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): August 31, 2021

Acumen Pharmaceuticals, Inc.
(Exact name of registrant as specified in its Charter)

Delaware	**001-40551**	**36-4108129**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

427 Park St., Charlottesville, Virginia	**22902**
(Address of Principal Executive Offices)	**(Zip Code)**

(434) 297-1000
(Registrant's Telephone Number, Including Area Code)

Not Applicable
(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 7.01 Regulation FD Disclosure.

On August 31, 2021, Acumen Pharmaceuticals, Inc. (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. A copy of the corporate presentation is attached as Exhibit 99.1 to this Current Report on Form 8-K (this "***Report***").

The information in this Report, including Exhibit 99.1 hereto, is furnished pursuant to Item 7.01 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, 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.

This Report and Exhibit 99.1 hereto contain forward-looking statements within the meaning of the federal securities laws. These forward-looking statements are based on current expectations and are not guarantees of future performance. Further, the forward-looking statements are subject to limitations listed in Exhibit 99.1 and in the other reports of the Company filed with the Securities and Exchange Commission, including that actual events or results may differ materially from those in the forward-looking statements.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Corporate Presentation, dated August 2021
104	Cover Page Interactive Data File (the cover page XBRL tags are embedded within the inline XBRL document)

SIGNATURES

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

Acumen Pharmaceuticals, Inc.

Dated: August 31, 2021

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

Corporate Overview

3Q 2021

Acumen Safe Harbor Statement

NOTES REGARDING THIS PRESENTATION

This presentation may contain "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 current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our clinical results, our development plans, our intellectual property and other future conditions. Words such as, but not limited to, "look forward to," "believe," "expect," "anticipate," "estimate," "intend," "plan," "would," "should" and "could," and similar expressions or words, identify forward-looking statements.

These risks and uncertainties are more fully described in our filings with the Securities and Exchange Commission, including in the section entitled "Risk Factors" in our Quarterly Report on Form 10-Q filed with the SEC on August 16, 2021 and subsequent reports that we file with the Securities and Exchange Commission. Moreover, we operate in a very competitive and rapidly changing environment. New risks emerge from time to time. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our 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. In light of these risks, uncertainties and assumptions, we cannot guarantee future results, levels of activity, performance, achievements, or events and circumstances reflected in the forward-looking statements will occur.

Forward-looking statements represent our beliefs and assumptions only as of the date of this presentation. Except as required by law, we undertake no duty to update any forward-looking statements contained in this release as a result of new information, future events, changes in expectations or otherwise.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

Acumen: Advancing a Potential Best-In-Class Antibody for Early Alzheimer's disease (Early AD)

AD Represents an Enormous Market Driven by High Unmet Need and Recent Scientific and Regulatory Momentum

Growing Scientific Consensus Supports Amyloid-Beta Oligomers (AβOs) as the Most Neurotoxic Form of Aβ and a Novel Target for Effective AD Treatment

ACU193: First, Clinical-Stage monoclonal antibody (mAb) to Selectively Target AβOs and has Promising Pre-Clinical Evidence supporting its Differentiation

Experienced Team of Industry Leaders with AD Drug Discovery, Development, and Regulatory Expertise from Eli Lilly & Co.

Phase 1 Clinical Trial Initiated in 2Q 2021 with Proof of Mechanism / Biomarker Data by YE 2022

July 2021 $184M IPO WITH HIGH QUALITY INVESTOR SYNDICATE

RA CAPITAL · BlackRock · DEEP TRACK CAPITAL · LOGOS CAPITAL · LAURION CAPITAL MANAGEMENT LP · PBM CAPITAL · ROCK SPRINGS CAPITAL · SANDS CAPITAL

Experienced in AD drug development

ACUMEN LEADERSHIP TEAM

DANIEL O'CONNELL
President & CEO

ACUMEN
neuroventures

ERIC SIEMERS, MD
Chief Medical Officer

ACUMEN
Lilly

JANICE HITCHCOCK, PHD
VP Regulatory Affairs

ACUMEN
Lilly

MATT ZUGA
Chief Financial Officer &
Chief Business Officer

ACUMEN
HighCape Partners

RUSSELL BARTON
Chief Operating Officer

ACUMEN
Lilly

ROBERT DEAN, MD, PHD
Sr. Development Advisor

ACUMEN
Lilly

GEORGE VAUGHN, CPA
VP, Finance and Accounting
Vaughn & Assoc.

JAMES SENETAR, MS, PHARM.D., PMP
Sr. Clinical Operations Manager

ACUMEN
Lilly

JASNA JERECIC, PHD
Analytical Methods
Leader, Research Scientist

ACUMEN

Over a decade of experience working towards a shared goal.

Aduhelm Approved under Accelerated Approval Pathway

FDA NEWS RELEASE

FDA Grants Accelerated Approval for Alzheimer's Drug

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For Immediate Release: June 07, 2021

Today, the U.S. Food and Drug Administration approved Aduhelm (aducanumab) for the treatment of Alzheimer's, a debilitating disease affecting 6.2 million Americans. Aduhelm was approved using the accelerated approval pathway, which can be used for a drug for a serious or life-threatening illness that provides a meaningful therapeutic advantage over existing treatments. Accelerated approval can be based on the drug's effect on a surrogate endpoint that is reasonably likely to predict a clinical benefit to patients, with a required post-approval trial to verify that the drug provides the expected clinical benefit.

