



***Breakthrough antibody
technology to broaden therapeutic
window of anti-cancer drugs***

January 2021

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The Company

Technology

- Proprietary CAB technology creates antibodies that conditionally and reversibly bind to tumors, but not normal cells, enabling increased antibody potency and reduced toxicity
- Strong intellectual property rights, with 257 patents issued, 8 allowed, and 212 pending applications

Clinical and Team

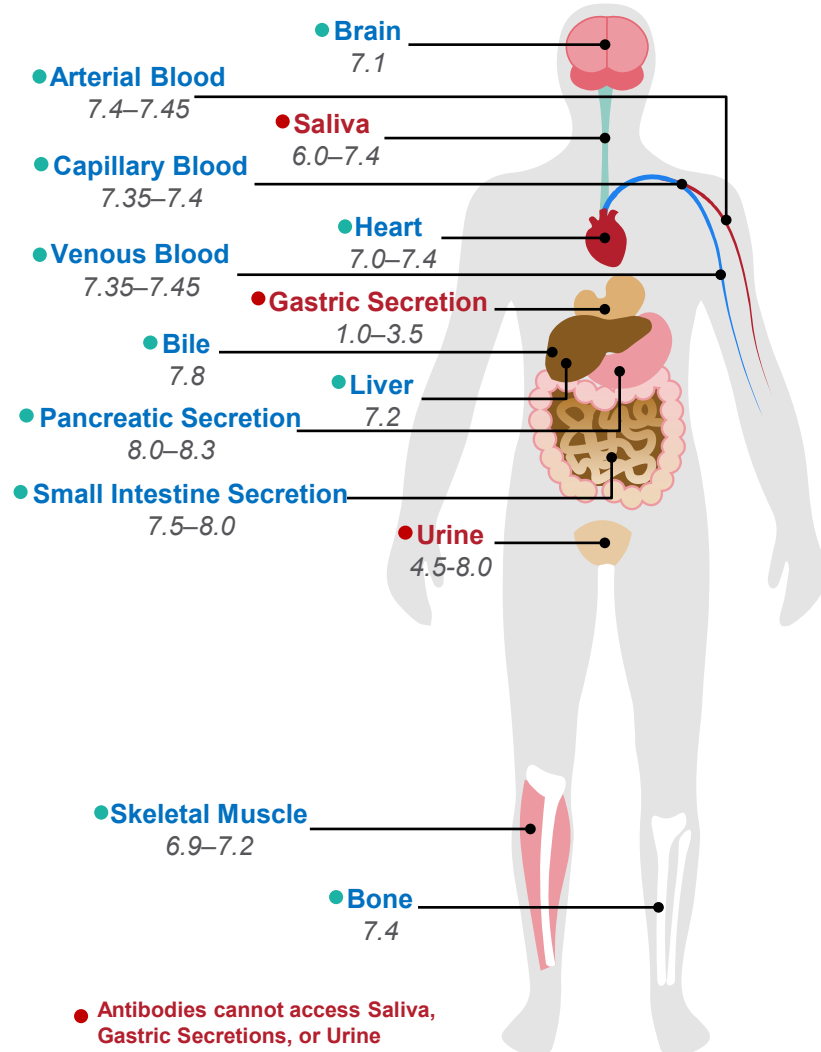
- Clinical stage company with two P2 CAB antibodies for multiple indications and one partnered CAB antibody entering P1 clinical studies
- 54 employees and contractors with exceptional experience in innovative research and clinical development

Finance and Infrastructure

- Launched successful IPO on December 16th raising over \$217 MM in gross proceeds with \$383 million raised to date
- Committed BeiGene collaboration with \$25 million received to date, and eligible to receive up to \$225.5 million in future milestone payments
- Headquartered in San Diego in a ~43,000 square foot office and lab facility with a contract lab in Beijing

CAB Technology Leverages Differential pH of Cancer Cells

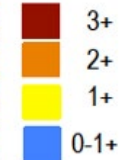
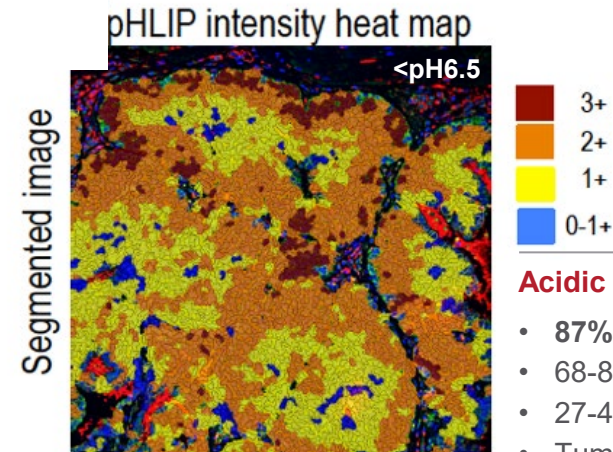
Normal Human Environments Typically Have Alkaline pH



● Antibodies cannot access Saliva, Gastric Secretions, or Urine

● pH of normal environments that antibodies access

Tumor Environments Are Acidic With Lower pH Levels



Acidic Cells

- 87% oxygenated cells
- 68-82% replicating cells
- 27-41% non-dividing cells
- Tumor-stroma boundary

1

CAB antibodies allow for selective targeting of cancer tumor cells, **based on pH**, while generally not binding to normal tissue, thereby eliminating or reducing “on-target off-tumor” toxicity

