AXL inhibitors for aggressive disease

SEB Annual Healthcare Seminar

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Oslo Børs: BGBIO

www.bergenbio.com

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BerGenBio Corporate Overview

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 aggressive diseases

2 selective AXL inhibitors in clinical development

Bemcentinib (oral once a day pill)
Tilvestamab (mAb)

Bemcentinib broad Phase II program
Monotherapy and combos with CPI, targeted & chemo

Biomarker correlation, parallel CDx development

Bemcentinib clinical data points 2020:
AML (chemo-combo)
NSCLC (KEYTRUDA combo) **COVID19** (mono)

Resourced to deliver milestones

Listed on Oslo Børs: BGBIO

Clinical trial collaborations
Merck, UKRI, and leading academic centres EU & USA

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

Cash Q3'20 NOK778m ($82m)

Introduction AXL biology

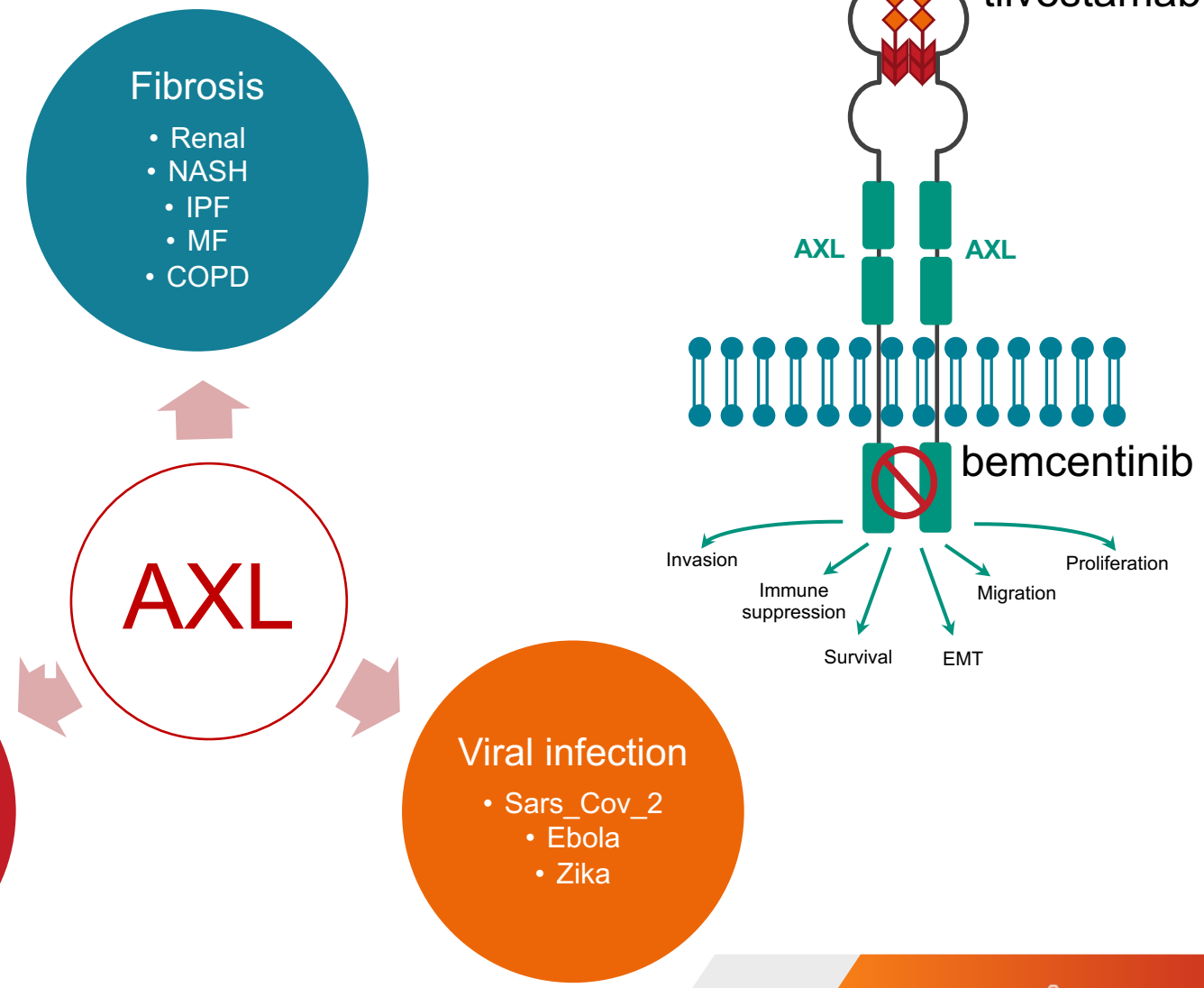
BerGenBio

Our drugs selectively inhibit AXL signaling

Axl regulates cellular plasticity implicated in fibrotic pathologies e.g. EMT, EndMT, Macrophage polarity

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



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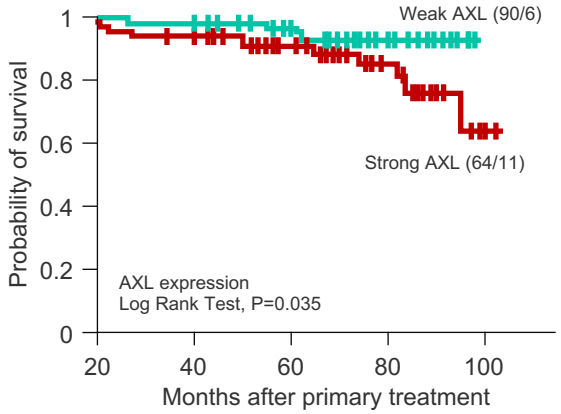
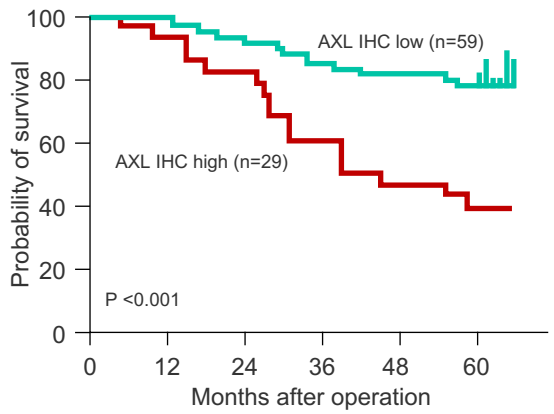
AXL is an independent negative prognostic factor in a broad variety of cancers

Strong AXL expression correlates with poor survival rate

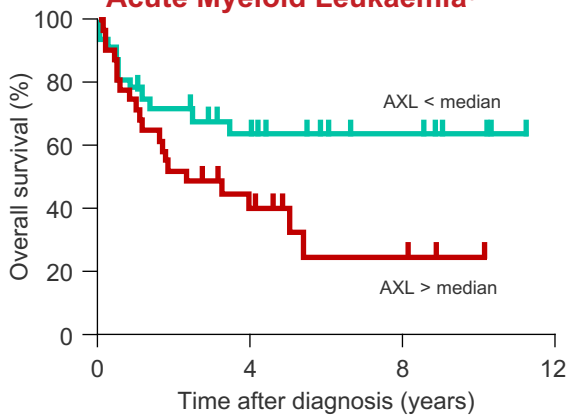
Breast carcinoma[1]



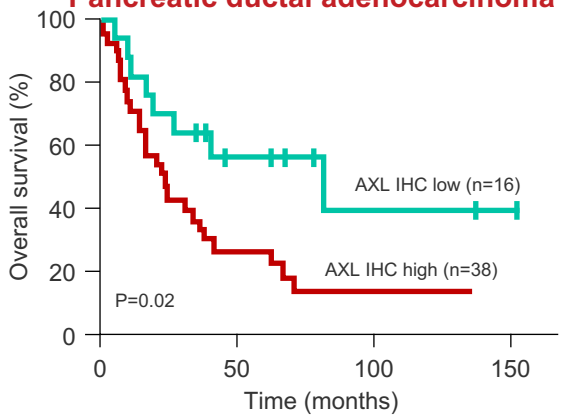
Lung adenocarcinoma (NSCLC)[2]



Acute Myeloid Leukaemia[3]



Pancreatic ductal adenocarcinoma[4]



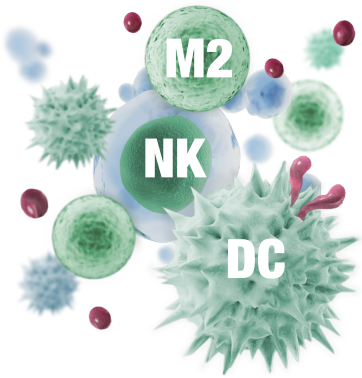
Broad evidence of AXL linked with poor prognosis[5]

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

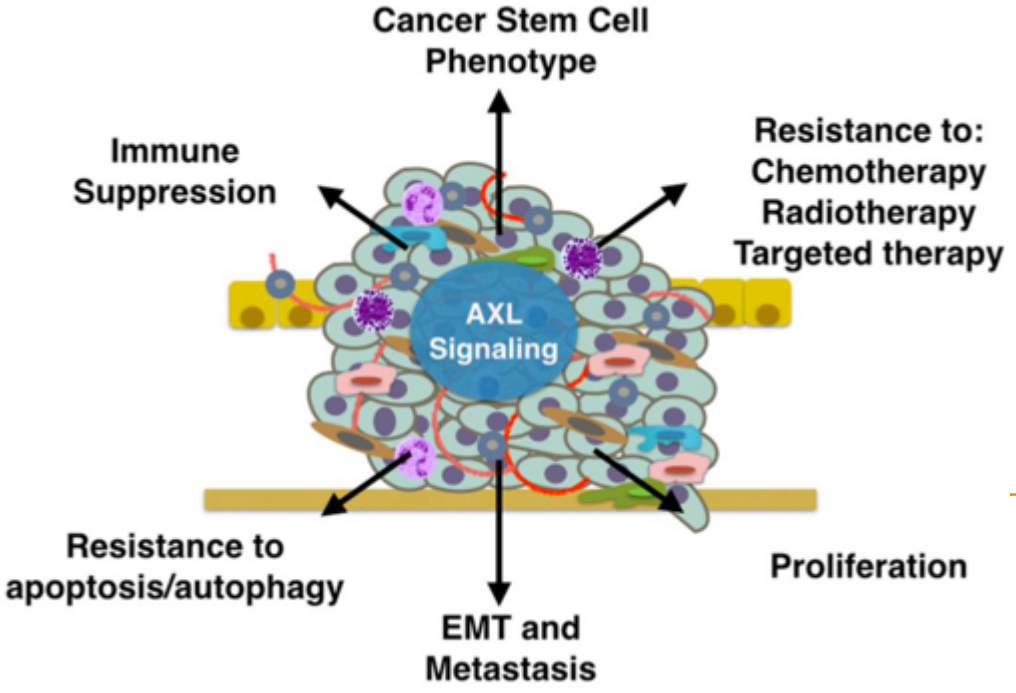
AXL is a key survival mechanism ‘hijacked’ by aggressive cancers and drives drug resistance, immune-suppression & metastasis

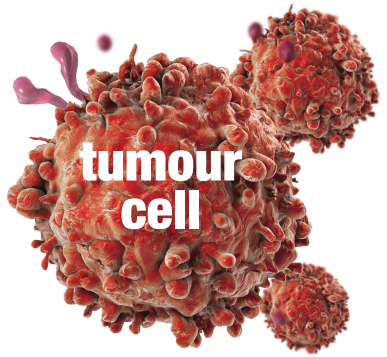
very low expression under healthy physiological conditions → overexpressed in response to hypoxia, inflammation, cellular stress & drug treatment → overexpression correlates with worse prognosis in most cancers



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





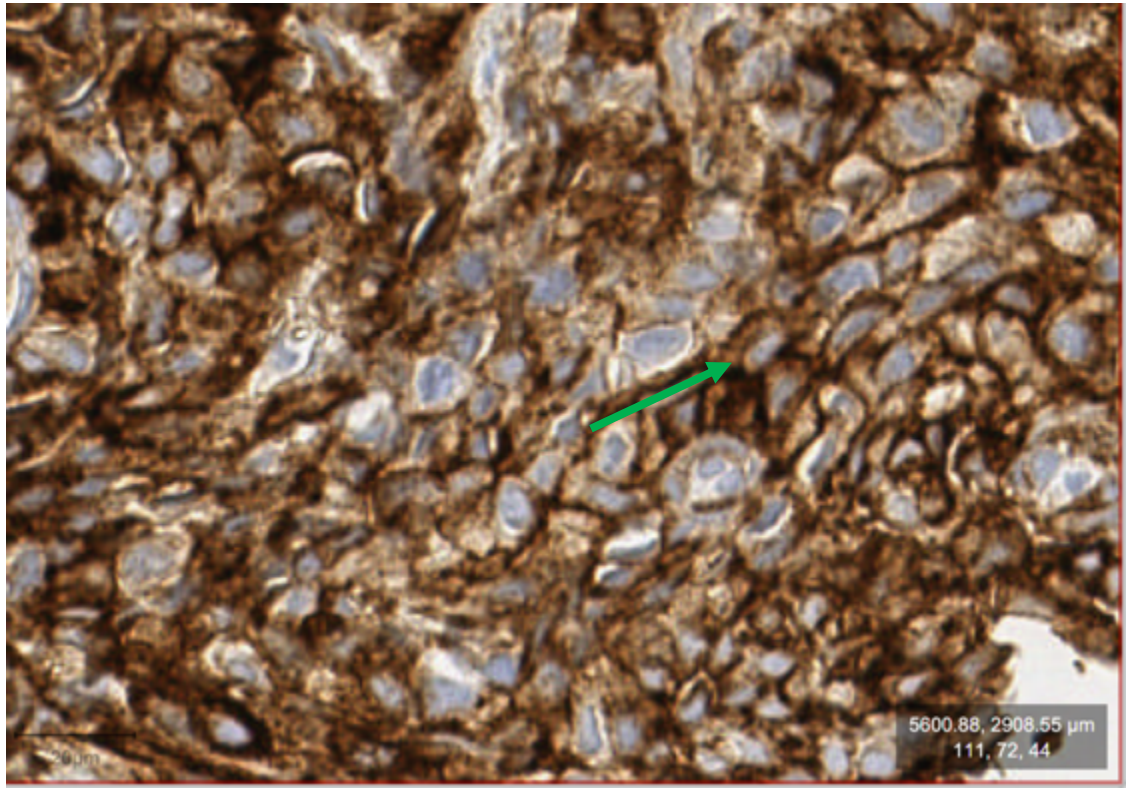
AXL increases on the tumor cell and causes cancer escape and survival

