UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): December 20, 2024

BIOATLA, INC.
(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39787**	**85-1922320**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

11085 Torreyana Road San Diego, California	**92121**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: 858 558-0708

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.0001 par value per share	BCAB	Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

BioAtla, Inc. (the "Company") updated its corporate presentation, which presentation is attached as Exhibit 99.1 to this Current Report on Form 8-K ("Current Report"). The Company may use such presentation from time to time in meetings with investors and analysts.

The information set forth in Item 7.01 of this Current Report, including Exhibit 99.1 attached hereto, is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of such section. The information set forth in Item 7.01 of this Current Report, including Exhibit 99.1 attached hereto, shall not be incorporated by reference into any filing under the Securities Act of 1933, as amended, or the Exchange Act, regardless of any incorporation by reference language in any such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Corporate Presentation.
104	Cover Page Interactive Data File-the cover page XBRL tags are embedded within the Inline XBRL document.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

BioAtla, Inc.

Date: December 20, 2024

By: /s/ Jay M. Short

Name: Jay M. Short
Title: Chief Executive Officer

Exhibit 99.1

Conditionally **A**ctive **B**iologics: Transforming Cancer Therapy

Investor Presentation
Non-Confidential

December 2024

bioatla

Important Notices & Disclaimers

This presentation (the "Presentation") by BioAtla, Inc. ("we", "us", "our", "BioAtla", or the "Company") contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 relating to our business, operations and financial conditions, including but not limited to statements regarding business plans and prospects and whether our clinical trials will support registration; achievement of milestones; results, conduct, progress and timing of our research and development programs and clinical trials; expectations with respect to enrollment and dosing in our clinical trials, plans and expectations regarding future data updates, clinical trials, regulatory meetings and regulatory submissions; plans to form collaborations or other strategic partnerships for selected assets; the potential regulatory approval path for our product candidates; expectations about the sufficiency of our cash and cash equivalents to fund operations. Words such as, but not limited to, "anticipate", "believe", "could", "estimate", "expect", "intend", "may", "plan", "potential", "predict", "project", "should", "will", "would" or the negative of those terms, and similar expressions that convey uncertainty of future events or outcomes, identify forward-looking statements.

These forward-looking statements reflect management's beliefs and views with respect to future events and are based on estimates and assumptions as of the date of this Presentation and are subject to risks and uncertainties, including those described in the Company's filings with the SEC, including but not limited to the Company's latest Annual Report on Form 10-K and any subsequently filed Quarterly Reports on Form 10-Q. Moreover, the Company operates in a very competitive and rapidly changing environment. New risks emerge from time to time. It is not possible for management to predict all risks, nor can the Company assess the impact of all factors on its business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. Given these uncertainties, you should not place undue reliance on these forward-looking statements. The Company qualifies all the forward-looking statements in this Presentation by these cautionary statements. Except as required by law, the Company undertakes no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

Statements contained herein are made as of the date of this Presentation unless stated otherwise, and this Presentation shall not under any circumstances create an implication that the information contained herein is correct as of any time after such date or that the information will be updated or revisited to reflect information that subsequently becomes available or changes occurring after that date hereof.

Certain information contained in this Presentation relates to or is based on statistical and other industry and market data obtained from independent industry publications and research, surveys and studies conducted by independent third parties as well as the Company's own estimates of the prevalence of certain diseases and conditions. The market data used in this Presentation involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data. Industry publications and third-party research, surveys and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. The Company's estimates of the patient population with the potential to benefit from treatment with any product candidates the Company may develop include several key assumptions based on its industry knowledge, industry publications and third-party research, which may be based on a small sample size and may fail to accurately reflect the addressable patient population. While the Company believes that its internal assumptions are reasonable, no independent source has verified such assumptions.

This Presentation may contain trademarks, trade names, or service marks belonging to other entities. The Company does not intend the use or display of other parties' trade names, trademarks or service marks to imply a relationship with, or endorsement or sponsorship of, or by these other parties.

None of the Company or any of its directors, officers, employees, contractors, agents, consultants, advisors or other representatives makes any representation or warranty, express or implied, as to the accuracy or completeness of the information contained in this Presentation.

BioAtla Overview

Clinical stage company harnessing the tumor microenvironment for targeted cancer therapy

- Advancing a platform of targeted "Conditionally Active Biologics (CAB)" monoclonal antibodies that bind epitopes exposed in **the acidic pH microenvironment of tumor cells,** but are otherwise shielded at normal pH of healthy cells
- Unlike prodrugs, no irreversible activation via enzymatic cleavage required for CAB antibody to bind in the acidic pH microenvironment
- CAB technology **enables enhanced (1) therapeutic exposure; (2) tumor selectivity; and (3) reduced toxicity** relative to traditional antibodies
- **Four clinical-stage and one pre-clinical stage** programs will be advanced directly or via corporate partners
- **Clinical readouts for multiple indications** with current runway into early 2026
- Headquartered in San Diego, CA with approximately 60 employees

Leadership Team

Jay Short, Ph.D.

Chairman, CEO and Cofounder

Richard Waldron, M.B.A.

Chief Financial Officer

Eric Sievers, M.D.

Chief Medical Officer

Sheri Lydick

Chief Commercial Officer

William Boyle, Ph.D.

Sr. Research Fellow

Monica Sullivan

Sr. VP, Intellectual Property & Contracts

Susie Melody

Sr. VP, Human Resources

Board of Directors and Scientific Advisors

Jay Short, Ph.D.
Chairman, Chief Executive Officer & Cofounder
Director

Lawrence Steinman, MD
Lead Director

Mary Ann Gray, Ph.D.
Director

Sylvia McBrinn
Director

Susan Moran, MD, MSCE
Director

Scott Smith
Director

Eddie Williams
Director

James Allison, Ph.D.
MD Anderson Cancer Center
Scientific Advisor

Padmanee Sharma, MD, Ph.D.
MD Anderson Cancer Center
Scientific Advisor

Lawrence Fong, MD
Cancer Immunotherapy Program, UCSF
Scientific Advisor

CAB Technology Summary

- All cancer cells are acidic (pH5.3-pH6.7)
  - The most acidic regions are oxygenated, not anaerobic
  - Acidity is a result of the need for precursor molecules from glycolysis for continuous cell replication
  - Cancer cells use acidity promote metastasis and defend against immune response
- CAB mechanism uses Protein-associated Chemical Switches (PaCS)
  - PaCS typically are negatively charged, naturally occurring small molecules (*e.g.* bicarbonate, hydrogen sulfide)
  - PaCS shield positive charges on normal tissues, which are neutralized by hydrogen ions, displacing the PaCS molecule, thereby unveiling novel epitopes for antibody binding
  - PaCS selective binding enables cancer-specific targeting of antibodies

Pre-clinical Evidence Summary

- Preclinical evidence of CAB selectivity
  - Differential EGFR tumor vs. skin binding (12.6-fold improved TI)
  - AXL-ADC reduced TMDD yielding
    - $T_{1/2}$ and exposure in NHP (>2-fold increase in $T_{1/2}$)
    - Reduced liver enzymes (>10-fold in ALT levels)
  - CTLA4 reduction of peripheral immune response while maintaining efficacy
    - Maintains efficacy at same dose, while enabling higher and extended dosing
    - Significant reduction in colitis in NHP compared to ipi
    - MTD not reached at 30 mg/kg in NHP
    - Selective reduction of activated T cells in the periphery or normal tissues
  - EpCAM DualCAB TCE maintained efficacy with highly reduced toxicity
    - MTD not reached
    - >100-fold improvement in TI
  - B7H3 DualCAB TCE associated with high acidity via hyper-glycolysis
    - MTD not reached in NHP
    - Significantly improved safety profile compared to other B7H3 TCEs in development

Clinical Evidence Summary

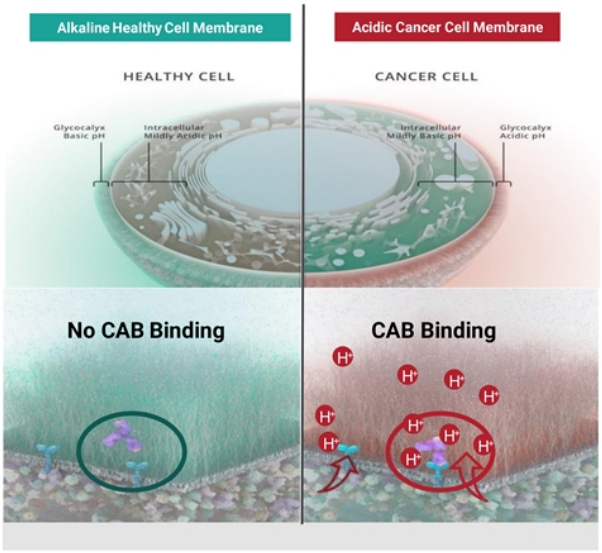
- Clinical evidence of CAB selectivity
  - AXL-ADC good risk/benefit ratio
    - Two non-CAB AXL-targeting ADCs terminated in P1
    - Potent and durable response in mKRAS NSCLC patients
  - ROR2-ADC good risk/benefit ratio
    - Good tolerability with only 7% treatment-related discontinuation rate
    - Potent and durable response in SCCHN patients
  - CTLA4 I/O enables higher and prolonged dosing with reduced immune-meditated AEs
    - Maintains PK and efficacy at similar dose, while enabling more intensive dosing
    - MTD not reached at 14.3 mg/kg
    - Extended dosing (>2x over ipi) and at higher doses, ongoing
    - Reduced grade 3 AEs such as colitis; no grade 4 or 5 AEs, even at higher doses
  - EpCAM DualCAB TCE
    - Non-CAB EpCAM TCE (BiTE) terminated in P1
    - Most advanced EpCAM TCE in the clinic showing tumor-reduction, ongoing in P1
    - MTD not yet reached

