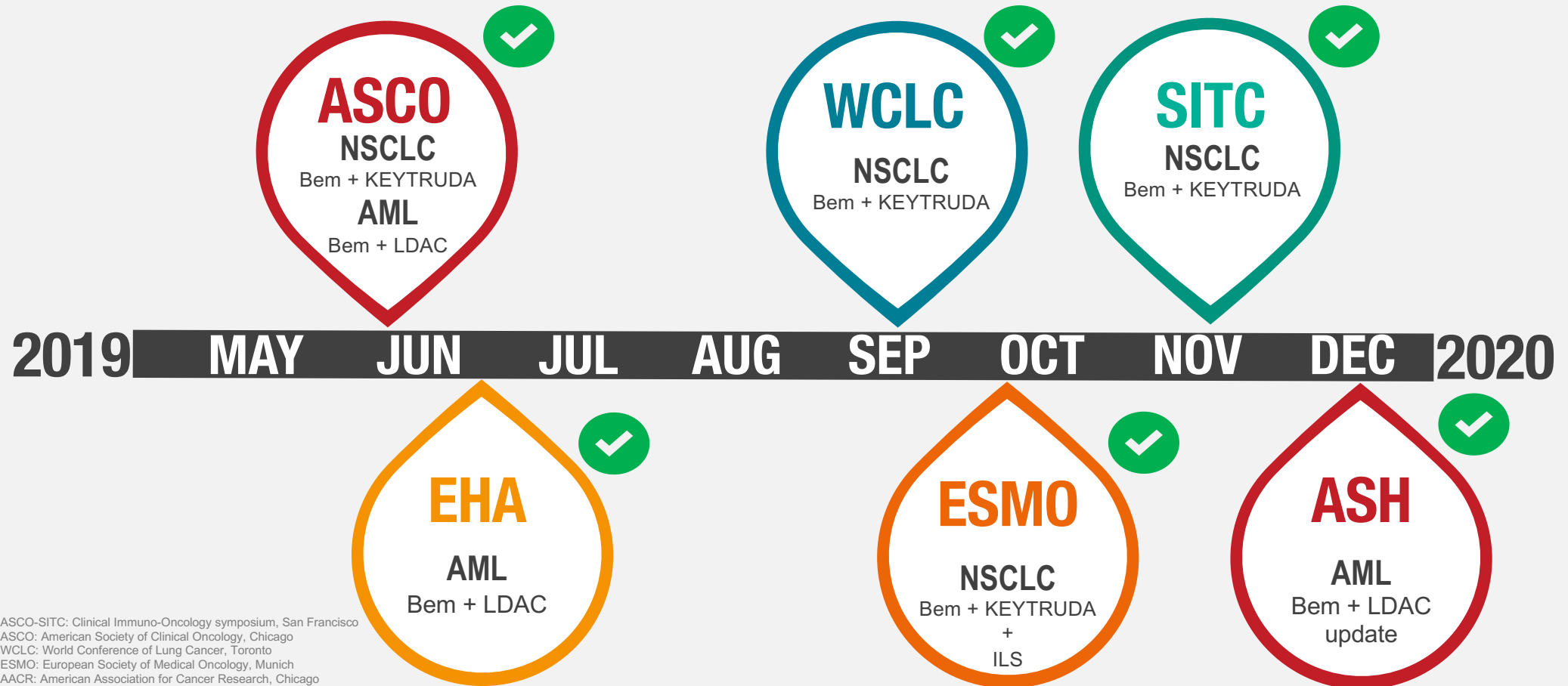
Jan
2020

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

Feb
2020

Cash and Cash Equivalents at end of Q4'19 + PIPE funding Jan '20 NOK 470m
Operating loss of NOK 59.1m in Q4'19 and NOK 204.4m full year 2019

Newsflow 2019



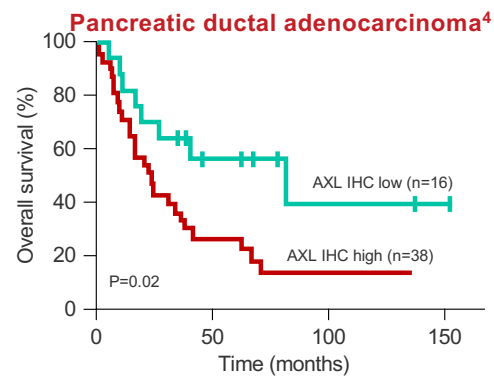
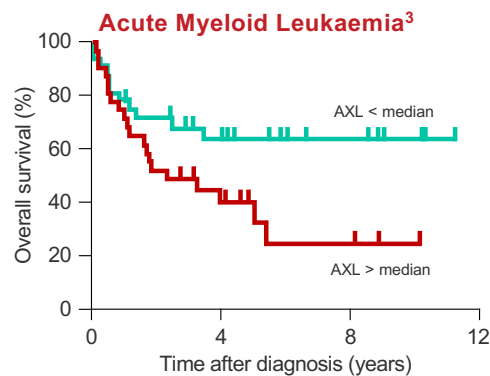
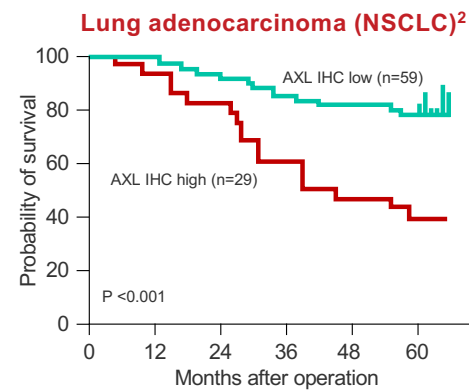
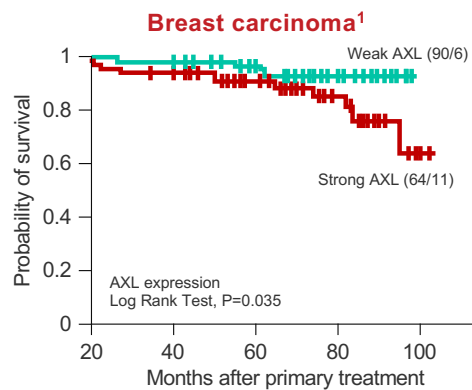
ASCO-SITC: Clinical Immuno-Oncology symposium, San Francisco
 ASCO: American Society of Clinical Oncology, Chicago
 WCLC: World Conference of Lung Cancer, Toronto
 ESMO: European Society of Medical Oncology, Munich
 AACR: American Association for Cancer Research, Chicago
 EHA: European Hematology Association, Stockholm
 SITC: Society for Immunotherapy of Cancer, DC
 ASH: American Society for Hematology, San Diego

AXL drives aggressive cancer



AXL +ve patients have a poor prognosis in most 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