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

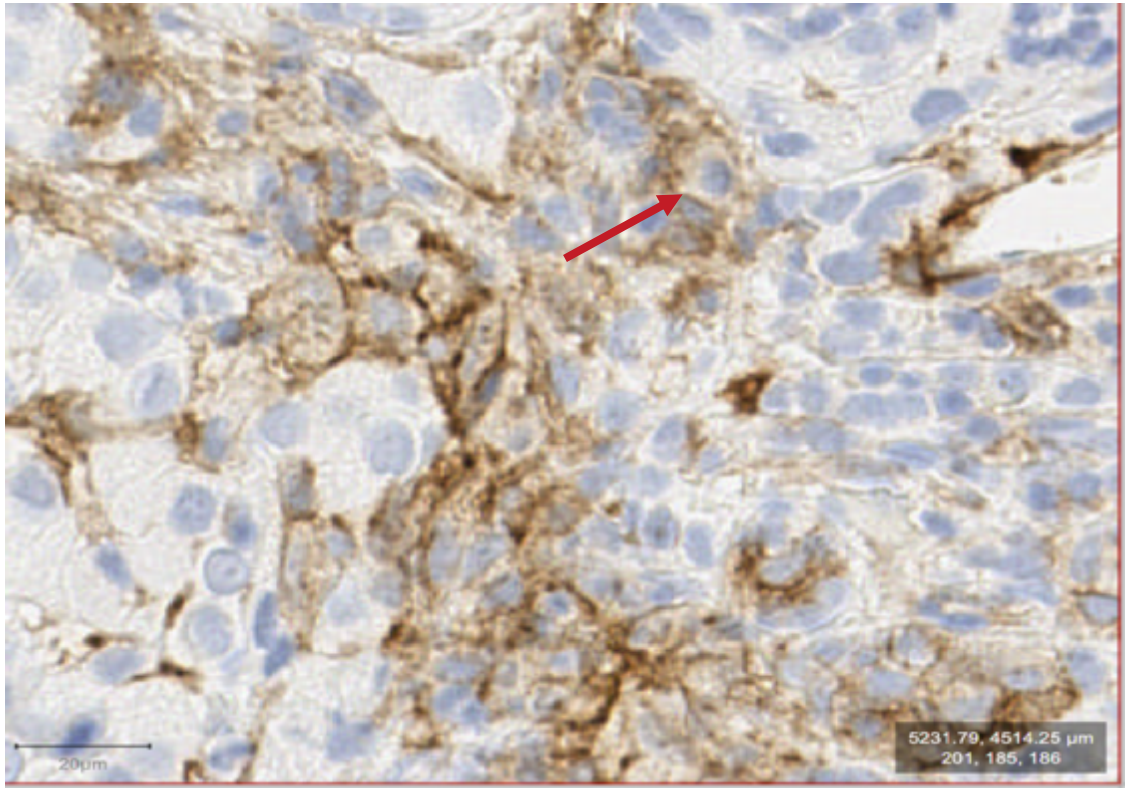
DC- dendritic cells Treg – Regulatory T Cell

Composite AXL score (cAXL) - status defined by presence of AXL on membranes of tumor + immune cells

Example of high AXL expression on tumour cells: cAXL status of this patient is positive



Example of tumour with a high number of AXL positive immune cells: cAXL status of this patient is positive



- Arrows directed at examples of positively-stained **tumour** and **immune** cell, respectively
- Both patients experienced significant tumour shrinkage on bemcentinib + pembrolizumab treatment combination

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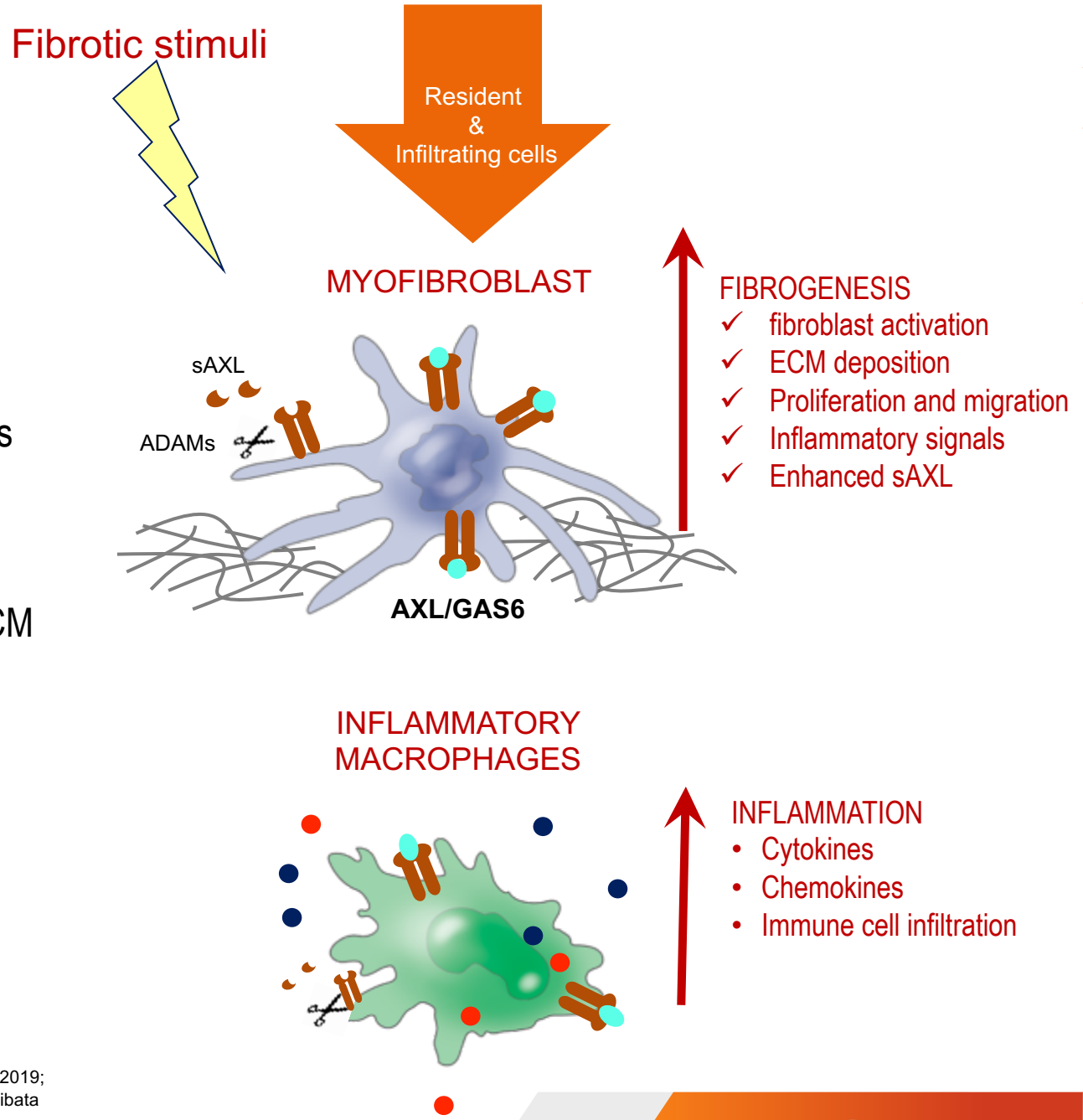
The role of AXL in fibrosis

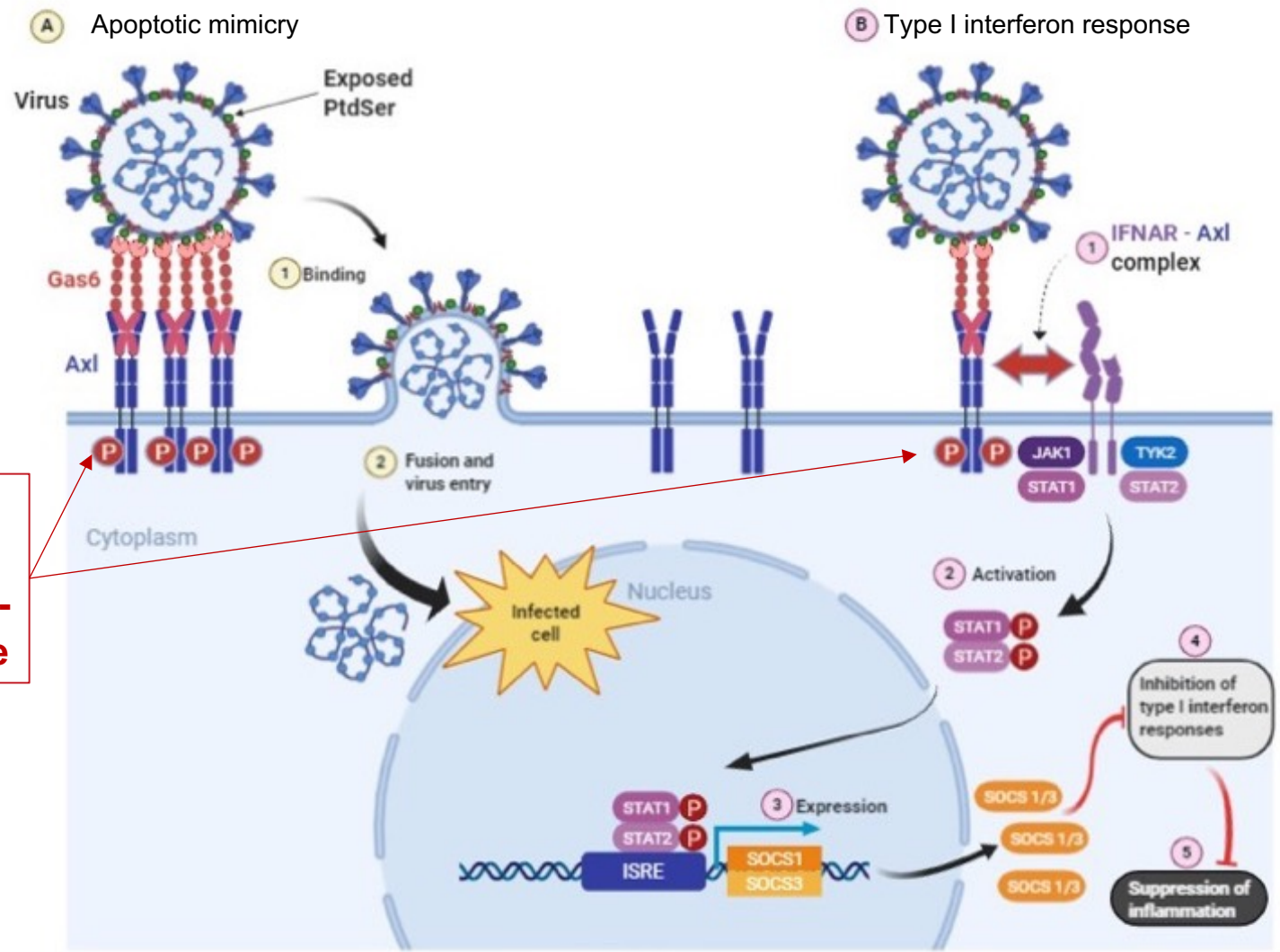
- AXL Regulates and modulates key fibrogenic pathways
  - TGFb signaling[1,2]
  - Mechanosensing Hippo pathway[3]
  - Peroxisome proliferator-activated receptor[4]
- Axl regulates cellular plasticity implicated in fibrotic pathologies e.g. EMT, EndMT, Macrophage polarity
- AXL is a negative regulator 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 fibrosis development:
  - Liver ($CCl4_6$/$HighFatDiet_7$),
  - Renal (UUO_8)
  - Pulmonary (Asthma[9], Bleo[10], IPF[10]) / COPD



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 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”.



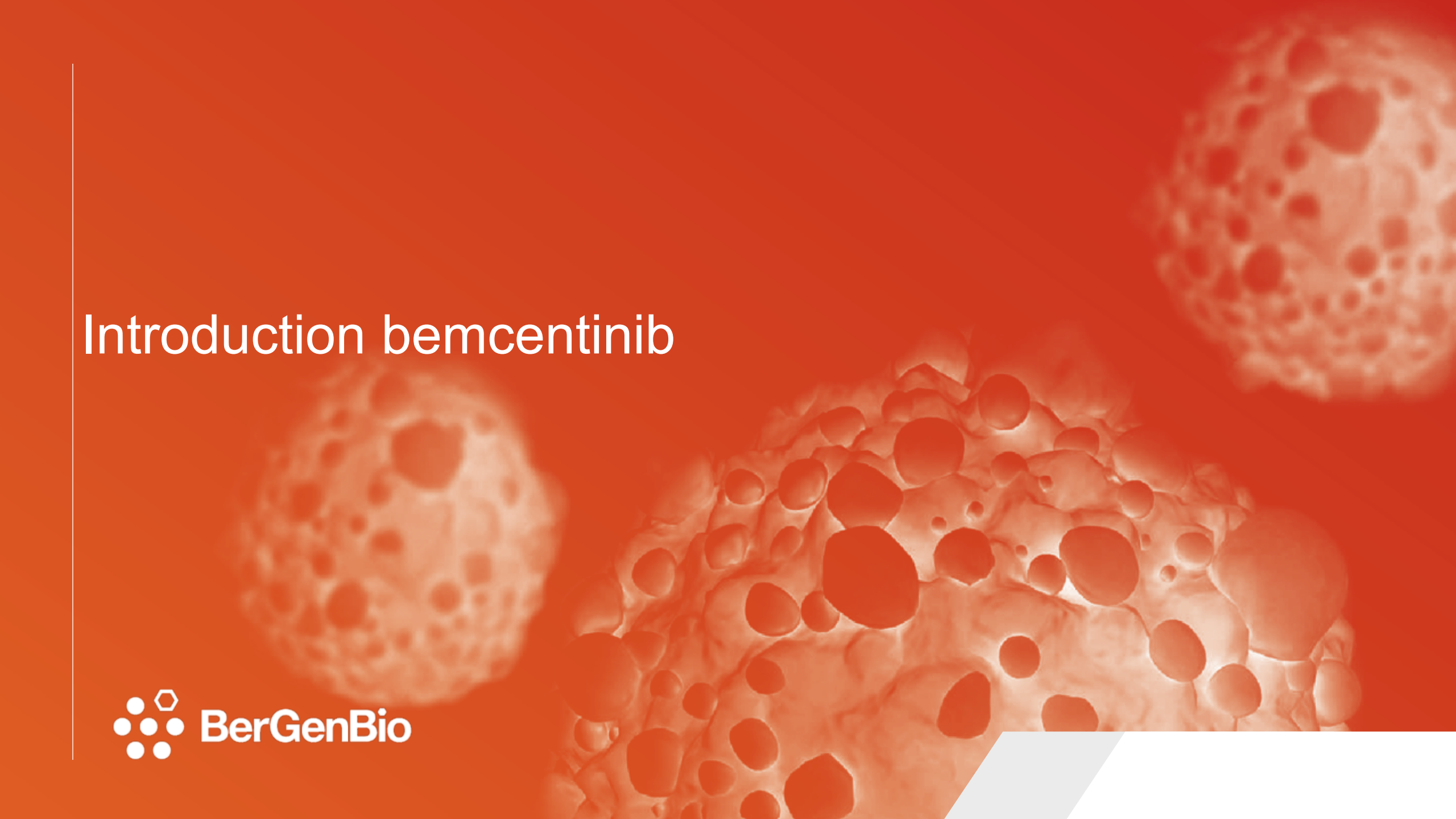
Viral-mediated AXL receptor activation dampens type I interferon responses, a key anti-viral defence mechanism for all cells

