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): March 31, 2026

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	The Nasdaq Capital 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 2.02 Results of Operations and Financial Condition.

On March 31, 2026, BioAtla, Inc. (the "Company") issued a press release announcing its financial results for the fourth quarter and fiscal year ended December 31, 2025. A copy of the press release is attached hereto as Exhibit 99.1 and is incorporated herein by reference. The information set forth in Item 2.02 of this Current Report on Form 8-K ("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 2.02 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 (the "Securities Act"), or the Exchange Act, regardless of any incorporation by reference language in any such filing.

Item 7.01 Regulation FD Disclosure.

On March 31, 2026, the Company updated its corporate presentation (the "Corporate Presentation"), which it intends to use at various meetings with investors, investment banks, industry analysts, potential strategic partners and other interested parties. The Corporate Presentation is attached hereto as Exhibit 99.2 and is incorporated herein by reference.

The information set forth in Item 7.01 of this Current Report, including Exhibit 99.2 attached hereto, is being furnished and shall not be deemed "filed" for purposes of Section 18 of 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 99.2 attached hereto, shall not be incorporated by reference into any filing under the Securities Act 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	Description
99.1	Press Release dated March 31, 2026
99.2	BioAtla, Inc. Corporate Presentation, dated March 31, 2026
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: March 31, 2026

By: /s/ Christian Vasquez
Christian Vasquez
Chief Financial Officer

Exhibit 99.1

BIOATLA REPORTS FOURTH QUARTER AND FULL YEAR 2025 FINANCIAL RESULTS AND BUSINESS HIGHLIGHTS

- *Initiated a formal process to monetize assets*
- *Implemented a restructuring plan to significantly reduce operating expenses and extend runway*

SAN DIEGO, March 31, 2026 – BioAtla, Inc. (Nasdaq: BCAB), a global clinical-stage biotechnology company focused on the development of Conditionally Active Biologic (CAB) antibody therapeutics for the treatment of solid tumors, today announced its financial results for the full year and fourth quarter ended December 31, 2025 and provided business highlights.

"As we evaluate strategic paths forward, our focus remains on supporting the advancement of the ongoing Phase 1 study of BA3182 in adenocarcinomas, while preserving capital, and ensuring we maintain the flexibility to pursue all potential value-creating options," said Jay M. Short, Ph.D., Chairman, Chief Executive Officer and co-founder of BioAtla.

Corporate Updates
On March 2, 2026, the Company announced that its Board of Directors initiated a formal process to explore and evaluate strategic options to maximize shareholder value, including the sale of preclinical and clinical assets, licensing transactions, strategic partnerships or other corporate transactions. The Company engaged Tungsten Advisors as the Company's exclusive strategic financial advisor to assist with this process. There can be no assurance that this process will result in any agreements or transactions. BioAtla does not intend to provide updates until the Board of Directors approves a specific action or otherwise determines whether disclosure is appropriate or required.

In connection with the evaluation of strategic options, the Company also implemented a reduction in force and other cost-containment measures intended to better align resources with its near-term priorities. In order to continue to preserve capital during this period, the Company is re-evaluating the timing and scope of its clinical development programs, including the appropriate timeline and pacing of additional enrollment in the Phase 1 study of BA3182, as well as the timeline to commence a Phase 3 Study for ozuriftamab vedotin (Oz-V) (CAB-ROR2-ADC) in 2L+ oropharyngeal squamous cell carcinoma (OPSCC). While the Company remains focused on conducting the ongoing Phase 1 study and is committed to its clinical development programs, there can be no assurances that clinical development of its programs will not be limited or delayed pending the outcome of the strategic process.

Fourth Quarter and Full Year 2025 Financial Results
Research and development (R&D) expenses were $8.0 million for the fourth quarter of 2025, compared to $11.7 million for the same period in 2024. For the full year ended December 31, 2025, R&D expenses were $43.6 million, compared to $63.1 million for 2024. The decreases for both the quarter and full year were primarily driven by lower program development costs due to the prioritization of clinical programs, reduced headcount-related expenses following the March 2025 workforce reduction, and lower non-cash stock-based compensation. The Company continues to expect R&D expenses to decline in 2026 following its March 2026 workforce reduction and as it concentrates resources on its prioritized programs.

Collaboration and other revenue was $2.0 million for the fourth quarter and full year 2025, reflecting the Context Therapeutics milestone payment under the license agreement for the CAB-Nectin4-TCE program, compared to zero collaboration revenue in the fourth quarter of 2024 and $11.0 million for the full year 2024 related to the initial upfront payment from Context Therapeutics license agreement.

General and administrative (G&A) expenses were $3.3 million for the fourth quarter of 2025, compared to $4.6 million in the fourth quarter of 2024. For the full year ended December 31, 2025, G&A expenses were $17.7 million, compared to $21.8 million for 2024. The decreases were primarily attributable to reduced personnel-related expenses following the March 2025 workforce reduction and lower non-cash stock-based compensation expense. The Company expects its G&A personnel related expenses to decline in 2026 following its March 2026 workforce reduction.

Net loss for the fourth quarter of 2025 was $9.8 million, compared to a net loss of $14.9 million for the same period in 2024. For the full year 2025, net loss was $59.6 million, compared to $69.8 million for the full year 2024. The year-over-year improvements were primarily driven by lower operating expenses and a non-cash gain on its warrant liability, partially offset by lower collaboration revenue in 2025, a non-cash loss associated with the liability related to Pre-paid Advance Agreements (“PPAs”), and other expenses primarily related to our PPAs financing in 2025. As of March 2026, the PPAs have been converted in full into shares of the Company’s common stock, and no amounts remain outstanding under the PPAs. Cash and cash equivalents were $7.1 million as of December 31, 2025. The Company is currently running the formal process, and is re-evaluating the structure and timing of completion of the previously announced SPV transaction pending the outcome of this process. Through meaningful cost reductions and utilization of the Standby Equity Purchase Agreement, the Company expects to extend its runway to advance through the formal process being led by Tungsten Advisors toward maximizing shareholder value.

About BioAtla®, Inc.
BioAtla is a global clinical-stage biotechnology company with operations in San Diego, California. Utilizing its proprietary CAB platform technology, BioAtla develops novel, reversibly active monoclonal and bispecific antibodies and other protein therapeutic product candidates. CAB product candidates are designed to have more selective targeting, greater efficacy with lower toxicity, and more cost-efficient and predictable manufacturing than traditional antibodies. BioAtla has a robust pipeline consisting of ADCs and T cell engagers (TCEs) that utilize its conditionally active platform technology utilizing pH sensitivity to minimize on-target, off-tumor toxicity. BioAtla has extensive and worldwide patent coverage for its CAB platform technology and products with greater than 780 active patent matters, more than 500 of which are issued patents. Broad patent coverage in all major markets include methods of making, screening and manufacturing CAB product candidates in a wide range of formats and composition of matter coverage for specific products. To learn more about BioAtla, Inc., visit www.bioatla.com.

Clinical stage pipeline:

- Ozuriftamab vedotin (CAB-ROR2-ADC) - Phase 3 in OPSCC
- Mecbotamab vedotin (CAB-AXL-ADC) - Phase 2 in Sarcoma (soft tissue and bone) and mKRAS NSCLC
- Evalstotug (CAB-CTLA-4) - Phase 2 in Unresectable and/or Metastatic Cutaneous Melanoma
- BA3182 (dual CAB-EpCAM x CAB-CD3 T cell engager) - Phase 1 in adenocarcinoma - BioAtla will continue to conduct the Phase 1 clinical study.

Pre-clinical stage pipeline:

- BA3361 (CAB-Nectin4-ADC) - data in breast cancer (BT474, T47D), lung cancer (NCI-H322), bladder cancer (HT1376) and pancreatic cancer models; IND-approved.
- BA3151 (CAB-B7H4-ADC) - data in breast cancer (MX-1) models.
- BA3142 (dual CAB-B7H3 x CAB-CD3 TCE) - IND ready; data in melanoma (A375) and pharyngeal cancer (Detroit 562) models.
- BA3311 (EGFR x CAB-CD3 TCE) - data in lung cancer (A549, HCC827), breast cancer (BT474), and colon cancer (HCT116) models.
- BA3241 (dual CAB-Trop2 x CAB-CD3 TCE) - data in epidermoid cancer (A431)

Partnered Program:

- BA3362 (dual CAB-Nectin4 x CAB-CD3 TCE) - out-licensed to Context Therapeutics for up to $133.5 Million plus royalties.

About BA3182 (CAB-EpCAM x CAB-CD3 Bispecific T-cell Engager Antibody)
BioAtla is developing BA3182 as a potential anticancer therapy for patients with advanced adenocarcinoma. BA3182 is a (CAB) EpCAM x (CAB) CD3 bispecific T cell engager antibody with binding sites for EpCAM and CD3ε designed to bind their respective targets specifically and reversibly under the conditions found in the tumor microenvironment (TME) and to have reduced binding outside of the TME. The CAB selective binding to both the CAB EpCAM and CAB CD3ε arms are required to activate the T cell engagement against the tumor, thus enabling the combined selectivity of each CAB binding arm in the bispecific antibody. BioAtla continues to advance the ongoing Phase 1 study to evaluate the safety, pharmacokinetics, and efficacy of BA3182 in advanced adenocarcinoma patients.

About Ozuriftamab Vedotin (Oz-V)
Oz-V, CAB-ROR2-ADC, is a conditionally and reversibly active antibody drug conjugate directed against ROR2, a transmembrane receptor tyrosine kinase that is present across many different solid tumors including head and neck, lung, cervical, triple-negative breast cancer, and melanoma. Overexpression of ROR2, a non-canonical wnt5A signaling receptor, forms a cancer axis that is associated with poor prognosis and resistance to chemo- and immunotherapies. This Phase 3 stage clinical asset is targeting the treatment of OPSCC patients who have previously progressed on PD-1/L1 therapies with or without platinum chemotherapy. HPV associated expression of E6 and/or E7 oncoproteins drives cancer progression by upregulating ROR2 expression. As such, there is potential to expand the application of Oz-V more broadly beyond OPSCC to all HPV+ cancers, which represents a market opportunity of over $7 billion worldwide. The FDA granted Fast Track Designation to Oz-V for the treatment of patients with recurrent or metastatic squamous cell carcinoma of the head and neck (SCCHN).

About OPSCC
OPSCC is a subset of squamous cell carcinoma of the head and neck (SCCHN) arising from the squamous cells that line the oropharynx, the middle part of the throat. This anatomic region is located behind the oral cavity and OPSCC typically involves tonsils, soft palate, pharyngeal walls, and/or the base of the tongue. A striking year-to-year increase in OPSCC is due to the rapidly increasing incidence of HPV infections which currently represents approximately 80% of OPSCC in the United States. The prognosis is

currently poor for patients with recurrent/metastatic OPSCC who have previously received standard treatments including surgery, radiation, platinum-based chemotherapy, and PD-1 inhibitor therapy.

About Mecbotamab Vedotin (Mec-V)
Mecbotamab vedotin (Mec-V), CAB AXL-ADC, is a conditionally and reversibly active antibody drug conjugate targeting the receptor tyrosine kinase AXL. This Phase 2 stage clinical asset is targeting multiple solid tumor indications, including the treatment of mKRAS NSCLC and soft tissue sarcoma. The Office of Orphan Products Development (OOPD) at FDA granted Orphan Drug Designation to Mec-V for the treatment of soft tissue sarcoma.

About Evalstotug
Evalstotug, is a CAB anti-CTLA-4 antibody that is anticipated to enable safer anti-CTLA-4 antibody combination therapies, such as with anti-PD-1 antibody checkpoint inhibitors. Like our other CAB candidates, this Phase 2 clinical asset is designed to be conditionally and reversibly active in the TME. Evalstotug is being developed as a potential therapeutic for multiple solid tumor indications that are known to be responsive to CTLA-4 treatment in combination with a PD-1 blocking agent.

Forward-looking Statements
Statements in this press release contain "forward-looking statements" that are subject to substantial risks and uncertainties. Forward-looking statements contained in this press release may be identified by the use of words such as "anticipate," "expect," "believe," "will," "may," "should," "estimate," "project," "outlook," "forecast" or other similar words. Examples of forward-looking statements include, among others, statements we make regarding BioAtla's business plans and prospects; statements concerning the intended benefits of the formal process; expected benefits and outcomes of our strategic partnerships and transactions; statements regarding the expected benefits related to the reduction in force and cost containment measures, ability to extend our cash runway, the expected timing and pacing of enrollment in our clinical trials and the potential regulatory approval path for our product candidates. Forward-looking statements are based on BioAtla's current expectations and are subject to inherent uncertainties, risks and assumptions, many of which are beyond our control, difficult to predict and could cause actual results to differ materially from what we expect. Further, certain forward-looking statements are based on assumptions as to future events that may not prove to be accurate. Factors that could cause actual results to differ include, among others: factors that raise substantial doubt about our ability to continue as a going concern and that we will need additional funding to continue development of our CAB technology platform and our CAB product candidates; the risk that preliminary or interim clinical results may not be indicative of results from later cohorts or larger populations; potential delays in clinical and preclinical trials; the uncertainties inherent in research and development, including the ability to meet anticipated clinical endpoints, commencement and/or completion dates for clinical trials, regulatory submission dates, or regulatory approval dates, as well as the possibility of unfavorable new clinical data and further analyses of existing clinical data; whether regulatory authorities will be satisfied with the design of and results from the clinical studies or take favorable regulatory actions based on results from the clinical studies; our dependence on the success of our CAB technology platform; our ability to enroll patients in our ongoing and future clinical trials; the successful selection and prioritization of assets to focus development on selected product candidates and indications; our ability to form collaborations and partnerships with third parties and the success of such collaborations and partnerships; our reliance on third parties for the manufacture and supply of our product candidates for clinical trials; our reliance on third parties to conduct our clinical trials and some aspects of our research and preclinical testing;

potential adverse impacts due to geopolitical or macroeconomic events outside of our control, including health epidemics or pandemics; and those other risks and uncertainties described in the section titled "Risk Factors" in our Annual Report on Form 10-K filed with the Securities and Exchange Commission (the “SEC”) on March 31, 2026 and our other reports filed with the SEC. Forward-looking statements contained in this press release are made as of this date, and BioAtla undertakes no duty to update such information except as required under applicable laws.

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External Contact:
Joyce Allaire
LifeSci Advisors, LLC
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BioAtla, Inc.
Unaudited Consolidated Statements of Operations and Comprehensive Loss
(in thousands, except share and per share amounts)

	Three Months Ended December 31,		Twelve Months Ended December 31,	
	2025	2024	2025	2024
Collaboration and other revenue	$ 2,000	$ —	$ 2,000	$ 11,000
Operating expenses:				
Research and development expense	$ 7,995	$ 11,650	$ 43,573	$ 63,095
General and administrative expense	3,260	4,594	17,732	21,848
Total operating expenses	11,255	16,244	61,305	84,943
Loss from operations	(9,255)	(16,244)	(59,305)	(73,943)
Other income (expense):				
Interest income	73	554	843	3,369
Gain on warrant liability	831	807	295	807
Loss on PPAs liability	(406)	—	(406)	—
Other expense	(1,027)	(1)	(1,034)	(9)
Total other income (expense)	(529)	1,360	(302)	4,167
Consolidated net loss and comprehensive loss	$ (9,784)	$ (14,884)	$ (59,607)	$ (69,776)
Net loss per common share, basic and diluted	$ (0.16)	$ (0.30)	$ (1.01)	$ (1.44)
Weighted-average shares of common stock outstanding, basic and diluted	59,796,673	49,646,078	58,827,934	48,573,364

BioAtla, Inc.
Consolidated Balance Sheet Data
(in thousands)

	December 31, 2025	December 31, 2024
Cash and cash equivalents	$ 7,118	$ 49,046
Total assets	13,828	52,422
Total current liabilities	21,922	14,540
Total liabilities	50,017	38,157
Total stockholders' equity (deficit)	(36,189)	14,265
Total liabilities and stockholders' equity (deficit)	13,828	52,422

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

Corporate Presentation

March 2026

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 we make regarding BioAtla's business plans and prospects; whether our clinical trials will support registration; achievement of milestones; results, progress and timing of our research and development programs and clinical trials; expectations with respect to enrollment and dosing in our clinical trials; the anticipated clinical benefits, safety, efficacy, and market potential of our product candidates; plans and expectations regarding future data updates, clinical trials, regulatory meetings and regulatory submissions; the timing of and the ability to establish collaborations or other strategic partnerships for selected assets; the potential regulatory approval path for our product candidates; and expectations about the sufficiency of our cash and cash equivalents to fund operations and expectations regarding R&D expenses and cash burn. 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.