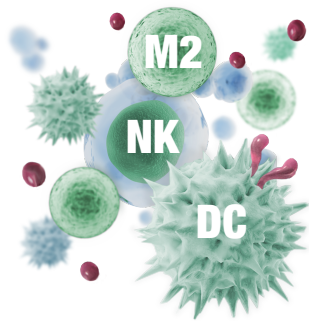
- RCC

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

very low expression under healthy physiological conditions

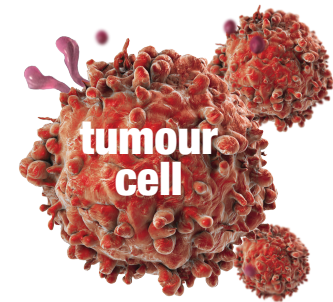
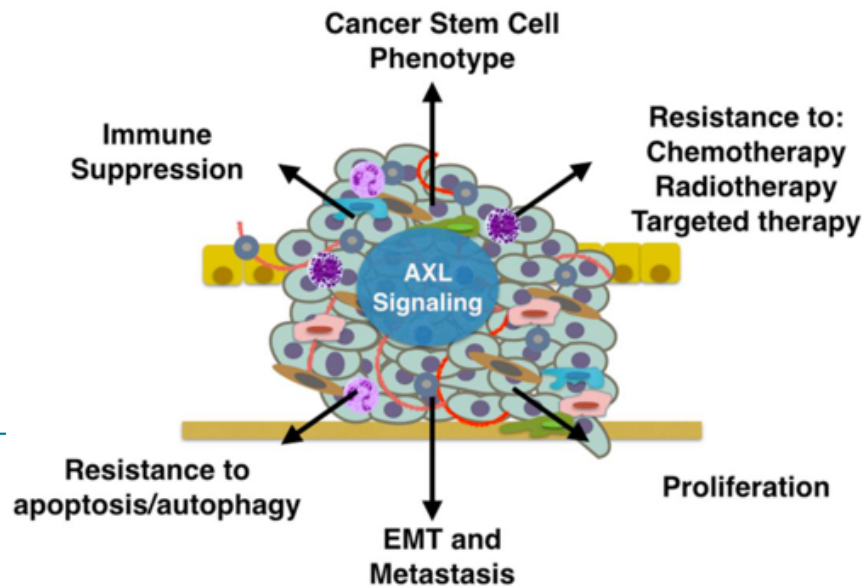
overexpressed in response to hypoxia, inflammation, cellular stress & drug treatment

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



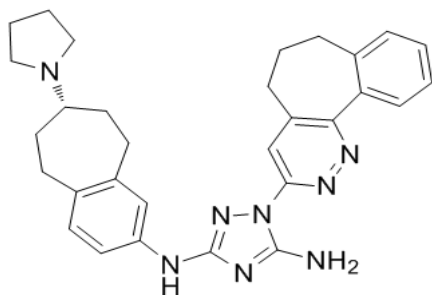
AXL increases on the tumor cell and causes cancer escape and survival

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

Bemcentinib



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



- ✓ Potent ($IC_{50} = 14 \text{ nM}$)
- ✓ Selective (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 cancer drugs 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

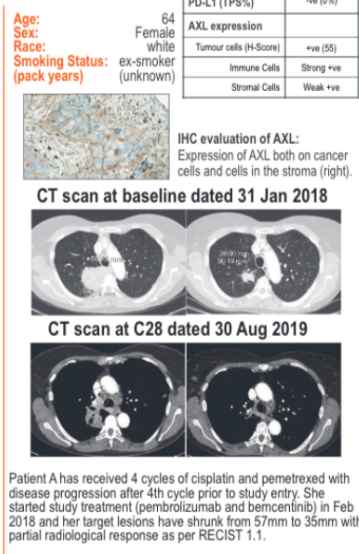
Multiple attractive opportunities in AML and NSCLC

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				

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

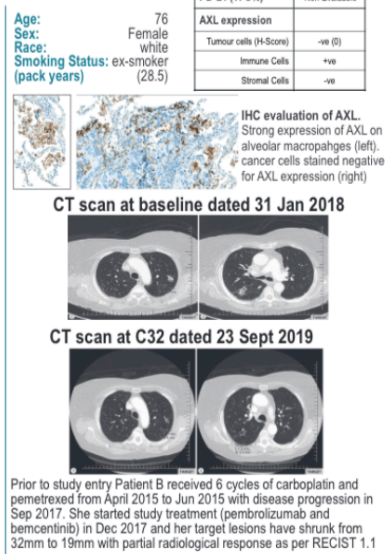
Patient A



Patient A: RESPONDER

- AXL stained +ve on tumor cells
- 61% tumor shrinkage

Patient B



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 Acute Myeloid Leukaemia (AML)

Objective: to develop a well tolerated, effective and convenient drug for this difficult to treat, elderly & frail patient population.

- ✓ Monotherapy $\geq 2L$ patients >75 yrs
43% ORR in AXL +ve R/R AML
- ✓ LDAC chemo combination 2L R/R patients >60 yrs



Acute Myeloid Leukaemia (AML)

Most common type of acute leukaemia in adults¹

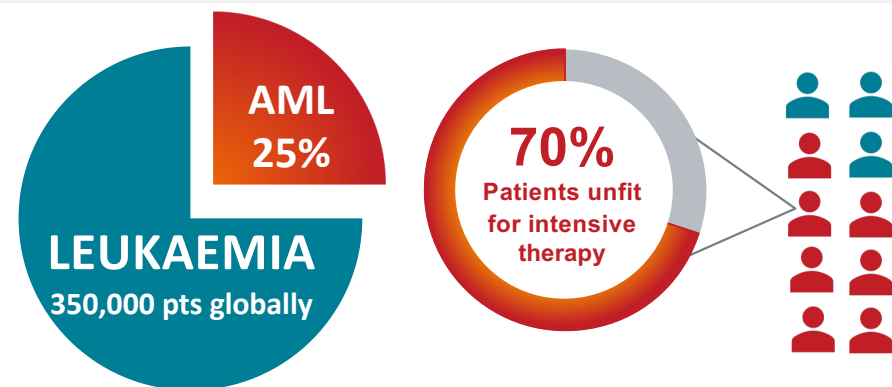
AML is a rare aggressive cancer of the blood and bone marrow characterised by difficult to treat malignancies

~ 20,000 new cases diagnosed and >10,000 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

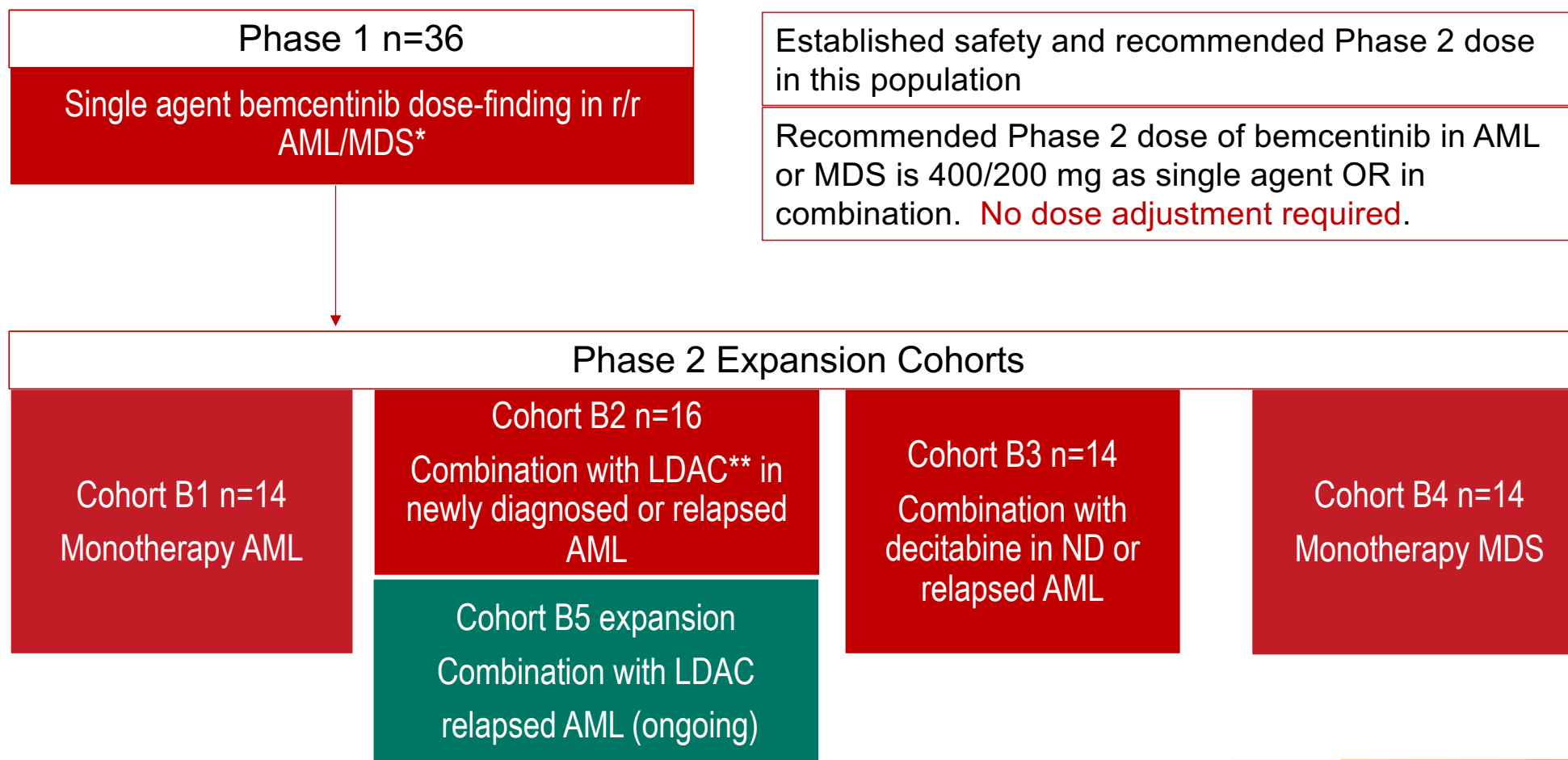
- Clinical trial
- No approved therapy, but options may include HMA, LDAC or single agent venetoclax
- Best supportive care (BSC) or palliative care

