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

Corporate Presentation

August 2025

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 and 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, 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; 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.

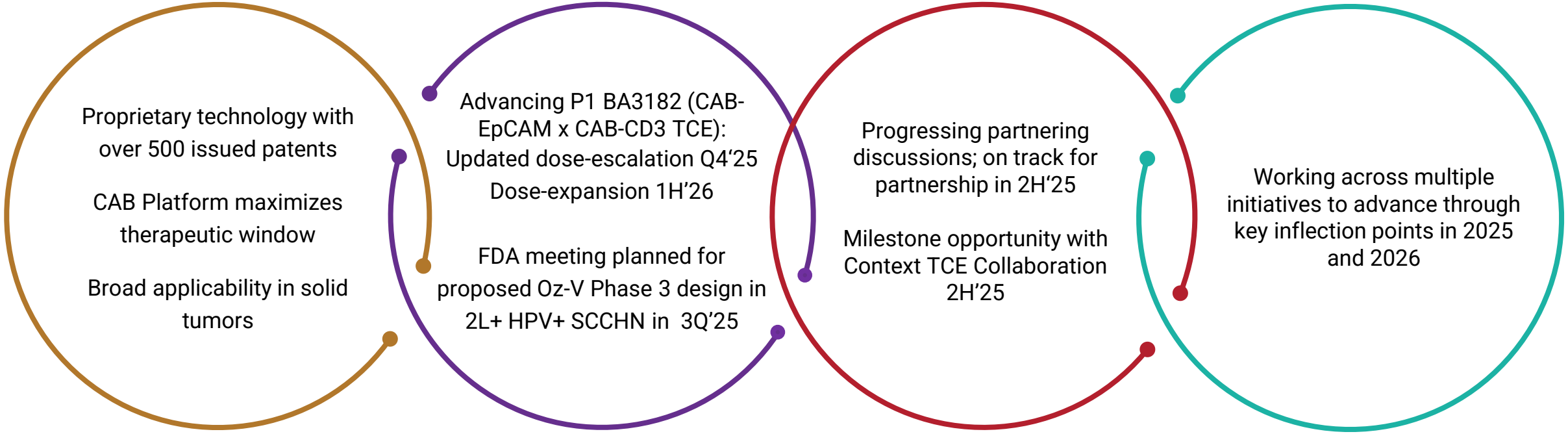
Certain information contained in this Presentation relates to or is based on statistical and other industry and market data obtained from independent industry publications and research, surveys and studies conducted by independent third parties as well as the Company's own estimates of the prevalence 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.

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

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



TCE – T-cell engager

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

Selective and Targeted CAB Platform Technology Widens Therapeutic Window

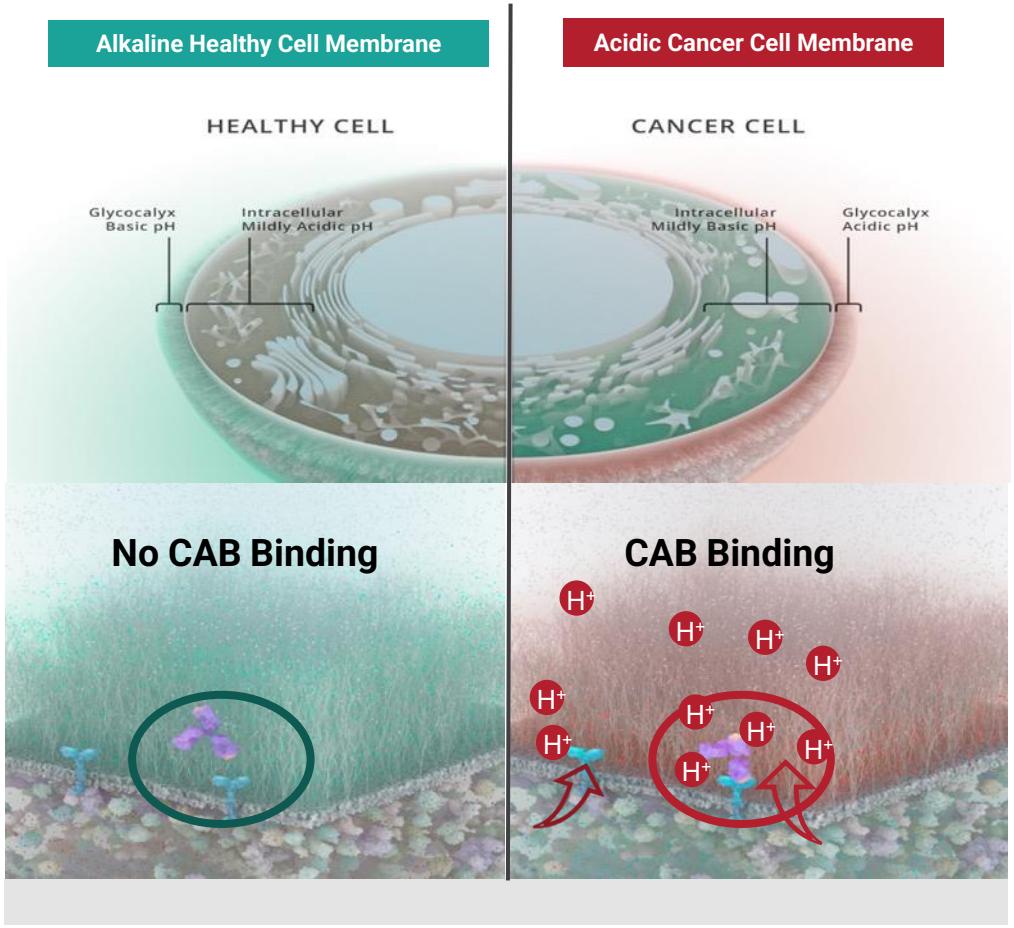
Thus has the potential to enhance clinical outcomes in multiple tumor types

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

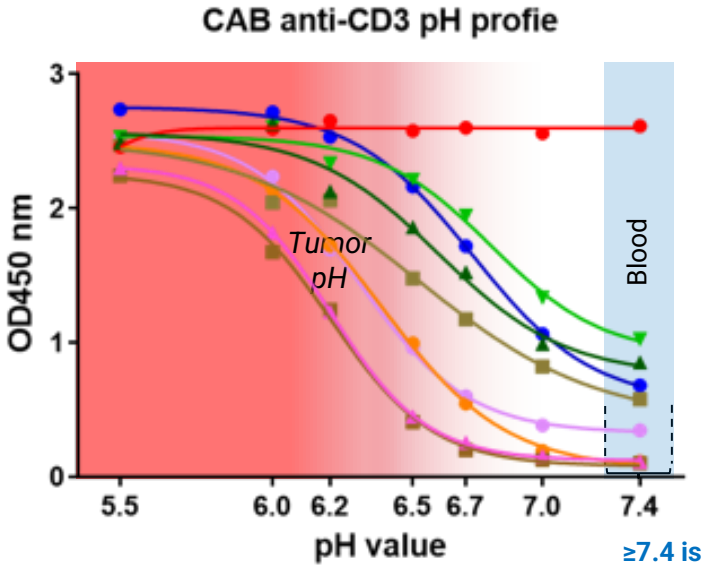
BioAtla's Solution: Conditionally Active Biologics (CAB)

Myth	Fact
Not all tumors are acidic	ALL tumors and cancer cells are acidic.
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 pH fluctuates, CABs are designed to bind any cancer cell at or below a predetermined pH.
Tumor size influence acidic environments 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)

- CAB technology generates a library of pH selective molecules from the original template
- Enhancing exposure and reducing toxicity

CABs Bind Selectively in the Lower pH TME



≥7.4 is pH of normal cell

Non-CAB Parent

Non-CABs Bind to Normal Tissues, Causing Toxicity

CABs

CABs Reduce or Eliminate, as needed, Binding to Normal Tissues, Enhancing Safety and Efficacy

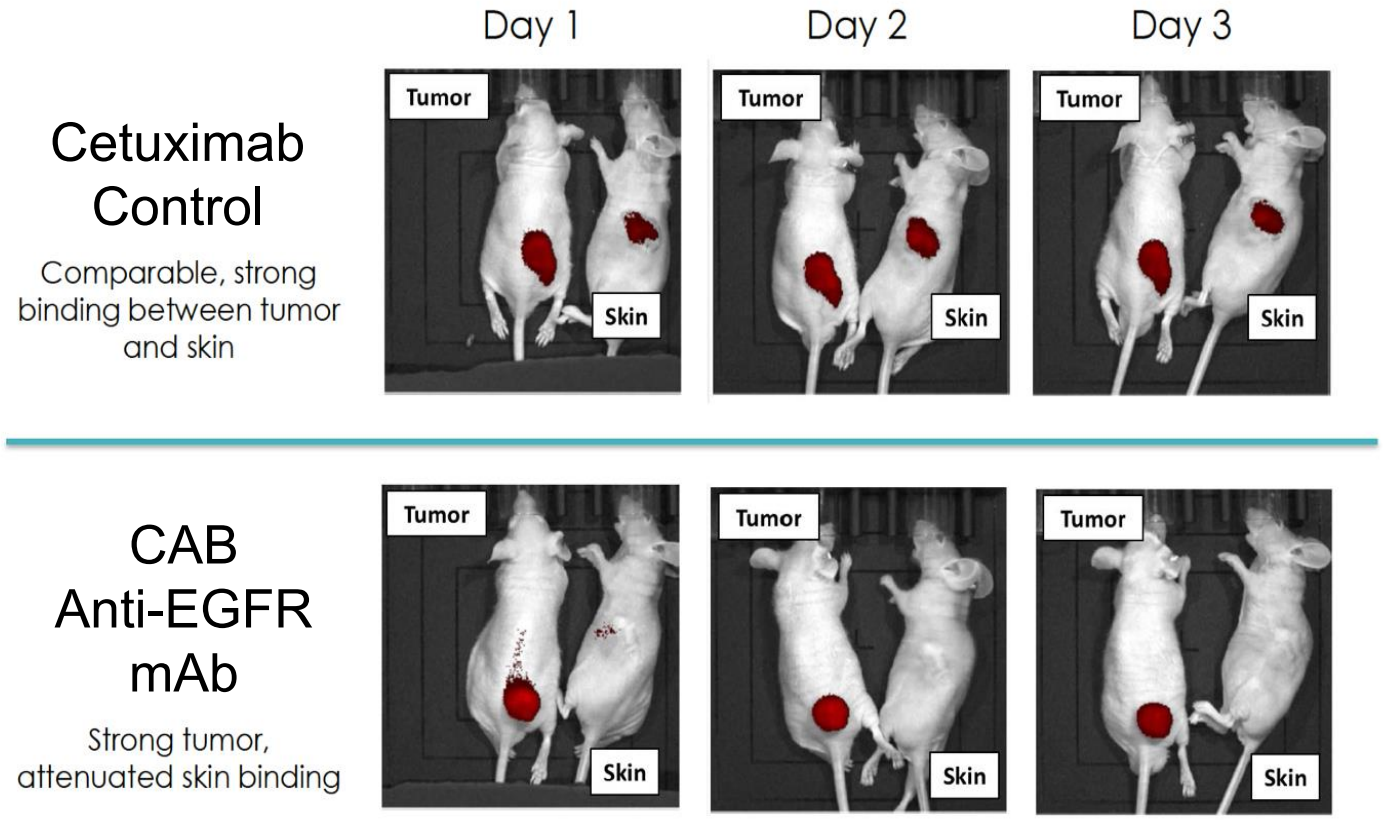
Selective Binding → Focused Tumor Killing → Normal Cells Preserved

Note: OD450nm = optical density measurements using a microplate reader with a 450nm filter; TME = Tumor MicroEnvironment; 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 ADC 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-fold in 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 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

Clinical Evidence Summary

- Clinical evidence of CAB selectivity
  - AXL-ADC promising risk/benefit ratio
    - Two other companies' non-CAB AXL-targeting ADCs terminated in P1 due to toxicity
    - Potent and durable response with differentiated OS in mKRAS NSCLC patients
  - ROR2-ADC promising risk/benefit ratio
    - Good tolerability
    - Potent and durable response in SCCHN patients, including in refractory HPV+ patients
  - EpCAM Dual CAB TCE
    - One other company with 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-mediated 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; no grade 4 or 5 AEs, even at higher doses

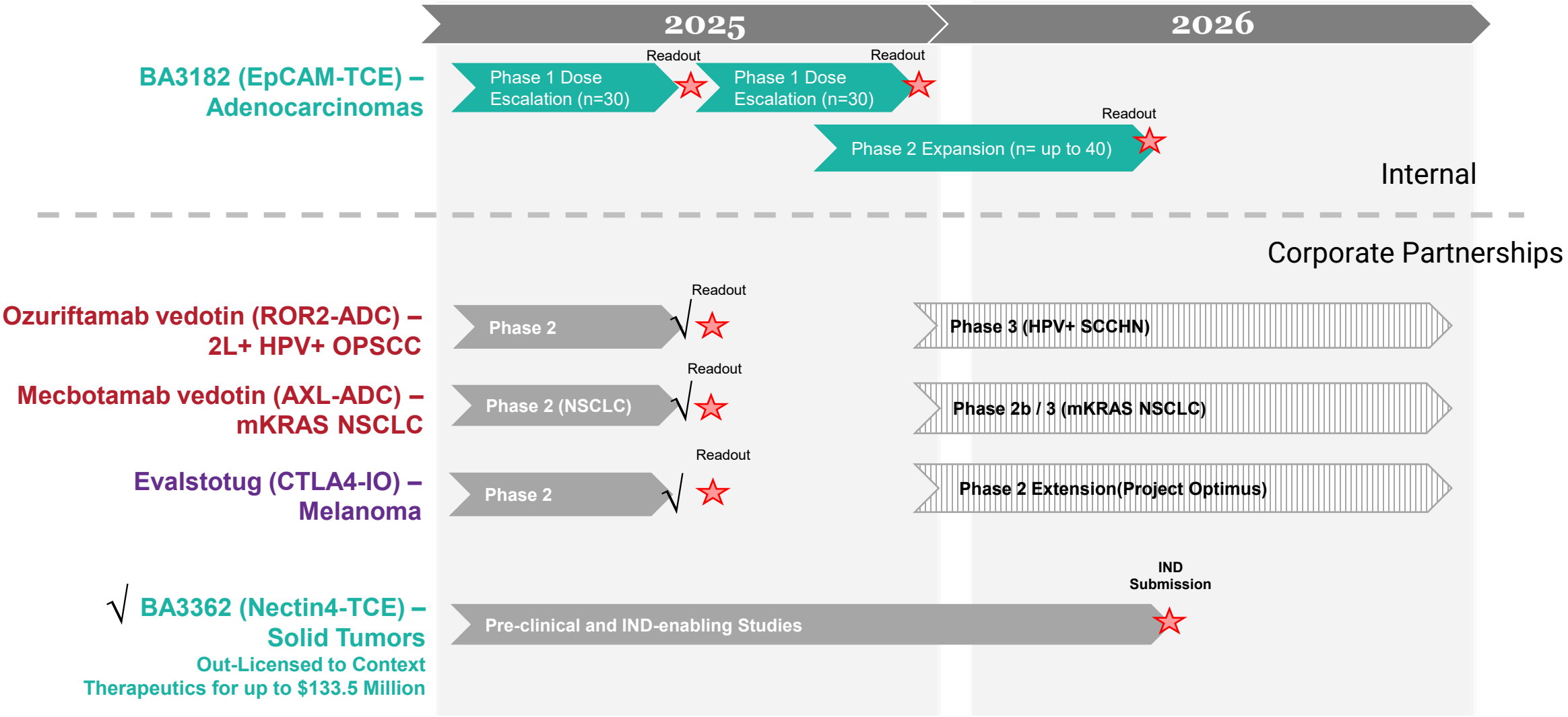
CABs demonstrate universal improvement in TI and enable therapeutic development for "undruggable" targets

Key Advantages of the CAB Platform

Widening The Therapeutic Index

- ✓ **Conditional and reversible binding increases efficacy and improves safety**
- ✓ **Not dependent on enzymatic activation**
- ✓ **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**

BioAtla's Catalysts Expected to be Achieved through Internal and Corporate Partnership Advancement



√ = Phase completed or asset partnered

Advancement anticipated through corporate partnership

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

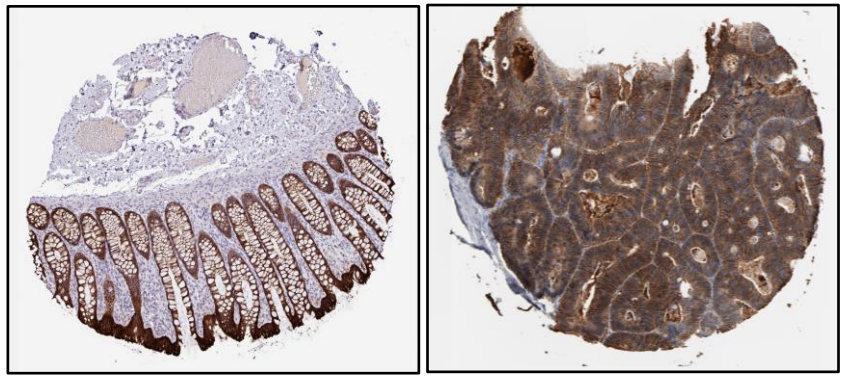
	Estimated Number of New Cancer Cases in 2025	EpCAM Expression (TIS 1 to 12)[2]
Breast Cancer	319,750	81%
Prostate Cancer	313,780	99%
Lung Cancer	226,650	93% NSCLC 80% SCLC
Colon Cancer	154,270	100%
Pancreatic Cancer	67,440	99%
Thyroid Cancer	44,020	97%
Ovarian	20,890	92%
Gallbladder & other biliary	12,610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