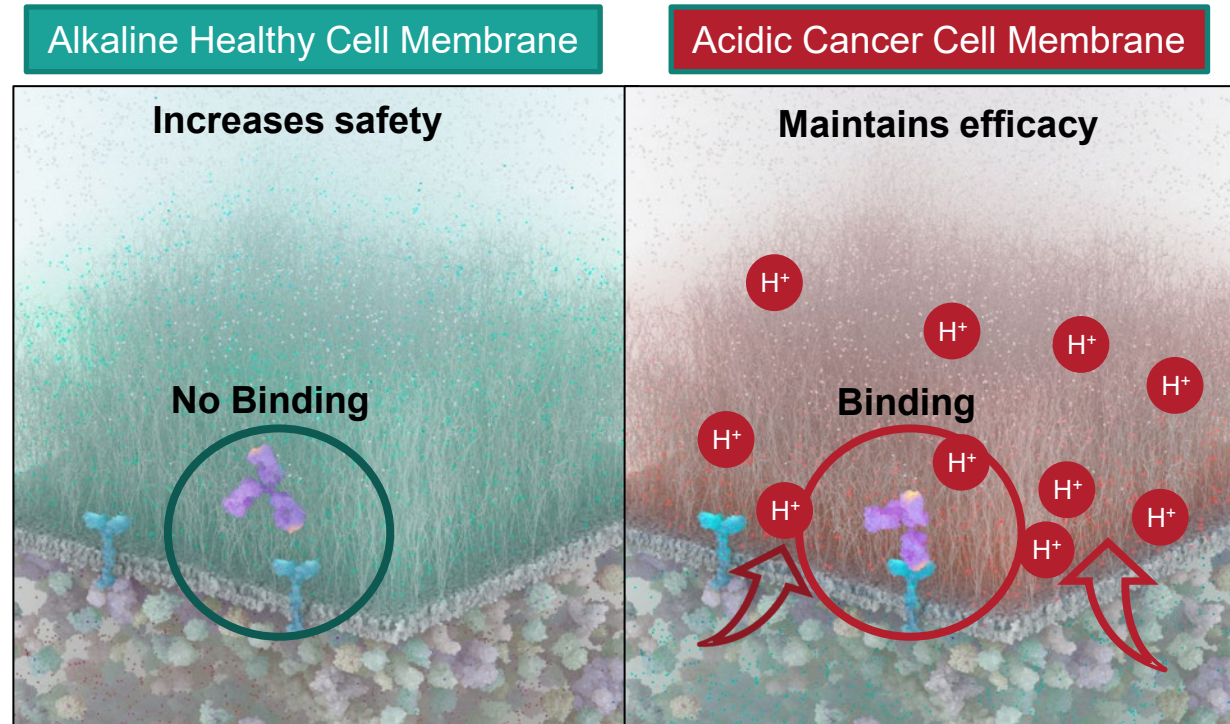
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This leads to the potential for **enhanced therapeutic exposure & reduced toxicity** compared to traditional antibodies with the goal of widening the “therapeutic index”

CAB Antibodies Selectively Bind in Acidic Tumor Microenvironment

Tumor acidity due to high glycolytic metabolism
(Warburg Effect)

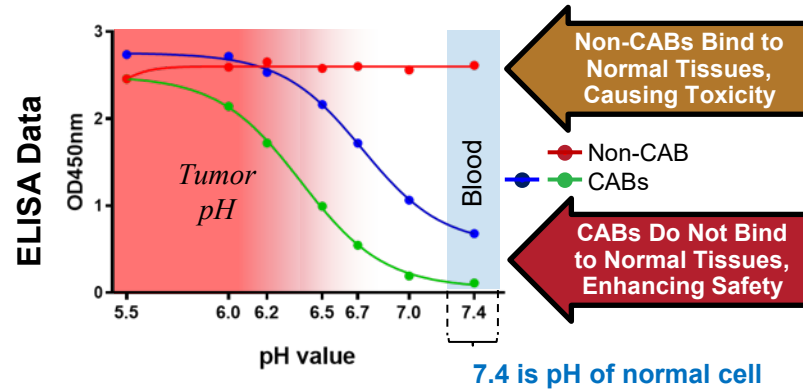
- Basis of PET scanning for tumor identification
- Lactic acid secretion lowers external pH
- Generates precursors for tumor cell replication
- Acidic environment reduces immune defenses



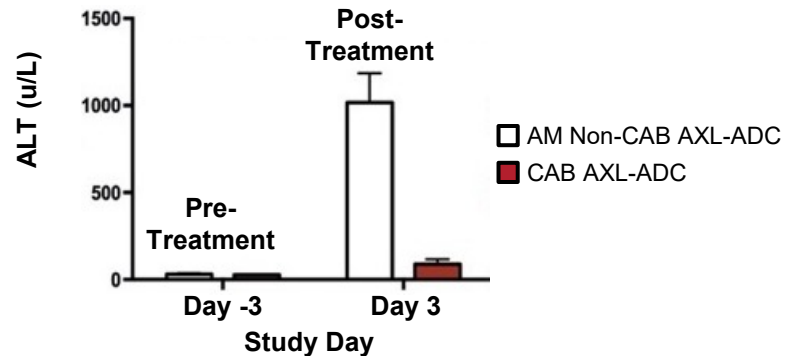
CABs utilize rapid, reversible chemical activation via H^+

CABs Bind Selectively and Reversibly Based on the TME, Enhancing Exposure and Reducing Toxicity

CABs Bind Selectively in the Lower pH TME



Reduced Toxicity in Non-human Primates



- CAB ADC resulted in minimal increase in ALT, supporting that on-target, off-tumor toxicity is reduced with the CAB ADC

CABs Widen Therapeutic Index

- Eliminates or reduces off-tumor tox
- Avoid TMDD, improves pharmacokinetics (PK)
- Only CDR modification, reducing immunogenicity
- Rapid, reversible activation via chemical switch (H⁺)
- Efficient development and manufacturing
- Expands target universe
- Increased safety and potency

Unlike prodrugs, CABs are reversible, enhancing the therapeutic index

Note: Data above based on non-human primate studies; OD450nm = optical density measurements using a microplate reader with a 450nm filter; TME = Tumor Microenvironment; AM = affinity matched; CDR = Complementarity-determining regions; TMDD = Tissue Mediated Drug Deposition; ALT or alanine aminotransferase elevation is a sign of liver toxicity