- First disease-modifying drug approved for Alzheimer's
- Approved under accelerated approval based on reduction in amyloid beta plaques[1]

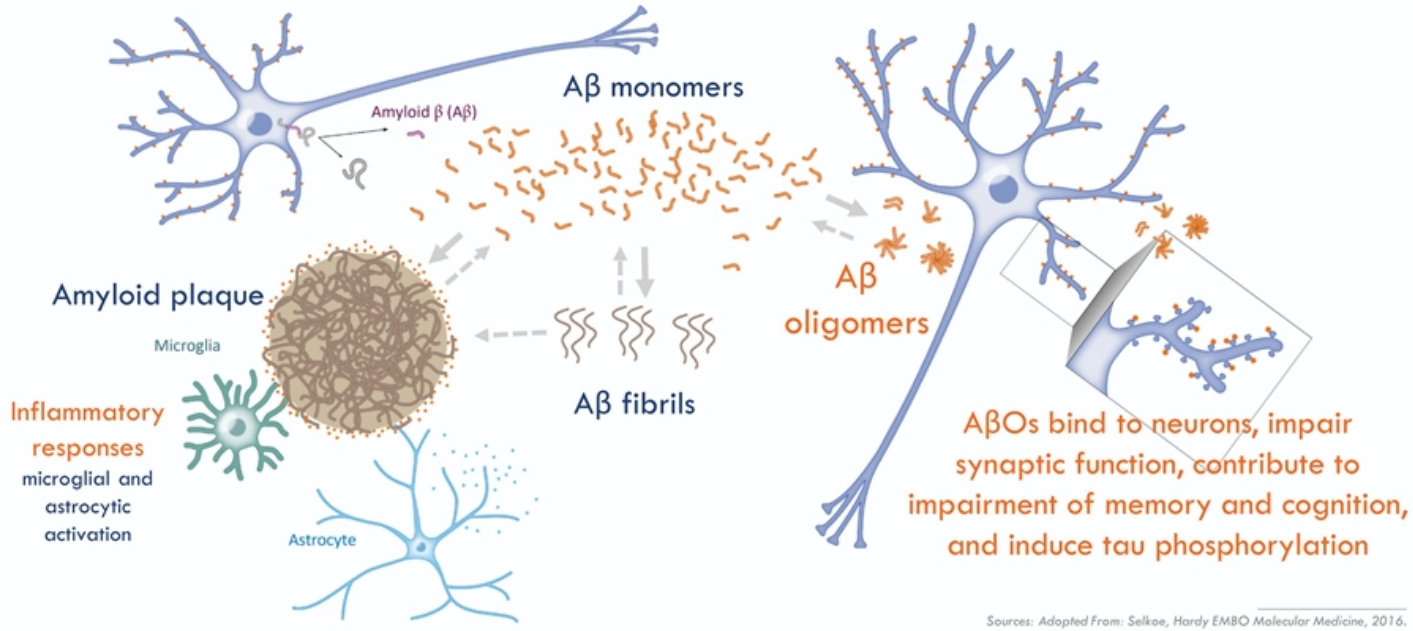
1https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/761178s000lbl.pdf

Aduhelm approval ushers in new era and regulatory environment for AD drug development
Acumen will evaluate biomarkers to support future regulatory submissions

ACUMEN

Growing Interest in the Anti-AβO Hypothesis

Growing understanding of disease mechanisms indicate that AβOs are the most toxic Aβ species and have the potential to be an ideal target for effective AD therapy

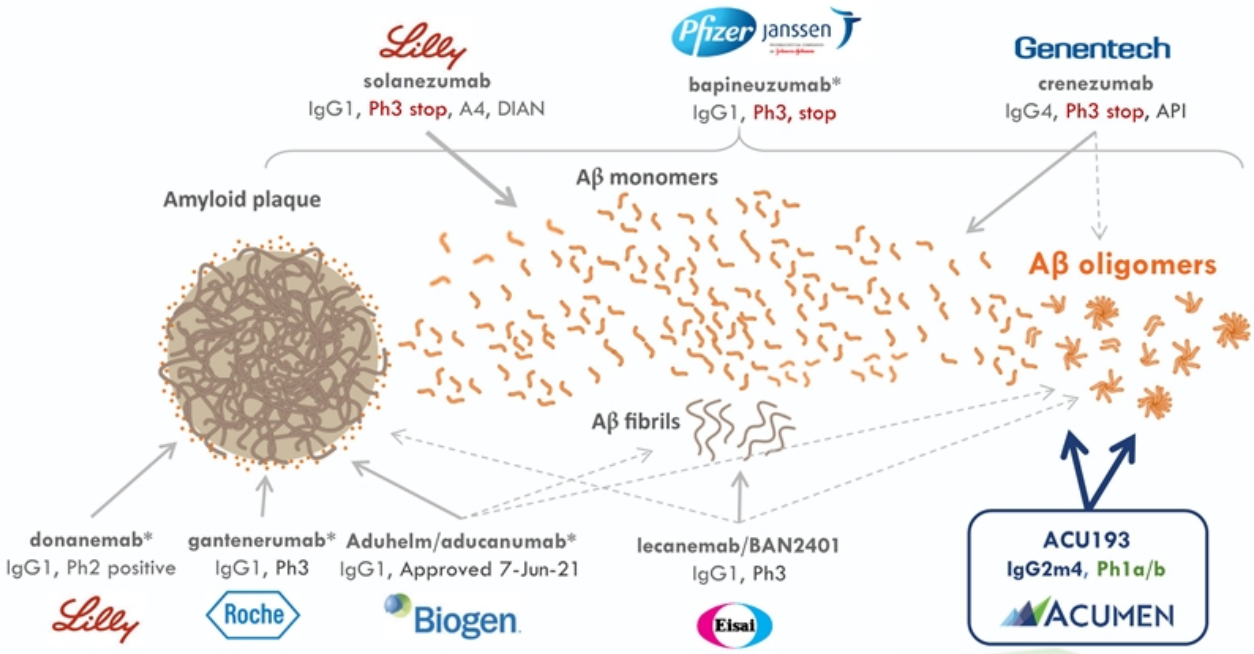


Sources: *Adopted From: Selkoe, Hardy EMBO Molecular Medicine, 2016.*

The only approved antibody for AD preferentially targets amyloid plaques with only limited effects on oligomeric forms of Aβ. Acumen's drug candidate ACU193 targets AβOs.

