

ACUMEN PHARMACEUTICALS, INC.

FORM 8-K (Current report filing)

Filed 10/26/26 for the Period Ending 10/26/26

Address	1210-1220 WASHINGTON STREET SUITE 210 NEWTON, MA, 02465
Telephone	617-344-4190
CIK	0001576885
Symbol	ABOS
SIC Code	2836 - Biological Products, (No Diagnostic Substances)
Industry	Biotechnology & Medical Research
Sector	Healthcare
Fiscal Year	12/31

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): **October 26, 2026**

Acumen Pharmaceuticals, Inc.
(Exact name of Registrant as Specified in Its Charter)

Delaware
(State or Other Jurisdiction
of Incorporation)

001-40551
(Commission
File Number)

36-4108129
(IRS Employer
Identification No.)

**1210-1220 Washington Street, Suite 210
Newton, Massachusetts**
(Address of Principal Executive Offices)

02465
(Zip Code)

Registrant's Telephone Number, Including Area Code: **(617) 344-4190**

(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 (see General Instructions A.2. below):

- ☐ 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	ABOS	The Nasdaq Global Select 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 October 26, 2026, Acumen Pharmaceuticals, Inc. (the “**Company**”) reported financial results and business highlights for the quarter ended September 30, 2026. A copy of this press release (the “**Earnings Press Release**”) is furnished as Exhibit 99.1 to this Current Report on Form 8-K (this “**Report**”) and is incorporated by reference.

The information in this Item 2.02 of this Report (including Exhibit 99.1) 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 that Section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended (the “**Securities Act**”), or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 7.01 Regulation FD Disclosure.

On October 26, 2026, the Company posted an updated corporate presentation to its website at <https://investors.acumenpharm.com/news-events/presentations>, which the Company may use from time to time in communications or conferences. The corporate presentation was updated to reflect the Company’s cash, cash equivalents and marketable securities balance as of September 30, 2026. A copy of the corporate presentation is attached as Exhibit 99.2 to this Report.

The information in this Item 7.01 of this Report (including Exhibit 99.2), 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 that Section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such filing. The Company’s submission of this Report shall not be deemed an admission as to the materiality of any information required to be disclosed solely to satisfy the requirements of Regulation FD.

Item 9.01 Financial Statements and Exhibits.

(d). Exhibits

Exhibit No.	Description
99.1	Earnings Press Release, dated October 26, 2026
99.2	Corporate Presentation, dated October 26, 2026
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

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

Acumen Pharmaceuticals, Inc.

Dated: October 26, 2026

By: /s/ Matthew Zuga
Matthew Zuga
Chief Financial Officer and Chief Business Officer



**Acumen Pharmaceuticals Reports Third Quarter 2026 Financial Results
and Business Highlights**

- Expect to report topline results for ALTITUDE-AD, a Phase 2 study to investigate sabirnetug (ACU193) for the treatment of early Alzheimer’s disease, in November 2026
- IND filing for the Enhanced Brain Delivery™ (EBD®) program lead clinical candidate targeted for mid-2027
- Cash, cash equivalents and marketable securities of \$78.6 million as of September 30, 2026, expected to support current clinical and operational activities into early 2027
- Company to host conference call and webcast today at 8:00 a.m. ET

NEWTON, Mass., Oct. 26, 2026 – [Acumen Pharmaceuticals, Inc.](#) (NASDAQ: ABOS) (“Acumen” or the “Company”), a clinical-stage biopharmaceutical company developing novel therapeutics that target toxic soluble amyloid beta oligomers (AβOs) for the treatment of Alzheimer’s disease (AD), today reported financial results for the third quarter of 2026 and provided a business update.

“In the third quarter, we continued readying for the readout from our ALTITUDE-AD Phase 2 study investigating the efficacy, safety and tolerability of sabirnetug for the treatment of early AD, and today I’m pleased to announce that we expect to report topline results in November 2026. We remain encouraged by the potential for sabirnetug to become a treatment of choice, with a differentiated benefit-risk profile driven by its unique attributes as an anti-AβO IgG2 monoclonal antibody,” said Daniel O’Connell, Chief Executive Officer of Acumen. “Another important piece of Acumen’s value proposition is our Enhanced Brain Delivery™ program, combining a validated blood-brain barrier-penetrating technology with an AβO-selective therapeutic antibody. We continue to make progress on IND-enabling activities in support of a mid-2027 IND submission for a lead candidate and remain encouraged about the potential it holds as a next-generation Alzheimer’s disease treatment.”

Anticipated Milestones

- The Company expects to report topline results from ALTITUDE-AD in November 2026.
 - ALTITUDE-AD is a 3-arm Phase 2 study investigating sabirnetug in 542 participants (N=180 per arm) for the treatment of early Alzheimer’s disease.



- ALTITUDE-AD includes two active doses (35 mg/kg, 50 mg/kg, IV Q4W) vs. placebo over an 18-month double-blinded period.
- Topline results are expected to include the iADRS (Integrated Alzheimer's Disease Rating Scale), our primary clinical efficacy endpoint, as well as key secondary endpoints, such as CDR-SB (Clinical Dementia Rating – Sum of the Boxes), certain safety measures such as adverse event rates, including ARIA rates, and key biomarkers.
- The Company is targeting the submission of an IND filing with respect to a lead clinical candidate in its EBD program in mid-2027.

Third Quarter 2026 Financial Results

- **Cash Balance.** As of Sept. 30, 2026, cash, cash equivalents and marketable securities totaled \$78.6 million compared to cash, cash equivalents and marketable securities of \$110.2 million as of June 30, 2026. Cash is expected to support current clinical and operational activities into early 2027.
- **Research and Development (R&D) Expenses.** R&D expenses were \$21.1 million for the quarter ended Sept. 30, 2026, compared to \$22.0 million for the quarter ended Sept. 30, 2025. The decrease was primarily due to reductions in manufacturing and materials costs, and CRO costs, which are both associated with our ALTITUDE-AD clinical trial, and partially offset by increased spending for our EBD program.
- **General and Administrative (G&A) Expenses.** G&A expenses were \$4.7 million for the quarter ended Sept. 30, 2026, compared to \$4.5 million for the quarter ended Sept. 30, 2025.
- **Loss from Operations.** Loss from operations was \$25.8 million for the quarter ended Sept. 30, 2026, compared to \$26.5 million for the quarter ended Sept. 30, 2025. This decrease was due to the decreased R&D expenses over the prior year period.
- **Net Loss.** Net loss was \$25.4 million for the quarter ended Sept. 30, 2026, compared to \$26.5 million for the quarter ended Sept. 30, 2025.

Conference Call Details

Acumen will host a conference call and live audio webcast today, Oct. 26, 2026, at 8:00 a.m. ET.

To participate in the live conference call, please register using this [link](#). After registration, you will be informed of the dial-in numbers including PIN. Please register at least one day in advance.

The webcast audio will be available via this [link](#).

An archived version of the webcast will be available for at least 30 days in the Investors section of the Company's website at www.acumenpharm.com.



About Sabirnetug (ACU193)

Sabirnetug (ACU193) is a humanized monoclonal antibody (mAb) discovered and developed based on its selectivity for soluble amyloid beta oligomers (AβOs), which are a highly toxic and pathogenic form of Aβ, relative to Aβ monomers and amyloid plaques. Soluble AβOs have been observed to be potent neurotoxins that bind to neurons, inhibit synaptic function and induce neurodegeneration. By selectively targeting toxic soluble AβOs, sabirnetug aims to address the hypothesis that soluble AβOs are an early and persistent underlying cause of the neurodegenerative process in Alzheimer's disease (AD). Sabirnetug has been granted Fast Track designation for the treatment of early AD by the U.S. Food and Drug Administration and is currently being evaluated in a Phase 2 study in patients with early AD.

About ALTITUDE-AD (Phase 2)

Initiated in 2024, ALTITUDE-AD is a Phase 2, multi-center, randomized, double-blind, placebo-controlled clinical trial designed to evaluate the efficacy and safety of sabirnetug (ACU193) infusions administered once every four weeks in slowing cognitive and functional decline as compared to placebo in participants with early Alzheimer's disease. The study has enrolled 542 individuals with early Alzheimer's disease (mild cognitive impairment or mild dementia due to AD) at multiple investigative sites located in the United States, Canada, the European Union and the United Kingdom. Topline results are expected in November 2026. More information can be found on www.clinicaltrials.gov, NCT identifier NCT06335173.

About Acumen Pharmaceuticals, Inc.

Acumen Pharmaceuticals is a clinical-stage biopharmaceutical company developing novel therapeutics that target toxic soluble amyloid beta oligomers (AβOs) for the treatment of Alzheimer's disease (AD). Acumen's scientific founders pioneered research on AβOs, which a growing body of evidence indicates are early and persistent triggers of Alzheimer's disease pathology. Acumen is currently focused on advancing its lead investigational product candidate, sabirnetug (ACU193), a humanized monoclonal antibody that selectively targets toxic soluble AβOs, in its ongoing Phase 2 clinical trial ALTITUDE-AD (NCT06335173) in early symptomatic Alzheimer's disease patients, following positive results in its Phase 1 trial INTERCEPT-AD. Acumen is also investigating a subcutaneous formulation of sabirnetug using Halozyme's proprietary ENHANZE® drug delivery technology. Acumen is also collaborating with JCR Pharmaceuticals to develop an Enhanced Brain Delivery™ (EBD®) therapy for Alzheimer's disease utilizing a transferrin-receptor-targeting blood-brain barrier-penetrating technology. The company is headquartered in Newton, Mass. For more information, visit www.acumenpharm.com.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. Any statement describing Acumen's goals, expectations, financial or other projections, intentions or beliefs is a forward-looking statement and should be considered an at-risk statement. Words such as "believes," "expects,"



“anticipates,” “could,” “should,” “would,” “seeks,” “aims,” “plans,” “potential,” “will,” “milestone” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Forward-looking statements include statements concerning Acumen’s business, and Acumen’s ability to achieve its strategic and financial goals, including its projected use of cash, cash equivalents and marketable securities and the expected sufficiency of its cash resources into early 2027, the therapeutic potential of Acumen’s product candidate, sabirnetug (ACU193), including against other antibodies, the timing of anticipated topline results of ALTITUDE-AD, Acumen’s plans to develop a candidate to treat Alzheimer’s Disease utilizing EBD technology, including its expectations with respect to timing for the submission of an IND, the potential of its EBD candidates and the potential for developing a best-in-class therapeutic candidate for people living with Alzheimer’s Disease. These statements are based upon the current beliefs and expectations of Acumen management, and are subject to certain factors, risks and uncertainties, particularly those inherent in the process of discovering, developing and commercializing safe and effective human therapeutics. Such risks may be amplified by the impacts of geopolitical events and macroeconomic conditions, such as rising inflation and interest rates, supply disruptions and uncertainty of credit and financial markets. These and other risks concerning Acumen’s programs are described in additional detail in Acumen’s filings with the Securities and Exchange Commission (“SEC”), including in Acumen’s most recent Annual Report on Form 10-K, and in subsequent filings with the SEC. Copies of these and other documents are available from Acumen. Additional information will be made available in other filings that Acumen makes from time to time with the SEC. These forward-looking statements speak only as of the date hereof, and Acumen expressly disclaims any obligation to update or revise any forward-looking statement, except as otherwise required by law, whether, as a result of new information, future events or otherwise.

