BioArctic AB

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Company Presentation

Nasdaq Stockholm: BIOA B

November 15, 2018

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Helping Patients with Disorders in the Central Nervous System by Developing Innovative Treatments

Alzheimer's Disease

Parkinson's Disease

Complete Spinal Cord Injury

Three key areas with high unmet medical needs – all lacking effective treatments today

Disease modifying treatment in Alzheimer's and Parkinson's Disease – areas with huge and growing markets due to aging populations

BAN2401 Phase 2b study in early AD in collaboration with Eisai – first late stage study demonstrating potential disease modifying effect on both cognition and biomarkers

BAN0805 for PD in collaboration with AbbVie – preparing for clinical development and IND in the U.S.

SC0806 a unique regenerative treatment for patients with Complete Spinal Cord Injuries in Phase 1/2

Attractive combination of fully financed partner projects and innovative pipeline with substantial market and out-licensing potential

Strong science based research and highly educated engaged teams with vast experience in drug development and great track record of high quality deliverables

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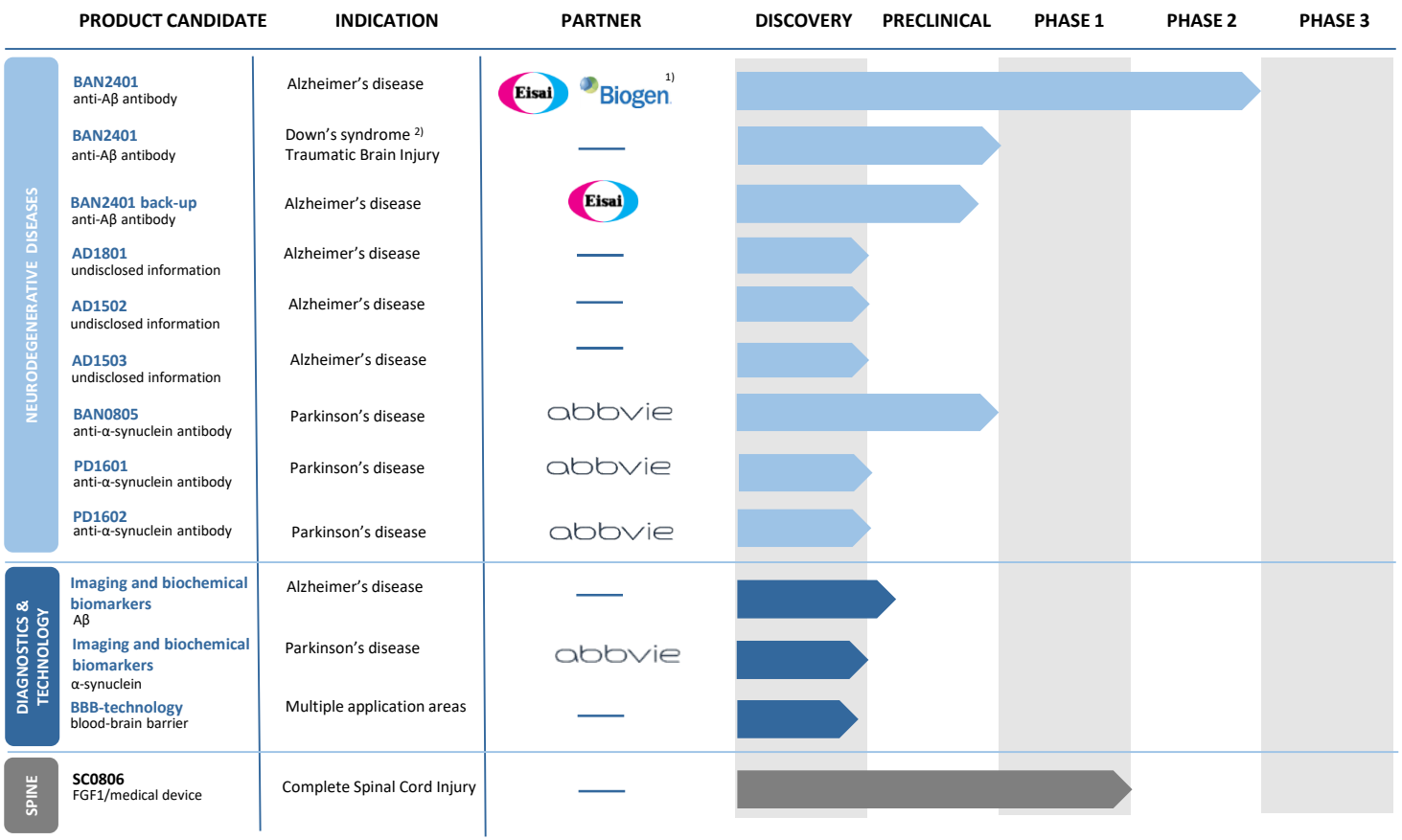
Strategic Partnerships and Cutting-Edge Proprietary R&D

per September 30, 2018



1) Partner with Eisai on BAN2401 for treatment of AD. Since 2014, Eisai partnered with Biogen in AD
2) Dementia and cognitive impairment associated with Down's syndrome and Traumatic Brain Injury