2nd Relapse

- Clinical trial
- BSC or palliative care

Opportunity for Single
Agent Bemcentinib

Bemcentinib clinical trial in Acute Myeloid Leukemia, (BGBC003)

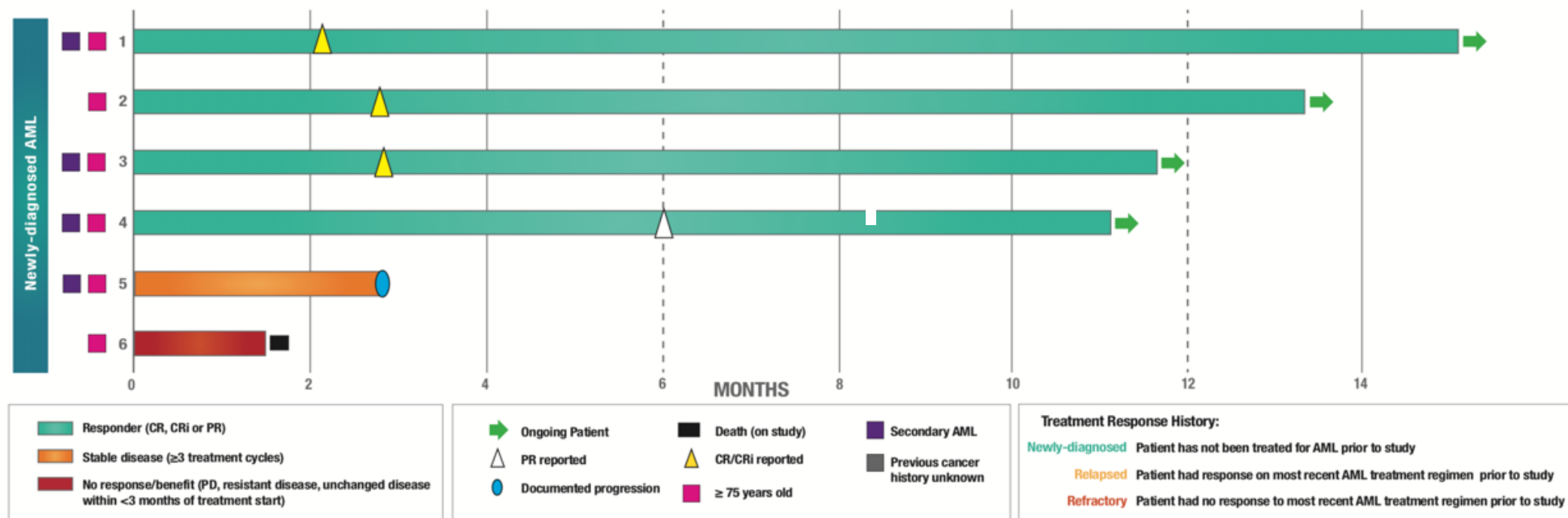


Bemcentinib + LDAC combination is active and effective in 1L newly diagnoses unfit/elderly AML patients

- 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

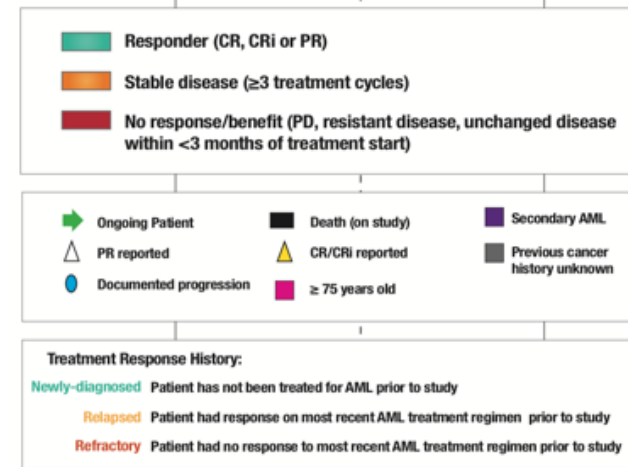
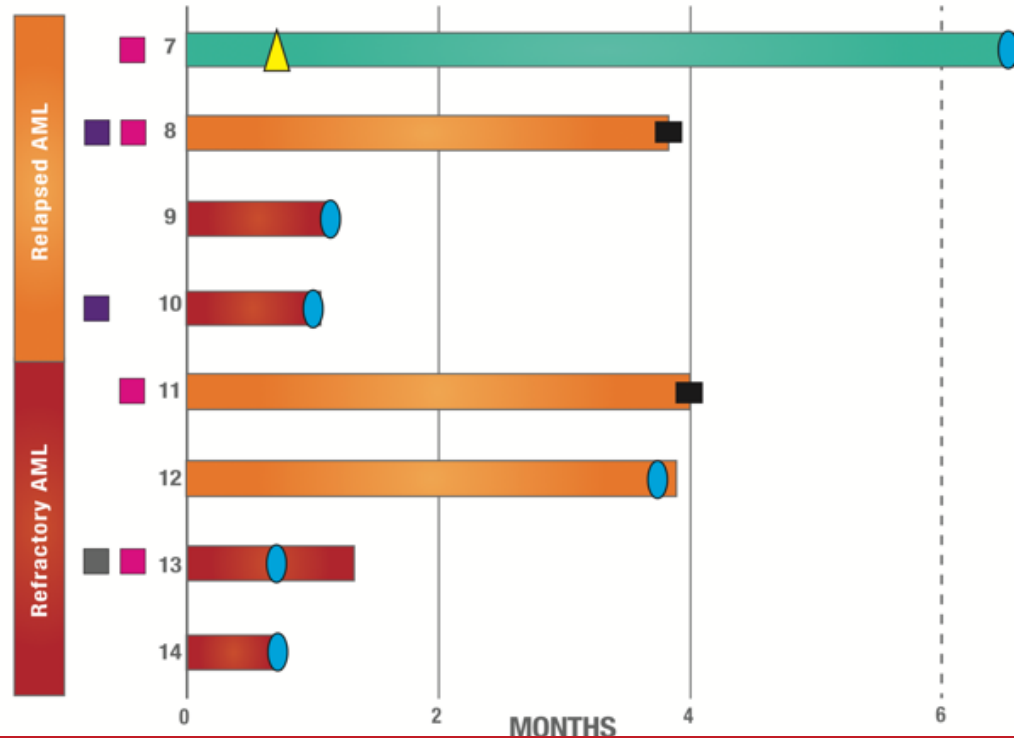
Time on treatment



