Corporate Overview

2Q 2022

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, Acumen's ability to achieve its strategic and financial goals, including its projected use of cash, cash equivalents and marketable securities and the sufficiency of its cash resources, and the therapeutic potential of Acumen's product candidate, ACU193, including its potential for improved safety and efficacy as compared to other monoclonal antibodies in development, as well as the expectations concerning the INTERCEPT-AD trial and Acumen's planned Phase 2/3 clinical trial, including the expected timing of initiation, enrollment and reporting data, and risks and uncertainties relating to the progression and duration of the COVID-19 pandemic and responsive measures thereto and related effects on Acumen. 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's10-K for the year ended December 31, 2021, 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 a Potential Best-/First-In-Class Antibody Product for Early Alzheimer's disease (Early AD)

Alzheimer's Represents an Enormous Market Driven by High Unmet Need and Recent Scientific and Regulatory Momentum

Scientific Consensus Supports **Amyloid-Beta Oligomers (AβOs)** as the **Most toxic** 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 Leadership Comprised of Industry Leaders with AD Drug Discovery, Development, and Regulatory Expertise from **Eli Lilly & Co.**

Strong Balance Sheet: ~$225M in cash at 12/31/21

July 2021 IPO ~$184M Gross

RA Capital
Deep Track
Sands Capital
PBM Capital
BlackRock

Phase 1 Clinical Trial in Early AD Patients Ongoing **Proof of Mechanism** / Target Engagement / Safety Data **Topline Results Expected 1H 2023**

We believe Acumen has the organizational expertise and fiscal resources to advance ACU193 through multiple anticipated clinical development milestones during 2022 through 2025

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 a significant symptom of early 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
- AD is the sixth leading cause of death in the US
- Patients suffering in the later stages of AD require nearly full-time care, resulting in a significant societal and economic burden.
- The direct cost of caring for individuals with AD and other dementias in the United States were expected to total $355 billion in 2021

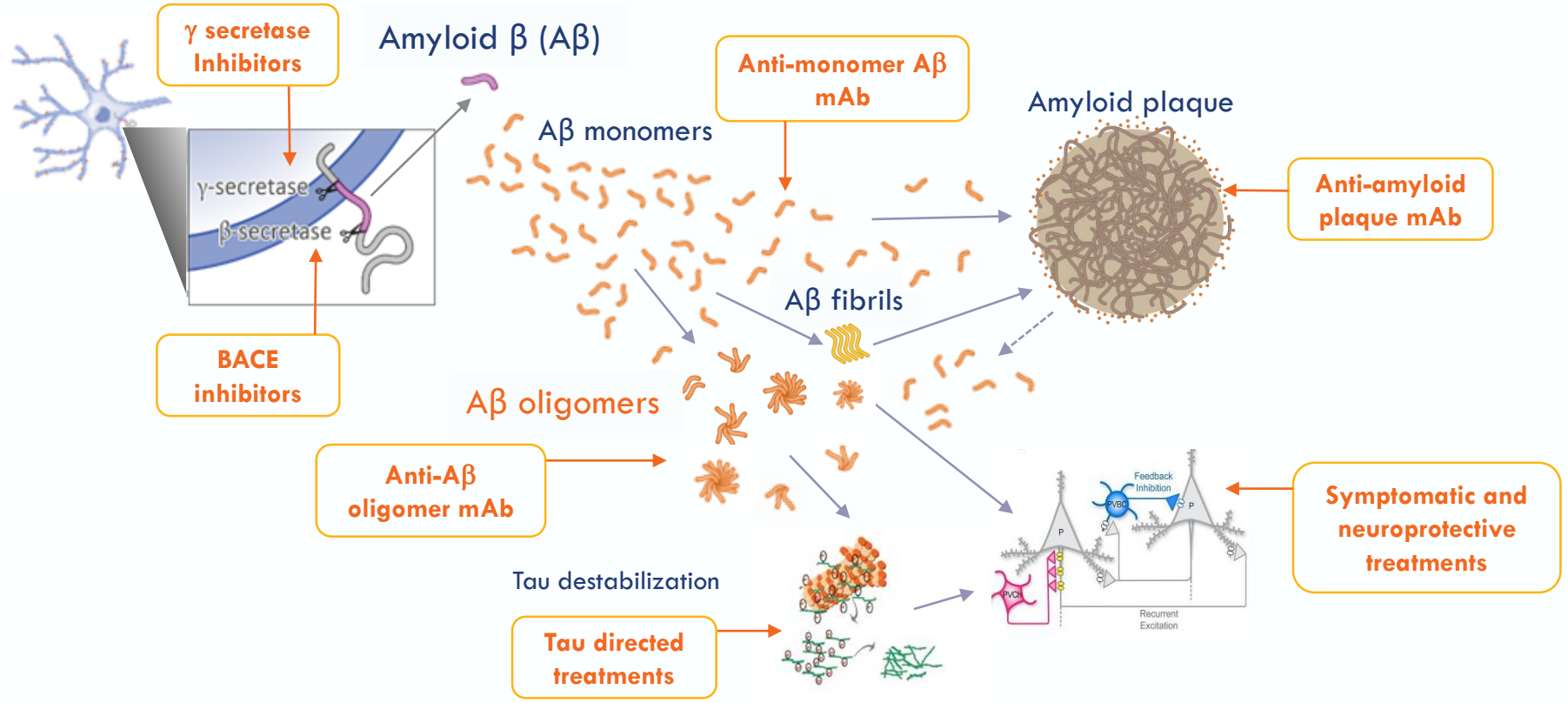
UNMET NEEDS

- Treatment options include cholinesterase inhibitors and an NMDA receptor antagonist, aimed to reduce symptomatic burden, which have modest benefit along with supportive care
- FDA approved Aduhelm™ using the accelerated access pathway based on a surrogate endpoint; Controversy over risk-benefit and CMS' CED reimbursement decision led to challenges culminating in Biogen's recent decision to terminate commercial support for the drug.