Leadership Team

Jay Short, Ph.D.

Chairman, CEO and Cofounder

Chris Vasquez

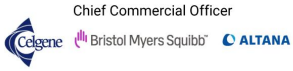
Chief Financial Officer

Eric Sievers, M.D.

Chief Medical Officer

Sheri Lydick

Chief Commercial Officer



Ji Hwan Lee

VP Head of Portfolio Management

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

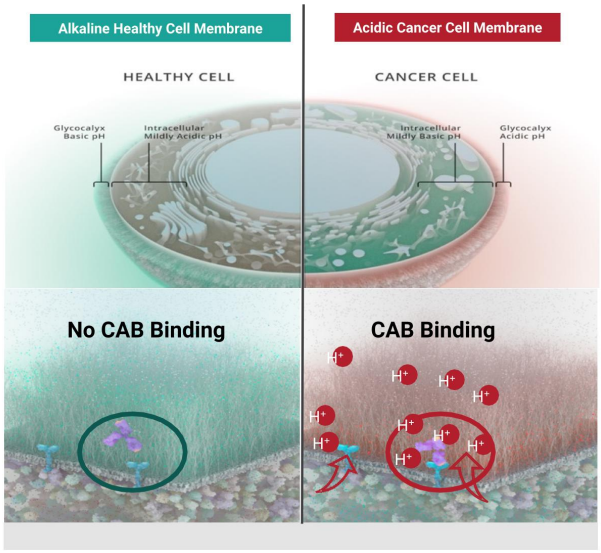
Padmanee Sharma, MD, Ph.D.
MD Anderson Cancer Center
Scientific Advisor

Lawrence Fong, MD
Cancer Immunotherapy Program, UCSF
Scientific Advisor

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 Platform 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 to promote metastasis and defend against immune response
- CAB mechanism
  - Leverages naturally occurring, negatively charged molecules (*e.g.* bicarbonate, hydrogen sulfide) to differentiate between targets on cancer cells versus normal cells
  - These physiological molecules underpin the CAB mechanism and are referred to as Protein-associated Chemical Switches (PaCS)™
  - In normal tissues, PaCS shield epitopes, so CAB antibodies cannot bind. In contrast, cancer cells produce H+ ions that remove PaCS molecules from the epitopes, enabling cancer-specific binding.

Myths vs Facts of pH Therapies in Cancer

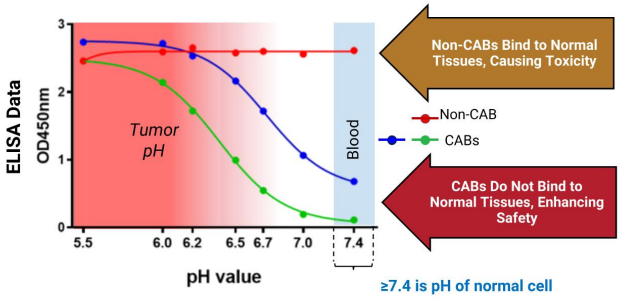
BioAtla's Solution: Conditionally Active Biologics (CAB)

Myth	Fact
Not all tumors are acidic	ALL tumors and cancer cells are acidic (*i.e.* pH5.3-6.7).
Cancer cells are ATP limited	Cancer cells are NOT generally ATP deficient but are limited in other precursor molecules whose synthesis depends upon glycolysis.
pH technology will miss tumor cells because tumor pH is variable	While tumor degree of pH acidity can vary, CABs are designed to bind any cancer cell at or below a predetermined pH.
Tumor size influences the acidic environment so pH technology will not work	• Larger tumors with larger anaerobic regions are not necessarily more acidic since oxygenated regions have higher acidity due to the higher concentration of hydrogen ions from rapid glycolysis. • Cancer cells – as opposed to the average pH of a tumor – are more acidic, especially at the membrane of the cancer cell.

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



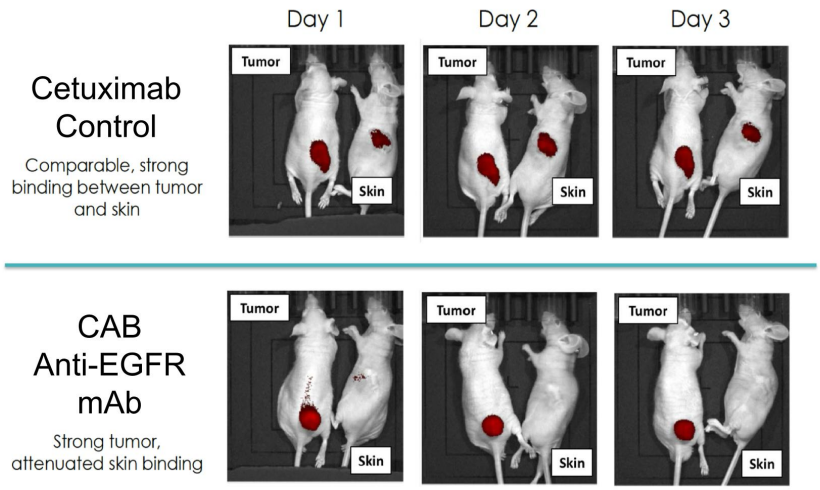
Selective Binding → Focused Tumor Killing → Normal Cells Preserved

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 Technology Eliminates On-target, Off-tumor Binding

Thus, widening the therapeutic index by 12.6-fold over cetuximab

Enhanced Tumor Selectivity
(12.6-fold increase in TI)



PET scan images

Conditional Binding Approaches: Prodrug vs BioAtla's CAB Platform Technology

Feature	Proteolytic cleavage of prodrug	Conditionally Active Biologic (CAB)
Mechanism of Action	**Mask** obscuring antibody binding site is cleaved by tumor-associated proteases	Biologic is engineered to bind **only in TME at low pH***
Trigger Type	**Enzymes** overexpressed in tumors	**TME** (*i.e.*, low pH)
Bioengineering	**Addition of foreign sequence**	**No foreign sequences**
Activation Precision	**Requires activation**; dependent on expression and enzymatic efficiency	**No activation required** – maximizes potency
Risk of Off-Target Effects	Irreversible activation - **can bind** target in **normal tissues**	Reversible binding - **will not bind** target in **normal tissues**

TME = Tumor microenvironment
*Warburg effect

Preclinical Evidence Summary

- Preclinical evidence of CAB selectivity
  - Differential EGFR tumor vs. skin binding (12.6-fold improved TI)
  - AXL-ADC reduced TMDD yielding
    - Increased $T_{1/2}$ and exposure in NHP (>2-fold increase in $T_{1/2}$)
    - Reduced liver enzymes (<10% of non-CAB ALT levels)
  - EpCAM DualCAB TCE maintained efficacy with highly reduced toxicity
    - MTD not reached in NHP
    - >100-fold improvement in TI
  - B7H3 Dual CAB TCE; target expression associated with high acidity via hyper-glycolysis
    - MTD not reached in NHP
    - Encouraging safety profile compared to other B7H3 TCEs in development
  - CTLA4 reduced 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

CAB, Conditionally Active Biologics; TI, Therapeutic Index; $T_{1/2}$, Half-life; TMDD, Target—Mediated Drug Disposition; NHP, Non-Human Primate; ALT, Alanine Aminotransferase; TCE, T-Cell Engager; MTD, Maximally Tolerated Dose; ipi, ipilimumab

Clinical Evidence Summary

- Clinical evidence of CAB selectivity
  - AXL-ADC good risk/benefit ratio
    - Two non-CAB AXL-targeting ADCs terminated in P1 due to toxicity
    - Potent and durable response with differentiated OS in mKRAS NSCLC and sarcoma patients
  - ROR2-ADC good risk/benefit ratio
    - Good tolerability with only 7% treatment-related discontinuation rate
    - Potent and durable response in SCCHN patients
  - 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
  - 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
    - Reduced grade 3 AEs such as colitis even at higher doses

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

Key Advantages of the CAB Platform

Widening The Therapeutic Index

- ✓ **Conditional and reversible binding increases clinical activity and improves safety**
- ✓ **Not dependent on enzymatic activation for selective binding**
- ✓ **Enhances pharmacologic properties**
- ✓ **Broadly applicable to antibody formats including ADCs, Bispecific TCEs, CAR-Ts and other proteins**
- ✓ **Provides ability to create new therapies and combinations against targets that had previously been limited due to toxicity**

Clinical and Preclinical Pipeline of Differentiated CAB Assets Across Multiple Solid Tumors

	CAB Program	Target	Indications	IND Enabling Pre-Clinical	Phase 1 Clinical	Phase 2 Clinical	Phase 3 Clinical
CAB-Bispecific TCE	BA3182	EpCAM x CD3	Adenocarcinomas				
	CT-202 (Out licensed to Context Therapeutics)	Nectin-4 x CD3	Solid Tumors				
	BA3142	B7H3 x CD3	Solid Tumors				
	BA3241	Trop2 x CD3	Solid Tumors				
	BA3311	EGFR x CD3	Solid Tumors				
CAB-ADCs	BA3021 *Ozuriftamab Vedotin*	ROR2	2L+ OPSCC				
	BA3011 *Mecbotamab Vedotin*	AXL	NSCLC Sarcoma				
	BA3361	Nectin-4	Solid Tumors				
CAB-I/O	BA3071 *Evalstotug*	CTLA-4	1L and 2L Melanoma				

IND, investigational new drug; OPSCC, oropharyngeal squamous cell carcinoma; NSCLC, Non-small Cell Lung Cancer; TCE, T cell engager

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

Why EpCAM (Epithelial Cell Adhesion Molecule) As a Target?

Targeting EpCAM has potential to serve over 1 Million patients (potential Pan-cancer drug)

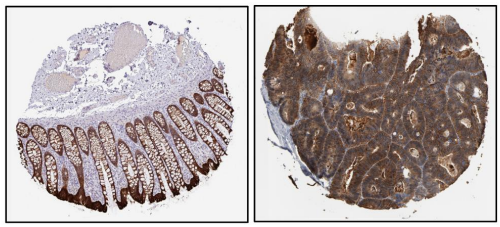
	Estimated Number of New Cancer Cases in 2025	EpCAM Expression (TIS 1 to 12)[2]
Breast Cancer	319,750	81%
Prostate Cancer	313,780	99%
Lung Cancer	226,650	93% NSCLC 80% SCLC
Colon Cancer	154,270	100%
Pancreatic Cancer	67,440	99%
Thyroid Cancer	44,020	97%
Ovarian	20,890	92%
Gallbladder & other biliary	12,610	97%

[1]Siegel RL, Kratzer TB, Giaquinto AN, Sung H, Jemal A. Cancer statistics, 2025. *CA Cancer J Clin*. 2025.
[2]G. Spizzo, et al. J Clin Pathol 2011;64:415e420.

Challenges of targeting EpCAM

All normal epithelia express EpCAM which with traditional antibodies would lead to on-target, off-tumor toxicities

CABs are essential for targeting EpCAM



Normal Colon **Colon Cancer**

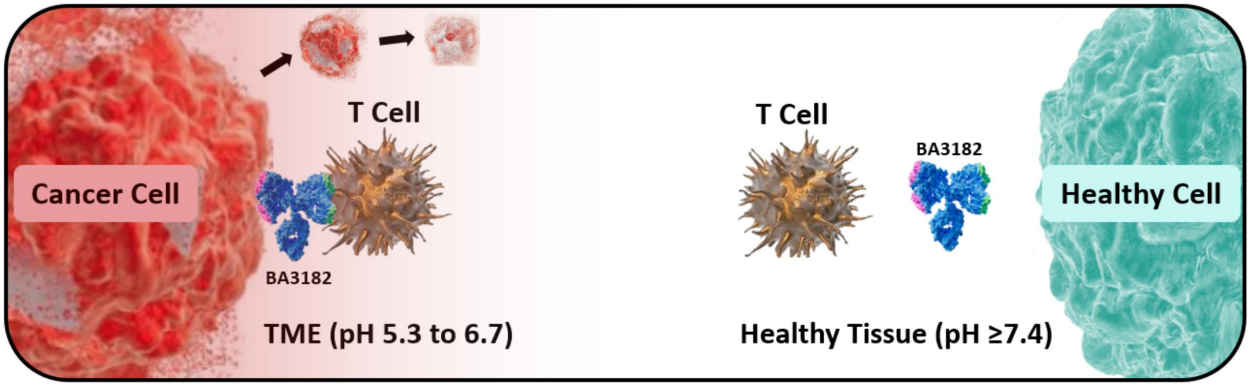
EpCAM IHC

Proposed Mechanism of Action of BA3182, a Dual-CAB EpCAM x CD3 bispecific T-cell Engager

CAB-TCEs redirect all T cells in the TME to attack cancer cells, but not in healthy tissues

Dual-conditional binding technology drives selective ablation of cancer cells in the acidic TME

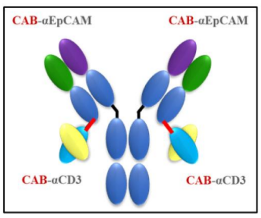
No binding in healthy tissue, reducing CRS and on-target, off-tumor toxicities



Mabs. 2024 Mar 6;16(1):2322562

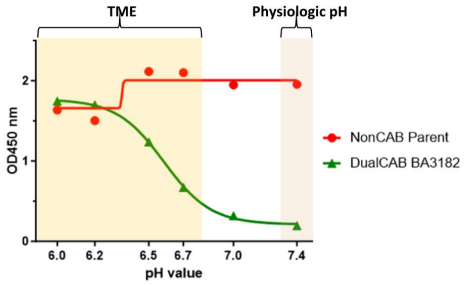
BA3182 – First Dual-CAB T-cell Engager Targeting EpCAM

Potent Lysis of EpCAM Positive Cancer Cells by BA3182



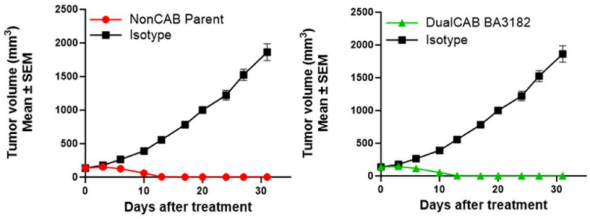
Tetravalent T-cell engager EpCAM*

pH Range ELISA



Highly reduced binding to both targets at normal physiological pH

HCT116 CDX Model



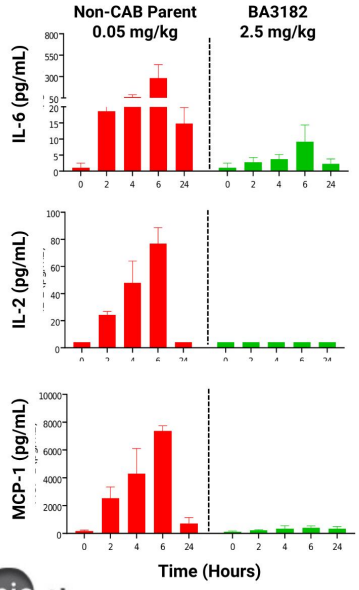
Similar efficacy in mouse xenograft models compared to Non-CAB parent molecule

*(2+1) functional format (only one anti-CD3scFv can engage a T cell based on steric hindrance)

Mabs. 2024 Mar 6;16(1):2322562

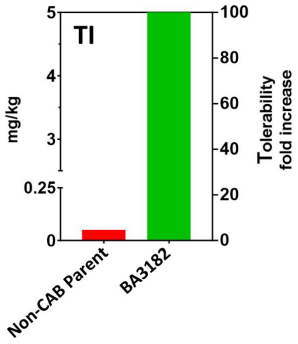
CAB-EpCAM-TCE, BA3182, is Well Tolerated at High Doses in Non-human Primates

Cytokine Levels Associated with Toxicity



Low cytokine levels with DualCAB vs Non-CAB Parent even at significantly higher doses

Increase in Safety and Tolerability

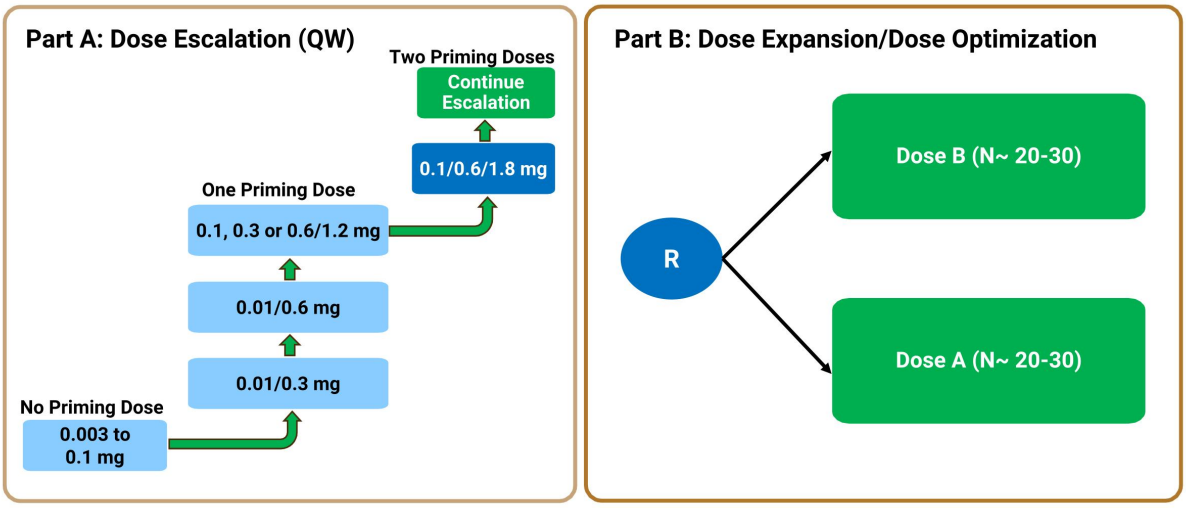


Test Article	Non-CAB Parent	BA3182
Dose	0.05 mg/kg	5 mg/kg
Clinical Outcome	All Euthanized on Day 8	No Clinical Findings

Mabs. 2024 Mar 6;16(1):2322562

Phase 1 Dose Escalation Study of BA3182 in Advanced Adenocarcinomas

Trial ongoing; evaluating various dosing and treatment schedules



Treatment notes:

- Prophylactic acetaminophen and diphenhydramine delivered prior to all doses and prophylactic tocilizumab (no corticosteroids) given prior to Cycle 1 Day 1 treatment dose
- Post treatment ondansetron guided for nausea
- Ongoing weekly treatment dosing continued after DLT observation interval concluded

Presented at ESMO Berlin, 2025.