Robust Pipeline of Antibody-Based Therapeutics

Type	CAB Program	Target	Indications	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3	Expected Upcoming Milestones
ADC	BA3011 (AXL-ADC)	AXL Positive	STS & Bone Sarcoma, NSCLC, Ovarian Cancer* (Mono & Combo w/ PD-1)						<ul style="list-style-type: none"> Ph2 interim data 2021 Ph2 registration data 2022
	BA3021 (ROR2-ADC)	ROR2 Positive	NSCLC, Melanoma, Ovarian Cancer* (Mono & Combo w/ PD-1)						<ul style="list-style-type: none"> Ph2 interim data 2021 Ph2 registration data 2022
CTLA-4	BA3071 (CTLA-4)	CTLA-4	RCC, NSCLC, SCLC, HCC, Melanoma, Bladder, Gastric, Cervical Cancer (Mono & Combo w/ PD-1)						<ul style="list-style-type: none"> Ph1 dose escalation trial to be initiated 2020 or early 2021 Ph1 data 2021
Bispecific	BA3182 (Bispecific)	EpCAM / CD3	NSCLC, SCLC, Colorectal, Ovarian, TNBC, Prostate Cancer**						<ul style="list-style-type: none"> US IND in 1H 2022
	BA3142 (Bispecific)	B7-H3 / CD3	NSCLC, SCLC, HNC, Melanoma, Sarcoma, Pancreatic, Prostate Cancer**						<ul style="list-style-type: none"> US IND in 2022
	EGFR (Bispecific)	EGFR / CD3	NSCLC, HNC, Pancreatic, TNBC, Colorectal Cancer**						<ul style="list-style-type: none"> US IND in 2022
	Nectin-4 (Bispecific)	Nectin-4 / CD3	Bladder, TNBC, Pancreatic Cancer**						<ul style="list-style-type: none"> US IND in 2022

Abbreviations: STS = Soft Tissue Sarcoma, NSCLC = Non-small Cell Lung Cancer, RCC = Renal Cell Carcinoma, SCLC = Small Cell Lung Cancer, HCC = Hepatocellular Carcinoma, TNBC = Triple-Negative Breast Cancer, HNC = Head and Neck Cancer; * Ph2 investigator-initiated trial for Ovarian Cancer expected to be initiated by the end of 2020 or early 2021
 ** Anticipated indications based upon tumor target expression

Disease Progression

1

AXL's higher expression associated with disease progression in several indications, including:

- Sarcoma, NSCLC, ovarian cancer, breast cancer, pancreatic cancer, glioblastoma, melanoma, RCC, prostate cancer, and esophageal cancer

Tumor Resistance

2

AXL expression associated with tumor resistance to:

- Chemotherapy, PD-1/L1 inhibitors, molecular targeted therapy, and radiation therapy

Clinical Validation

3

AXL has been clinically validated as a target:

- Multiple assets in the clinic including non-specific small molecules and antibody ADCs
- Some anti-AXL antibodies in the clinic have shown encouraging signs of antitumor activity; however, adverse events may limit clinical utility and/or potency

Initial US Addressable Patient Population

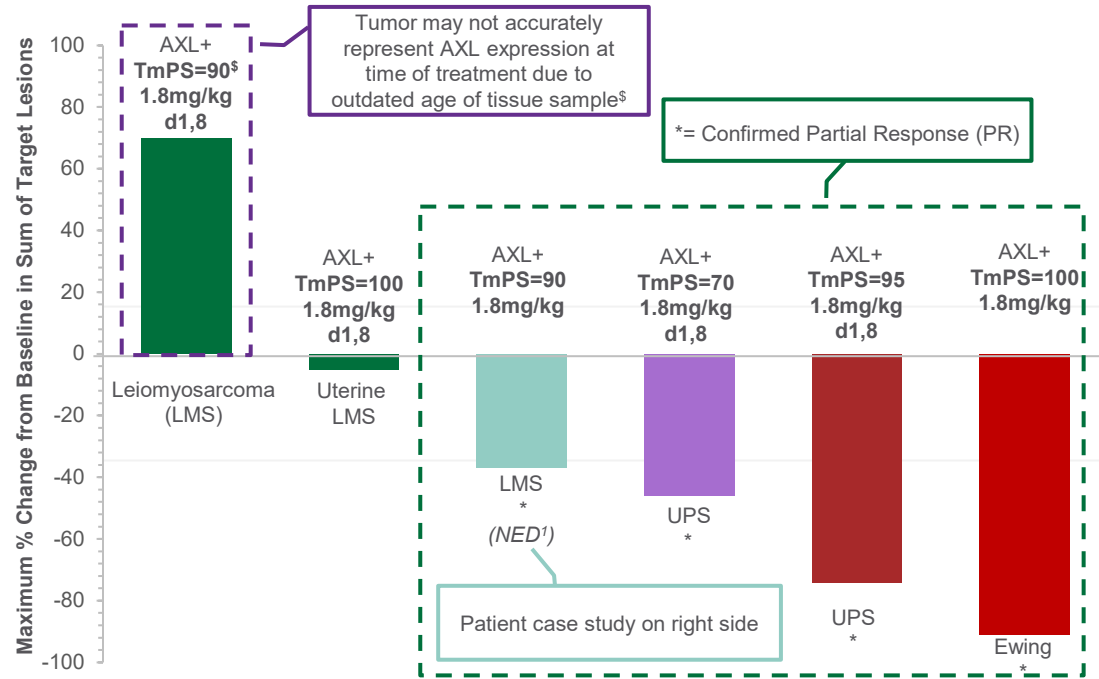
Tumor type	Patient treatment phase	Est. corresponding US patient population	Est. AXL positivity rate ¹	Est. US target population at launch
Sarcoma (STS & Bone)	Stage III/IV	10,000 – 15,000	50%	5,000 – 7,500
NSCLC	Stage III/IV (PD-1/L1 experienced)	66,000 ²	30%	15,000
Ovarian Cancer	Stage III/IV Platinum resistant	12,000	30 – 40%	4,000

Source: BioAtla IHC assay validation results & phase 1 AXL testing data, GlobalData-Opportunity Analysis and Forecasts, SEER database

¹Based on TmPS (Tumor membrane Percent Score) ²75% of these patients generally switch to a new therapy

BA3011: Encouraging Results in AXL High (TmPS ≥ 70) Sarcoma Patients at 1.8mg/kg

Sarcoma (confirmed TmPS** ≥ 70 ; 1.8mg/kg Q3W or 2Q3W)



4 partial responses out of 6 for sarcoma patients with TmPS ≥ 70 at optimal dosing levels