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

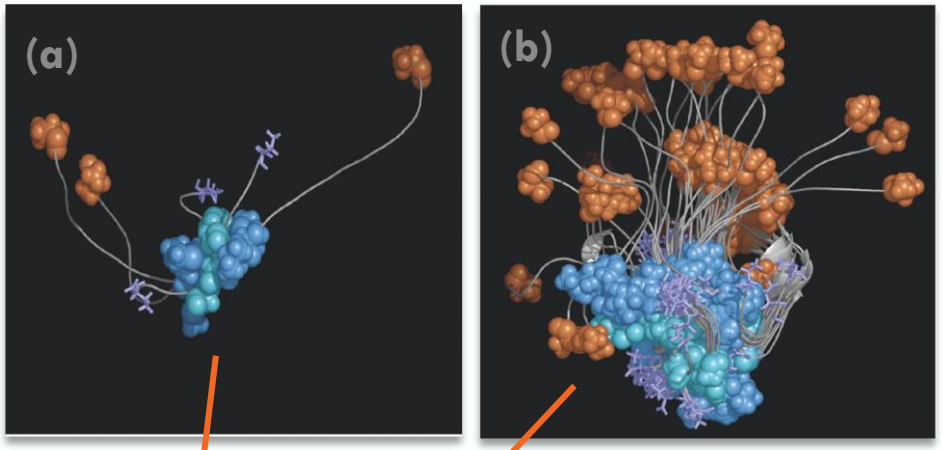


Emerging data indicate that soluble amyloid β oligomers (AβOs) are the most toxic species and should be preferentially targeted for removal

What is an Aβ Oligomer?

AβOs may consist of 2 to >200 Aβ peptides.

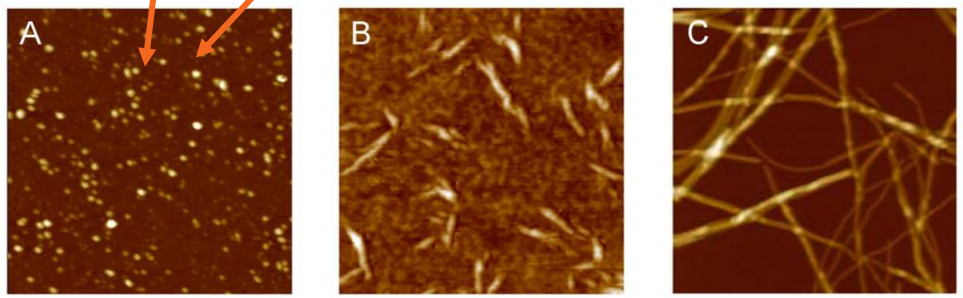
Figure 1. AβOs composed of 3 (a) and 18 (b) Aβ peptides are depicted below.



Sources: Kelley et al. J Chem Physics 2008.

Quaternary structures of Aβ oligomers, protofibrils and fibrils

Figure 2. Atomic force microscopy images of representative steps of amyloid aggregation: (A) oligomers; (B) protofibrils; (C) mature fibrils. Scan size 1.0 μm. Z range (A) 8.0 nm; (B) 15 nm; (C) 20 nm.



Source: Relini et al. Biomolecules 2014

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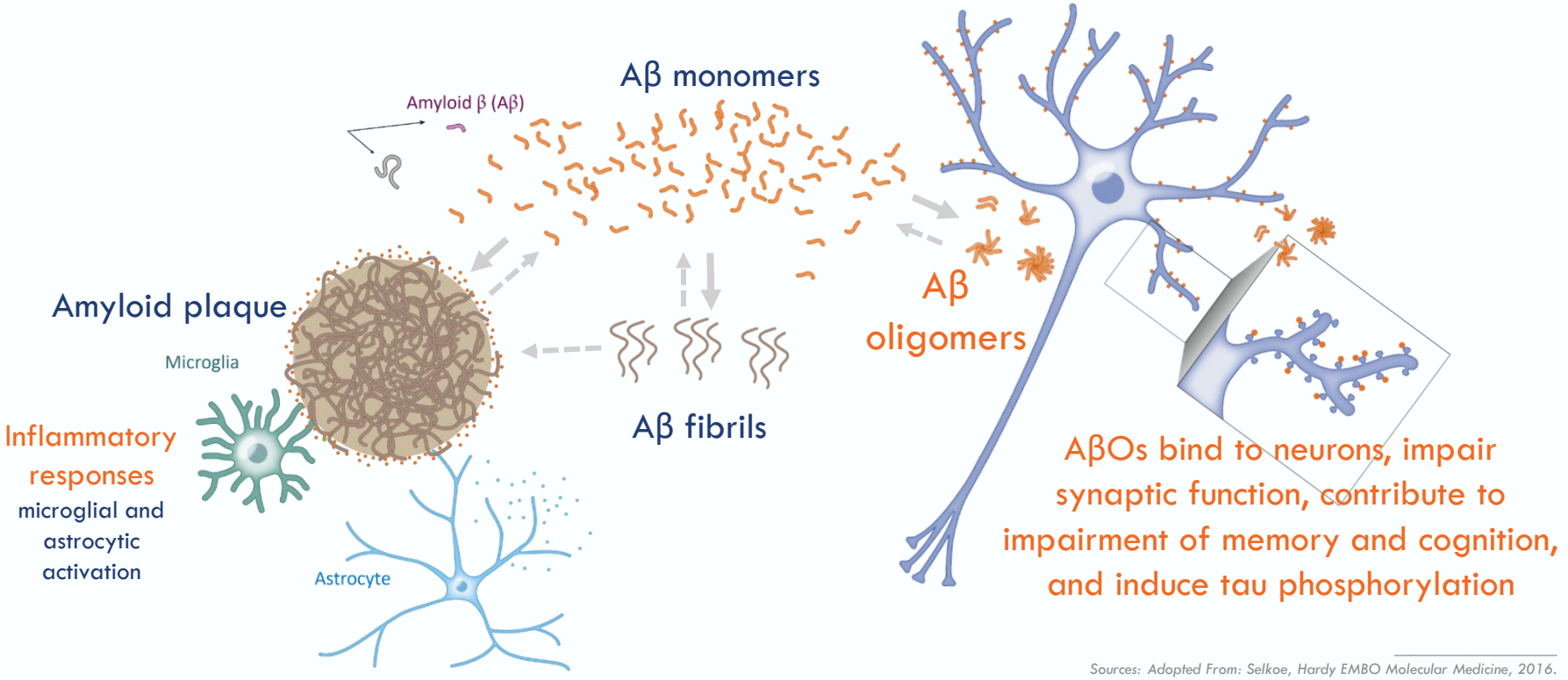
Scientific Consensus Supports Anti-AβO Hypothesis