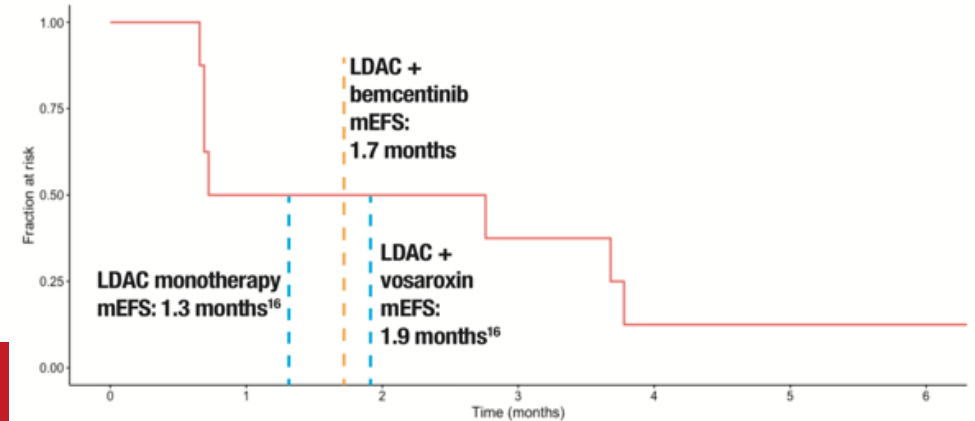
Bemcentinib + LDAC in r/r AML patients

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 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

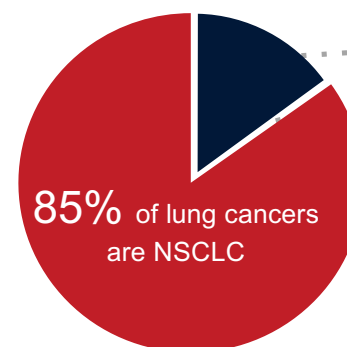
The largest cancer killer, most patients depend on drug therapy

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*

Bemcentinib Opportunities

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

Platinum doublet chemotherapy
+/- checkpoint inhibitor

Checkpoint
inhibitor
monotherapy

Targeted therapy

Deepening 1L responses,
particularly PD-L1 negative/low

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

Checkpoint refractory:
SoC: Docetaxel or clinical trial

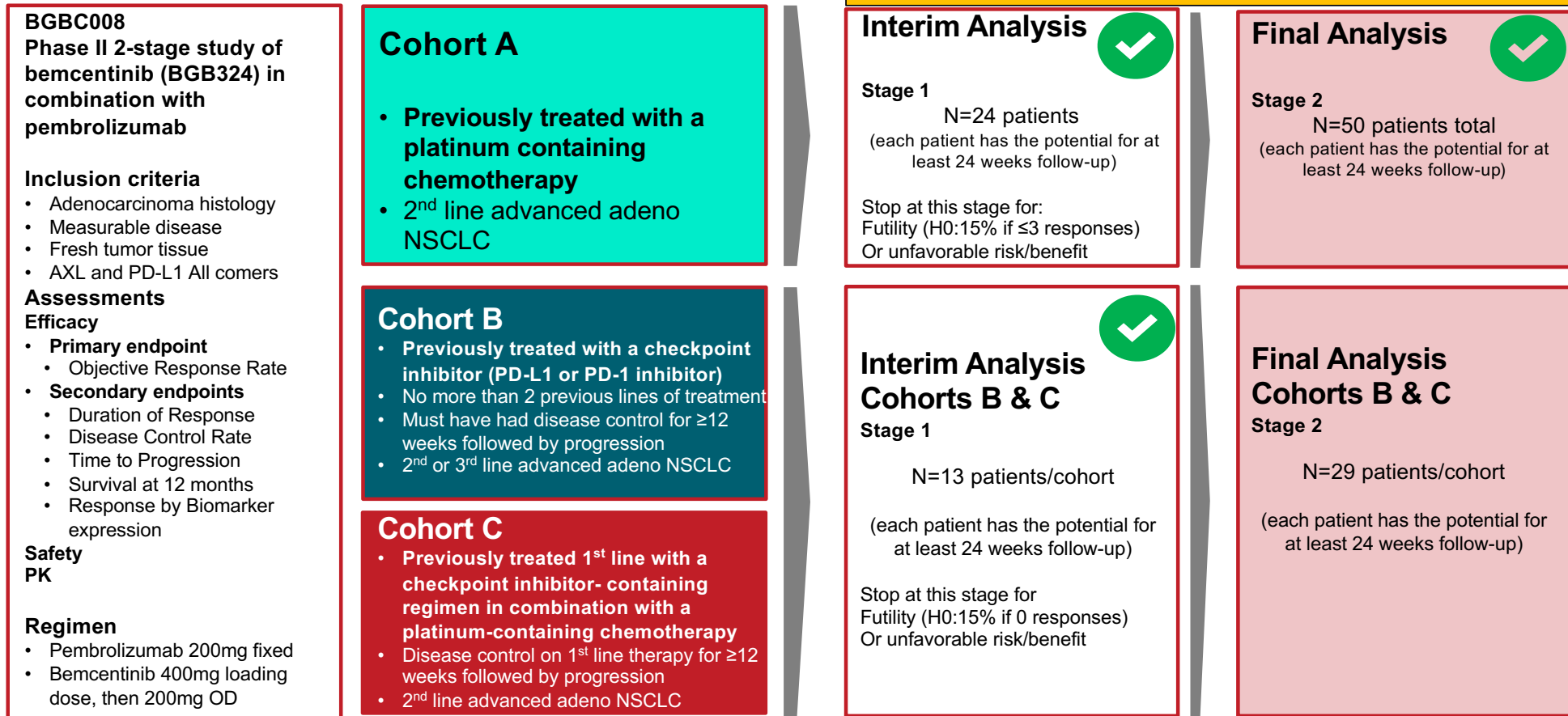
Platinum doublet chemotherapy

Bemcentinib + Keytruda
(BGBC008)

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

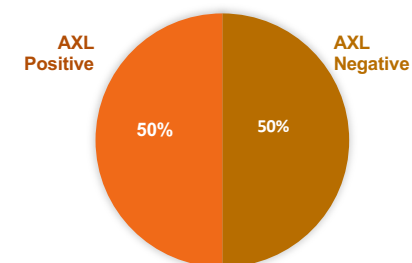
Treatment related adverse events were considered to be less severe and better tolerated than for other TKIs or CPI combinations used in NSCLC

Most frequent TRAEs (≥10% dosed pts)

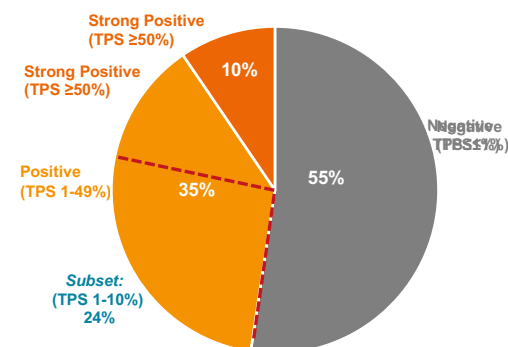
Event Terms	All Grades		Grade≥3	
	n	%	n	%
Transaminase increased*	19	38 %	7	14%
Asthenia / Fatigue	15	30 %	4	8%
Diarrhoea	12	24 %	0	0%
Nausea	7	14 %	0	0%
Anaemia	6	12 %	1	2%
Blood creatinine increased	6	12 %	0	0%
Decreased appetite	6	12 %	0	0%
Pruritus	5	10 %	0	0%