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

Bemcentinib potently inhibits SARS-CoV-2 infection of cells.[1]

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

Introduction bemcentinib



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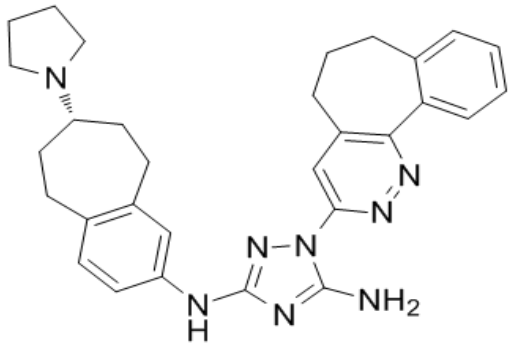
Bemcentinib, a first-in-class, potent, oral, highly selective AXL inhibitor



- ✓ Nanomolar in vitro potency (IC_{50} = 14 nM)
- ✓ Uniquely selective for AXL
  - ✓ 50-100 fold selective *cf.* TAM kinases

- ✓ Manufacturing at increased scale for late stage regulatory filing
- ✓ Size 0 100mg HPMC capsules
- ✓ 3 years stability confirmed
- ✓ Life cycle development
  - ✓ Enhanced solubility
  - ✓ Nano dispersion

- ✓ Once daily oral dosing
- ✓ Extensive Phase I & II experience
  - ✓ >350 patients
- ✓ Safety and tolerability profile supports use in combination with other drugs
- ✓ MOA is synergistic with other therapies, enhancing response

Pipeline of sponsored clinical trials

Candidate	Targeted Indication	Preclinical	Phase I	Phase II	Registrational
Bemcentinib monotherapy	>2L AML & MDS				
Bemcentinib combination with LDAC	2L AML				
Bemcentinib combination with Pembrolizumab MERCK	2L NSCLC chemo refratcory				
	2L NSCLC CPI refractory				
	2L NSCLC CPI+chemo refractory				
Bemcentinib monotherapy	Hospital COVID19 patients				
Tilvestamab (BGB149)	Phase I				

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Pipeline of Investigator Sponsored Trials (ISTs)

Candidate	Targeted Indication	Phase I	Phase II	Registrational	Sponsor
Bemcentinib	COVID-19	Monotherapy			Uni. Hospital Southampton/UKRI funded
	2L AML	Monotherapy			European MDS Cooperative Group
	2L NSMDS	Monotherapy			European MDS Cooperative Group
	Recurrent Gioblastoma	Monotherapy			Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins
	Relapse Mesothelioma	+ pembrolizumab			University of Leicester / MERCK
	1L Metastatic Melanoma	+ pembrolizumab or +Dabrafenib/Trametinib			Haukeland University Hospital
	2-4L Stage 4 NSCLC	+ docetaxel			UT Southwestern Medical Center
	1L metastatic or recurrent PDAC	+ Nab-paclitaxel +Gemcitabine +Cisplatin			UT Southwestern Medical Center

Ongoing Trial | Completed Trial

AXL inhibitor treatment of AML with bemcentinib

- FDA granted Orphan status in AML
- FDA granted Fast Track Designation in AML

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Acute Myeloid Leukaemia (AML)

Most common type of acute leukaemia in adults[1]

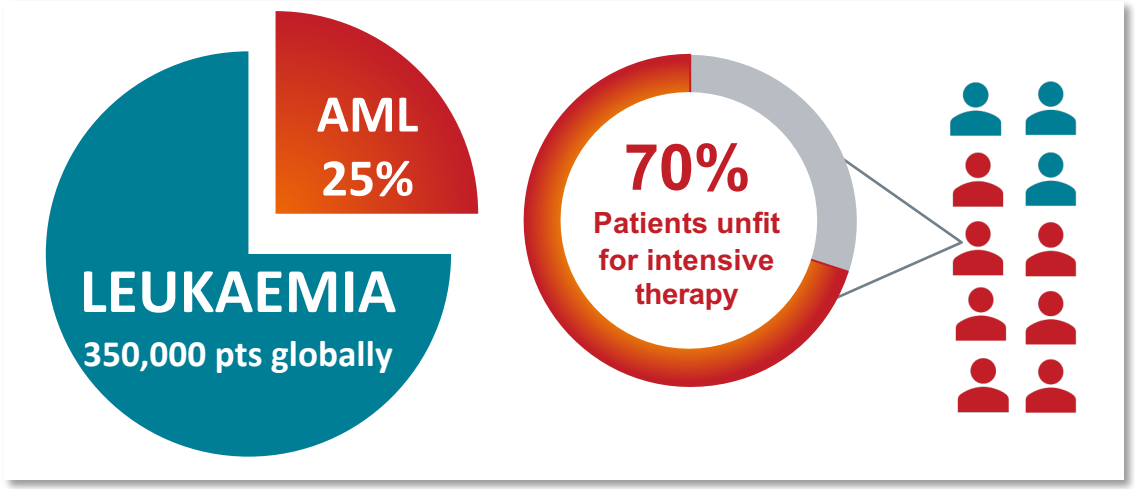
AML is a rare aggressive cancer of the blood and bone marrow characterised by difficult to treat malignancies

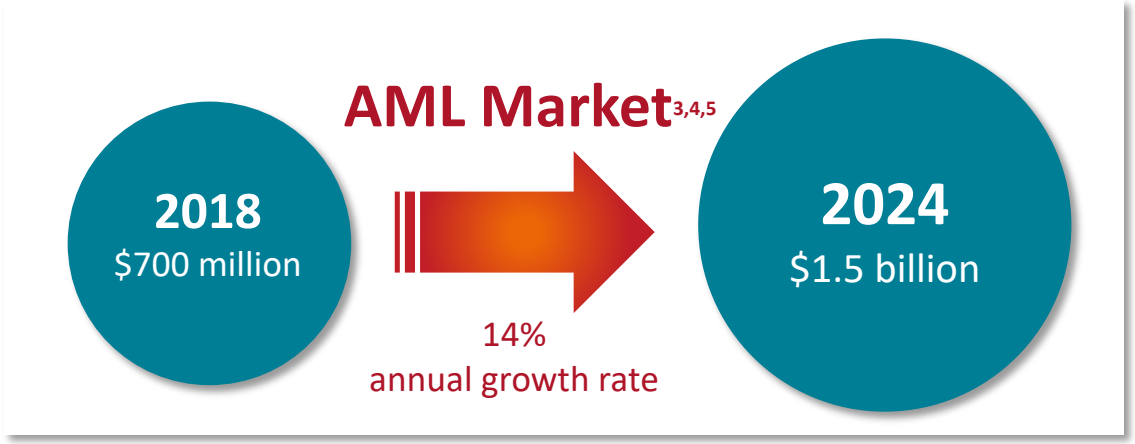
~ 20,000 new cases diagnosed and >10,000 deaths in the US in 2018[2]

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]

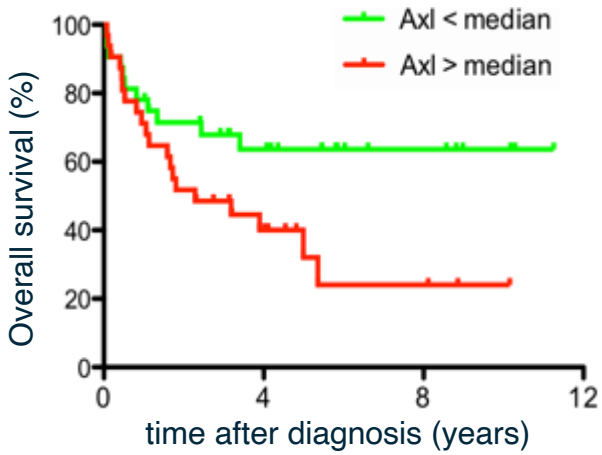




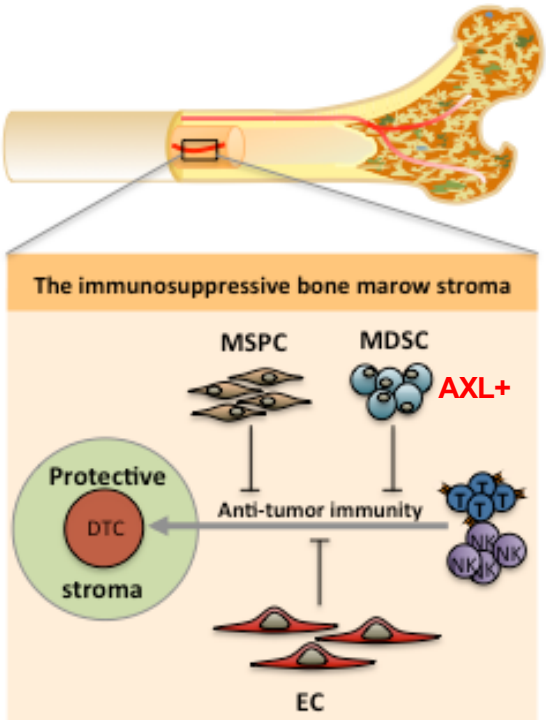
(1) Cancer.gov; (2) SEER; (3) https://www.who.int/selection_medicines/committees/expert/20/applications/AML_APL.pdf?ua=1ble
(4) https://www.cancer.net/cancer-types/leukemia-acute-myeloid-aml/statistics (5) https://www.businesswire.com/news/home/20190319005442/en/ (6) http://asheducationbook.hematologylibrary.org/content/2010/1/62.long,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

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

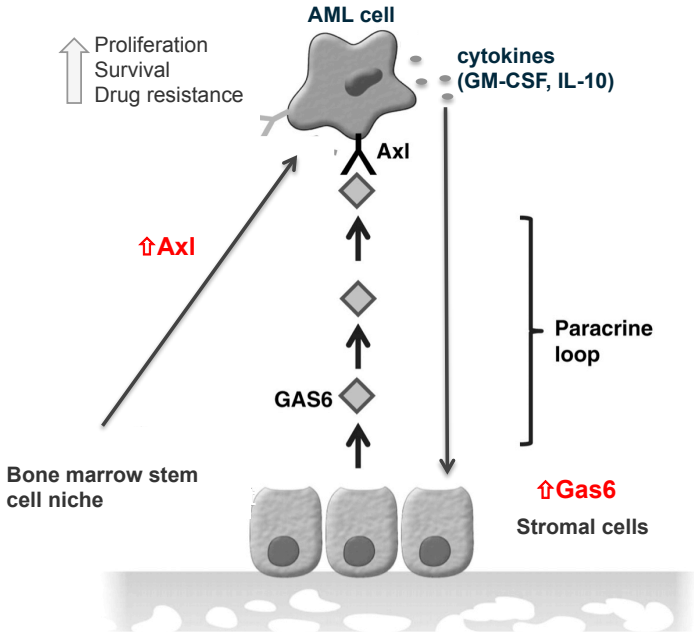
Axl is associated with therapy resistance and poor overall survival in AML patients.



Immunosuppressive niches in the bone marrow show enhanced AXL on AML, MDS progenitor and myeloid cells

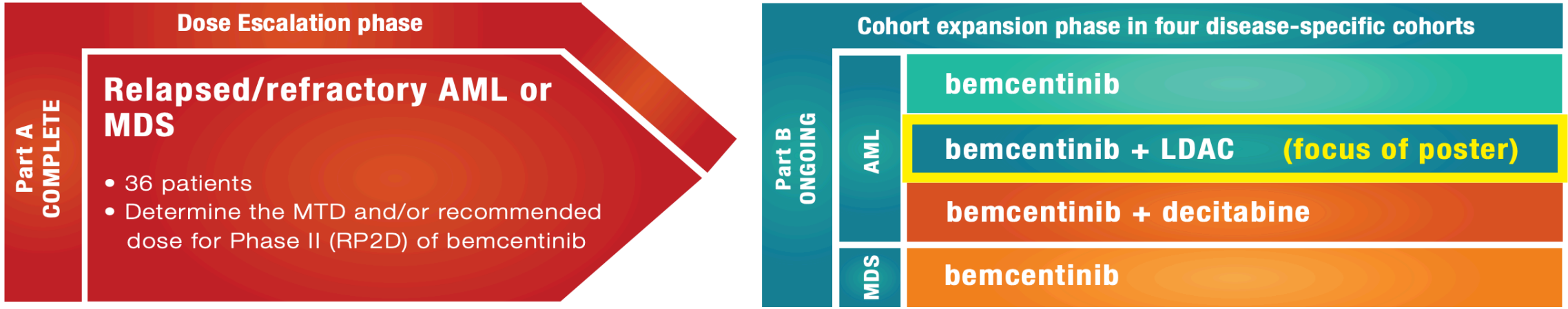


A paracrine axis between AML cells and the BM stroma establishes an immune and therapy- protective tumor cell niche



Source: Loges, 2015; Ben-Battala, 2011

Study Design
Focus on bemcentinib+LDAC combination in patients with relapsed AML



Key inclusion criteria
- Patients with AML (with exception of AML M3) who are not suitable for intensive chemotherapy as a result of advanced age or co-morbidities
- Are suitable to receive treatment with cytarabine

