BIOARCTIC AB (PUBL)
NASDAQ STOCKHOLM: BIOA B

Presentation of BioArctic

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BIOARCTIC

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BIOARCTIC

BioArctic – a unique Swedish biopharma company
Improving life for patients with central nervous system disorders

High unmet need for disease-modifying treatments for Alzheimer's and Parkinson's diseases creates **large commercial opportunity**

World-class research and development driven organization with basis in founder's breakthrough discoveries and fruitful collaborations with leading **academic researchers** and **pharma companies** generating and developing **innovative projects**

Attractive and well-balanced project portfolio with projects from discovery through Phase 3 and combination of both proprietary projects with substantial marketing and out-licensing potential and partnered projects generating income

Well-financed with approximately BSEK 1 (MUSD >100[1]) in cash, **net profitable** during the last seven years and **valuable collaboration agreements** totaling BSEK 9.6[2] (BUSD ~1) plus royalties

1) FX as per September 30, 2020
2) FX as per December 31, 2019

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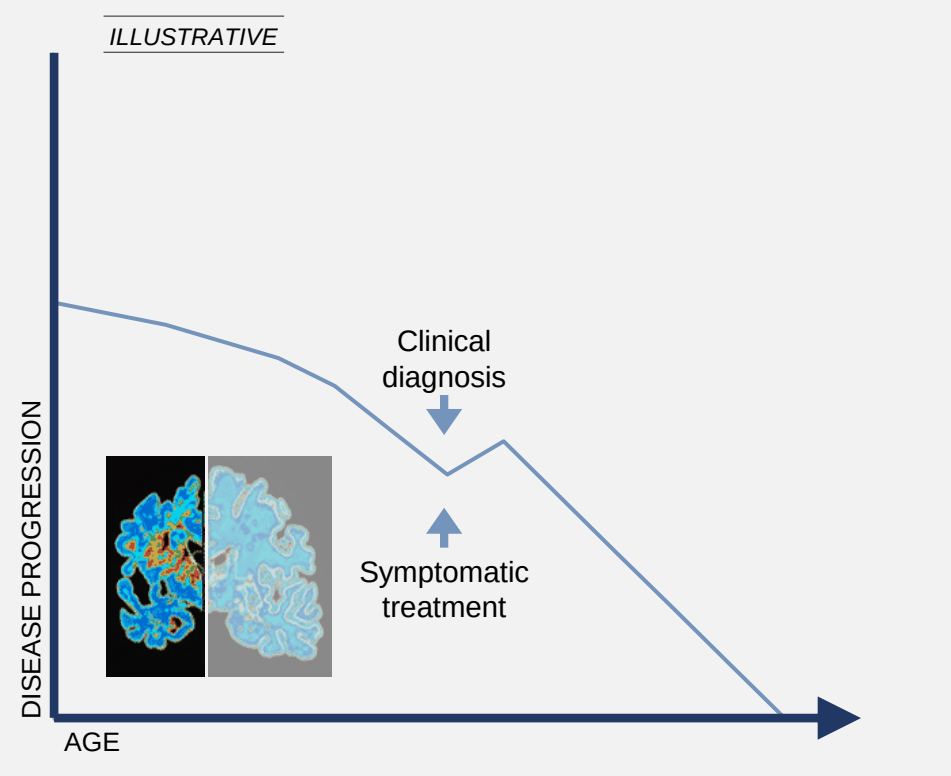
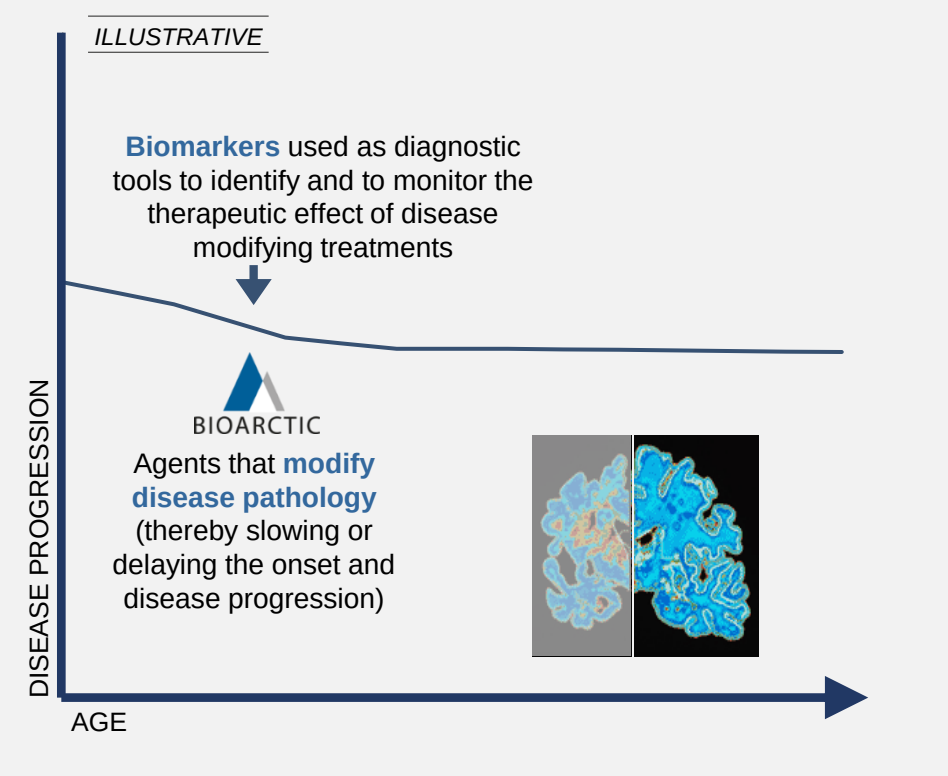
We focus on disease modifying agents and diagnostics/biomarkers for neurodegenerative diseases

Neurodegenerative disease therapy TODAY



Neurodegenerative disease therapy TOMORROW



Significant unmet medical need to be addressed by disease modifying agents and reliable diagnostics/biomarkers

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Attractive and well-balanced project portfolio combines fully-financed partner projects and cutting-edge proprietary projects

	Project	Partner	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
ALZHEIMER'S DISEASE	BAN2401 *(Clarity AD)*	Eisai[1]	Early Alzheimer's disease[4]				
	BAN2401 *(AHEAD 3-45)*	Eisai[1]	Preclinical (asymptomatic) Alzheimer's disease[5]				
	BAN2401 back-up	Eisai					
	AD1801						
	AD1502						
	AD1503						
	AD2603						
PARKINSON'S DISEASE	ABBV-0805[2]	AbbVie					
	PD1601	AbbVie					
	PD1602	AbbVie					
OTHER CNS DISORDERS	BAN2401		Down's syndrome[3] Traumatic brain injury[3]				
	ND3014						
BLOOD-BRAIN BARRIER TECHNOLOGY	BBB technology platform						
DIAGNOSTICS	Imaging and biochemical biomarkers – Alzheimer's disease						
	Imaging and biochemical biomarkers – Parkinson's disease	AbbVie					

as of September 30, 2020

1) Partnered with Eisai for BAN2401 for treatment of Alzheimer's disease. Eisai entered partnership with Biogen regarding BAN2401 in 2014
2) AbbVie in-licensed BAN0805 in late 2018 and develops the antibody with the designation ABBV-0805
3) Dementia and cognitive impairment associated with Down´s syndrome and with traumatic brain injury
4) Mild cognitive impairment due to Alzheimer's disease and mild Alzheimer's disease
5) Normal cognitive function with intermediate or elevated levels of amyloid in the brain

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Long-standing and extensive partnerships

Alzheimer's disease

Partner track record

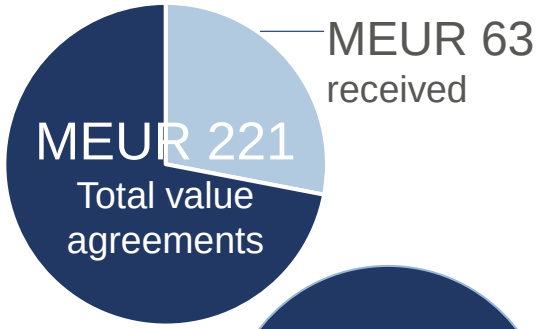
Eisai

Discovered and developed world's best-selling medicine for symptoms in Alzheimer's