CABs demonstrate universal clinical improvement in TI and enable therapeutic development "undruggable" targets

Selective and Targeted CAB Technology Widens Therapeutic Window

Thus has the potential to enhance clinical outcomes in multiple tumor types

- BioAtla discovered that acidic pH at the cancer cell surface unveils binding sites that are shielded at normal pH of healthy cells
- BioAtla invented CAB technology, creating antibodies that bind ***only*** to these unveiled sites on cancer cells
- CAB binding region is not masked or caged and thus different from prodrugs that require irreversible enzymatic cleavage to become activated
- CAB antibodies have the potential for increased efficacy with improved safety relative to traditional antibodies

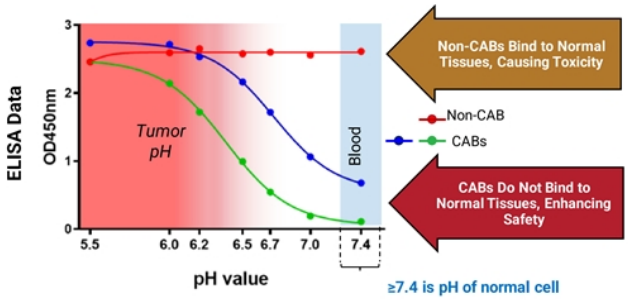


Chang, H.W., Frey, G., Liu, H., Xing, C., Steinman, L, Boyle, B.J., & Short, J.M. (2021) PNAS 118(9): 1-10, Suppl. 1-19.

CAB Antibodies Bind Selectively and Reversibly Based on the Tumor Microenvironment (TME)

Enhancing exposure and reducing toxicity

CABs Bind Selectively in the Lower pH TME



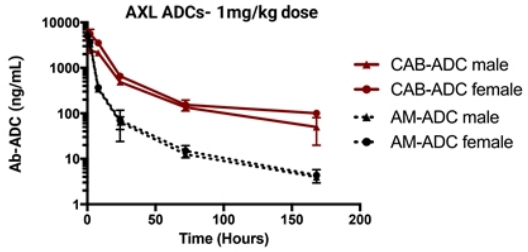
Selective Binding → Focused Tumor Killing → Normal Cells Preserved

Note: OD450nm = optical density measurements using a microplate reader with a 450nm filter; TME = Tumor Micro Environment; mABs = monoclonal antibodies; Data above based on non-human primate studies

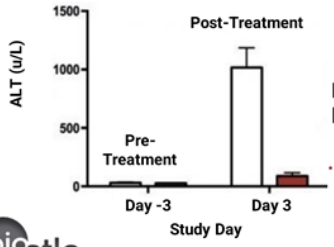
CAB ADCs Show Strong Selectivity and Have an Improved Therapeutic Window vs Traditional ADCs

Unlike prodrugs, CABs are reversible and selective enhancing the therapeutic index

Enhanced ADC Therapeutic Exposure

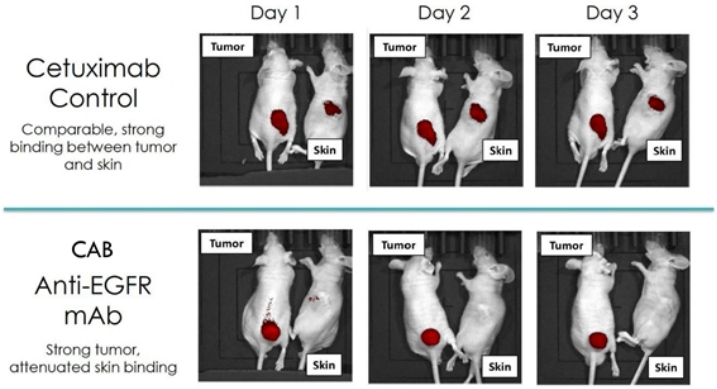


Reduced ADC Toxicity



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

Enhanced ADC Tumor Selectivity (12.6-fold increase in TI)



Normal (skin patch) versus Tumor Binding by anti-EGFR versus CAB-anti-EGFR

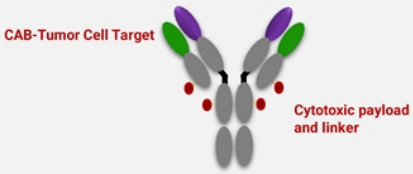
Note: OD450nm = optical density measurements using a microplate reader with a 450nm filter; Data above based on non-human primate studies; AM = affinity matched; ALT or alanine aminotransferase is a sign of liver toxicity

Broad Applicability of BioAtla's CAB Platform Across Several Antibody Types

ADCs

Targets: ROR2, AXL

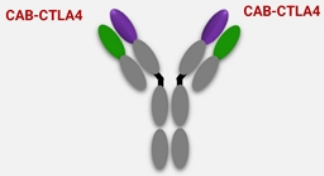
Widely expressed in a variety of tumor types, ROR2 and AXL overexpression correlates with poor prognosis, metastasis, and drug resistance to PD-1 and EGFR therapies



I/O Antibodies

Target: CTLA-4

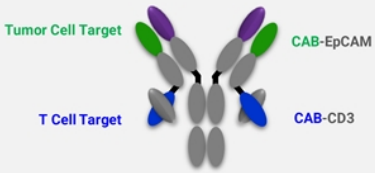
CTLA-4 blockade activates effector T cells, thereby enhancing anti-tumor immunity



Bispecific TCE

Target: EpCAM & CD3

Bispecific antibodies bridge cancer cells and cytotoxic T lymphocytes, activating T cells and promoting cancer cell lysis



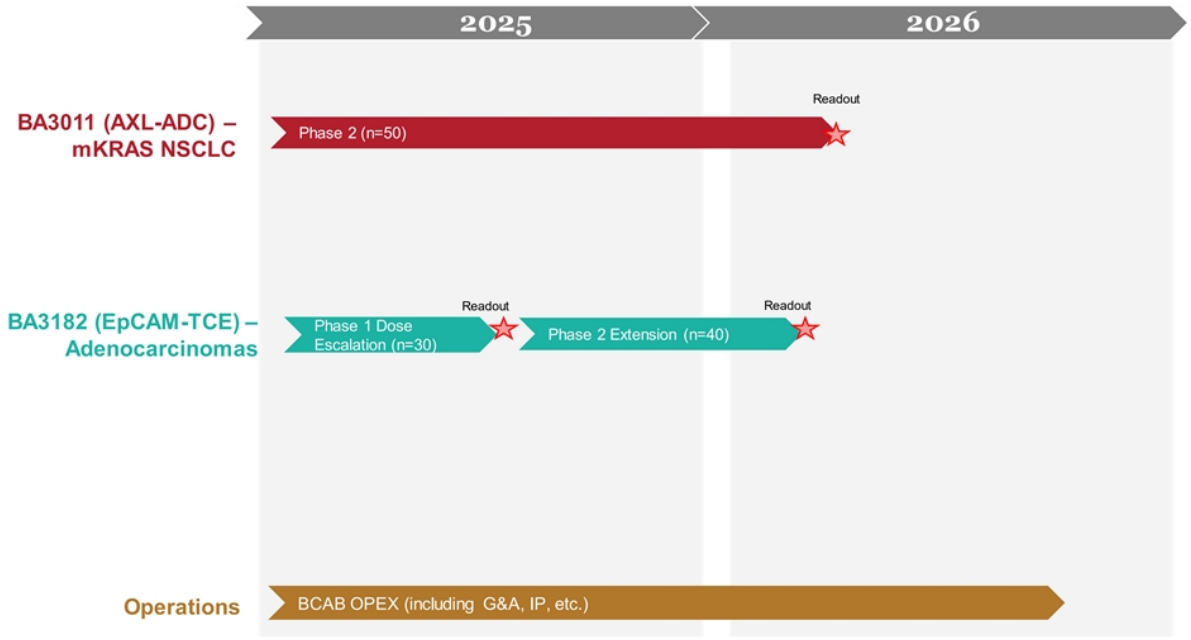
ADC – antibody drug conjugate; IO – immuno-oncology; TCE – T-cell engager

Focused Pipeline with Broad Applicability of Differentiated CAB Assets Designed to Deliver Near-term value