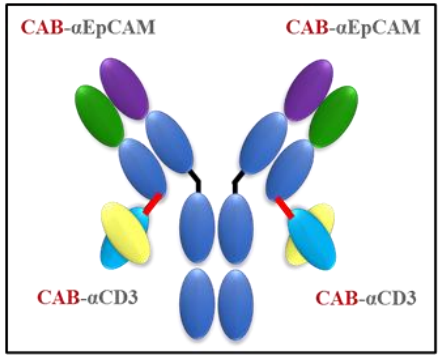


Normal Colon **Colon Cancer**

EPCAM IHC

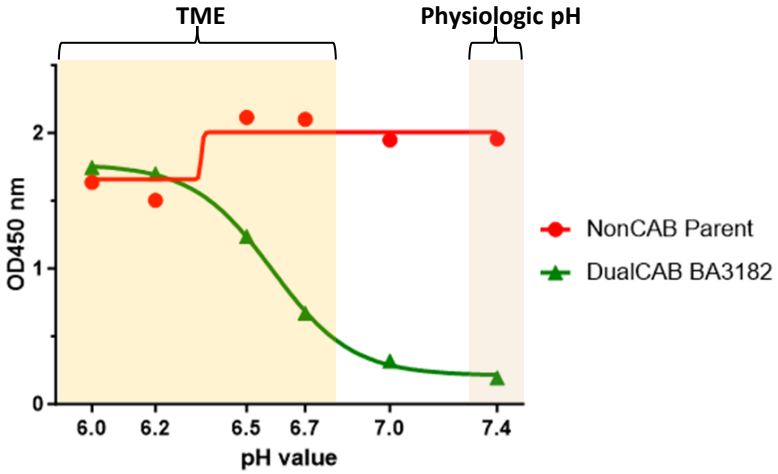
BA3182 – First DualCAB T-cell engager targeting EpCAM

Potent Lysis of EpCAM Positive Cancer Cells by BA3182



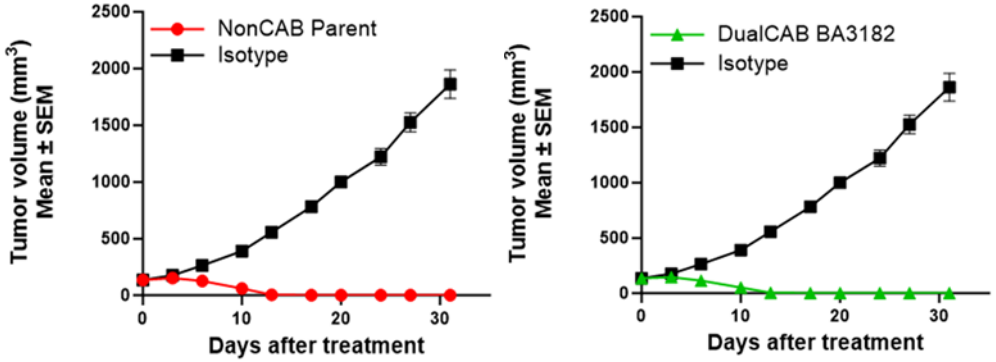
Tetravalent T-cell engager EpCAM

pH Range ELISA



Highly reduced binding to both targets at normal physiological pH

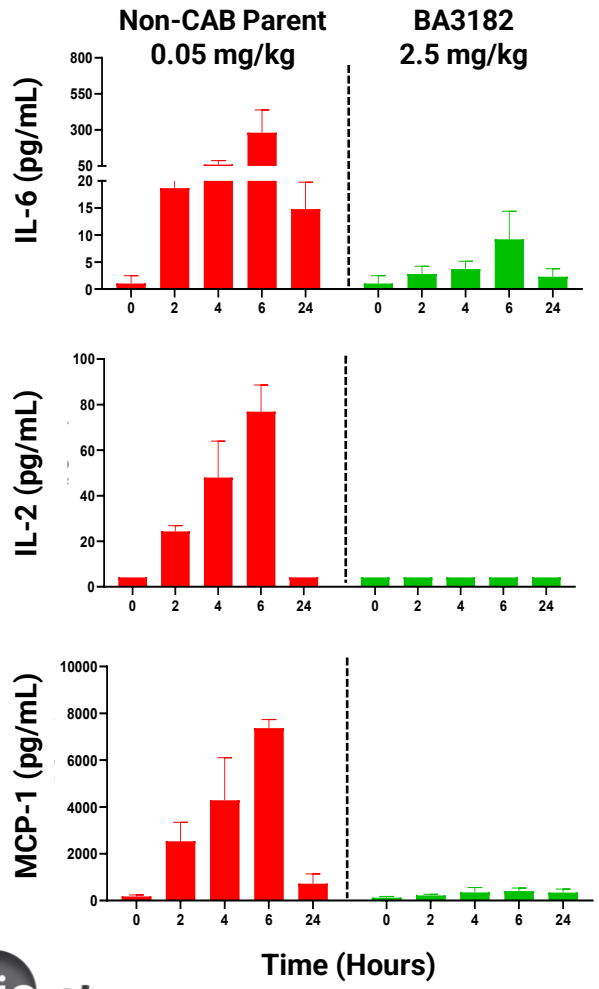
HCT116 CDX Model



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

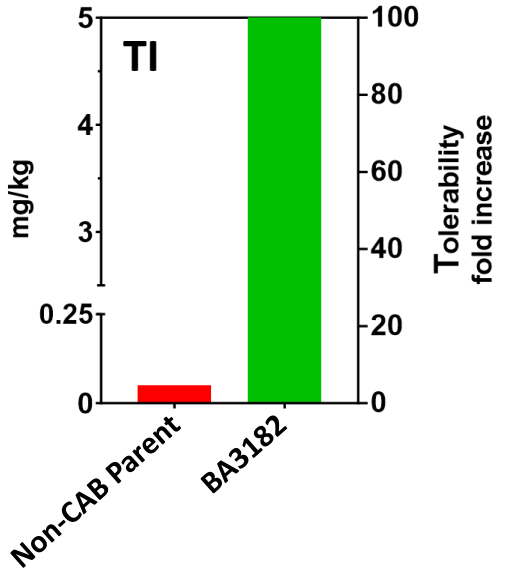
BA3182 is Well Tolerated at High Doses in Non-human Primates

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

BA3182 Phase 1 Dose Escalation: Design and Current Status

Now focusing enrollment among colorectal carcinoma; dose escalation actively continues

Primary objectives:

Characterize safety, tolerability, and define recommended phase 2 dose

Secondary objectives:

Characterize antitumor activity, pharmacokinetics, and immunogenicity

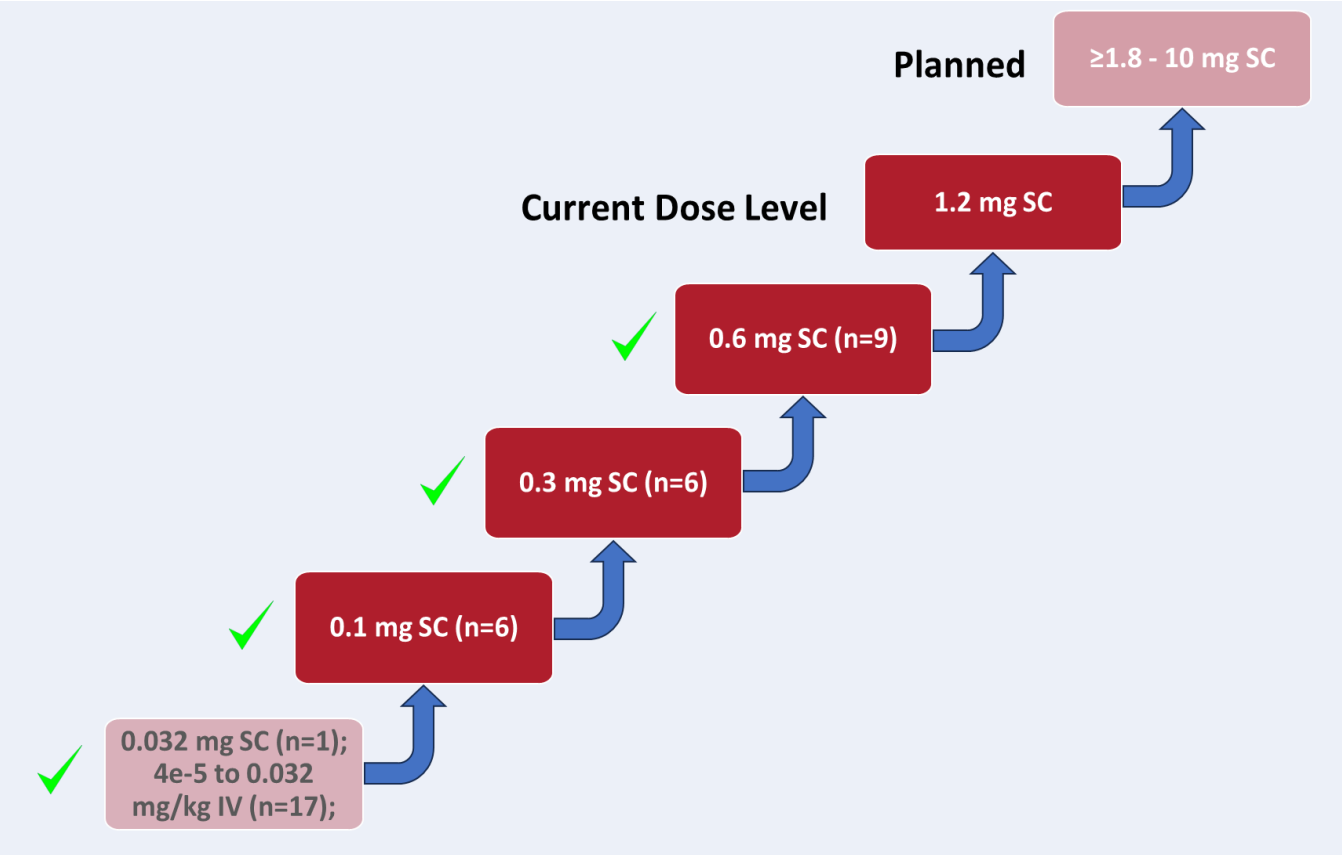
Disposition:

39 pts treated per protocol as of 20 June 2025 data cutoff:

Intravenous (IV), weight-based dosing (N=17)*

Subcutaneous (SC), flat dosing (N=22)

*Poster presented at: European Society for Medical Oncology Gastrointestinal Cancers Congress (ESMO GI); July 2–5, 2025; Barcelona, Spain.



Treatment notes:
0, 1, or 2 smaller priming doses (0.00012 mg/kg, 0.0001 mg, 0.003 mg, 0.1 mg, or 0.3 mg) delivered 4-7 days prior to weekly larger treatment doses
Ongoing weekly treatment dosing continues after DLT observation interval concludes

BA3182 Phase 1 Patient Demographics

Patients dosed SC per protocol as of June 20, 2025: N=22

Patient Characteristic	N=22
Age, mean (SD), y	58 (10)
Male	9 (50)
Female	9 (50)
ECOG performance	
0	13 (59)
1	9 (41)
Number of prior lines of therapy, median	3

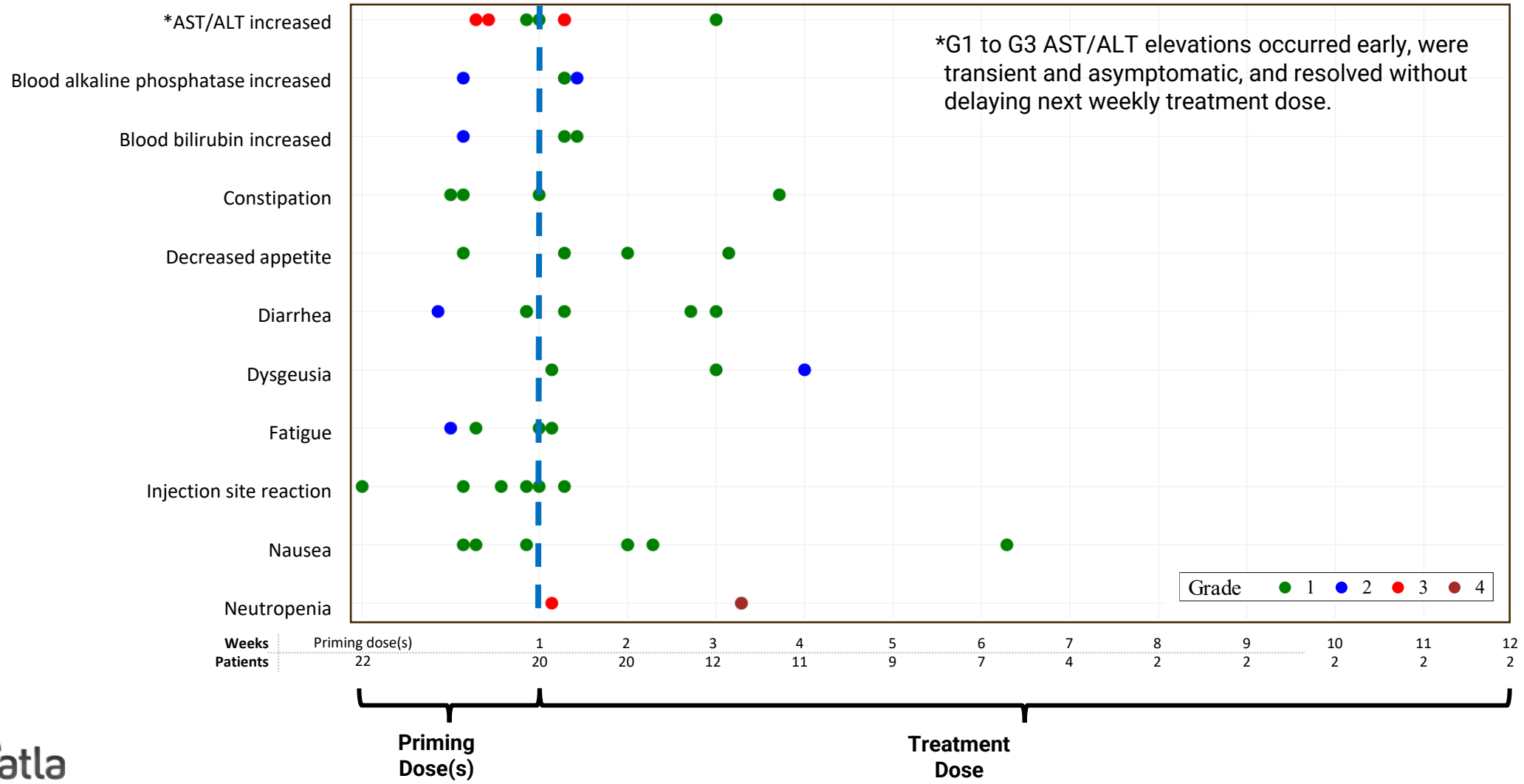
Tumor	N=22
Adenoid Cystic Carcinoma	1 (5)
Cholangiocarcinoma	1 (5)
Colon/Rectum	11 (50)
Gallbladder	1 (5)
Ovarian	1 (5)
Pancreas	6 (27)

Presented at ESMO TAT, 2025.

Data cut as of 13Jun25

BA3182 Demonstrating an Encouraging Safety Profile To Date

Temporal occurrence of ALL (related and unrelated) treatment emergent AEs occurring in >10% among patients who received SC dosing (n=22)



Presented at ESMO TAT, 2025.

Data cut as of 20Jun25

BA3182 Adverse Events, Including CRS, Generally Low-Grade, Transient and Readily Manageable

Safety of subcutaneous dosing (N=22)

Characteristic	N=22
Any Adverse Events (AEs) (n, %):	20 (91)
Related AEs of CTCAE[1] Grade 3 (n, %)	6 (32)
Related AEs of CTCAE[1] Grade 4[2] (n, %)	1 (5)[3]
Any related serious AEs[2] (n, %)	3 (14)
Related AEs leading to death[2] (n, %)	0
Related AEs leading to treatment discontinuation[2] (n, %)	0

[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] Possibly related to tocilizumab

Characteristic	Any	G3+
ALT increase (n, %)^	8 (36)	3 (14)
AST increase (n, %)^	7 (32)	3 (14)
Nausea (n, %)	7 (32)	0
Diarrhea (n, %)	6 (27)	0
Injection site reaction (n, %)	6 (27)	0
Constipation (n, %)	4 (18)	0
Decreased appetite (n, %)	4 (18)	0
Fatigue (n, %)	4 (18)	0
Alk Phos increased (n, %)	4 (18)	0
Non-febrile neutropenia (n, %)	3 (14)	2 (9)
Bilirubin increased (n, %)	3 (14)	0
Dysgeusia (n, %)	3 (14)	0

^Early, transient

Presented at ESMO TAT, 2025.