Aricept donepezil HCl tablet

Industry-leading pipeline in dementia area

Collaboration and license



Royalties
High single digit %

- BioArctic retains rights to BAN2401 in other indications and option to market in the Nordics

Parkinson's disease

Partner track record

abbvie

World's all-time best-selling medicine (BUSD 20)

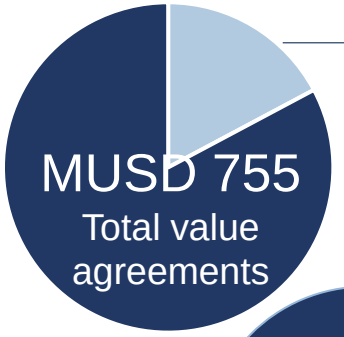
HUMIRA adalimumab

10 different indications in immunology

Approved product for symptoms associated with Parkinson's disease

Duopa carbidopa/levodopa

Collaboration and license



Royalties
Tiered %

- AbbVie global rights to alpha-synuclein portfolio for all indications

Sources: Eisai, AbbVie and BioArctic corporate information

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BAN2401 (lecanemab): potential disease modifying antibody for Alzheimer's disease with unique binding profile

High unmet medical need

No existing disease-modifying treatment

IN 20 YEARS
doubling

TODAY
>30 million
people with Alzheimer's

BAN2401 unique profile – selectively binding toxic protofibrils

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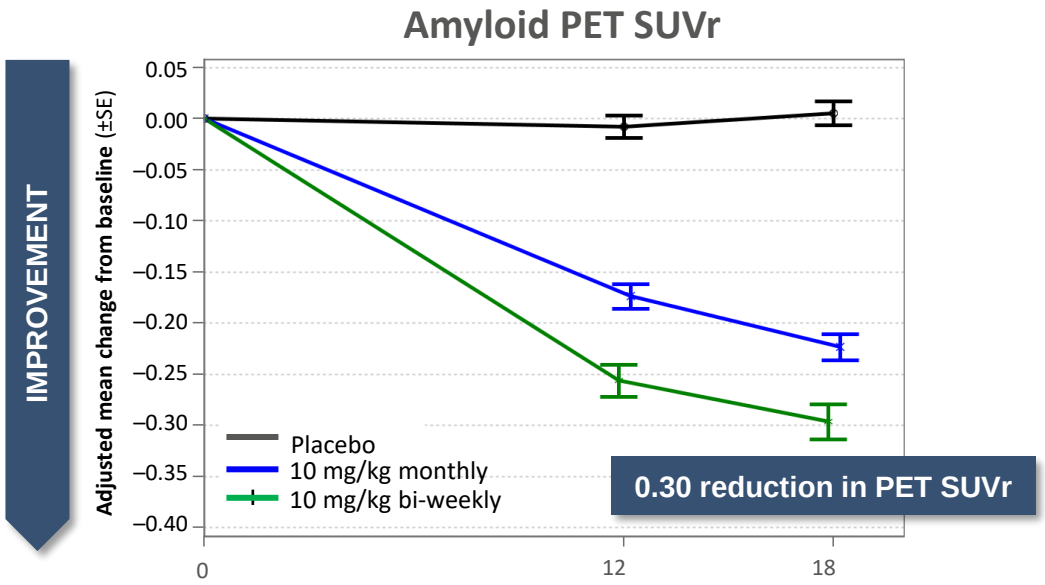
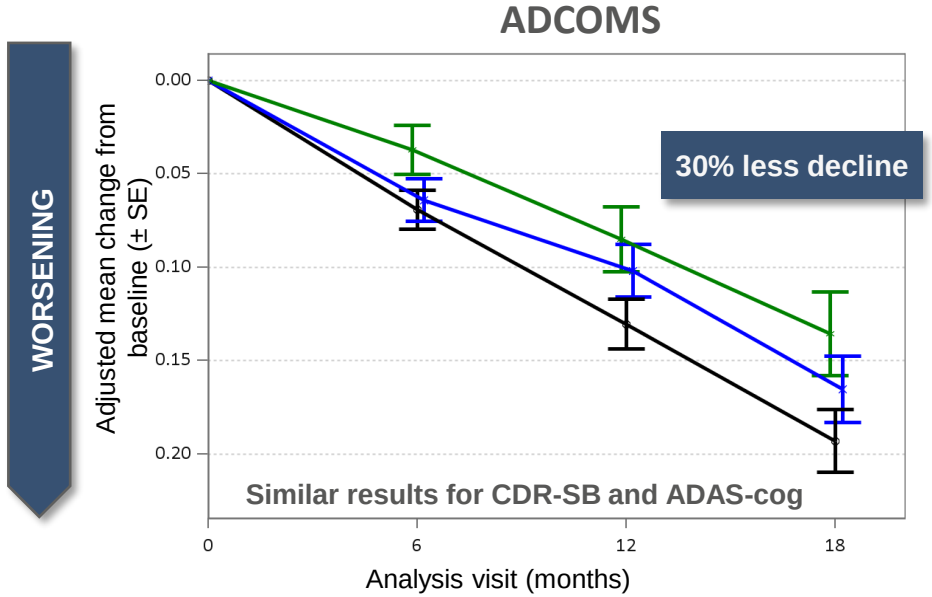
BAN2401: potential disease modifying antibody for Alzheimer's disease with positive Phase 2b results





BAN2401 has positive Phase 2b results

- **Large trial:** 856 early Alzheimer's patients
- **Consistent effects** on clinical outcomes, imaging and neurodegenerative biomarkers
- **Rapid onset** of clinical effect
- **Effect increase over time**
- **Good safety profile –** no titration required due to low frequency of ARIA–E (<10%)

PET: positron emission tomography

Presented at the Clinical Trials on Alzheimer's Disease Conference 2018; Barcelona, Spain. October 25, 2018

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Broad BAN2401 clinical program – driven by BioArctic's partner Eisai

Disease stage

Preclinical AD	MCI	Mild AD	Moderate	Severe

FDA guideline

Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6

AHEAD 3-45 Phase 3 Program – ongoing

- Eisai and ACTC started new Phase 3 study in 2020
- A total of 1400 participants will be enrolled in the study.
- Pre-clinical, asymptomatic, AD subjects with intermediate or elevated amyloid levels in the brain

Clarity AD Phase 3 confirmatory study – ongoing

- Primary endpoint readout expected 2022
- Target of 1566 early AD patients

OLE Phase 2b open-label extension study – ongoing

- Approximately 200 patients with early AD
- Baseline data presented in December 2019 showed maintenance of benefit after BAN2401 treatment conclusion

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Recent highlights
Another Phase 3 study in Alzheimer's disease and new data at CTAD

BAN2401

- Recruitment of early Alzheimer patients for Clarity AD confirmatory Phase 3 study ongoing
  - CTAD presentation showed baseline characteristics consistent with Phase 2b study, and representative of an early Alzheimer's disease population
- Phase 2b open label extension study data presented at CTAD
  - Rapid reduction of amyloid in the brain (already at 3 months)
  - Consistent low level of adverse event ARIA-E (<10%)
- Phase 3 study in preclinical AD, AHEAD 3-45, started
  - CTAD presentation of study design and initial screening of individuals

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Why should we succeed where many others have failed?