Biomarker

cAXL status n = 30



PD-L1 status n = 37

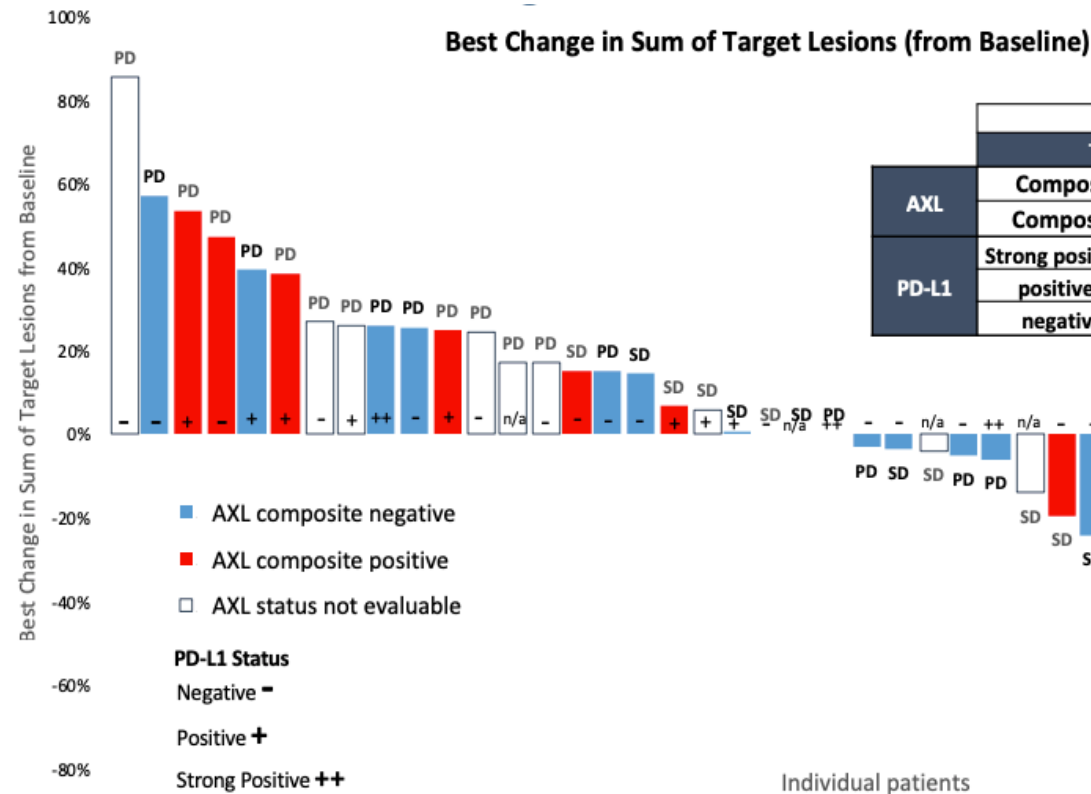


*Data cutoff (30 Sep 2019)

Cohort A: Anti-tumor activity of bemcentinib in combination with pembrolizumab

Change in tumour size from baseline by RECIST 1.1

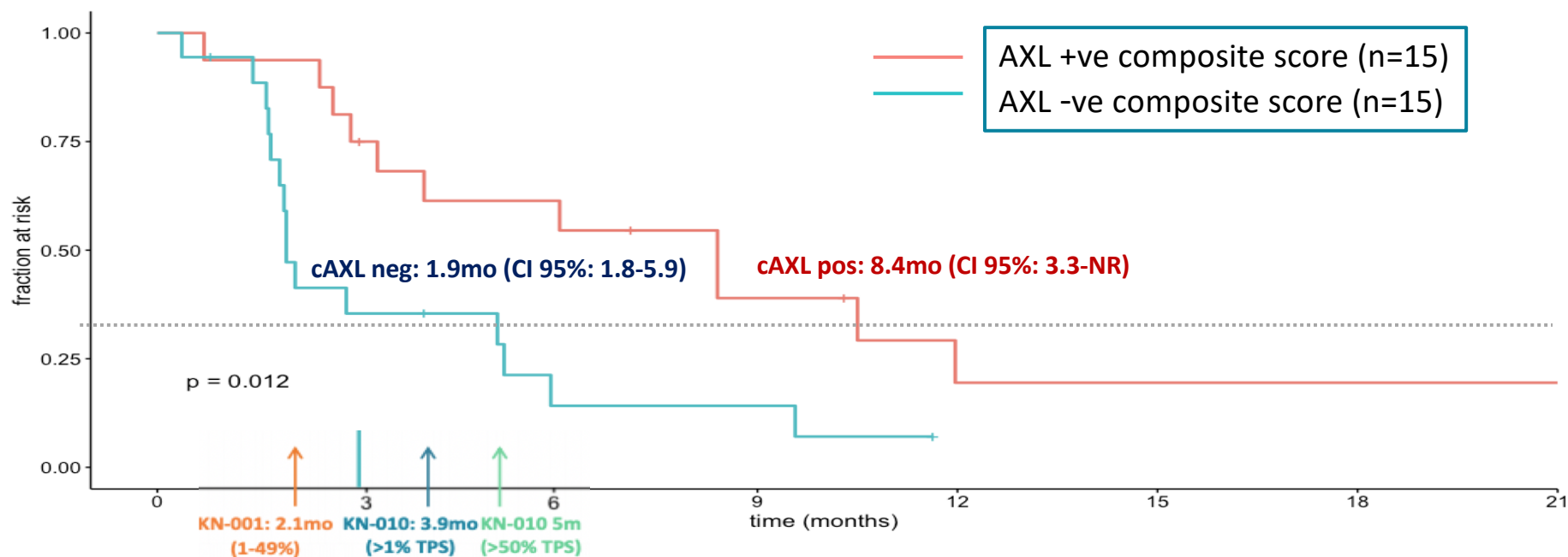
Primary endpoint met:
Overall Response Rate
cAXL^{+ve} 5X > cAXL^{-ve}



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

Individual patients

Cohort A: >4 X improvement in mPFS* in composite 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