BA3182 Phase 1 Patient Demographics

Patients dosed SC per protocol as of September 10, 2025: N=35

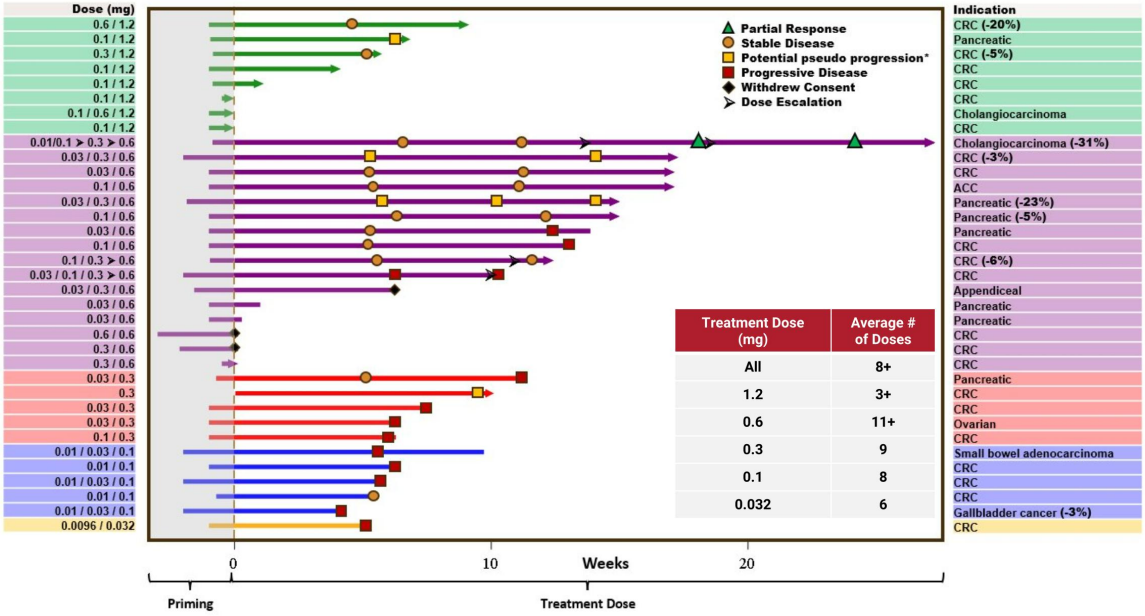
Patient Characteristic	N=35
Age, mean (SD), y	57 (10)
Male (n, %)	19 (54)
Female (n, %)	16 (46)
ECOG performance status	
0 (n, %)	24 (69)
1 (n, %)	11 (31)
Presence of liver metastases (n, %)	22 (63)

Carcinoma Type (n, %)	N=35	Median # of prior treatments
Adenoid Cystic Carcinoma	1 (3)	2
Appendiceal	1 (3)	2
Cholangiocarcinoma	2 (6)	1
Colorectal	22 (63)	4
Gallbladder	1 (3)	3
Ovarian	1 (3)	8
Pancreas	7 (20)	3

Presented at ESMO Berlin, 2025.

Data cut as of 10Sep25

BA3182 Preliminary Prolonged Tumor Control With Increasing Doses



*Pts with unconfirmed PD were treated beyond initial progression per protocol when pseudo-progression was suspected

Presented at ESMO Berlin, 2025.

Live Data cut as of 10Sep2025

BA3182 Achieved Objective Tumor Size Reductions Across Multiple Tumor Types

Preliminary assessment of anti-tumor activity among pts receiving treatment doses of ≥0.6 mg (N=24)

- 14 CRC pts: among 7 pts who had one scan – 5 achieved SD at 1st scan; among the 7 pts without scans –2 withdrew consent before the 1st scan and 5 are pending 1st scan
- 6 pancreatic cancer pts: 2 pts had SD at 1st scan and 2 pts continued treatment beyond potential pseudo-progression
- 2 cholangiocarcinoma pts: 1 pt achieved a confirmed PR and 1 pt is pending first scan
- 1 ACC pt experienced SD
- Among pts treated with ≥0.6 mg , 9/10 pts achieved SD at a higher rate and remained on active treatment for prolonged intervals, generally longer than those who received lower doses

CRC, colorectal cancer; SD, stable disease; PR, partial response; ACC, adenoid cystic carcinoma

Presented at ESMO Berlin, 2025.

Live Data cut as of 10Sep2025

BA3182 Adverse Events Generally Transient and Readily Manageable

Preliminary safety of subcutaneous dosing

Characteristic	N=35 (n, %)
Any Adverse Events (AEs)	29 (83)
Related AEs of Grade 3-4	14 (40)
Related AEs of Grade 3 hepatic analytes[1]	11 (31)
Related AEs of Grade 4 hepatic analytes[1]	1 (3)
Related AEs of Grade 3-4 non-febrile neutropenia[2]	2 (6)
Related AEs of Grade 3 excluding hepatic analytes[1] and non-febrile neutropenia[2,4]	3 (9)
Any related serious AEs[3]	5 (14)
Related CRS of any grade (per ASTCT grading)	2 (6)
Related AEs leading to death	0
Related AEs leading to treatment discontinuation	1 (3)

[1]Early, transient elevation of hepatic analytes: AST, ALT, bilirubin, and/or alkaline phosphatase
[2]Non-febrile, transient neutropenia, possibly related to tocilizumab
[3]G2 pancreatitis, G2 atrial fibrillation, G3 diarrhea, G3 acute kidney injury, and G2 CRS
[4]G3 diarrhea; G3 diarrhea/lymphocyte count decrease; G3 acute kidney injury/white blood cell count decrease

TRAE >10% (N=35)	TRAE Any (n, %)	TRAE G3+ (n, %)
Alanine aminotransferase increased*	15 (42.9)	7 (20.0)
Aspartate aminotransferase increased*	15 (42.9)	9 (25.7)
Nausea	14 (40.0)	0
Injection site reaction	12 (34.3)	0
Diarrhea	11 (31.4)	2 (5.7)
Fatigue	9 (25.7)	0
Blood bilirubin increased*	8 (22.9)	0
Neutrophil count decreased	7 (20.0)	2 (5.7)
Blood alkaline phosphatase increased*	6 (17.1)	1 (2.9)
Decreased appetite	6 (17.1)	0
Dysgeusia	5 (14.3)	0
Vomiting	5 (14.3)	0
Abdominal pain	4 (11.4)	0
Constipation	4 (11.4)	0

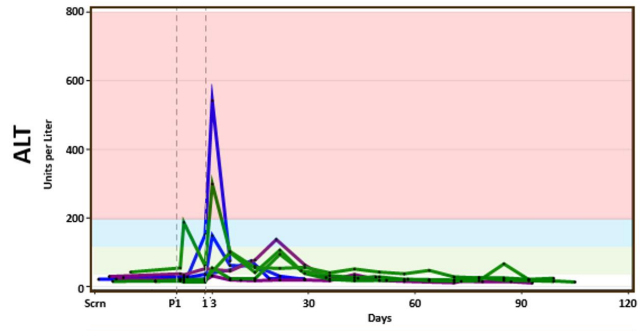
*Transient laboratory changes; resolved

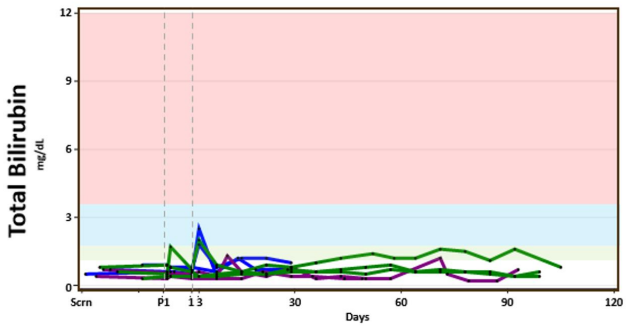
Data cut as of 10Sep25

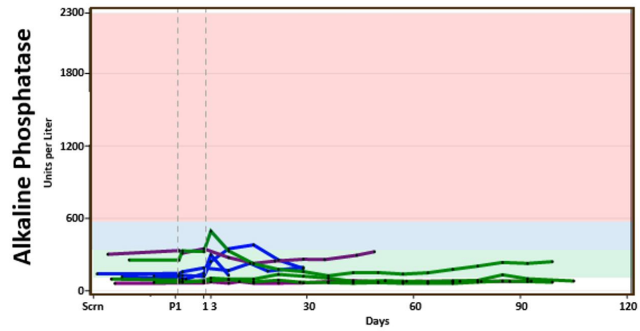
Presented at ESMO Berlin, 2025.

Early, Transient Increases in Hepatic Analytes Resolved with Continued Dosing

Patients primed with 0.1 mg prior to treatment dose (N=9; treatment doses: 0.3, 0.6, and 1.2 mg)

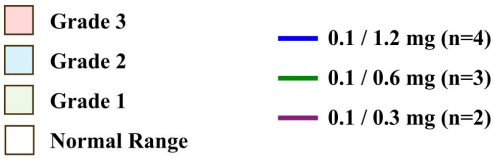






Summary of hepatic analyte changes:

- Early, transient, and asymptomatic
- Enabling on-time weekly treatment dosing
- Consistent with cholestasis (not on target toxicity)

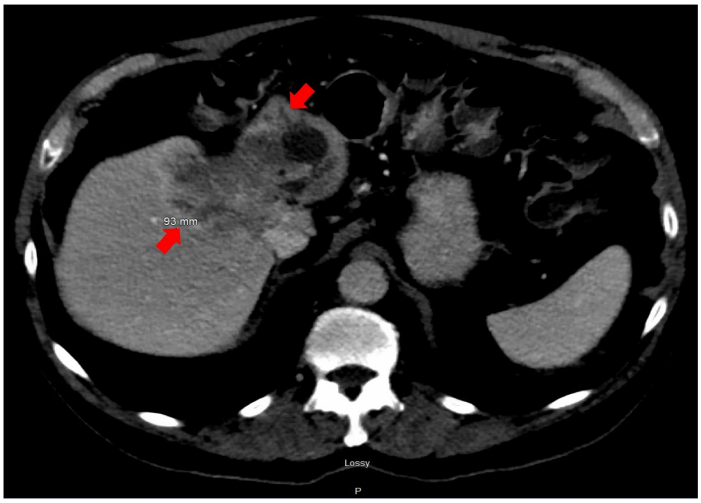


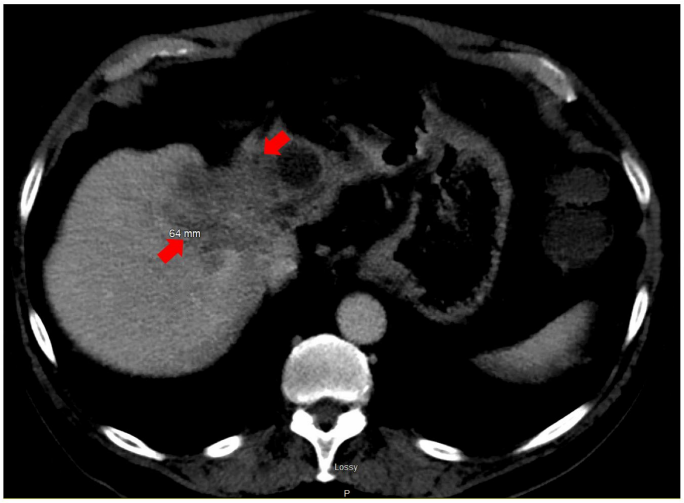
Presented at ESMO Berlin, 2025.

Live Data cut as of 10Sep2025

Confirmed Partial Response (31% Tumor Reduction) BA3182 at 0.6 mg in Patient with Intrahepatic Cholangiocarcinoma without Progression for >6 months

71-year-old male with stage IV cholangiocarcinoma previously treated on clinical trial with gemcitabine, cisplatin, durvalumab, and investigational agent.





Presented at ESMO Berlin, 2025.