ACUMEN

ACU193 Positioning Relative to Late-stage and Approved Anti-Aβ/plaque mAbs



ACU193's High Selectivity for AβOs Combined with an Expected Lack of ARIA-related Safety Concerns Is Anticipated to Provide Superior Cognitive Efficacy Compared to Peers

* IgG1 monoclonal antibodies that bind amyloid plaque are associated with high rates of ARIA-E

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Upcoming Milestones: ACU193 Development Plan Expected to Demonstrate Proof of Mechanism by YE 2022

ACU193 Phase 1 Proof of Mechanism results are expected to inform dose selection and regulatory strategy for the Phase 2/3 trial

AD Drug Development: Amyloid Hypothesis

AD is One of the World's Largest Unmet Medical Needs

DISEASE OVERVIEW

- AD is a progressive, uniformly fatal neurodegenerative disorder and is the most common form of dementia
- Memory loss is the key symptom of AD
- In advanced stages of the disease, complications from severe loss of brain function — such as dehydration, malnutrition or infection — result in death

DISEASE BURDEN

- AD affects >6M people in the United States and >32M people worldwide
- Patients suffering in the later stages of AD require nearly full-time care, resulting in a significant societal and economic burden, with direct healthcare costs estimated to be $355 billion annually in 2021

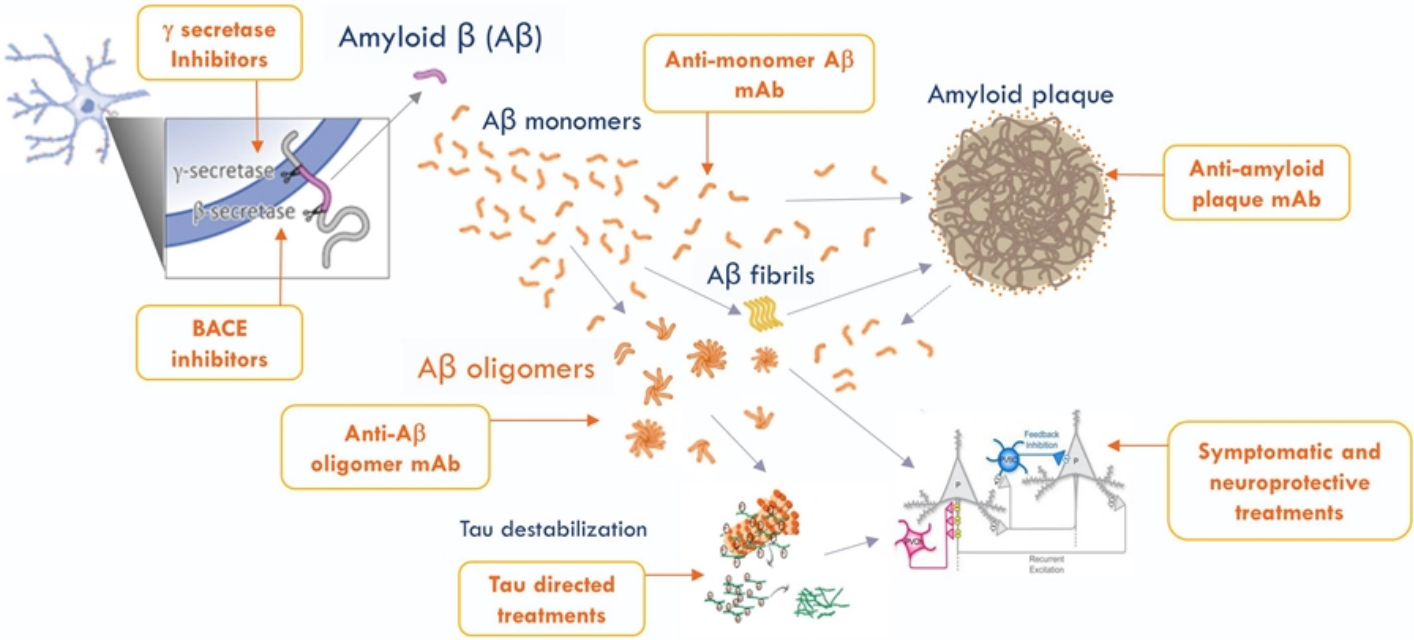
UNMET NEEDS

- AD is the sixth leading cause of death in the US
- Treatment options include cholinesterase inhibitors and an NMDA receptor antagonist, aimed to reduce symptomatic burden, which have modest benefit along with supportive care
- Aduhelm (aducanumab) was approved through accelerated access pathway based on a surrogate endpoint

Alzheimer's Pathophysiology

Build-up of amyloid-beta (Aβ) is believed to lead to neurodegeneration and dementia

Previous and current anti-amyloid and related drug targets have attempted to intervene



Emerging data indicate that amyloid β oligomers are the most toxic species and should be preferentially targeted for removal

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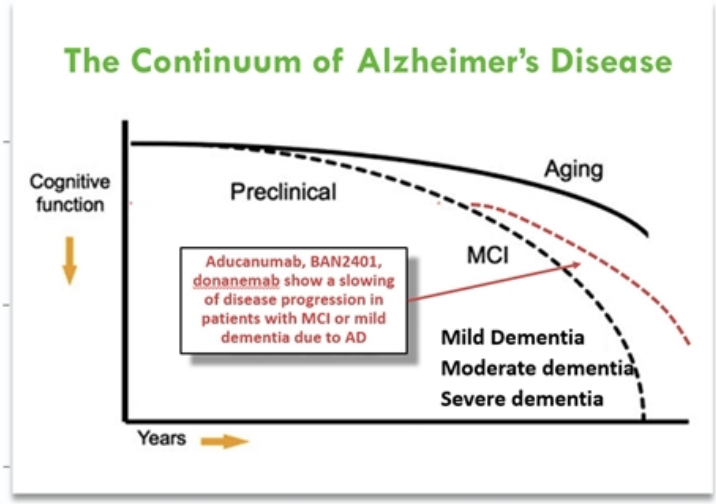
Recent anti-amyloid mAb results (anti-Ab/plaque) establish biological foothold for treating disease

EFFICACY: Reduced cognitive decline ~11% - 47% at ~18 months

TARGET ENGAGEMENT: Positive effects on imaging and fluid biomarkers

SAFETY: ARIA-E rates ~10% to ~35%, and higher in genetically predisposed APOE4+ for plaque targeting mAbs

Potential for current generation anti-Aβ/plaque mAbs to serve as 'first-in-class' drugs providing foothold for treating patients that can be built upon and/or improved



Abbreviations: ARIA-E - Amyloid Related Imaging Abnormalities – Edema; BACE - Beta Amyloid Cleavage Enzyme

Anti-Aβ/plaque mAbs, as a class, appear to have positive signal in early AD and leave significant room for improvement

Positive Signals and Proof of Concept from Recent Phase 2-3 AD Anti-Amyloid mAb Studies