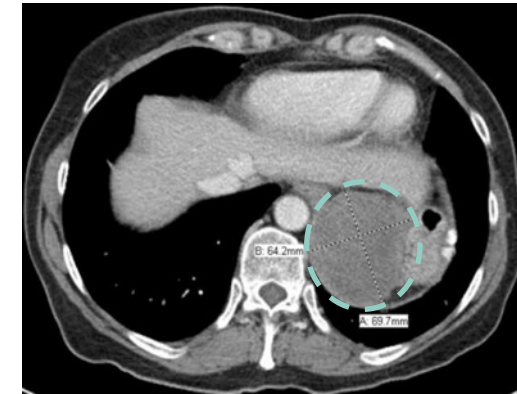
Note: **AXL Tumor membrane Percent Score or TmPS = % Score $\geq 1+$

[§]Tissue biopsy from resection, over 1 year old prior to trial entry

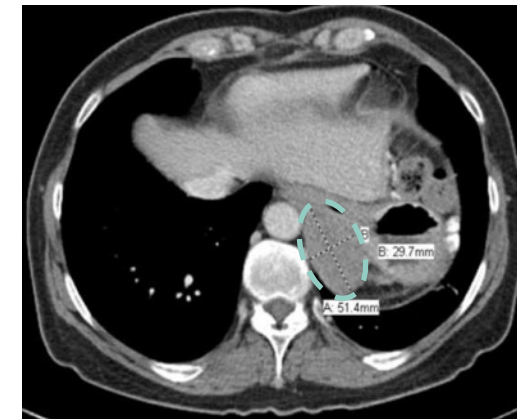
All patients: Multiple cycles of antineoplastic agents received prior to starting treatment with BA3011;

¹NED = No evidence of disease; study is on-going as of Dec 31, 2020

LMS Patient Case Study



Pre-treatment

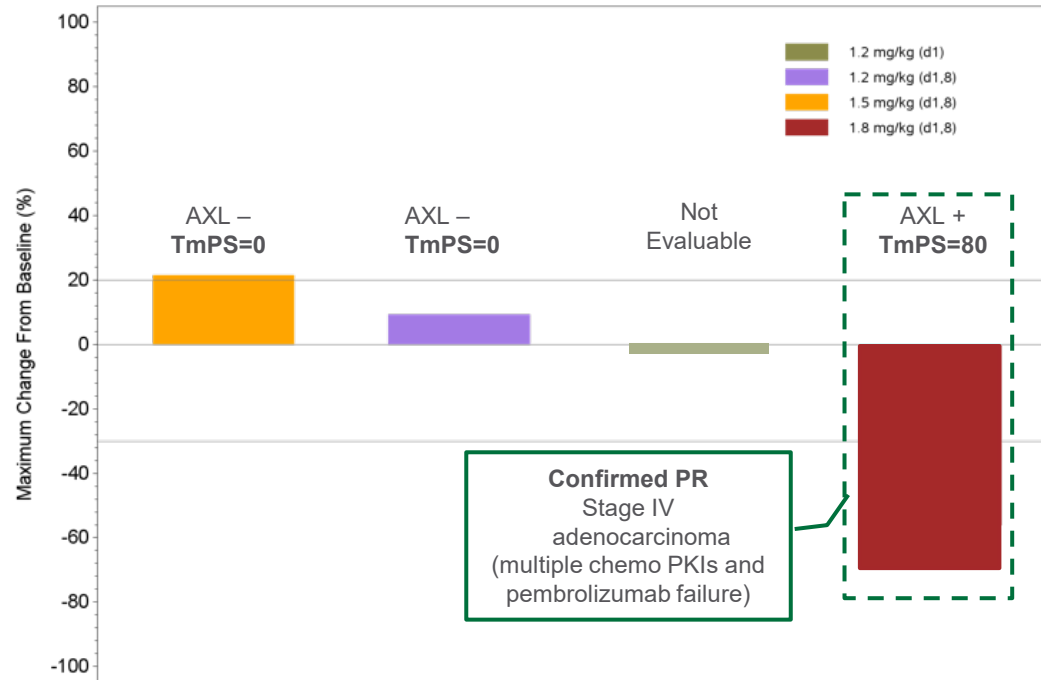


Post-treatment, week 18 Scan

- 37% tumor reduction
- Tumor mass reduced enough to enable successful surgical resection resulting in NED¹

BA3011: Encouraging Results in NSCLC AXL High (TmPS ≥70) Patient at 1.8mg/kg

NSCLC (All Patients)



Stage IV adenocarcinoma patient case study

- Patient experienced multiple failures of prior treatments
- Prior treatment with PD-1 inhibitor (pembrolizumab) failed
- ~70% tumor reduction after BA3011 dosed at 1.8 mg/kg 2Q3W

Out of 4 NSCLC patients, partial response achieved in the one patient with TmPS ≥ 70

Note: All patients: Multiple cycles of antineoplastic agents received prior to starting treatment with BA3011

ROR2 Over-expression

1

Over-expressed across many solid tumors, including NSCLC, melanoma, ovarian, TNBC, and HNC

Enhanced ROR2 Expression

2

Enhanced ROR2 expression with prior PD-1/L1 treatment

Nascent Competition

3

No other ROR2 ADC or small molecules in the clinic yet, but competition is emerging