Data cut as of 10Sep25

BA3182 Summary / Upcoming Milestones

- BA3182, a dual-conditionally binding CAB-EpCAM x CAB-CD3 T-cell engager continues to demonstrate a manageable safety profile with preliminary evidence of antitumor activity
- Early cytokine increases appear to cause brief, reversible cholestasis – consistent with tumor-selective targeting
- BA3182 treatment achieved an ongoing, confirmed partial response and multiple patients have experienced prolonged tumor control; currently testing dose levels up to 1.8mg and dose varying frequencies
- Dose escalation read out anticipated 1H’26

Ozuriftamab Vedotin (CAB-ROR2-ADC): Oropharyngeal Squamous Cell Carcinoma (OPSCC)

Significant Opportunity for Oz-V in OPSCC

Rapidly Growing Patient Population

- OPSCC incidence is increasing, largely due to HPV-driven disease[1,2]
- Up to 80% of OPSCC cases in the United States caused by HPV infection[3]
- By 2030, OPSCC will be the most common subtype of head and neck cancer in the US[1,2]

Potential to Address Significant Unmet Need

- OPSCC poorly served by SOC, including EGFR inhibitors[4-10]
- Oz-V has a compelling and differentiated profile in HPV+ OPSCC

Large Commercial Opportunity with Potential to Expand

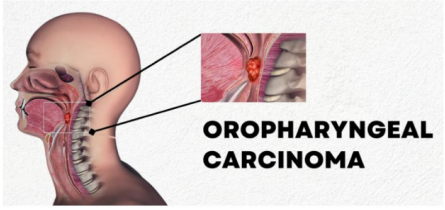
- $800mm WW peak sales projections in 2L+ OPSCC[11]
- Total WW OPSCC market value projected to be ~$3Bn by 2032[12]
- Total HPV+ solid tumors (ie, cervical) worldwide market valued at >$7Bn[13]

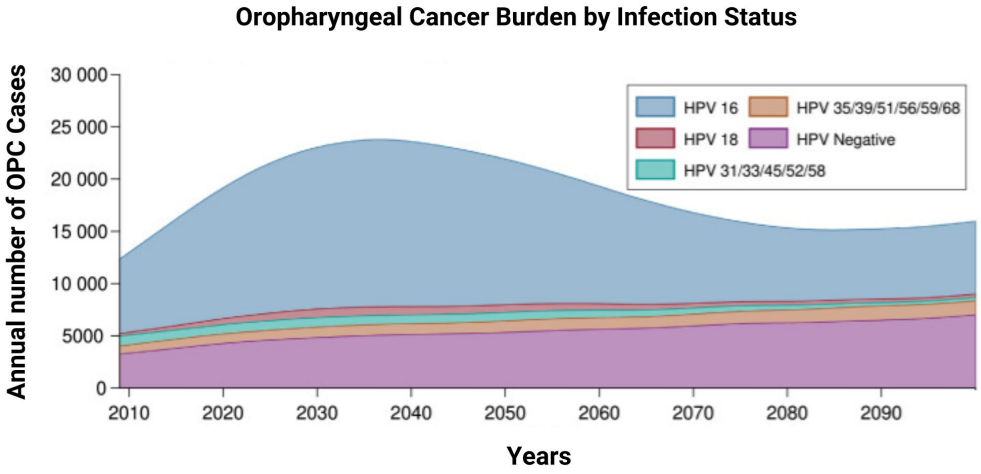
[1]Med Sci (Basel). 2023 Jun 13;11(2):42.; [2]Oral Oncol. 2021 Apr:115:105177.; [3]Dela J Public Health. 2023 Apr 22;9(1):26-28.[4]N Engl J Med 2016;375:1856-1867. [5]Journal of Clinical Oncology 2018; 36(15): 1551-1558. [6]Cohen E, et al. (2019) Lancet 393, 156–167. [7]British Journal of Cancer (2018) 119:153–159; https://doi.org/10.1038/s41416-018-0131-9. [8]2008 Jun 15;112(12):2710-9. doi: 10.1002/cncr.23442, [9]Erbitux USPI accessed 2024., [10]INTERLINK-1: Phase 3 study of cetuximab ± monalizumab /Volume 34, Supplement 2S554-S555 October 2023., [11]Internal BioAtla projections.; [12]https://www.coherentmi.com/industry-reports/oropharyngeal-cancer-market; [13]Cervical Cancer Therapeutics Market Size, Demand, Report to 2033; 23-cervix-uteri-fact-sheet.pdf

OPSCC, Oropharyngeal Squamous Cell Carcinoma; SOC, Standard of Care; WW, worldwide

OPSCC Incidence is Increasing

Proportion of new HPV+ OPSCC diagnoses now approach 80% in the US



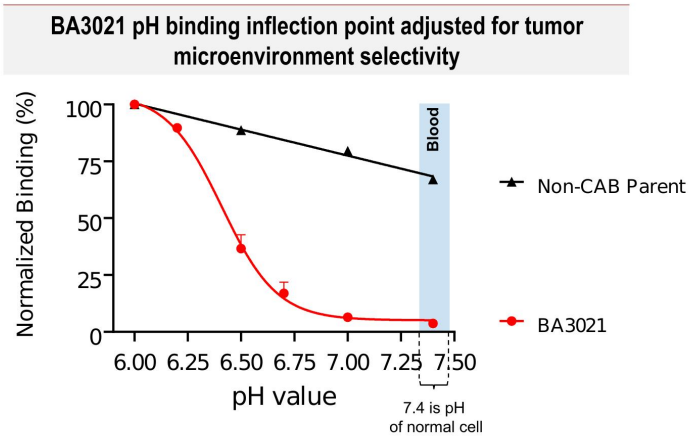


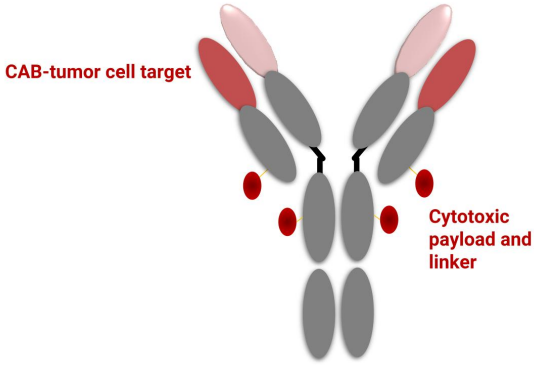
- Oropharyngeal cancer has now surpassed cervical cancer as the most common HPV-related malignancy
- More than 21,000 U.S. cases yearly compared to nearly 12,000 cervical cancer cases*

Damgacioglu H, Sonawane K, Chhatwal J, et al. Long-term impact of HPV vaccination and COVID-19 pandemic on oropharyngeal cancer incidence and burden among men in the USA: A modeling Study. The Lancet Regional Health – Americas. 2022;8:100143.
*CDC report.

Ozuriftamab Vedotin (Oz-V): CAB-ROR2-ADC

ROR2 is expressed in a variety of tumor types, with overexpression associated with metastasis, tumor resistance to chemotherapy, and poor prognosis





- MMAE-containing ADC (DAR4) with cleavable linker
- Humanized anti-ROR2 (N-terminal) IgG1
- ~2nM affinity (pH 6)
- MMAE-containing ADC (DAR4) with cleavable linker
- Epitope in Ig loop region

ROR2 Overexpression is Driven by HPV E6 / E7 Oncoproteins

80% of OPSCC cases in the United States caused by HPV infection

- HPV driven cancers
  - **Highest** ROR2 expression among SCCHN
  - HPV E6 and E7 oncoproteins **drive ROR2 overexpression**
  - ROR2 overexpression results in increased proliferation and invasiveness
- Oz-V conditionally and selectively eliminates ROR2-expressing cells

HPV infection drives ROR2 overexpression

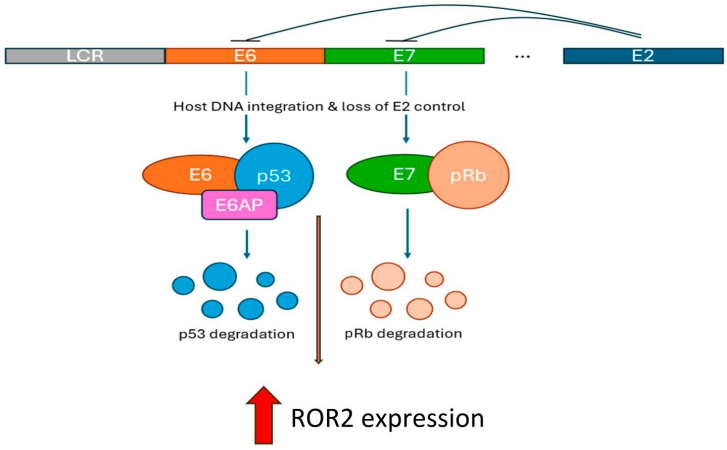


Figure adapted from Z. Lu, et al. Cancers. 2024, 16, 3474.

Carrero, et al. Oncogene (2019) 38:3551–3568; https://doi.org/10.1038/s41388-018-0659-4
Avincsal, et al. Oncol Rep. 2021 Jul;46(1):148. doi: 10.3892/or.2021.8099. Epub 2021 Jun 3. PMID: 34080643.

Demographics and Baseline Clinical Characteristics

HPV p16 positive and all patients

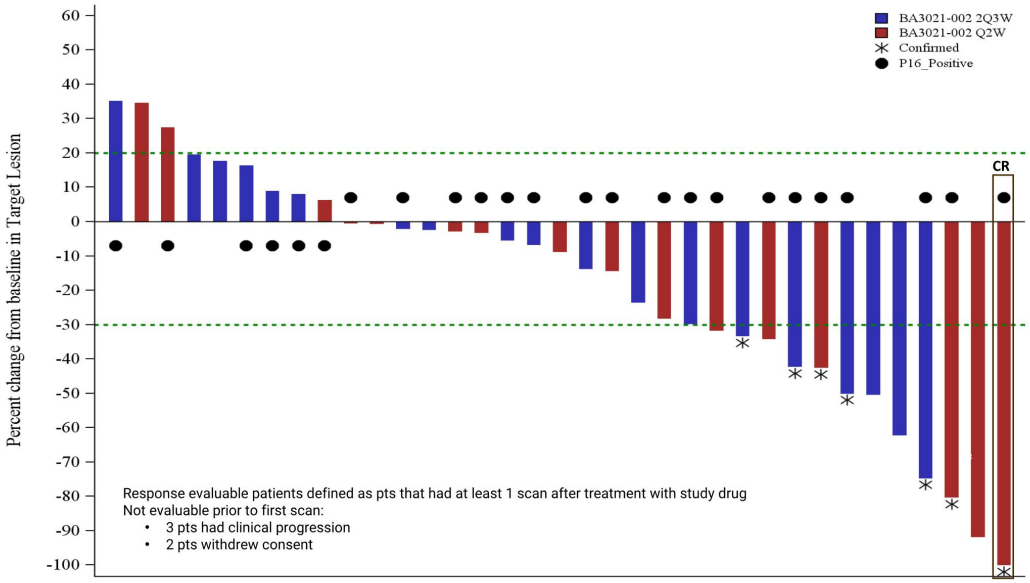
Ozuriftamab vedotin 1.8 mg/kg	OPSCC p16+[a]		Full Analysis
	2Q3W (n=10)	Q2W (n=13)	2Q3W and Q2W (N=40)
Age, mean (SD), y	65 (5)	62 (7)	65 (8)
Sex, n (%)			
Male	10 (100)	12 (92)	36 (90)
Female	0	1 (8)	4 (10)
ECOG performance, n (%)			
0	3 (30)	6 (46)	15 (38)
1	7 (70)	7 (54)	25 (63)
Number of prior lines of therapy, median	3	3	3
Prior anti–PD-1 exposure, n (%)	**10 (100)**	**13 (100)**	**40 (100)**
Prior platinum-based chemotherapy exposure, n (%)	**10 (100)**	**11 (85)**	**34 (85)**
Prior taxane exposure, n(%)	7 (70)	7 (54)	26 (65)

[a]HPV status was determined using p16 immunohistochemistry.
2Q3W = Days 1 and 8 of 21-day cycle

Data Cut Date: 31Oct2025

Oz-V in SCCHN Continues to Demonstrate Clinical Responses in a Heavily Pretreated Population

1.8 mg/kg Q2W and 2Q3W; Median of 3 prior lines of therapy



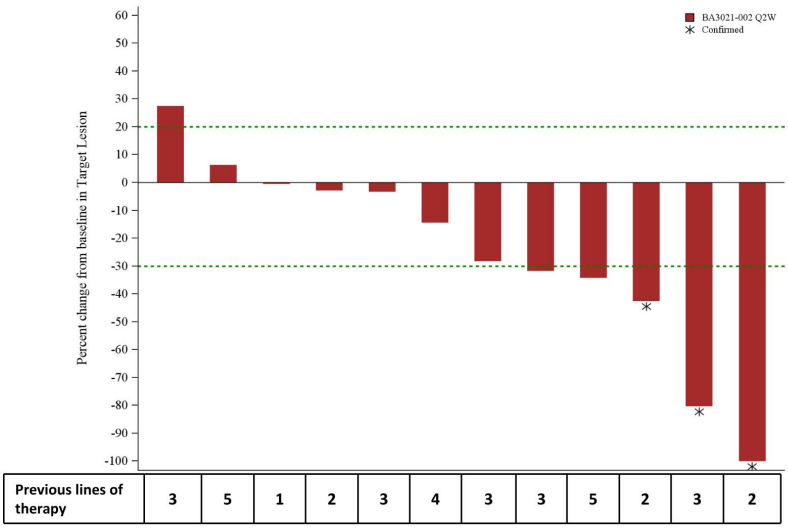
Response evaluable patients defined as pts that had at least 1 scan after treatment with study drug
Not evaluable prior to first scan:

- 3 pts had clinical progression
- 2 pts withdrew consent

Data Cut Date: 31Oct2025

Oz-V in SCCHN p16+^ OPSCC

1.8 mg/kg Q2W; Median of 3 prior lines of therapy



Previous lines of therapy	3	5	1	2	3	4	3	3	5	2	3	2

	Q2W
Responders (confirmed & unconfirmed)	**42%** (5/12*)
Responders (confirmed)	**25%** (3/12*)
DCR	**92%** (11/12*)
Median DOR (months)	***9.9 ongoing***
Median PFS (months)	***4.7 ongoing***
Median OS (months)	***11.6 ongoing***

^ p16 is strongly associated with HPV; HPV testing in progress for unknown patients

* Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Not evaluable prior to first scan:

- 1 patients had clinical progression

Data Cut Date: 31Oct2025

2L+ HPV+ OPSCC: Cross Trial Comparisons of ORR and OS

Considerably improved response rate and survival among a heavily pretreated trial population

Cross trial comparisons**	Median prior lines of therapy	ORR (%)	OS (months)***
Ozuriftamab vedotin monotherapy (1.8 mg/kg Q2W) *Study Ongoing**	**3**	**42% ORR 25% cORR**	**11.6 ongoing**
SOC (methotrexate, docetaxel, or cetuximab)[1,2]	2	3.4%	4.4
Cetuximab monotherapy[3,4,5]	1	0%	NA
Not approved			
Petosemtamab (1500 mg Q2W)	2	13%	NA

Accelerated Approval Opportunity (ORR)

Anti-EGFR therapies have inferior therapeutic outcomes among HPV+ patients

*Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug;
Not evaluable prior to first scan: 1 patients had clinical progression

**The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

***From Dr. Alan Ho at MSKCC: "...extrapolating the OS of CheckMate 141 patients after progression on Nivo, we are figuring the OS of these patients to be about 7 months (OS of 9 -PFS of 2 months) post PD-1. Some of the retrospective papers (https://pubmed.ncbi.nlm.nih.gov/31574417/ & https://pubmed.ncbi.nlm.nih.gov/31864957/ we reviewed post-PD1 saw that upper limit of OS was of about 7-8.5 months post PD-1. **Figuring what the upper and lower estimates are, we think 6 months OS is a fair estimate for the null.**"

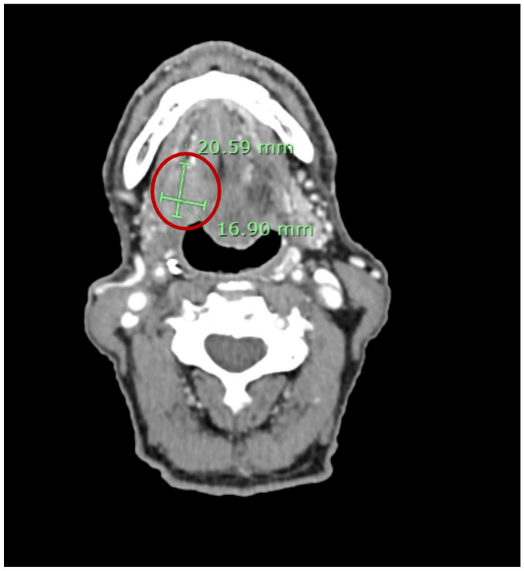
[1]N Engl J Med 2016;375:1856-1867. [2] Journal of Clinical Oncology 2018; 36(15): 1551-1558. [3]2008 Jun 15;112(12):2710-9. doi: 10.1002/cncr.23442, [4]Erbitux USPI accessed 2024., [5]INTERLINK-1: Phase 3 study of cetuximab ± monalizumab /Volume 34, Supplement 2S554-S555 October 2023
SOC, Standard of Care (Cetuximab, Methotrexate or Docetaxel); NA, not available

Data Cut Date: 31Oct2025

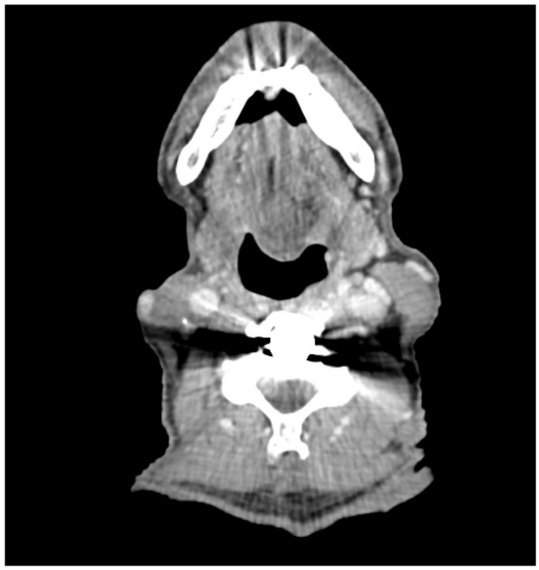
Confirmed Complete Response Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

76-year-old male, stage IV – recurred after surgery and radiation therapy; prior treatments: pembrolizumab; clinical trial bispecific anti-PD1/CD47; patient remains in complete response >16 months after Oz-V treatment initiation