Data cut as of 13Jun25

BA3182 Achieved Objective Tumor Size Reductions Across Multiple Tumor Types

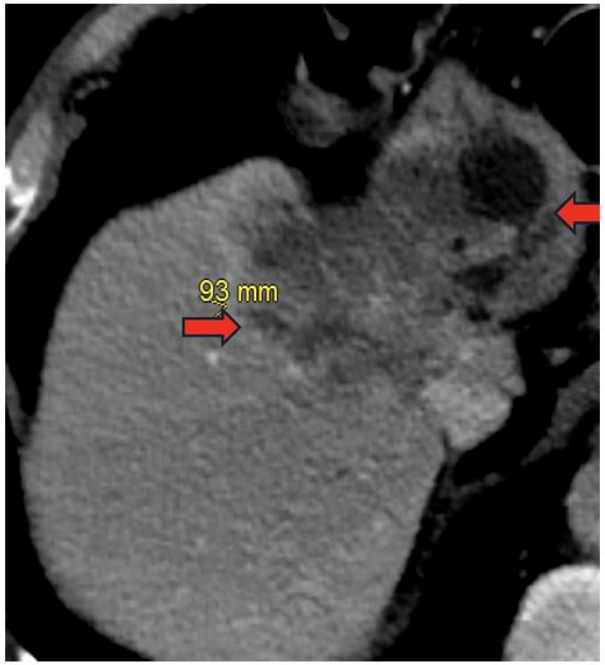
Preliminary assessment of anti-tumor activity among those with available scans

- Seven patients achieved objective tumor size reductions:
  - Pancreas (-5%)
  - CRC (-6%, -8%, -10%)
  - Breast (-11%)
  - Cholangiocarcinoma (-13%)
  - NSCLC (-25%)
- All five evaluable patients in the 0.6 mg cohort achieved stable disease on first scan and continue on therapy
- Prolonged progression-free intervals observed in 2 CRC pts: 11 mo and 16 mo.

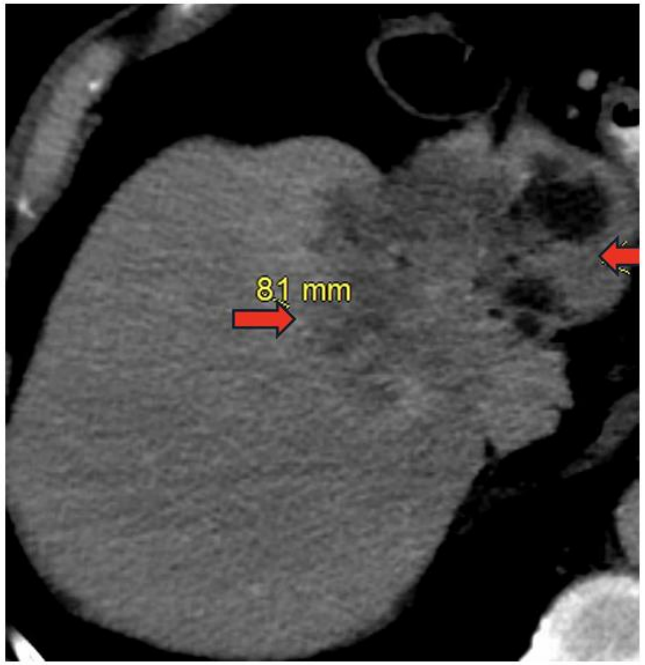
13% Tumor Reduction in Intrahepatic Cholangiocarcinoma From BA3182 at 0.1 mg SC QW Without Progression for 21 weeks, Ongoing

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

Baseline – Week 0



Week 12



Since being on study, patient has experienced measured tumor reduction and symptom improvement with resolution of pain and resumption of activities of daily living. Patient dose-escalated to 0.3 mg for 15th weekly treatment dose.

Presented at European Society for Medical Oncology Gastrointestinal Cancers Congress 2025 (ESMO GI) and ESMO TAT, 2025.

BA3182 Conclusions / Milestones

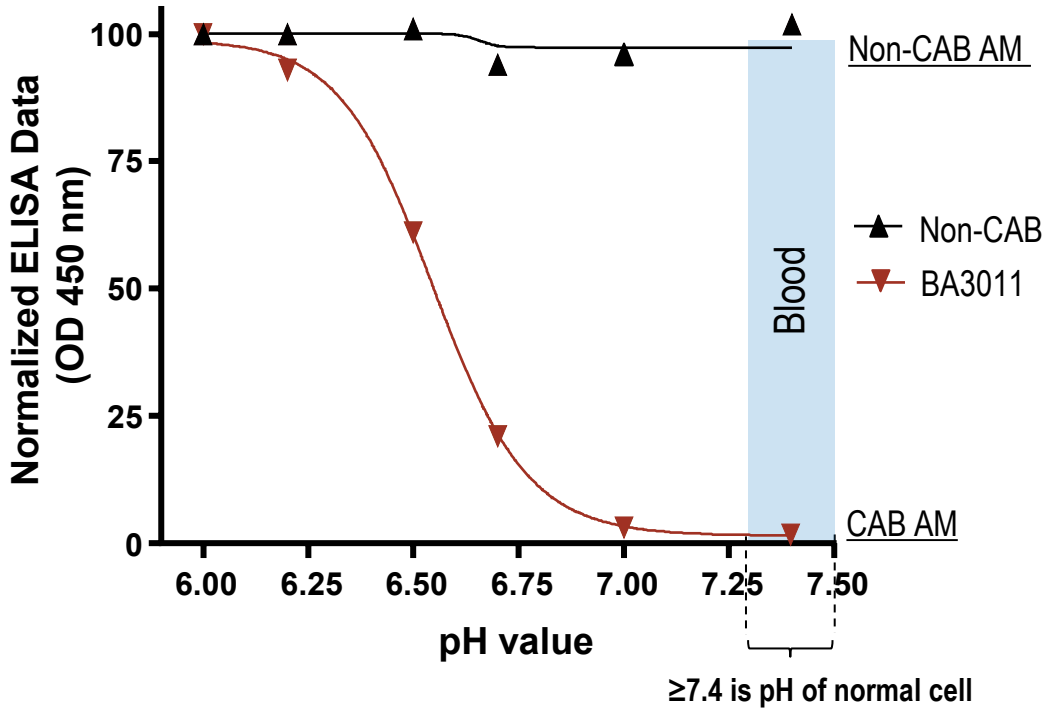
- BA3182 conditionally binds EpCAM and CD3 at low pH, thus restricting activity to the TME while avoiding damage to normal EpCAM-expressing tissues
- Related AEs were generally low-grade, transient, and readily manageable
- Evidence of prolonged tumor control and reductions achieved among heavily pretreated pts
- Dose escalation read out anticipated 2H’25
- Phase 2 dose expansion (up to 40 pts) anticipated 1H’26

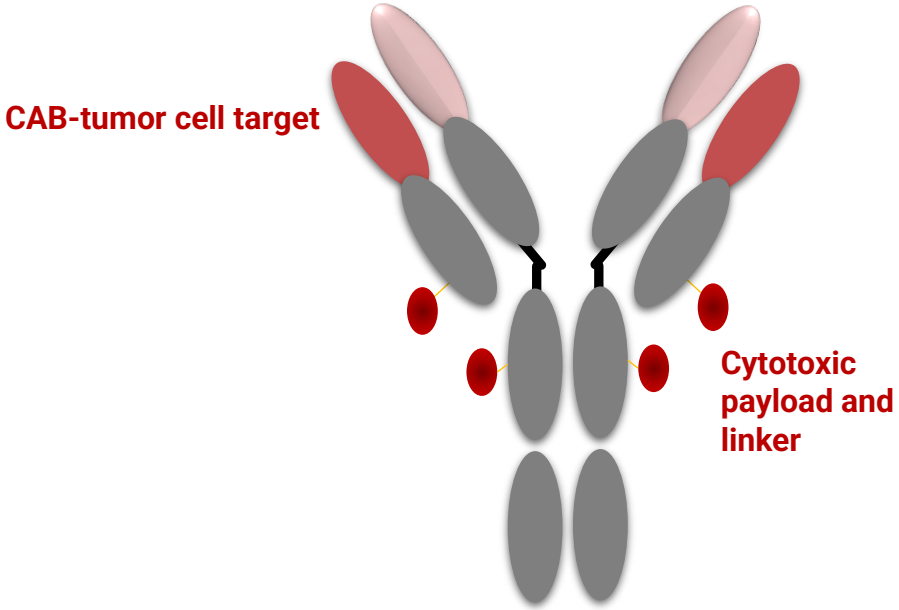
Mecbotamab Vedotin (CAB-AXL-ADC): mKRAS Non-Small Cell Lung Cancer (NSCLC)

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





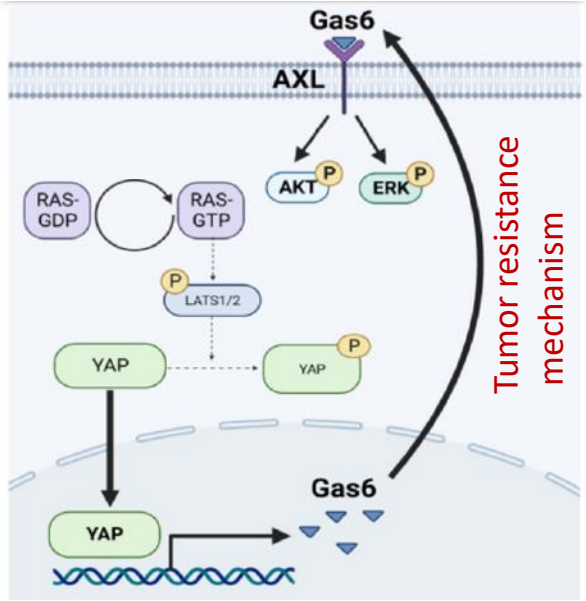
- Humanized anti-AXL IgG1
- ~100 pM affinity (pH 6.5)
- VC-MMAE (DAR 4) linker and payload
- Epitope in Ig loop region

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

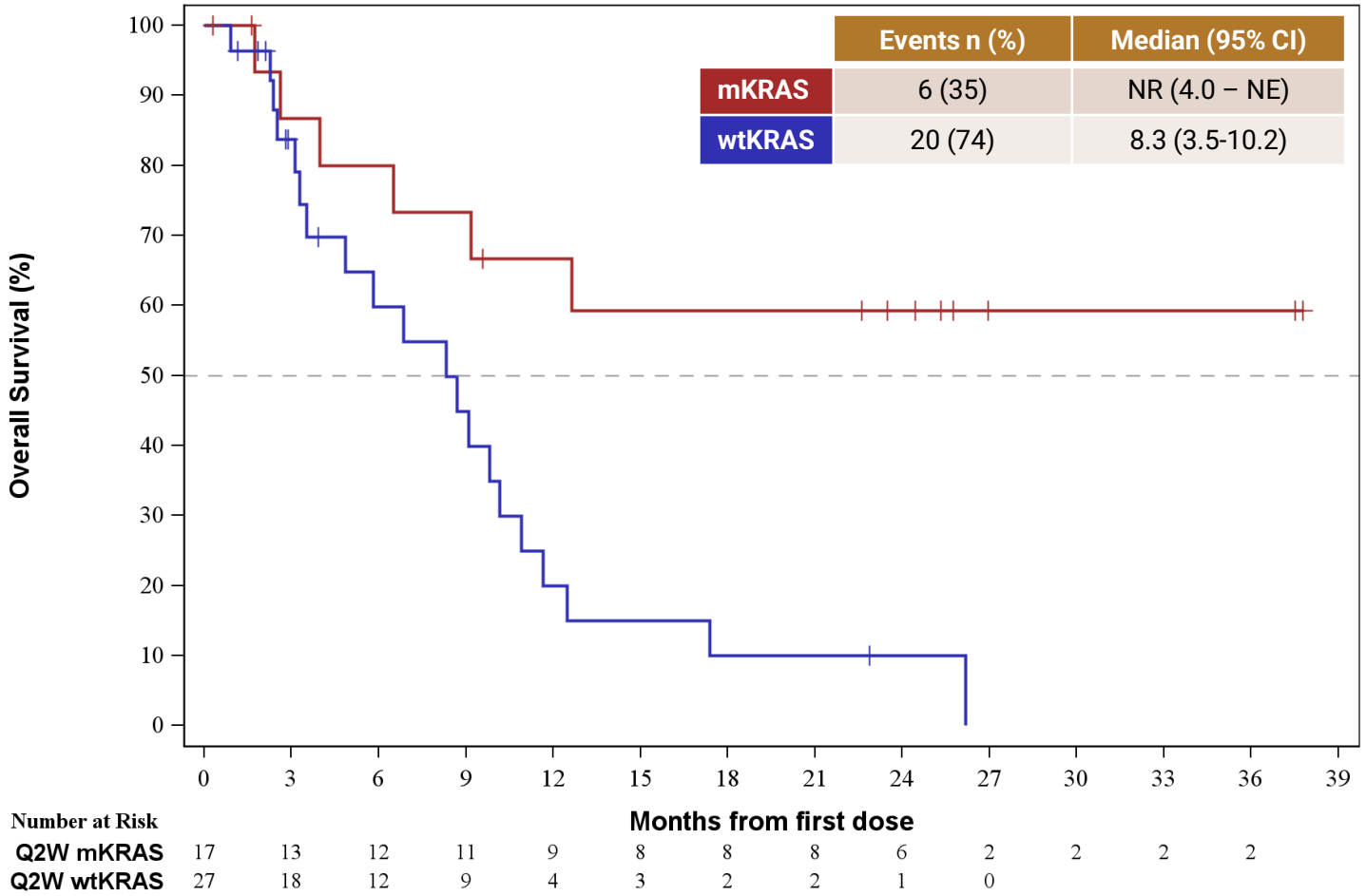


Adapted from Morimoto et al. Cancer Letters 587 (April 2024)

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

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

Median of 3 prior lines of tx



Median 3 prior lines of therapy	mKRAS (Q2W only)
Responders (confirmed & unconfirmed)	31% (5/16^)
Responders (confirmed)	25% (4/16^)
DCR	81% (13/16^)
Median DOR	5.9 months
Median PFS	4.5 months
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: Live database as of 16Apr25

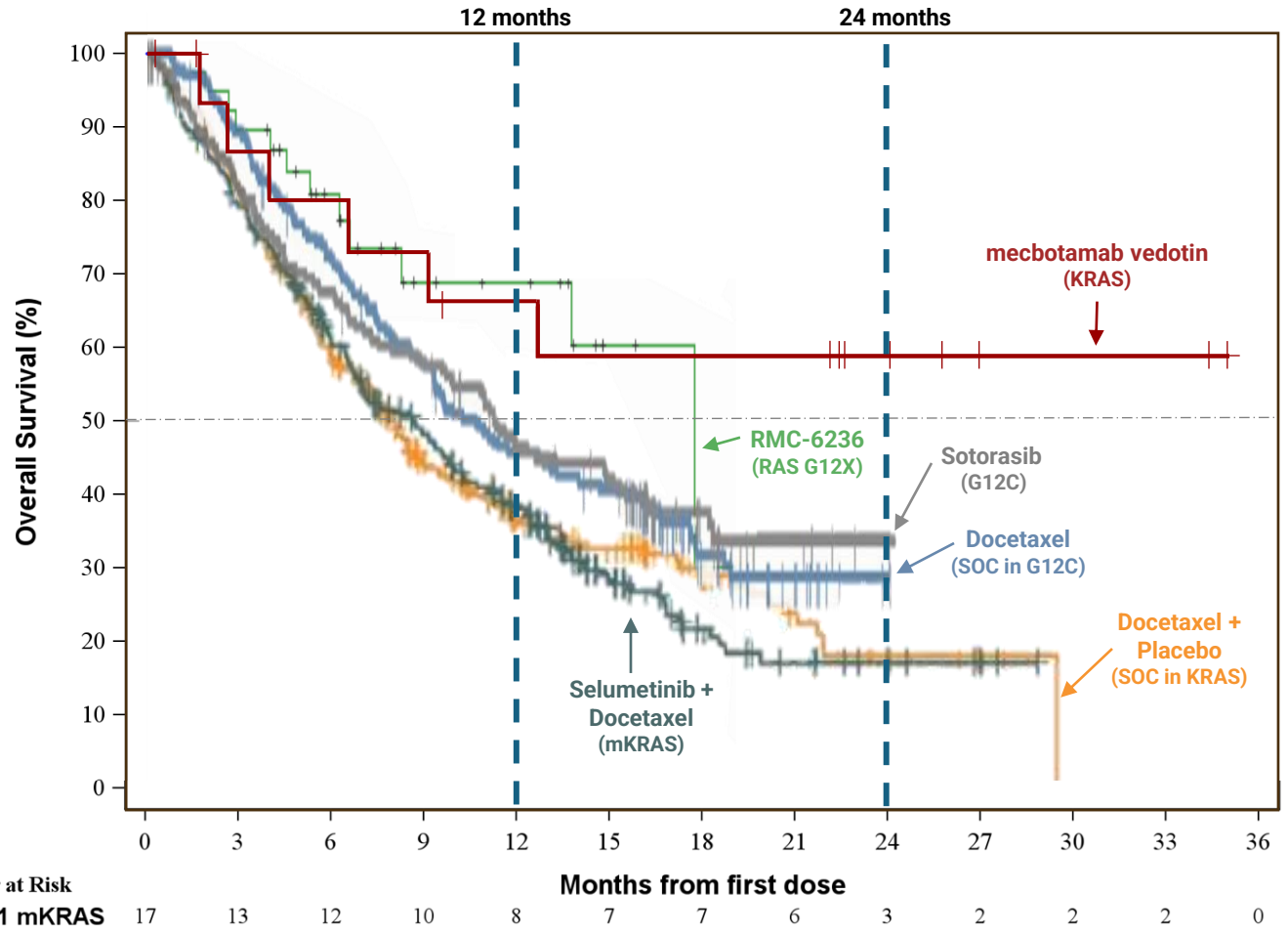
Mec-V 1.8 mg/kg Q2W Associated with Exceptional Overall Survival in mKRAS NSCLC

Overall survival cross trial comparison*

	mecbotamab vedotin (1.8 mg/kg Q2W)	RMC-6236[1]	Sotorasib[2]	Docetaxel[3]
Population	mKRAS	RAS G12X	G12C	mKRAS
Number of patients	17	73	171	256
Prior Lines of tx	**3**	**2**	**2**	**1**
Median OS	Not reached	17.7 months	10.6 months	7.9 months
Survival at 12 months	67%	69%	51%	<40%
Survival at 24 months	59%	Not Reported	33%	<20%

*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.