CONTACTS:

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ICR Healthcare
AcumenPR@icrhealthcare.com



Acumen Pharmaceuticals, Inc.
Condensed Balance Sheets
(in thousands, except share and per share data)

	September 30, 2026 (unaudited)	December 31, 2025
ASSETS		
Current assets		
Cash and cash equivalents	\$ 53,075	\$ 53,989
Marketable securities	25,529	62,876
Prepaid expenses and other current assets	6,530	5,387
Total current assets	85,134	122,252
Restricted cash	231	231
Other assets, long-term	200	350
Total assets	<u>\$ 85,565</u>	<u>\$ 122,833</u>
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities		
Accounts payable	\$ 6,056	\$ 554
Accrued clinical trial expenses	8,459	10,616
Accrued expenses and other current liabilities	10,168	10,072
Debt, short-term	20,404	8,765
Total current liabilities	45,087	30,007
Debt, long-term	5,868	22,396
Total liabilities	50,955	52,403
Commitments and contingencies		
Stockholders' equity		
Preferred stock, \$0.0001 par value; 10,000,000 shares authorized and no shares issued and outstanding as of September 30, 2026 and December 31, 2025	—	—
Common stock, \$0.0001 par value; 300,000,000 shares authorized as of September 30, 2026 and December 31, 2025; 72,327,320 and 60,575,369 shares issued and outstanding as of September 30, 2026 and December 31, 2025, respectively	7	6
Additional paid-in capital	559,914	516,803
Accumulated deficit	(525,286)	(446,462)
Accumulated other comprehensive (loss) income	(25)	83
Total stockholders' equity	34,610	70,430
Total liabilities and stockholders' equity	<u>\$ 85,565</u>	<u>\$ 122,833</u>



Acumen Pharmaceuticals, Inc.
Condensed Statements of Operations and Comprehensive Loss
(in thousands, except share and per share data)
(unaudited)

	Three Months Ended September 30,		Nine Months Ended September 30,	
	2026	2025	2026	2025
Operating expenses				
Research and development	\$ 21,113	\$ 21,968	\$ 65,406	\$ 84,359
General and administrative	4,700	4,545	14,112	14,274
Total operating expenses	25,813	26,513	79,518	98,633
Loss from operations	(25,813)	(26,513)	(79,518)	(98,633)
Other income (expense)				
Interest income	812	1,639	2,987	6,125
Interest expense	(999)	(1,068)	(3,162)	(3,137)
Change in fair value of embedded derivatives	650	(440)	950	(290)
Other expense, net	(19)	(69)	(81)	(262)
Total other income	444	62	694	2,436
Net loss	(25,369)	(26,451)	(78,824)	(96,197)
Other comprehensive gain (loss)				
Unrealized (loss) gain on marketable securities	(12)	86	(108)	157
Comprehensive loss	\$ (25,381)	\$ (26,365)	\$ (78,932)	\$ (96,040)
Net loss per common share, basic and diluted	\$ (0.35)	\$ (0.44)	\$ (1.14)	\$ (1.59)
Weighted-average shares outstanding, basic and diluted	72,321,459	60,573,425	69,297,155	60,557,668



Acumen Pharmaceuticals, Inc.
Condensed Statements of Cash Flows
(in thousands)
(unaudited)

	Nine Months Ended September 30,	
	2026	2025
Cash flows from operating activities		
Net loss	\$ (78,824)	\$ (96,197)
Adjustments to reconcile net loss to net cash used in operating activities:		
Depreciation	35	46
Stock-based compensation expense	7,626	7,433
Amortization of premiums and accretion of discounts on marketable securities, net	(198)	(919)
Change in fair value of embedded derivatives	(950)	290
Amortization of right-of-use asset	104	93
Realized gain on marketable securities	(6)	(17)
Noncash interest expense	1,048	942
Changes in operating assets and liabilities:		
Prepaid expenses and other current assets	(1,143)	1,205
Other long-term assets	11	42
Accounts payable	5,502	(4,966)
Accrued clinical trial expenses	(2,157)	(5,460)
Accrued expenses and other liabilities	96	1,066
Net cash used in operating activities	(68,856)	(96,442)
Cash flows from investing activities		
Purchases of marketable securities	(40,823)	(38,056)
Proceeds from maturities and sales of marketable securities	78,266	145,827
Purchases of property and equipment	—	(88)
Net cash provided by investing activities	37,443	107,683
Cash flows from financing activities		
Proceeds from private placement, net of offering costs	35,441	—
Repayments of debt	(4,987)	—
Proceeds from exercise of stock options	194	37
Repurchase of common shares to pay employee withholding taxes	(149)	(73)
Net cash provided by (used in) financing activities	30,499	(36)
Net change in cash and cash equivalents and restricted cash	(914)	11,205
Cash and cash equivalents and restricted cash at the beginning of the period	54,220	35,859
Cash and cash equivalents and restricted cash at the end of the period	\$ 53,306	\$ 47,064



Corporate Presentation

October 2026

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. Any statement describing Acumen's goals, expectations, financial or other projections, intentions or beliefs is a forward-looking statement and should be considered an at-risk statement. Words such as "believes," "expects," "anticipates," "could," "would," "seeks," "aims," "plans," "potential," "will" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Forward-looking statements include statements concerning Acumen's business, and Acumen's ability to achieve its strategic and financial goals, including its projected use of cash, cash equivalents and marketable securities and the expected sufficiency of its cash resources into early 2027, the therapeutic potential of Acumen's product candidate, sabirnetug (ACU193), including against other antibodies, the timing of anticipated topline results of ALTITUDE-AD, the potential for additional development to support a subcutaneous dosing option of sabirnetug, and the potential to develop a candidate to treat Alzheimer's Disease utilizing Enhanced Brain Delivery™ (EBD™) technology. These statements are based upon the current beliefs and expectations of Acumen management, and are subject to certain factors, risks and uncertainties, particularly those inherent in the process of discovering, developing and commercializing safe and effective human therapeutics. Such risks may be amplified by the impacts of the COVID-19 pandemic. These and other risks concerning Acumen's programs are described in additional detail in Acumen's filings with the Securities and Exchange Commission ("SEC"), including in Acumen's most recent Annual Report Form 10-K and future filings and reports by Acumen. Copies of these and other documents are available from Acumen. Additional information will be made available in other filings that Acumen makes from time to time with the SEC. These forward-looking statements speak only as of the date hereof, and Acumen expressly disclaims any obligation to update or revise any forward-looking statement, except as otherwise required by law, whether, as a result of new information, future events or otherwise. In this presentation, references to cash also include cash equivalents.

Advancing Next Generation Treatments for Early Alzheimer's Disease (AD)

Targeting Toxic Amyloid Beta Oligomers (A β Os)



With passion, expertise, and perseverance, we are forging a path toward innovative treatments that preserve quality time for all people impacted by Alzheimer's and other neurodegenerative diseases.



Large and growing market in need of additional treatment options



Sabirnetug (ACU193): monoclonal antibody (mAb) **highly selective for toxic A β Os** with positive Phase 1 clinical trial results in AD patients; **Phase 2 (IV) topline results expected November 2026**



Enhanced Brain Delivery™ (EBD®) program to develop oligomer-targeted antibodies with BBB-penetrating technology; **pre-clinical candidate (PCC) data announced early 2026 & lead candidate IND targeted for mid-2027**



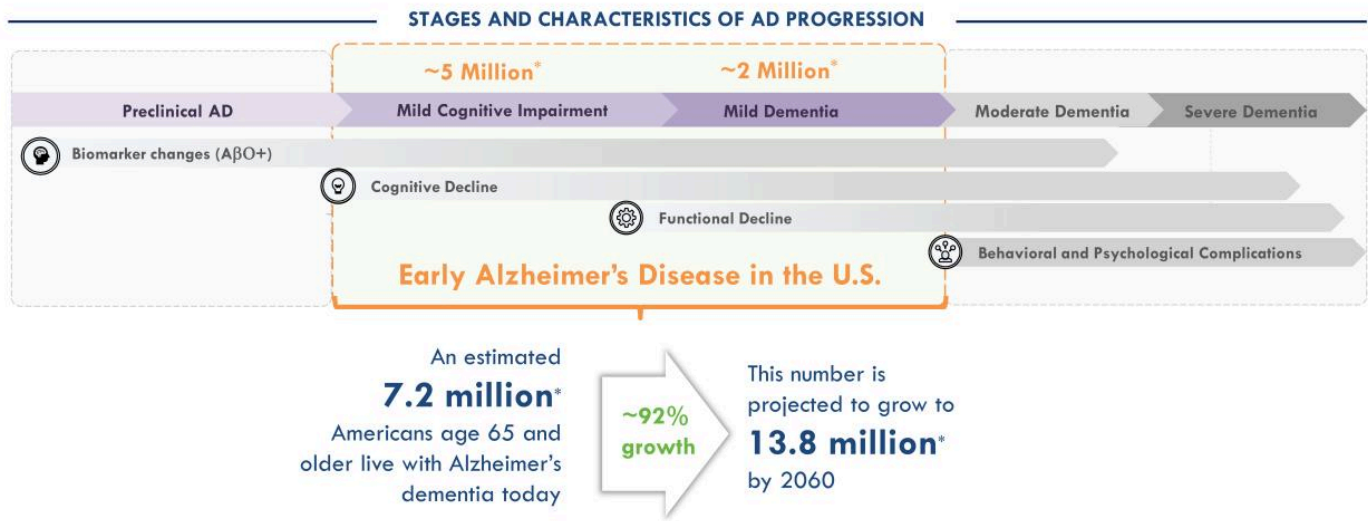
Experienced leadership team with extensive AD drug development experience



Strong balance sheet supporting clinical development plans for sabirnetug (~\$79M at 9/30/26)

BBB: blood-brain-barrier

Early Alzheimer's Disease (AD) Patient Population Represents Significant and Growing Market; Preclinical AD a Potential Future Addressable Population