Baseline - July 14, 2023



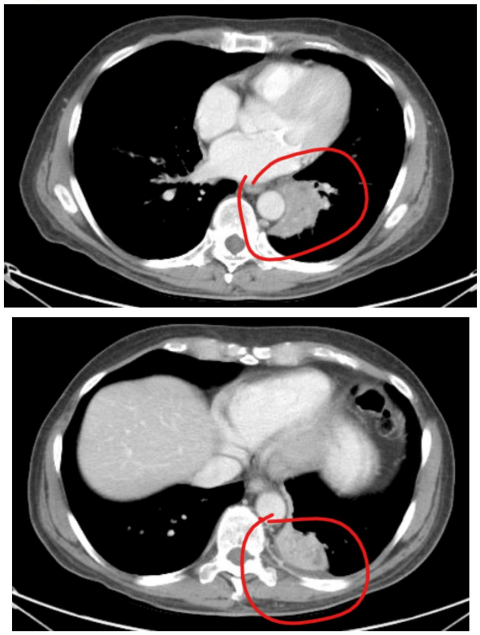
On Treatment – December 8, 2023



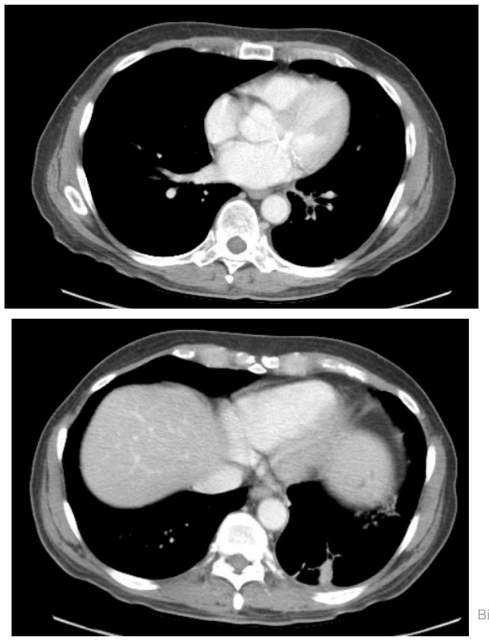
Partial Response (-80%) Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

63-year-old male, stage IV – recurred after surgery and radiation therapy; prior treatments: platinum, investigational agents including pembrolizumab

Baseline – December 4, 2025

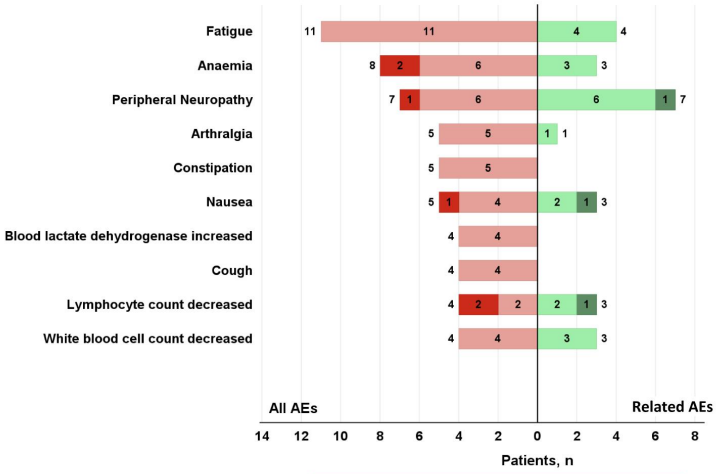


On Treatment – February 15, 2025



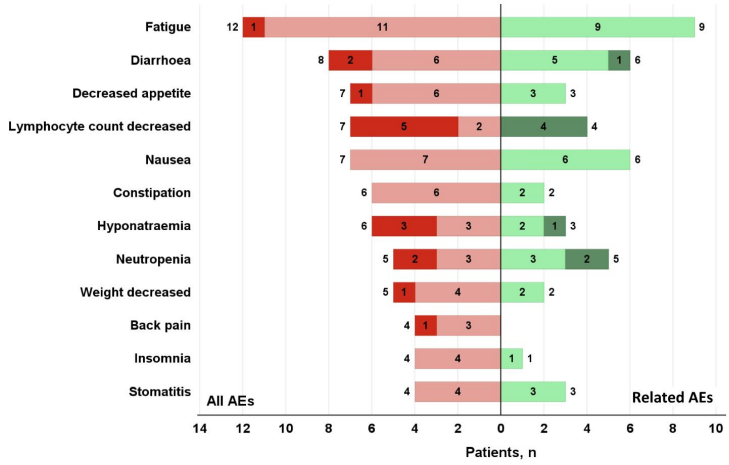
Most frequent AEs of any grade (>15% of patients) (N=40)

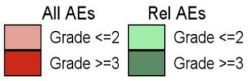
Q2W (n=20)



Treatment-Related Adverse Events	
Related Grade 3	3 (15%)
Related Grade 4	0 (0%)
Related Serious	0 (0%)

2Q3W (n=20)





Treatment-Related Adverse Events	
Related Grade 3	6 (30%)
Related Grade 4	2 (10%)
Related Serious	2 (10%)

Data Cut Date: 31Oct2025

Ph2 Oz-V: Overall Safety Summary of SCCHN patients

Investigator Choice[3] related AEs G3 or 4 = 35% - Considerably higher than Oz-V 1.8 mg/kg Q2W

	1.8 mg/kg Q2W (N=20)	1.8 mg/kg 2Q3W (N=20)[4]	Total (N=40)[4]
Any Adverse Events (AEs)	19 (95%)	20 (100%)	38 (95%)
Related AEs with CTCAE[1] Grade 3 or 4[2]	**3 (15%)**	7 (35%)	10 (25%)
Any related serious AEs[2]	0	2 (10%)	2 (5%)
Possibly Related AEs leading to death[2]	0	0	0
Related AEs leading to treatment discontinuation[2]	1 (5%)	1 (5%)	2 (5%)

[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.
[3]N Engl J Med 2016;375:1856-1867. [2] Journal of Clinical Oncology 2018; 36(15): 1551-1558
[4]One patient from Phase 1 not included

Phase 2 Oz-V Safety Data

Most frequent treatment-emergent related adverse events (>15%)

	1.8 mg/kg Q2W (N=20)		1.8 mg/kg 2Q3W (N=20)^		Total (N=40)^	
Preferred Term	**All Grades n (%)**	**Grade 3-4 n (%)**	**All Grades n (%)**	**Grade 3-4 n (%)**	**All Grades n (%)**	**Grade 3-4 n (%)**
Number of subjects with at least one TRAE	19 (95)	12 (60)	20 (100)	16 (80)	39 (98)	28 (70)
Fatigue	11 (55)	0	12 (60)	1 (5)	23 (58)	1 (3)
Nausea	5 (25)	1 (5)	7 (35)	0	12 (30)	1 (3)
Anaemia	8 (40)	2 (10)	3 (15)	1 (5)	11 (28)	3 (8)
Constipation	5 (25)	0	6 (30)	0	11 (28)	0
Lymphocyte count decreased	4 (20)	2 (10)	7 (35)	5 (25)	11 (28)	7 (18)
Decreased appetite	3 (15)	0	7 (35)	0	10 (25)	1 (3)
Peripheral neuropathy¥	7 (35)	1 (5)	3 (15)	1 (5)	10 (25)	2 (5)
Diarrhoea	1 (5)	0	8 (40)	2 (10)	9 (23)	2 (5)
Hyponatraemia	3 (15)	1 (5)	6 (30)	4 (20)	9 (23)	5 (13)
Arthralgia	5 (25)	0	3 (15)	0	8 (20)	0
Weight decreased	3 (15)	0	5 (25)	1 (5)	8 (20)	1 (3)
Cough	4 (20)	0	3 (15)	0	7 (18)	0
Blood lactate dehydrogenase increased	4 (20)	0	3 (15)	0	7 (18)	0
Hypercalcaemia	3 (15)	1 (5)	3 (15)	1 (5)	6 (15)	2 (5)
Neutropenia*	1 (5)	0	5 (25)	2 (10)	6 (15)	2 (5)
White blood cell count decreased	4 (20)	0	2 (10)	0	6 (15)	0

^ One patient from Phase 1 not included

* Derived from neutropenia, and neutrophil count decreased

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

Data Cut Date: 31Oct2025

FDA End of Phase 2 Meeting: Key Outcomes

Dual primary endpoints of ORR and OS to support potential accelerated approval followed by full approval

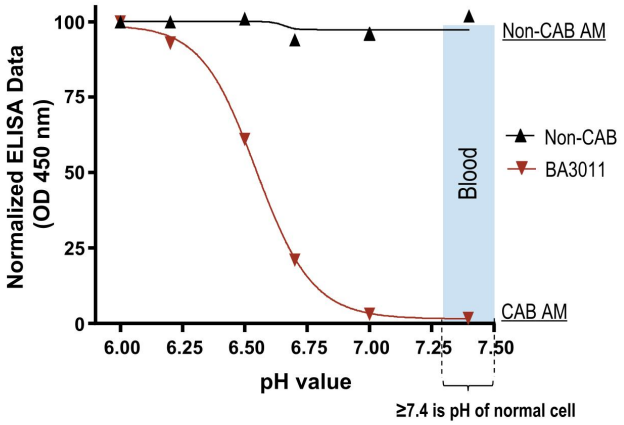
- **Pivotal Trial Design:** For full approval, approximately 300 patients prospectively randomized and stratified, one to one between two open label treatment arms
- **Oz-V Dose and Regimen:** Patients randomized to the investigational arm will receive 1.8 mg/kg every other week
- **Investigator's Choice (IC) control arm:** Patients randomized to the control arm will receive either cetuximab, docetaxel, or methotrexate monotherapy
- **Accelerated Approval Endpoint:** Based on interim analysis of enrolled patients, statistically significant improvement of confirmed Overall Response Rate (ORR) by Blinded Independent Central Review (BICR) supported by an adequately characterized Duration of Response (DOR) without detriment in OS
- **Full Approval Endpoint:** Statistically significant improvement of OS

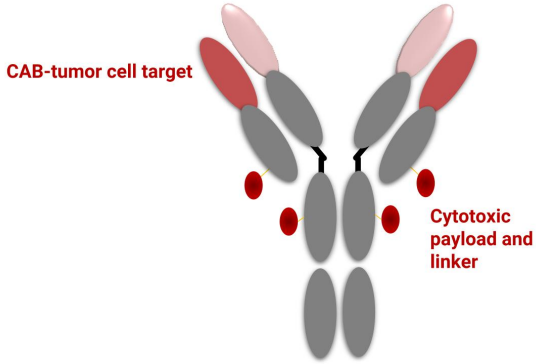
Mecbotamab Vedotin (CAB-AXL-ADC): mKRAS Non-Small Cell Lung Cancer (NSCLC)

Mecbotamab Vedotin (Mec-V): 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

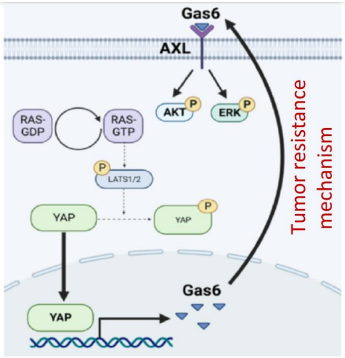
1. Gay CM, Balaji K, Byers LA. *Br J Cancer*. 2017;116(4):415-423. 2. Zhang G, Wang M, Zhao H, Cui W. *Oncol Lett*. 2018;15(3):2726-2734.

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.

AXL Plays a Crucial Role in the Survival of mutated KRAS (mKRAS) NSCLC Cells

- mKRAS represents 30% of all NSCLC patients
- 70% to 85% of mKRAS NSCLC express AXL by IHC and higher by mRNA analysis
- AXL over-expression drives aggressive tumor characteristics, resistance to therapies, and poor patient outcomes
- Significant opportunity for Mec-V (CAB-AXL-ADC)

mKRAS leads to upregulation and activation of AXL expression

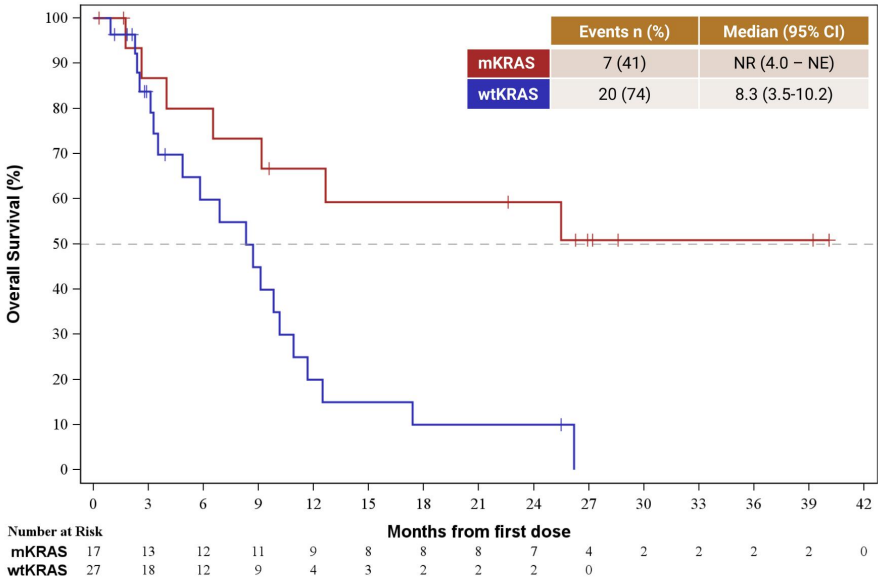


Adapted from Morimoto et al. Cancer Letters 587 (April 2024)

* Morimoto et al., Cancer Letters 587 (2024) 216692 and BioAtla study BA3011-002
IHC- Immunohistochemical assay

Mec-V 1.8 mg/kg Q2W Overall Survival mKRAS vs wtKRAS NSCLC

Median of 3 prior lines of tx



median of 2 prior lines of tx	mKRAS (Q2W only)
Responders (confirmed & unconfirmed)	31% (5/16^)
Responders (confirmed)	19% (3/16^)
DCR	81% (13/16^)
PFS	4.6
One-year landmark OS	67%

^Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug
Prior to first scan: one patients withdrew consent

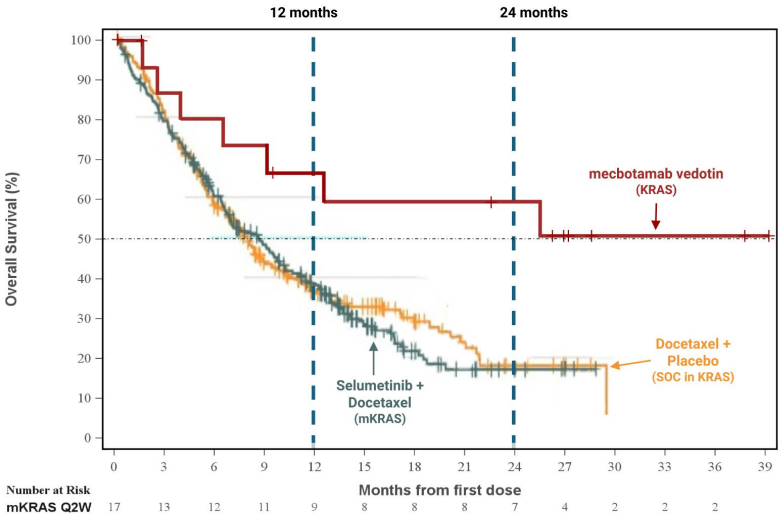
Data Cut Date: 19Aug2025

Mec-V 1.8 mg/kg Q2W Associated with Exceptional Overall Survival in mKRAS NSCLC

Overall survival cross trial comparison[1]

	mecbotamab vedotin (1.8 mg/kg Q2W)	Docetaxel[3]
Population	mKRAS	mKRAS
Number of patients	17	256
Prior Lines of tx	2	1
Prior Taxane	**53%**	**0%**
ORR	31%[2]	14%
PFS (months)	4.5	2.8
OS (months)	Not reached	7.9
Survival at 12 months	67%	<40%
Survival at 24 months	59%	<20%

FDA guidance: 2L+ NSCLC Phase 3 trial will be randomized mecbotamab vedotin versus docetaxel (full-approval = OS)



[1] The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

[2] Confirmed and unconfirmed responders.