[1]Revolution Medicines Corporate Presentation February 2025;
[2]Lancet 2023; 401: 733–46.;
[3]JAMA. 2017 May 9;317(18):1844–1853.



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

Data Cut Date: Live database as of 16Apr25

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.

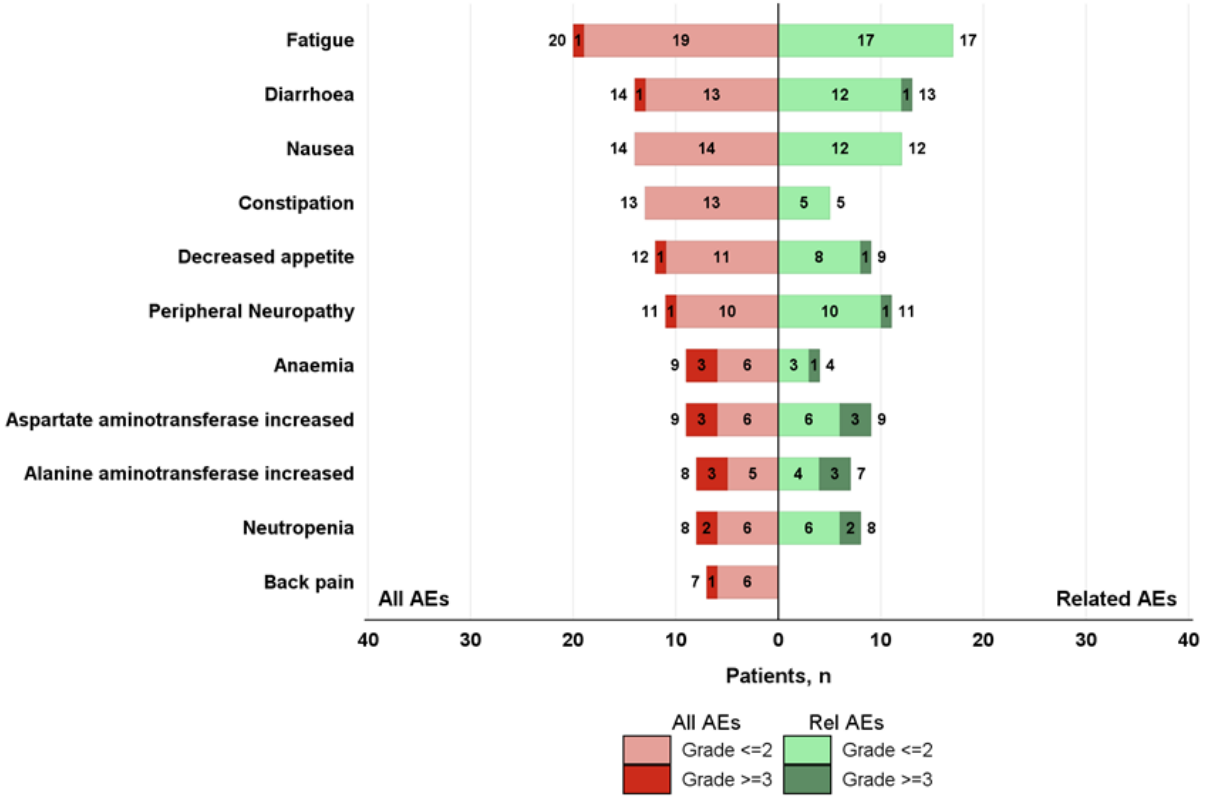
***Confirmed and Unconfirmed

Phase 2 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]	5 (11)
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: 18Jan2025

Potential for Mec-V to Address All mKRAS NSCLC

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

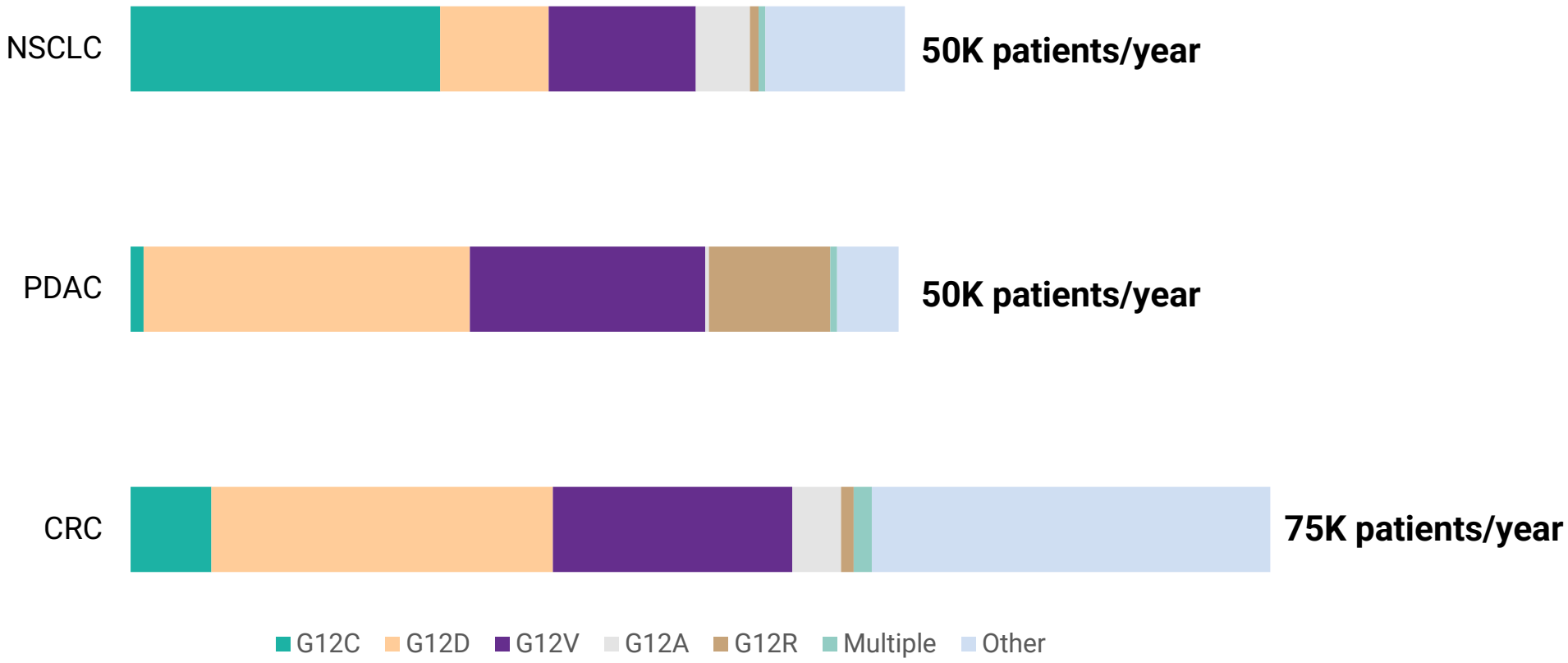
- Promising anti-tumor activity among patients whose tumors express KRAS mutations
  - mKRAS represents 30% of all NSCLC patients and is associated with increased AXL expression
  - 1.8 mg/kg Q2W associated with exceptional overall survival even in heavily pretreated patients
  - 67% alive at a landmark 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

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

Potential for Oz-V to be the First Approved Treatment Specifically for HPV-associated Oropharyngeal Squamous Cell Carcinoma (HPV+ OPSCC)

Rapidly Growing Patient Population

- In the US, OPSCC is the most frequent HPV-associated cancer[1]
- By 2030, the majority of all head and neck cancer will be HPV-associated[1]

Potential to Address Significant Unmet Need

- HPV+ OPSCC poorly served by SOC, including EGFR inhibitors[2-8]
- Oz-V has a compelling and differentiated profile in HPV+OPSCC

Large Commercial Opportunity with Potential to Expand

- 2L+ HPV+ OPSCC worldwide market valued at ~$1Bn[9]
- Total HPV+ solid tumors (ie, cervical) worldwide market valued at >$7Bn[10]

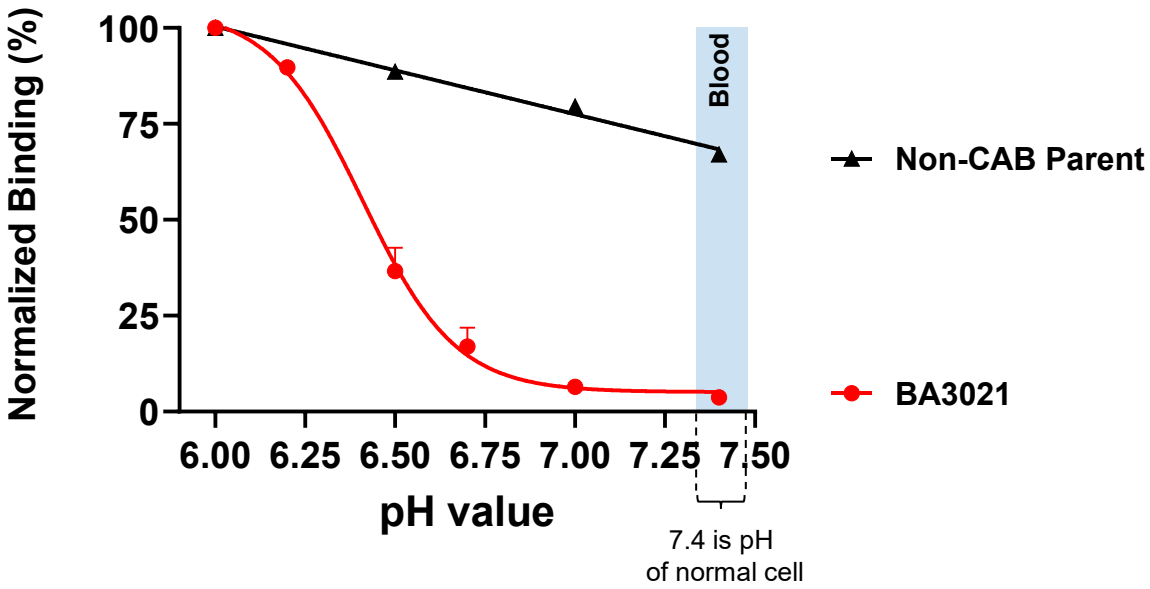
[1]Oral Oncol. 2021 Apr:115:105177.; [2]N Engl J Med 2016;375:1856-1867. [3]Journal of Clinical Oncology 2018; 36(15): 1551-1558. [4]Cohen E, et al. (2019) Lancet 393, 156–167. [5]British Journal of Cancer (2018) 119:153–159; https://doi.org/10.1038/s41416-018-0131-9. [6]2008 Jun 15;112(12):2710-9. doi: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

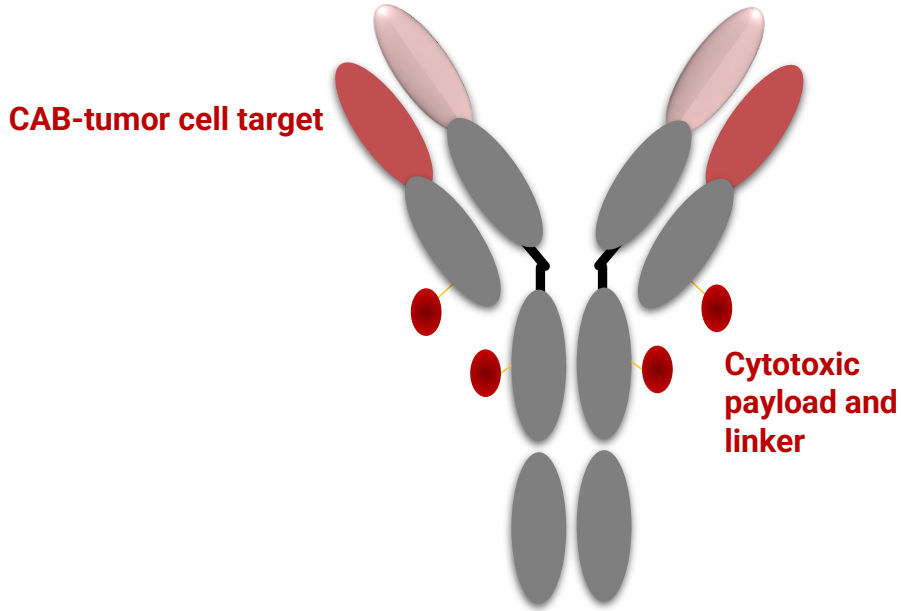
OPSCC, Oropharyngeal Squamous Cell Carcinoma; SOC, Standard of Care

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

BA3021 pH binding inflection point adjusted for tumor microenvironment selectivity





- 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

HPV+ E6/E7 Proteins Upregulate ROR2 and Drive Proliferation and Invasion

Provides compelling rationale for targeting ROR2 with Oz-V

- ROR2 is expressed by SCCHN at high rates
- 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

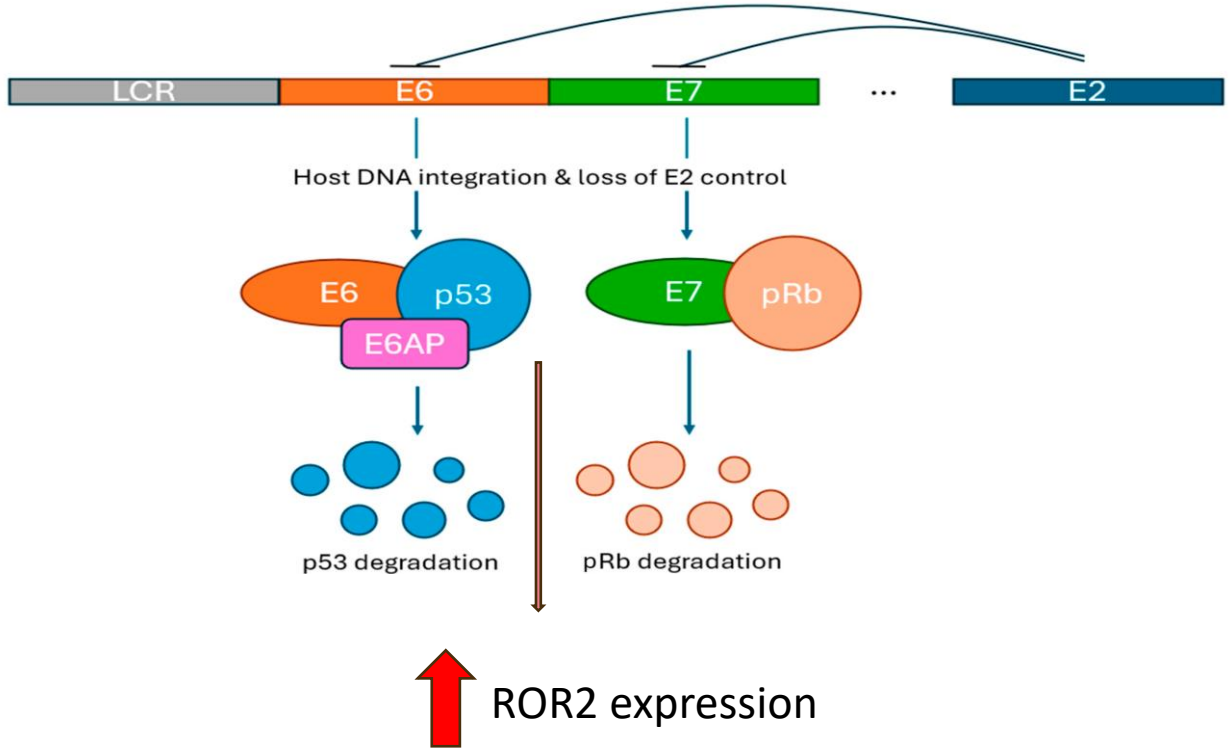


Figure adapted from Z. Lu, et al. Cancers. 2024, 16, 3474.