Right target	Right patient population	Right dose & exposure	Right measurements	Right safety
• Address selectively the toxic protofibrils – soluble aggregated form of amyloid	• Early Alzheimer's – MCI due to AD & Mild AD • Identify right patients – biomarkers	• Select dose with effect and acceptable side-effect profile demonstrated in Phase 2 • No titration, same dose to all patients	• Sensitive cognition and function scales • Biomarkers for disease progression and disease modification	• Well tolerated with benign safety profile • Low risk for amyloid related imaging abnormalities (ARIA) and no expected cardiovascular risk

Right study design and performance	
	• Build confirmatory Phase 3 program on robust Phase 2b results with dose selection • Same patient population, dose, measurements • No interim analysis • Monitor variability to ensure study power

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ABBV-0805: potential disease modifying antibody for Parkinson's disease – in Phase 1 preparing for Phase 2

High unmet medical need

No existing disease-modifying treatment

Younger patient group, still at working age

TODAY

>6 million[1]

people with Parkinson's

Unique profile

Unique and targeted binding profile

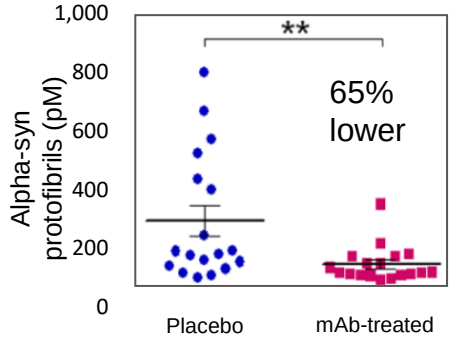
- Highly selective for toxic forms of misfolded alpha-synuclein (oligomers/protofibrils)

Built on genetic and pathology rationale

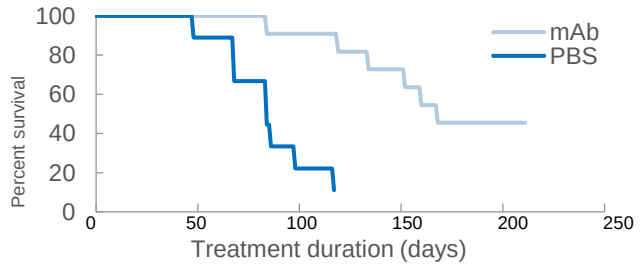
- Alpha-synuclein mutations lead to Parkinson's
- Alpha-synuclein oligomers/protofibrils are elevated in Parkinson's

Preclinical proof of concept

Reduction of neurotoxic alpha-synuclein oligomers/protofibrils



Delays disease progression and increases lifespan



1) Dorsey and Bloem, JAMA Neurology 2018;75:9-10

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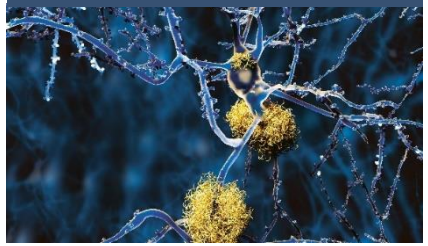
Early-stage portfolio continues to develop well

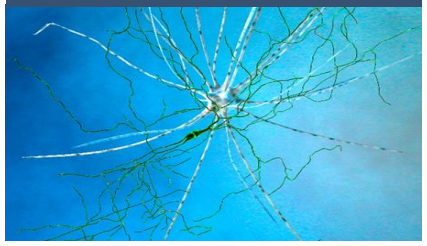
Alzheimer's disease



Discovery stage programs

- 4 fully-owned disease modifying antibody projects in Alzheimer's disease
- Each project has a different mechanism from the others

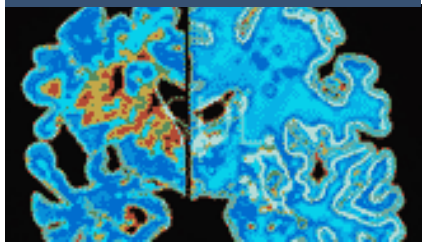
Parkinson's disease



Discovery stage projects

- Preclinical stage alpha-synuclein projects in research collaboration with abbvie

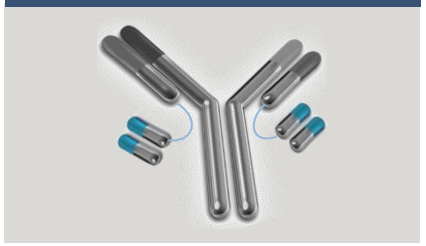
Other CNS disorders



Neurodegeneration research

- BAN2401 in indications other than Alzheimer's disease
- Research project in neurodegeneration ("ND") with potential in various CNS disorders

Blood-brain barrier technology



Blood-brain barrier technology platform

- Continued development with expanded and enhanced capabilities
- Collaboration with Uppsala University under Vinnova grant

Diagnostics

Diagnostics

- Continued development of imaging and biochemical biomarkers

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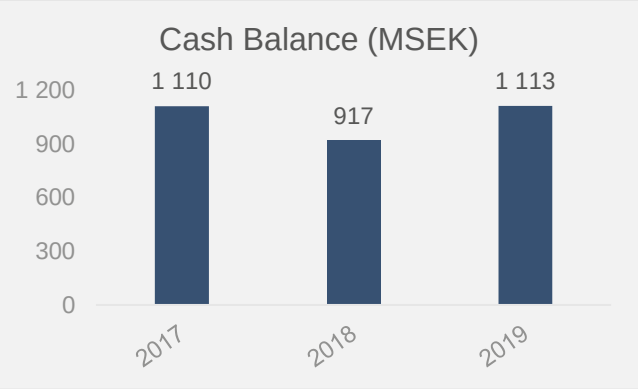
BioArctic has a strong financial profile

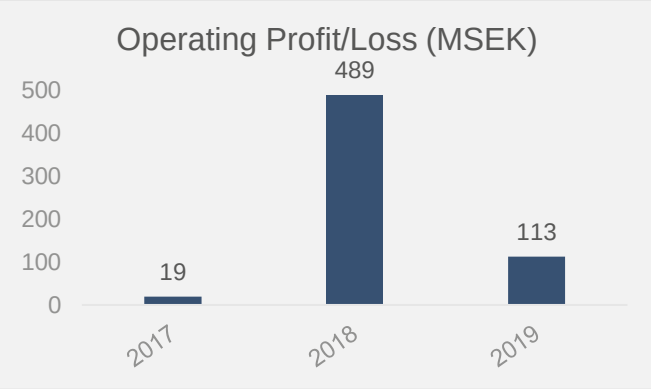
- Listed on Nasdaq Stockholm Mid Cap, market capitalization of SEKbn 6.1[1] (~650 MUSD)[2]

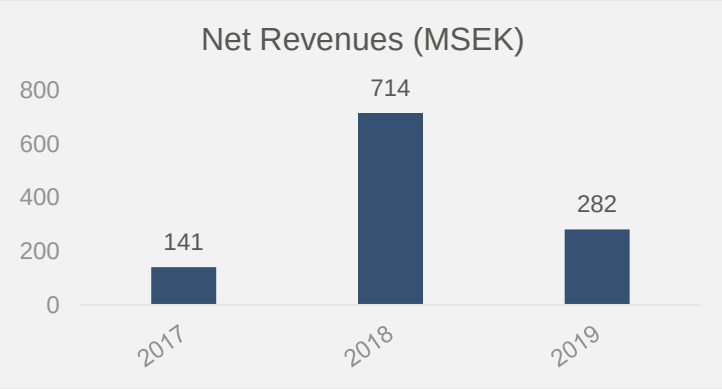
- More than 1 billion SEK (100 MUSD) in cash

- Net profit during the last 7 years
- Expected 2020 operating costs 150-170 MSEK

- Significant funding from partner research collaborations and license agreements, as well as grants
- Total potential collaboration deal value[3] of ~SEKbn 9.6 (~1 BUSD) of which ~SEKbn 1.9 (~0.2 BUSD) received
- Additional future royalties potential
- Milestone payments one-time nature explain fluctuations in financial results







1) As of April 21, 2020.
2) Calculated using relevant exchange rate as of April 21, 2020.
3) Calculated using relevant exchange rate as of December 30, 2019.