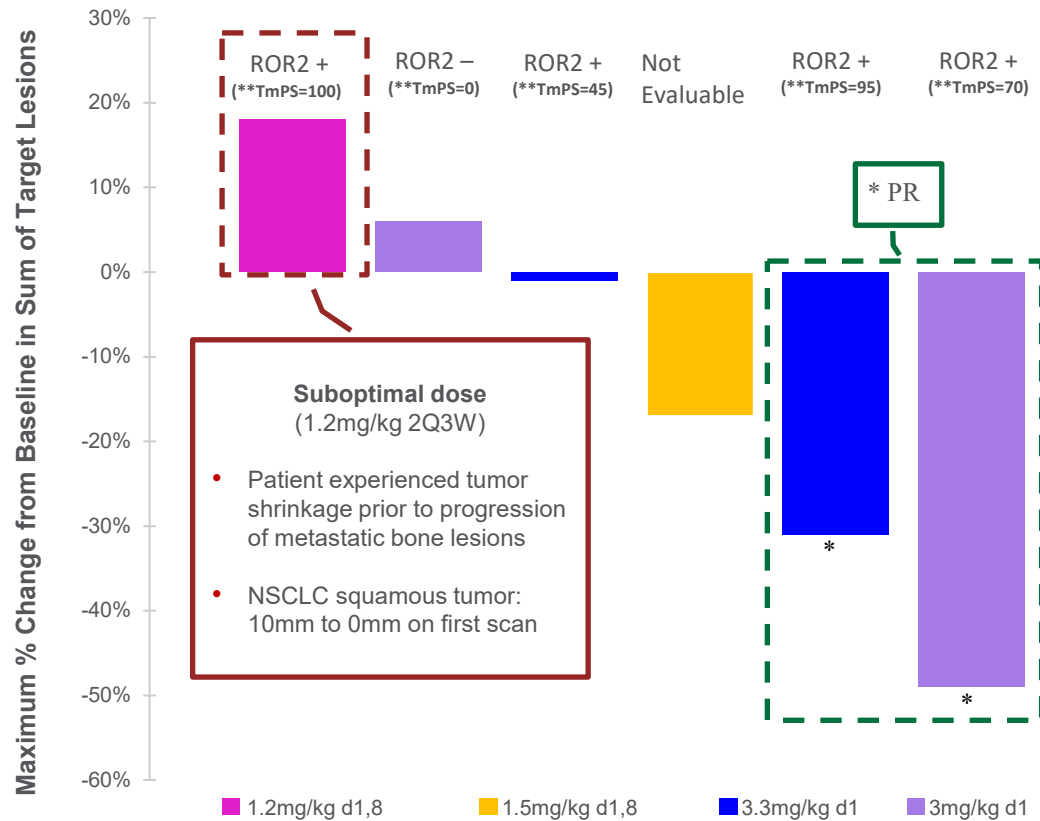
Initial US Addressable Patient Population

Tumor type	Patient treatment phase	Est. corresponding US patient population	Est. ROR2 positivity rate ¹	Est. US target population at launch
NSCLC	Stage III/IV (PD-1/L1 inhibitor)	66,000 ²	30%	15,000
Melanoma	Immune checkpoint inhibitor	25,000 ²	20 – 30%	5,000
Ovarian Cancer	Stage III/IV Platinum resistant	12,000	30 – 40%	4,000

Source: BioAtla IHC assay validation results & phase 1 AXL testing data, GlobalData-Opportunity Analysis and Forecasts, SEER database; ¹Based on TmPS (Tumor membrane Percent Score) ²75% of these patients generally switch to a new therapy

BA3021: Encouraging Results in Stage IV PD-1 Refractory NSCLC Patients

All evaluable NSCLC patients enrolled in BA3021 Phase 1 trial



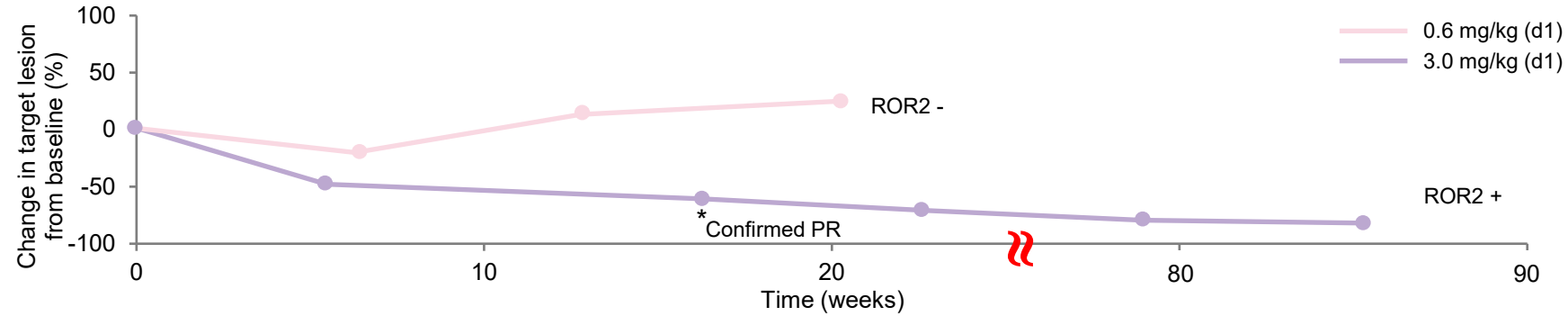
Clinical results show promise in refractory patients

- 1 All NSCLC patients who enrolled in this trial had previously been treated with PD-1 therapy
- 2 ROR2 expression strongly correlates with anti-tumor response

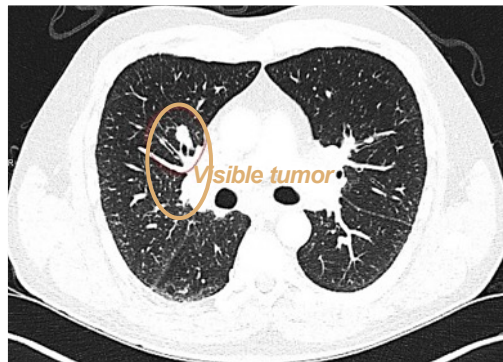
Note: Not Evaluable (Strong, extensive fibroblastic stromal positivity reported)

**TmPS= Tumor membrane Percent Score- Tumor membrane target expression calculated by summing the percentages of intensities at either $\geq 1+$, $\geq 2+$ or $\geq 3+$. Scores range from 0 to 100.