*Alzheimer's Association

AD Treatment Landscape Continues to Evolve

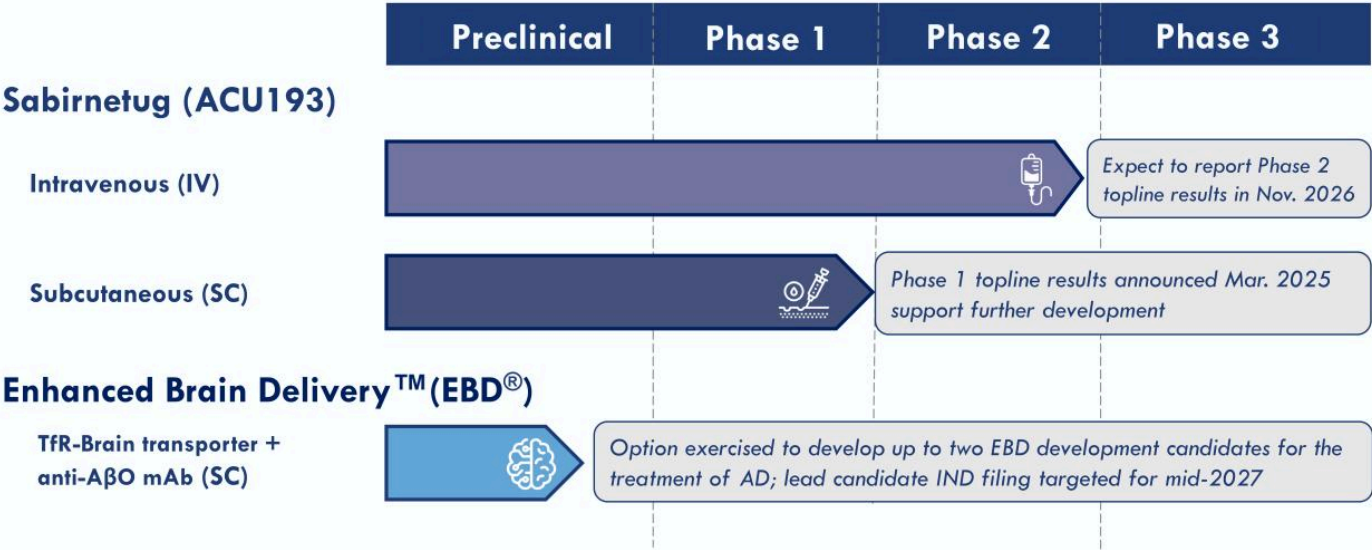
Approved Therapies Establish Momentum and Next-generation Approaches Expand Impact



*ARIA-E: Amyloid-related imaging abnormalities - edema

2026 a Pivotal Year for Acumen's Pipeline

Sabirnetug Global Phase 2 Study Results & Next Generation EBD Candidate Selection



Acumen Leadership Team

Experienced in AD/Neuro Drug Development



DANIEL O'CONNELL
Chief Executive Officer
ACUMEN
neuroVentures



JAMES DOHERTY, PHD
President &
Chief Development Officer
ACUMEN
Sage Therapeutics AstraZeneca



ERIC SIEMERS, MD
Chief Medical Officer
ACUMEN
Lilly



MATT ZUGA
Chief Financial Officer &
Chief Business Officer
ACUMEN
HIGHCAPE PARTNERS



RUSSELL BARTON
Chief Operating Officer
ACUMEN
Lilly



LIANE SCHENK
SVP, Head of CMC
ACUMEN
Lilly Lonza
NOVAVAX



LAURA ROSEN, MD, PHD
SVP, Clinical Development
ACUMEN
Takeda Shire
MERCK



AMY SCHACTERLE, PHD
Chief Regulatory Officer,
Head of Quality
ACUMEN
Sage Therapeutics
sunovion



SIEW TIN GAN
Head of Clinical
Operations
ACUMEN
Lundbeck Takeda



JASNA JERECIC, PHD
Disease Area Strategy Lead
ACUMEN



PAUL SHUGHRUE, PHD
VP, Research & Strategy
ACUMEN
MERCK prothena
Allergan



DEREK MEISNER, JD
Chief Legal Officer
ACUMEN
X4



JULIE BOCKENSTETTE
Chief People Officer
ACUMEN
Roche Lilly

Acumen team has decades of experience in Alzheimer's drug discovery and development

Milestones Achieved and Upcoming

MILESTONES	TIMING	STATUS
Initiation of ALTITUDE-AD Phase 2 trial	2Q2024	✓
Completion of enrollment of ALTITUDE-AD	1Q2025	✓
Phase 1 subcutaneous topline results	1Q2025	✓
EBD [®] non-clinical data package	Early 2026	✓
ALTITUDE-AD topline results	November 2026	☐
EBD lead candidate IND filing	Mid-2027	☐

~\$79M

Cash, cash equivalents and
marketable securities as of
Sept. 30, 2026

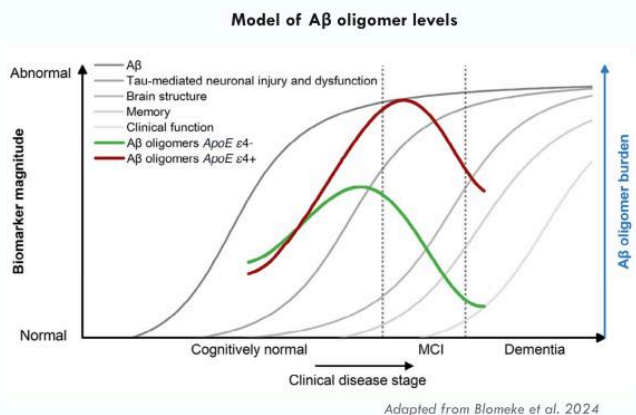
Acumen expects its cash runway to extend into early 2027

Amyloid Beta Oligomers in AD

Sabirnetug (ACU193): monoclonal antibody (mAb)
highly selective for toxic A β O_s

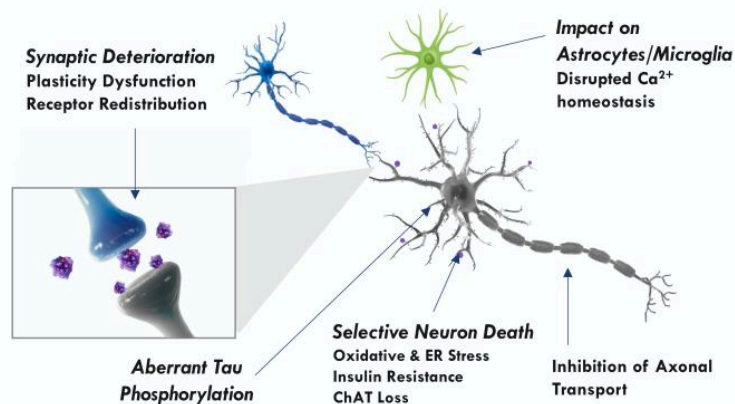


Soluble A β O_s Contribute to Pathophysiological Processes Associated with Alzheimer's Disease



- Soluble A β forms appear early in the course of disease pathophysiology
- Production of toxic soluble A β persists after plaque removal

- Toxic consequences of soluble A β oligomer production include synapse dysfunction and loss, tau hyperphosphorylation, immune cell activation and functional impairment
- Reduced neuronal toxicity and intervention at the synaptic level may prevent irreversible neuronal cell death



Sabirnetug: Potential Next Generation Immunotherapy for Early AD

Designed for Improved Efficacy & Safety

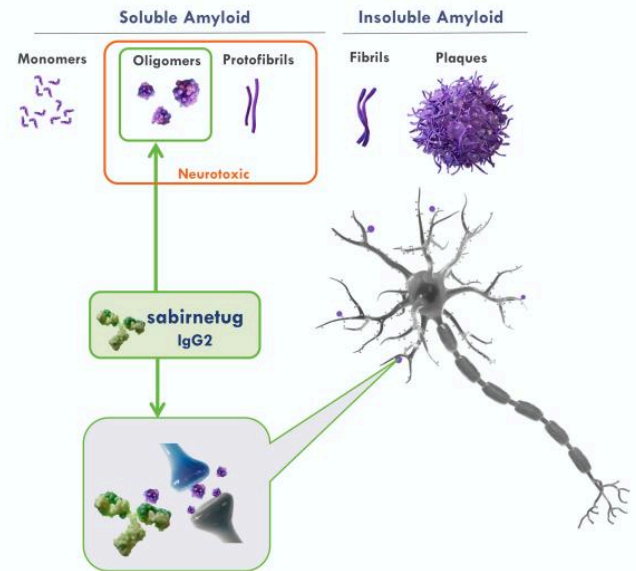
- Humanized, affinity matured mAb developed to target toxic A β oligomers
- IgG2 subclass mAb with reduced effector function

Regulatory Interactions

- FDA Fast Track designation for the treatment of early AD
- Phase 2 implemented as a registration-quality study

Positive Ph1 in AD Patients & Encouraging Ph2 Enrollment

- Successful Phase 1 exclusively in early AD patients
 - ✓ Safety, target engagement, biomarker effects
- Phase 2 (n=542) enrollment complete in March 2025; topline results expected November 2026



Sabirnetug

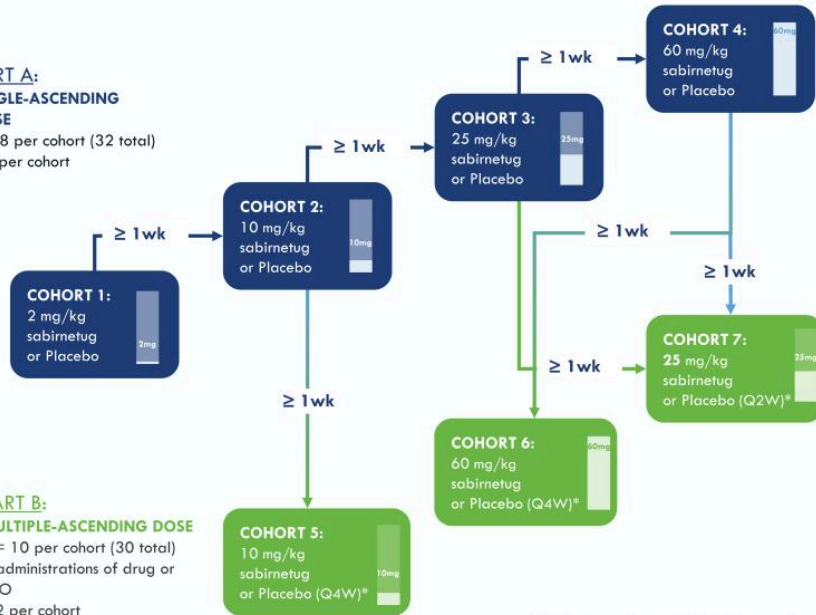


INTERCEPT-AD: A Randomized Placebo Controlled Phase 1 in Early AD Patients

PART A:

SINGLE-ASCENDING DOSE

n = 8 per cohort (32 total)
6:2 per cohort



PART B:

MULTIPLE-ASCENDING DOSE

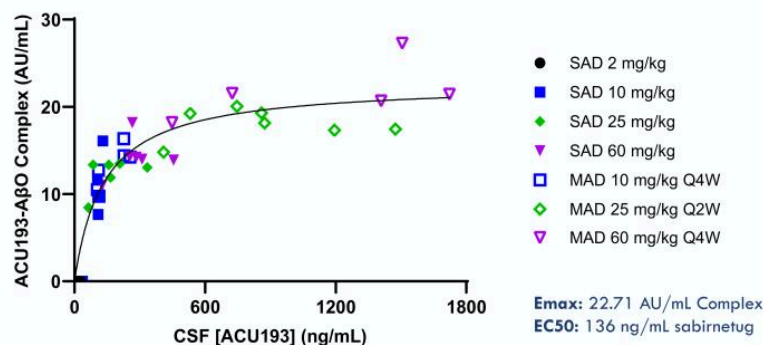
n = 10 per cohort (30 total)
3 administrations of drug or
PBO
8:2 per cohort



Q2W: Dosing every two weeks; Q4W: Dosing every four weeks.