Percent Slowing of Cognitive/Functional Decline*

Measured Outcome**	solanezumab EXPEDITION 3 (Phase 3)	aducanumab EMERGE (Phase 3)	aducanumab ENGAGE (Phase 3)	lecanemab BAN2401 (Phase 2)	donanemab (Phase 2)
ADAS-cog	-11%	-27%	-12%	-47%	-39%
ADCS-ADL	-15%	-40%	-18%	N.A.	-23%
CDR-SB	-15%	-23%	2%	-26%	-23%
MMSE	-13%	-15%	3%	N.A.	-21%
iADRS	-11%	N.A.	N.A.	N.A.	-32%

* Percent Slowing = P[1- [(endpoint score-baseline score)active/(endpoint score-baseline score)placebo]]*100%*(-1)

** ADAS-cog: Alzheimer's Disease Assessment Scale – Cognitive Subscale
ADCS-ADL: Alzheimer's Disease Cooperative Study – Activities of Daily Living
CDR-SB: Clinical Dementia Rating – Sum of Boxes
MMSE: Mini-Mental State Examination
iADRS: Integrated Alzheimer's Disease Rating Scale

Note: ENGAGE Post-Protocol Version 4 – at least 14 doses of 10 mg/kg, High Dose cohort achieved 27% improvement on CDR-SB compared to placebo

"We're looking for a biological foothold against Alzheimer's that we can build on. And so, these effects are small, but I think they are meaningful, and I hope they're the beginning of a process that we can add to." ***- Stephen Salloway, MD** of Brown University*[+]

+Source: Wall Street Journal, Biogen Details Case for Controversial Alzheimer's Drug, published December 5, 2019

Anti-plaque mAbs demonstrate dose-related ARIAs that will limit use

Percent of ARIA Events for Anti-Aβ/plaque mAbs*

	TARGETING AB MONOMERS		TARGETING AMYLOID PLAQUES									
	solanezumab EXPEDITION 3 (Phase 3)		aducanumab EMERGE (Phase 3)			aducanumab ENGAGE (Phase 3)			lecanemab BAN2401 (Phase 2)		donanemab (Phase 2)	
	PC	Treated	PC	Low	High	PC	Low	High	PC	High	PC	Treated
ARIA-E	0.2%	0.1%	2.2%	26.1%	34.4%	3.0%	25.6%	35.7%	0.8%	9.9%	0.8%	27.5%
ApoE ε4 carriers			1.9%	29.8%	42.5%	2.4%	28.7%	41.8%	1.2%	14.6%	3.6%	44.0%
ApoE ε4 non-carriers			2.9%	18.1%	17.9%	4.3%	17.5%	27.7%	0.0%	8.0%		
Any ARIA E or H			10.3%	32.8%	41.2%	9.8%	30.7%	40.3%		N.A.	8.0%	38.9%

* PC = Placebo, Low = Low Dose; High = High Dose

Shows the absence of ARIA after treatment with antibodies targeting Aβ monomers (solanezumab) in comparison to the increasing presence of ARIA after treatment at increasing dose levels with antibodies targeting amyloid plaques (aducanumab, BAN2401, and donanemab), indicate that ARIA results from the removal of amyloid plaques around blood vessels and likely does not result, from treatment with antibodies that target other species of Aβ, i.e. Aβ monomers and AβOs.

ARIA-E represents a dose limiting adverse effect for mAbs with plaque binding. Antibodies that avoid ARIAs should be safer and more feasible to administer and possibly at higher doses.

ACU193's High Selectivity for AβOs, Combined with its Expected Lack of ARIA-related Safety Concerns, Is Anticipated to Provide Superior Efficacy Compared to Peers

		TARGET SELECTIVITY+				SAFETY PROFILE
Company	Asset	Amyloid plaque	Aβ fibrils	Aβ monomers	Aβ oligomers	Lack of ARIA
ACUMEN	**ACU193**	✗	untested	✗	✓	✓
Biogen	Aduhelm aducanumab	✓	✓	✗	✓	✗
Eisai	lecanemab BAN2401	✓	✓	✗	✓	✗
Roche	gantenerumab	✓	✓	✗	✓	✗
Lilly	donanemab	✓	untested	✗	✗	✗
Lilly	solanezumab*	✗	✗	✓	✗	✓
Genentech	crenezumab*	✓	✓	✓	✓	✓
Pfizer janssen	bapineuzumab*	✓	✓	✓	✓	✗

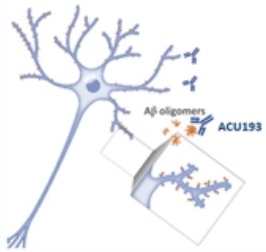
*Phase 3 discontinued for primary AD indication

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

ACU193:
Our differentiated approach

Target Product Profile: ACU193 Best-in-Class, 1st line, anti-AβO, Disease-modifying Immunotherapy for Early AD

DRUG:	ACU193 is a humanized, affinity-matured, mAb with high selectivity for toxic **AβOs vs. Aβ monomers (>500x)** and amyloid plaques. **ACU193 is an IgG2m4 subclass** mAb which lacks inflammatory effector functions of other IgG subclasses.
POPULATION:	Early AD - Mild Cognitive Impairment and Mild Dementia due to AD (amyloid positive by PET)
DOSING:	IV infusion every 4 weeks
DURATION:	Chronic therapy for duration of Early AD
VALUE PROPOSITION:	**Selectivity for toxic AβOs is expected to provide superior cognitive efficacy** and **improved safety and tolerability** relative to non-selective anti-Aβ/plaque mAbs • Slow decline of memory and cognition in Early AD • Decrease AβOs induced synaptic and neuronal network toxicity • Slow disease progression and downstream effects on tau, neurodegeneration, and neuro-inflammation • Low rate of ARIA expected • **Effective as stand-alone therapy or potentially in combination with other symptomatic, anti-inflammatory, and/or tau directed therapies**