All evaluable metastatic melanoma patients enrolled in BA3021 Phase 1 trial by ROR2 TmPS

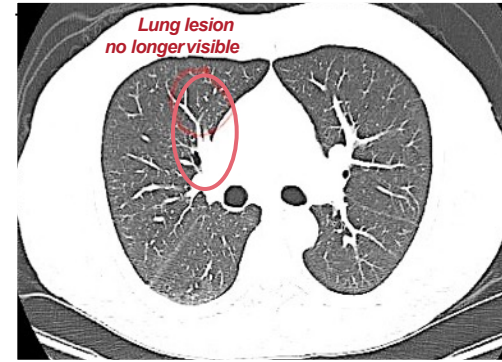


- One of two melanoma patients enrolled in the BA3021 Phase 1 dose escalation trial achieved a partial response (Purple line)
- Patient with PR experienced failure of both nivolumab & nivolumab + ipilimumab; now continuing BA3021 > 1 yr, consistent with results below



Pre-treatment CT scan

Pre-treatment posterior occipital lymph node biopsy: **Active melanoma**



On-treatment; Week 6 Scan

On-treatment posterior occipital lymph node biopsy: **No melanoma detected**

“Biopsy: consistent with metastatic melanoma... consists of fibrous stroma and a relatively **pure population of malignant melanoma cells...**”

“Final pathology results: dense fibrous connective tissue with abundant melanin-laden macrophages, **no melanoma seen...**”

Overview of adverse events in Phase 1 trials

AEs consistent with MMAE-based toxicity, including:

- reversible myelosuppression
- transient liver enzyme elevation
- metabolic disturbances

Few grade 3 or greater AEs

None of the related AEs led to treatment discontinuation

Patients administered 1.8mg/kg 2Q3W (d1,8) (safety population)

Characteristic	BA3011 (N=20)	BA3021 (N=7)
Any Adverse Events (AEs)	17 (85%)	7 (100%)
Related AEs with CTCAE ¹ Grade 3 or 4 ²	6 (30%)	3 (43%)
Any related serious AEs ²	3 (15%)	2 (29%)
AEs leading to death	0	0
Related AEs leading to death ²	0	0
Related AEs leading to treatment discontinuation ²	0	0

BA3011 (CAB AXL-ADC)
(all patients n=55)

No clinically meaningful on-target toxicity observed

Constipation

- Grade 1-2 **(16%)**
- Grade 3-4 **(4%)**

Constipation is believed to be an on-target mediated effect

Differentiated profile due to advantageous pharmacokinetic characteristics of CAB ADC

Peripheral Neuropathy & Diarrhea

- PN rates **(18%) (All Grade 1-2)**
- Diarrhea rates **(20%) (16% Grade 1-2)**

Note: ¹CTCAE: Common Terminology Criteria for Adverse Events. The NCI Common Terminology Criteria for Adverse Events is a descriptive terminology which is utilized for Adverse Event (AE) reporting. A grading (severity) scale is provided for each AE term. ²As assessed by the investigator. Missing responses are counted as related. Studies are on-going

Potentially Registration-Enabling Phase 2 Trials

CAB AXL-ADC (BA3011)



Sarcoma (STS# & Bone)
mono & combo with PD-1; AXL TmPS* \geq 70 ;
3rd Line (n=165-240)

Interim analysis

★
Data read-out



NSCLC
mono & combo with PD-1; AXL TmPS* \geq 50
in PD-1 refractory patients (n=40)

★
Proof Of Concept read-out

Ovarian Investigator-Initiated Trial (IIT) not shown



CAB ROR2-ADC (BA3021)



NSCLC
mono & combination w PD-1
ROR2 TmPS* \geq 50 ; PD1-refractory patients (n=200)



Melanoma
mono & combination w PD-1
ROR2 TmPS* \geq 50 ; PD1-refractory patients (n=200)

Interim analysis

★
Data read-out

Ovarian Investigator-Initiated Trial (IIT) not shown

Note: # STS= Soft Tissue Sarcoma; *TmPS= Tumor membrane Percent Score- Scores range from 0 to 100

Opportunity exists for a “safer” CTLA-4 inhibitor

- Traditional combination of anti-PD-1 and anti-CTLA-4 checkpoint inhibitor led to **improved outcomes**
- Combination associated with increase in **adverse events** and **treatment discontinuations**
- A safe combo of PD-1/CTLA-4 has **potential** across many immunogenic tumors

Clinical Endpoint	Nivolumab (PD-1) ¹	Nivolumab + Ipilimumab ¹
Progression Free Survival	6.9 months	11.5 months
Grade 3 or 4 Adverse Events	16.3%	55.0%
Discontinued Treatment	7.7%	36.4%

Source: ¹Larkin et al., New Eng. J. Med.,373: 23-34, 2015



Global Strategic Collaboration

- BeiGene holds an exclusive global license to BA3071
- BioAtla has received \$25 million in upfront payments & reimbursement
- BioAtla eligible to receive up to \$225.5 million for subsequent regulatory and development milestones
- BioAtla eligible to receive tiered royalties of up to low 20's on worldwide sales

Clinical Development

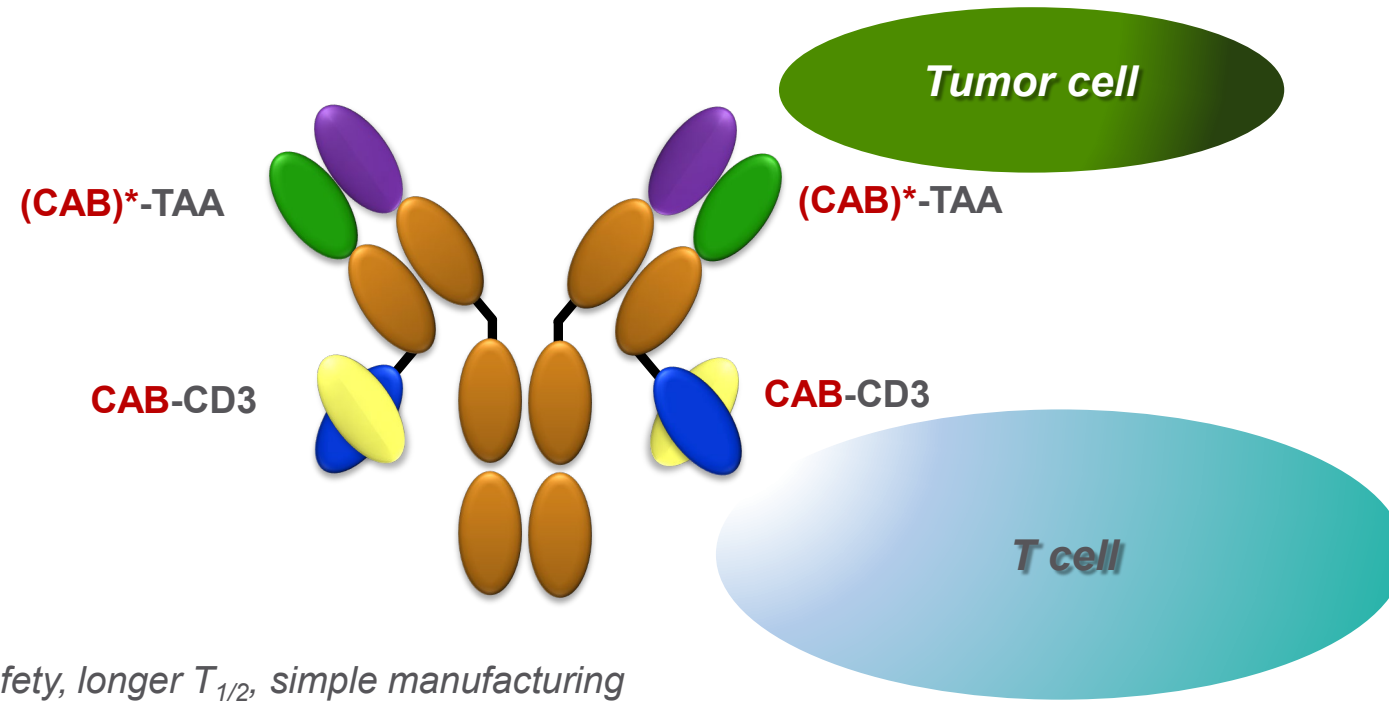
- Expected Phase 1 dose escalation trial early 2021
- Doses of 7mg Q3W to 700mg Q3W¹ as monotherapy and in combination with tislelizumab²