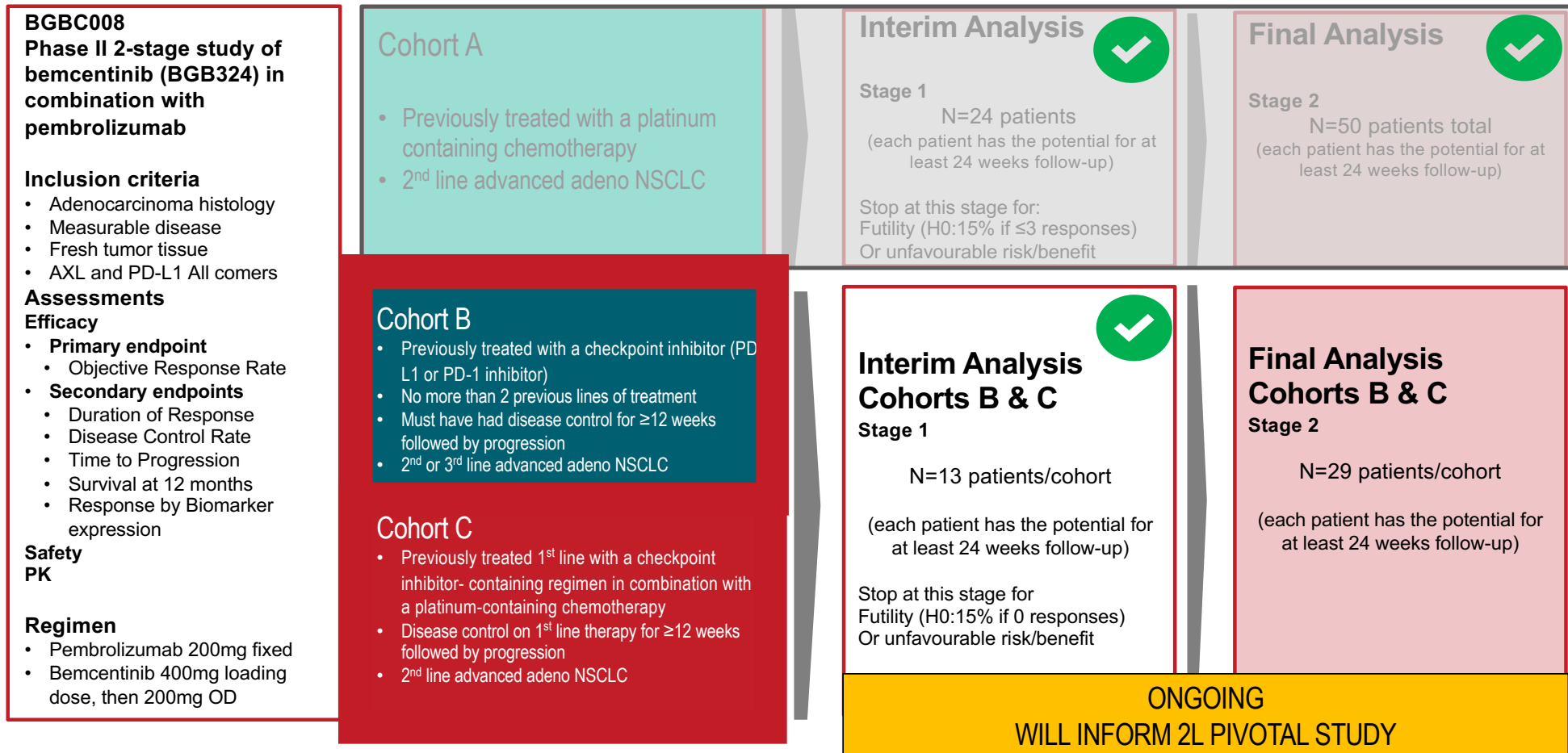
Cohort A results update : January 20, 2020

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Source: KN001; Garon *et al* NEJM 2015; KN-010 Herbst *et al*, Lancet 2016 *Progression-free survival

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



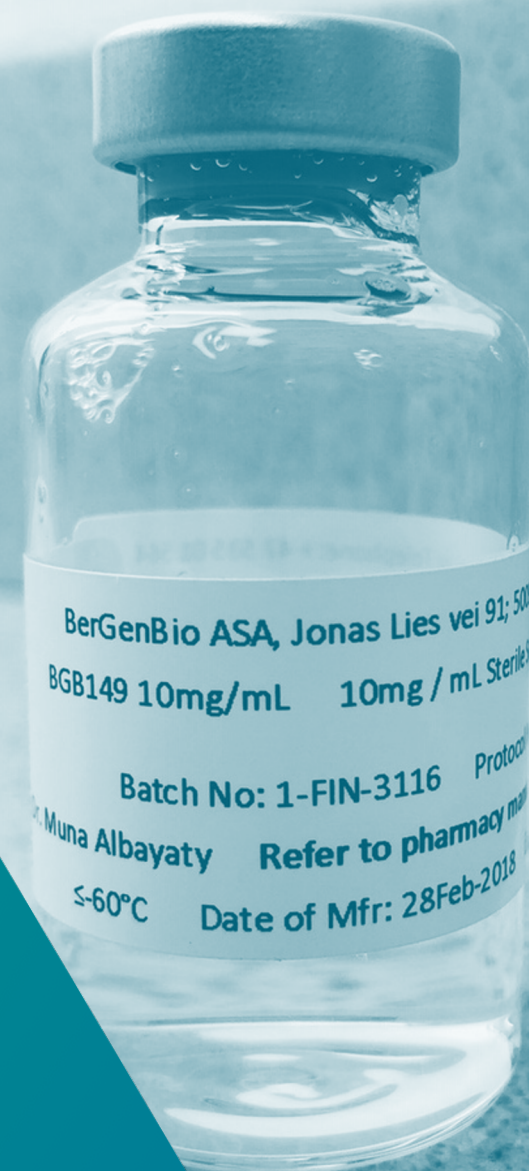
Development strategy for Bemcentinib in NSCLC (ad. & Sc.)

Clinical Position	Patient Population	Concept	Development Plan – Aim for 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/A A) 6 & 12mn OS, OS (for full approval) 2° endpoints: ORR, DoR, Safety, tolerability.	<ol style="list-style-type: none"> 1. Pending data from 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

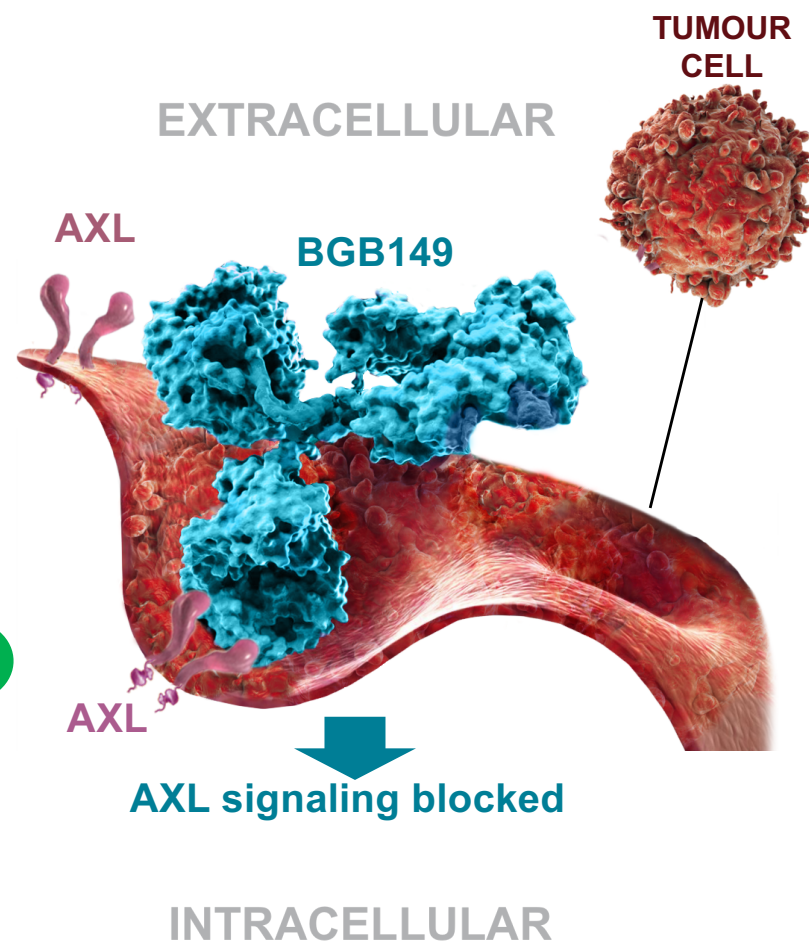
Phase Ia healthy volunteer SAD study complete