[3] JAMA. 2017 May 9;317(18):1844–1853

Data Cut Date: 19Aug2025

Competitive Response Rate in 2L+ mKRAS NSCLC

Median Overall Survival ranges from 6 to 11 months in mKRAS NSCLC when treated with docetaxel in the 2L+

Cross trial comparisons**	Pts	Median prior lines of therapy	ORR	Median OS (months)
Mecbotamab Vedotin (1.8 mg/kg Q2W regimen) *Study Ongoing**	**17**	**3**	**31%*** **	**Not Reached (1 year landmark at 67%)**
SELECT-1[1] (all mKRAS variants)	256	1	13.7%	7.9
Real-life ESME cohort[2] (all mKRAS variants)	1000+	1	NA	6.1 to 10.6*
Codebreak 200[3] (mKRAS G12C)	174	2	13.2%	11.3
KRYSTAL-12[4] (mKRAS G12C)	152	2	9.2%	NA

Docetaxel Studies

1. Janne, P et al. JAMA. 2017 May 9;317(18):1844–1853. (2) Thomas QD, et al. ESMO Open, Volume 9, Issue 6, 103473. (3) de Langen AJ, et al. Lancet 2023; 401:733-746; (4) Mok TS, Journal Clinical Oncology 2024; 42(17_suppl):LBA8509.

* PD-1/PD-L1 monotherapy, PT-based CT without a PD-1/PD-L1, or Docetaxel monotherapy or combination

**The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

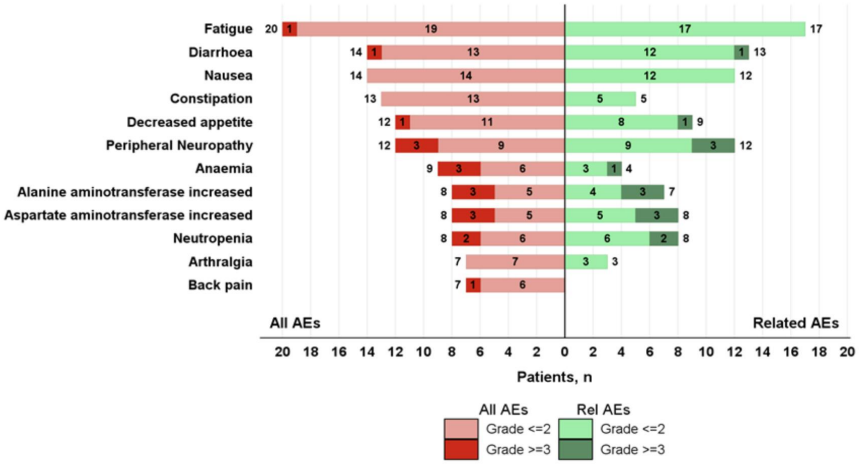
***Confirmed and Unconfirmed

Ph2 Mec-V: Overall Safety Summary of NSCLC patients

1.8 mg/kg Q2W with or without nivolumab generally well-tolerated; only 7% discontinuation due to related AEs

	N=45 (%)
Any Adverse Events (AEs)	45 (100)
Related AEs with CTCAE[1] Grade 3 or 4[2]	14 (31)
Any Related Serious AEs[2]	6 (13)
Possibly Related AEs leading to death[2]	0
Related AEs leading to treatment discontinuation[2]	3 (7)

Most frequent AEs any grade occurring at a rate >15%



[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: 19Aug2025

Potential for Mec-V to Address All mKRAS NSCLC

1.8 mg/kg Q2W associated with improved overall survival and favorable benefit / risk profile

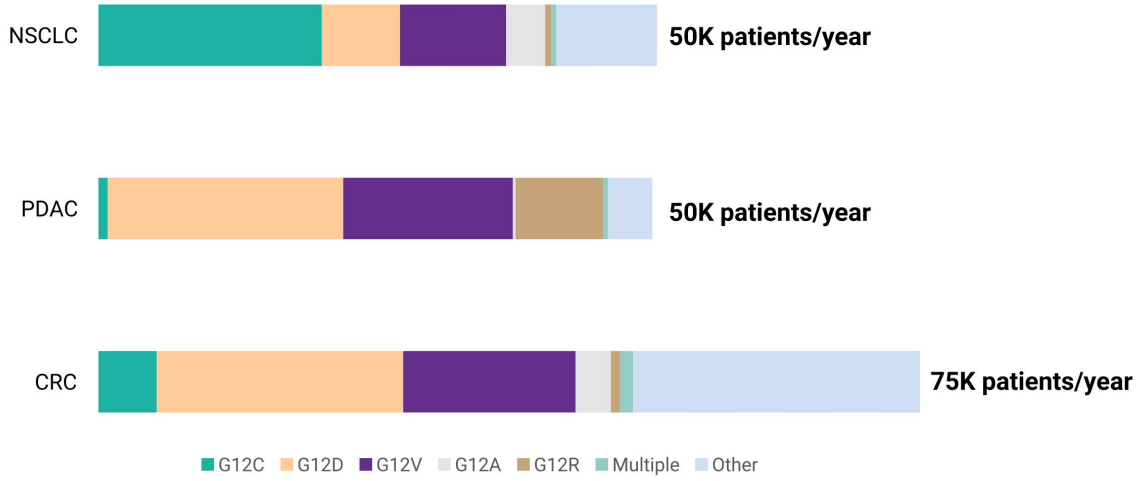
- Promising anti-tumor activity among patients whose tumors express KRAS mutations
  - mKRAS represents 30% of all NSCLC patients and is associated with increased AXL expression
  - 1.8 mg/kg Q2W associated with exceptional overall survival even in heavily pretreated patients
  - 67% alive at a landmark of one year
  - 59% alive at a landmark of two years; standard of care agents result in less than 20% alive at the two year*
  - 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 positioning for a future pivotal trial

*The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

Significant Opportunity for Mec-V to Expand Beyond mKRAS NSCLC

KRAS mutations most commonly found in CRC, NSCLC and PDAC

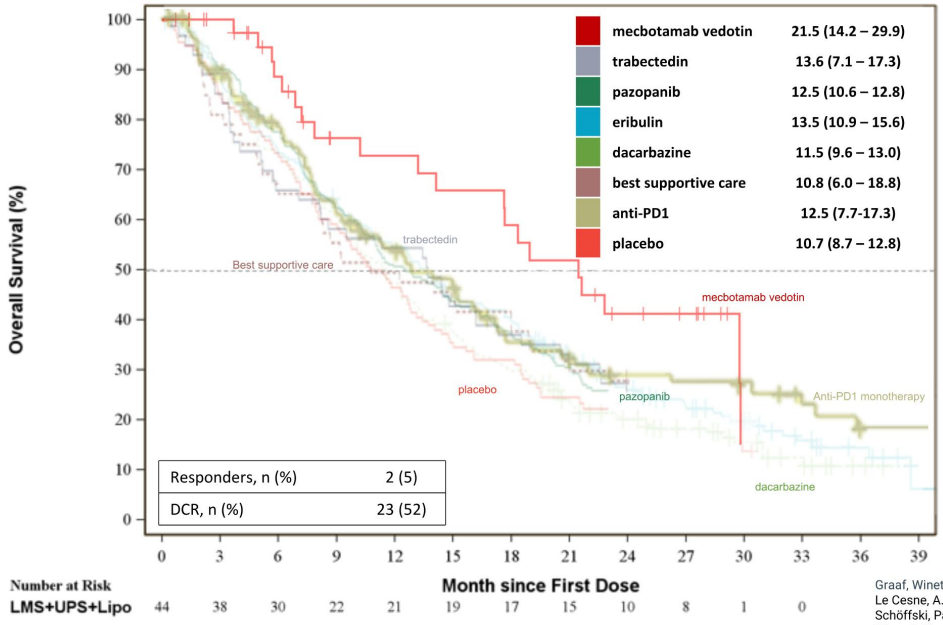
Estimated US incidence of select mKRAS cancers and distribution of selected mKRAS variants



Lee J., Sivakumar S., Schrock A., et al. NPJ Precision Oncology, 2022. PMID: 36494601.
CRC: colorectal cancer; NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma

Improved Overall Survival also Observed in Soft Tissue Sarcoma Mirrors Overall Survival Observed in mKRAS NSCLC

LMS, UPS, and Liposarcoma: 1.8 mg/kg Q2W Mono and anti-PD-1 Combo; cross-trial comparison*



Data Cut Date: 24Mar2025

Mec-V in Sarcoma:

Median of 2 prior lines of Tx

Median OS of 21.5 months

Landmark OS at 1 year 73% (54-85)

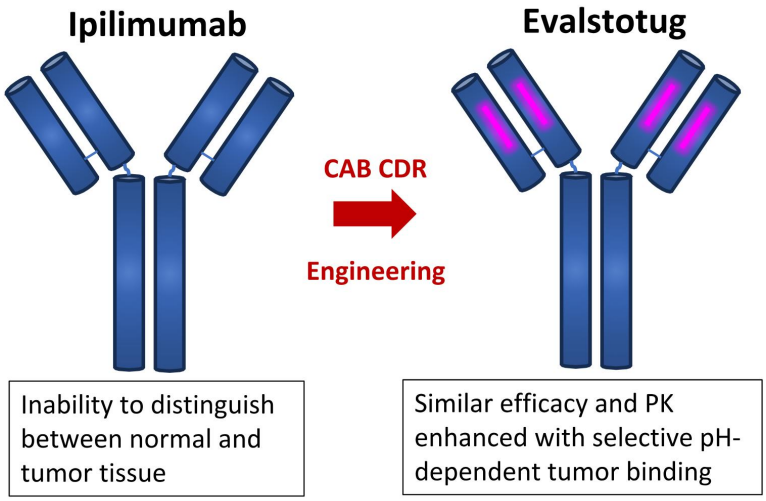
Graaf, Winette van der et al. *The Lancet* 379 (2012): 1879-1886.
Le Cesne, A. et al Annals of Oncology, Volume 32, Issue 8, 1034 - 1044
Schöffski, Patrick et al. The Lancet, Volume 387, Issue 10028, 1629 – 1637
Miao, R. *Cancer Immunol Immunother* 72, 2521–2527 (2023)

*The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials unreliable.

Evalstotug (CAB-CTLA-4)

Evalustotug is a Next Generation Adaptation of Ipilimumab

CAB-CTLA4 selectively active in tumor microenvironment, thereby reducing immune mediated adverse events (imAEs)



Evalstotug is a “CABified” Ipilimumab: A Next Generation CTLA-4 Inhibitor

Preserved efficacy with reduced toxicity in combination with PD-1

- Ipilimumab (ipi) CDRs modified to bind at tumor cell acidic pH, but not at normal pH leading to evalstotug:
  - Preserved affinity and epitope
  - Equivalent E_{Max} and EC_{50} in preclinical models
  - However, observed substantially reduced G.I. toxicity in primates

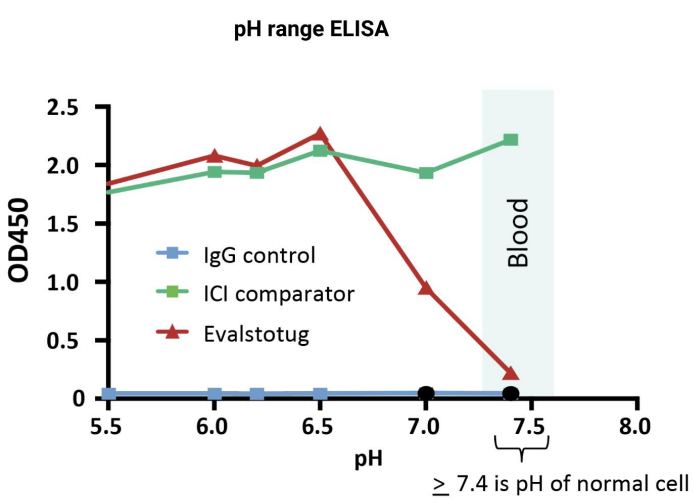
- CAB CTLA-4, evalstotug, enables targeted exposure in TME enabling lower imAE relative to ipi

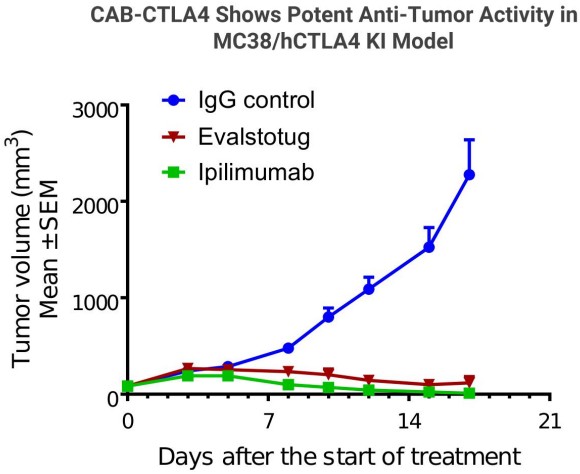
Abbreviations: ADCC, antibody-dependent cellular cytotoxicity; CAB, Conditionally Active Biologic; CD, cluster of differentiation; CDR, complementarity-determining region; CTLA-4, cytotoxic T-lymphocyte associated protein 4; EC50, concentration producing 50% Emax; Emax, maximum effect; imAE, immune mediated adverse event; PD-1, programmed cell death protein 1; t1/2, half-life; Treg, regulatory T cells.

1. Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.

Reversible Binding in the TME

Comparison of evalstotug binding to CTLA-4 in different pH conditions

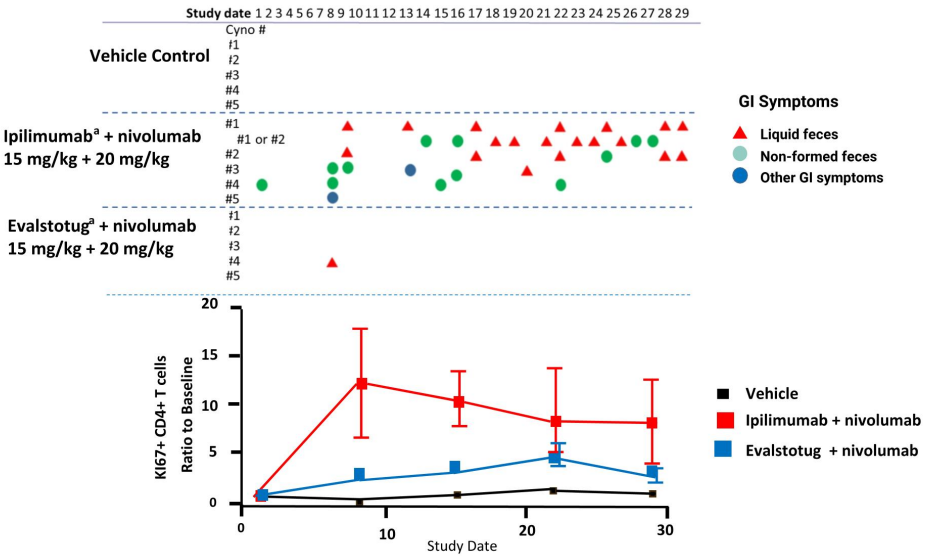




BA3071 induces complete tumor regression in mouse tumor model. Mice were dosed with IgG control and anti-CTLA antibodies at 10mg/kg (equivalent to 1mg/kg anti-CTLA-4 human dose), IP, BIW, N=12 mice/group.

Abbreviations: CTLA-4, cytotoxic T-lymphocyte associated protein 4; ICI, immune checkpoint inhibitor; IgG, immunoglobulin G; OD450, optical density at 450 nm; TME, tumor microenvironment.
Note: Figures modified from Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.

Evalstotug Reduced GI Toxicity in Primates



Abbreviations: CD, cluster of differentiation; Cyno, cynomolgus macaque; GI, gastrointestinal; QW, once weekly.
Note: Ipilimumab and evalstotug had the same half-life and exposure in this model. Figure modified from Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.
[a]Ipilimumab analog or evalstotug 15 mg/kg (≈11 mg/kg human dose) + nivolumab 20 mg/kg (≈14.6 mg/kg human dose) both administered QW for 4 weeks.