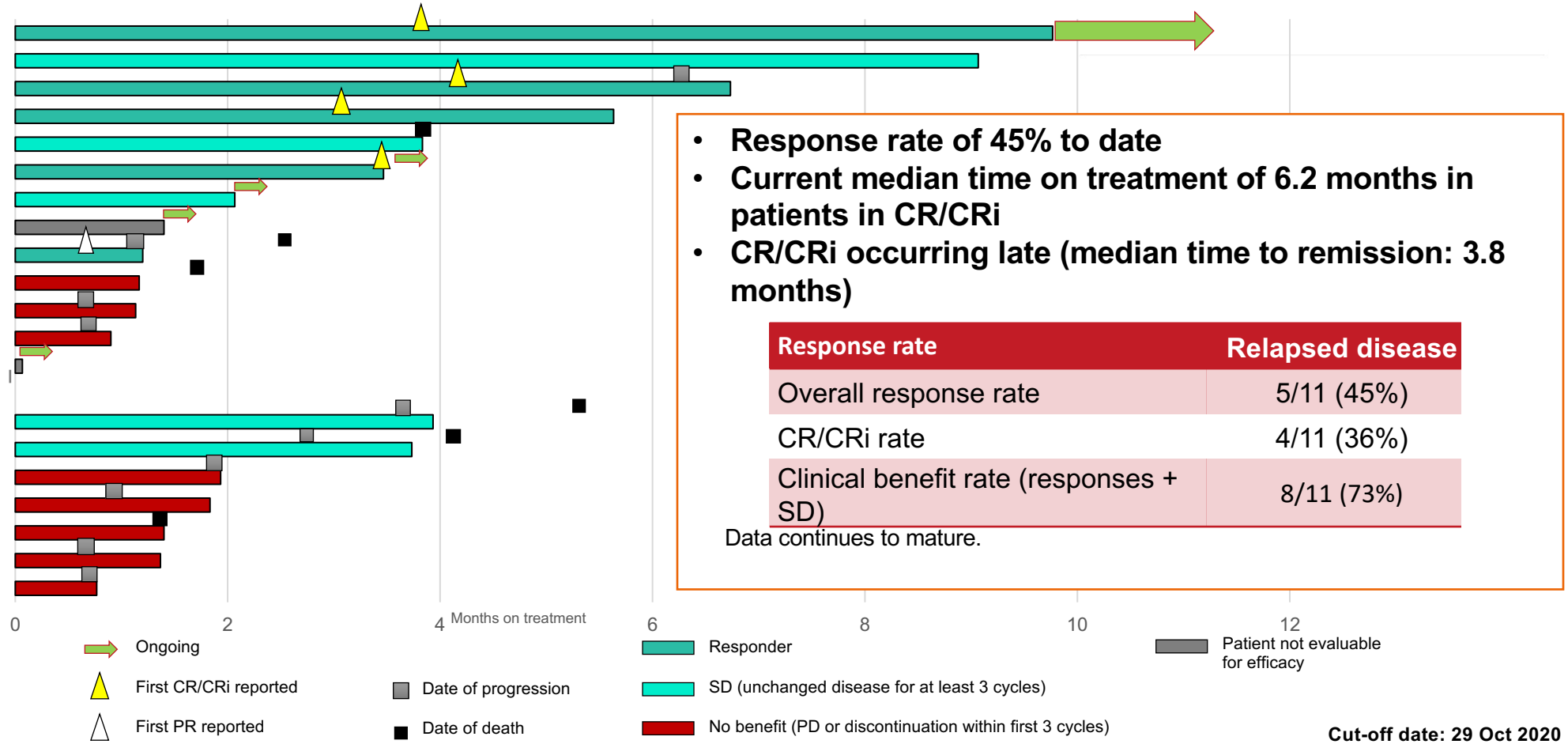
Endpoints
- ***Primary:*** safety and tolerability
- ***Secondary:*** ORR, RFS (relapse-free survival), OS, PK profile

Encouraging clinical activity observed in bemcentinib + LDAC combination in relapsed AML (Recruitment is ongoing)

	Age	Disease Type	Cytogenetic profile
Relapsed disease	78	P	F
	78	P	A
	76	P	I
	75	P	I
	78	P	I
	72	P	I
	72	P	Not done
	86	P	F
	66	S	I
	74	P	A
	74	P	A
	75	S	A
	73	P	I
	74	P	I
Refractory disease	71	S	I
	76	P	I
	81	P	A
	74	S	I
	75	P	I
	75	P	I
	75	P	A

P – de novo / primary disease
S – secondary disease

F – favorable risk
I – intermediate risk
A – poor / adverse risk



- **Response rate of 45% to date**
- **Current median time on treatment of 6.2 months in patients in CR/CRi**
- **CR/CRi occurring late (median time to remission: 3.8 months)**

Response rate	Relapsed disease
Overall response rate	5/11 (45%)
CR/CRi rate	4/11 (36%)
Clinical benefit rate (responses + SD)	8/11 (73%)

Data continues to mature.

Cut-off date: 29 Oct 2020

Response assessed according to IWG revised recommendations in reporting AML (Cheson, et al. 2003)

Efficacy-evaluable: subjects completed 1 cycle of treatment and have bone marrow blast count at screening and at Cycle 2 or after

AXL inhibitor treatment of MDS with bemcentinib

BerGenBio

Rationale for targeting AXL in MDS with bemcentinib

1. Patients failing HMA still have a dismal outcome with median survivals of less than 6 months[1,2] and very limited available treatment options

2. AXL mediates proliferation and survival of leukemic cells, innate immune cell suppression and resistance to chemotherapeutic agents[3]

3. Bemcentinib (BEM) is a selective small molecule inhibitor of AXL, a surface membrane protein kinase receptor overexpressed on leukemic (stem) cells[3]

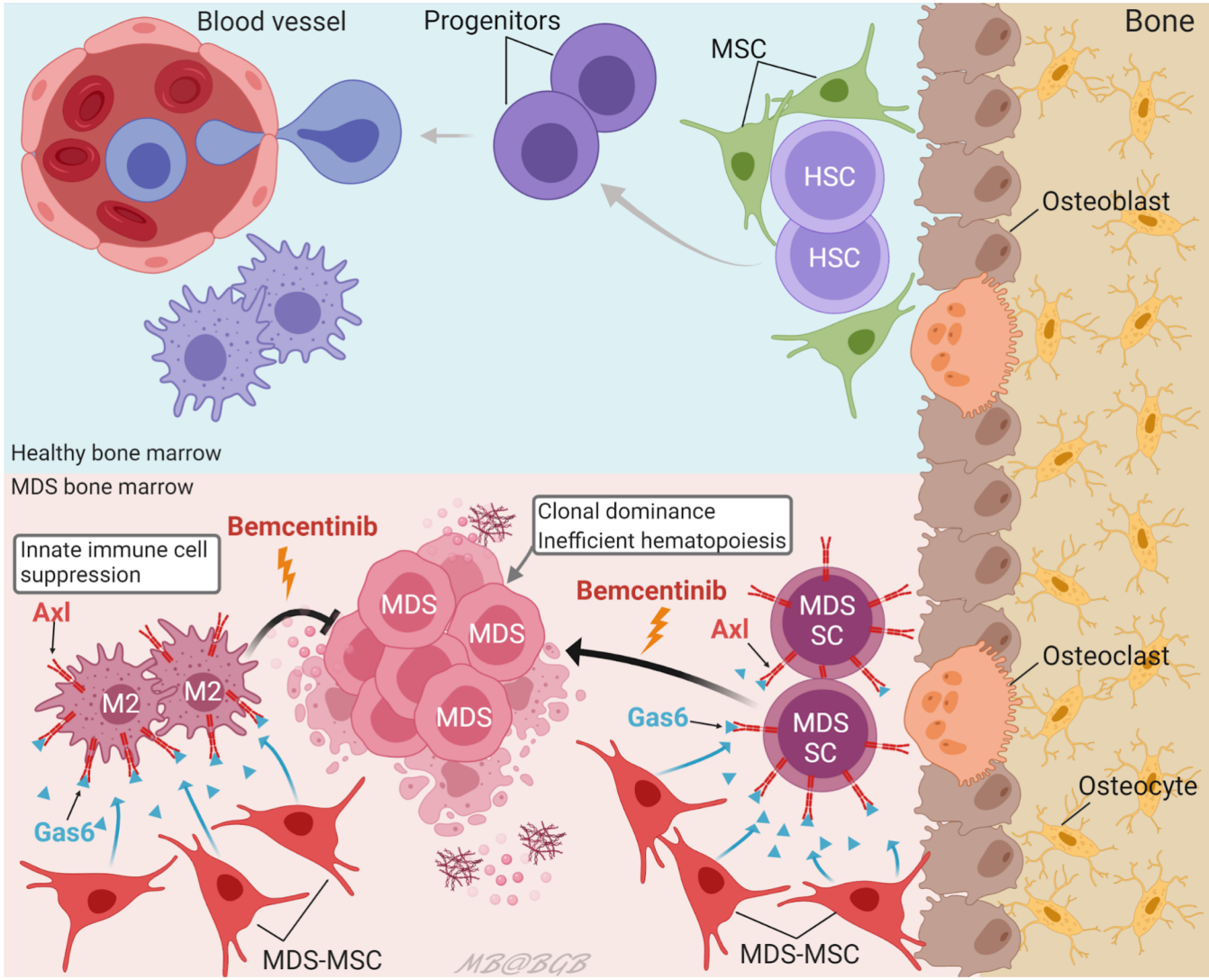


Figure: BerGenBio, Bergen, Norway

Abbreviations: HMA: Hypomethylating agents, AXL: Axl Receptor Tyrosine Kinase Gas6: Growth arrest-specific Protein 6, SC: stem cell, MSC: mesenchymal stem cells

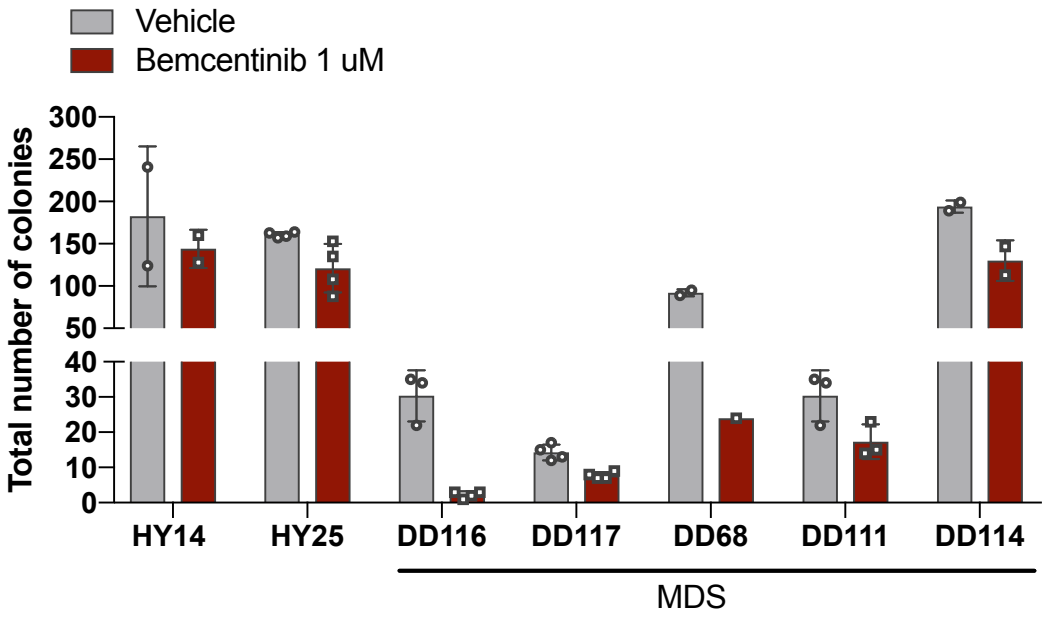
1 Prebet *et al.* American Society of Clin. Onc. (2011);
2 Komrokji et al., Clin. Lymphoma Myeloma Leuk., 2015
3 Medyouf H, Annals of Oncology, 2018, Abstract 5735

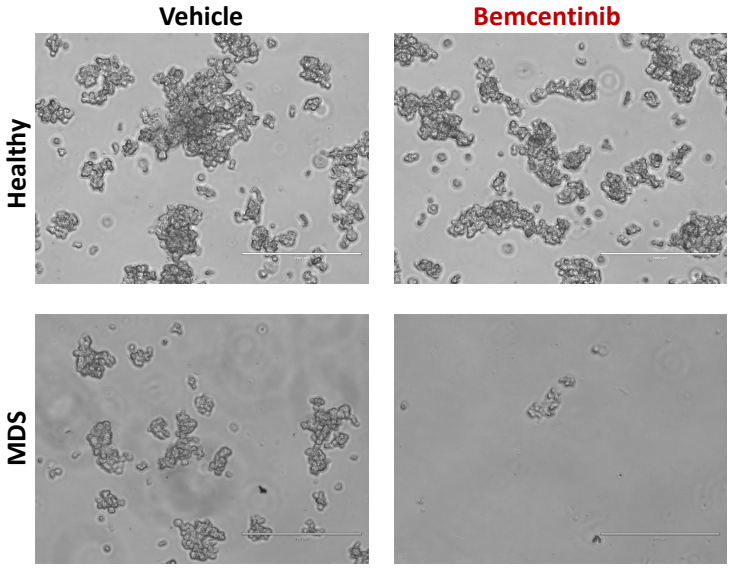
Bemcentinib: Background and Rationale

4

Blockade of the Gas6/AXL signalling axis selectively impaired MDS growth in an ex-vivo stroma-dependent co-culture setting