Doses Approaching Maximal Target Engagement Support Sabirnetug A β O Mechanism and Helped Guide Dose Selection for Next Study Phase

Single & Multiple Dose Cohorts - Exposure Response Relationship (Emax Model)



Sabirnetug-A β O Complex

- Acumen developed a novel assay to measure the complex of A β O bound to sabirnetug in cerebrospinal fluid
- Observed target engagement with oligomers that increased across a range of doses
- Achieved saturation point between 25 mg/kg and 60 mg/kg

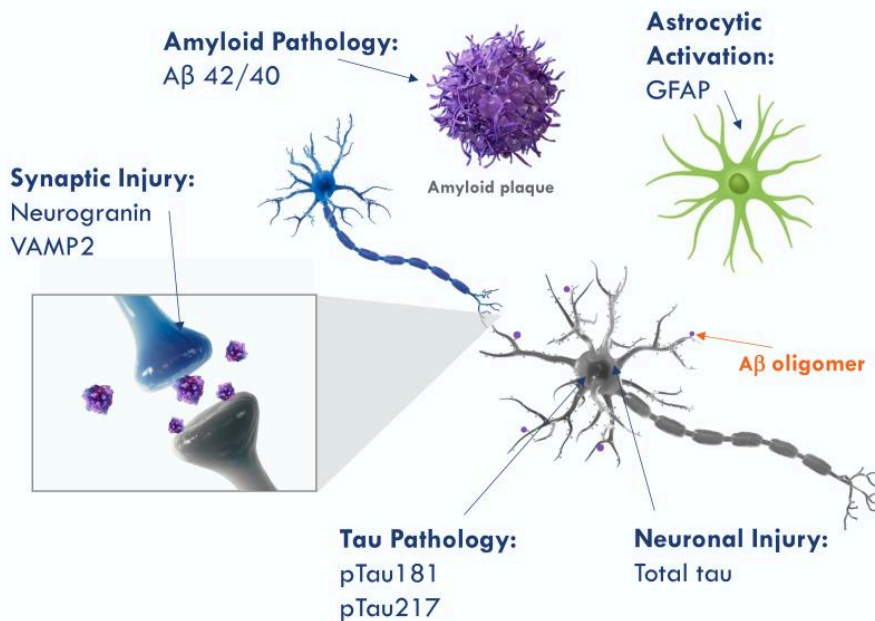
*One patient from Cohort 5 (10 mg/kg Q4W) excluded because only received one administration of drug (study drug discontinued after lacunar infarct).

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

Importance of Key Fluid Biomarkers Associated with AD Pathology

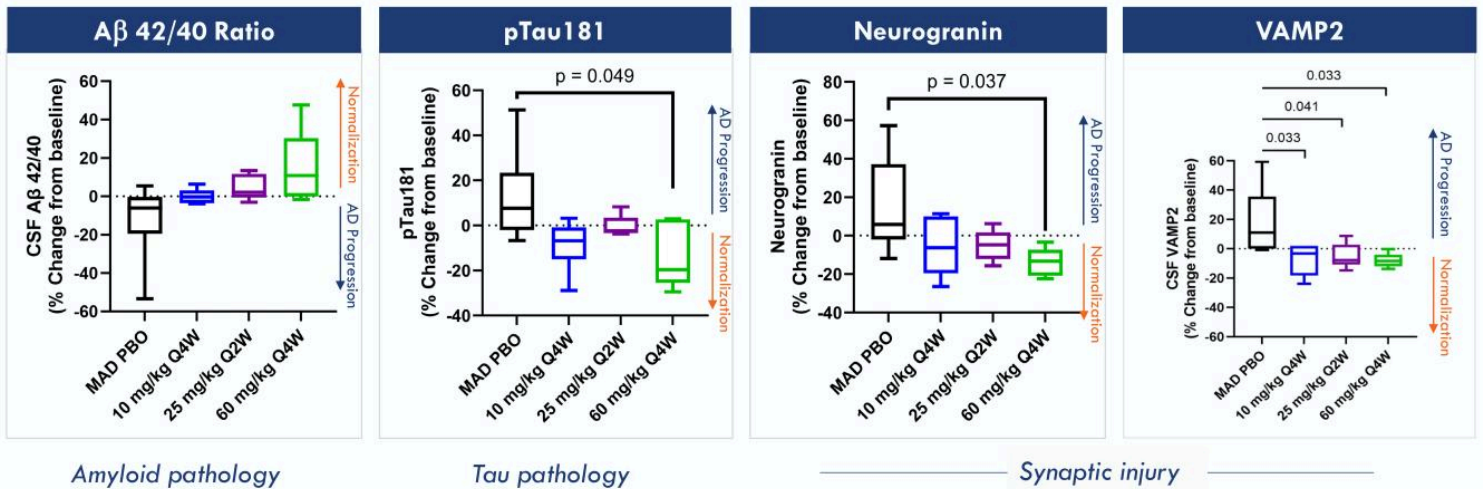
- Biomarkers from cerebrospinal fluid and plasma capture neuronal, synaptic, and axonal injury and reflect the cumulative outcome of different pathological substrates in AD¹
- Evidence suggests that biomarkers are likely to be better predictors of the underlying pathology of AD than imaging alone²

- **After just three administrations of sabirnetug, patients with early AD demonstrated improvements in biomarkers associated with AD pathology**



1. Tarawneh, R. Biomarkers: Our Path Towards a Cure for Alzheimer Disease. Biomarker Insights Volume 15: 1–15. 2020; 2. Blennow K, Zetterberg H. The Past and the Future of Alzheimer's Disease Fluid Biomarkers. J Alzheimer's Dis. 2018;62(3):1125-1140.

Consistent Improvement in CSF Amyloid, Tau and Synaptic Biomarkers Indicate Downstream Pharmacology of Sabirnetug After Only Three Doses



E. Cline, et al, Biofluid biomarker changes following treatment with sabirnetug (ACU193) in INTERCEPT-AD, a phase 1 trial in early Alzheimer's disease, JPAD 2025.

n = 8 subjects/treated group; 6 subjects in pooled placebo (PBO); p-values from unpaired, 2-sided Student's t test

Sabirnetug Demonstrates Potential for Best-in-Class Safety Profile

Compelling Overall Safety Profile, with Low Incidence of ARIA-E in the INTERCEPT-AD study

INTERCEPT-AD Phase 1 Safety Data

5

Total ARIA-E cases,
or ~10%

0

Cases of ARIA-E in
ApoE4 homozygotes
N=6

0

Deaths, SAEs Related
to Study Drug

✓ **Limited incidence of ARIA-E**

- 10 mg/kg Q4W: 1 asymptomatic case
- 25 mg/kg Q2W: 1 asymptomatic case
- 60 mg/kg Q4W: 2 asymptomatic cases; 1 symptomatic case

✓ **No ARIA-E observed in ApoE4 homozygotes (n=6), despite comprising 13% of study**

- Differentiated from other antibodies that have ARIA-E rates ~30% to ~40% in participants who are E4-homozygotes

✓ **Broad therapeutic index** with convenient monthly dosing

- Safety profile may support attractive benefit/risk option for large portion of patients

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

ApoE4: ε4 allele of Apolipoprotein E

INTERCEPT-AD Phase 1 Data Support Potential for Sabirnetug to Offer Next Generation Efficacy and Safety

Key Takeaways from INTERCEPT-AD

Potential for Differentiated Efficacy

- ✓ First mAb to demonstrate selective target engagement of A β O $_2$ s (most toxic form of A β)
- ✓ Rapid, meaningful plaque reduction comparable to the current market front-runners at similar timepoints
- ✓ Improvement of AD biomarkers in CSF and plasma are a strong indication of downstream effects

Potential for Differentiated Safety

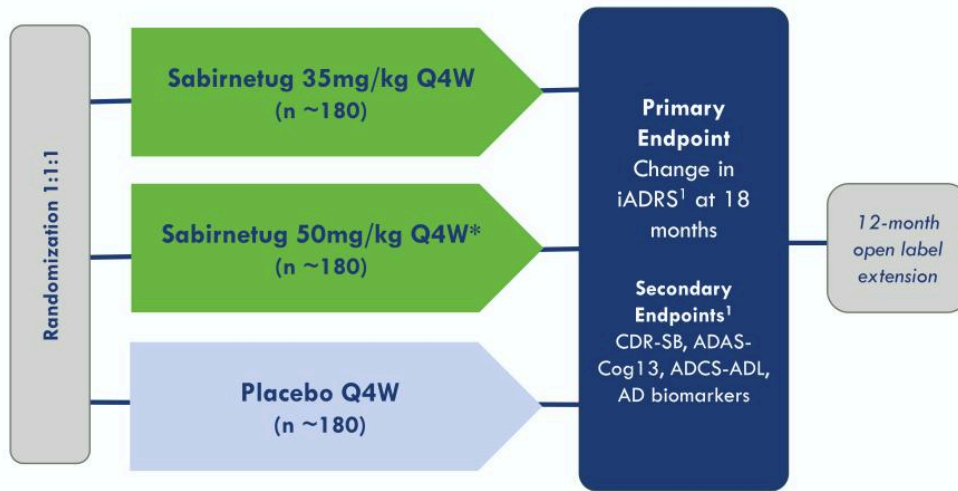
- ✓ Compelling safety profile with low incidence of ARIA-E
- ✓ Absence of ARIA-E observed in ApoE4 homozygotes
- ✓ Broad potential therapeutic index with convenient monthly dosing



ALTITUDE-AD Phase 2 Study Results Expected in Late 2026

Objective: To evaluate the clinical efficacy, safety and tolerability of sabirnetug

Patient population: Patients with early AD (MCI or mild dementia due to early AD)



*Titration of sabirnetug 35mg/kg Q4W for two doses.