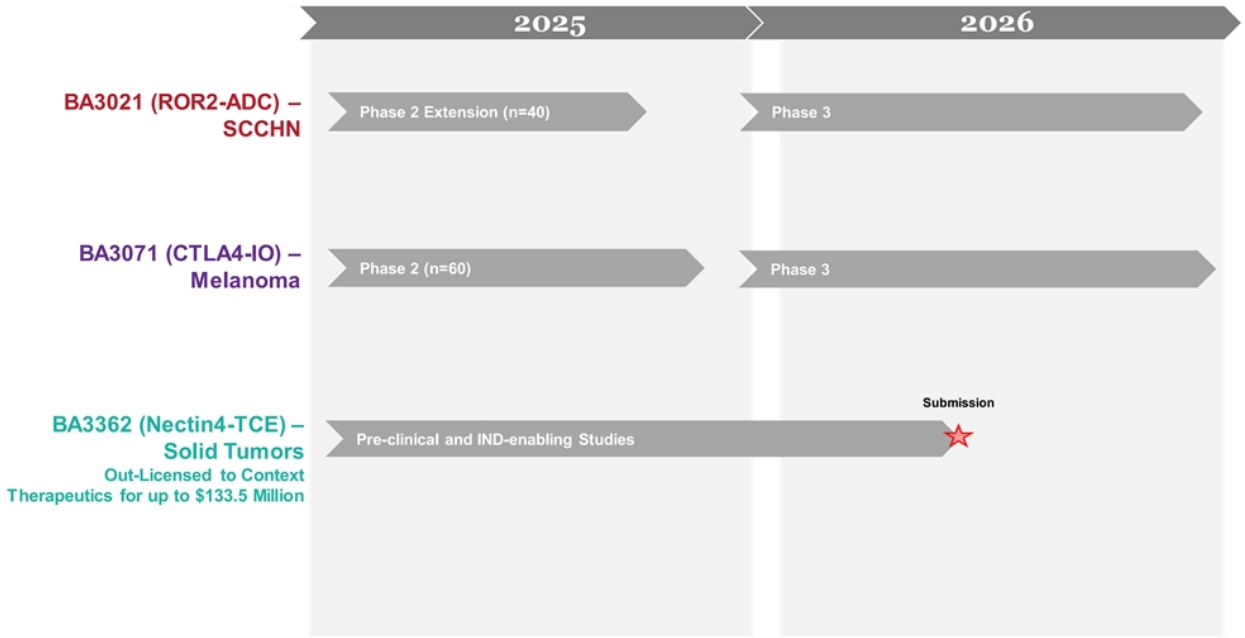
	CAB Program	Target	Indications	IND Enabling Pre-Clinical	Phase 1 Clinical	Phase 2 Clinical
CAB-ADCs	*Mecbotamab Vedotin BA3011*	AXL	NSCLC UPS			
	Ozuriftamab Vedotin BA3021	ROR2	SCCHN			
CAB-I/O	*Evalstotug BA3071*	CTLA-4	Melanoma NSCLC			
CAB-Bispecific TCE	BA3182	EpCAM x CD3	Adenocarcinomas			
CAB-Bispecific TCE	BA3142	B7H3 x CD3	Multiple tumor types			
CAB-Bispecific TCE	BA3382 (Out-Licensed to Context Therapeutics for up to $133.5 Million)	Nectin4 x CD3	Multiple tumor types			

IND, investigational new drug; UPS, Undifferentiated Pleomorphic Sarcoma; NSCLC, Non-small Cell Lung Cancer; SCCHN, Squamous Cell Carcinoma of the Head and Neck

BioAtla's Near-Term Catalysts



BioAtla's Near-Term Catalysts with Potential Partnering Opportunities

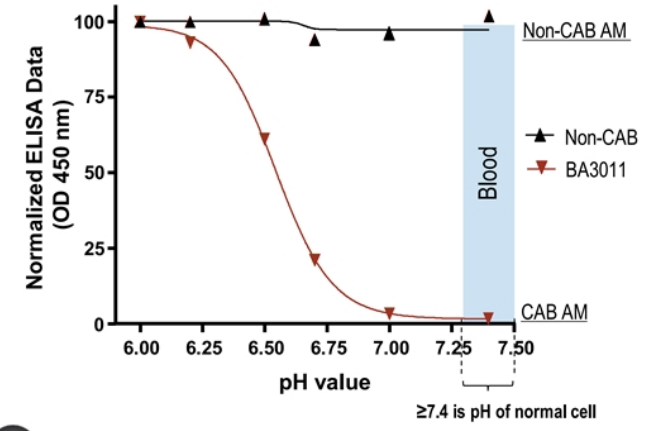


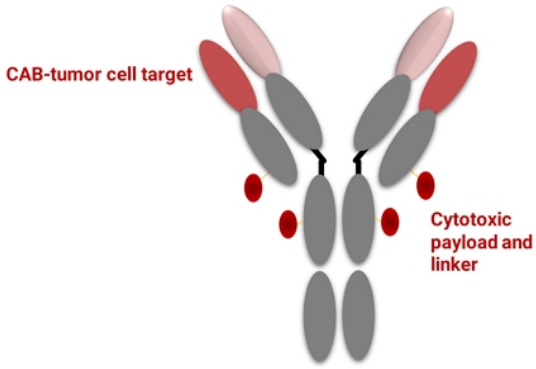
Mecbotamab Vedotin (CAB-AXL-ADC): mKRAS Non-Small Cell Lung Cancer (NSCLC)

Mecbotamab Vedotin: CAB-AXL-ADC

AXL is expressed in a variety of tumor types, with overexpression associated with metastasis, tumor resistance to chemotherapy, and poor prognosis

BA3011 pH binding inflection point adjusted for tumor microenvironment selectivity





- Humanized anti-AXL IgG1
- ~100 pM affinity (pH 6.5)
- VC-MMAE (DAR 4) linker and payload
- Epitope in Ig loop region

1. Gay CM, Balaji K, Byers LA. *Br J Cancer*. 2017;116(4):415-423. 2. Zhang G, Wang M, Zhao H, Cui W. *Oncol Lett*. 2018;15(3):2726-2734.

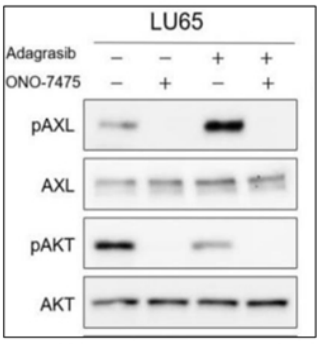
Abbreviations: ADC, antibody-drug conjugate; AM, affinity matched; CAB, conditionally active biologic; DAR, drug antibody ratio; ELISA, enzyme linked immunosorbent assay; Ig, immunoglobulin; OD, optical density; VC-MMAE, valine-citrulline monomethylauristatin E.

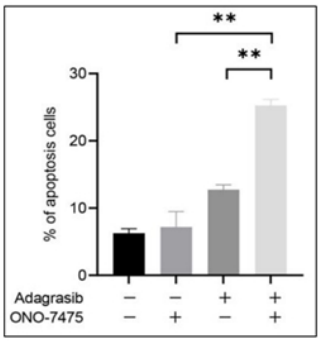
Co-expression of mutant KRAS and AXL is Significant and Functionally Linked

- In lung cancer cells, AXL over-expression is involved in resistance to chemotherapy and targeted therapies and drives epithelial to mesenchymal transition
- Prevalence of AXL expression in KRAS mutant NSCLC is significant, the majority of these tumors exhibit high levels of AXL
    - Upregulation is associated with aggressive tumor characteristics, resistance to therapies, and poor patient outcomes.
- AXL signaling mediates the adaptive resistance to mKRAS inhibitors adagrasib and sotorasib (G12C) in lung cancer (85% AXL positive) (Morimoto et al., Cancer Letters 587 (2024) 216692)
- Potential significant opportunity for mecbotamab vedotin to improve outcomes in the mKRAS population across mKRAS variants

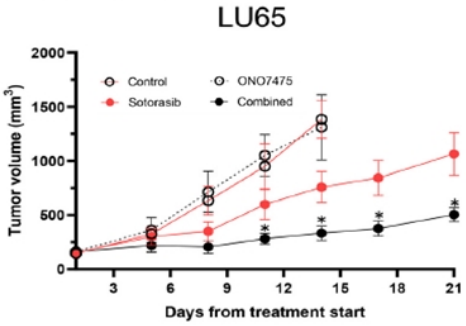
AXL Plays a Crucial Role in the Survival of KRAS G12C mutant NSCLC Cells Treated with KRAS G12C Inhibitors

AXL driving resistance to KRAS inhibitors

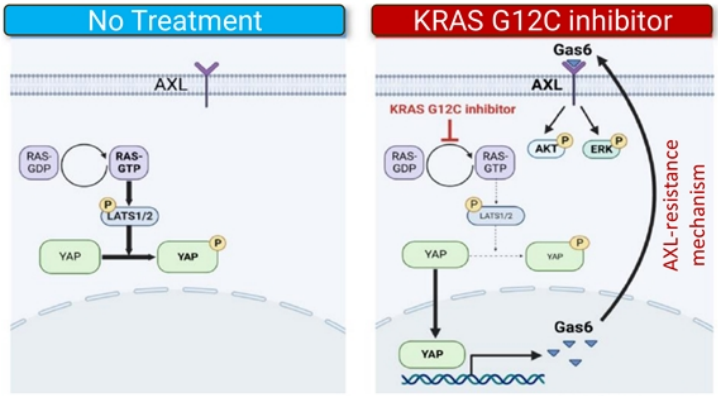




p = Phosorylated form of protein



- **KRAS inhibition leads to upregulation and activation of AXL expression**
  - Autocrine/paracrine of increased GAS6 expression
- **AXL inhibition alone does not lead to tumor killing, but potentiates anti-tumor effects of KRAS inhibition**
  - These results indicated pivotal roles for the YAP-GAS6-AXL axis and its inhibition in the intrinsic resistance to KRAS G12C inhibitors
- **AXL-ADC improves outcomes via tumor killing**



Morimoto et al. Cancer Letters 587 (April 2024)