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

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

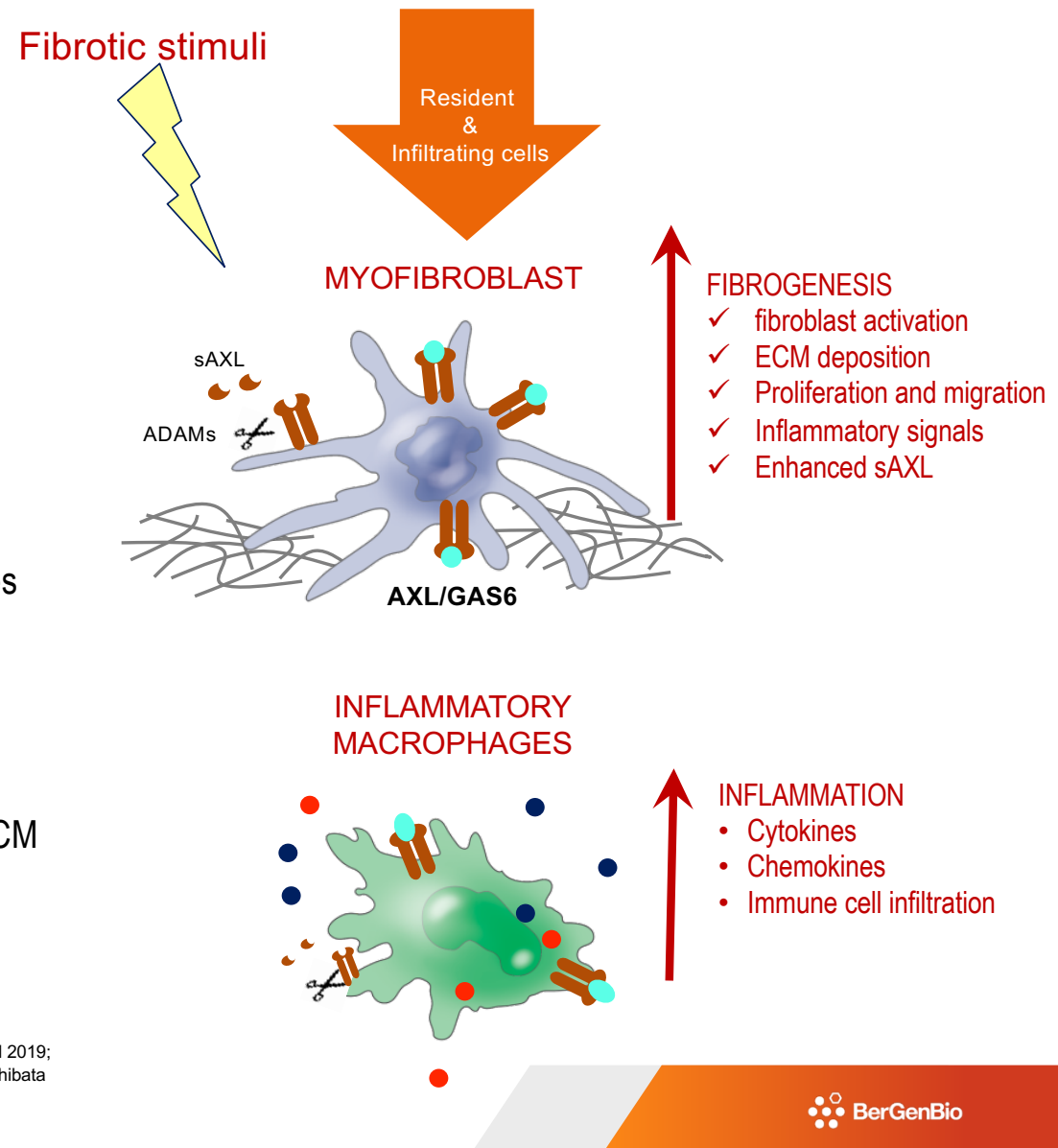
Confirmatory evidence of *in vivo* target engagement with sAXL -- stabilisation in circulation

First-in-patient trial expected in H2 2019



The role of AXL in fibrosis

- AXL Regulates and modulates key fibrogenic pathways
 - TGFb 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



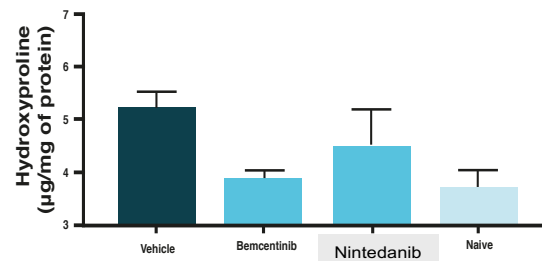
1 Gilbane ART 2015; 2 Reichl Hep. 2015; 3 Gibault ChemMed 2017; 4 Zhu AJTR 2016; Fujino Lab invest 2017, J Exp Med 2019; 6 Barcena J. Hep 2015; 7 Tutusaus A. Cell Mol Gastroenterol 2019 Hepatol. 2019; 8 Landolt L. Physiol Reports 2019; 9 Shibata J Immunology 2014; 10 BerGenBio ASA, unpublished; 11 Espindola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

AXL inhibition prevents fibrosis in a panel of preclinical models

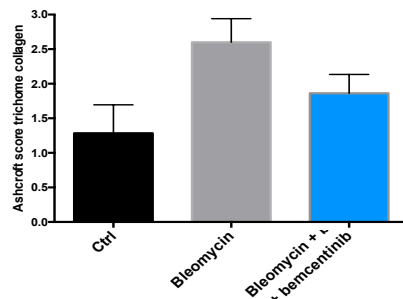
Pharmacological modulation of Axl with bemcentinib inhibits pre-clinical development of Liver (CCl₄/HighFatDiet₇), Renal (UUO₈) and Pulmonary (Asthma⁹, Bleo¹⁰, IPF¹⁰) fibrosis

Lung

Bemcentinib reduces fibrosis in a human xenograft model of IPF¹

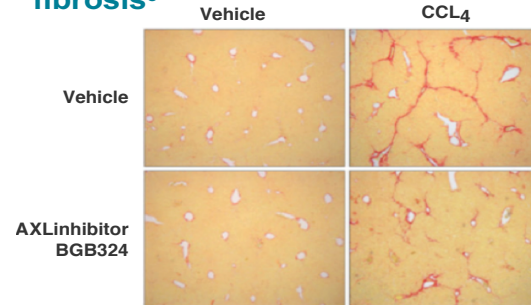


Bemcentinib reduces bleomycin induced fibrosis²

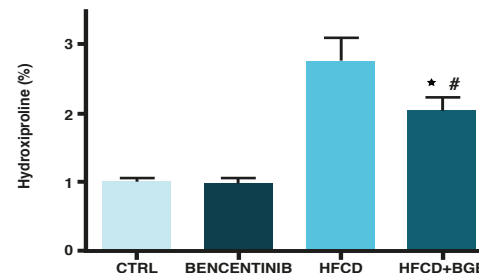


Liver

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



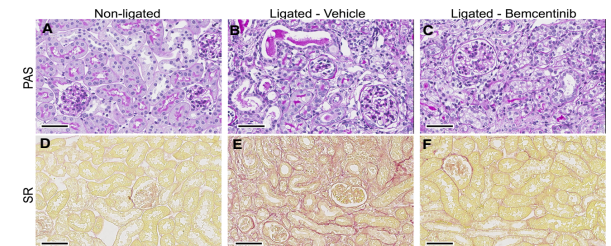
Bemcentinib reduces fibrosis in a diet induced model of NASH⁴



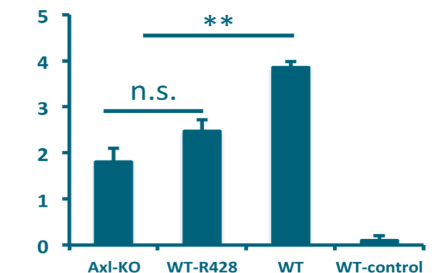
HFCD = high-fat, choline deficient diet
Leads to NASH in animal models