Key Highlights

- 542-participant study fully enrolled in March 2025 (U.S., Canada, U.K., Germany, Spain)
- Open label extension portion of ALTITUDE-AD initiated in November 2025
- Topline results expected in November 2026

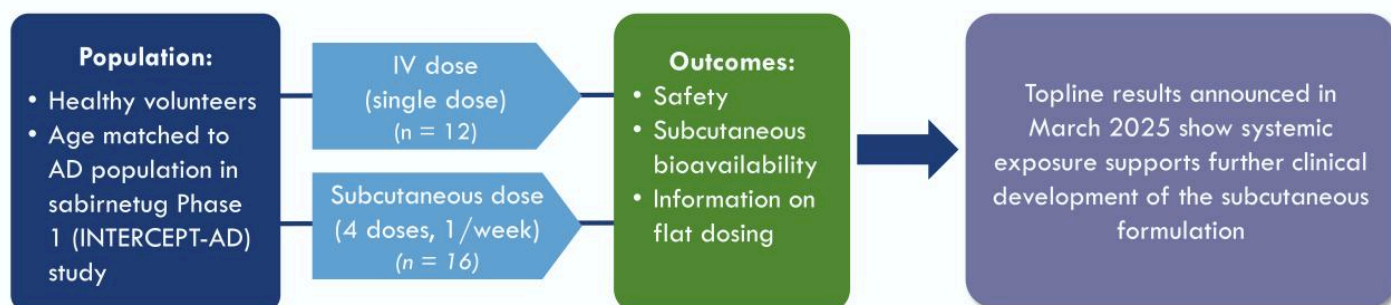
1. iADRS: Integrated Alzheimer's Disease Rating Scale; CDR-SB: Clinical Dementia Rating – Sum of Boxes; ADAS-cog: Alzheimer's Disease Assessment Scale – Cognitive Subscale; ADCS-ADL: Alzheimer's Disease Cooperative Study – Activities of Daily Living

Subcutaneous Formulation Well-Tolerated in Phase 1 Healthy Volunteer Study

Potential to Dose Once-Weekly with Single Injection to Broaden Patient Access and Increase Treatment Convenience

Phase 1 Subcutaneous Healthy Volunteer Study

Phase 1 study to compare the pharmacokinetics of subcutaneous form of sabirnetug to the IV form



Announced partnership with Halozyne in November 2023 to develop subcutaneous dosing option for sabirnetug using Halozyne's drug delivery technology, ENHANZE®

Sabirnetug IP & Market Exclusivity

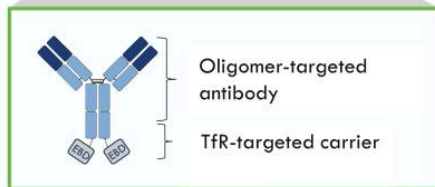
- Exclusive, perpetual, irrevocable, worldwide, royalty-free license from Merck to its Amyloid Derived Diffusible Ligand (ADDL) IP including issued sabirnetug patents
- Sabirnetug Global IP estate:
 - ✓ Issued patents in 19 countries
 - ✓ Composition of matter patents and methods of use run into July 2031
 - ✓ Patent term extensions may be available, 3-5 years depending on jurisdiction
- Biologics market exclusivity is expected for sabirnetug as a novel biologic drug
 - ✓ US provides 12 years market exclusivity for novel biologics
 - ✓ Europe provides 10 years of market exclusivity for novel biologics
- EBD program includes Acumen wholly-owned A β O-selective mAbs expected to generate novel IP including Composition of Matter Patent(s)

Enhanced Brain Delivery™ (EBD®)



Aim to Widen the Therapeutic Window to Increase Efficacy, Safety, and Convenience

Aim to Widen the Therapeutic Window to Increase Efficacy, Safety, and Convenience



Opportunities:

- Increased brain exposure may enhance efficacy
- Wider capillary distribution may reduce ARIA-E risk
- Lower dose volumes enable convenient subcutaneous administration



- ✓ Collaboration, option and license agreement announced in July 2025 with JCR Pharmaceuticals
- ✓ Acumen holds exclusive option to license and develop up to two candidates under terms of agreement

Image courtesy of JCR Pharmaceuticals

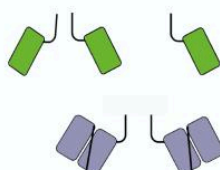
Acumen's EBD May Improve Efficacy, Safety and Delivery Compared to Non-A β O Selective Antibodies

Differentiated Cargo (A β O-selective mAb)



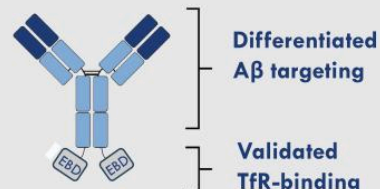
- **Targets synaptotoxic A β O species**
- **Robust fluid biomarker results** in Phase 1b INTERCEPT-AD trial
- Demonstrated **low ARIA-E** overall in Phase 1b, including no ARIA-E observed in APOE 4/4 carriers
- **Low rate of infusion related reactions** observed to date clinically
- Candidate mAbs: ACU193 and ACU234

Validated BBB Carrier Technology (TfR-targeting)



- **Enhance brain penetration** versus native antibody to enable low-volume SC delivery
- **Potential for lower ARIA rate** due to TfR prevalence on capillary bed
- **Little to no anemia** observed with JCR technologies

Acumen EBD Candidates



Enhanced efficacy: Leverage improved brain delivery/distribution and A β oligomer targeting



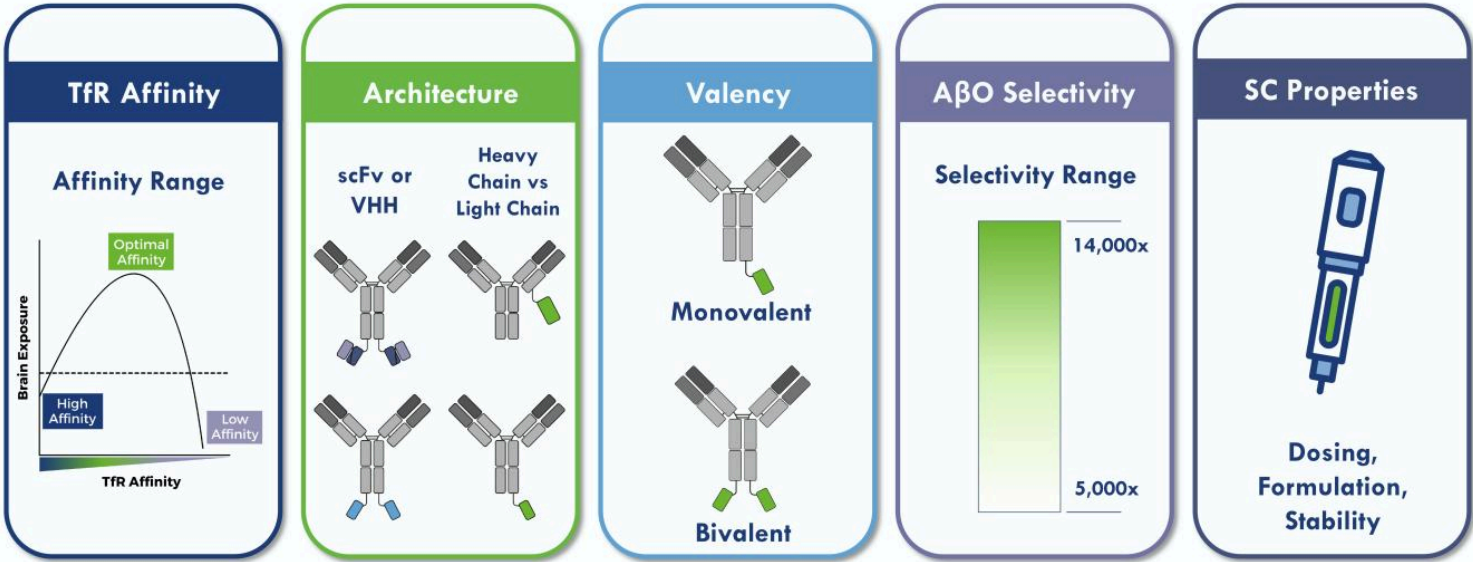
Best-in-class safety: Lower ARIA, anemia, IRRs



Designed for SC administration: Low dose, formulation, stability

ApoE4: ϵ 4 allele of Apolipoprotein E

Acumen Explored Broad Parameters to Develop a TfR-Mediated AβO Product



TfR: transferrin receptor; scFv: single chain variable fragment antibodies; VHHs: variable heavy domain antibodies; SC: subcutaneous

Phase 1 Results Provide Foundation for EBD Program



Half-life/Dosing Interval

Once monthly dosing



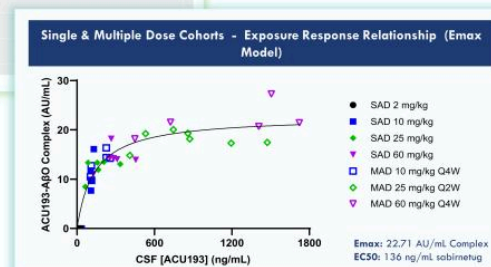
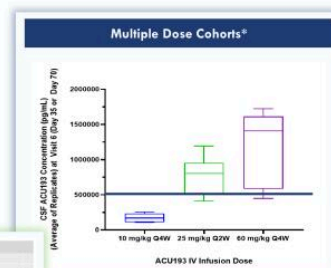
Dosing Exposure

35 mg/kg and 50 mg/kg in Phase 2 IV



Target Engagement

Maximal TE of A β O_s observed



*One patient from Cohort 5 (10 mg/kg Q4W) excluded because only received one administration of drug (study drug discontinued after lacunar infarct).

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabinmetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.



Mouse Surrogate Antibodies Developed to Explore EBD and Target Engagement in a Mouse Model of Alzheimer’s Disease

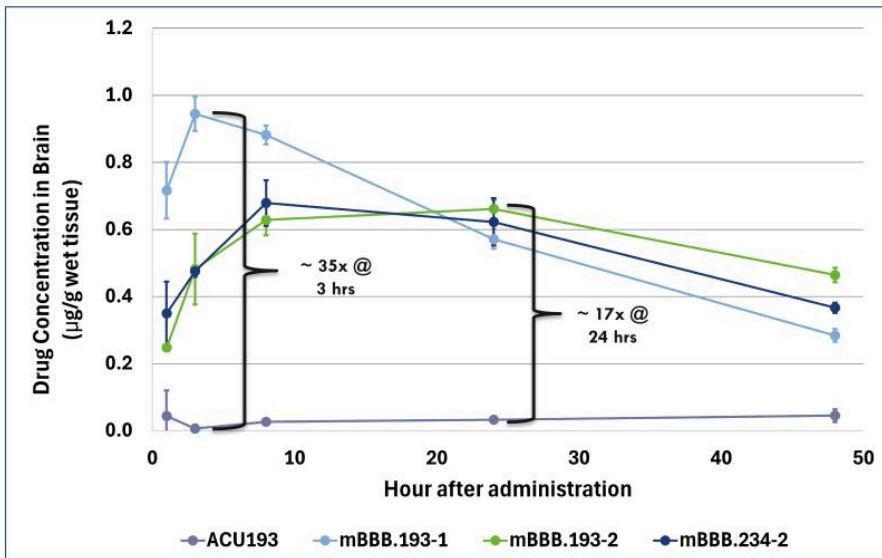
No.	mBBB.193-1	mBBB.193-2	mBBB.234-2	ACU193
Candidate Molecules	ACU193 bivalent mscFv #1	ACU193 bivalent mscFv #2	ACU234 bivalent mscFv #2	
Affinity to mTfR*	0.33 nM	5.34 nM	3.52 nM	0 nM
Oligomer Binding	0.60 nM	0.45 nM	2.48 nM	0.319 nM *
Monomer Binding	6.91 µM	5.87 µM	8.60 µM	3.76 µM