BioArctic: With Patients in Mind

Built on science

Projects in focus

	Project	Partner	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
ALZHEIMER'S DISEASE	BAN2401 (*Clarity AD*)	Eisai[1]	Early Alzheimer's disease[4]				
	BAN2401 (*AHEAD 3-45*)	Eisai[1]	Preclinical (asymptomatic) Alzheimer's disease[4]				
	BAN2401 back-up						
	AD1801						
	AD1502						
	AD1503						
	AD2603						
PARKINSON'S DISEASE	ABBV-0805[2]	AbbVie					
	PD1601	AbbVie					
	PD1602	AbbVie					
OTHER CNS DISORDERS	BAN2401		Down's syndrome[3] Traumatic brain injury[3]				
	ND3014						
BLOOD-BRAIN BARRIER TECHNOLOGY	BBB technology platform						
DIAGNOSTICS	Imaging and biochemical biomarkers – Alzheimer's disease						
	Imaging and biochemical biomarkers – Parkinson's disease	AbbVie					

Value-driven leadership

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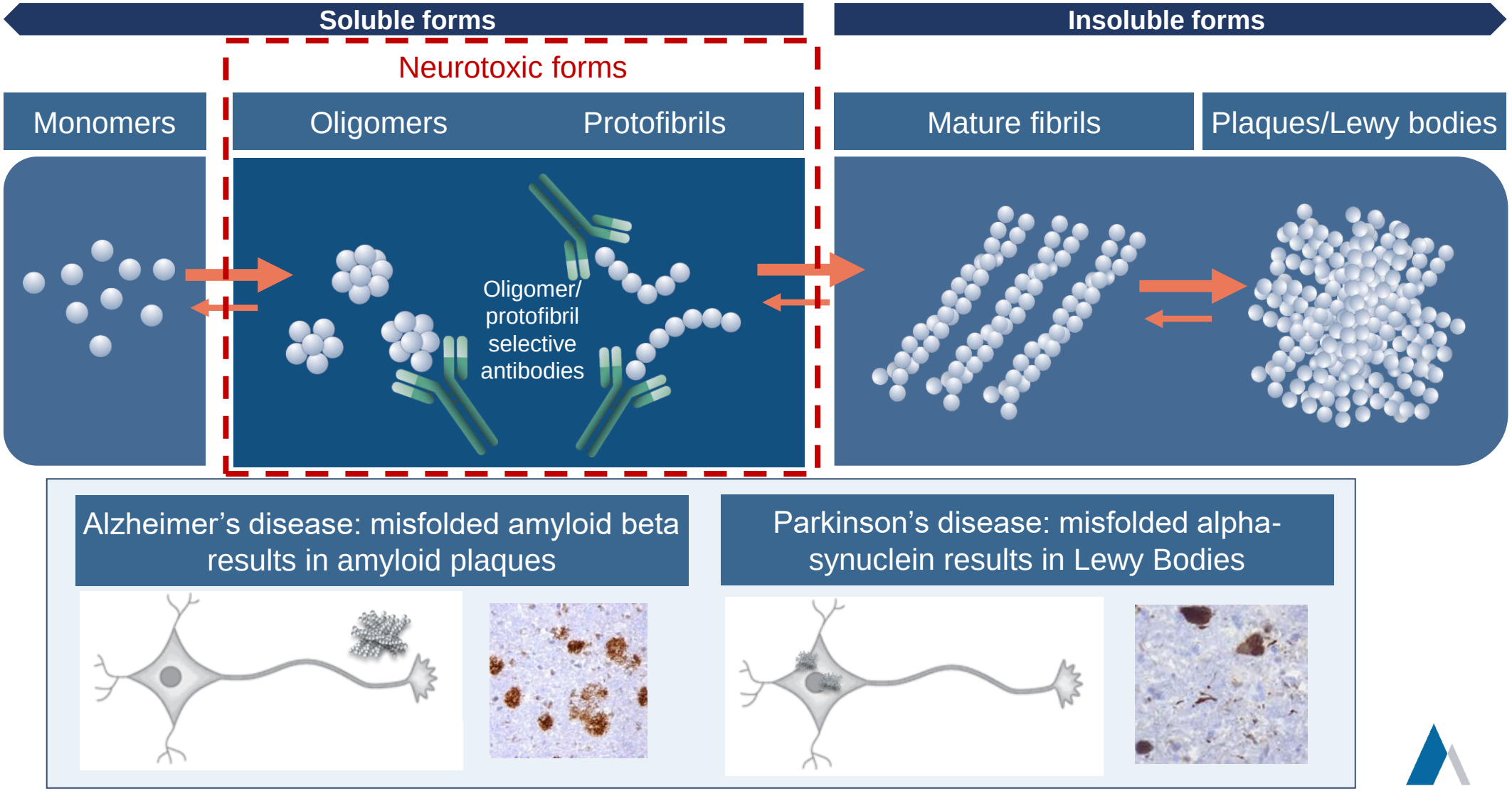
Questions?

Back up slides

Targeting neurotoxic forms of aggregated misfolded proteins is important when designing therapies for neurodegenerative diseases



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BAN2401 – Innovative Phase 2b study design
Positive 18-month results reported by Eisai

IMPORTANT PARAMETERS

Right target	Right patient population	Right dose & exposure	Right measurements	Right safety
• Address the soluble protofibrils – a toxic form of amyloid	• Early Alzheimer's – MCI due to AD & Mild AD • Identify right patients – biomarkers	• Selecting doses with exposures above preclinical IC50 • Adaptive design testing several doses and dose regimens	• More sensitive cognition scales • Biomarkers for disease progression and disease modification	• Well tolerated with benign safety profile • Low risk for amyloid related imaging abnormalities (ARIA) and no expected cardiovascular risk

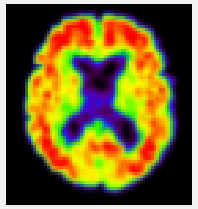
PHASE 2B STUDY DESIGN

Patient inclusion

Multinational recruitment:
- 100 clinical centers

Inclusion criteria:
- MMSE >22-30
- Stable concomitant medication
- Positive amyloid PET/CSF

Inclusion completed with 856 patients



Treatment 12 months

Double-blind, placebo controlled, parallel-group study with Bayesian adaptive design

- Placebo
- 2.5 mg/kg twice a month
- 5 mg/kg once a month
- 5 mg/kg twice a month
- 10 mg/kg once a month
- 10 mg/kg twice a month

Primary analyses:
- Change from baseline in ADCOMS at 12 months
- Safety and tolerability

Treatment 18 months

Key analyses:
- Change from baseline in ADCOMS, CDR-SB, ADAS-cog at 18 months
- Change from baseline in brain amyloid as measured by amyloid PET
- Change from baseline in CSF biomarkers
- Safety and tolerability

Source: Company information
Note: ADCOMS = Alzheimer's Disease Composite Score, an evaluation tool developed by Eisai

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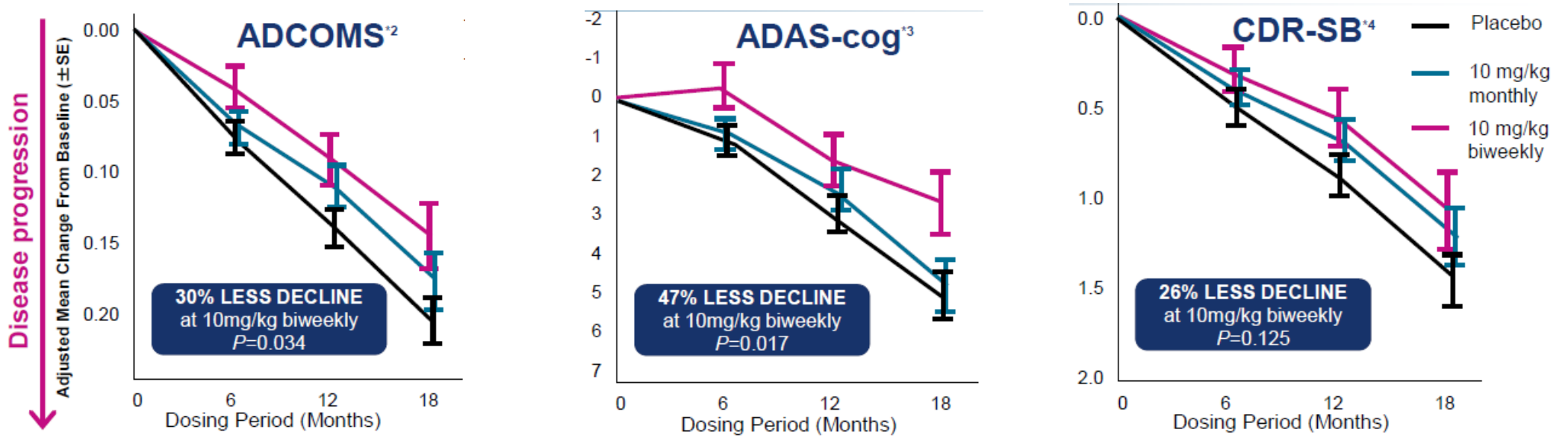
BAN2401 Showed Effect on Clinical Parameters