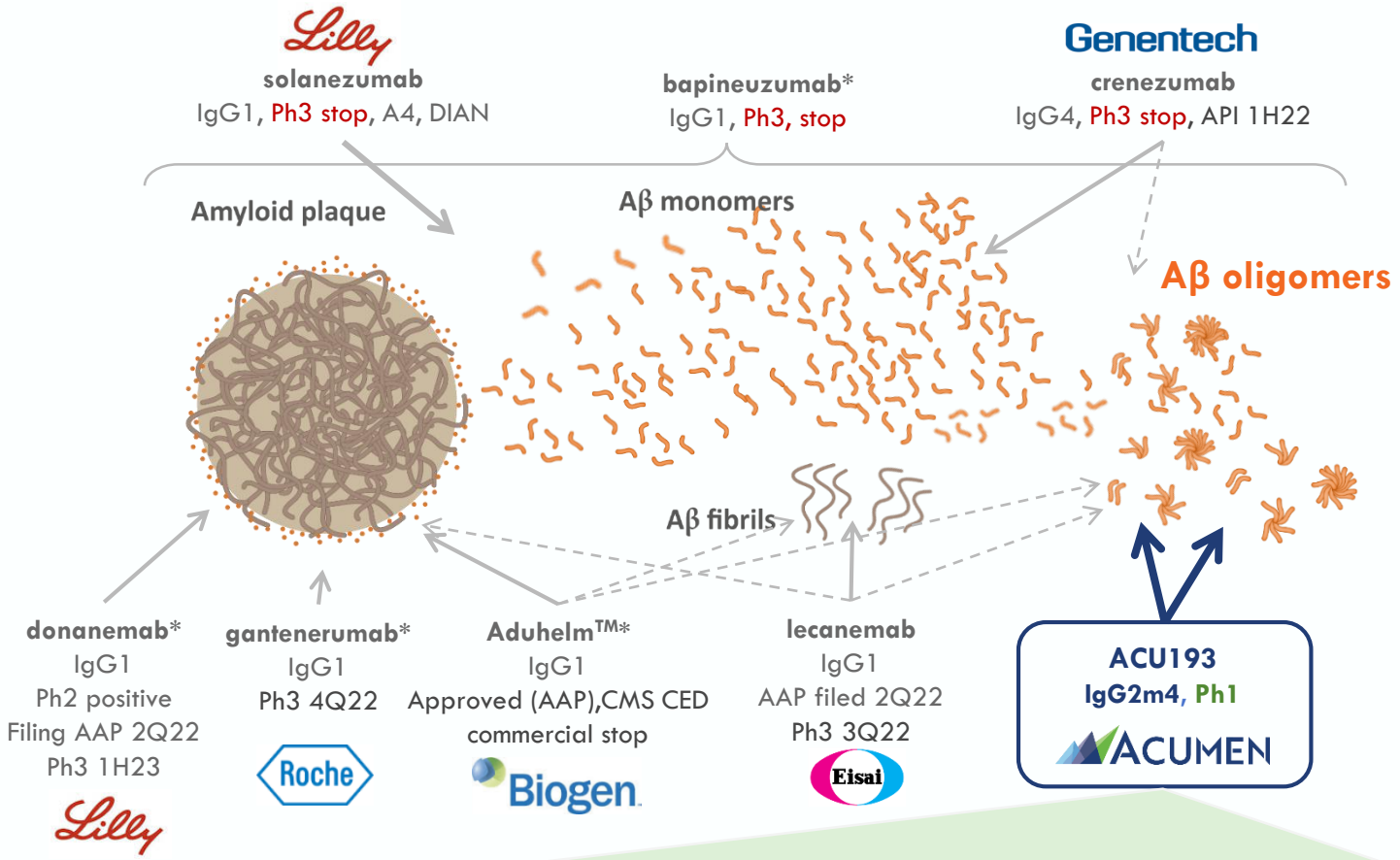
Growing understanding of disease mechanisms indicate that AβOs are the most toxic Aβ species and have the potential to be an ideal target for effective AD therapy