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

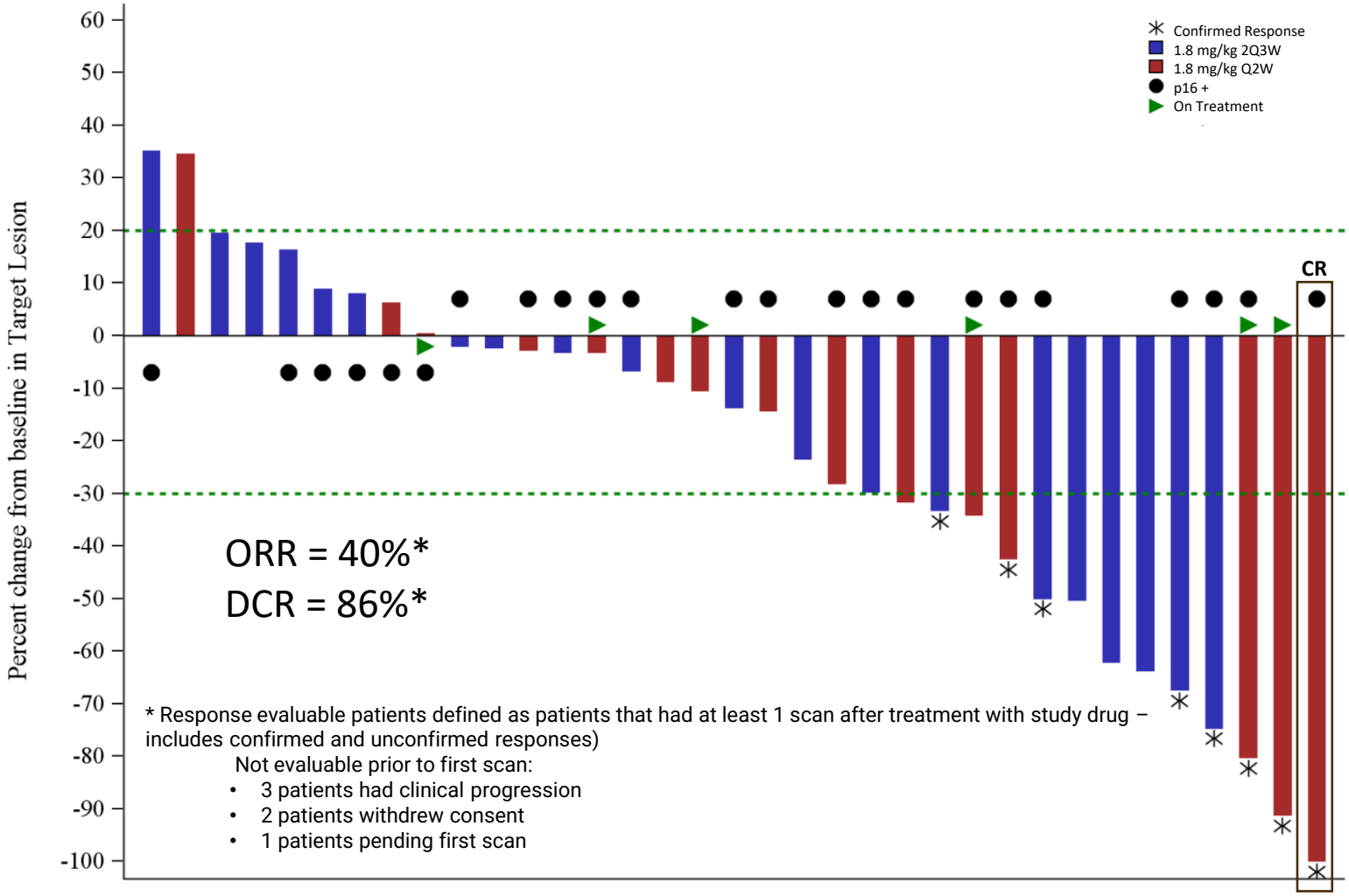
HPV p16 positive and all patients

	p16+ analysis	Full analysis
Median age, (range) years	64 (51-76)	65 (47-84)
Sex		
Male	24 (92)	37 (90)
Female	2 (8)	4 (10)
ECOG		
0	10 (39)	16 (39)
1	15 (58)	25 (61)
Prior lines of Therapy		
1	2 (8)	4 (10)
2	8 (31)	13 (33)
3+	16 (61)	22 (57)
PD-(L)1 inhibitor	26 (100)	41 (100)
Platinum-based therapy	23 (88)	34 (83)
Taxanes	13 (65)	
Cetuximab	8 (31)	20 (49)
Smoking Status		
Yes	0	1 (3)
Ex-smoker	14 (54)	22 (55)
Never	12 (46)	17 (43)

Data Cut Date: Live Database as of May 13, 2025

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 patients that had at least 1 scan after treatment with study drug – includes confirmed and unconfirmed responses)

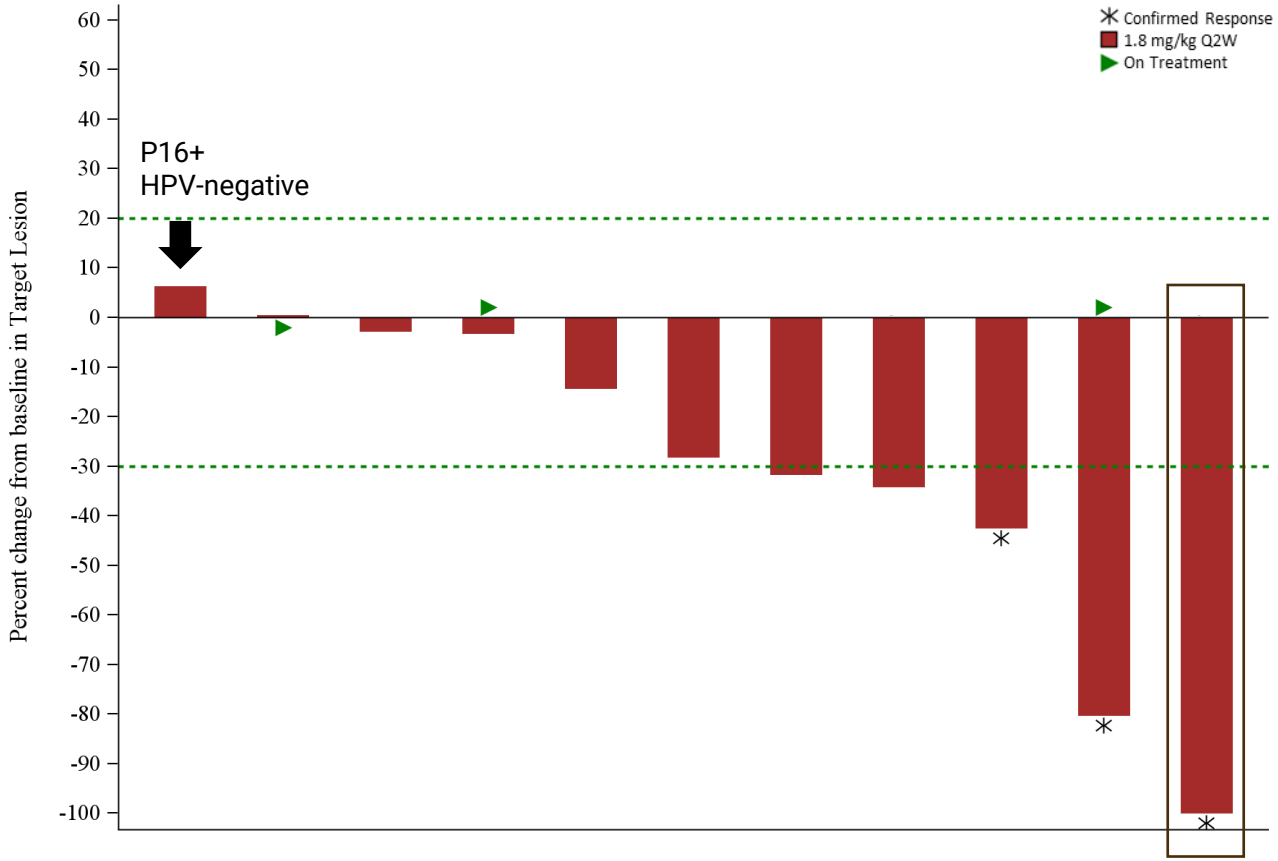
Not evaluable prior to first scan:

- 3 patients had clinical progression
- 2 patients withdrew consent
- 1 patients pending first scan

Data Cut Date: Live Database as of May 13, 2025

Oz-V in SCCHN p16+^ OPSCC

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



	Q2W
Responders (confirmed & unconfirmed)	**45%** (5/11*)
Responders (confirmed)	**27%** (3/11*)
DCR	**100%** (11/11*)
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

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**	**45% ORR 27% 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**."

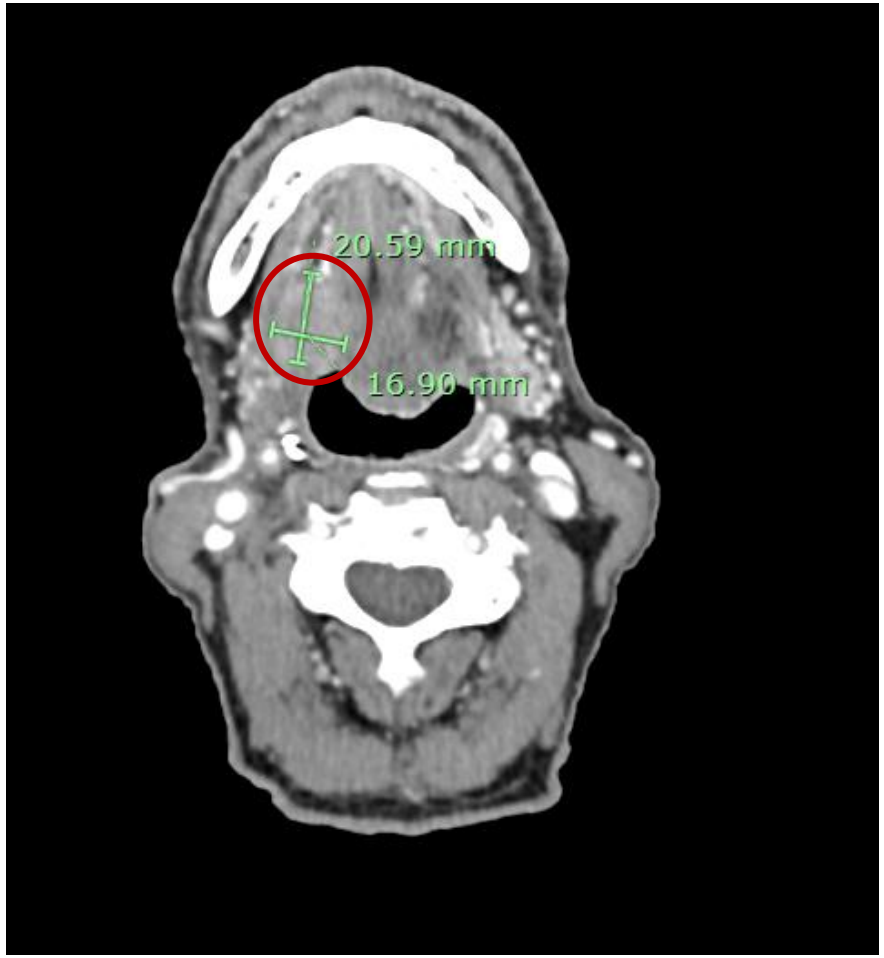
[1]N Engl J Med 2016;375:1856-1867. [2] Journal of Clinical Oncology 2018; 36(15): 1551-1558. [3]2008 Jun 15;112(12):2710-9. doi: 10.1002/cncr.23442, [4]Erbitux USPI accessed 2024., [5]INTERLINK-1: Phase 3 study of cetuximab ± monalizumab /Volume 34, Supplement 2S554-S555 October 2023
SOC, Standard of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

Data Cut Date: Live Database as of 14 May 2025

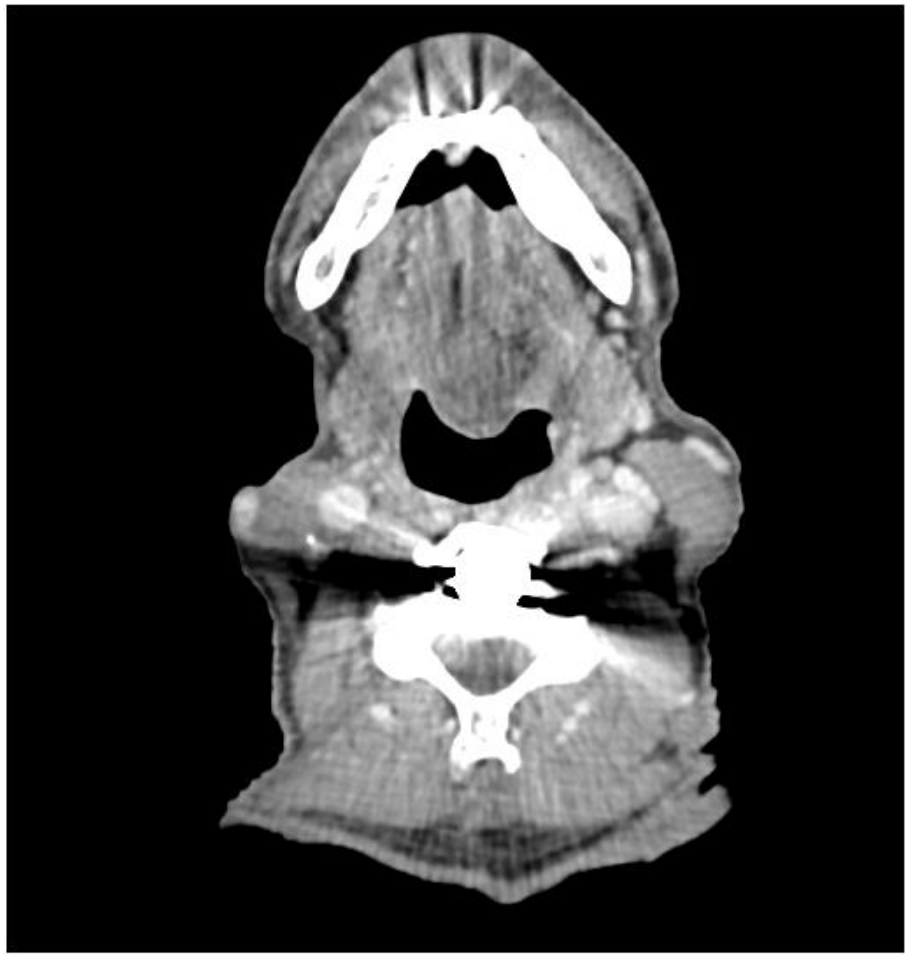
Confirmed Complete Response Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