Evalstotug Across Multiple Tumor Types

Evalstotug at 350mg Q3W (5mg/kg based on 70 kg pt) in Combination With PD1: Demographics – Across Multiple Tumor Types; n=17

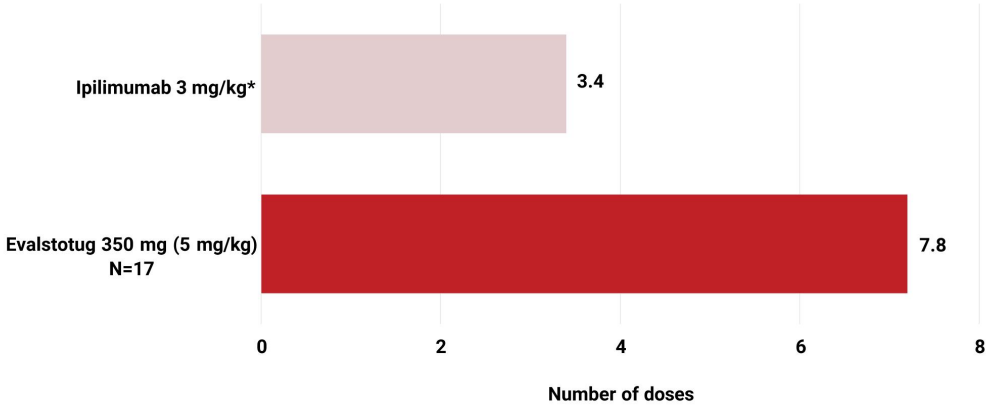
82% patients experienced failure of prior PD-1 treatment

	Total (N=17)
Age, y, mean (SD)	60 (14)
Sex, n (%)	
Female	8 (47)
Male	9 (53)
White race, n (%)	14 (82)
ECOG, n (%)	
0	12 (71)
1	5 (29)
Prior Anti-PD-1 Therapy, n (%)	14 (82)

	Total (N=17)	Prior # of treatments
Tumor type, n (%)		
Melanoma	11	0 - 1
Gastric	2	3 - 5
Renal cell	1	3
Cervical	1	3
NSCLC	1	3
aHCC	1	5

Patients Treated with Evalstotug Received More Than Twice as Many Doses Compared with Reported Ipi Dosing

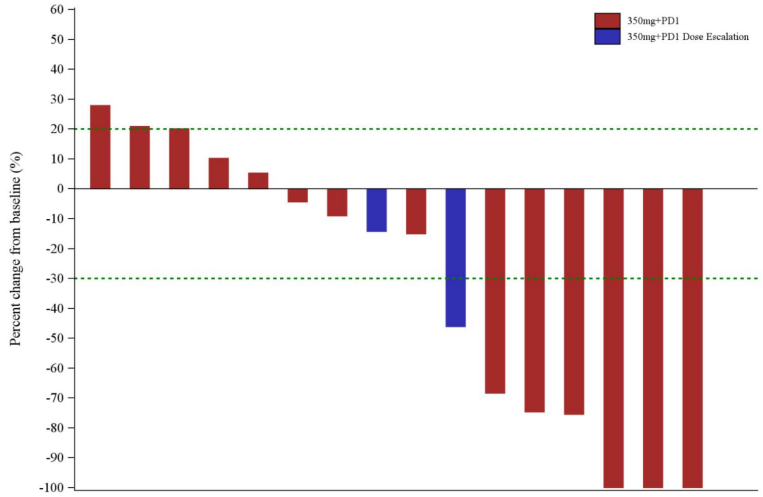
Mean number of evalstotug 350 mg doses vs ipilimumab when in combination with PD1



Note: Mean number of doses for ipilimumab is based on a retrospective observational study.
Evalstotug 350 mg and 700 mg are dose-equivalent to ipilimumab 5 mg/kg and 10 mg/kg, respectively.
*Mohr P, et al. J Eur Acad Dermatol Venereol. 2018;32(6):962-971.

Evalstotug at 350mg Q3W (5mg/kg based on 70 kg pt) in Combination with PD1 Across Multiple Tumor Types

3 Complete Responders with 2 in melanoma and 1 in cervical cancer



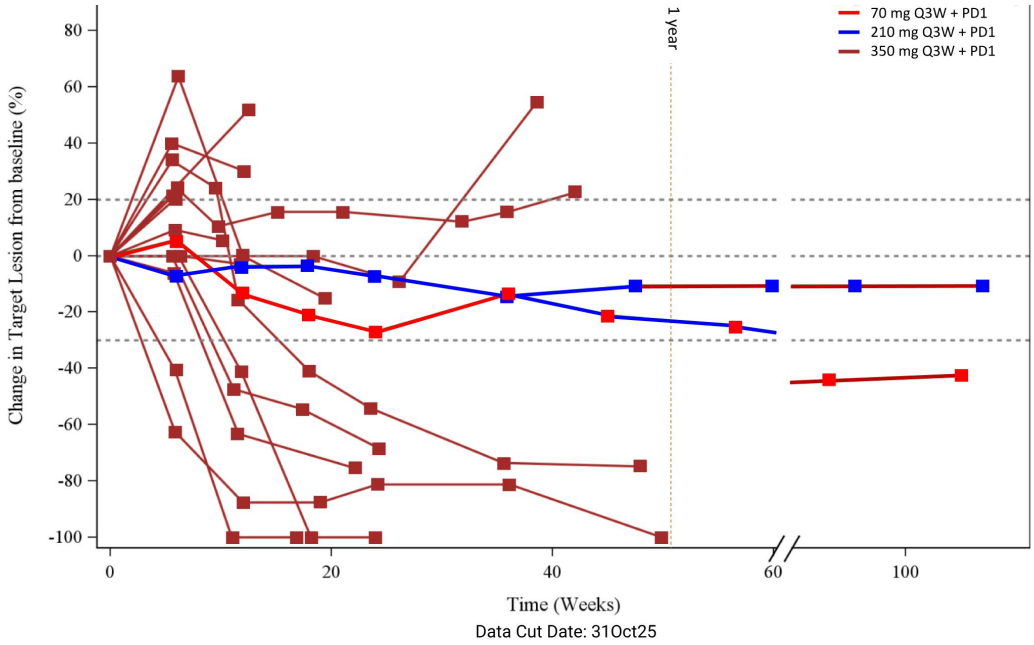
	Total
Responders (confirmed & unconfirmed)	**44%** (7/17[1])
Responders (confirmed)	**41%** (7/17[1])
DCR	**76%** (13/17[1])
OS	***ongoing***

[1]Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Data Cut Date: 31Oct25

Evalstotug in Combination with PD1: 7 of 17 Achieved Response

Durable antitumor activity across multiple solid tumor types



Data Cut Date: 31Oct25

Evalstotug Melanoma (with or without prior ICI treatment)

Evalstotug In Combination with PD1 in Melanoma (with or without prior ICI treatment; n=16): Demographics

Evalstotug 350 mg or 700 mg Q3W (5 mg/kg or 10 mg/kg based on 70 kg patient)

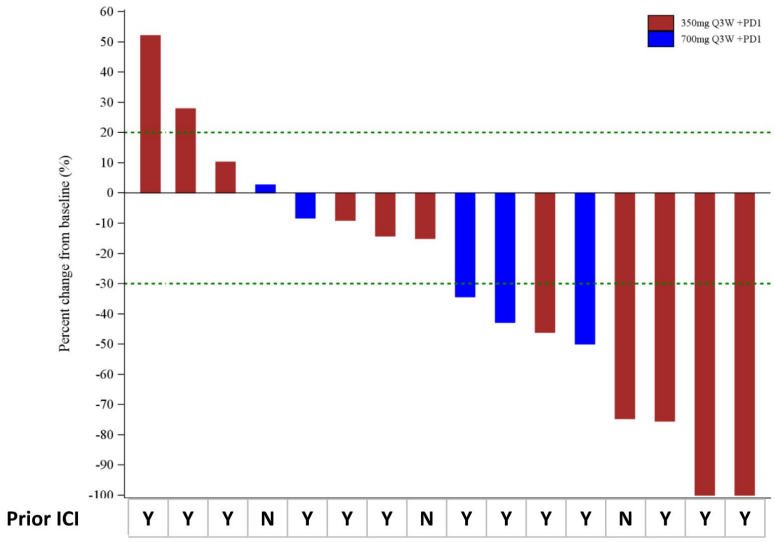
	Total (N=16)
Age, y, mean (SD)	61 (14)
Sex, n (%)	
Female	10 (62)
Male	6 (38)
White race, n (%)	16 (100)
ECOG, n (%)	
0	12 (75)
1	4 (25)
Prior ICI Therapy Status, n (%)	
No prior ICI treatment	**3 (19)**
Adjuvant	**10 (62)**
Metastatic setting	**3 (19)**

ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

Evalstotug in Combination with PD1 in Melanoma (with or without prior ICI treatment)

8 responders among 16 patients; 81% of patients had prior ICI



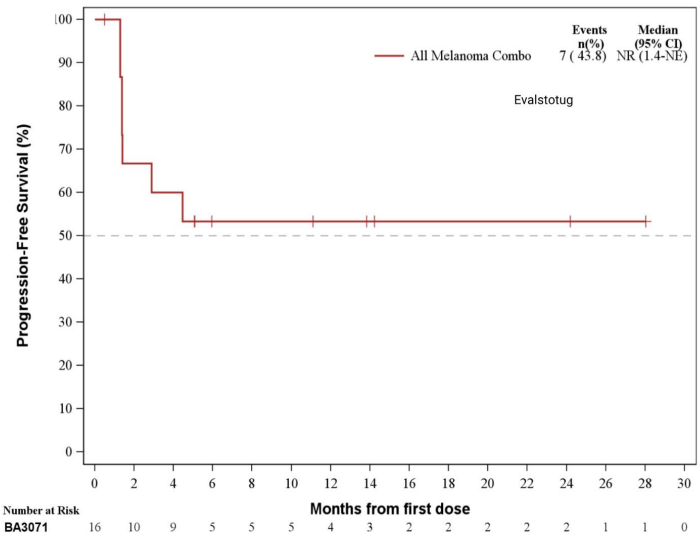
	Total
Responders (confirmed & unconfirmed)	**50%** (8/16[1])
Responders (confirmed)	**50%** (8/16[1])
DCR	**94%** (15/16[1])
PFS	***Not Reached***
OS	***Not Reached***

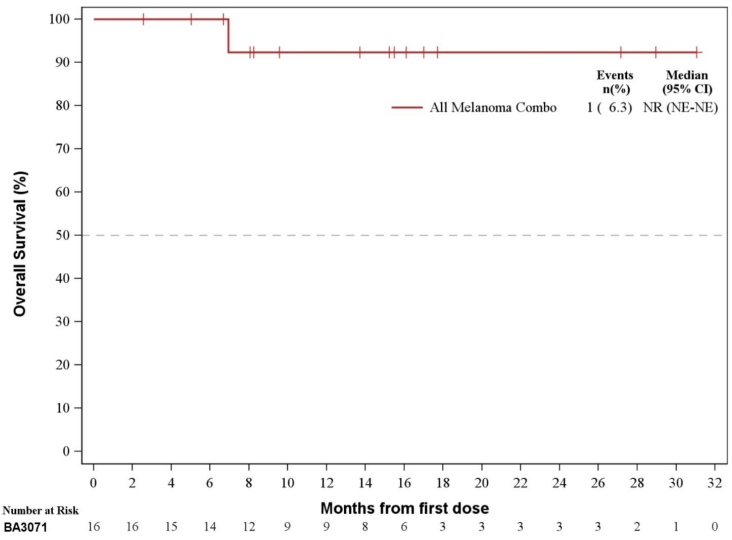
[1]Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Data Cut Date: 31Oct25

50% ORR and 94% DCR in Melanoma (with or without prior ICI treatment)

81% patients had received prior PD1





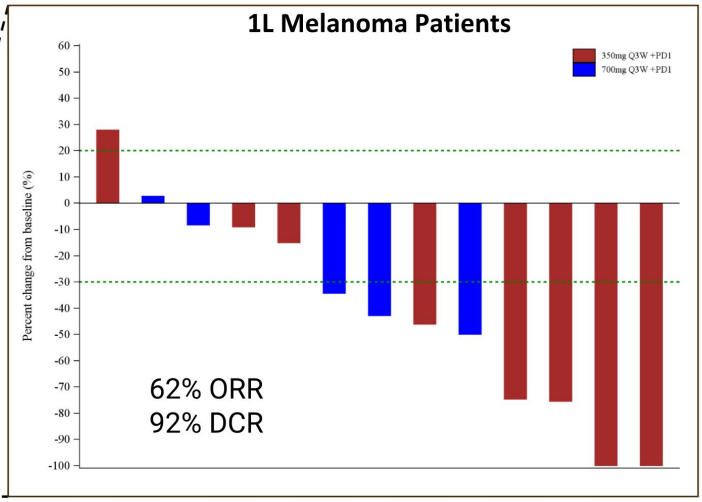
Data Cut Date: 31Oct25

Evalstotug 1L Melanoma – prior adjuvant therapy or no prior ICI treatment

Evalstotug In Combination with PD1 in Melanoma (no prior treatment or prior adjuvant therapy; n=13): Demographics and Waterfall

Evalstotug 350 mg or 700 mg Q3W (5 mg/kg or 10 mg/kg based on 70 kg patient)

	Total (N=16)
Age, y, mean (SD)	61 (14)
Sex, n (%)	
Female	10 (62)
Male	6 (38)
White race, n (%)	16 (100)
ECOG, n (%)	
0	12 (75)
1	4 (25)
Prior ICI Therapy Status, n (%)	
No prior ICI treatment	**3 (19)**
Adjuvant	**10 (62)**
Metastatic setting	**3 (19)**

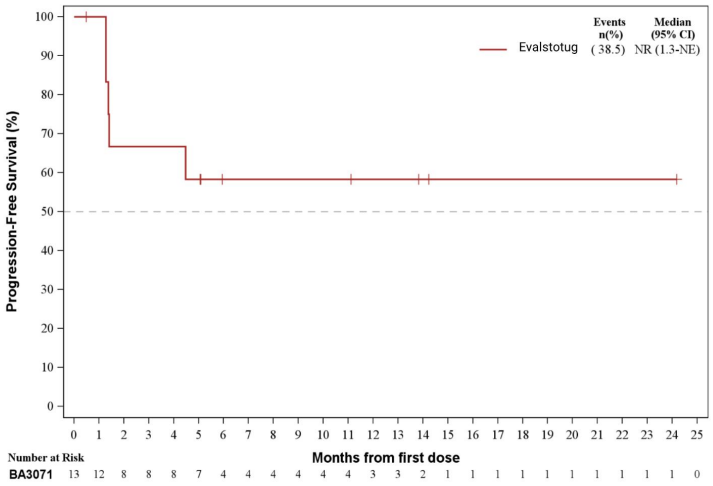


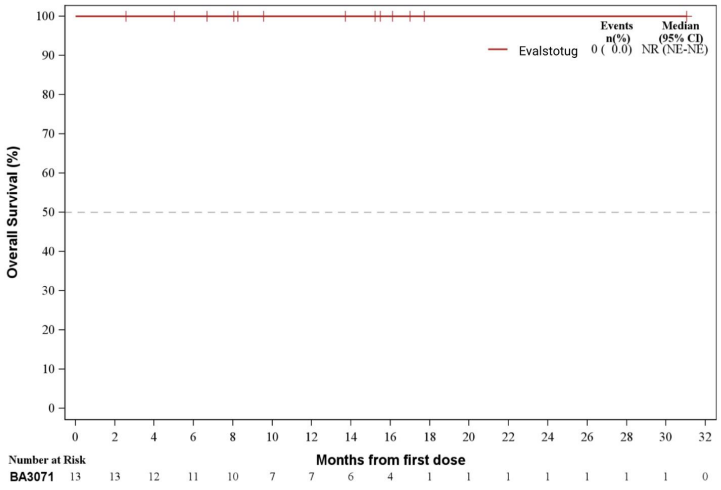
ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

62% ORR and 92% DCR in Melanoma (with or without prior ICI treatment; n=13) Evalstotug in Combination with PD1

81% patients had received prior PD1 adjuvant treatment





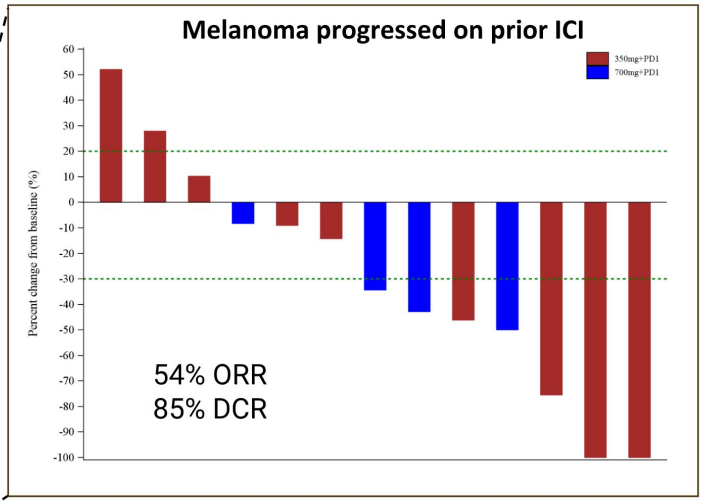
Data Cut Date: 31Oct25

Evalstotug 1L and 2L+ Melanoma who received prior ICI treatment

Evalstotug In Combination with PD1 in Melanoma (received prior ICI treatment; n=13): Demographics and Waterfall