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

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

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 anti-plaque mAbs

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

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ACU193's High Selectivity for toxic 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™	✓	✓	✗	✓	✗
Eisai	lecanemab	✓	✓	✗	✓	✗
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

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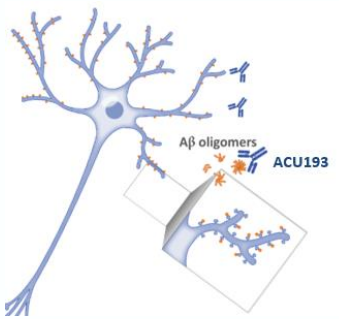
Target Product Profile: ACU193 Best-in-Class, 1st line, anti-AβO, Disease-modifying Immunotherapy for Early AD

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 ongoing

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

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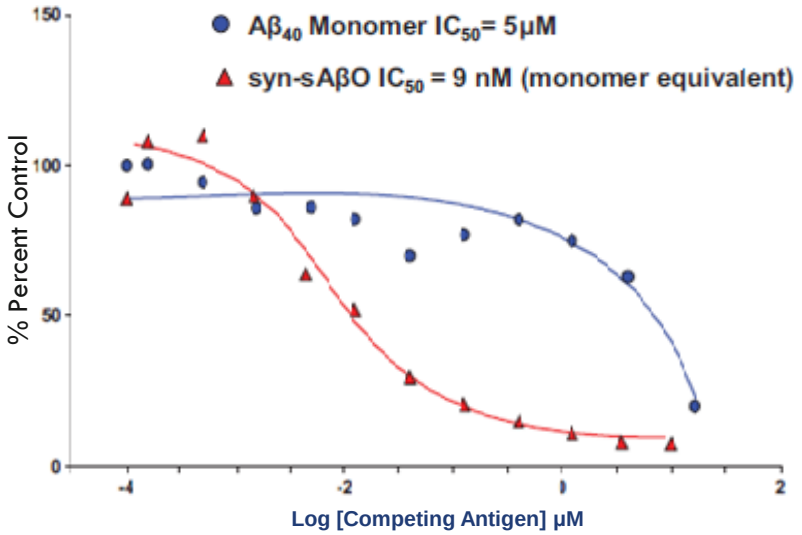
ACU193 is the First mAb Developed to Selectively Target AβOs

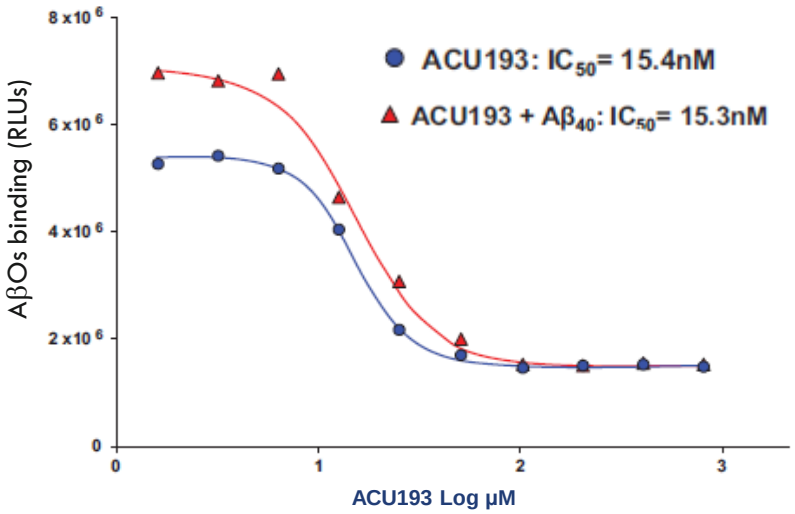
Highly selective for Aβ oligomers versus Aβ monomers