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



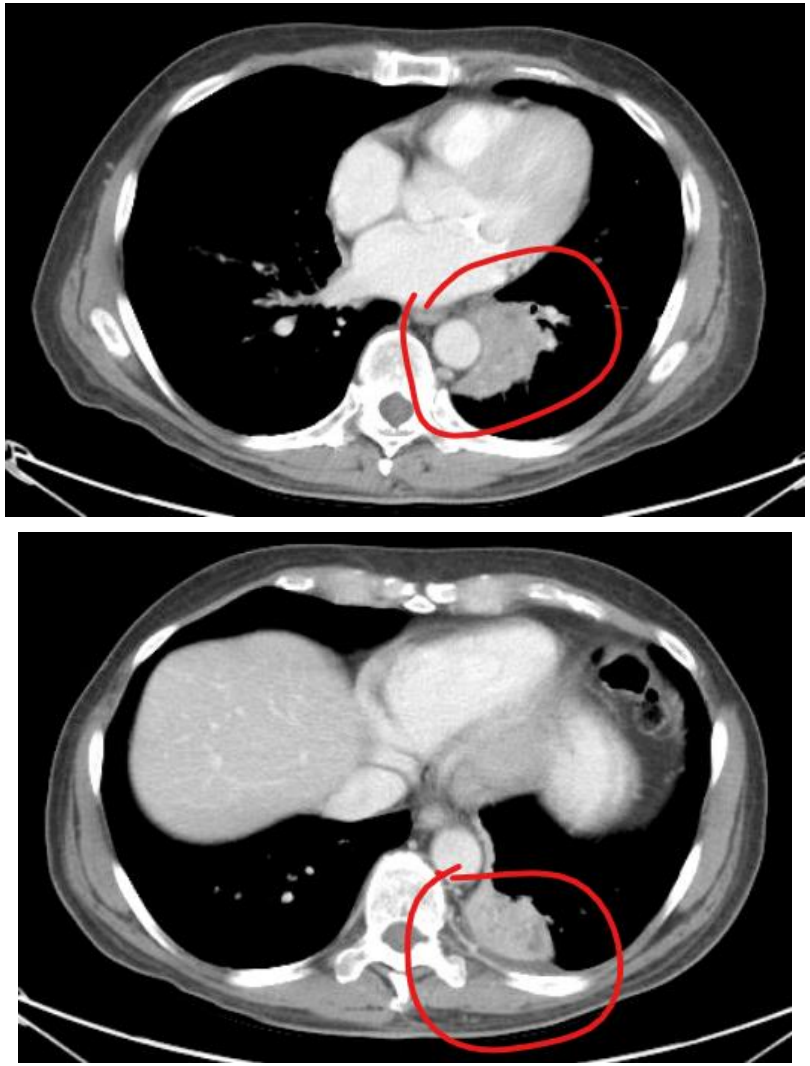
On Treatment – December 8, 2023



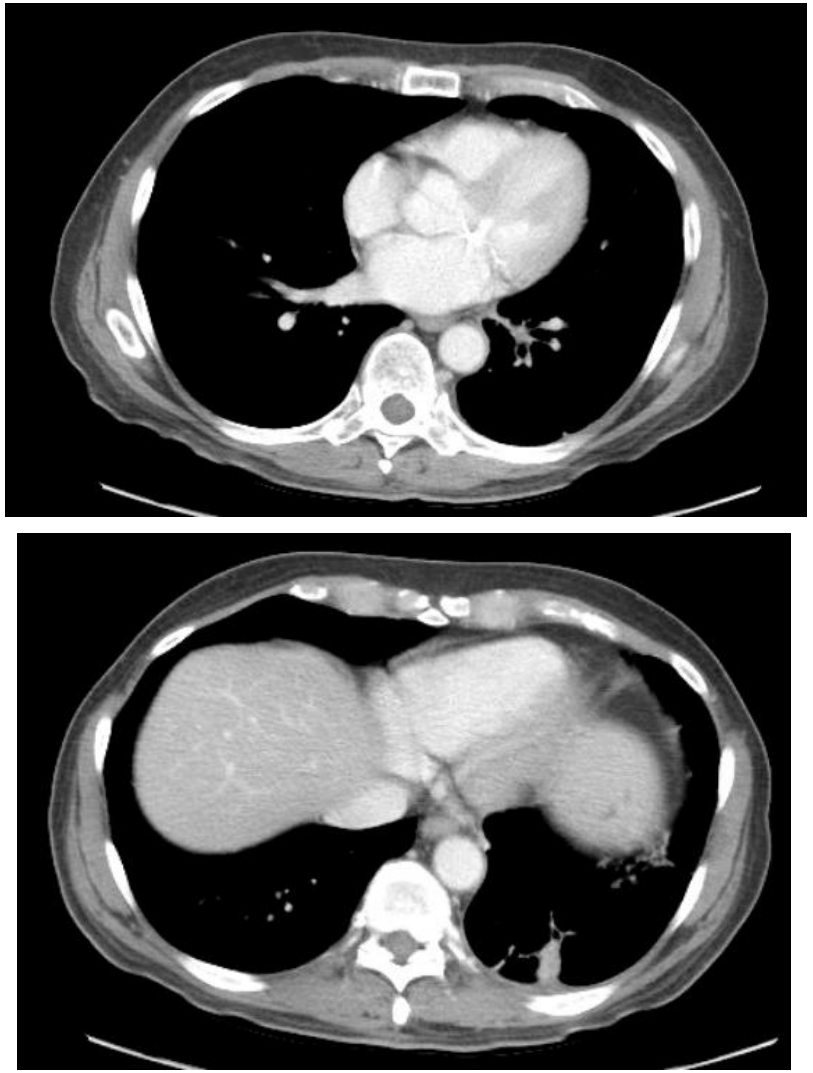
Partial Response (-80%) Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

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



Ph2 Oz-V: Overall Safety Summary of SCCHN patients

Q2W dosing regimen was well-tolerated

	1.8 mg/kg Q2W (N=20)	1.8 mg/kg 2Q3W (N=20)[3]	Total (N=40)[3]
Any Adverse Events (AEs)	18 (90%)	20 (100%)	38 (95%)
Related AEs with CTCAE[1] Grade 3 or 4[2]	3 (15%)	9 (45%)	12 (30%)
Any related serious AEs[2]	1 (5%)	4 (20%)	5 (13%)
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] One patient from Phase 1 not included

Data Cut Date: Live Database as of March 26, 2025

Phase 2 Oz-V Safety Data

Most frequent treatment-emergent related adverse events

	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	13 (65)	3 (15%)	19 (95%)	9 (45%)	32 (80%)	12 (30%)
Fatigue	5 (25%)	0	9 (45%)	0	14 (35%)	0
Nausea	3 (15%)	1 (5%)	6 (30%)	0	9 (23%)	1 (3%)
Lymphocyte count decreased	3 (15%)	1 (5%)	5 (25%)	5 (25%)	8 (20%)	6 (15%)
Diarrhoea	1 (5%)	0	6 (30%)	1 (5%)	7 (18%)	1 (3%)
Anaemia	3 (15%)	0	3 (15%)	0	6 (15%)	0
White blood cell count decreased	3 (15%)	0	3 (15%)	0	6 (15%)	0
Decreased appetite	1 (5%)	0	3 (15%)	0	4 (10%)	0
Localised oedema	1 (5%)	0	2 (10%)	0	3 (8%)	0
Hyponatraemia	0	0	3 (15%)	1 (5%)	3 (8%)	1 (3%)
Arthralgia	1 (5%)	0	2 (10%)	0	3 (8%)	0
Weight decreased	1 (5%)	0	2 (10%)	0	3 (8%)	0
Aspartate aminotransferase increased	1 (5%)	1 (5%)	2 (10%)	0	3 (8%)	1 (3%)
Myalgia	1 (5%)	0	2 (10%)	0	3 (8%)	0
Vomiting	2 (10%)	0	1 (5%)	0	3 (8%)	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: Live Database as of March 24, 2025

Ozuriftamab Vedotin Summary / Next Steps

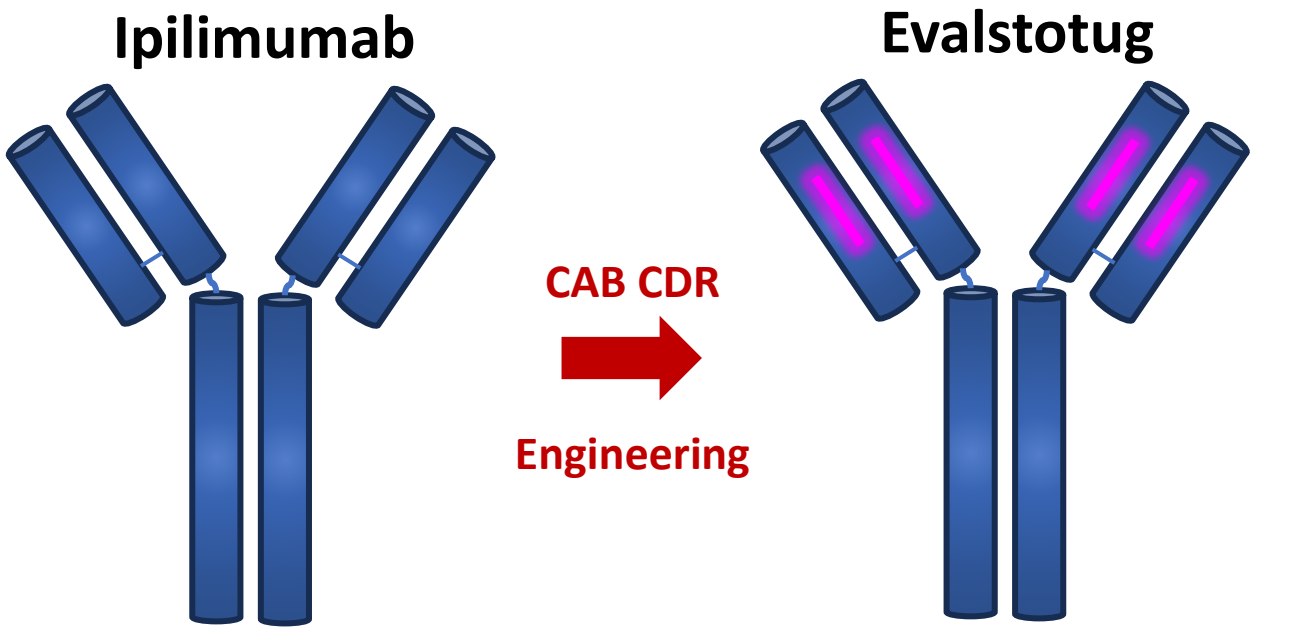
Oz-V has Fast Track Designation

- Previously received FDA actionable guidance on pivotal trial in 2L+ SCCHN; FDA supportive of:
  - Proposed pivotal randomized, controlled trial of Oz-V monotherapy vs investigator's choice (cetuximab, docetaxel, or methotrexate)
  - Dual primary endpoints
    - Overall Response Rate (potential for accelerated approval with acceptable DOR)
    - Overall Survival (supports full approval)
- Meeting planned with FDA in 3Q 2025 to confirm proposed Phase 3 study design in 2L+ HPV+ OPSCC
- Partnering discussions ongoing with updated Oz-V data in 2L+ HPV+ OPSCC

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)



Inability to distinguish between normal and tumor tissue

Similar efficacy and PK enhanced with selective pH-dependent tumor binding

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

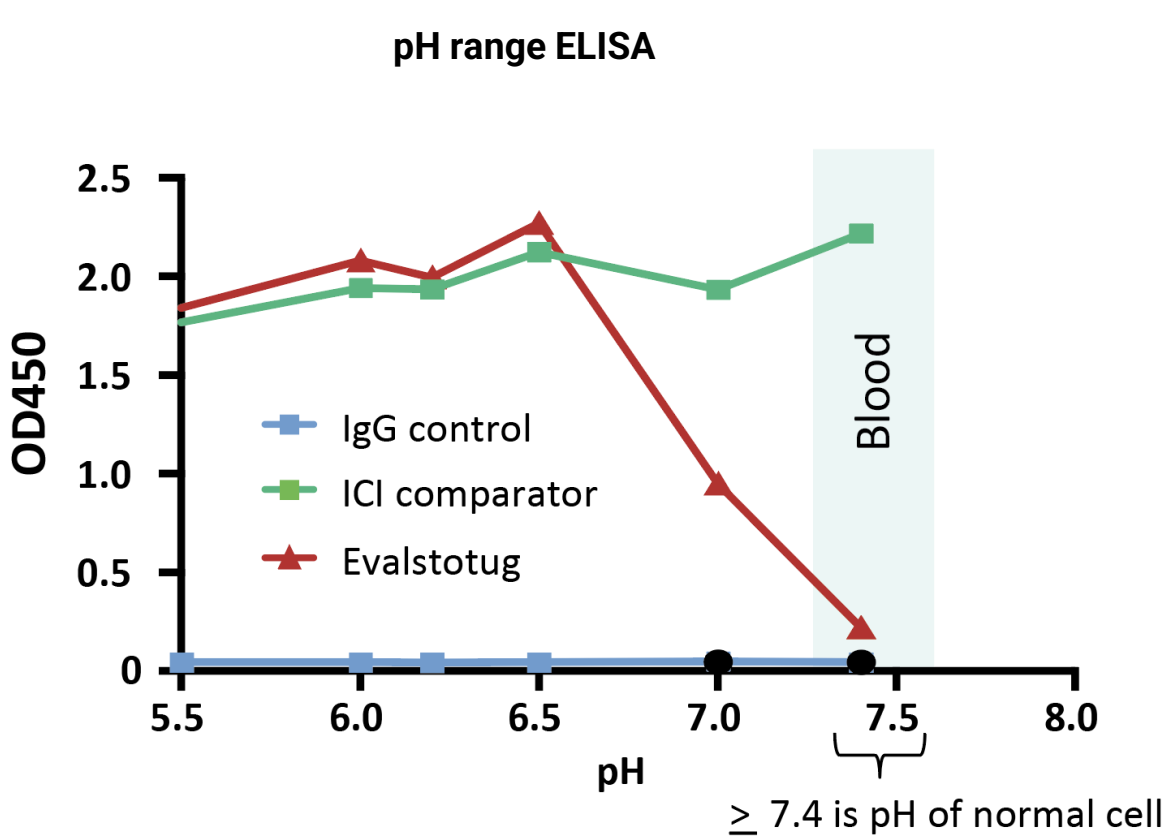
- CAB CTLA-4, evalstotug, enables targeted exposure in TME enabling lower imAE relative to ipi

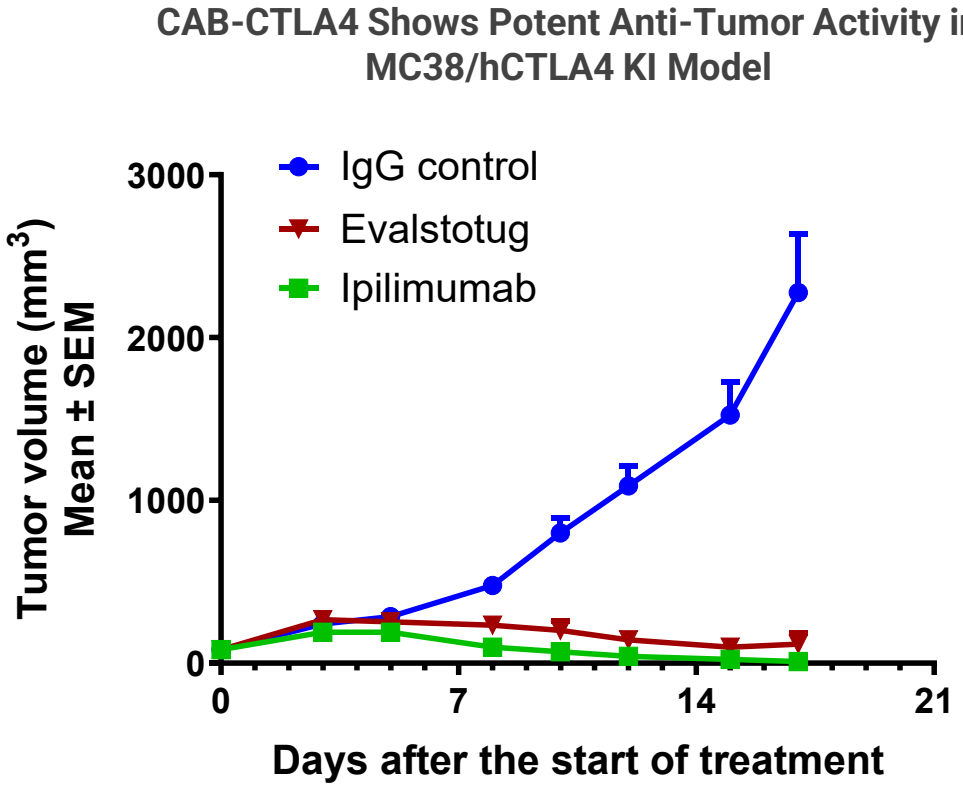
Abbreviations: ADCC, antibody-dependent cellular cytotoxicity; CAB, Conditionally Active Biologic; CD, cluster of differentiation; CDR, complementarity-determining region; CTLA-4, cytotoxic T-lymphocyte associated protein 4; EC50, concentration producing 50% Emax; Emax, maximum effect; imAE, immune mediated 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