Collaboration & Support

- BeiGene leads Development, Manufacturing and Commercialization activities

¹Equivalent to 10mg/kg of ipilimumab

²Tislelizumab is an anti-PD-1 antibody from BeiGene in late-stage development



Higher safety, longer $T_{1/2}$, simple manufacturing



CABs have potential to reduce systemic activation for greater safety and efficacy



Reduced cytokine release syndrome and neurological toxicity



Enables T cell engaging therapies with high potency while limiting T cell exhaustion

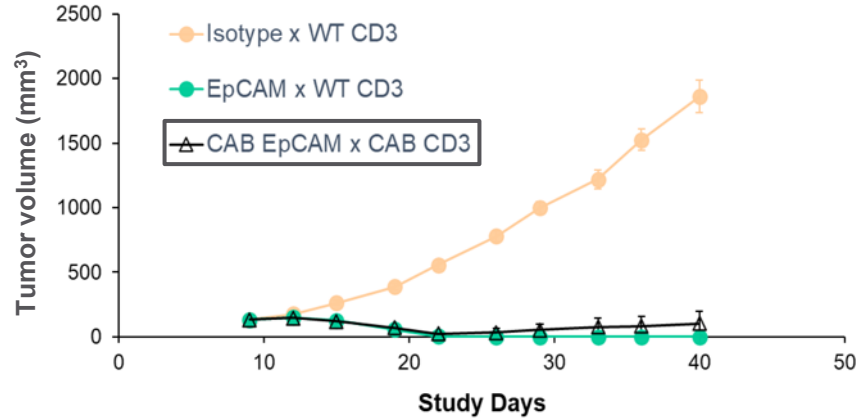


Four active bispecific programs (BA3182, BA3142, EGFR, Nectin-4)

Note: *Optional CAB directed against the tumor associated antigen (TAA)

CAB-EpCAM x CAB-CD3 Bispecific Antibody Exhibits Comparable Antitumor Activity, While Maintaining Superior Safety Profile

CAB EpCAM x CAB CD3 bispecific demonstrates efficient tumor shrinkage

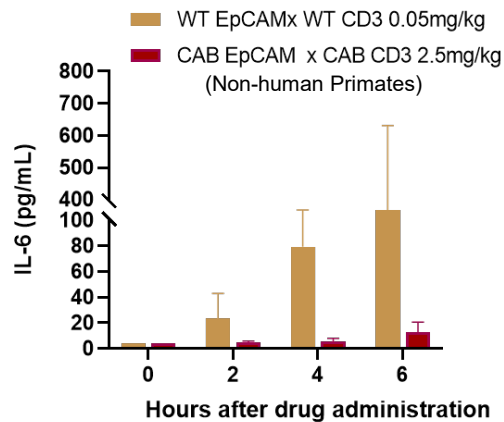


MiXeno Model with HCT116 = Colorectal Cancer Cell Line
1mg/kg twice/week in mice
(equivalent to 0.25mg/kg in non-human primates)

Summary

- CAB-EpCAM x CAB-CD3 bispecific antibodies have comparable antitumor activity to wild type bispecific
- Low toxicity observed, characterized by lower levels of IL-6
- > 10x Higher Therapeutic Index

CAB EpCAM exhibits lower IL-6 levels associated with severe cytokine-related toxicities



Bispecific Safety Results (Non-human Primates)