→ Effects were especially observed in the CD34+ MDS stem cell compartment



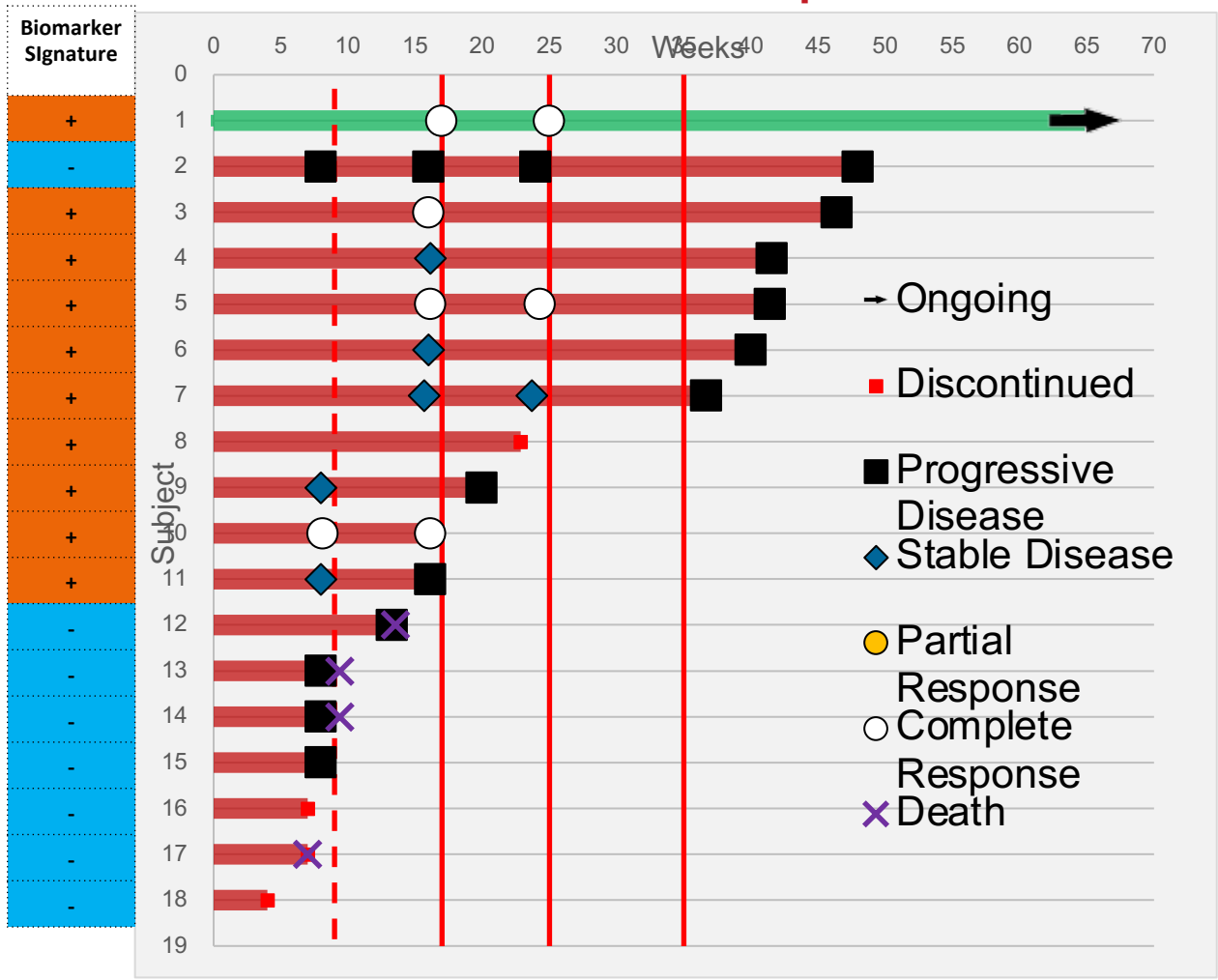


Abbreviations: HMA: Hypomethylating agents, AXL: Axl Receptor Tyrosine Kinase Gas6: Growth arrest-specific Protein 6, SC: stem cell, MSC: mesenchymal stem cells

Figures: Courtesy of Medyouf H., Frankfurt, Germany, unpublished data

Encouraging clinical activity observed with bemcentinib in relapsed MDS

Best Response	Number (%); Median [range]
ORR (CR, CRi, PR, SD) (SAF, n=46)	10 (22%)
HR-MDS (n=22)	8 (36%)
AML (n=24)	2 (8%)
CR/ CRi	
HR-MDS	4 (18%) CR:1 (4%); CRi:3 (14%)
AML	0 (0%)
PR	
HR-MDS	1 (5%)
AML	0 (0%)
SD	
HR-MDS	3 (14%)
AML	2 (8%)



- Median response duration at cut-off* in patients who have discontinued treatment: **269 days**

→ *Data continuous to mature*

- Treatment is still ongoing in **3 patients** as of the cut-off

*data cut-off June 2020

Bemcentinib clinical development in 2L Non Small Cell Lung Cancer (NSCLC)

BerGenBio

Cohort B1

Study Design

Open-label multi-center single arm phase II study

Cohort A

- Previously treated with a platinum containing chemotherapy
- CPI-naïve
- Has PD at screening

Interim Analysis
Cohort A
Stage 1

N=22 patients
(each patient has the potential for at least 24 weeks follow-up)

Final Analysis
Cohort A
Stage 2

N=48 patients
(each patient has the potential for at least 24 weeks follow-up)

Cohort B

- Previously treated with a mono therapy PD-L1 or PD-1 inhibitor
- Must have had disease control on most recent treatment
- Has PD at screening

Interim Analysis
Cohorts B
Stage 1

N=16 patients
(each patient has the potential for at least 24 weeks follow-up)

Final Analysis
Cohorts B
Stage 2

N=29 patients
(each patient has the potential for at least 24 weeks follow-up)

Cohort C

- Previously treated 1st line with a combination of checkpoint inhibitor + platinum-containing chemotherapy
- Must have had disease control on 1st line therapy
- Has PD at screening

Interim Analysis
Cohorts C
Stage 1

N=13 patients
(each patient has the potential for at least 24 weeks follow-up)

Final Analysis
Cohorts C
Stage 2

N=29 patients
(each patient has the potential for at least 24 weeks follow-up)

Cohort A

Patient Disposition and Demographics

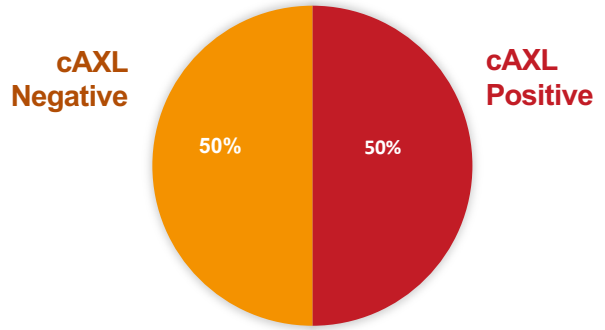
Patient disposition	N
Screened	74
Enrolled	50
Evaluable	44
Ongoing	4

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

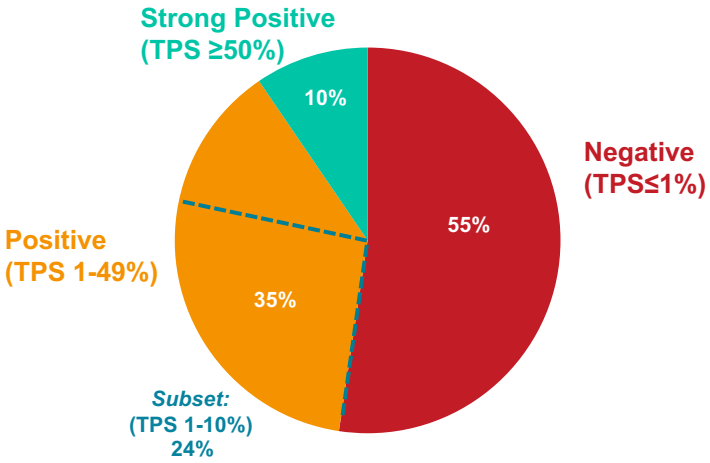
Patient demographics		N (%)
Age	Median	65
	Range	39-82
ECOG at screen	0	22 (44)
	1	28 (56)
Sex	Female	20 (40)
Smoking Status	Smoker	10 (20)
	Ex-smoker	29 (58)
	Never smoked	10 (20)
	Unknown	1 (2)

Biomarkers

cAXL status
n = 30



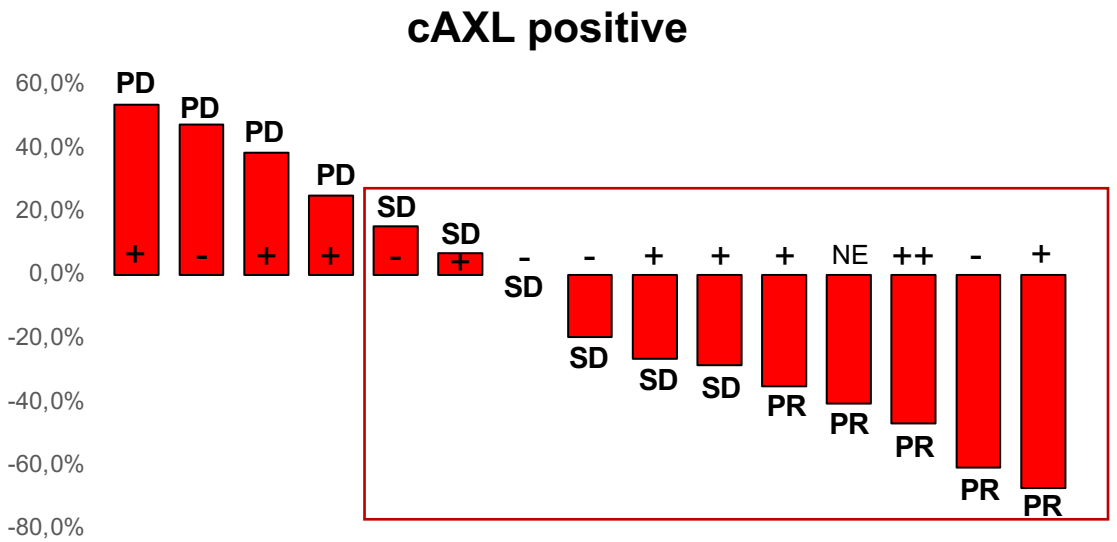
PD-L1 status
n = 37

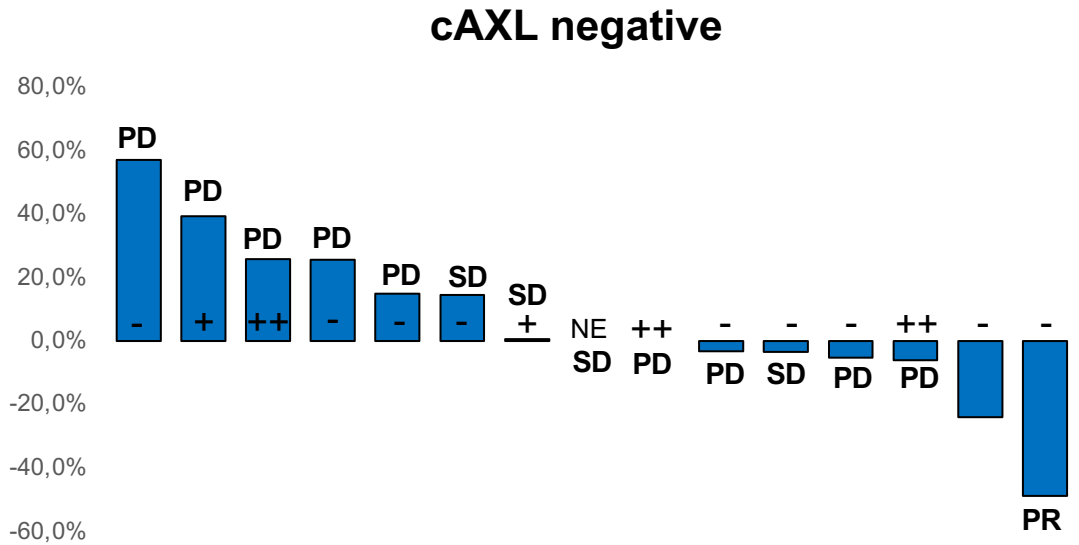


*Data cutoff:17 Apr 2020

Cohort A

Change in tumour size from baseline in cAXL
(evaluable patients only)





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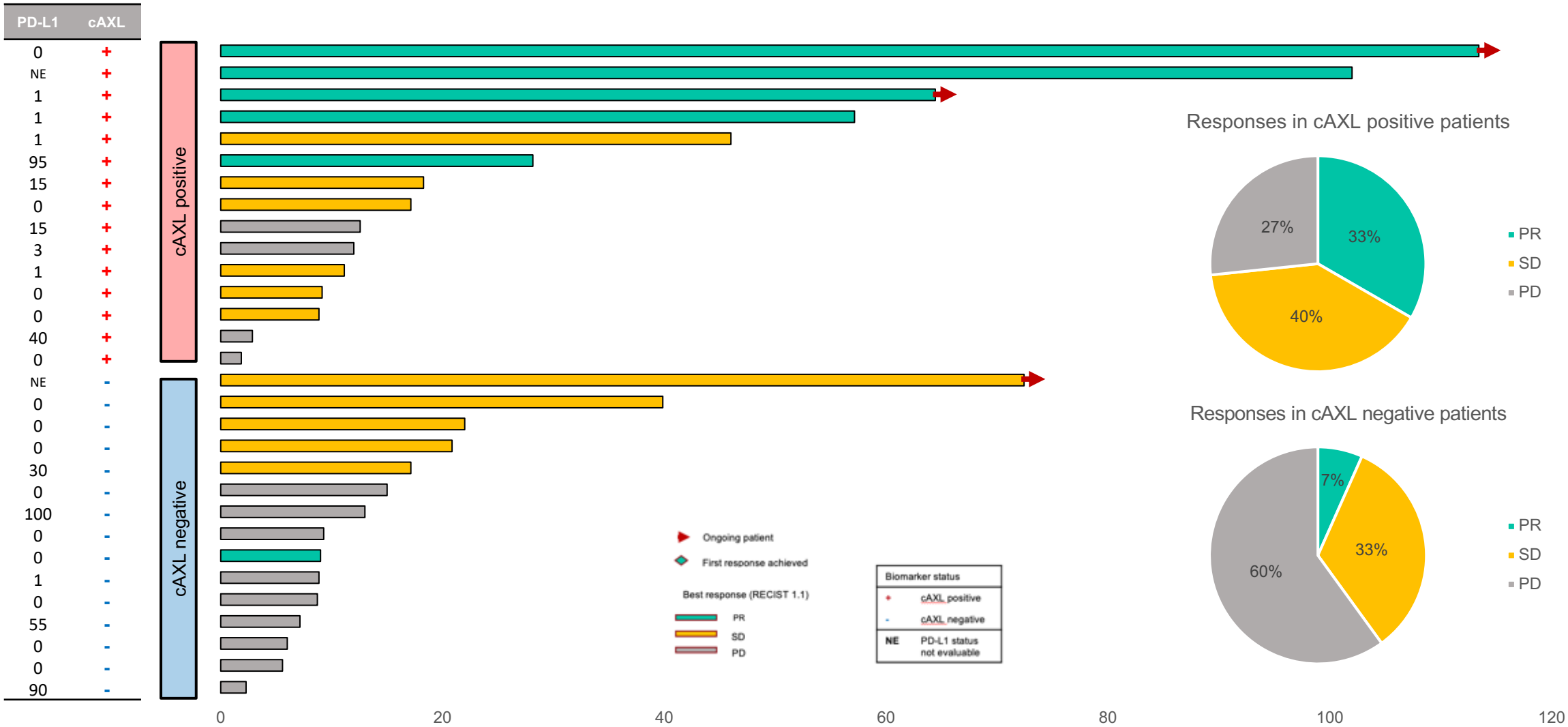
Cohort A