ACU193: Extensive Data Package Supporting Development

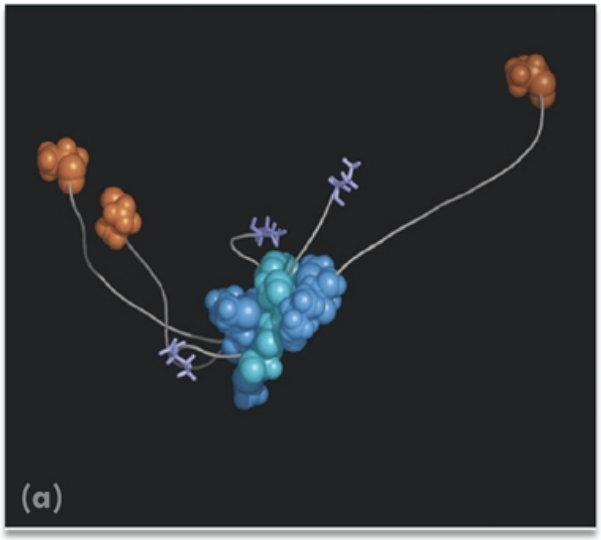
SELECTIVITY	• Nanomolar affinity for AβOs, >500-fold greater selectivity for AβOs over Aβ monomer, with limited or no discernable binding to vascular amyloid or dense core amyloid plaques • Binds broad range of endogenous AβOs present in transgenic mice and human AD samples (binds dimers to mid-sized molecular weight AβOs)
PHARMACOLOGY	• Dose-dependent effects in multiple in vitro neuroprotection assays • Positive memory and behavioral effects in multiple in vivo transgenic mouse models for AD
PK/PD	• Brain penetration and biodistribution demonstrated in multiple species • Performs like other peripherally administered CNS mAbs
SAFETY	• IgG2m4 subclass lacks inflammatory effector function signaling (C1q, FcγR1, FcγRIII) • Microhemorrhage studies show no increased risk of microhemeorrhage • GLP studies demonstrated acceptable safety margin for clinical dosing plans
REGULATORY	• Active IND • Phase 1 started 2Q 2021

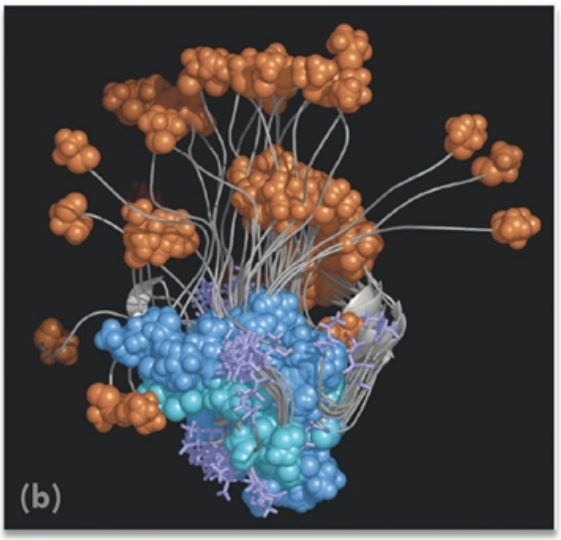
ACU193 is a promising immunotherapy for Early AD expected to provide meaningful cognitive and functional benefits, slow disease progression, and offer an attractive safety profile

ACUMEN

What is an Aβ Oligomer?

AβOs may consist of 2 to >200 Aβ peptides.
AβOs composed of 3 (a) and 18 (b) Aβ peptides are depicted below.





Sources: Kelley et al. J Chem Physics 2008.

AβOs are present in brain in a wide range of sizes

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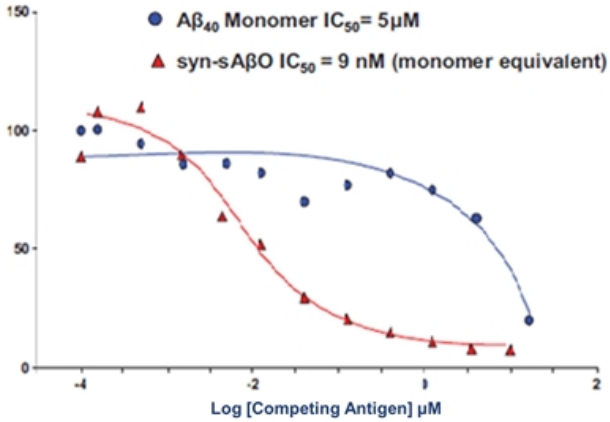
ACU193 is the First mAb Developed to Selectively Target AβOs

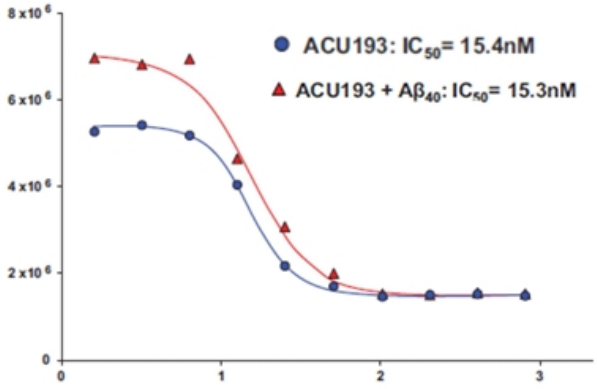
Highly selective for Aβ oligomers versus Aβ monomers

ACU193 Selectivity



Binding of ACU193 to AβOs >500x binding to Aβ monomer

ACU193 Selectivity in presence of 5μM monomeric Aβ



Even in the presence of a large excess of Aβ monomer, binding of ACU193 to AβOs is unchanged

ACU193 selective binding to AβOs is preserved even in the presence of a large excess of Aβ monomer which is present in brain – limited target distraction