WT-EpCAM x WT-CD3

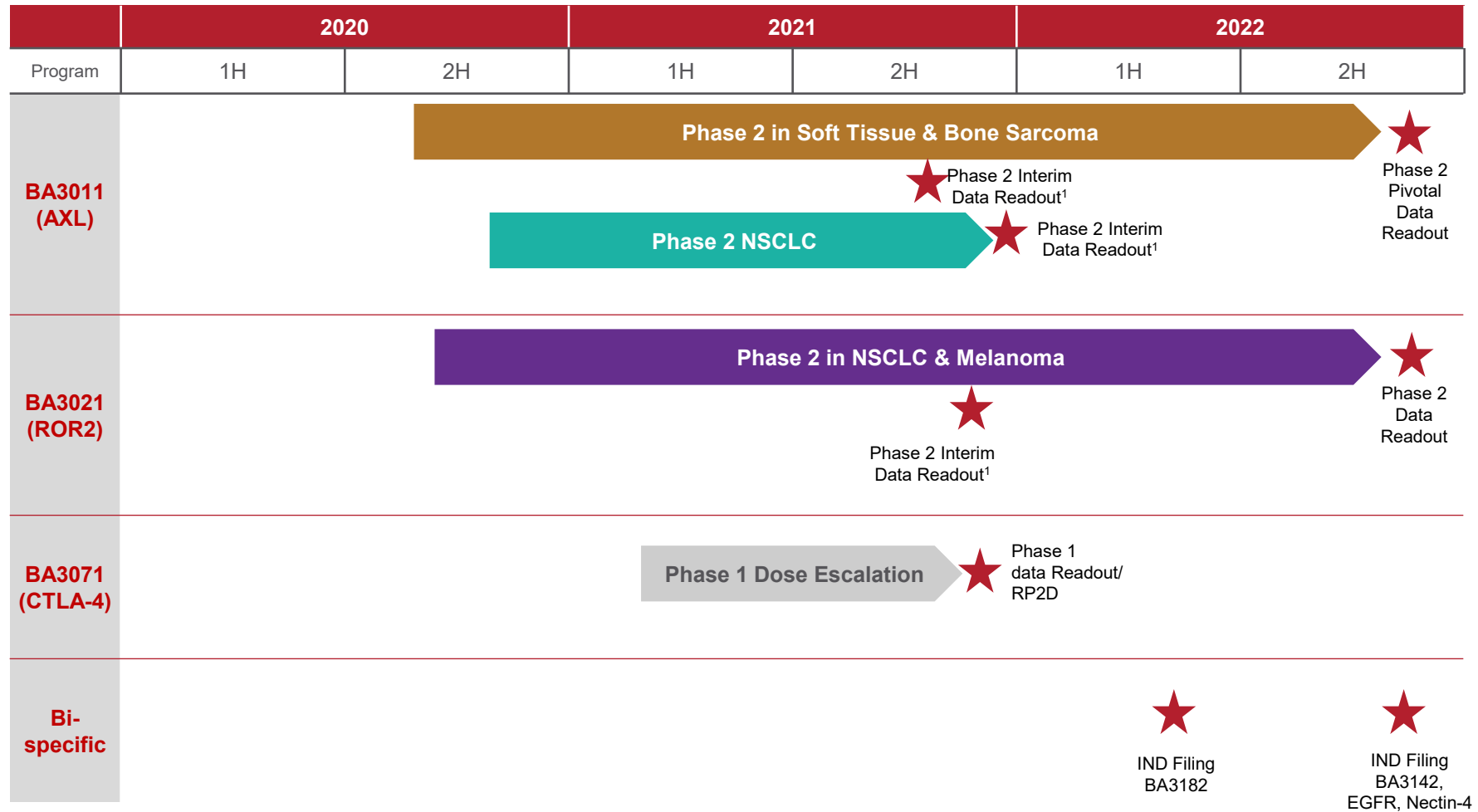
0.05 mg/kg = 2 expired
*0.025mg/kg = 2 ill

WT = wild type; *from independent experiments

CAB-EpCAM x CAB-CD3

0.25mg/kg = 2 normal
1.0 mg/kg = 2 normal
2.5 mg/kg = 2 normal

Upcoming Data Readouts and Inflection Points



Note: ¹Ovarian Investigator Initiated Trial not shown

Y/E 2020 cash on hand of \$239 mm;
Sufficient capital to get through all listed inflection points and well into 2023

BioAtla is well positioned to develop a strong franchise of CAB-enabled treatments



Innovative CAB technology platform, with clinically-validated antibodies, that conditionally activate at optimal exposure levels, exhibit high potency, and possess ideal safety profiles



Multiple clinical assets demonstrating differentiated CAB technology and strong results for challenging targets, leading to novel therapeutics that can fulfill previously unmet patient needs, and resulting in a broad and diverse pipeline



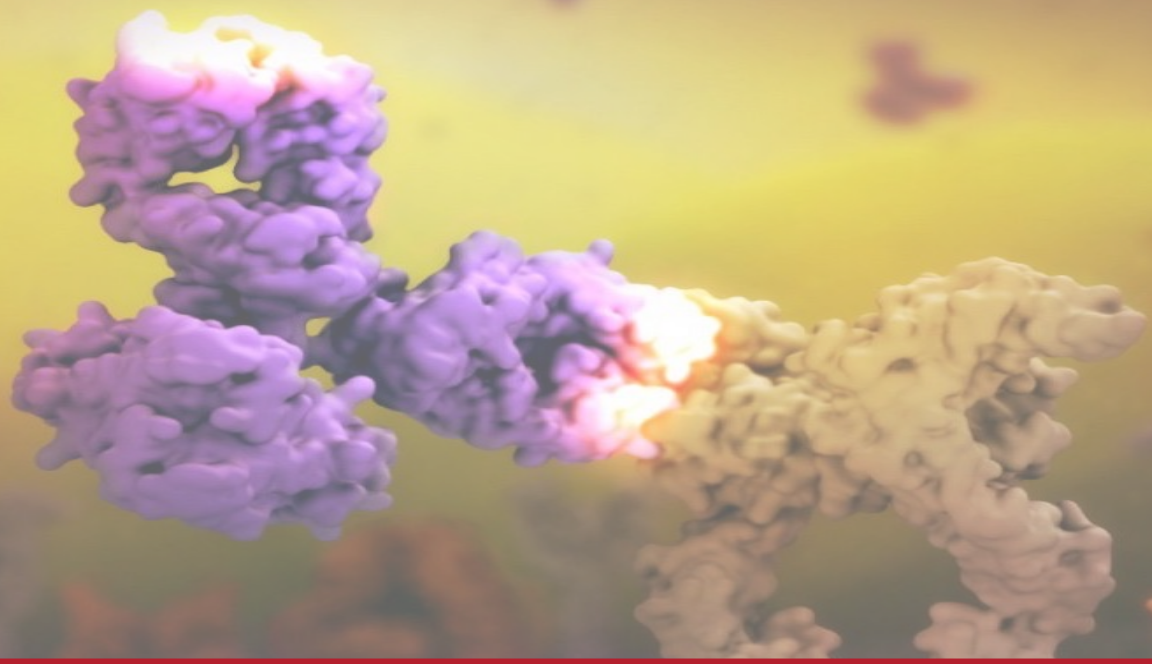
Strong intellectual property foundation that provides worldwide coverage and multiple diversified patents for each product



Talented and experienced management team, with a strong track record and over 20 years of experience on average with leading biopharmaceutical companies



Strong financial support through a global collaboration with BeiGene and backing of reputable investors



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