Docetaxel (expected control arm) has modest efficacy in 2L+ NSCLC

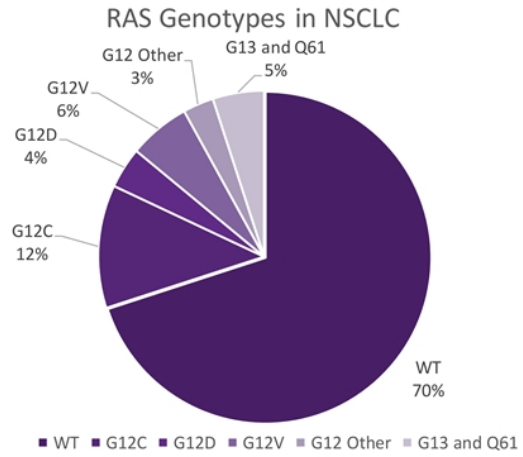
Codebreak and Krystal has G12C docetaxel 2L+ data

No pan-KRAS docetaxel 2L+ data

Study with Docetaxel in 2L+ NSCLC	Timing relative to Immune checkpoint inhibitors	Pts	ORR	Median PFS	Median OS
REVEL[1]	Prior	625	14%	3.0	9.1
CheckMate 057[2]	Prior	290	12%	4.2	9.4
OAK[3]	Prior	425	13%	4.0	9.6
POPLAR[4]	Prior	143	14.7%	3.0	9.7
TROPION-Lung-01[6]	After	305	13%	3.7	11.8
Codebreak 200[5] **(KRAS G12C)**	After	174	13.2%	4.5	11.3
KRYSTAL-12[7] **(KRAS G12C)**	After	152	9.2%	3.8	NA

(1) Garon EB, et al. Lancet 2014;384:665-673; (2) Borghaei H, et al. N Engl J Med 2015; 373:1627-1639; (3) Rittmeyer A, et al. Lancet 2017;389:255-265; (4) Fehrenbacher L, et al. Lancet 2016;387:1837-1846; (5) de Langen A.J, et al. Lancet 2023;401733-746; (6) Ahn MJ, J Clin Oncol. 2024 Sep 9;JCO2401544. doi: 10.1200/JCO-24-01544. ; (7) Mok TS, J Clin Oncol. 2024;42(17_suppl):LBA8509.
NSCLC, non-small cell lung cancer; CPI, check point inhibitor; ORR, objective response rate; PFS, progression-free survival; OS, overall survival; 1L, first line; 2L, second line.

- 234,580 new cases of NSCLC expected in US for 2024
- 30% or 70,374 new cases of NSCLC with RAS mutations in US for 2024



Emerging Opportunity for Mecbotamab Vedotin in Patients with Mutated KRAS (mKRAS) Variants

mKRAS constitutes 30% of all NSCLC patients

mutant KRAS; all NSCLC (SQ+NSQ); median of approximately 3 prior lines of tx for both mKRAS and wtKRAS

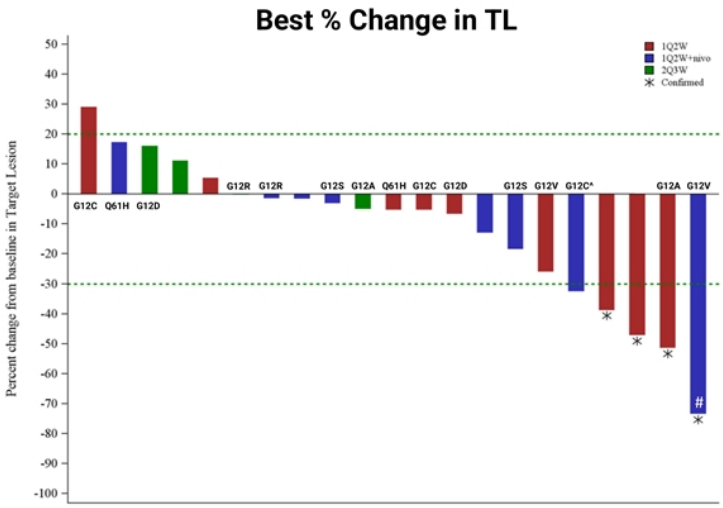
	Total (N=78)
Age, y, mean (range)	**67 (46-82)**
KRAS Status, n (%)	
WtKRAS	**50 (64)**
mKRAS	**24 (31)**
Unknown KRAS status	**4 (5)***

* Two responders with no additional biopsy sample for KRAS mutation assessment

Data Cut Date: Live database as of 24Oct2024

Confirmed Responses with Mecobotamab Vedotin Across mKRAS Variants - *ongoing*

N=21 of 24**; 1.8 mg/kg Q2W, 2Q3W, and Q2W+nivo



^ Patient was previously treated with Sotorasib
# Complete Response as defined by disappearance of all pathologic lymph nodes
*Confirmed responses

Median of 3 prior lines of tx	mKRAS N=21
BOR all	5 (24%)
BOR confirmed	4 (19%)
DCR	76%
DOR	4.8 months
PFS	4.6 months
OS	12.6 months

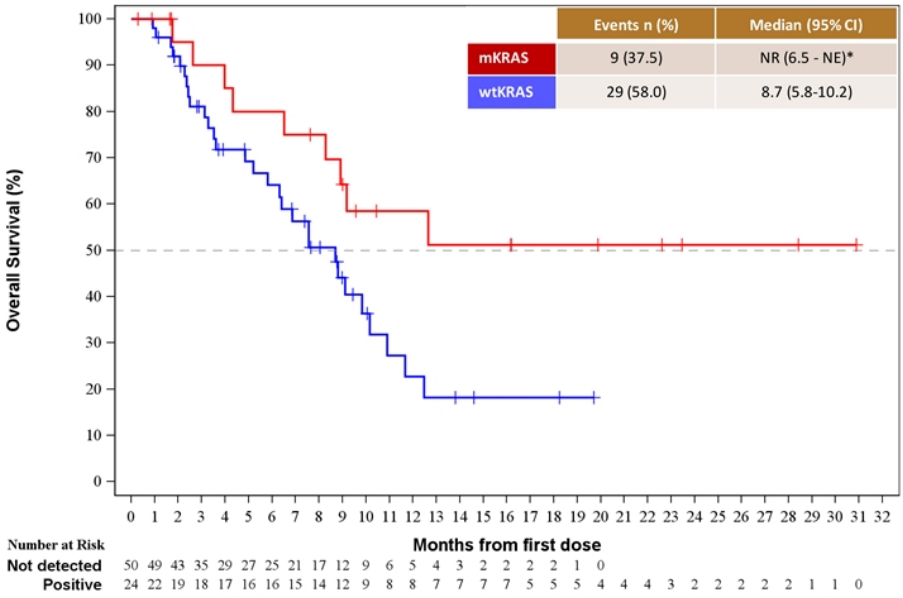
**Evaluable patients defined as patients that had at least 1 scan after treatment with study drug
Prior to first scan:
Two patient withdrew consent
One patient DC due to AE

Data Cut Date: Live database as of 24Oct2024

ORR: overall response rate; AEs: adverse events; DOR: duration of response; PFS: progression-free survival; BOR: best overall response; DCR: disease control rate; TTR: time to response; OS: overall survival

Improved Overall Survival with Mecbotamab Vedotin among NSCLC Patients with Tumors Expressing mutant KRAS Variants Compared to KRAS Wildtype

Median of 3 prior lines of tx

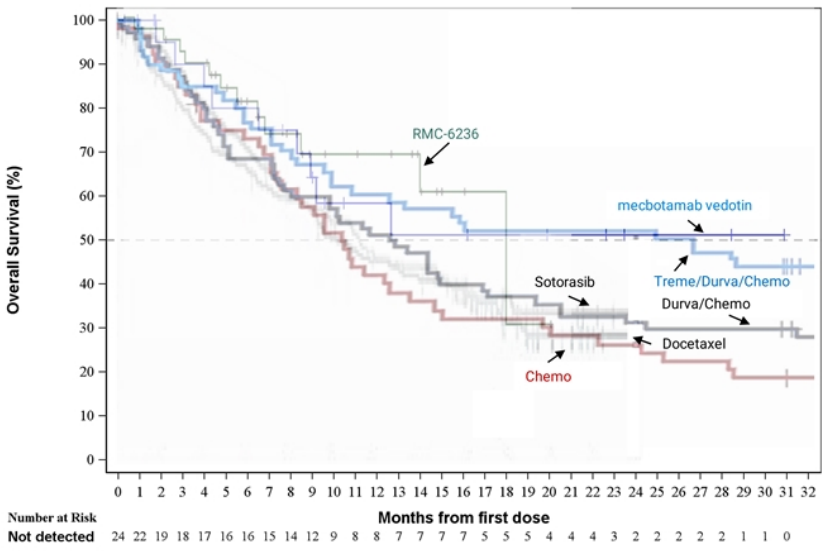


*Ongoing, >50% survival @ 1yr.
Data Cut Date: Live database as of 24Oct2024

Overall Survival comparison between mecbotamab vedotin vs competition

mKRAS median of 3 prior lines of tx

	mecbotamab vedotin	RMC-6236	Treme/Durva/Chemo	Sotorasib
Population	mKRAS	RAS G12X	mKRAS	G12C
Number of patients	24	73	113	171
Prior Lines of tx	2-3	1	0	1
ORR	19% / 29%	38% / ?	50%	28 to 36%
OS	Not reached	17.7 months	25.7	10.6
Median prior lines (range)	3 (1 to 9)	2 (0 to 6)	0	2
Related Gr3-Gr4 events	33%	16%	51.8%	33%
Related AEs leading to treatment d/c	7%	4%	10.9%	9%



Data Cut Date: 21Oct24 Live Database

Ph2 Mecbotamab Vedotin: Overall Safety Summary of NSCLC patients

Generally well-tolerated

	1.8 mg/kg Q2W (N=26)	1.8 mg/kg 2Q3W (N=33)	1.8 mg/kg Q2W + Nivo (N=19)	Total (N=78)
Any Adverse Events (AEs)	26 (100)	33 (100)	19 (100)	**78 (100)**
Related AEs with CTCAE[1] Grade 3 or 4[2]	10 (39)	12 (36)	4 (21)	**28 (33)**
Any Related Serious AEs[2]	4 (15)	3 (9)	1 (5)	**8 (9)**
Possibly Related AEs leading to death[2]	0	0	0	**0**
Related AEs leading to treatment discontinuation[2]	1 (4)	4 (12)	1 (5)	**6 (7)**

[1]CTCAE: 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.
[2]As assessed by the investigator. Missing responses are counted as related.