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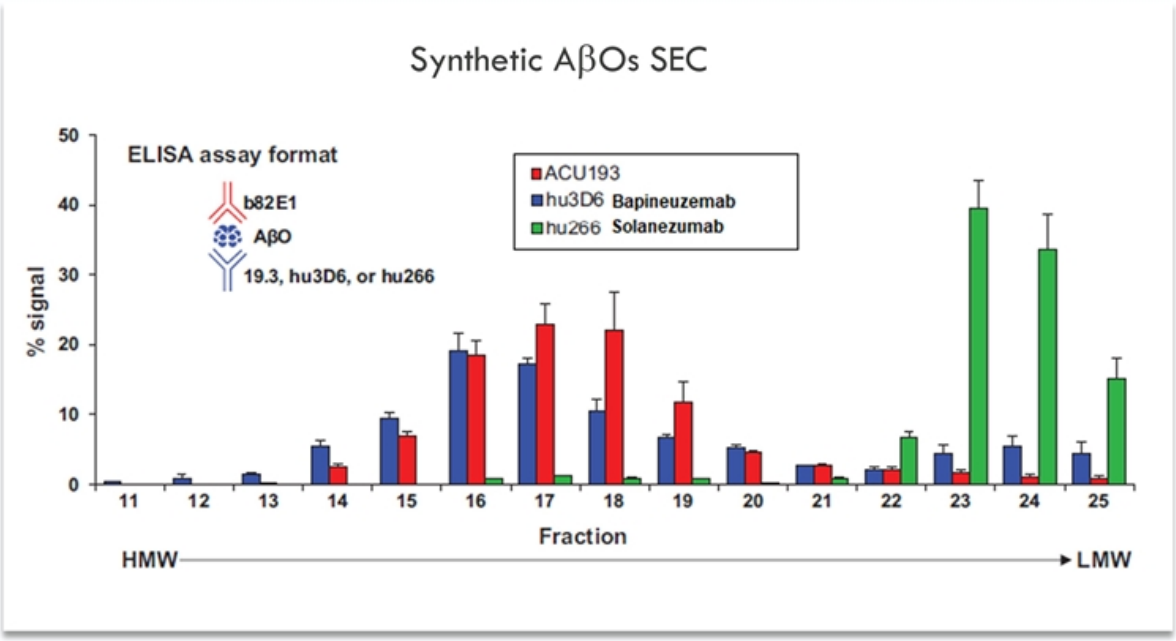
ACU193 has a greater preference for AβOs than other mAbs

Comparison of Aβ species-mAb complex signals across SEC fractions

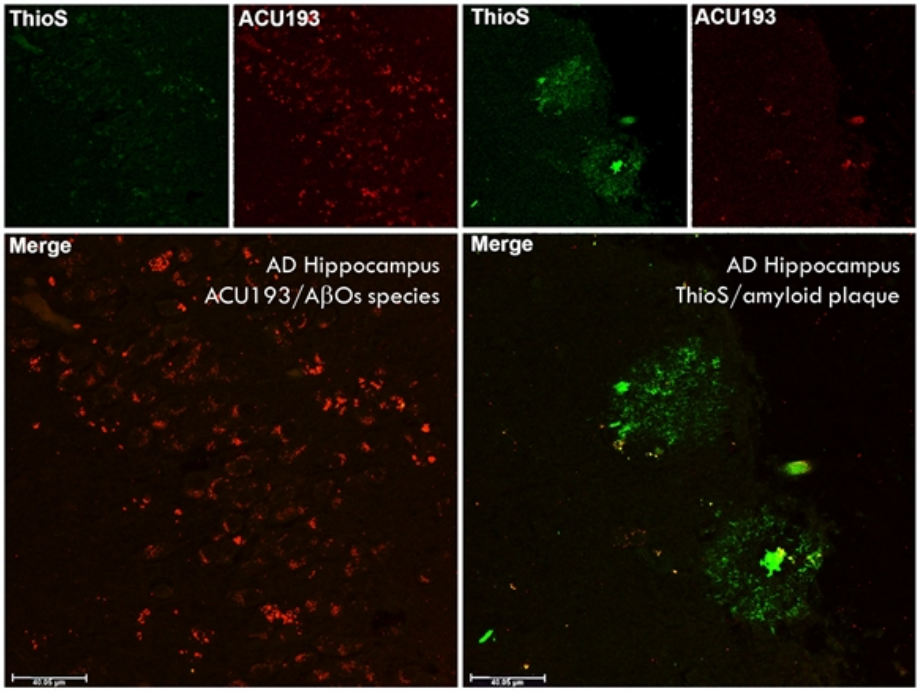


ACU193 binds to a wide range of oligomeric species of Aβ that are differentiated from those bound by hu266 (solanezumab) or hu3D6 (bapineuzumab)

SELECTIVITY

ACU193 is highly selective for AβOs versus Aβ plaques

ACU193 staining in human AD brain slices ACU193 (red) binds non-Thioflavin S positive Aβ (green)



ACU193 has little or no binding to thioflavin S positive fibrillar Aβ plaque in human AD brain tissue

Sources: E. Cline et al. CTAD 2019.

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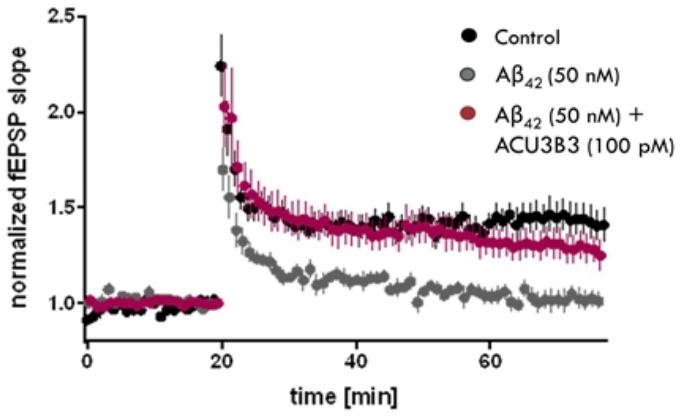
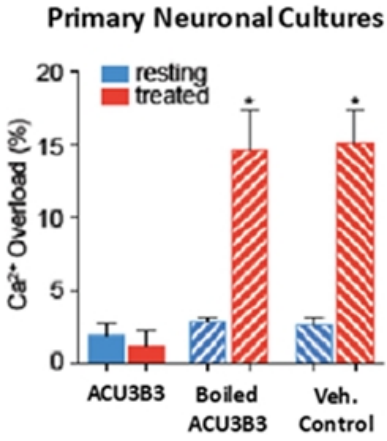
AβOs Bind to Neurons and are Toxic; mouse analogue of ACU193 prevents toxicity

After binding to neurons, AβOs disrupt Long Term Potentiation (LTP) and cause pathologic increases in intracellular calcium that is destructive to cells.