Time on treatment in patients evaluable for cAXL



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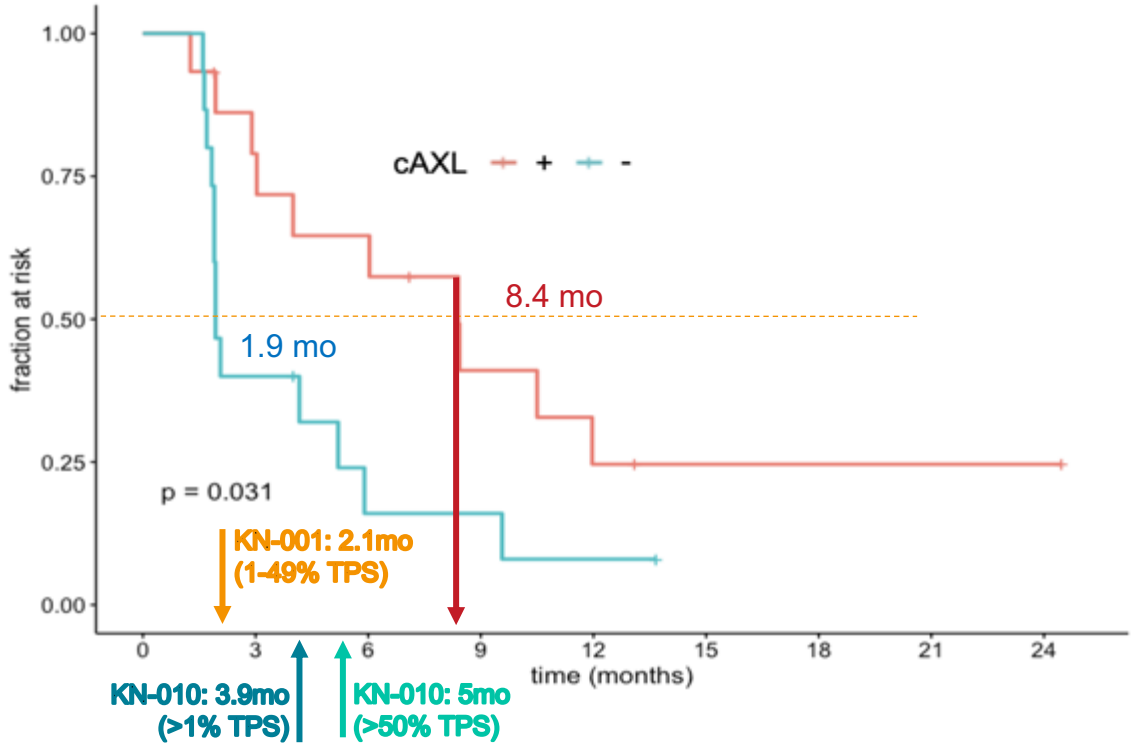
Enhanced survival in cAXL +ve patients with addition of bemcentinib to pembrolizumab

AXL is an adverse prognostic biomarker

mPFS 8.4 months in cAXL+ patients



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

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

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

Data cut-off: 17-April-2020

Source: KN-001: Garon et al NEJM 2015; KN-010: Herbst et al, Lancet 2016; CheckMate-057: Borghaei et al, NEJM 2015

Cohort B1

Bemcentinib + KEYTRUDA in CPI refractory patients

CHECK POINT INHIBITOR REFRACTORY PATIENTS: precise and specific definition

Initial clinical benefit on prior anti-PD(L)1 therapy

- Anti-PD(L)1 monoclonal antibody as **monotherapy or combination** therapy
- **Clinical benefit** (CR, PR, SD) lasting for at least **12 weeks**
- At least **2 doses** of anti-PD(L)1

Progression

- Confirmed PD (RECIST v1.1)
- Progression occurred within 12 weeks of last dose of anti-PD(L)1 therapy

IO-refractory patient enters trial

- Enrolment **within 12 weeks** of initial PD
- No administration of other therapy between last dose of anti PD-1/L1 mAb and commencement of clinical trial agent

Cohort B1

Patient Disposition and Demographics

Patient disposition	N
Screened	21
Enrolled	16
Evaluable*	15
Ongoing	3

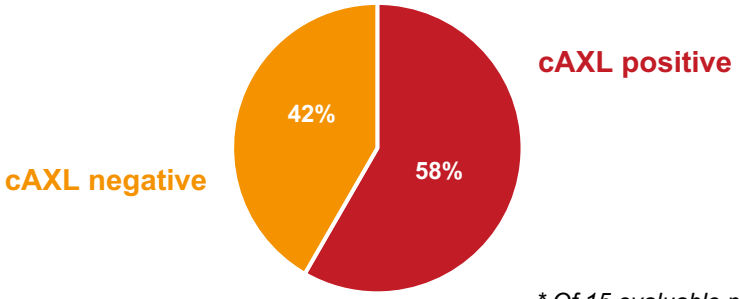
* with at least 1 post-baseline scan assessment

Disease mutations	N (%)
None	13 (81)
KRAS	2 (13)
BRAF	1 (6)

Patient demographics		N (%)
Age	Median	64,5
	Range	40-76
ECOG at screen	0	6 (38)
	1	10 (63)
Sex	Female	3 (19)
	Male	13 (81)
Smoking status	Smoker	6 (38)
	Ex-smoker	8 (50)
	Never smoked	0 (0)
	Unknown	1 (6)

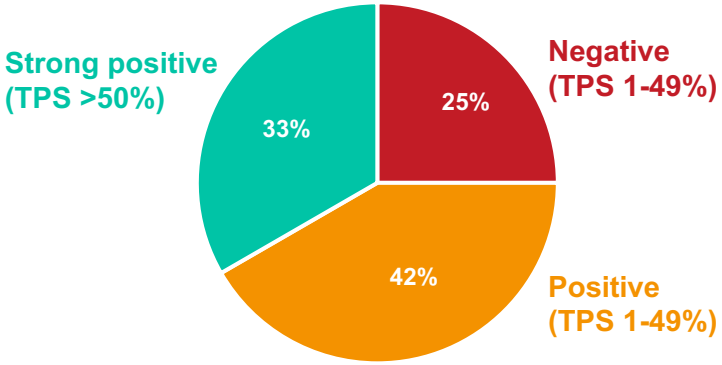
Biomarkers

cAXL status
n = 12*



** Of 15 evaluable patients, 3 not evaluable for AXL*

PD-L1 status
n = 12**



*** Of 15 evaluable patients, 3 not evaluable for PD-L1*

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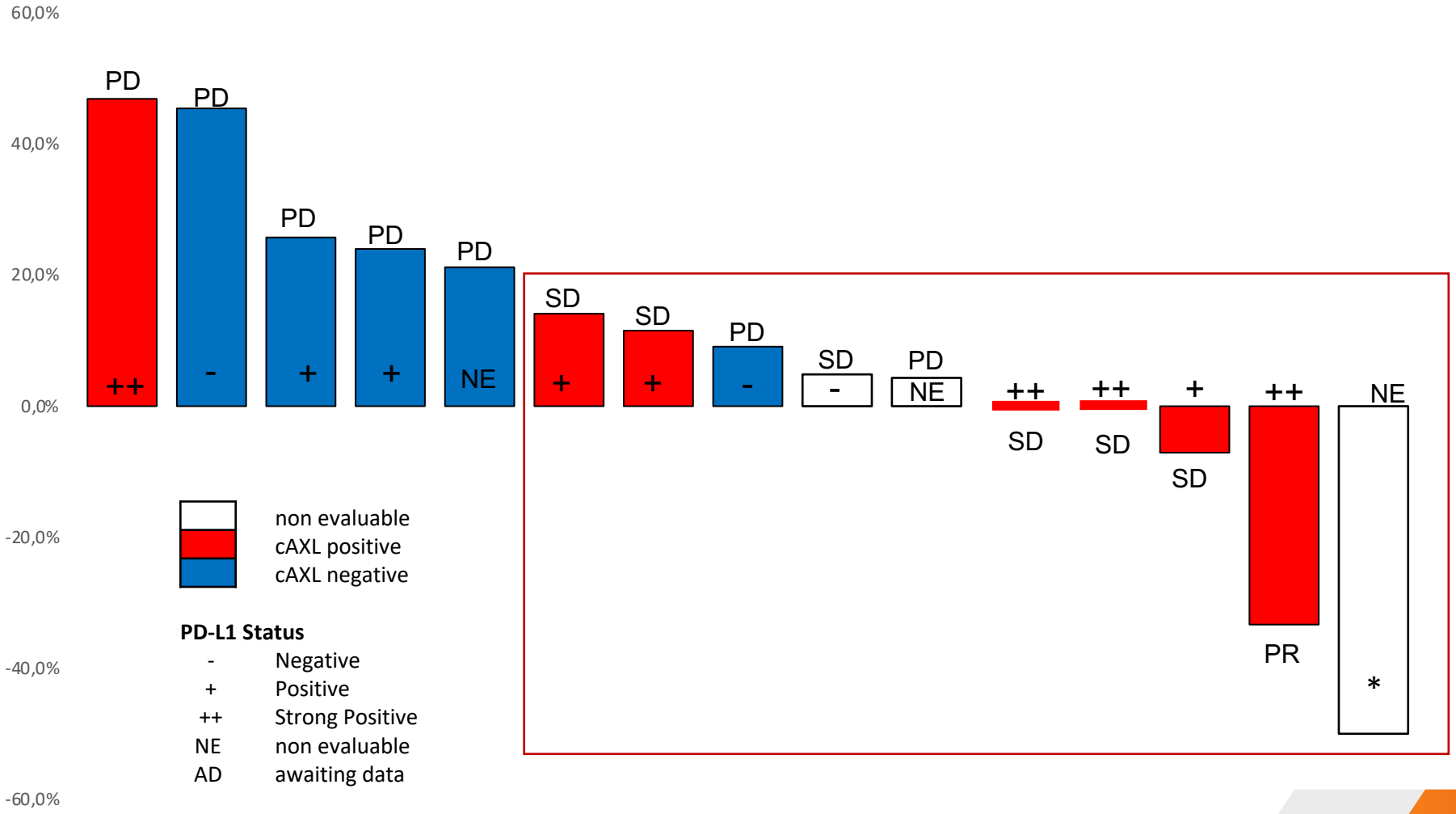
Best % change in sum of target lesions from baseline

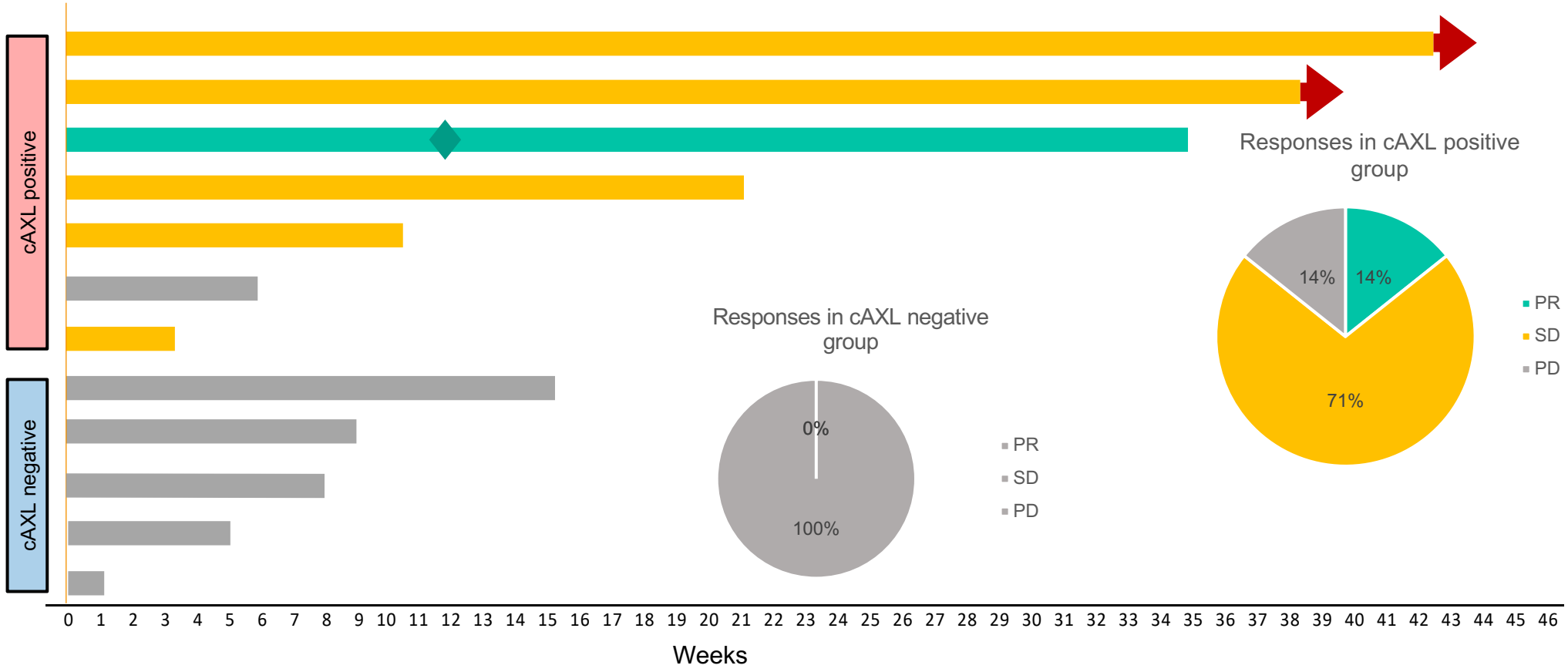


Data cut-off: 17-April-2020

Cohort B1

Time on treatment in patients evaluable for cAXL

Pre Tx	PD-L1	cAXL
1L P	20	+
2L A/O	65	+
1L A	95	+
2L P	15	+
2L N	100	+
2L P	95	+
1L I/N	2	+
2L P	35	-
2L N	30	-
2L A	0	-
2L C	NE	-
2L P/O	0	-