Evalstotug 350 mg or 700 mg Q3W (5 mg/kg to 10 mg/kg based on 70 kg patient)

	Total (N=16)
Age, y, mean (SD)	61 (14)
Sex, n (%)	
Female	10 (62)
Male	6 (38)
White race, n (%)	16 (100)
ECOG, n (%)	
0	12 (75)
1	4 (25)
Prior ICI Therapy Status, n (%)	
No prior ICI treatment	**3 (19)**
Adjuvant	**10 (62)**
Metastatic setting	**3 (19)**

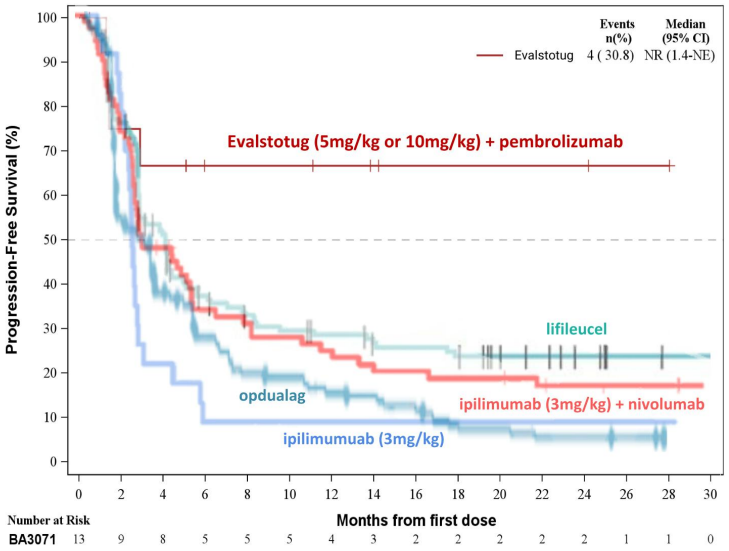


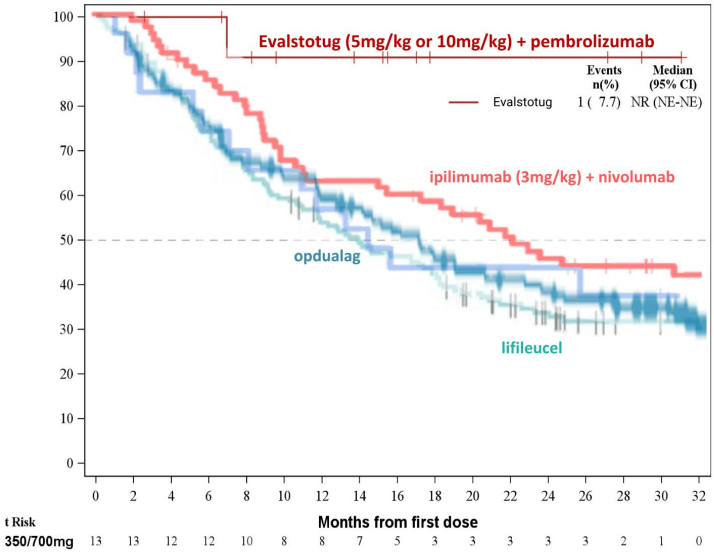
ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

54% ORR and 85% DCR in 1L and 2L+ Melanoma who received Prior ICI

All 13 patients received prior ICI; evalstotug at higher doses achieves greater efficacy





Data Cut Date: 31Oct25

Safety

Evalstotug vs Ipilimumab: Cross Trial Comparison

Lower imAE rate vs Ipilimumab despite more patients previously treated with ICI

Treatment	Evalstotug (5 mg/kg) + PD1 Q3W N=17	Evalstotug (5 mg/kg or 10 mg/kg) + PD1 Q3W N=25	Ipilimumab (3 mg/kg) + nivolumab Q3W N=178-314[1,2,3]
Doses	**1 – ≤18 weeks exposure** (≤6 doses)		**1 – ≤12 weeks exposure** (≤4 doses)
Tumor Types	**Multiple tumor types**		**Melanoma**
% Patients w/ Prior Tx	**90%**		**15%[4]**
imAE (G3-4)	**12%** (no G4)	**16%** (no G4)	**40%**

[1]Wolchok, J; Five-Year Survival with Cmbined Nivolumab and Ipilimumab in Advanced Melanoma; N Engl J Med 2019;381:1535-1546; [2]Lebbe, C; Evaluation of Two Dosing Regimens for Nivolumab in Combination With Ipilimumab in Patients With Advanced Melanoma: Results From the Phase IIIb/IV CheckMate 511 Trial; J Clin Oncol. 2019 Feb 27;37(11):867–875; [3]Larkin, J. Combined Nivolumab and Ipilimumab or Monotherapy in Untreated Melanoma; N Engl J Med 2015;373:23-34; [4]Allouchery, M; Safety of immune checkpoint inhibitor rechallenge after discontinuation for grade ≥2 immune-related adverse events in patients with cancer; J Immunother Cancer. 2020 Dec;8(2):e001622.

Data Cut Date: 31Oct25

Evalstotug in Combination with PD1: Overall Safety Summary

Generally well-tolerated; All G3 and G4 resolved

Related AE Summary	Total (N=17)	Total (N=8)	Total (N=25)
Any Related Adverse Events (AEs)	16 (94%)	8 (100%)	24 (96%)
Related AEs with CTCAE[1]			
Grade 3[2]	6 (36%)	4 (50%)	12 (48%)
Grade 4[2] transient hypercalcemia	1 (6%)	1 (13%)	8 (32%)
imAE Grade 3 or 4	5 (29%)	2 (25%)	7 (28%)
Any related serious AEs[2]	5 (29%)	5 (63%)	10 (40%)
Possibly Related AEs leading to death[2]	0	0	0
Related AEs leading to treatment discontinuation[2]	5 (29%)	3 (38%)	7 (28%)

[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.

Evalstotug in Combination with PD1 Safety Data

Treatment-emergent related adverse events >15%

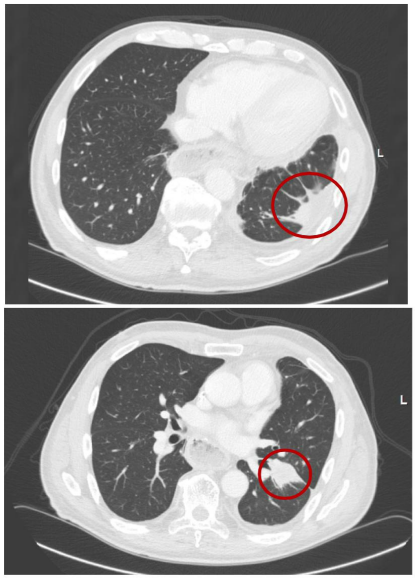
	Evalstotug 350 mg + PD1 (N=17)		Evalstotug 700 mg + PD1 (N=8)		Total (N=25)	
AE	All	Gr3+	All	Gr3+	All	Gr3+
Chills	11 (64.7)	0	4 (50.0)	0	15 (60.0)	0
Fatigue	11 (64.7)	0	3 (37.5)	1 (12.5)	14 (56.0)	1 (4.0)
Nausea	8 (47.1)	0	4 (50.0)	0	12 (48.0)	0
Cytokine release syndrome	6 (35.3)	0	5 (62.5)	0	11 (44.0)	0
Vomiting	9 (52.9)	0	2 (25.0)	0	11 (44.0)	0
Arthralgia	9 (52.9)	0	1 (12.5)	1 (12.5)	10 (40.0)	1 (4.0)
Diarrhoea	6 (35.3)	1 (5.9)	3 (37.5)	0	9 (36.0)	1 (4.0)
Abdominal pain	6 (35.3)	1 (5.9)	2 (25.0)	0	8 (32.0)	1 (4.0)
Pruritus	4 (23.5)	0	4 (50.0)	0	8 (32.0)	0
Rash	6 (35.3)	0	2 (25.0)	0	8 (32.0)	0
Anaemia	5 (29.4)	3 (17.6)	2 (25.0)	0	7 (28.0)	3 (12.0)
Headache	6 (35.3)	0	1 (12.5)	0	7 (28.0)	0
Oedema peripheral	3 (17.6)	0	3 (37.5)	0	6 (24.0)	0
Hypokalaemia	4 (23.5)	1 (5.9)	1 (12.5)	0	5 (20.0)	1 (4.0)
Decreased appetite	4 (23.5)	0	1 (12.5)	0	5 (20.0)	0
Infusion related reaction	2 (11.8)	0	3 (37.5)	0	5 (20.0)	0
Lipase increased	3 (17.6)	2 (11.8)	1 (12.5)	0	4 (16.0)	2 (8.0)
Pneumonia	1 (5.9)	0	3 (37.5)	2 (25.0)	4 (16.0)	2 (8.0)
Dehydration	3 (17.6)	1 (5.9)	1 (12.5)	0	4 (16.0)	1 (4.0)
Back pain	3 (17.6)	0	1 (12.5)	0	4 (16.0)	0
Cough	4 (23.5)	0	0	0	4 (16.0)	0
Dyspnoea	3 (17.6)	0	1 (12.5)	0	4 (16.0)	0
Hypomagnesaemia	3 (17.6)	0	1 (12.5)	0	4 (16.0)	0
Influenza like illness	4 (23.5)	0	0	0	4 (16.0)	0
Pyrexia	3 (17.6)	0	1 (12.5)	0	4 (16.0)	0

Data Cut Date: 31Oct25

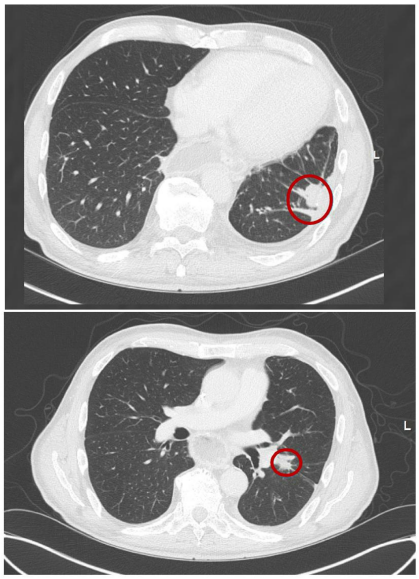
Confirmed PR - Gastro-esophageal Cancer

63-year-old male, stage IV gastro-esophageal cancer HER2 negative, post-FOLFOX, taxane, TKI, anti-PD1 and anti-VEGFI

Baseline - July 31,2023



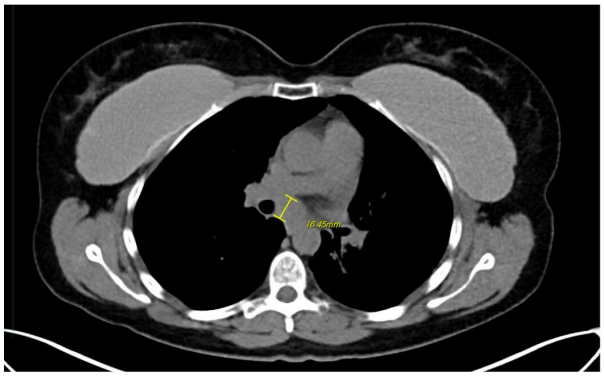
On Treatment - October 23, 2023



Confirmed CR - Cervical Cancer

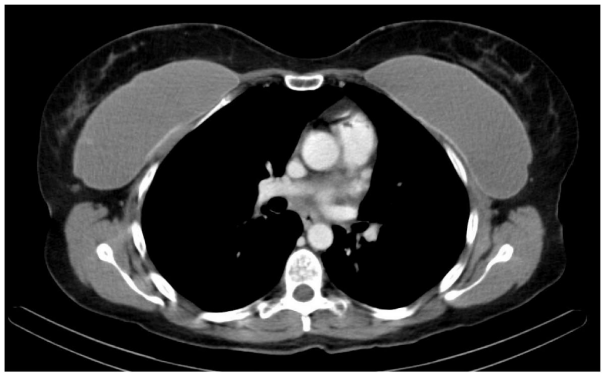
43-year-old female, stage IV cervical cancer HPV+16 positive, post-platinum, taxane, anti-PD1 and anti-VEGF

Baseline – March 23, 2023



"Multiple enlarged mediastinal, paraesophageal, and right hilar lymph nodes…"

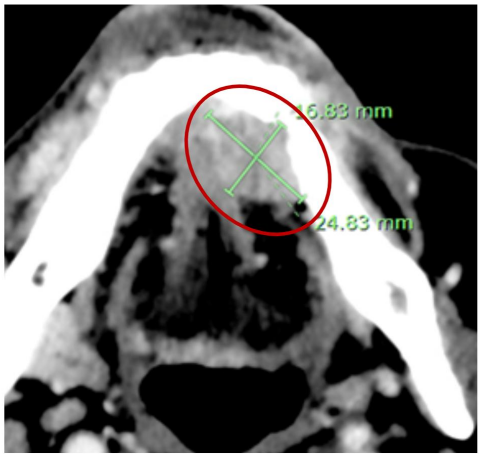
On Treatment – August 9, 2023

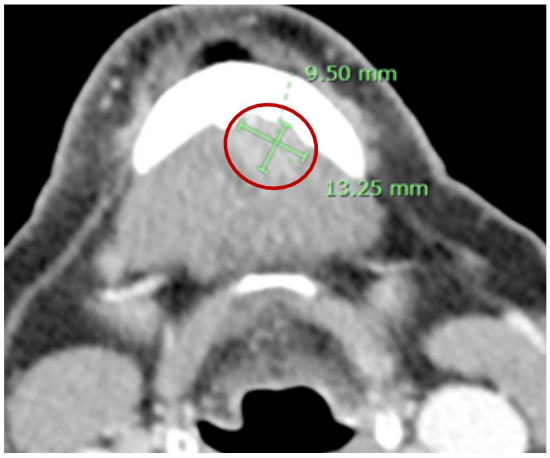


"No enlarged mediastinal, hilar or axillary lymph nodes are present. There is persistent resolution of previously noted enlarged mediastinal and paraesophageal lymph nodes."

Evalstotug Enables the Combination with ADC and PD1 Antibodies
PR (-50%) – Well Tolerated Triplet; Oz-V + Evalstotug + PD1

69 yo M with oral cavity, floor of mouth/ mandibular, mucoepidermoid carcinoma; neo-adjuvant patient





Disease had recurred after prior surgery and chemoradiation (platinum / taxane / pembrolizumab as well as cetuximab). Prior to Oz-V triplet patient had tumor filling the maxillary sinus measuring 5.3cm in longest diameter. After triplet, the tumor nearly resolved and is difficult to measure. A transient, clinically asymptomatic elevation of hepatic transaminases was documented that didn't recur with subsequent dosing.

FDA Guidance Regarding Evalstotug Pivotal Trial in 1L Unresectable and / or Metastatic Melanoma

- Centrally reviewed PFS acceptable as primary endpoint
- General agreement with proposed study population and sample size
- Additional guidance received on ongoing dose optimization and control arm:
  - IO-based combination regimen should be included in the control arm
  - Project Optimus should guide determination of Phase 3 evalstotug dose

Overall Summary: Evalstotug + PD1

Greater efficacy and lower imAE rate vs ipilimumab + PD1

- Preclinical & clinical data demonstrate that
  - ipi & evalstotug are similar, *i.e.* epitope, affinity, $T_{1/2}$ and tumor exposure, and efficacy
  - ipi & evalstotug are NOT similar with respect to normal tissue environment and safety; *e.g.*, reduced imAEs and extended treatment
- 54% ORR and 85% DCR in 1L and 2L+ melanoma patients who received prior ICI treatment
- Higher dosing yields encouraging efficacy with low incidence and severity of imAEs, consistent with CAB-driven tumor selectivity for potential best-in-class CTLA4
- Significant opportunity for an effective and better tolerated CTLA-4 in combination regimens

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