Data Cut Date: 10Jul2024

Mecbotamab Vedotin: Phase 2 Safety Data of NSCLC patients

Most frequent treatment-emergent adverse events irrespective of causality (≥15%)

	BA3011 Q2W (N=26)		BA3011 2Q3W (N=33)		BA3011 Q2W + Nivo (N=19)		TOTAL (N=78)	
Preferred Term	**All Grades**	**Grade 3-4**	**All Grades**	**Grade 3-4**	**All Grades**	**Grade 3-4**	**All Grades**	**Grade 3-4**
Number of Subjects with at Least One TEAE	26 (100)	17 (65)	33 (100)	18 (55)	19 (100)	9 (47)	78 (100)	44 (56)
Fatigue	12 (46)	1 (4)	8 (24)	2 (6)	8 (42)	0	28 (36)	3 (4)
Diarrhoea	8 (31)	1 (4)	12 (36)	2 (6)	6 (32)	0	26 (33)	3 (4)
Decreased Appetite	6 (23)	1 (4)	13 (39)	0	6 (32)	0	25 (32)	1 (1)
Neuropathy¥	8 (31)	1 (4)	12 (36)	0	4 (21)	0	24 (31)	1 (1)
Nausea	6 (23)	0	9 (27)	0	8 (42)	0	23 (29)	0 (0)
Neutropenia*	9 (35)	3 (12)	8 (24)	7 (21)	1 (5)	0	18 (23)	10 (13)
Constipation	8 (31)	0	9 (27)	1 (3)	5 (26)	0	22 (28)	1 (1)
Anaemia	3 (12)	1 (4)	5 (15)	1 (3)	6 (32)	2 (11)	14 (18)	4 (5)
Aspartate Aminotransferase Increased	5 (19)	2 (8)	5 (15)	0	4 (21)	1 (5)	14 (18)	3 (4)
Alanine Aminotransferase Increased	5 (19)	2 (8)	5 (15)	0	3 (16)	1 (5)	13 (17)	3 (4)
Arthralgia	3 (12)	0	7 (21)	0	3 (16)	0	13 (17)	0 (0)
Back Pain	4 (15)	0	7 (21)	0	3 (16)	1 (5)	14 (18)	1 (1)

* Derived from neutropenia, and neutrophil count decreased

¥ Derived from neuropathy peripheral, peripheral motor neuropathy, and peripheral sensory neuropathy

Data Cut Date: 10Jul2024

Mecbotamab Vedotin NSCLC Summary

Median of 3 prior lines of tx

- Promising anti-tumor activity among patients whose tumors express KRAS mutations
  - mKRAS represents 30% of all NSCLC patients and is associated with increased AXL expression
  - Improved overall survival observed among treated patients with tumors expressing mutated KRAS variants (12.6 months) compared to KRAS wildtype (8.7 months)
  - Anti-tumor activity across nine different KRAS mutation variants
    - Partial response observed in a patient who had experienced prior failure of sotorasib
    - Patient treated with mecbotamab vedotin + anti-PD-1 antibody remains in complete response for >2 years
- Potential for a pan mKRAS strategy in NSCLC; currently determining the most efficient path forward for a future pivotal trial

BA3182 (EpCAM x CD3 Bispecific T-Cell Engager): Adenocarcinoma

CAB-EpCAM x CAB-CD3 Bispecific T-Cell Engager (BA3182)

Significant opportunity for safe and effective EpCAM x CD3 bispecific

- EpCAM is an attractive therapeutic target because it's expressed in most solid tumors, as well as in normal epithelial tissues, well suited for CAB technology
- BA3182 exhibits efficient tumor shrinkage with encouraging safety profile in vitro and in vivo[1]
- In non-GLP and GLP tox studies in NHP, dual selection results in high selectivity with 100-fold therapeutic index (TI) increase[1]
- Phase 1 dose escalation ongoing
  - Observed multiple patients with tumor reduction, including one colorectal cancer patient with stable disease for one year (ongoing)
  - Maximally tolerated dose has not yet been reached
  - Implemented priming dose to modulate cytokine release syndrome that is commonly observed with T-cell engagers and can also occur in patients with heavy tumor volume
- Given encouraging continued ongoing dose escalation with increasing antitumor activity, we now anticipate data readout of Phase 1 study around mid-2025

[1]Gerhard Frey, Ana Paula G. Cugnetti, Haizhen Liu, Charles Xing, Christina Wheeler, Hwai Wen Chang, William J. Boyle & Jay M. Short (2024) A novel conditional active biologic anti-EpCAM x anti-CD3 bispecific antibody with synergistic tumor selectivity for cancer immunotherapy, mAbs, 16:1, 2322562, DOI: 10.1080/19420862.2024.2322562

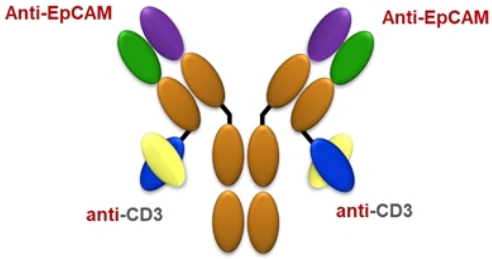
EpCAM is Widely Expressed Across Solid Tumors

4 most common new cases of cancers from American Cancer Society and EpCAM Expression

	ACS estimate for 2024 in US	EpCAM Expression (TIS 1 to 12)
Breast Cancer	310,720	81%
Prostate Cancer	299,010	99%
Lung Cancer	234,580	93% NSCLC/ 80% SCLC
Colon Cancer	106,590	100%
Pancreatic Cancer	66,440	99%
Thyroid Cancer	44,020	97%

Overview of the Structure and Activity of BA3182

Dual CAB EpCAM x CAB CD3 Bispecific antibody



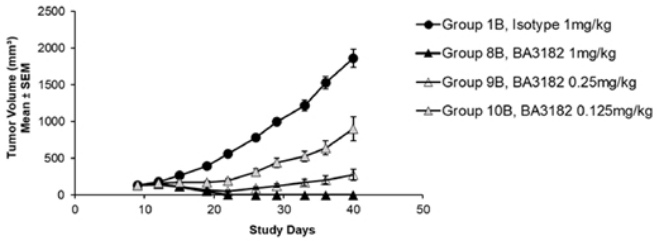
- BA3182 humanized anti-EpCAM IgG1 antibody with a humanized anti-CD3ε scFv fused to the C-terminus of the IgG1 kappa light chain constant domain.
- H1-L1 expression configuration
- CHO product MW = 198,981 Da

Potency of BA3182 in Mediating T Cell Activation in a Bioassay

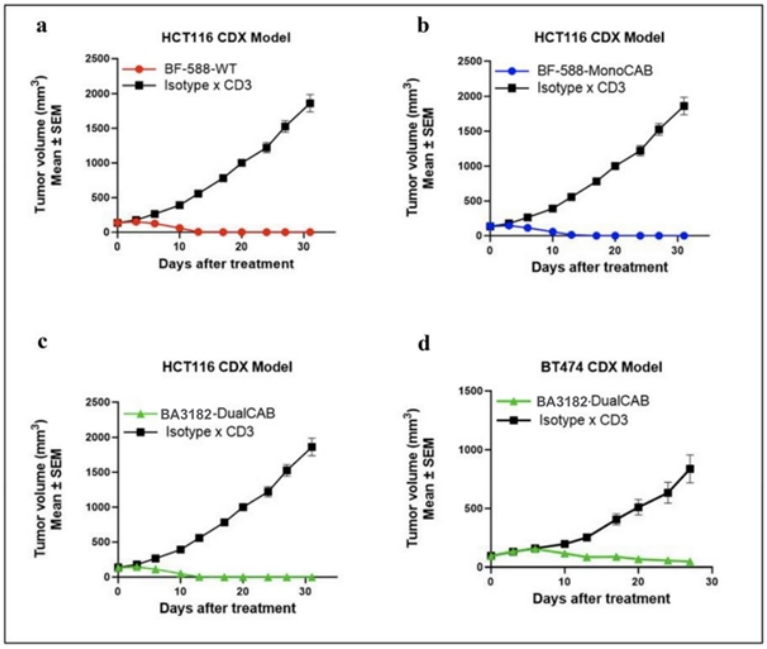
	EC_{50} (nM) pH6.0	EC_{50} (nM) pH7.4
HCT116	0.273 ± 0.030	1.99 ± 0.147
CHO huEpCAM	0.049 ±0 0.002	0.383 ± 0.023
CHO cynoEpCAM	0.576 ± 0.027	9.65 ± 0.932

Abbreviations: CHO, Chinese hamster ovary; EC_{50}, half maximal effective concentration.