ADCOMS cognition scale (the key efficacy parameter)	ADAS-Cog (well-established cognition scale)	CDR-SB (cognition and function scale)



- Showed effect already at 6 months – as well as after 12 and 18 months of treatment
- Slowing of disease progression observed across sub-groups[1]
- Clinical effect increased over time

1) MCI due to AD – mild AD, ApoE4 carriers – non-carriers, with or without symptomatic treatment
2) ADCOMS – Alzheimer's Disease Composite Score
3) ADAS-Cog – Alzheimer's Disease Assessment Scale, cognitive subscale
4) CDR-SB – Clinical Dementia Rating – sum of boxes

Presented at CTAD October 2018 by Eisai

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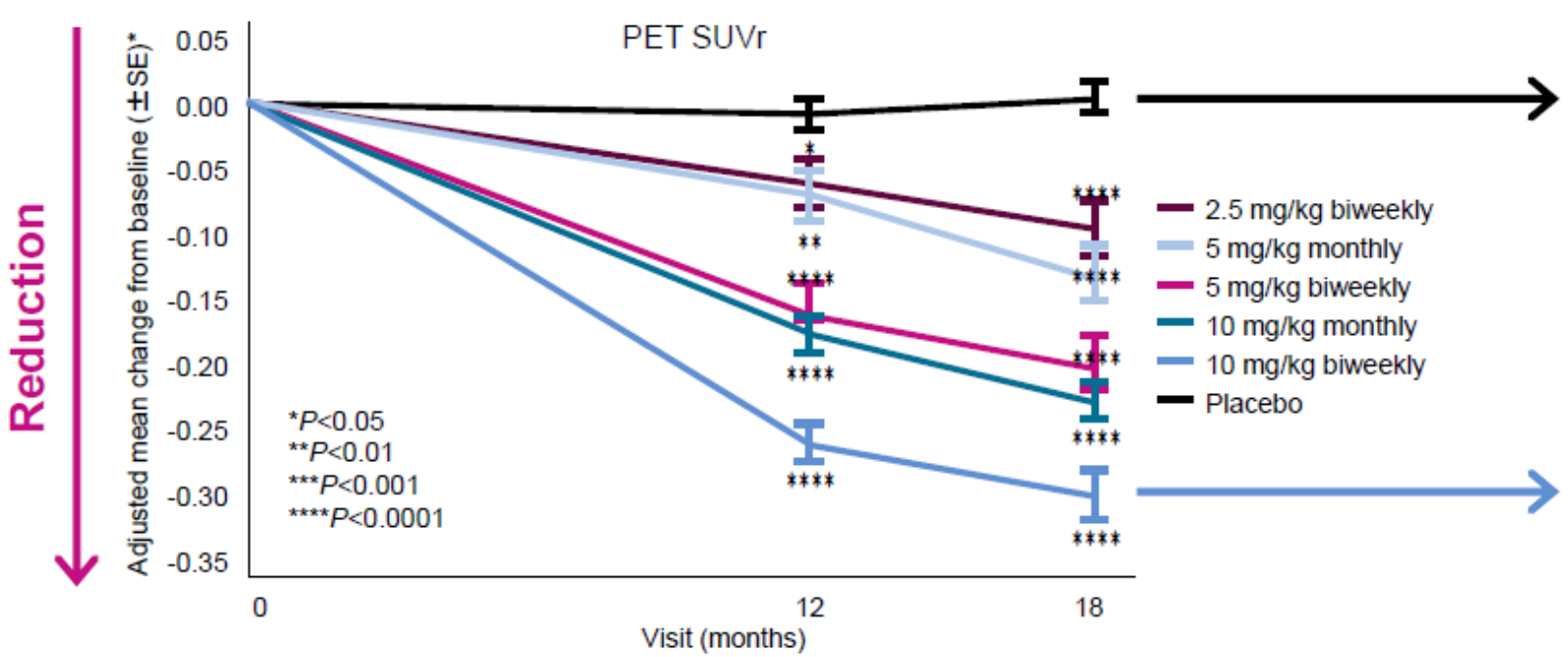
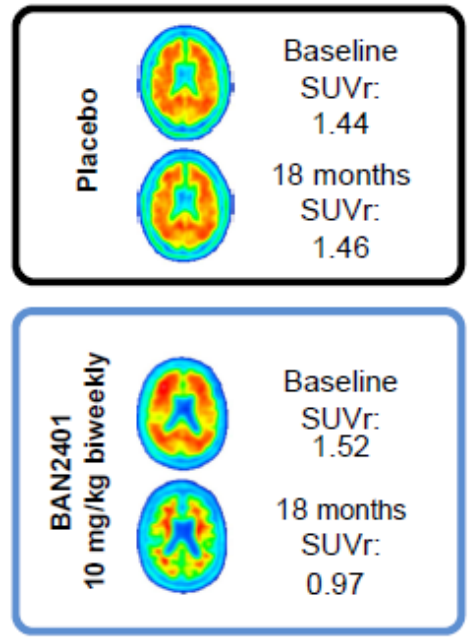
BAN2401 Reduced Amyloid Burden over 18 Months

Amyloid reductions were shown on two PET imaging assessments: PET visual read and PET SUVr

PET SUVr





93% of amyloid positive converted to negative at highest dose

Level for being amyloid positive: > 1.15 (SUVr)

PET visual read

81% of amyloid positive converted to negative at highest dose

Data presented at AD/PD March 2019 by Eisai

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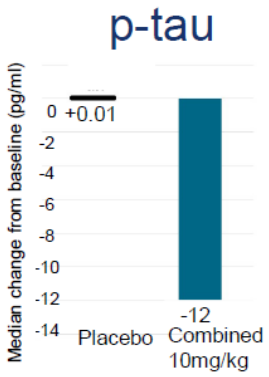
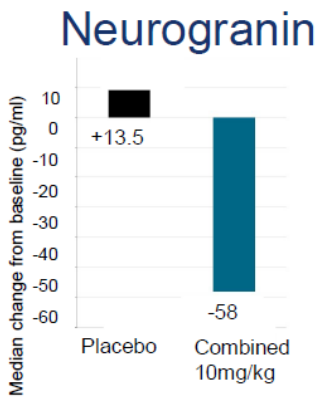
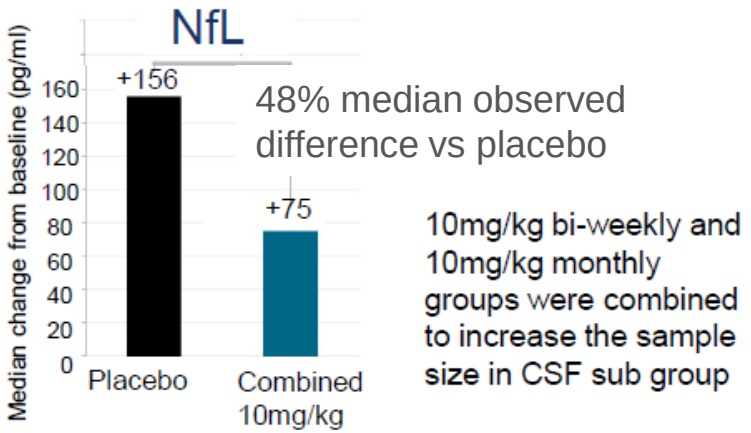
BAN2401 Showed Effects on CSF Markers of Neurodegeneration Supporting a Disease Modifying Treatment