ACU3B3 prevents AβO inhibition of hippocampal LTP *ex vivo*



ACU3B3 prevents AβO mediated Ca2+ elevation in cell cultures



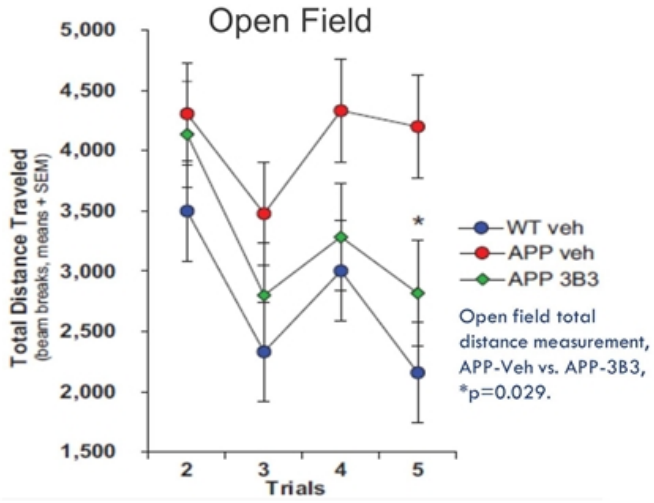
Note: (1) ACU3B3 is the mouse monoclonal antibody precursor to and equivalent of humanized ACU193

ACU3B3 prevents changes in aberrant neuronal activity underlying memory loss in AD and prevents AβO mediated disruption of calcium homeostasis in neuronal cultures

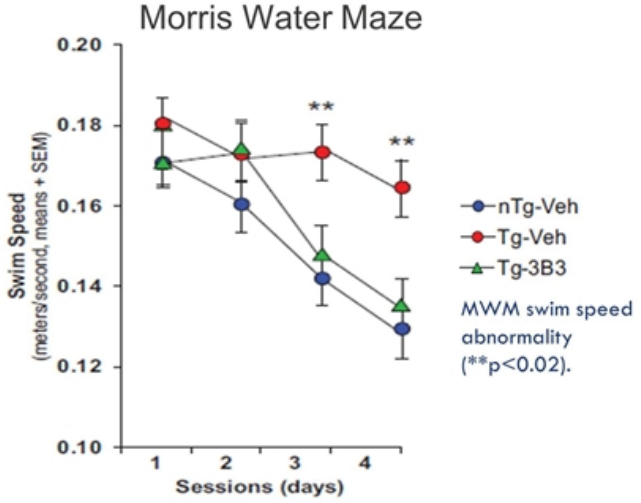
ACUMEN

Treatment of a Transgenic Mouse Model of AD results in Behavioral Improvements

Murine version of ACU193 (3B3) was used to treat younger mice with depositing plaque or older mice with abundant plaque



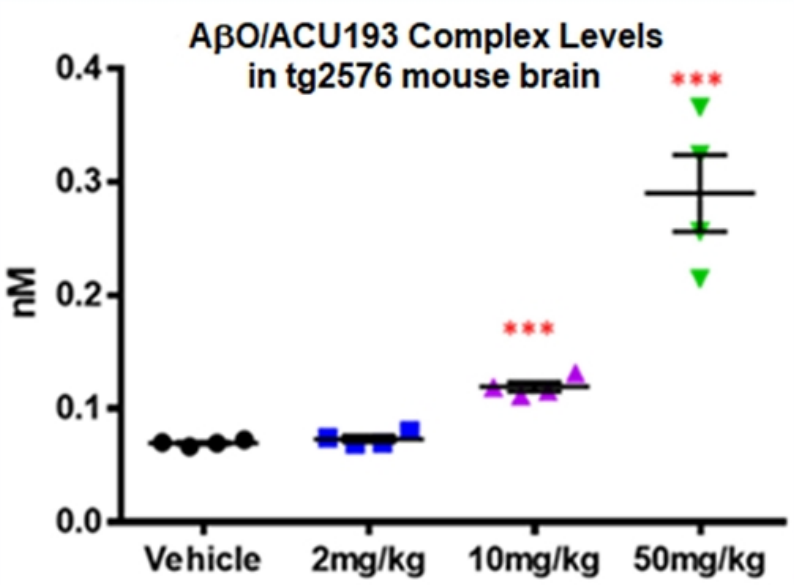
Deficits in younger (5-7 months) transgenic mice are markedly reduced with treatment



Deficits in older (9-10 months) transgenic mice are markedly reduced with treatment

ACUMEN

ACU193 Enters the CNS and Binds to AβOs in Transgenic Mice in Dose Dependent Manner



ACU193 engages target AβOs in transgenic mouse brain (tg2576) in dose dependent manner. Ability to push doses higher in patient clinical trials may provide increased target coverage.

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Clinical Development Plans

Phase 1 Overview

TRIAL DESIGN:

Randomized Placebo Controlled Phase 1a/b

- Part A : Single-Ascending Doses
- Part B : Multiple-Ascending Doses

ENROLLMENT CRITERIA:

Early AD

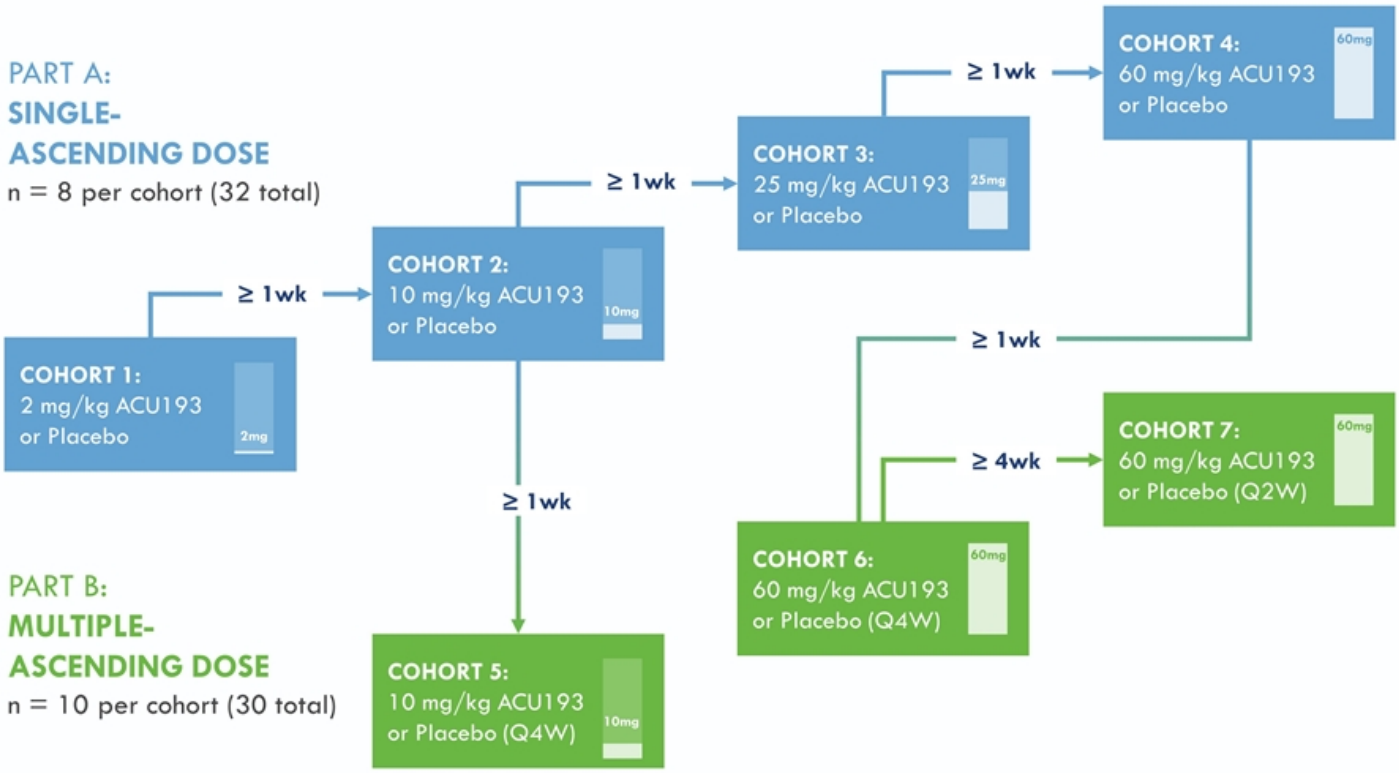
- Mild Cognitive Impairment and Mild Dementia due to AD (amyloid positive by PET)

TRIAL OBJECTIVES:

Proof of Mechanism (PoM)

- Safety and tolerability
- Pharmacokinetics
- Target Engagement
- Biomarkers; cognition

Randomized Placebo Controlled Phase 1a/b in Early AD patients - Started 2Q 2021



Phase 1 Objectives: Proof of Mechanism

1. SAFETY AND TOLERABILITY
- Assessment of ARIA-E
- Absence of problematic immunogenicity

2. PHARMACOKINETICS
- Peripheral and Central

3. EVIDENCE OF TARGET ENGAGEMENT
- CSF level of ACU193:AβO complexes (bound)

4. FLUID BIOMARKER EFFECTS
- Phospho-tau, Neurofilament light, et. al.

5. CLINICAL MEASURES
- Assessment of clinical cognitive measures, computerized tests (Cogstate Ltd.)

6. MRI EFFECTS
- Potential improvements in cerebral blood flow shown with MRI ASL pulse sequence

PROOF OF MECHANISM

Requirements for Phase 2/3

- ✓ Acceptable safety and tolerability
- ✓ Show ACU193 gets into central compartment
- ✓ Target engagement
- ✓ Other indicators of target mechanism of action

ACUMEN

ACU193 IP & Market Exclusivity; Commercial Considerations

- Exclusive, perpetual, worldwide, royalty-free license from Merck to all Merck Amyloid Derived Diffusible Ligand (ADDL) IP including, issued ACU193 patents
- ACU193 Global IP estate:
  - Issued patents in 17 countries, pending in 2 countries
  - Composition of matter patents and methods of use run into July 2031
  - Patent term extensions available, 3-5 years depending on jurisdiction
- Biologics market exclusivity is expected for ACU193 as a novel biologic drug
  - FDA currently provides 12 years market exclusivity for novel biologics
  - EMEA provides 10 years of market exclusivity for novel biologics
- Aduhelm list price of $56,000 sets framework for first approved disease-modifying drug

Acumen is Well Capitalized to Achieve Important Clinical Development Milestones

MILESTONES	STATUS/EXPECTED TIMING
IND submission	✓
Initiated Ph1a/b clinical trial	✓
Ph1a/b trial updates	Periodic 2021-2022
Ph1a/b Proof of Mechanism Top-Line results	YE 2022
Initiate Ph2/3 Clinical trial	2023

$235m

Pro forma cash balance as of July 8, 2021[(1)]

Note: Expected timelines subject to change.

(1) Cash, cash equivalents and marketable securities were $68.8 million as of June 30, 2021. The net proceeds of $169 million from the IPO resulted in total cash, cash equivalents and marketable securities increasing to more than $235 million as of July 8, 2021.

Experienced in AD drug development

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Acumen: Advancing a Potential Best-In-Class Antibody for Early Alzheimer's disease (Early AD)

AD Represents an Enormous Market Driven by High Unmet Need and Recent Scientific and Regulatory Momentum

Growing Scientific Consensus Supports Amyloid-Beta Oligomers (AβOs) as the Most Neurotoxic Form of Aβ and a Novel Target for Effective AD Treatment

ACU193: First, Clinical-Stage monoclonal antibody (mAb) to Selectively Target AβOs and has Promising Pre-Clinical Evidence supporting its Differentiation

Experienced Team of Industry Leaders with AD Drug Discovery, Development, and Regulatory Expertise from Eli Lilly & Co.

Phase 1 Clinical Trial Initiated in 2Q 2021 with Proof of Mechanism / Biomarker Data by YE 2022

July 2021 $184M IPO WITH HIGH QUALITY INVESTOR SYNDICATE

RA CAPITAL · BlackRock · DEEP TRACK CAPITAL · LOGOS CAPITAL · LAURION CAPITAL MANAGEMENT LP · PBM CAPITAL · ROCK SPRINGS CAPITAL · SANDS CAPITAL