***In Vivo* Efficacy of BA3182 Against HCT116 CDX Model in Humanized NOG Mice**

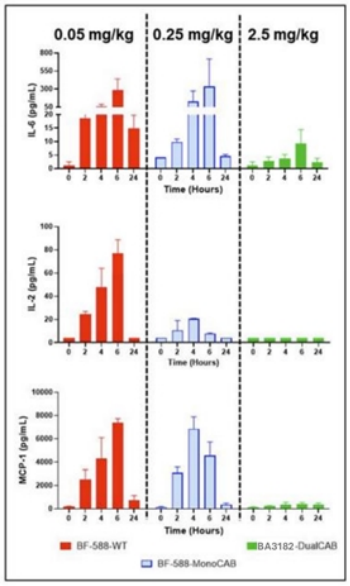


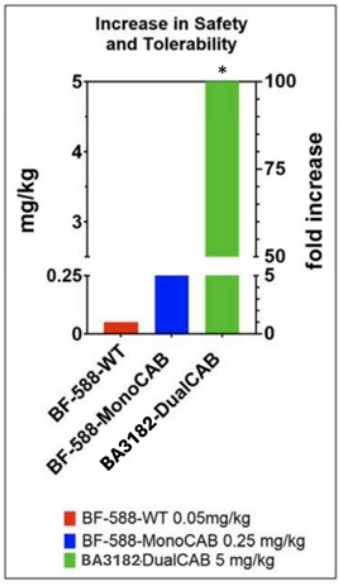
MonoCAB and BA3182 DualCAB Inhibit Tumor Growth *in vivo*



Source: MABS 2024, VOL. 16, NO. 1, 2322562 https://doi.org/10.1080/19420862.2024.2322562

BA3182 DualCAB Shows Significant Reduction of Immunotoxicity and Highly Increased Tolerability in Cynomolgus Monkey





Tox. Study Type	Single-Dose Non-GLP			Repeat-Dose GLP
Test Article	**BF-588-WT**	**BF-588-MonoCAB**	**BA3182 DualCAB**	**BA3182-DualCAB**
Dose	**0.05mpk***	**0.25mpk**	**2.5mpk**	**5mpk**
	Not tolerated	Maximum Tolerated Dose (MTD)	Did not reach MTD	Did not reach MTD
Clinical Outcome	Euthanized on Day 8 (2/2)[a]	Returned to colony (2/2)	Returned to colony (2/2)	No clinical signs, schedule euthanasia (10/10)
Findings	Inappetence, Emesis, Hunched Posture, BWL, Hepatocellular, Hepatobiliary, Kidney and Intestinal injury	Minimal elevation of liver enzymes; no clinical observations	No clinical observations	No clinical observations, no macroscopic and no microscopic findings

Source: MABS 2024, VOL. 16, NO. 1, 2322562 https://doi.org/10.1080/19420862.2024.2322562

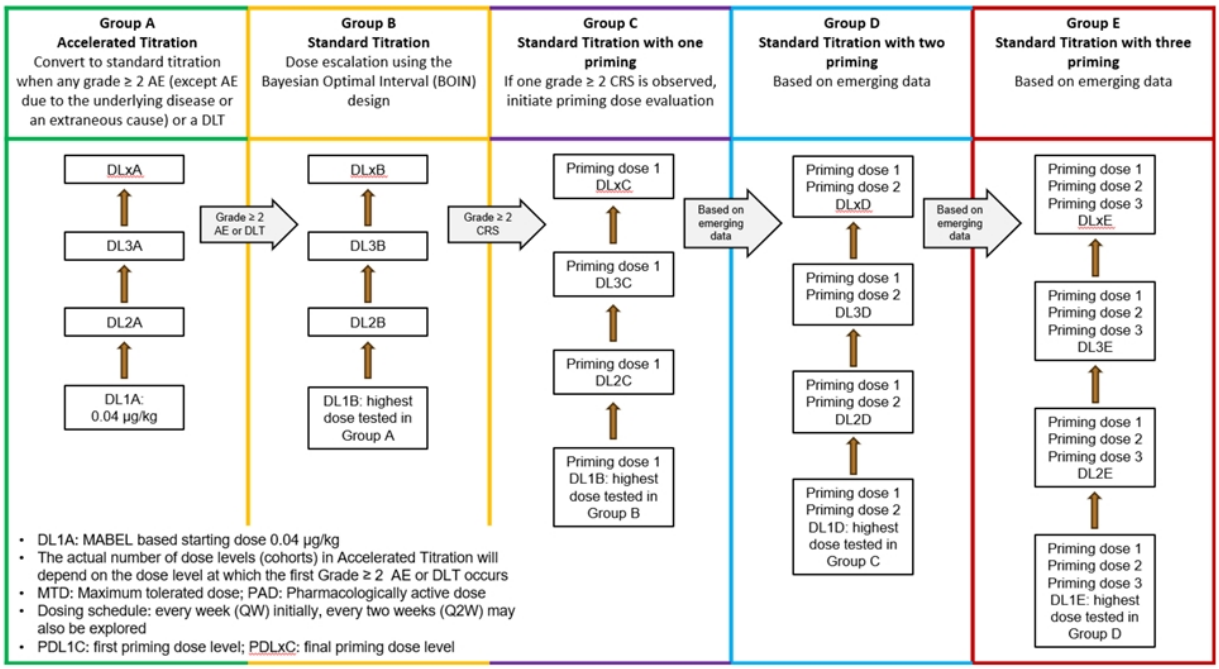
Amgen Discontinued Development Of Solitomab Due to DLTs Which Precluded Dose Escalation to Potentially Therapeutic Levels

Solitomab, a bispecific T-cell engager (BiTE®) antibody construct targeting EpCAM

- Sixty-five patients received solitomab at doses between 1 and 96 µg/day for ≥28 days
- Overall, 95% of patients had grade ≥3 treatment-related AEs, primarily diarrhea, elevated liver parameters, and elevated lipase
- Fifteen patients had dose-limiting toxicities (DLTs):
  - Eight had transient abnormal liver parameters shortly after infusion start or dose escalation (grade 3, n = 4; grade 4, n = 4)
  - One had supraventricular tachycardia (grade 3)
  - Six patients had a DLT of diarrhea: (grade 3, n = 4; grade 4, n = 1; grade 5, n = 1)
- Limited anti-tumor activity
  - 1 unconfirmed partial response
  - 17 stable disease
  - 28 disease progression
  - 19 were not evaluable

Kebenko, M., et al. (2018). A multicenter phase 1 study of solitomab (MT110, AMG 110), a bispecific EpCAM/CD3 T-cell engager (BiTE®) antibody construct, in patients with refractory solid tumors. *OncoImmunology*, *7*(8). https://doi.org/10.1080/2162402X.2018.1450710

BA3182 - Phase 1 Study Design



Abbreviations: AE, adverse event; CRS, cytokine release syndrome; DL, dose level; DLT, dose-limiting toxicity; MABEL, minimum anticipated biological effect level.

EpCAM Expression in Adenocarcinoma

Validated commercial antibody for EpCAM

Original article

EpCAM expression in primary tumour tissues and metastases: an immunohistochemical analysis

EpCAM expression was assessed in 2,291 primary tumor tissues and in 108 metastases using the EpCAM-specific antibody.

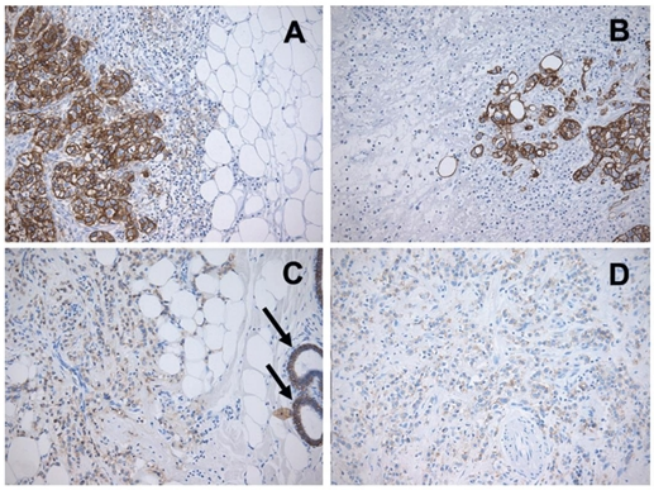


Figure 1 Comparison between immunohistochemical epithelial cell adhesion molecule (EpCAM) expression in primary breast cancer and metastases. (A) Invasive ductal carcinoma with strong EpCAM expression (total immunostaining score (TIS) 12). (B) Brain metastases from the same patient showing strong EpCAM expression (TIS 12). (C) Invasive lobular carcinoma with weak EpCAM expression (TIS 3); arrows: normal breast ducts showing EpCAM expression. (D) Peritoneal metastases from the same patient showing weak EpCAM expression (TIS 3).