CSF Biomarkers:
- t-tau
- p-tau
- neurogranin
- NfL

- Neurodegenerative markers show effect of BAN2401 on underlying pathophysiology
  - Reduction in t-tau (neuron loss)
  - Reduction in p-tau (neuronal damage)
  - Reduction in neurogranin (synaptic damage)
  - Reduction in increase of Neurofilament Light (NfL) (axonal degeneration)







Presented at AAIC July 2018 and CTAD Oct 2018 by Eisai

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Similar low levels of ARIA-E[1] in OLE as in BAN2401 Phase 2b core study ~10%

BAN2401 was generally **well-tolerated** in core study with infusion reactions and ARIA as the most common side effects (mostly mild to moderate)

Phase 2b study – ARIA-E incidence

- <10% at any dose
- <15% in APOE4 carriers at the highest dose
- Only ~10% of ARIA-E cases (5/48) reported symptoms including headache, visual disturbances or confusion
- Reversible and MRI findings typically resolved within 4-12 weeks

Phase 2b OLE study – ARIA-E incidence

Observed cases of ARIA-E in OLE (10 mg/kg biweekly)

- <8% to date
- <9% to date in core placebo-treated
- 13% in core placebo-treated ApoE4 carriers
- Reversible and MRI findings typically resolved within 4-12 weeks

[1] ARIA-E, Alzheimer's Related Imaging Abnormality-Edema

Presented at AAIC July 2018 by Eisai and at CTAD November 2020

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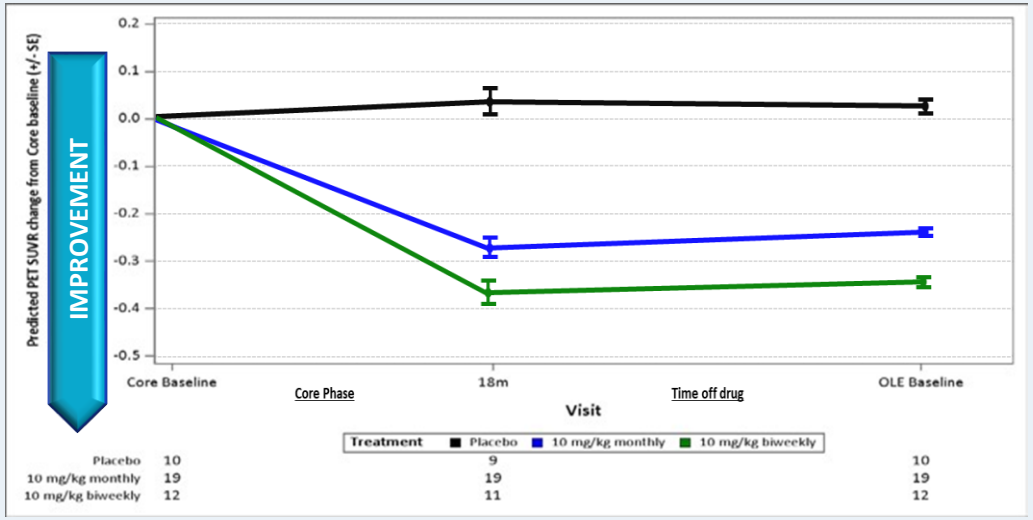
BAN2401 OLE data further supports effects in the core Phase 2b study

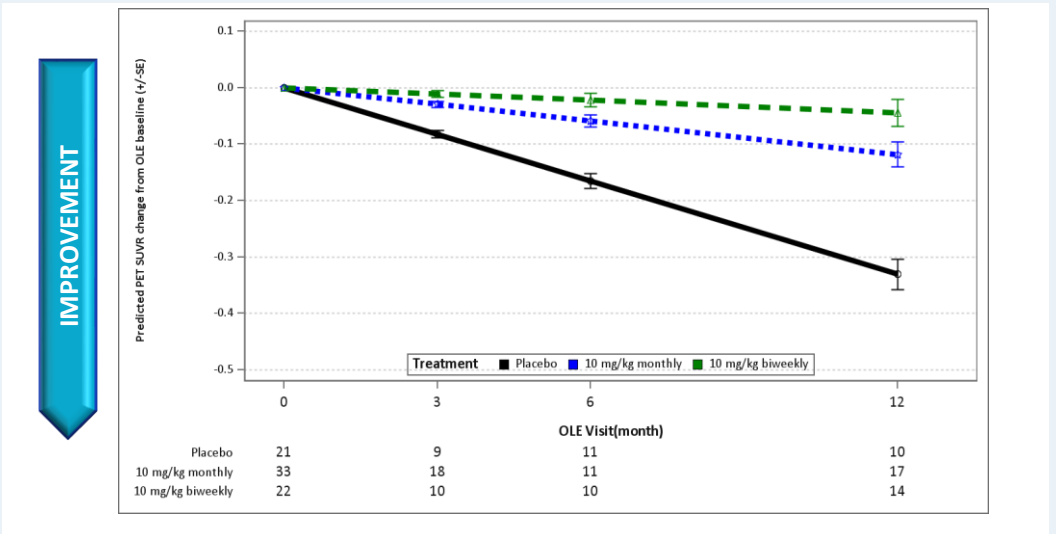
Amyloid reduction in the brain from BAN2401 treatment persisted after treatment conclusion

Amyloid PET SUVr in placebo and both 10 mg/kg groups: Phase 2b Core versus OLE Baseline



Rapid brain amyloid reduction from BAN2401 treatment in previous placebo patients

Amyloid PET SUVr in placebo and both 10 mg/kg groups: Phase 2b OLE Baseline versus OLE 3, 6 and 12 months



Phase 2b core study placebo patients entered OLE with high brain amyloid levels. A rapid decrease in amyloid levels was observed already after 3 months BAN2401 treatment. Further decreases were observed after 6 and 12 months treatment.

Phase 2b core study BAN2401 10 mg/kg patients entered OLE with low brain amyloid levels, which remained low .

Presented at CTAD December 2019 and at CTAD November 2020 by Eisai

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BAN2401 - Eisai´s Phase 3 study “Clarity AD” designed to confirm the positive Phase 2b results

IMPORTANT PARAMETERS

Right target

- Address the soluble protofibrils – a toxic form of amyloid

Right patient population

- Early Alzheimer’s – MCI due to AD & Mild AD
- Identify right patients – biomarkers

Right dose & exposure

- Top dose in Phase 2b study demonstrated positive effects

Right measurements

- Cognition scales
- Biomarkers for disease progression and disease modification

Right safety

- Well tolerated with a benign safety profile
- Low levels of amyloid related imaging abnormalities (ARIA), reversible and mostly without symptoms

PHASE 3 STUDY DESIGN

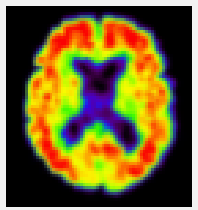
Patient inclusion

Global recruitment:
US, EU, Asia

Inclusion criteria:

- MCI due to AD or mild AD
- Positive amyloid PET/CSF

1566 Early Alzheimer patients



Treatment 18 months

Randomized, double-blind, placebo controlled, parallel-group study

- BAN2401 10 mg/kg twice a month
- Placebo

Read-outs at 18 months

Primary analysis:
Change from baseline in CDR-SB

Key analyses:

- Change from baseline in ADCOMS, ADAS-cog
- Change from baseline in brain amyloid measured by amyloid PET
- Change from baseline in CSF biomarkers: t-tau, p-tau, neurogranin, Neurofilament light
- Safety and tolerability

Source: Company information
Note: ADCOMS = Alzheimer’s Disease Composite Score, an evaluation tool developed by Eisai