Cline et al., CTAD, 2025
 *Cline et al., AAIC, 2025

The fusion of anti-oligomer antibodies and J-Brain Cargo (anti-TfR scFv) did not alter the ability of constructs to bind TfR or alter the preferential binding to AβOs

mTfR: murine transferrin receptor; mscFv: murine single chain variable fragment antibodies

In Wild-Type Mice, All EBD Fusion Proteins Showed Increased Brain Exposure Compared to Sabirnetug (ACU193)



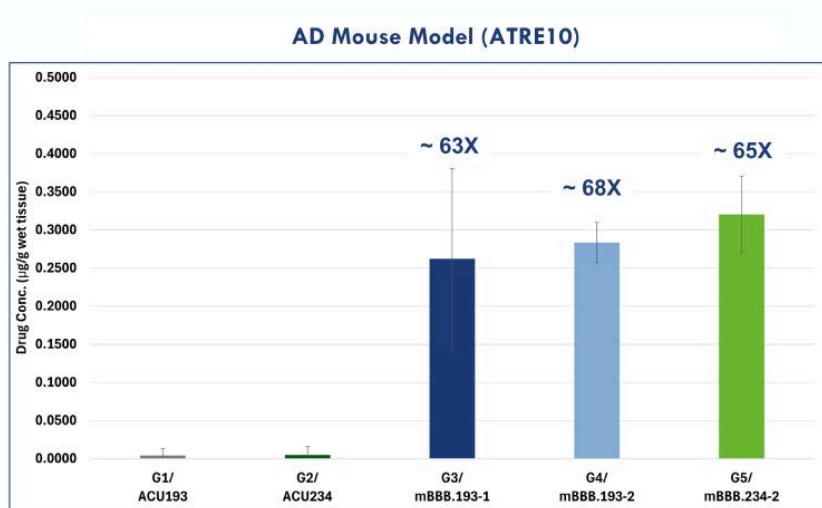
Animals dosed IV at 2 mpk

Cline et al., CTAD, 2025

- Higher brain exposure was observed for all fusion proteins compared to ACU193:
 - mBBB.193-1 exhibited **~35-fold higher** brain exposure at 3 hours
 - mBBB.193-2 and mBBB.234-2 exhibited **~17-fold higher** brain exposure at 24 hours
- mBBB.193-2 and mBBB.234-2 showed the greatest cumulative exposure:
 - **32- to 55-fold higher AUC** than ACU193

Surrogate Antibodies Show Enhanced Delivery to the AD Mouse Brain, when Compared with ACU193 and ACU234

- The brain exposure of the three fusion proteins was **63- to 68-fold higher** compared to ACU193 and ACU234
- Higher concentrations of all three fusion proteins seen in AD mouse brain compared to wild-type mice may be due to target engagement of A β O_s and retention in brain



Animals dosed at IV 2 mpk

Cline et al., CTAD, 2025

Robust Data Package Supporting EBD Construct Selection

Inclusive of NHP Primate Data



TfR Affinity



Stability &
Manufacturability



Pharmacokinetics



Brain Distribution



A β Oligomer Binding



Anemia Risk

Key Takeaways from NHP Study Results



✓ Enhanced Brain Penetration

EBD candidates achieved 14-40x higher brain levels in non-human primates compared to native antibodies 24 hours after dosing



✓ Low Anemia Risk

- Hematology data in non-human primates indicate low potential for anemia: At 24 hours after SC dosing, EBD candidates demonstrated no observed change in red blood cell count, hematocrit, hemoglobin or reticulocyte count
- No adverse events observed



✓ Subcutaneous Dosing Capability

Favorable stability profile and enhanced brain delivery support a path to subcutaneous administration with low-volume devices

Lead clinical candidate IND filing targeted for 2027

Acumen Positioned to Deliver Potential Next-Gen Treatment for Early AD

Key Takeaways

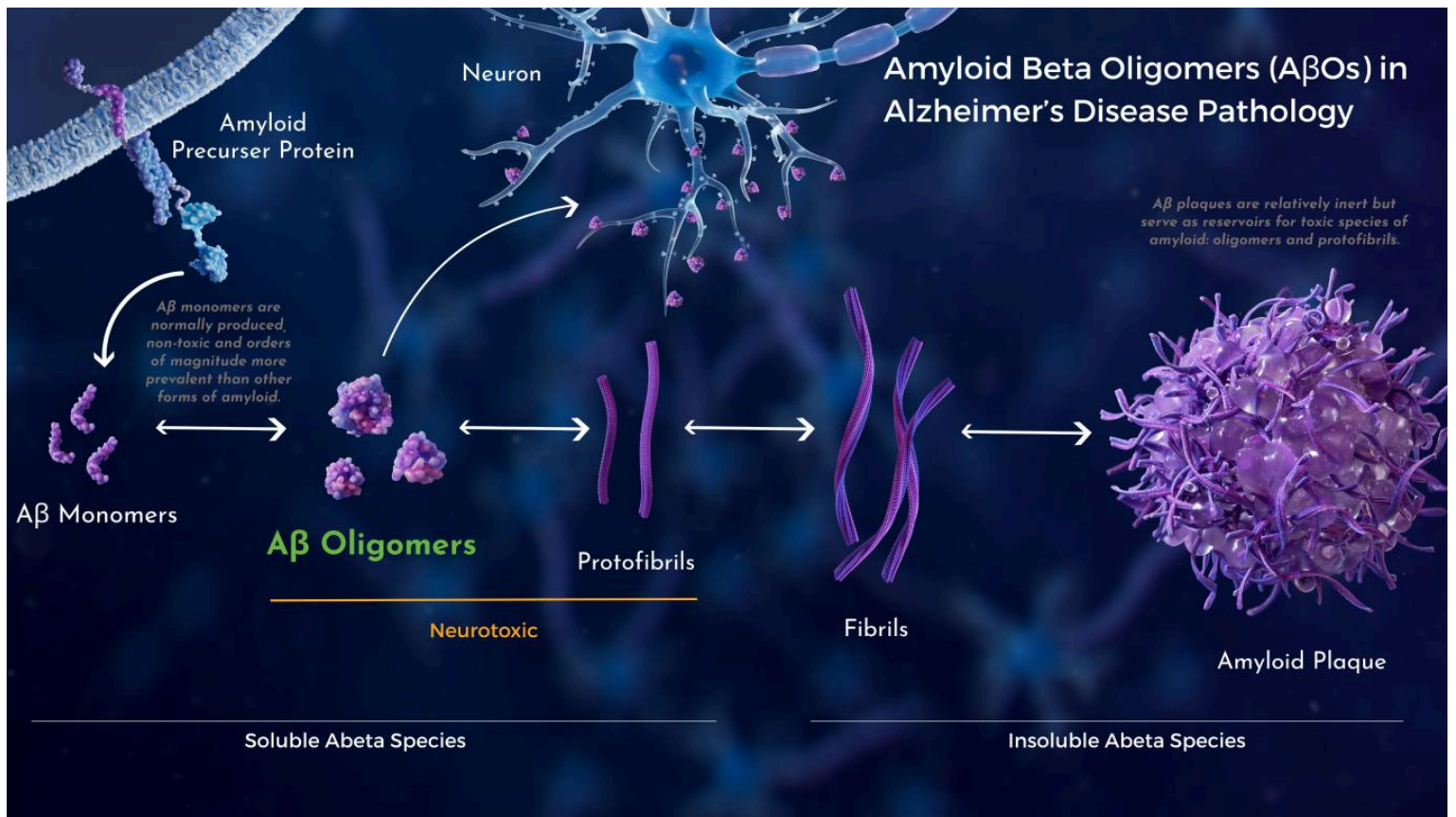
- ✓ **Significant and growing Alzheimer's population** in need of additional treatment options
- ✓ **Synaptotoxic A β O**s appear early in Alzheimer's Disease and contribute to its pathophysiological processes; sabirnetug demonstrates **high selectivity for A β O**s in AD patients
- ✓ **Positive Phase 1 data** strengthen potential for sabirnetug to offer **next-generation** efficacy and safety
- ✓ Significant interest in ALTITUDE-AD, a **Phase 2 study** investigating sabirnetug, evidenced by **rapid enrollment**
- ✓ **EBD[®]** program augments **portfolio optionality**, leveraging Acumen capabilities and assets

Next Steps

- ✓ EBD program pre-clinical candidate (PCC) data announced in **early 2026**
- ☐ Topline results from ALTITUDE-AD Phase 2 IV study evaluating the clinical efficacy and safety of sabirnetug in patients with MCI or mild dementia due to Alzheimer's expected in **November 2026**
- ☐ Lead clinical candidate IND filing for EBD program targeted for **mid-2027**

Appendix

Selection of Non-clinical Sabirnetug Data



Literature Selection: Soluble A β O_s Contribute to Pathophysiological Processes Associated with Alzheimer's Disease

Synapse deterioration

Zhao et al, 2006
Lacor et al, 2007
Shankar et al, 2007
Wu et al, 2010
Brito-Moreira et al, 2017
Actor-Engel et al, 2021
Sackmann & Hallbeck, 2020
Limegrover et al, 2021

Plasticity dysfunction

Lambert et al, 1998
Walsh et al, 2002
Wang et al, 2002
Townsend et al, 2006
Yasumoto et al, 2019

Receptor

Redistribution

Snyder et al, 2005
Roselli et al, 2005
Lacor et al, 2007
Zhao et al, 2008

Aberrant Tau

phosphorylation
De Felice et al, 2008
Ma et al, 2009
Tomiyama et al, 2010
Zempel et al, 2010
Bloom, 2014
Forný-Germano et al, 2020
Wakeman et al, 2022
Darricau et al, 2023

Impact on

astrocytes/microglia

Hu et al, 1998
Jimenez et al, 2008
Sondag et al, 2009
Tomiyama et al, 2010

Disrupted Ca²⁺ homeostasis

Demuro et al, 2005
De Felice et al, 2007
Alberdi et al, 2010
Wang et al, 2018

Selective

neuron death

Lambert et al, 1998
Kim et al, 2003
Florent et al, 2006
Ryan et al, 2009
Lee et al, 2017
Komura, 2019

Insulin

resistance

Zhao et al, 2008
Zhao et al, 2009
Ma et al, 2009
De Felice et al, 2009

ChAT loss

Heinitz et al, 2006
Nunes-Tavares et al, 2012

Oxidative stress

Longo et al, 2000
Sponne et al, 2003
Tabner et al, 2005
De Felice et al, 2007

ER stress

Resende et al, 2008
Nishitsuji et al, 2009

Inhibition of axonal transport

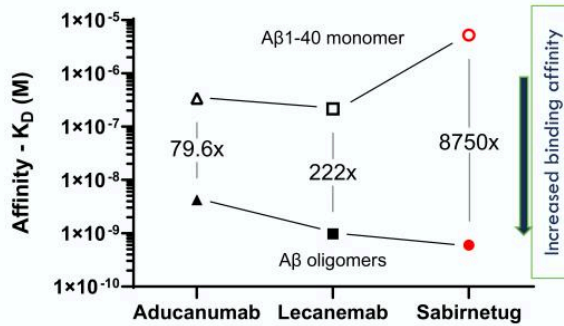
Pigino et al, 2009
Poon et al, 2009
Decker et al, 2010

Differences in the Profile of Sabirnetug Relative to Approved anti-A β DMTs:

Greater A β O selectivity against A β monomers and linear fibrillar species

Greater selectivity against monomeric A β

Sabirnetug vs Lecanemab and Aducanumab

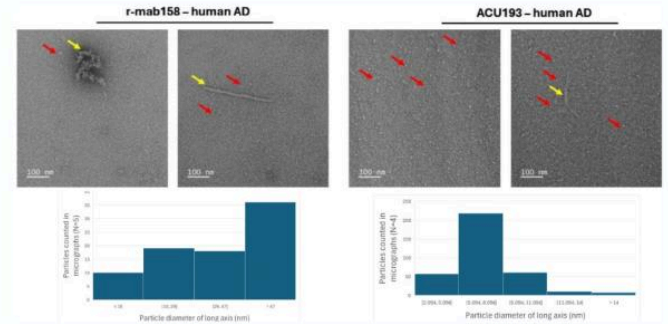


Cline et al, AAIC 2025.