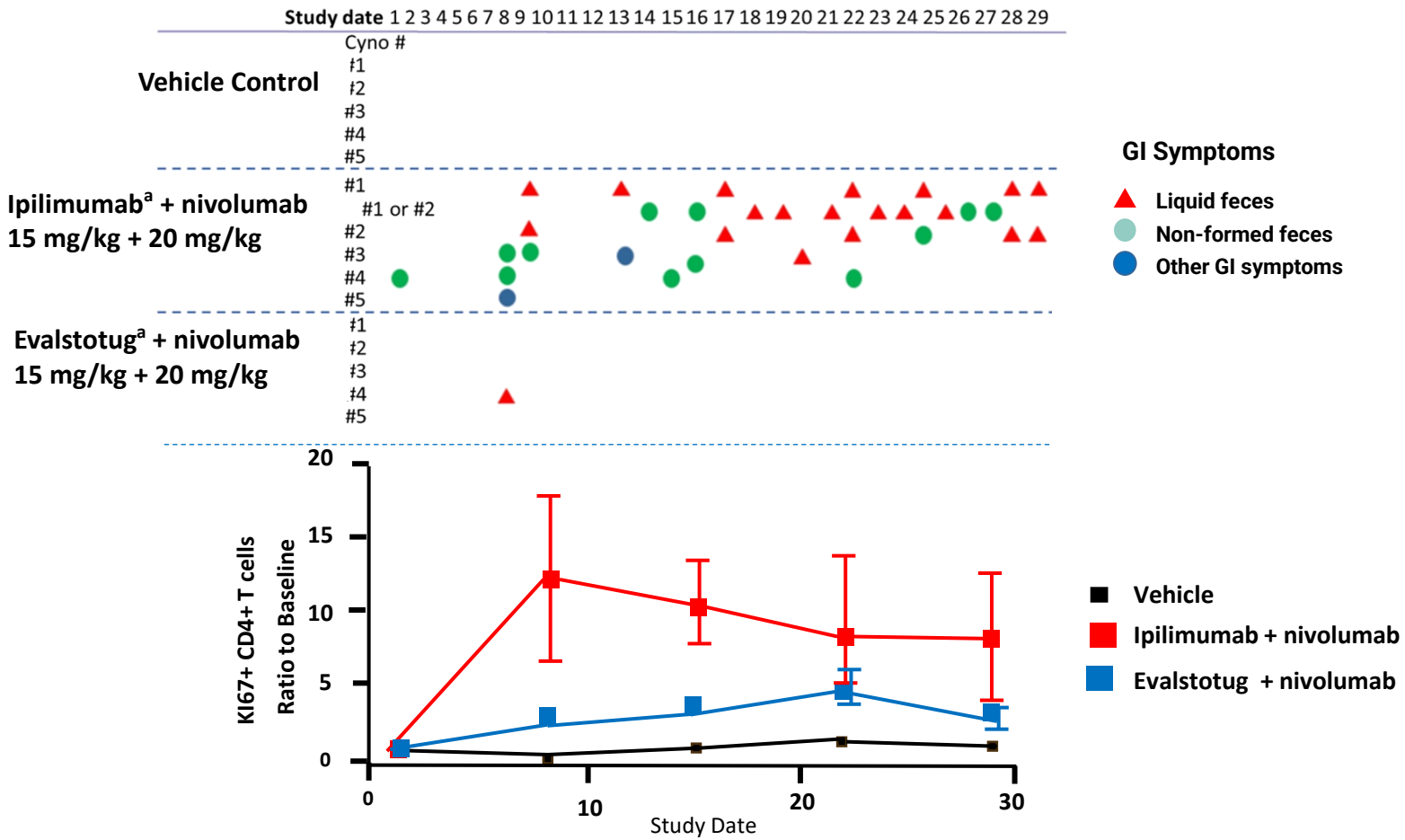


BA3071 induces complete tumor regression in mouse tumor model. Mice were dosed with IgG control and anti-CTLA antibodies at 10mg/kg (equivalent to 1mg/kg anti-CTLA-4 human dose), IP, BIW, N=12 mice/group.

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 at 350 mg (5mg/kg based on 70 kg pt) in Combination With PD1: Demographics – Tumor Types

82% patients experienced failure of prior PD-1 treatment

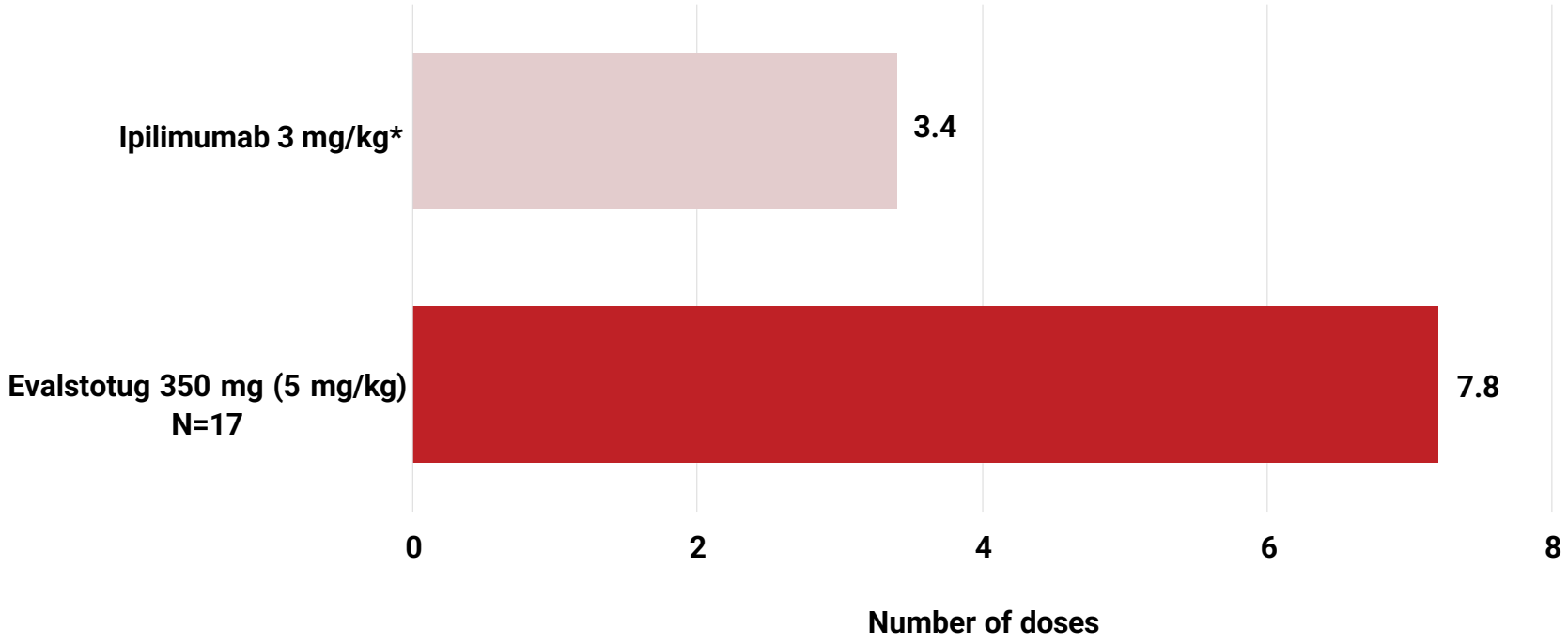
	Total (N=17)
Age, y, mean (SD)	60 (14)
Sex, n (%)	
Female	8 (47)
Male	9 (53)
White race, n (%)	13 (76)
ECOG, n (%)	
0	12 (71)
1	5 (29)
Prior Anti-PD-1 Therapy, n (%)	14 (82)

	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

Data Cut Date: 26Jul24

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

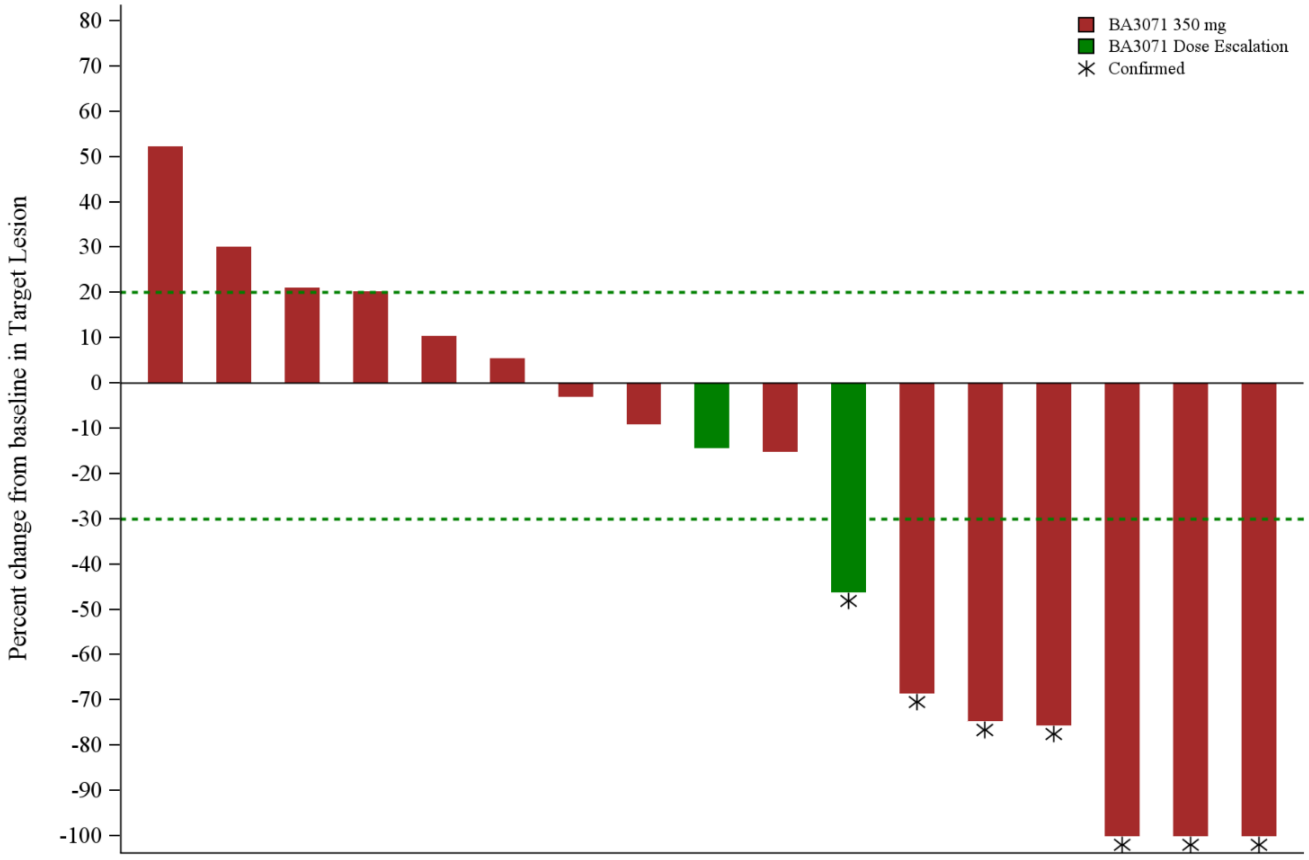


Note: Mean number of doses for ipilimumab is based on a retrospective observational study.
Evalstotug 350 mg and 700 mg are dose-equivalent to ipilimumab 5 mg/kg and 10 mg/kg, respectively.
*Mohr P, et al. J Eur Acad Dermatol Venereol. 2018;32(6):962-971.

Live Database 21Feb2025

Evalstotug at 350 mg in Combination with PD1 Across Multiple Tumor Types

17 patients treated at 350 mg; 3 Complete Responders with 2 in melanoma and 1 in cervical cancer



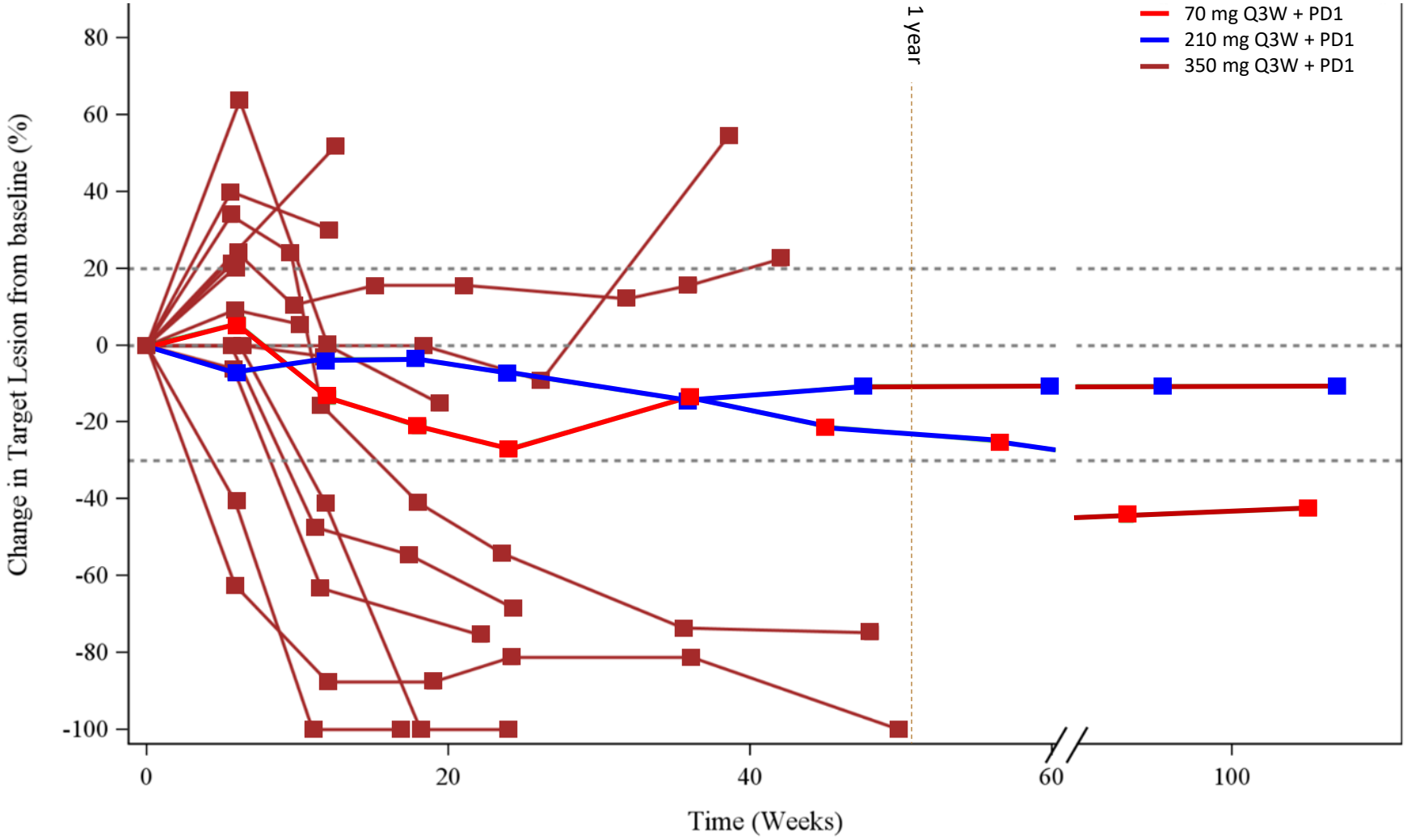
	Total
Responders (confirmed & unconfirmed)	**44%** (7/17[1])
Responders (confirmed)	**41%** (7/17[1])
DCR	**76%** (13/17[1])
DOR	***ongoing***
OS	***ongoing***

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

Live Database 28Apr2025

Evalstotug: 7 of 17 Achieved Response in the 5 mg/kg Q3W + PD1

Durable antitumor activity across multiple solid tumor types



Live Database 28Apr2025

67% ORR and 92% DCR in Unresectable and/or Metastatic Cutaneous Melanoma Patients Treated with Evalstotug (5—14.3 mg/kg) in combination PD-1 Antibody

10/12 patients had received prior PD1 adjuvant or neoadjuvant treatment

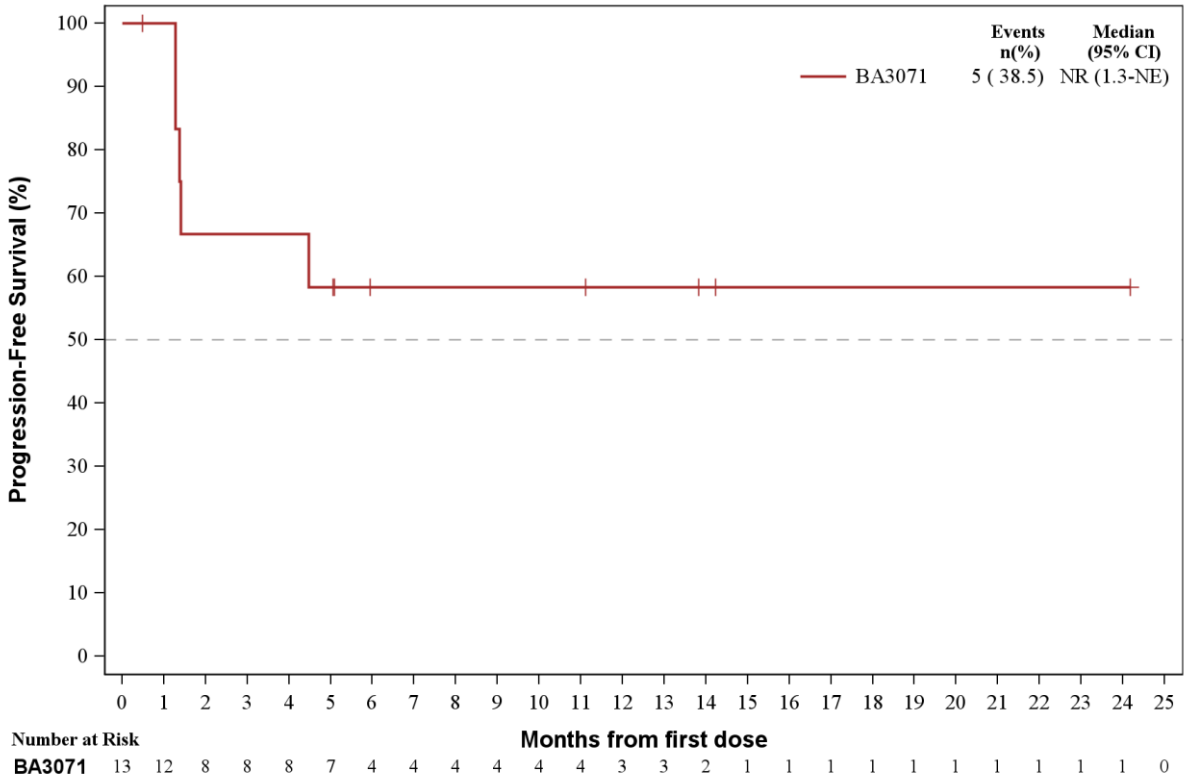
Evalstotug dose (mg)	Age (yrs)	Sex	ECOG	Prior Treatment	Prior PD1 exposure < 6 months	Best change in target lesion (%)	Response to evalstotug
350	34	M	0	None	No	-100%	CR*
350	54	F	0	Adjuvant anti-PD-1 for 3mos	Yes	-100%	**cCR**
350	63	M	0	Adjuvant anti-LAG-3/anti-PD-1 for 11mos	Yes	-9%	SD
350	59	F	0	Adjuvant anti-PD-1 for 12mos	No	-75%	cPR
350	67	F	0	Adjuvant anti-PD-1 for 12mos	Yes	-100%	**cCR**
350	89	F	0	None	No	0%	SD
350	34	M	0	Adjuvant anti-PD-1 for 6mos	No	-	PD
700	73	M	0	Adjuvant anti-PD-1 for 7mos	Yes	-50%	cPR
700	57	F	1	Neo-adjuvant anti-PD-1 for 12mos	No	-8%	SD
700	72	F	0	Adjuvant anti-PD-1 for 11mos	No	-34%	cPR
70 ➢210 ➢350	75	F	1	Adjuvant anti-PD-1 for 11mos	Yes	-46%	cPR
700 ➢1000	57	M	0	Adjuvant anti-LAG-3/anti-PD-1 for 3mos	Yes	-39%	PR**