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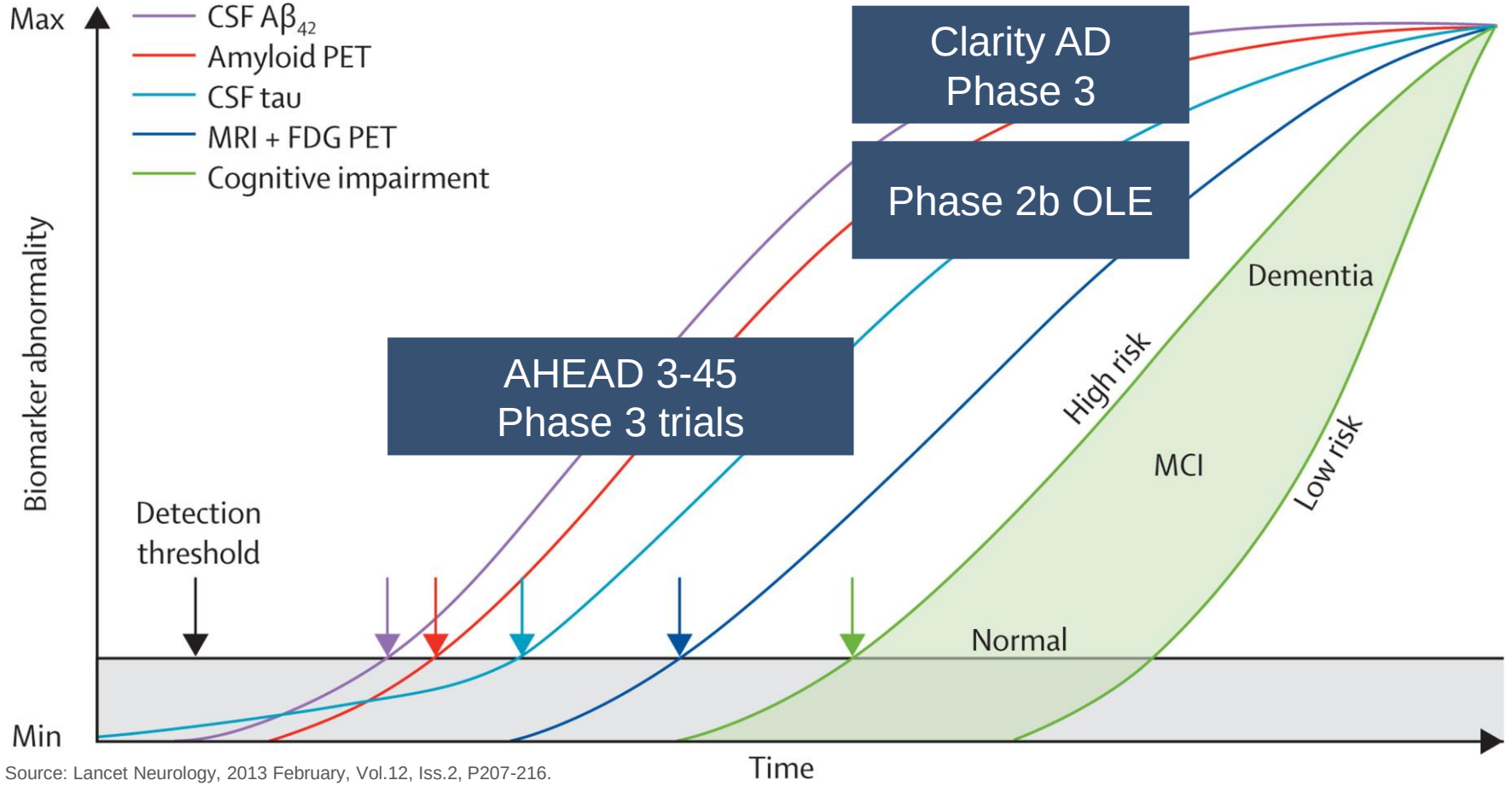
Broad BAN2401 clinical program



Source: Lancet Neurology, 2013 February, Vol.12, Iss.2, P207-216.

Populations:

Preclinical AD	Early AD (MCI and Mild AD)	Moderate	Severe

1) PACC5: Preclinical Alzheimer's Disease Cognitive Composite 5

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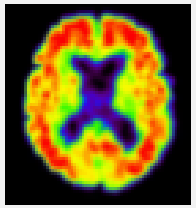
BAN2401 – Eisai and ACTC´s Phase 3 study “AHEAD 3-45” in pre-clinical Alzheimer´s disease

AHEAD 3-45 PHASE 3 STUDY DESIGN

Patient inclusion → Treatment 4 years → Read-outs

Preclinical stages of AD
Inclusion criteria:

- A45: no or limited cognitive decline and elevated amyloid
- A3: cognitively normal, intermediate amyloid in brain



Amyloid PET
A45: elevated
A3: intermediate

No of subjects

- A45 up to 1,000
- A3 up to 400

Recruitment geographies:
US, Japan, Australia, EU

Randomized, double-blind, placebo controlled, parallel-group study

A45	BAN2401	
	Placebo	
A3	BAN2401	
	Placebo	

Primary endpoints:
A45: PACC5
A3: Abeta PET

Other endpoints:
A45: Biomarkers
A3: Biomarkers, PACC5
Safety and tolerability

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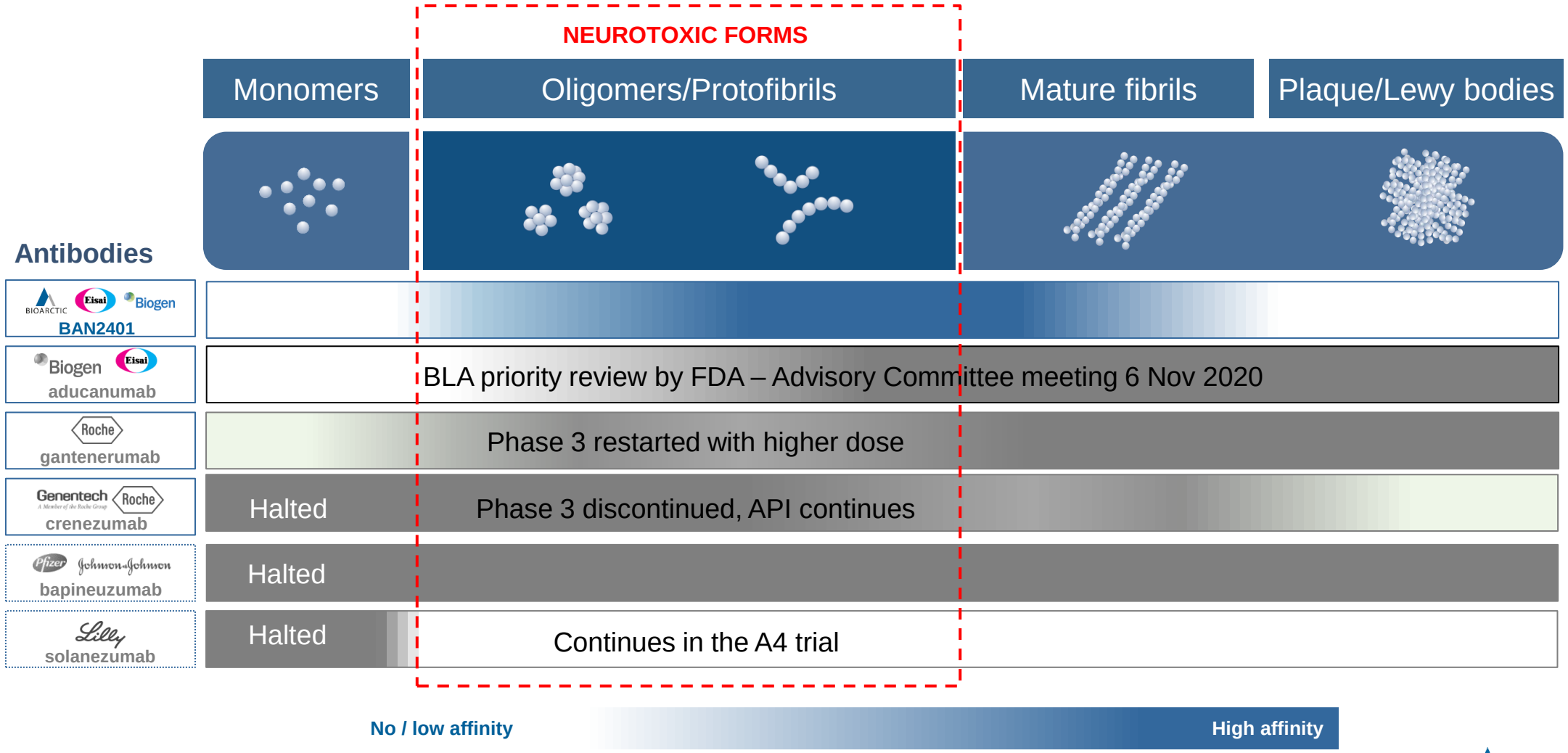
BAN2401 differentiated binding profile



Source: Binding profiles of aducanumab, gantenerumab, crenezumab, bapineuzumab and solanezumab are interpretations based on information disclosed by the respective company.