+ cAXL positive

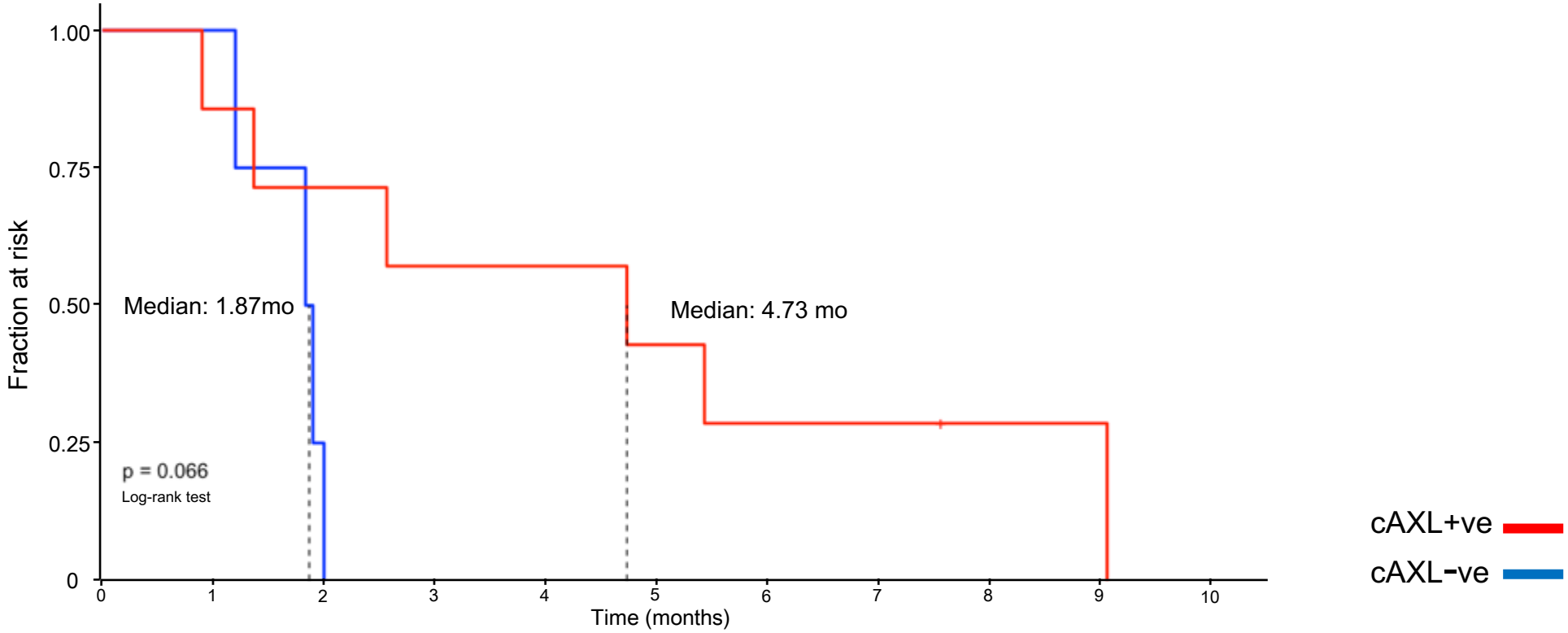
- cAXL negative

Previous immunotherapy (1 or 2L)
P: pembrolizumab; **A**: atezolizumab; **N**: nivolumab; **C**: cetrelimab; **I**: ipilumimab; **O**: other

Data cut-off: 17-April-2020

Cohort B1

mPFS improvement in cAXL +ve patients



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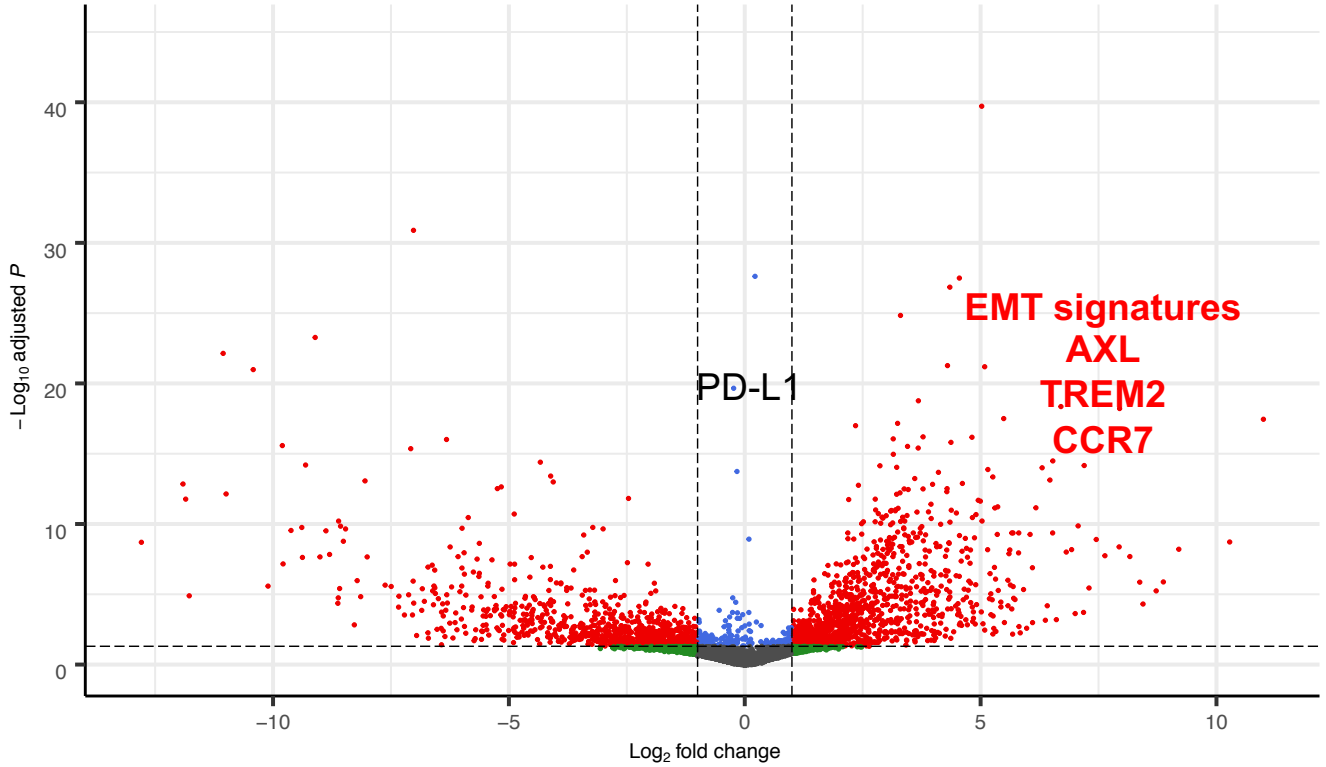
Cohort B1

Clinical translational findings

Whole tumor gene expression of Cohort B1 patients benefiting from bemcentinib-pembrolizumab



Volcano Plot: Differential gene expression analysis of patients showing benefit (n=5) vs patients with PD (n=3)

RNAseq analysis identifies gene signatures from benefiting patients:

- Increased AXL expression
- Genes associated with tumor cell EMT[1]
- Presence of TREM2+ TAMs[#,2]
- Presence of CCR7+ mregDC1[##,3]

[#] tumor-associated macrophages
[##]regulatory dendritic cells

[1]Liberzon, Cell Systems 2015;[2]Katzenelenbogen *Cell* 2020, Molgora *Cell* 2020; [3]Maier *Nature* 2020

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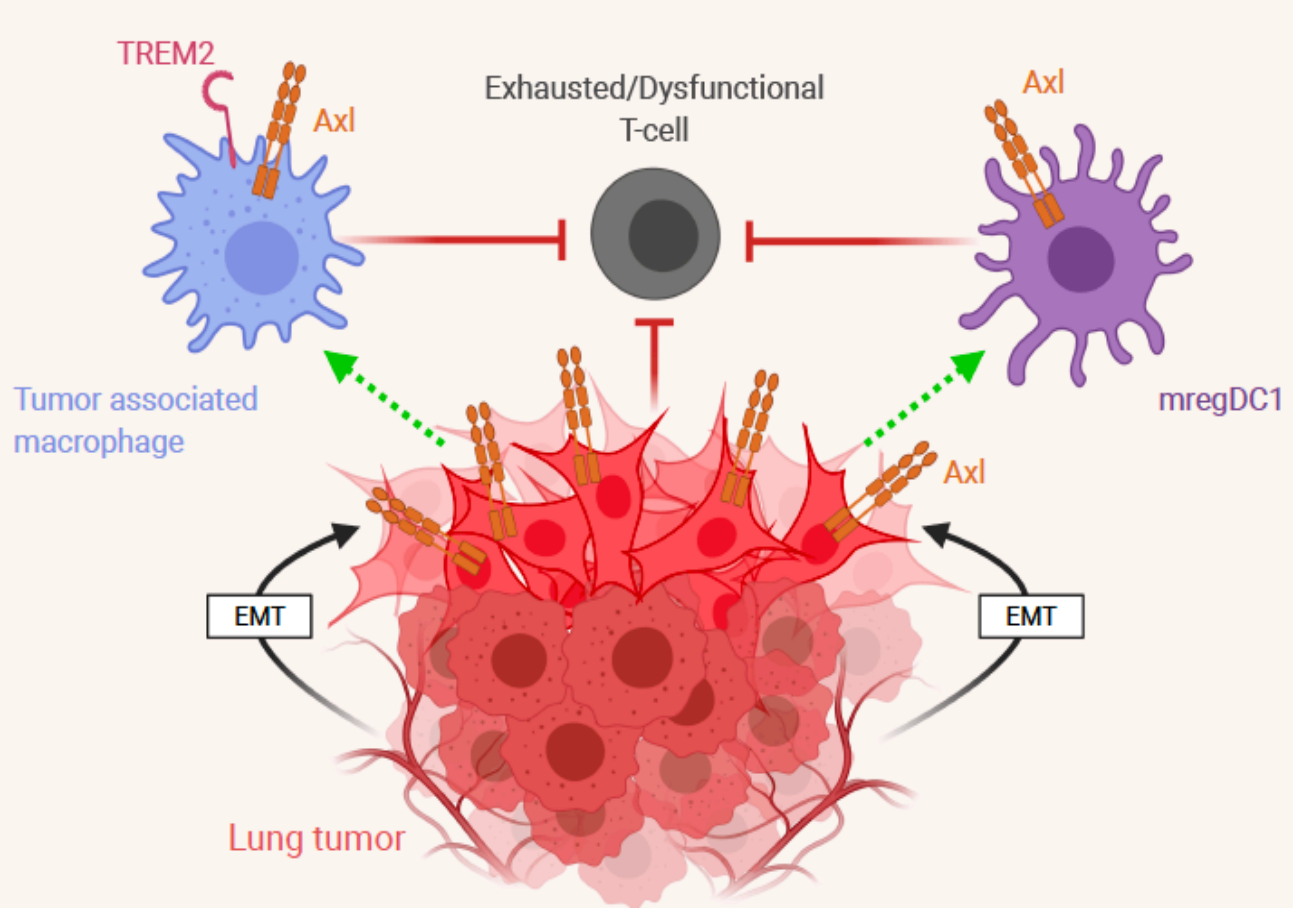
Proposed mechanism

AXL+ suppressive myeloid cells drive T cell dysfunction



- AXL promotes tumor-cell EMT and recently-described regulatory myeloid cells:
  - AXL⁺ TREM2⁺ Tumor Associated Macrophage[1,2]
  - AXL⁺ CCR7⁺ mregDC1[3]
- AXL expression in these cells promotes T cell dysfunction/exhaustion[2]
- Bemcentinib may reverse acquired resistance to checkpoint inhibition by targeting AXL+ TREM2 macrophages and regulatory DCs
- Bemcentinib inhibition of AXL reverses this state of immune suppression in the microenvironment, and promotes checkpoint inhibitor re-engagement

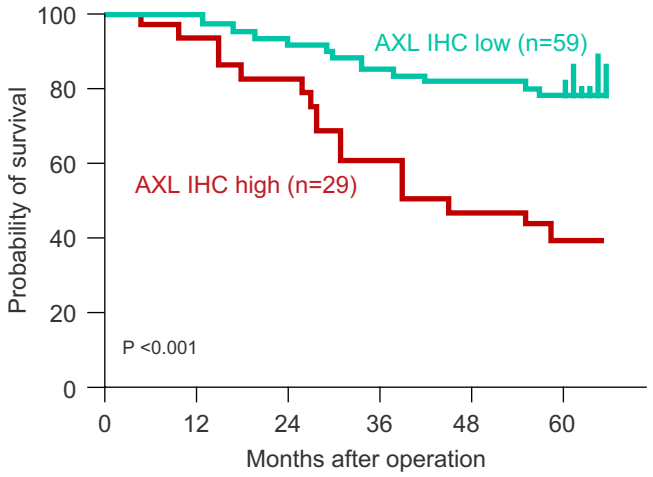
1. Katzenelenbogen *Cell* 2020; 2. Molgora *Cell* 2020; 3. Maier. et al. *Nature* 2020

TREM – Triggering receptor Expressed on Myeloid Cells
CCR7 - CC Chemokine receptor 7

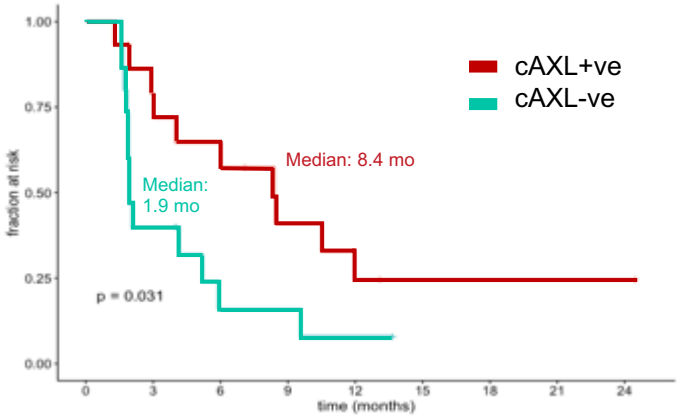
AXL expression defines a poor prognosis subgroup of NSCLC

cAXL+ patients have significantly enhanced survival with bemcentinib + pembrolizumab in CPI-naïve and refractory patients