Kidney

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



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



Finance Report

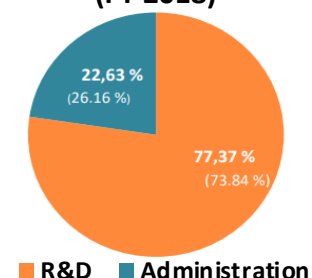
Rune Skeie - CFO



Key financial figures Q4 and full year 2019

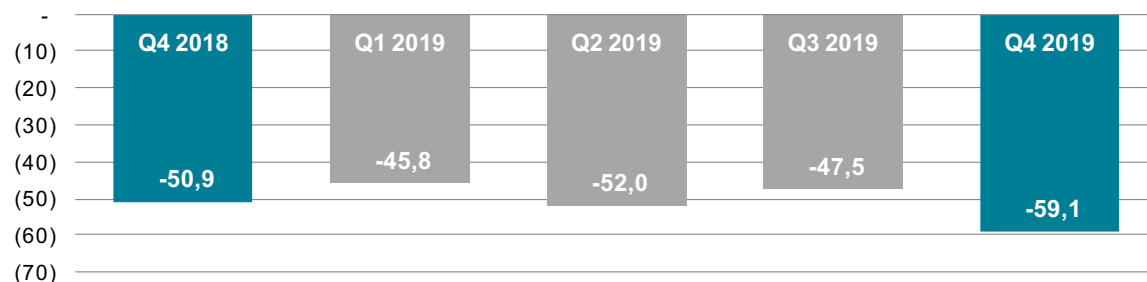
(NOK million)	Q4 2019	Q4 2018	FY 2019	FY 2018
Operating revenues	0,2	2,3	8,9	2,3
Operating expenses	59,3	53,2	213,3	196,9
Operating profit (-loss)	-59,1	-50,9	-204,4	194,5
Profit (-loss) after tax	-57,6	-51,1	-199,3	-191,7
Basic and diluted earnings (loss) per share (NOK)	-0,94	-0,93	-3,43	-3,60
Net cash flow in the period	-35,9	-37,8	-106,8	-9,9
Cash position end of period	253,6	360,4	253,6	360,4

Operating expenses FY 2019
(FY 2018)



■ R&D ■ Administration

Operating profit (-loss) million NOK

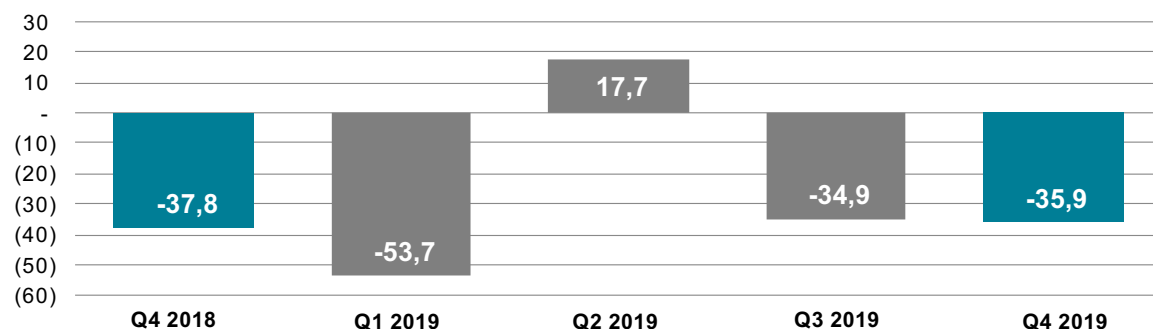


Increase in operation expenses due to expansion in clinical trials activity and as YE provisions

- Well managed overhead costs
- 77,37 % of operating expenses FY 2019 (FY 2018: 73.84 %) attributable to Research & Development activities.

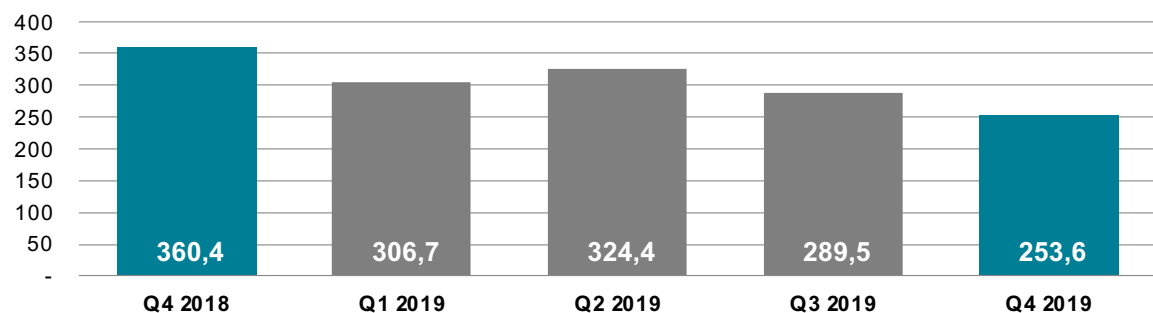
Cash flow and cash position

Cash flow (million NOK)



- Q4'19: variance between cash flow and operation loss:
 - NOK10.4m received from grants
 - NOK2.5m share option expenses
 - NOK10.3m provision for YE expenses
- Quarterly average cash burn (Q418 – Q419) NOK 45.1m (USD 5.2m)

Cash position (million NOK)



- Cash position YE 2019 NOK 253.6 m (USD 28.9m)
- Private Placement January 2020
 - Additional cash NOK 219.9m (USD 24.0m).
 - NOK 98.5m received by 10 February
 - NOK 121.3m subject to EGM approval (expected 25 February)

Cash Position strengthen following successful private placement

- NOK 220 million (USD 24 million) raised in gross proceeds through a private placement in January 2020 of 12,215,318 new shares at NOK 18 per share
 - The private placement was oversubscribed and attracted strong interest from existing shareholders and new institutional investors in Norway and internationally
 - Repair Offering of up to 1,500,000 new shares proposed to raise additional gross proceeds of approx. NOK 27 million (USD 3 million)
- Use of proceeds:
 - Execution of ongoing clinical trials of bemcentinib (2L AML and 2L NSCLC) and tilvestamab (phase Ib)
 - Manufacturing development activities for bemcentinib
 - General corporate purposes
- Current cash resources are expected to be sufficient to reach data read-out from ongoing trials in during 2020.

Private Placement and subsequent repair offering

- Subscription of 12,215,318 shares (20%) received and shares allocated.
- **Tranche 1 (completed):**
 - 5,475,136 shares issued and gross proceeds NOK 98.5 million received.
 - Total number of shares after Tranche 1: 66,551,726
- **Tranche 2 (expected to complete 25 February):**
 - Issue of 6,240,182 shares, subject to EGM approval (20 February 2020).
 - Gross proceeds NOK 121.3 million.
- **Extraordinary General Meeting 20 February 17:00 CET**
 - Place: Møllendalsbakken 9, Bergen.
 - Registration and Proxy form at: www.bergenbio.com/investors/general-meetings/
 - Agenda:
 - Approval of Tranche 2
 - Approval of subsequent repair offering
- **Subsequent Repair offering**
 - Issue up to 1,500,000 shares
 - Directed to shareholders at 29 January who did not participate in the Private Placement 29 January.
 - Shareholders at 29 January (reg. 31 January) will receive 0.045 subscription rights per share as of 29 Jan.
 - Completion of subsequent repair offering is subject to EGM approval, approved Prospectus and share price development.

Expected news flow 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: 2L IO+CHEMO refractory interim efficacy & mPFS combination with pembrolizumab
tivestamab	Healthy volunteers Phase Ia SAD study		Phase 1b/2a patient study initiate

BerGenBio – Investment Highlights

Pipeline of first-in-class anti AXL therapeutics: AXL is an increasingly validated novel target for aggressive cancers

Bemcentinib, first in class, selective, oral AXL inhibitor: once-a-day pill

Bemcentinib: attractive clinical profile targeting two large unmet medical needs: 2L AML and 2L NSCLC

Strategic options include go to market in select indications and significant attractive high value partnering opportunities

Proprietary CDx methods in parallel development for patient selection and personalised medicine approach for reimbursement

Experienced management team

Cash and Cash Equivalents at end of Q4'19 + PIPE funding Jan '20 NOK 470m.

Analyst coverage



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Link to reports from Trinity Delta:

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

Thank you