Source: company data

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Long-standing and Extensive Partnerships

Eisai collaboration and license agreements Alzheimer's Disease

Description of agreements

- Two research collaborations – disease modifying therapies for AD – resulted in two licenses for Aβ oligomer/protofibril antibodies: BAN2401 and BAN2401 Back-up
- Third research collaboration – new target as a disease modifying therapy for AD

Milestone / royalty potential

- Total aggregated value of the research collaborations and license agreements is approx. EUR 218m in signing fee and milestones, plus high single digit royalties
- BioArctic has received approx. EUR 47m for the research collaborations, signing fees and milestones

AbbVie collaboration agreement Parkinson's Disease

Description of agreements

- Research collaboration – alpha-synuclein antibodies as disease modifying therapies for PD incl. BAN0805 to IND, follow-up compounds and diagnostic
- Option for AbbVie for a license to develop and commercialize the antibodies

Milestone / royalty potential

- Total potential value of the agreement is up to USD 755m incl. an up-front fee, option exercise fee, and success-based milestones plus tiered royalties
- BioArctic has received an USD 80m up-front payment for the research collaboration
- Payment of USD 50m to be received when exercising option to license, pending US antitrust legislation clearance

Strategic collaborations with pharmaceutical industry validating potential value and commercialization potential for BioArctic with proven track record of delivering on research collaborations

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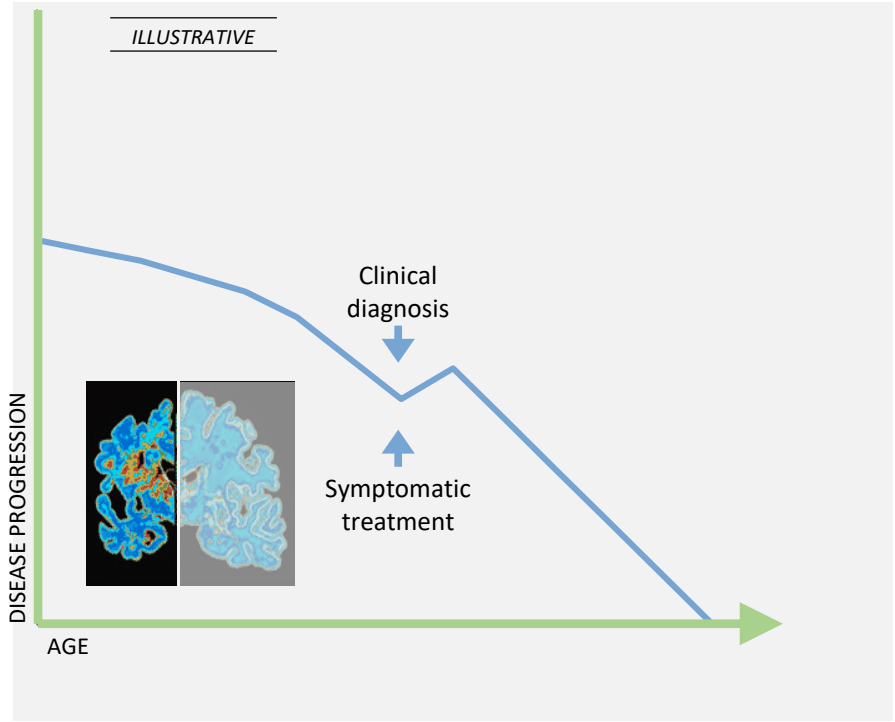
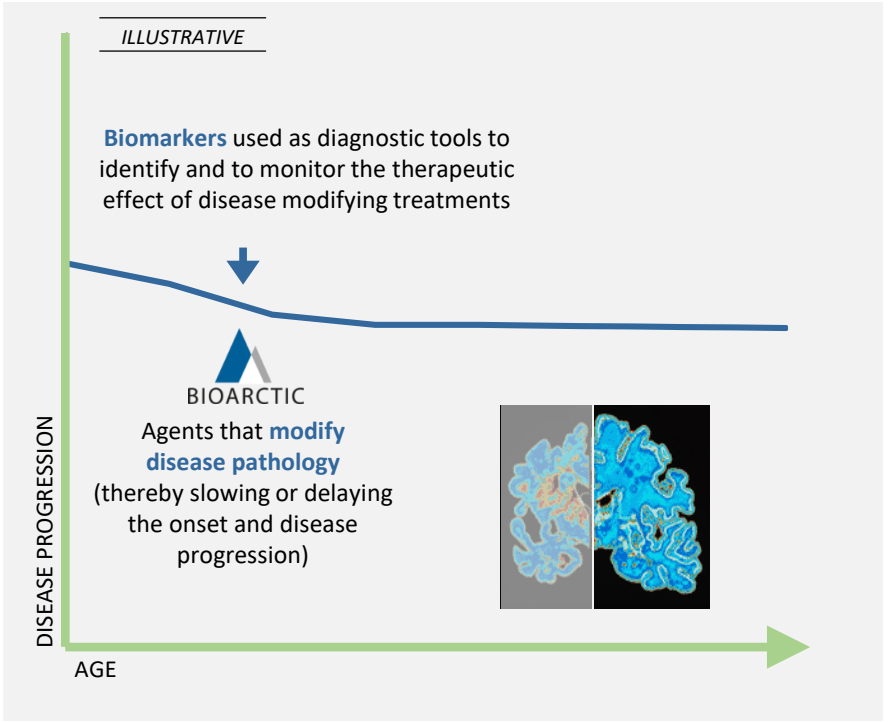
Disease Modifying Agents and Reliable Diagnostics/Biomarkers for Neurodegenerative Diseases