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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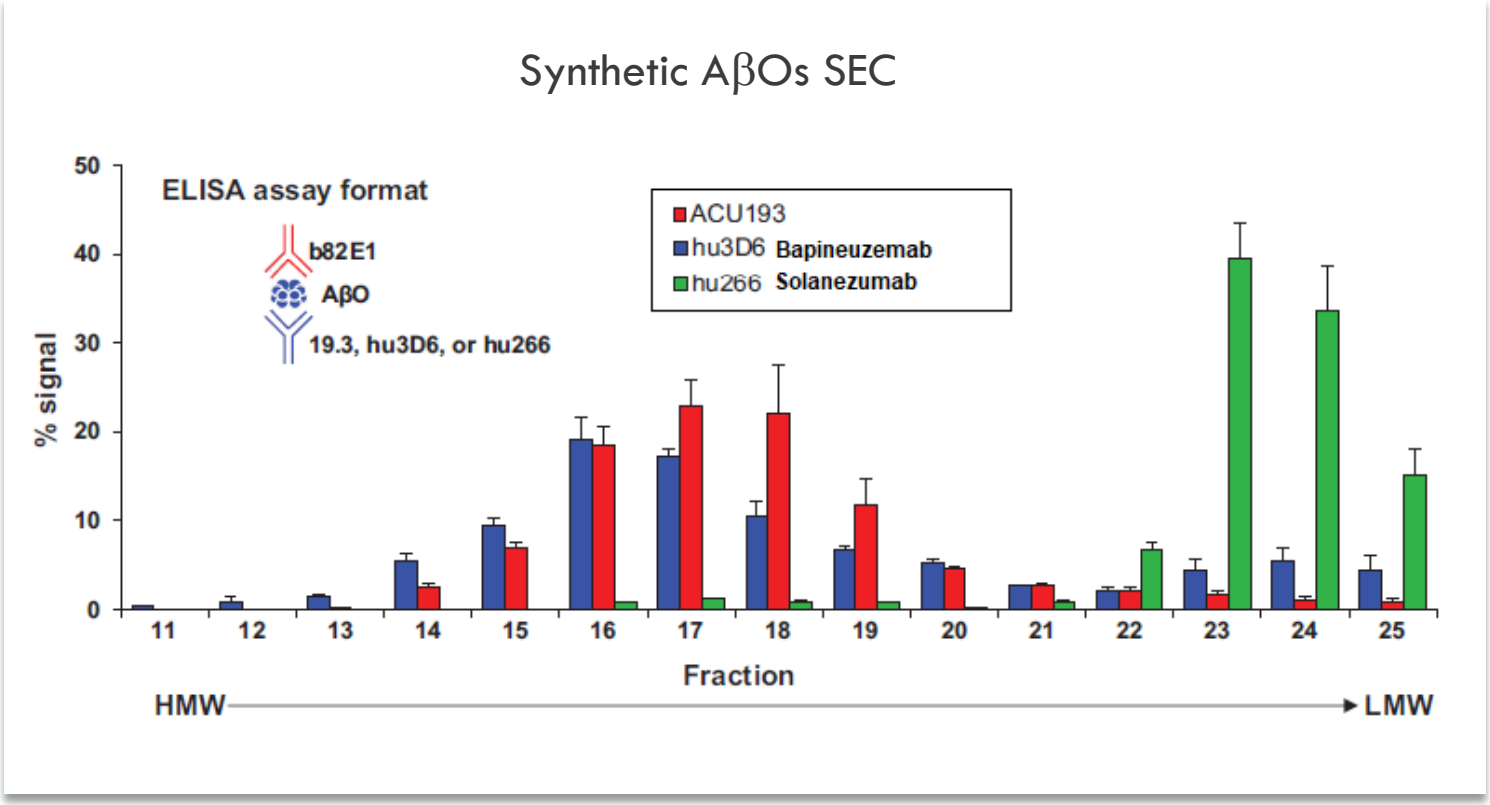
ACU193 has a greater preference for AβOs than other mAbs

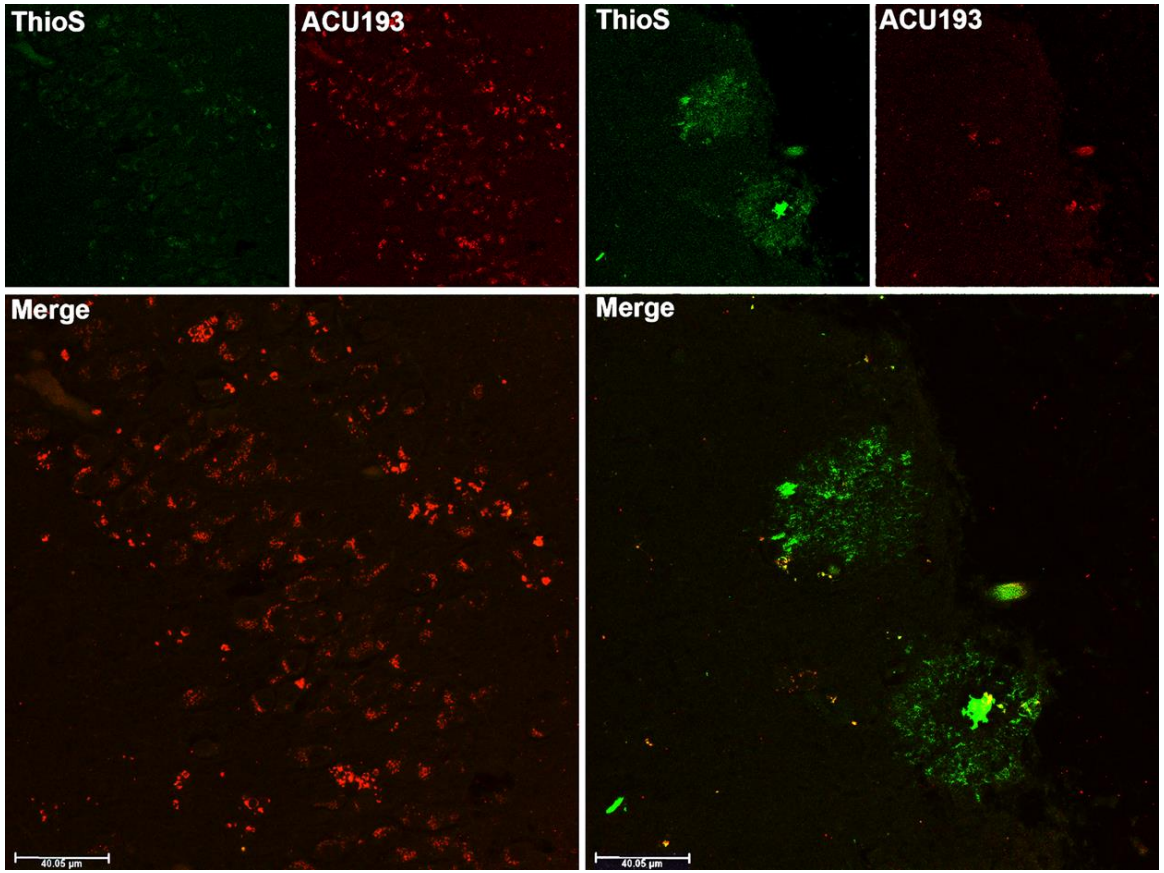
Comparison of Aβ species-mAb complex signals across SEC fractions