- **lecanemab** → 222x higher affinity to A β O than monomer
- **sabirnetug** → 8750x higher affinity to A β O than monomer

There have been no head-to-head clinical trials between the product candidates listed above. Study designs and protocols for each product candidate were different, and as a result, results may not be comparable between product candidates.

Greater selectivity for soluble A β species isolated from human AD brain

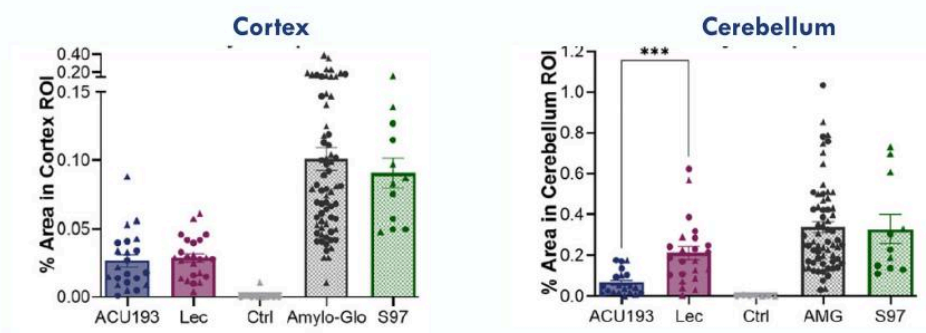


De Leon et al, AAIC 2025.

- Eluates from affinity chromatography using:
 - **r-mab158** → 36% globular, **64% fibrillar**, A β species
 - **sabirnetug** → 99% globular, **1% fibrillar** A β species

Vascular A β Binding Profile in a Murine Model of Vascular CAA

Immunohistochemical comparison of vascular A β binding in the APP:hE4 mouse brain



- Sabirnetug (ACU193) and lecanemab showed **qualitatively similar overall staining** in both cortex and cerebellum
- But sabirnetug had **significantly less cerebellar vascular labeling** than lecanemab ($p = 0.0001$)

Grenon, Martine et al., *Ex vivo comparison of ACU193 and lecanemab reveals binding differences in mouse brain*, *Alzheimer's & Dementia*, 2026.
There have been no head-to-head clinical trials between the product candidates listed above. Study designs and protocols for each product candidate were different, and as a result, results may not be comparable between product candidates.

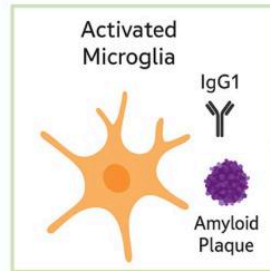
Sabirnetug is an IgG2 Antibody, Which May Lower Risk of ARIA and IRR

Differences in effector coupling may influence inflammatory risk profile

IgG1 antibodies

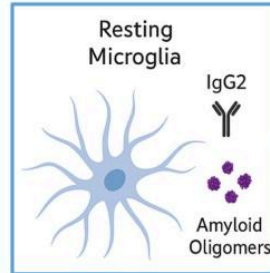
(*lecanemab, donanemab*)

strongly bind Fcγ1 receptors and C1q, triggering complement activation and antibody-dependent cellular phagocytosis (ADCP), causing inflammation and triggering strong cytokine release.



IgG2 antibodies,

like sabirnetug, weakly bind Fcγ1 receptors and avoid complement activation, potentially reducing the risk of inflammation and cytokine release.



Potential Impact on Safety:

- **IgG1-based anti-Aβ DMTs (lecanemab, donanemab) show substantial inflammatory risk, leading to ARIA and hypersensitivity reactions**
 - Including IRRs and anaphylaxis
 - This may limit the doses that can be used to drive efficacy
- **IgG2-based sabirnetug may enable amyloid clearance with reduced inflammation, cytokine release, and ADCP involvement**
 - IgG2 Fc attenuation may lower the frequency and severity of hypersensitivity reactions or ARIA

Maverakis E et al. Glycans in the immune system and The Altered Glycan Theory of Autoimmunity: a critical review. *J Autoimmun.*, 2015.

Wang, J., et al. A systemic view of Alzheimer disease — Insights from amyloid-β metabolism beyond the brain. *Nature Reviews Neurology*, 2017.

Shashidharamurthy R, et al. Dynamics of the interaction of human IgG subtype immune complexes with cells expressing R and H allelic forms of a low-affinity Fcγ receptor CD32A, 2009.

Phase 1 INTERCEPT-AD



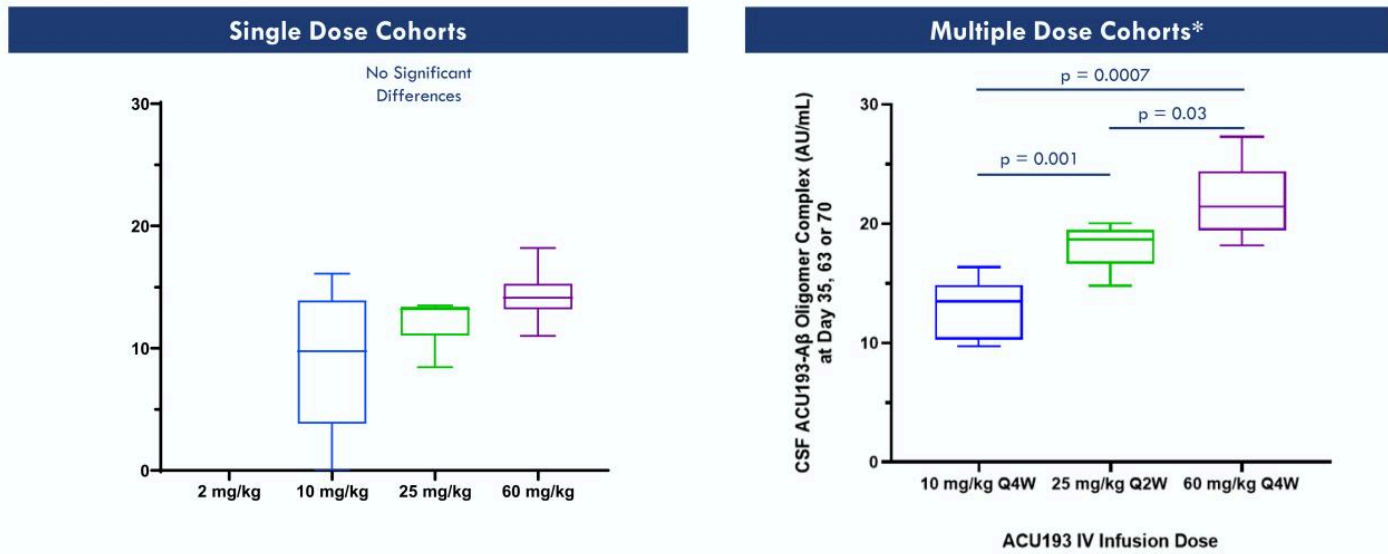
Target Engagement Assessed by Measuring Sabirnetug-A β O Complex in CSF

- Novel assay configuration tailored to selectively detect sabirnetug-A β O complex in CSF as direct measure of target engagement
- Translated for clinical use from a preclinical assay developed by Merck that showed sabirnetug engages target A β Os in transgenic mouse brain (tg2576) in dose dependent manner

MSD S-Plex (Turbo) Immunoassay



Target Engagement of Sabirnetug with A β O $_2$ is Dose Proportional

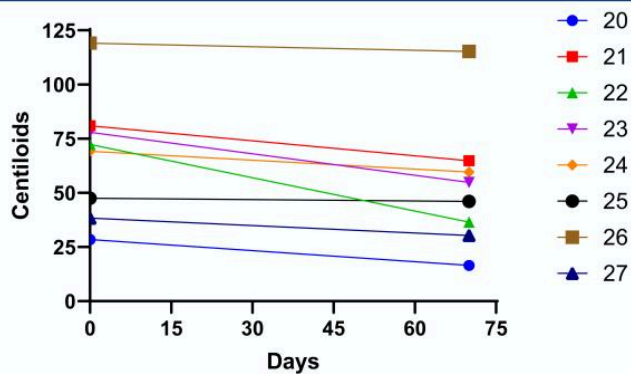


*One patient from Cohort 5 (10 mg/kg Q4W) excluded because only received one administration of drug (study drug discontinued after lacunar infarct).

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

Nearly All Sabirnetug-Treated Patients in High Dose MAD Cohorts Showed Reductions in Plaque Load After Three Doses at 63 or 70 days

25 mg/kg Q2W MAD



Mean reduction in amyloid plaque

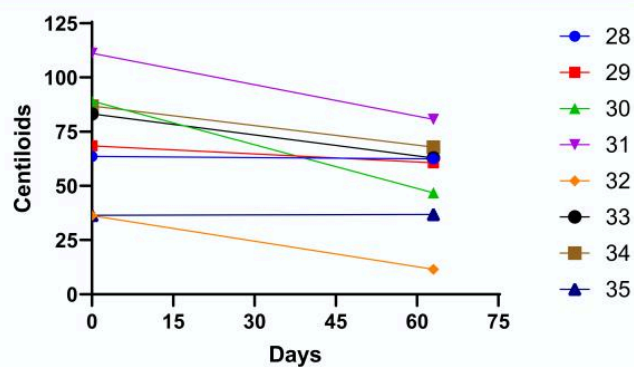
Δ (absolute value, centiloids)

13.7

Δ (% , centiloids)

20.6%

60 mg/kg Q4W MAD



Mean reduction in amyloid plaque

Δ (absolute value, centiloids)

18.1

Δ (% , centiloids)