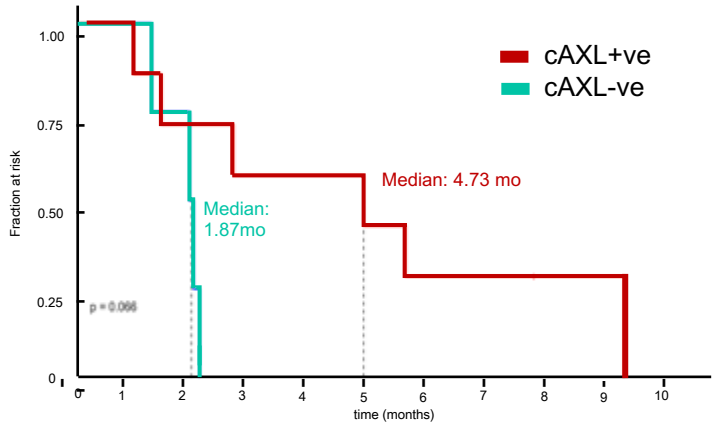
In NSCLC, the AXL expression encodes poor-prognosis[1]: defines expectations of the control arm



Cohort A PFS : CPI-naïve



Cohort B1 PFS: CPI-refractory



BIOLOGY = RATIONALE = OUTCOME

[1]Ishikawa, 2012

Bemcentinib clinical development in COVID 19

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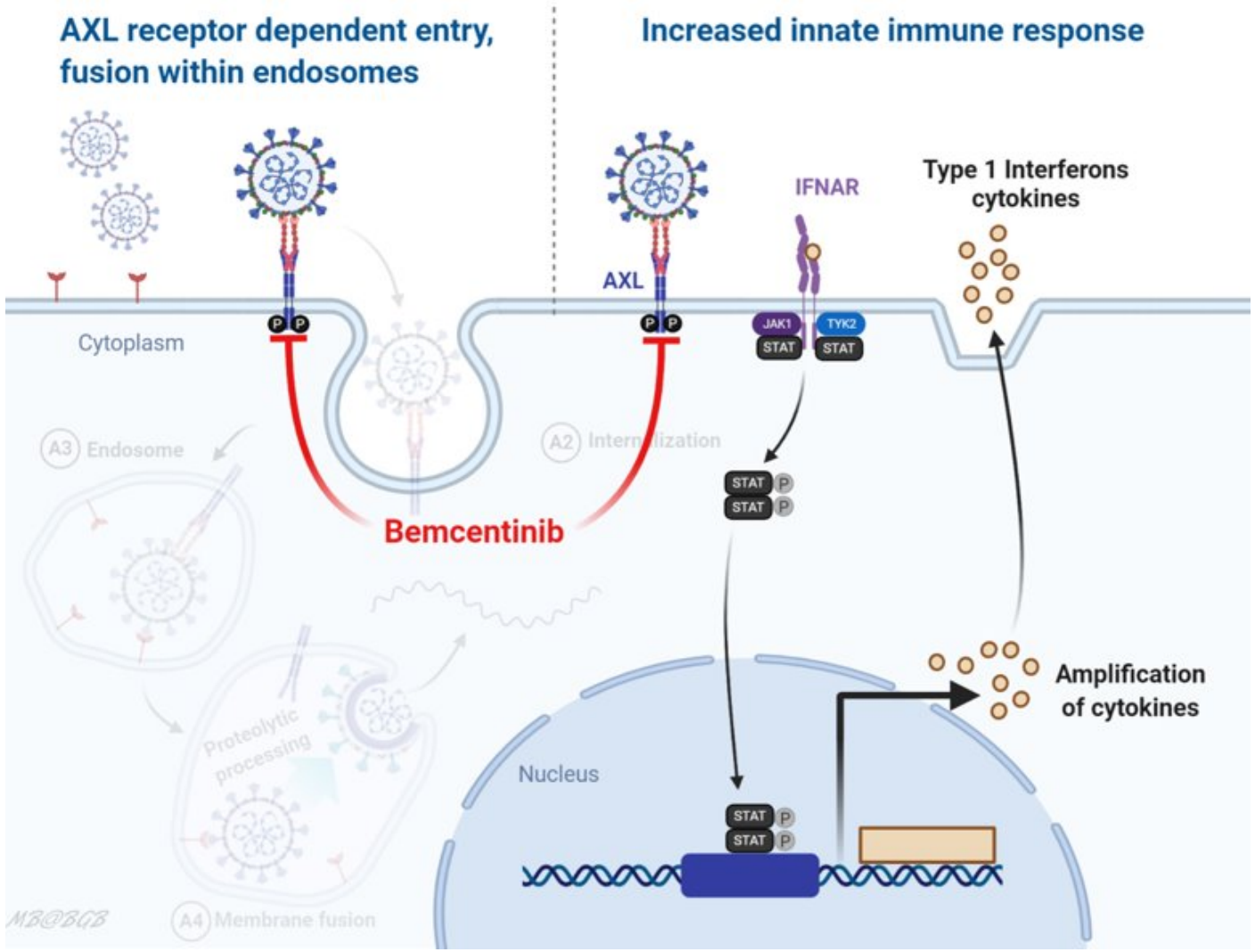
Potential of Bemcentinib on SARS-CoV-2 infection of host cells



- Utilization of AXL contributes to ACE2-dependent entry
- AXL enhances virus infection by facilitating virus entry via an endosomal pathway
- Bemcentinib control of virus infection likely involves both :
  - reduced viral entry and
  - enhanced interferon responses

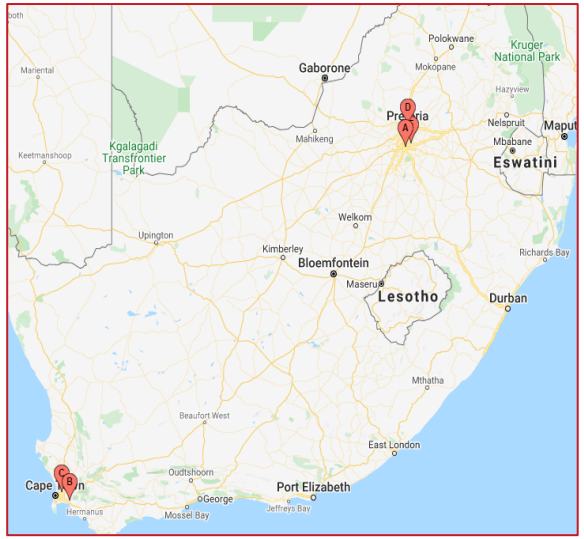
Two rPh II trials in hospitalized COVID-19 patients

- 2 x 120 patients
- Clinical and translational end points

ACCORD II STUDY

- Multicentre, seamless, Phase II adaptive randomisation platform trial
- Assessing the safety and efficacy of three candidate agents
- Up to 25 sites across the UK
- 60 patients will receive bemcentinib and 60 patients in a control group will receive standard of care treatment.

BGBC020 – SOUTH AFRICA



- Company sponsored randomises Phase II trial
- 60 patients will receive bemcentinib and 60 patients in a control group will receive standard of care treatment
- Assessing the safety and efficacy of bemcentinib
- 5 sites across South Africa
- 7 sites across India.
- .

BGBC020 - INDIA

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Bemcentinib randomised Phase II Studies in COVID-19

BGBC019 – ACCORD -120 pts & BGBC020 – 120 pts

COVID: BGBC020

Primary objective

To evaluate the efficacy of bemcentinib as add-on therapy to standard of care (SoC) in patients hospitalised with coronavirus disease 2019 (COVID-19).

Primary endpoint

Time to sustained clinical improvement of at least 2 points (from randomisation) on a 9-point category ordinal scale, live discharge from the hospital, or considered fit for discharge (a score of 0, 1, or 2 on the ordinal scale), whichever comes first, by Day 29 (this will also define the “responder” for the response rate analyses).

Key Secondary objectives

- To evaluate the ability to prevent deterioration according to the ordinal scale by 1, 2, or 3 points
- To evaluate the number of oxygen-free days
- To evaluate severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) viral load

Key Secondary objectives

- The proportion of patients not deteriorating according to the ordinal scale by 1, 2, or 3 points on Days 2, 8, 15, and 29
- Duration (days) of oxygen use and oxygen-free days
- Qualitative and quantitative polymerase chain reaction (PCR) determination of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in oropharyngeal/nasal swab while hospitalised on Days 1, 3, 5, 8, 11, 15, and 29

Exploratory objectives

- To evaluate PK of bemcentinib
- To evaluate SARS-CoV-2 viral load
- To collect samples for serology research, viral genomics, serum antibody production, and COVID-19 diagnostics

Exploratory objectives

- PK concentration and parameters
- Qualitative and/or quantitative PCR determination of SARS-CoV-2 in blood (on Day 1) and saliva
- Analysis of samples collected at baseline prior to treatment and at specific time points

Executing a broad development program for Bemcentinib

monotherapy	+checkpoint inhibitors	+ chemotherapy	+ targeted therapy	+ epigenetic Therapy (HMA)
r/r MDS	Relapsed NSCLC			
Recurrent Glioblastoma	Relapsed TNBC	1L Pancreas		
r/r AML	1L Metastatic Melanoma	>2L NSCLC	1L Melanoma	
1L COVID19	1L Metastatic Mesothelioma	Relapsed AML	1L & Relapsed NSCLC	r/r AML

Bemcentinib foundation for cancer therapy

Introduction Tilvestamab (BGB149) anti-AXL monoclonal antibody

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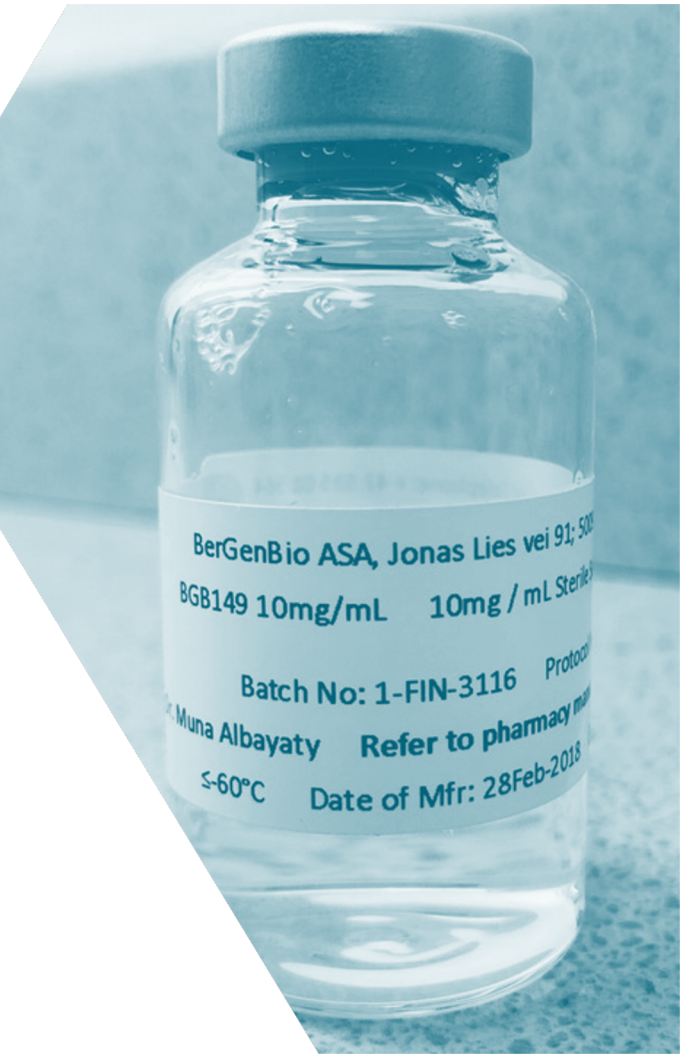
Ref. BGB149-101 / NCT03795142

TILVESTAMAB: Anti-AXL monoclonal antibody

Phase I clinical trial ongoing



Functional blocking fully-humanised IgG1 monoclonal antibody

Binds human AXL, blocks AXL signalling

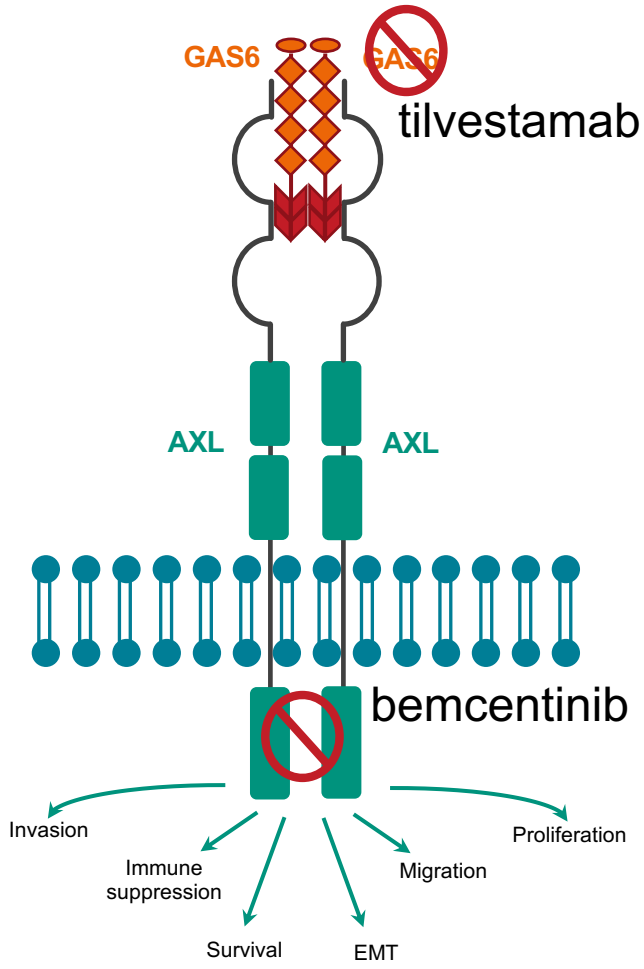
High affinity (KD: 500pM), displaces GAS6
Anti-tumour efficacy demonstrated *in vivo*

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 SAD trial complete
Phase Ib/IIa MAD in set up phase



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Well positioned for success ….

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Outlook

Strong cash position

- Well funded, Q3 cash position $82m (NOK 778 million)

Promising pipeline

- Two first-in-class drug AXL inhibitors in multiple Ph II clinical trials
- Pioneering biology and a substantial amount of favourable clinical POC, safety and translational data

Compelling Phase II POC

- Relapse AML in combination with LDAC
  - FDA Fast Track Designation and Orphan Status
- Relapse MDS monotherapy
  - predictive biomarkers
- 2L NSCLC: CPI combination,
  - significant survival benefit and predictive biomarkers

Strong science supporting COVID-19 treatment in 2 randomised phase II trials

- Anticipate top line clinical data Q1’21

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