J Clin Pathol 2011;64:415e420.
doi:10.1136/jcp.2011.090274

Frequent High-Level Expression of the Immunotherapeutic Target EpCAM in Colon, Stomach, Prostate and Lung Cancers

British Journal of Cancer (2006) 94, 128 – 135

www.bjcancer.com

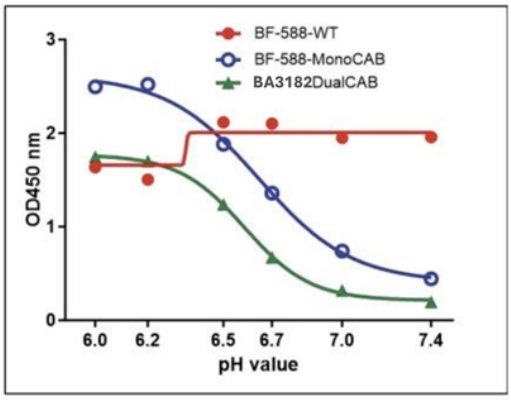
TABLE 1. Expression of EpCam in Normal Tissue

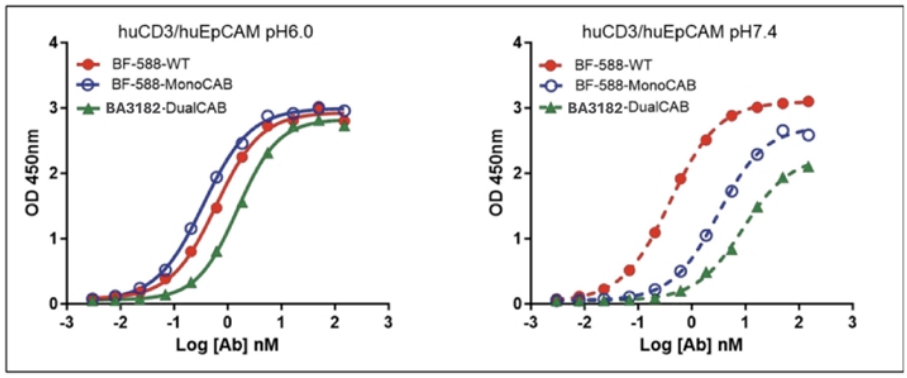
Normal tissue	Intensity
Breast, lobular epithelium	+
Cervix, epithelium	++
Colon, epithelium	+++
Endometrium, epithelium	+++
Esophagus, basal squamous cell epithelium	+
Gall bladder, epithelium	+++
Kidney, cortical collecting duct epithelium	++
Liver, small bile duct epithelium	+++
Lung, alveolar epithelium	+
Pancreas, acinar epithelium	+++
Parathyroid	++
Parotis, acinar epithelium	++
Prostate, secretory cells	++
Skin, basal cells	+
Small intestine, epithelium	+++
Stomach, epithelium	+
Submandibular gland, acinar epithelium	+
Testis, spermatogonia	+
Thymus, epithelial cells	+
Thyroid, follicular epithelium	++
Urinary bladder, epithelium	+

Table 2 Frequency of Ep-CAM overexpression in major human cancers

Tumour entity	*n*	Ep-CAM expression: Negative (%)	Weak/ moderate (%)	Strong (%)
All	3360	5.9	11.6	82.5
Colon				
Adenocarcinoma NOS	1086	0.3	1.7	98.0
Medullary carcinoma	5	20.0	0	80.0
Mucinous carcinoma	88	0	4.5	95.5
Signet ring cell carcinoma	4	0	0	100.0
Other types	3	0	0	100.0
Stomach				
Stomach carcinoma	473	2.5	6.8	90.7
Lung				
Adenocarcinoma NOS	317	5.3	13.9	80.8
Adenosquamous carcinoma	11	45.4	18.2	36.4
Bronchioloalveolar carcinoma	60	3.3	21.7	75.0
Large cell carcinoma	224	15.1	17.9	67.0
Neuroendocrine carcinoma	56	16.1	19.6	64.3
Squamous cell carcinoma	619	17.3	29.1	53.6
Prostate				
Prostate carcinoma	414	1.9	10.9	87.2

In vitro Characterization of BF-588-WT, BF-588-MonoCAB and BA3182-DualCAB





EC_{50} (nM)	pH6.0	pH7.4
BF-588-WT	0.61	0.40
BF-588-MonoCAB	0.35	3.14
BA3182-DualCAB	1.52	9.09

Source: MABS 2024, VOL. 16, NO. 1, 2322562 https://doi.org/10.1080/19420862.2024.2322562

BA3182 Bispecific TCE Antibody has >80-fold Higher Therapeutic Index Compared to non-CAB Bispecific

Maximum Tolerated Dose

Therapeutic Index (TI)

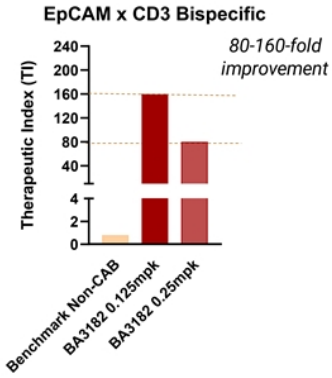
Minimum Effective Dose

- Minimal effective dose (MED) determined in the mouse-based animal models
- MTD/Highest Non-severely Toxic Dose (HNSTD) determined in non-human primate (NHP)*

TI = **HNSTD (mg/m²) in Non-human primate***
Mouse minimal tumor repression dose (mg/m²)

Antibody	Dose Type	mg/kg	mg/m2	Therapeutic Index (TI)
Benchmark	MED (mouse)	0.125	0.375	0.8
	MTD (NHP)	0.025	0.300	
BA3182	MED (mouse)	**0.125**	0.375	160
	MTD (NHP)	5	60	
BA3182	MED (mouse)	**0.25**	0.750	80
	MTD (NHP)	5	60	

**Maximum tolerated dose not achieved in NHP*



BA3142 (B7H3 x CD3 Bispecific T-Cell Engager): Pre-clinical

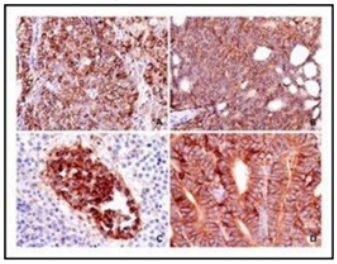
B7H3 Background

Large commercial potential

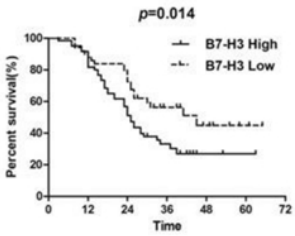
High expression across many different solid tumors

- B7H3 is a member of B7 family of immunoregulatory transmembrane glycoproteins expressed by T cells.
- B7H3 is overexpressed on many solid cancers and displays relatively high tumor-versus-normal tissue binding differential.
- B7H3 overexpression has been correlated with disease severity and poor prognosis in many cancer types including:
  - Head and Neck (male only 41,510 est. new US cases in 2024)*
  - Lung (234,580 est. new US cases in 2024)*
  - Melanoma (100,640 est. new US cases in 2024)*
  - Pancreatic (66,050 est. new US cases in 2024)*
  - Prostate (299,020 est. new US cases in 2024)*
- B7H3 it also has immune-independent roles outside T cells and plays a part in the metabolic reprogramming of cancer cells in vitro and in vivo B7-H3
  - Promotes the Warburg effect effecting extracellular tumor pH

B7-H3 Expression in Prostate



Lung

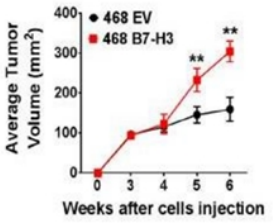


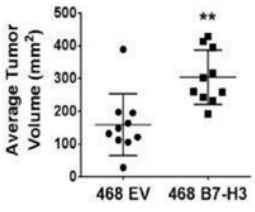
* @2024, American Cancer Society Inc., Surveillance and Health Equity Source

B7H3 is exceptional for CAB targeting

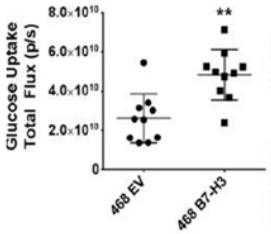
Enhances the Warburg effect in tumors

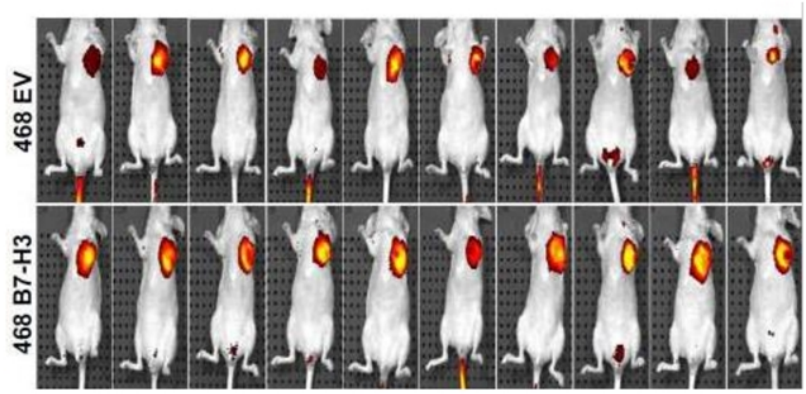
- **B7H3 expression increases tumor cell aerobic glycolysis and acidic pH in TME**
- **Regulates the expression of HIFα leading to elevated LDH and PDK1**





MDA-MB-468 B7-H3 overexpressing breast cancer cells (468 B&-H3) or transduced with empty vector (EV)