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

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

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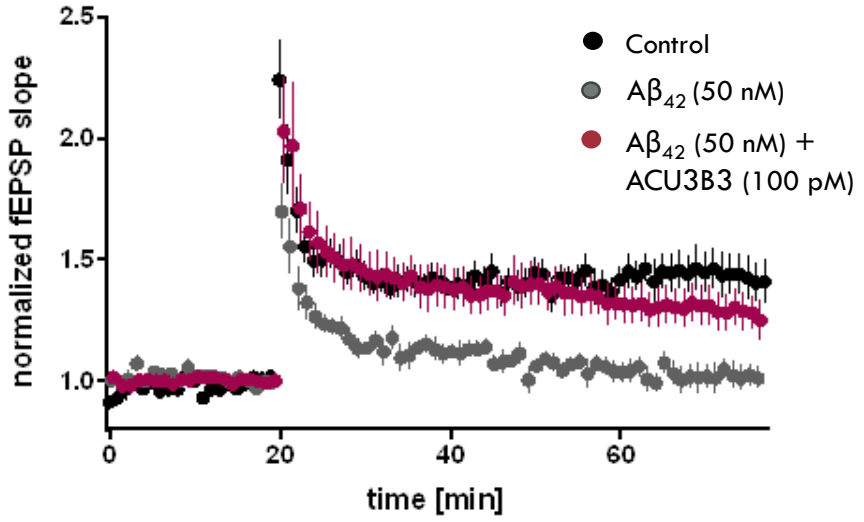
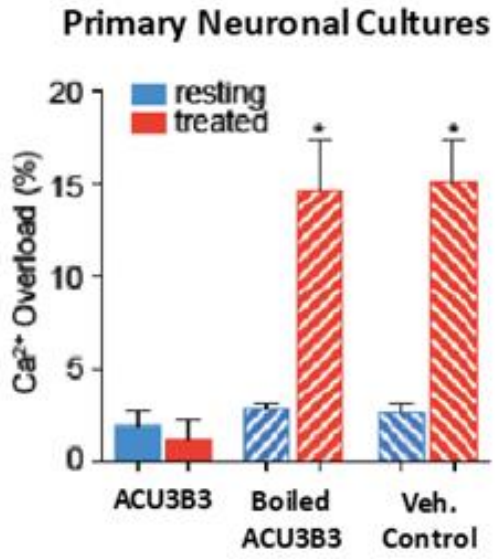
AβOs Bind to Neurons and are Toxic; mouse analogue of ACU193 prevents toxicity

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

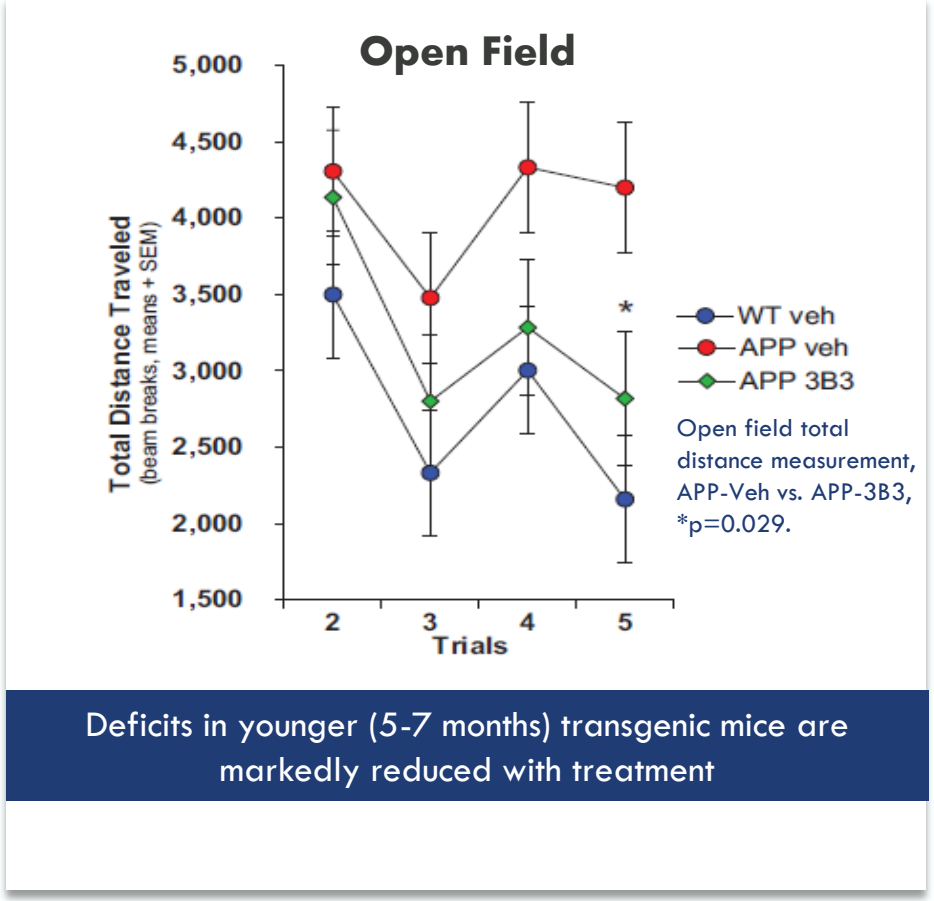


Note: (1) ACU3B3 is the mouse monoclonal antibody precursor to and equivalent of humanized ACU193