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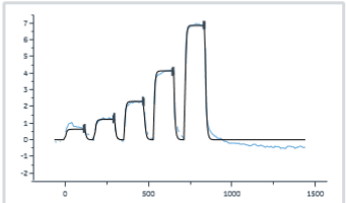
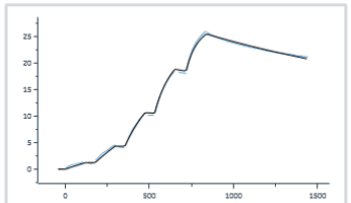
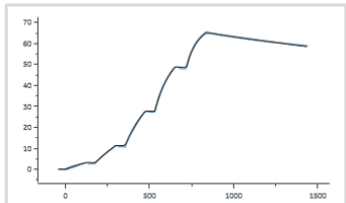
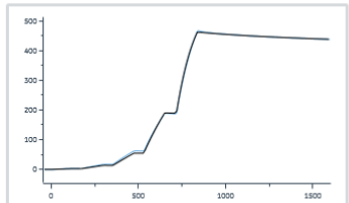
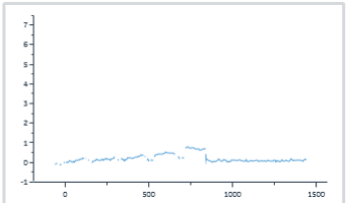
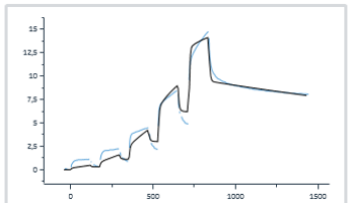
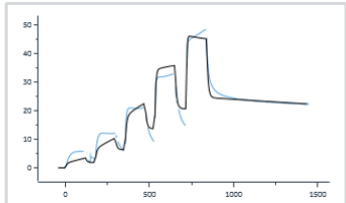
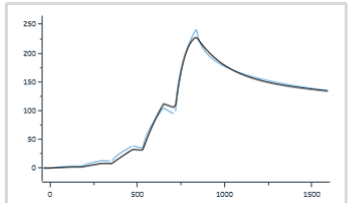
BAN2401 showed stronger binding than aducanumab to all Aβ species, especially to protofibrils

Surface Plasmon Resonance confirmed data from inhibition ELISA

	Monomers	Small protofibrils	Large protofibrils	Fibrils
BAN2401	 K_{D1} : 3980 nM	 K_{D1} : 1.32 nM	 K_{D1} : 0.54 nM	 K_{D1} : 6.82 nM
aducanumab	 K_{D1} : >5000 nM	 K_{D1} : 138 nM	 K_{D1} : 45 nM	 K_{D1}: 31.2 nM
Fold difference:	**-**	**105**	**83**	**4.4**

Presented by Lars Lannfelt at CTAD, December 7, 2019

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Continued progress in collaboration with AbbVie on alpha-synuclein

Collaboration highlights

- ABBV-0805 targeting disease modification in Parkinson's disease
- Potential to expand to earlier stage Parkinson's disease patients and other diseases where alpha-synuclein plays a role
- AbbVie is responsible for clinical development
- BioArctic is responsible for delivering follow-up antibodies in the continued collaboration with AbbVie

ABBV-0805 advancing in clinical trials

December 2018

Alpha-synuclein antibody portfolio licensed by AbbVie

Milestone of 50 MUSD for the license

January 2019

February 2019

ABBV-0805 IND-application approved by the US FDA

March 2019

AbbVie started Phase 1 with ABBV-0805

2020

Phase 1 study ongoing

Aim to evaluate safety and tolerability

Phase 1 MAD study canceled

Detailed plan for Phase 2 being prepared

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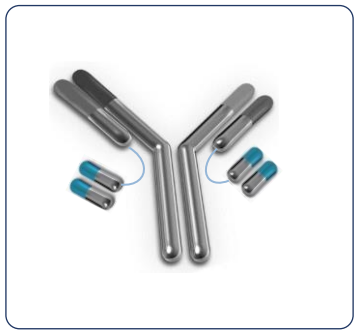
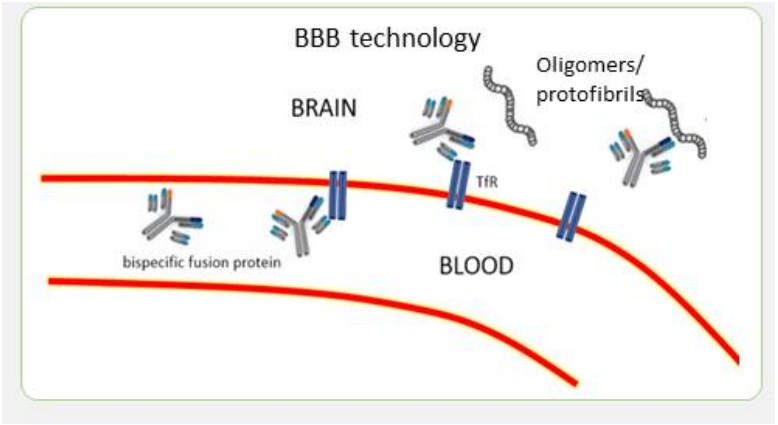
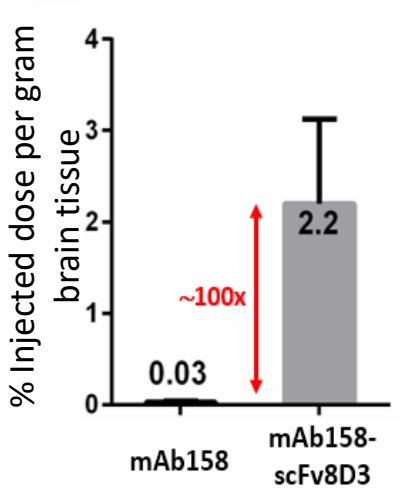
Blood-brain barrier technology platform potential across multiple diseases with promising preclinical results



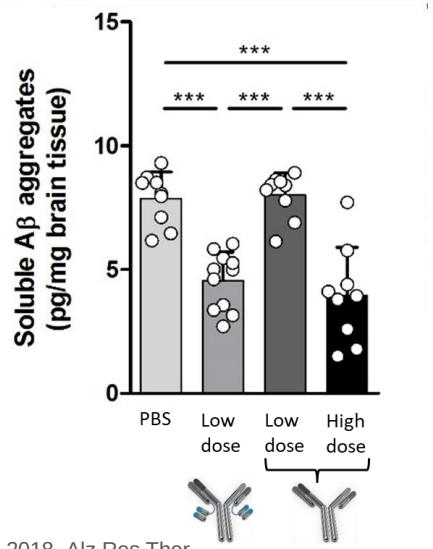


- Development of multi-specific antibodies with a transporter to facilitate passage across the blood brain barrier
- Collaboration with Uppsala University with a grant from Sweden's Innovation Agency, Vinnova

Substantially increased antibody brain uptake by BioArctic's Brain Transport technology



Hultqvist et al 2017, Theranostics.



Syvänen et al. 2018, Alz Res Ther

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