Overexpression of B7H3 in cancer cell lines

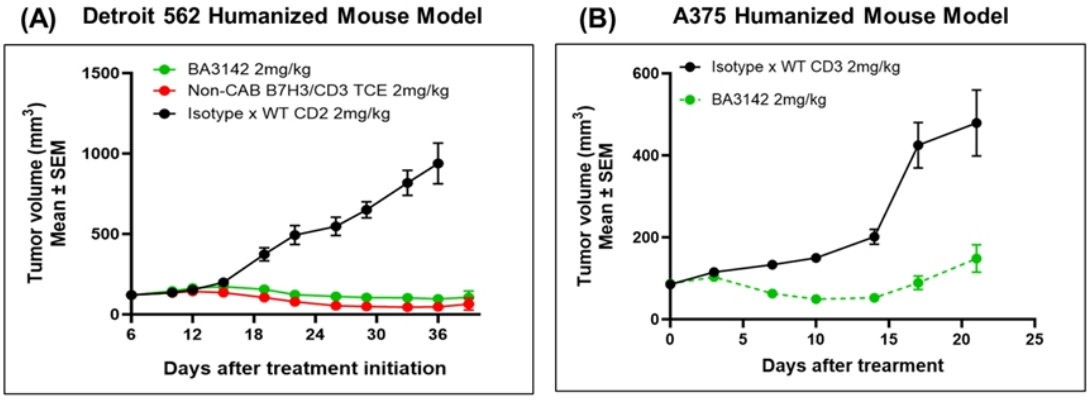
- increases extracellular acidification rate *in vitro*
- increases tumor growth *in vivo*
- Increases glucose uptake *in vivo*

Target biology unusually suited to create environment for CAB Technology

Lim et al, Cancer Res, 76:2231, 2016

BA3142 *in vivo* Efficacy

BA3142 dosed at 2mg/kg led to significant tumor regression in Detroit 562 and A375 humanized mouse model of human pharyngeal and melanoma cancers, respectively. The *in vivo* anti-tumor activity of BA3142 was comparable to the Non-CAB B7H3/CD3 TCE.



***In vivo* efficacy studies.** Triple immunodeficient mice were engrafted with human PBMCs and inoculated with Detroit 562 **(A)** or A375 **(B)** cells. Treatment was initiated when the tumor volume reached approximately 80-120 mm^3, and mice were dosed with TCEs at 2mg/kg BIW x 4 weeks.

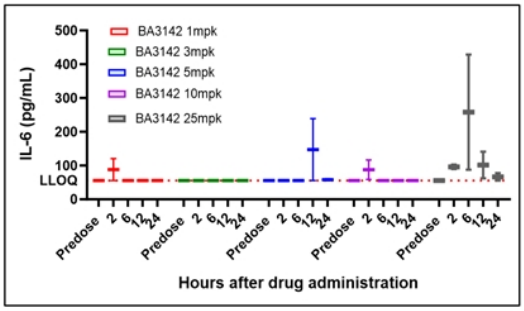
Competition Limited for Differentiated TCE as a Result of Clinical Failures

- **Terminated programs-**
  - Macrogenics terminated MGD009 **(B7H3 x CD3)** in Phase 1 a partial clinical hold due to hepatic safety concerns. Program discontinued in 2019.
  - Daiichi-Sankyo **discontinued** the development of its **fucosylated mAb** during Phase 1 ("business decision").
- **Remaining competition is limited to date-**
  - Macrogenics **abandoned TCE's** and is focused on its 2 remaining ADC programs:
    - Enoblituzumab (**Fc-mediated** killing): Efficacy signal observed in SCCHN & NSCLC combo w/ pembrolizumab in anti-PDL1 naïve and grade ≥3 TEAEs to watch for.
    - MGC018 (**ADC** duocarmycin-based linker payload): Phase 1 alone or in combo w/ PD1 underway.
    - Other **ADC**s emerging
  - Xencor (B7H3 x CD28) Phase 1 **initiated**, but yet to be validated
- **BioAtla differentiated CAB TCE program-**
  - BA3142 DualCAB B7H3 x CD3:
    - Higher potency and efficacy than MGD009 B7H3 x CD3 bispecific TCE (*i.e.* >10-fold lower EC50 in PBMC cell killing activity)
    - Better safety with no MTD reached even 25mg/kg (very high level for a TCE)
    - Ph 1 initiation in 2025

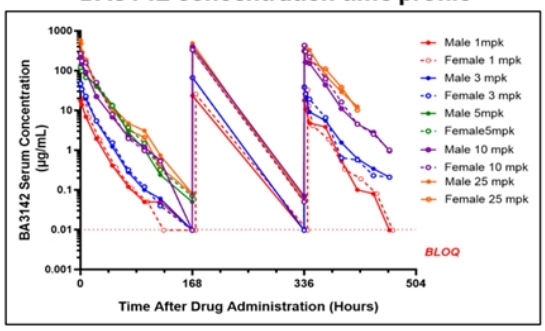
BA3142 – NHP Tox and PK study

- BA3142 was well-tolerated in non-human primates at 25 mg/kg; MTD not reached
- Sporadic elevations of IL-6 cytokine was observed without a clear relationship with BA3142 dosing.
- Systemic exposure was dose-proportional with no significant gender differences in TK parameters.

IL-6 levels in NHP serum



BA3142 concentration time profile



BA3142 Systemic Exposure

TK Parameters are shown as Mean ± SD				
BA3142 Dose	C_{max} (µg/mL)	$AUC_{0\text{-}168}$ (h*µg/mL)	Cl (mL/h/kg)	$T_{1/2}$ (h)
1mg/kg	19 ± 2	192 ± 11	5.2 ± 0.3	15 ± 2
3mg/kg	48 ± 2	520 ± 24	5.8 ± 0.3	14 ± 3
5mg/kg	129 ± 14	1280 ± 21	3.9 ± 0.1	16 ± 1
10mg/kg	243 ± 36	2970 ± 926	3.5 ± 1.1	13 ± 1
25mg/kg	795 ± 115	6780 ± 891	3.7 ± 0.5	25 ± 2

Toxicity study in NHP. Cynomolgus monkeys received a single or three intravenous administration of BA3142 at different doses. Serum was collected at different time points for cytokine and toxicokinetic analysis. **(A)** IL-6 cytokine levels in NHP serum, **(B)** BA3142 concentration time profile in NHP serum and **(C)** TK parameters after a single dose of BA3142.

BA3142 – CAB B7H3 TCE Summary

- B7H3 tumor expression correlated with exceptionally high glycolytic activity and acidity, making this target well suited for CAB technology
- Potent B7H3 x CD3 bispecific TCE with good safety profile and wide therapeutic margin
  - Exceptional HNSTD 25 mg/kg in NHPs; MTD not reached
- Multiple potential solid tumor indications in large markets
- Dual CAB format with simple 1H & 1L gene expression in mammalian cells
  - cGMP drug product available
  - Path to clinic in 2Q2025

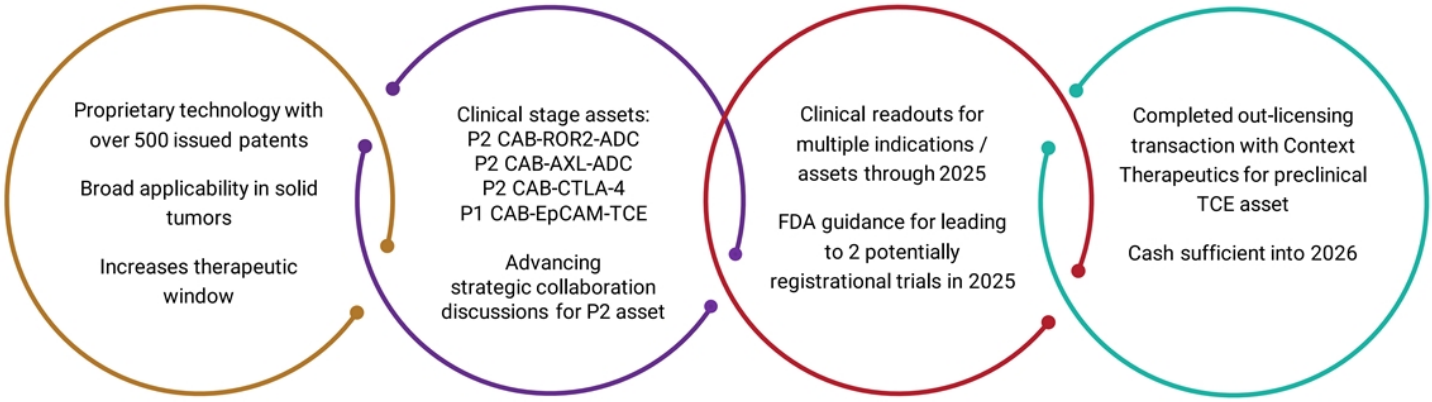
BA3142 – CAB B7H3 TCE

Path to IND - Potential IND submission 2Q 2025

- cGMP manufacturing completed
- Material for clinical trials available
- Tox lot *in vitro* characterization (in progress)
  - pH Affinity ELISA
  - pH Range ELISA
  - Cross Species ELISA
  - Specificity ELISA
  - pH SPR analysis
  - pH FACS
  - Promega T cell activation Assay
  - PBMC cytotoxicity assay
  - PBMC cytokine release assay
- GLP tox study including BioA development (cyno/human PK, ADA assays)
- Clinical protocol development
- IND preparation and publishing

BioAtla© Is A Clinical Stage Company Focused on Transforming Cancer Therapy

with **C**onditionally **A**ctive **B**iologics (CABs)



ADC – antibody drug conjugate; TCE – T-cell engager

Appendix