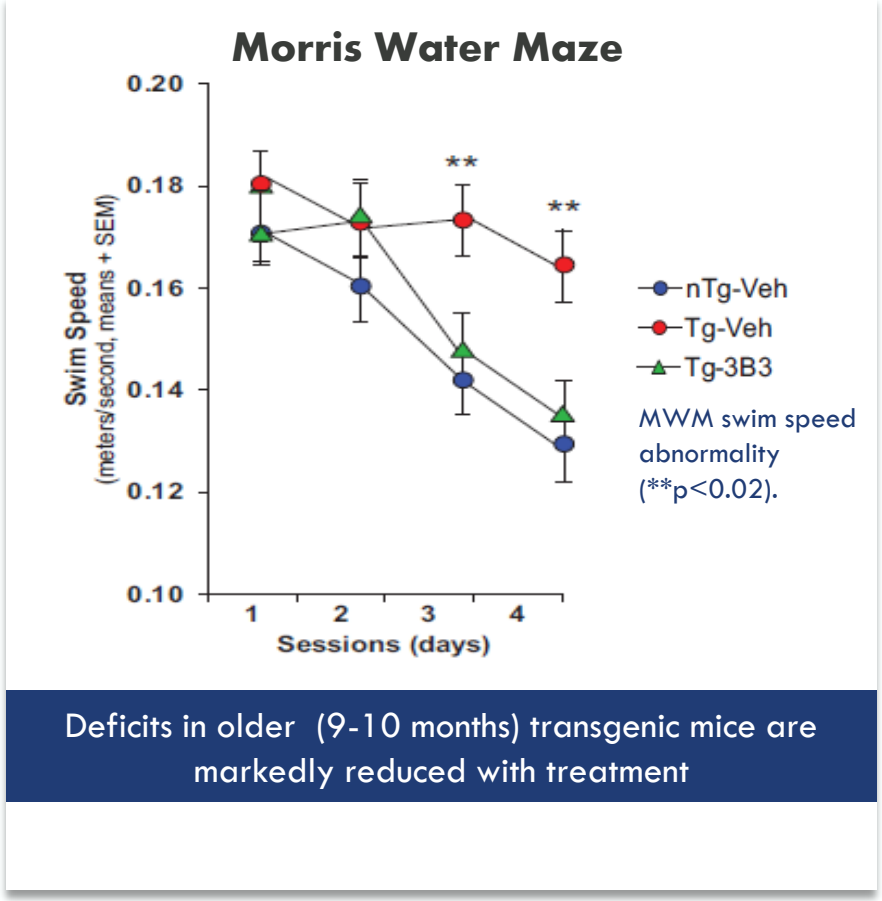
ACU3B3 prevents changes in aberrant neuronal activity underlying memory loss in AD and prevents AβO mediated disruption of calcium homeostasis in neuronal cultures

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

Murine parent version of ACU193 (ACU3B3) 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

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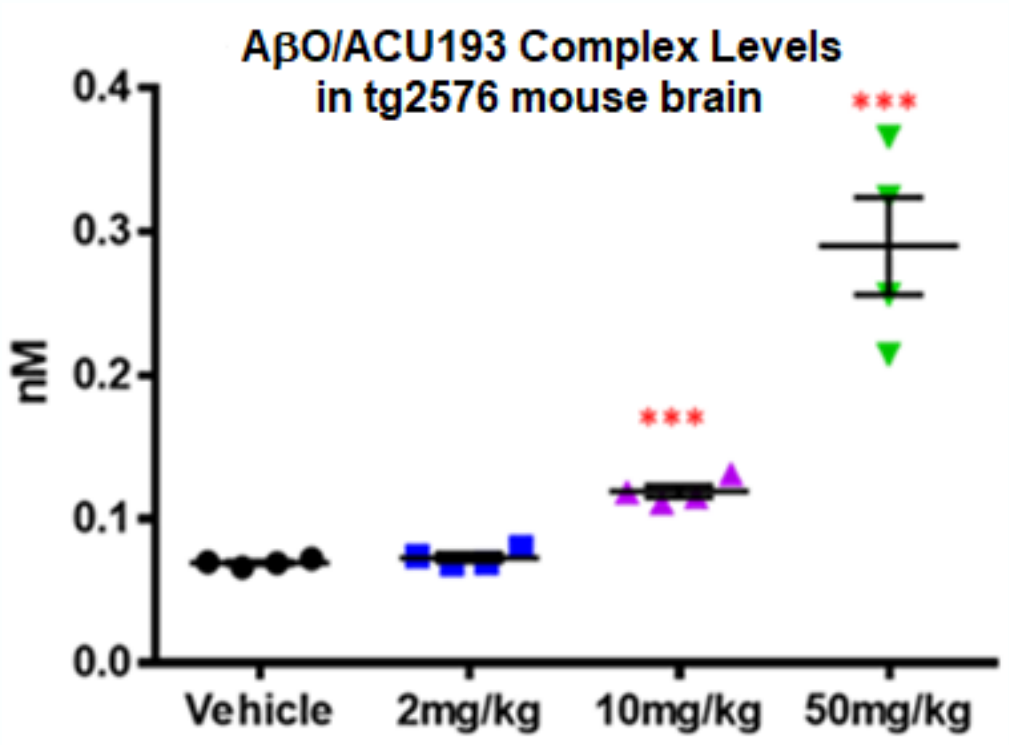
ACU193 Enters the CNS and Binds to AβOs in Transgenic Mice in Dose Dependent Manner



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

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(ACU-001) INTERCEPT-AD trial: Phase 1 Overview

TRIAL DESIGN:

Randomized Placebo Controlled Phase 1

- 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

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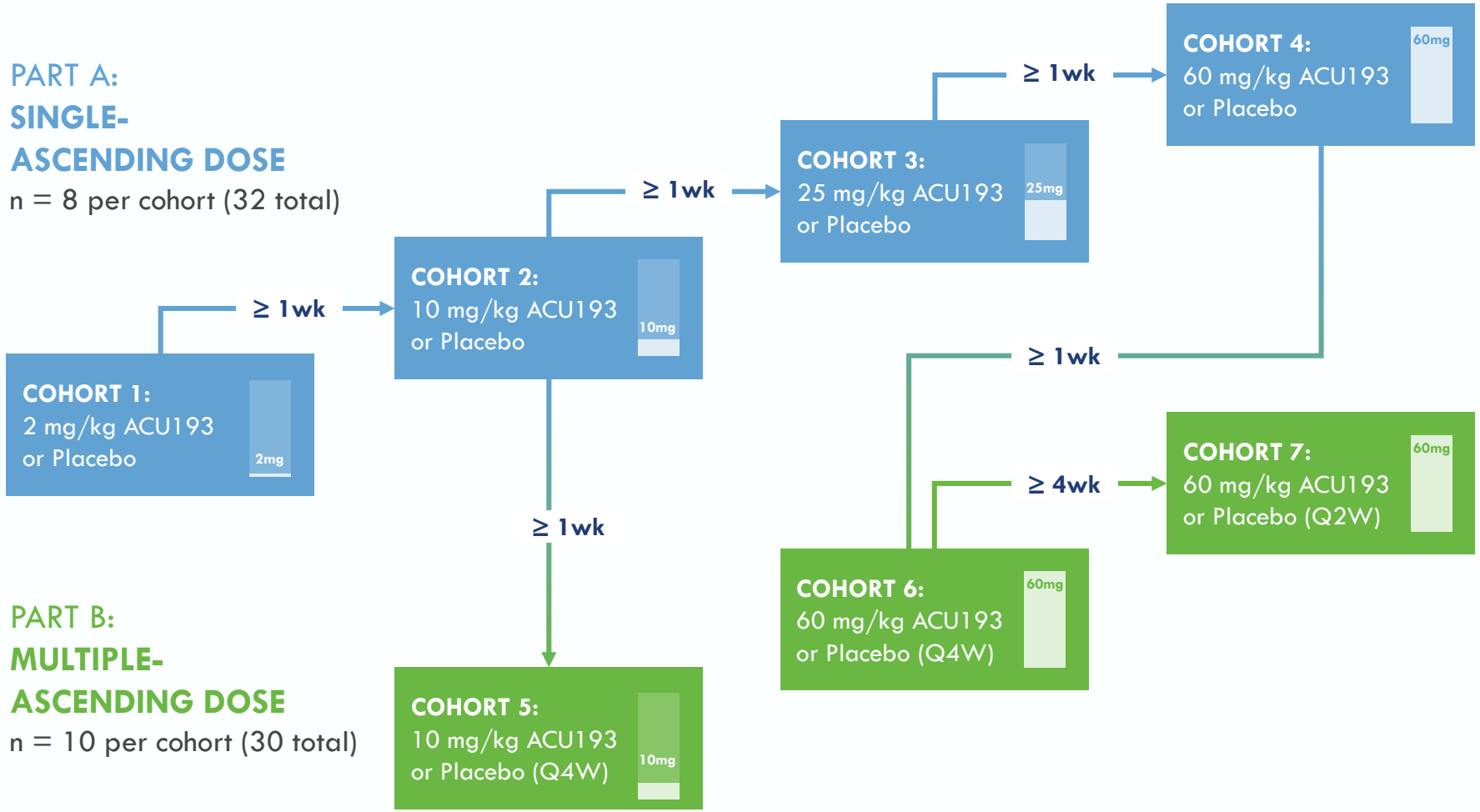
INTERCEPT-AD a Randomized Placebo Controlled Phase 1 in Early AD patients



Phase 1 Objectives: Proof of Mechanism –Ability to move to Phase 2/3

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

Topline Results anticipated in 1H 2023: primary outcomes Safety / ARIA-E, PK and Target Engagement. Detailed study results anticipated to be presented at major Alzheimer's meeting

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Business Considerations

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Experienced in AD/neuro drug development

ACUMEN LEADERSHIP TEAM

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ACUMEN
neuroventures

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Chief Medical Officer
ACUMEN
Lilly

JANICE HITCHCOCK, PHD
VP Regulatory Affairs
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MATT ZUGA
Chief Financial Officer &
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KENT IVERSON
CMC Leader
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AMGEN Genentech

SIEW TIN GAN
Head of Clinical
Operations
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Takeda Lundbeck

JASNA JERECIC, PHD
Analytical Methods
Leader, Research Scientist
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Acumen team has decades of experience in Alzheimer's drug discovery and development

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Experienced in AD Drug Development

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ACU193 IP & Market Exclusivity

- 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 19 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

Acumen is Well Capitalized, with Expected Cash Runway through 2025 to Achieve Multiple Anticipated Clinical Milestones

MILESTONES	STATUS/EXPECTED TIMING
Initiated Ph1 clinical trial INTERCEPT-AD	✓
INTERCEPT-AD Trial updates	2022
Proof of Mechanism Topline Results	1H 2023

~$225M

Cash and marketable securities as of December 31, 2021

We believe Acumen has the organizational expertise and cash and marketable securities on hand to advance ACU193 through multiple anticipated clinical milestones 2022 through 2025

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ABOS: Key Take aways

- Massive unmet need in AD
- Upcoming sector catalysts 2H22
- Differentiated Product Candidate
- Experienced team
- Blue chip investors, Very strong balance sheet and cash runway
- Value-inflection clinical data 1H2023

Thank you!

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