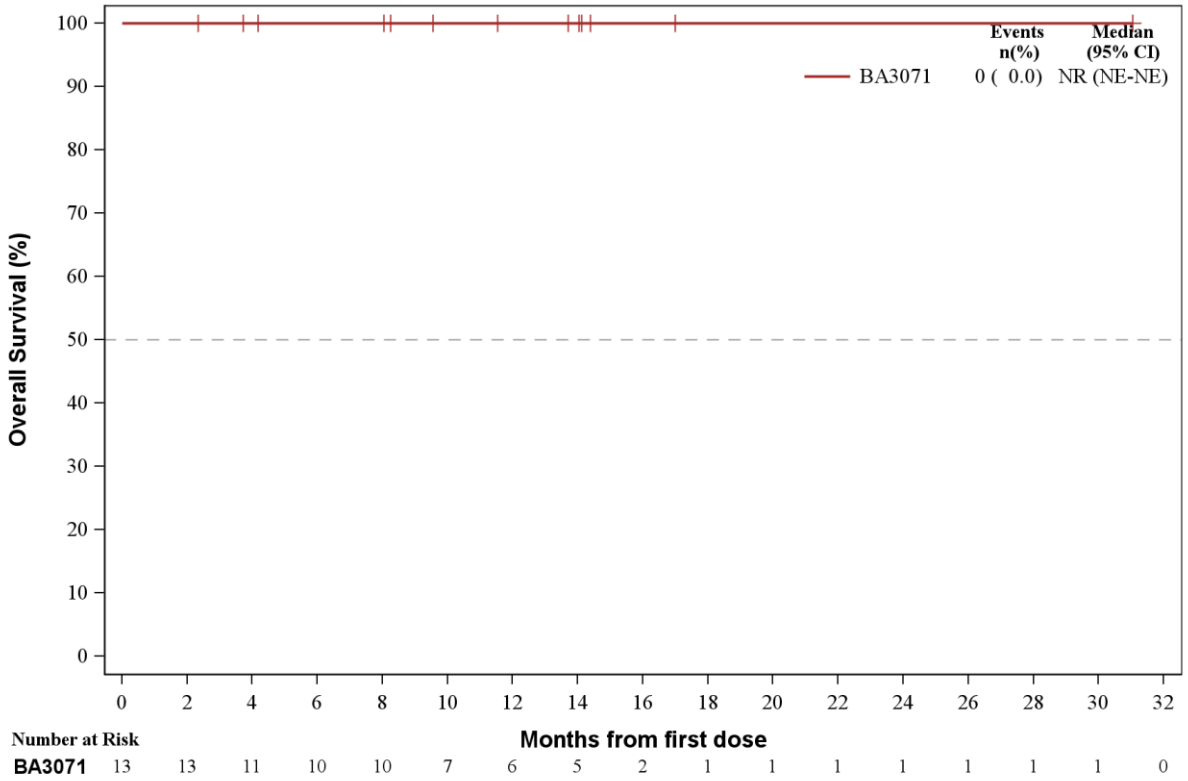
*Patient with 73% tumor reduction initially experienced tumor growth with new lesions and Investigator continued treatment per protocol resulting in complete metabolic response based on PET scan (May 5, 2025)
**Pending confirmation

Live Database 28Apr2025

Evalstotug in Combination with PD1 Across Prior IO 1L Melanoma

All patients still alive





Data Cut Date: 22Jul25

Evalstotug vs Ipilimumab TRAE, imAE, and Response

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

Doses	1 – ≤18 weeks exposure (≤6 doses)	1 - >100 weeks exposure (1 - >33 doses)	1 – ≤12 weeks exposure (≤4 doses)
Treatment (Q3W)	Evalstotug (5 mg/kg^) + PD1 Q3W N=17	Evalstotug (5 mg/kg^) + PD1 Q3W N=17	Ipilimumab (3 mg/kg) + nivolumab Q3W N=178-314[1,2,3]
Tumor Types	Multiple tumor types 90% with prior tx		Melanoma 15% with prior tx[4]
TRAE (G3-4)	29%	41%	48-59%
imAE (G3-4)	18% (no G4)	29% (no G4)	40%

^Two patients dose escalated
- 70 mg to 350 mg
- 210 mg to 350 mg

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.*

Evalstotug in Combination with PD1 Safety Data

Treatment-emergent related adverse events

	Total (N=17)		
Preferred Term	**All Grades [n (%)]**	**Grade 3 [n (%)]**	**Grade 4 [n (%)]**
Number of subjects with at least one TEAE	**16 (94%)**	**6 (36%)**	**1 (6%)**
Chills	12 (71%)	0 (0%)	0 (0%)
Nausea	6 (35%)	0 (0%)	0 (0%)
Vomiting	5 (29%)	0 (0%)	0 (0%)
Fatigue	5 (29%)	0 (0%)	0 (0%)
Cytokine release syndrome	5 (29%)	0 (0%)	0 (0%)
Pyrexia	4 (24%)	0 (0%)	0 (0%)
Arthralgia	4 (24%)	0 (0%)	0 (0%)
Infusion related reaction	4 (24%)	0 (0%)	0 (0%)
Rash	4 (24%)	0 (0%)	0 (0%)
Lipase increased	**3 (18%)**	**2 (12%)**	**0 (0%)**
Headache	3 (18%)	0 (0%)	0 (0%)
Pruritus	3 (18%)	0 (0%)	0 (0%)
Colitis	**2 (12%)**	**2 (12%)**	0 (0%)
Pancreatitis	**2 (12%)**	**2 (12%)**	0 (0%)
Diarrhoea	2 (12%)	0 (0%)	0 (0%)
Hypotension	2 (12%)	0 (0%)	0 (0%)
Influenza like illness	2 (12%)	0 (0%)	0 (0%)
Rash maculo-papular	2 (12%)	0 (0%)	0 (0%)
Hypercalcaemia	1 (6%)	0 (0%)	1 (6%)*

	Total (N=17)		
Preferred Term	**All Grades [n (%)]**	**Grade 3 [n (%)]**	**Grade 4 [n (%)]**
Chronic gastritis	**1 (6%)**	**1 (6%)**	**0 (0%)**
Hypertension	**1 (6%)**	**1 (6%)***	**0 (0%)**
Autoimmune encephalopathy	**1 (6%)**	**1 (6%)**	**0 (0%)**
Abdominal pain	1 (6%)	0 (0%)	0 (0%)
Decreased appetite	1 (6%)	0 (0%)	0 (0%)
Asthenia	1 (6%)	0 (0%)	0 (0%)
Troponin increased	1 (6%)	0 (0%)	0 (0%)
Muscle spasms	1 (6%)	0 (0%)	0 (0%)
Pain	1 (6%)	0 (0%)	0 (0%)
Immune-mediated adrenal insufficiency	1 (6%)	0 (0%)	0 (0%)
Dizziness	1 (6%)	0 (0%)	0 (0%)
Autoimmune thyroiditis	1 (6%)	0 (0%)	0 (0%)
Skin lesion	1 (6%)	0 (0%)	0 (0%)
Alanine aminotransferase increased	1 (6%)	0 (0%)	0 (0%)
Aphthous ulcer	1 (6%)	0 (0%)	0 (0%)
Thyroiditis	1 (6%)	0 (0%)	0 (0%)
Aspartate aminotransferase increased	1 (6%)	0 (0%)	0 (0%)
Tachycardia	1 (6%)	0 (0%)	0 (0%)
Tongue geographic	1 (6%)	0 (0%)	0 (0%)
Hyperthyroidism	1 (6%)	0 (0%)	0 (0%)
Hyperhidrosis	1 (6%)	0 (0%)	0 (0%)

*G4 Hypercalcaemia and G3 Hypertension are not imAE based on SITC criteria.

Evalstotug in Combination with PD1: Overall Safety Summary

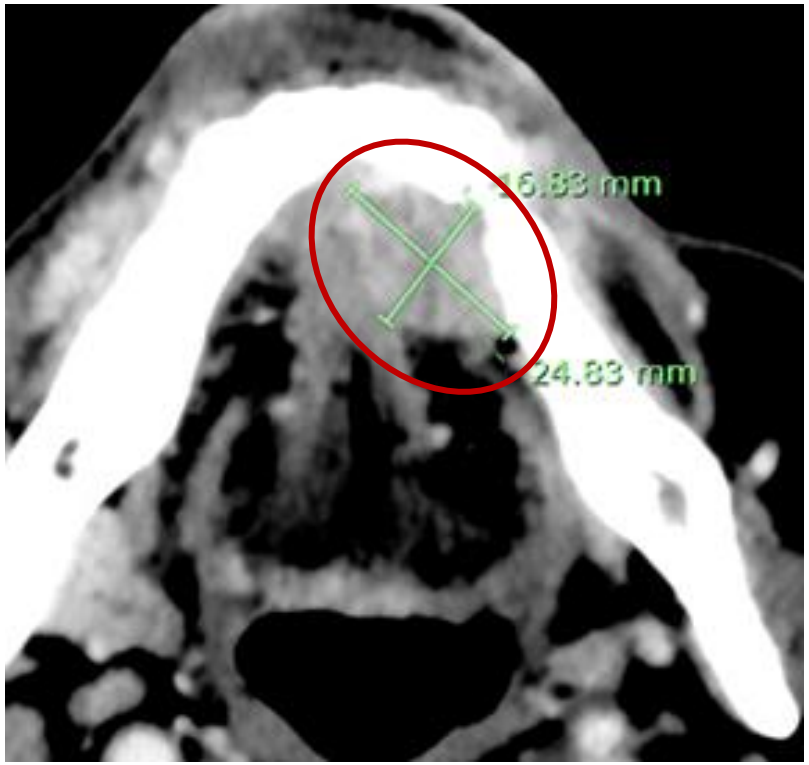
Generally well-tolerated; All G3 and G4 resolved

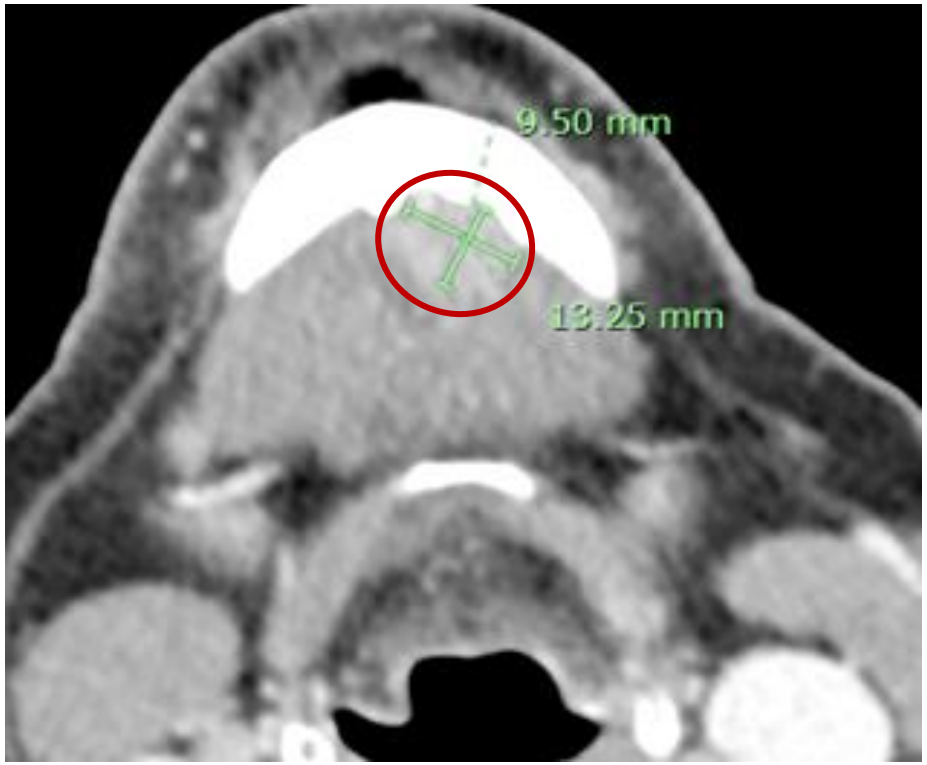
Related AE Summary	Total (N=17)
Any Related Adverse Events (AEs)	16 (94%)
Related AEs with CTCAE[1]	
Grade 3[2]	6 (36%)
Grade 4[2] transient hypercalcemia	1 (6%)
Any related serious AEs[2]	5 (29%)
Possibly Related AEs leading to death[2]	0
Related AEs leading to treatment discontinuation[2]	5 (29%)

[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 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

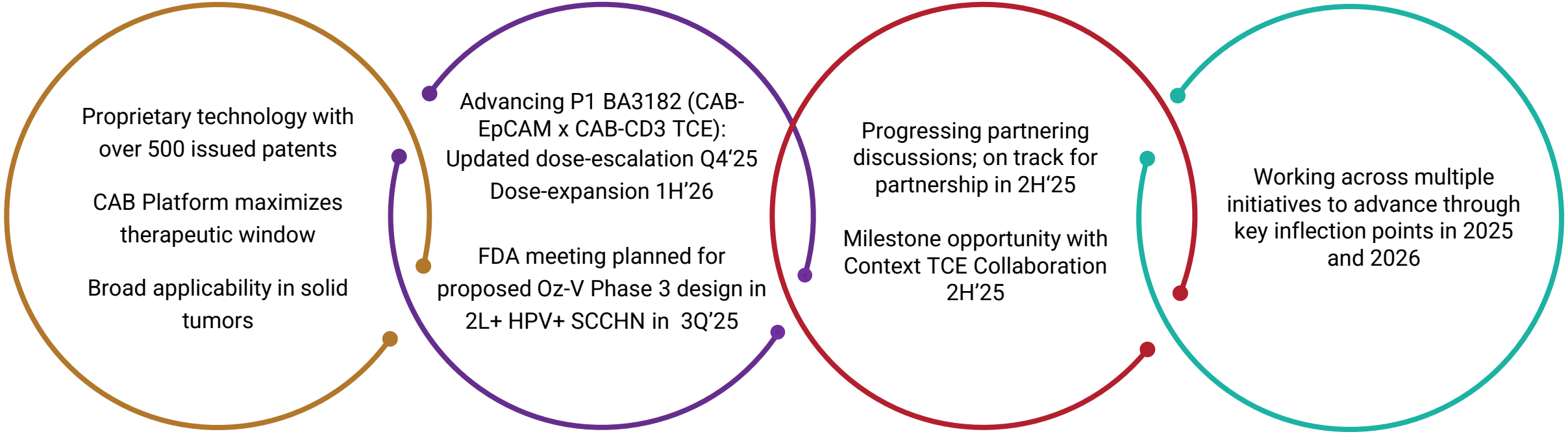
IO – immuno-oncology

Conclusions

- 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 irAEs and extended treatment
- 67% ORR and 92% DCR in unresectable and/or metastatic cutaneous melanoma patients treated with evalstotug (5 – 14.3 mg/kg) in combination with PD-1 antibody
- Higher and longer dosing relative to ipi 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
- Recently initiated partnering discussions

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

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



TCE – T-cell engager