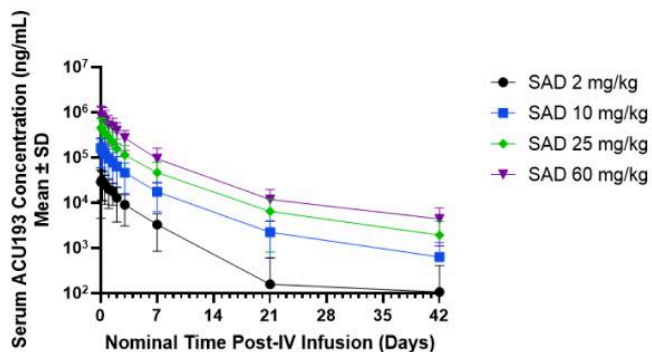
25.6%

Plaque load based on florbetapir PET

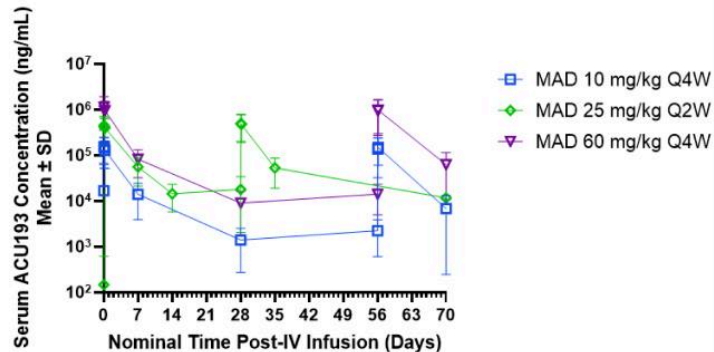
E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

Sabirnetug Serum Exposure is Dose Proportional Without Accumulation

Single Dose Cohorts



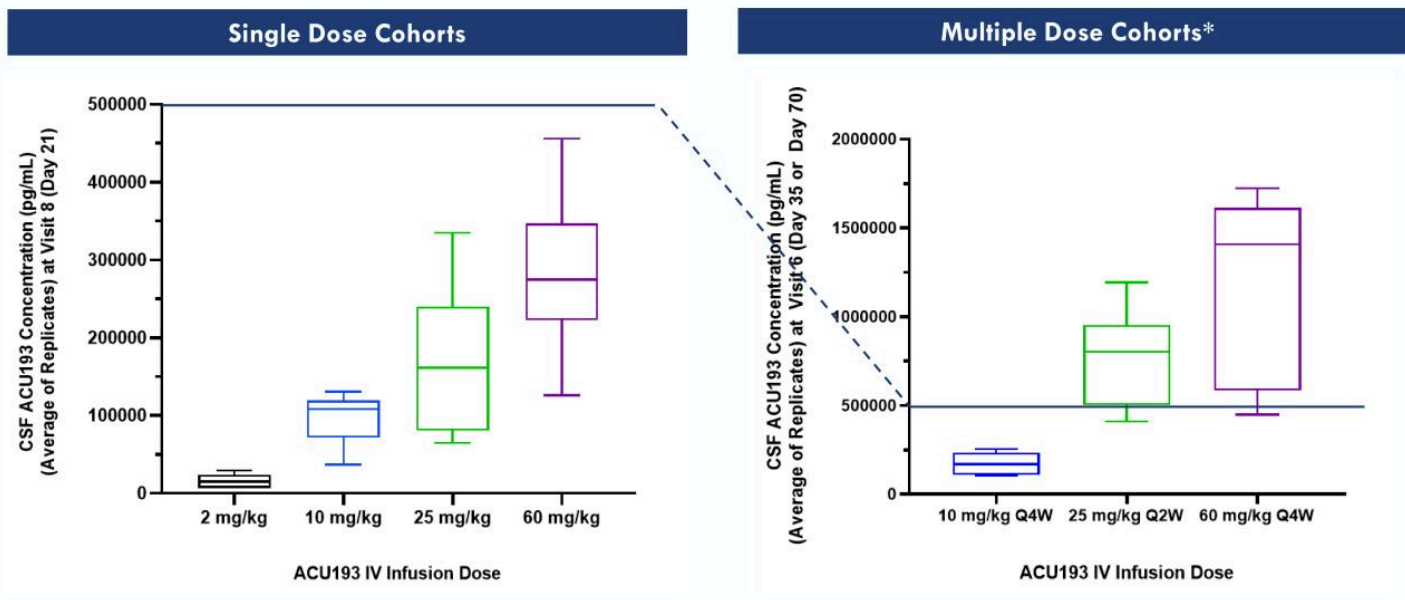
Multiple Dose Cohorts



Estimated serum terminal $T_{1/2}$ of 5-7 days

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

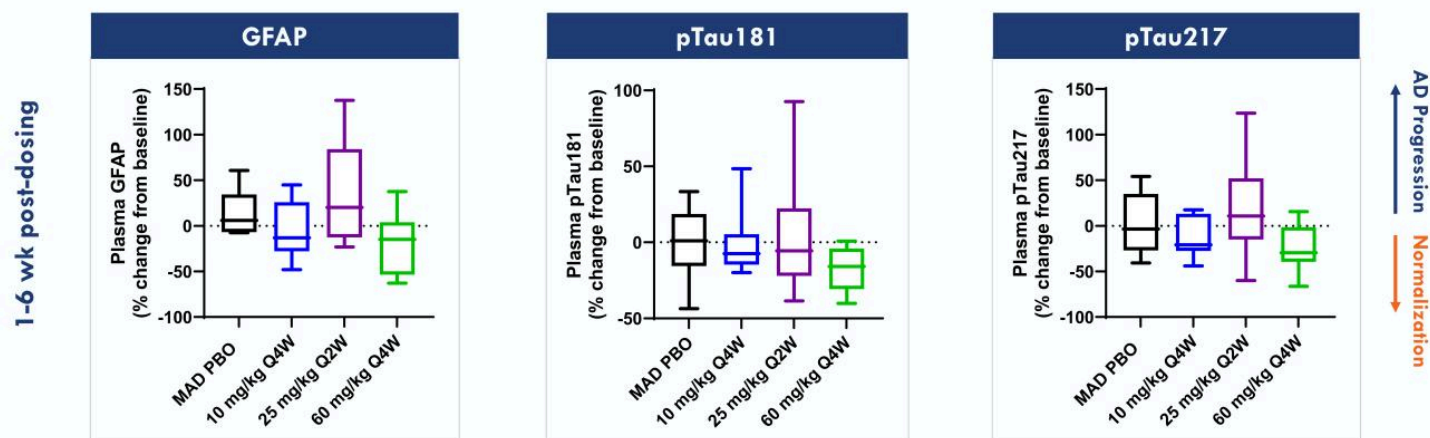
Sabirnetug CSF Exposure is Dose and Dose-Regimen Proportional



*One patient from Cohort 5 (10 mg/kg Q4W) excluded because only received one administration of drug (study drug discontinued after lacunar infarct).

E. Siemers, et al. INTERCEPT-AD, a phase 1 study of intravenous sabirnetug in participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. JPAD 2025.

Trend Toward Normalizing Plasma Biomarkers with 10 mg/kg and 60 mg/kg Q4W



- Plasma measurements of glial fibrillary acidic protein (GFAP), pTau181, and pTau217 in 10 mg/kg Q4W & 60 mg/kg Q4W groups were lower than placebo
- More impact to fluid biomarkers was observed with longer dosing duration
 - The 25 mg/kg Q2W cohort differed in dose and sample timing, with drug on board for less time than the 10 mg/kg & 60 mg/kg Q4W cohorts

E. Cline, et al, Biofluid biomarker changes following treatment with sabirnetug (ACU193) in INTERCEPT-AD, a phase 1 trial in early Alzheimer's disease. JPAD 2025.

n = 8 subjects/treated group; 6 subjects in pooled placebo (PBO); *p*-values from unpaired, 2-sided Student's *t* test

ARIA-E Summary for INTERCEPT-AD

SAD

2 mg/kg
Cohort 1

ApoE	D21	D140
3,4		
3,3	PBO	PBO
3,4		
2,3		
3,4	PBO	PBO
3,3		
3,3		
3,3		

10 mg/kg
Cohorts 2, 5

ApoE	D21	D140
3,4	PBO	PBO
3,3		
3,3		
3,4		
3,4	PBO	PBO
3,4		
3,4		
3,4		

25 mg/kg
Cohorts 3, 7

ApoE	D21	D140
3,3		
3,3	PBO	PBO
4,4		
3,3		
2,4		
3,3	PBO	PBO
3,4		
3,3		

60 mg/kg
Cohorts 4, 6

ApoE	D21	D140
4,4	PBO	PBO
3,4		
3,4	PBO	PBO
3,3		
3,3		
3,4		
2,4		
3,4		

NO ARIA-E

Asymptomatic ARIA-E

Symptomatic ARIA-E

Discontinued

MAD

ApoE	D28	D70	D196
2,3			
3,3			
3,3			
4,4			
3,3	PBO	PBO	PBO
3,4			
4,4			
3,4			
3,3			
3,4	PBO	PBO	PBO

ApoE	D28	D70	D98
3,3			
3,4			
3,4			
3,4			
3,4			
3,4	PBO	PBO	PBO
3,3			
3,4	PBO	PBO	PBO
4,4			

ApoE	D28	D63	D126
3,4			
3,3			
3,3			
4,4			
4,4	PBO	PBO	PBO
3,3			
3,4			
3,4			
3,4	PBO	PBO	PBO
3,3			

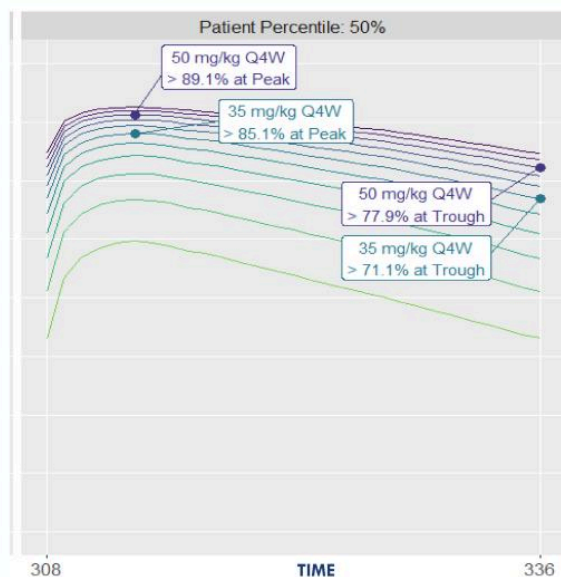
PBO: Patient on placebo

Phase 2 ALTITUDE-AD



Simulated CSF Target Engagement at Steady-State for ALTITUDE-AD Doses

- CSF target engagement was simulated at a candidate list of doses given Q4W at steady-state



Ph2 Dosing Strategy (ALTITUDE-AD)

upper dose: 50 mg/kg Q4W

lower dose: 35 mg/kg Q4W

- Notable **diminishing differentiation** as dose increases
- Doses were selected with **peak-trough** variation in mind: select doses based on trough (end of dosing interval) CSF engagement

CERTARA

Regimen 10 mg/kg Q4W 15 mg/kg Q4W 20 mg/kg Q4W 25 mg/kg Q4W 30 mg/kg Q4W 35 mg/kg Q4W 40 mg/kg Q4W 45 mg/kg Q4W 50 mg/kg Q4W 55 mg/kg Q4W 60 mg/kg Q4W