New therapy focus on disease pathogenesis – efforts to delay the neurodegenerative process

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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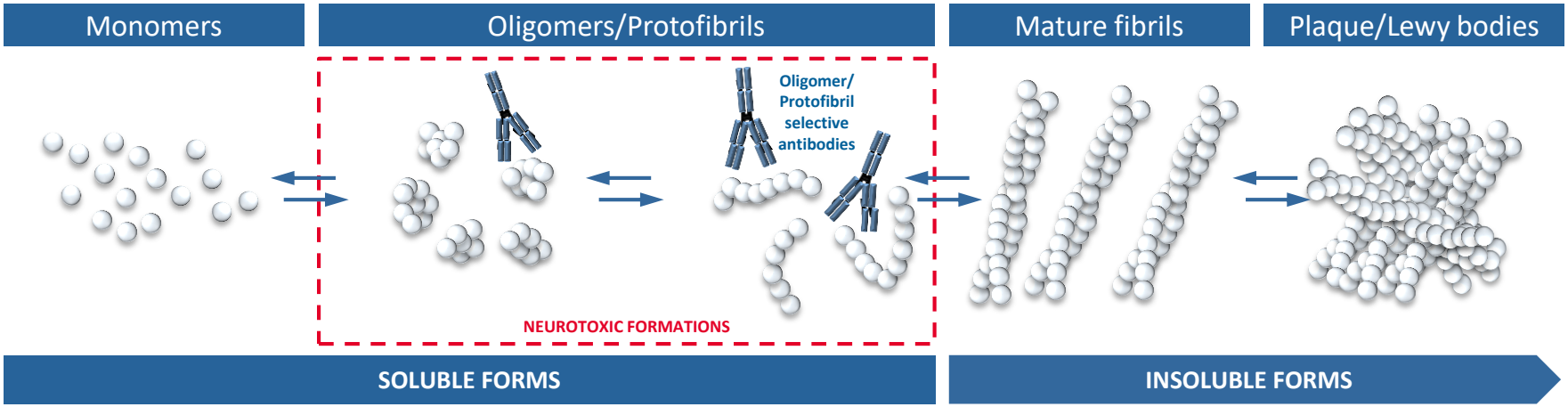
Protein Misfolding is Disease Causing in a Number of Neurodegenerative Diseases Including AD and PD



Aβ Plaque in AD

Alpha-synuclein Lewy bodies in PD

Source: company information.

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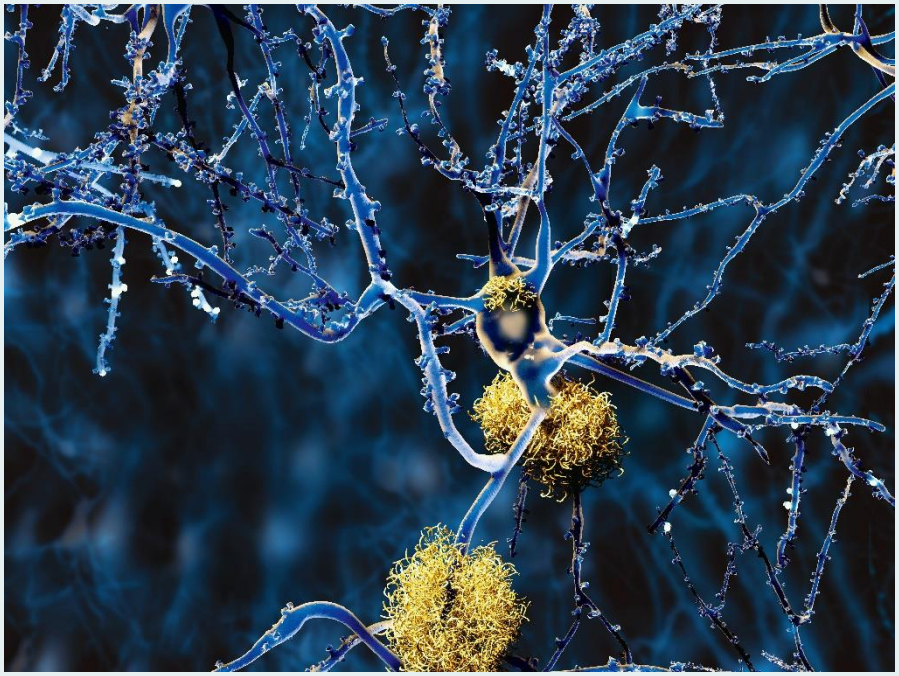
About Alzheimer’s Disease

Neurons with Amyloid Plaques in Alzheimer’s Disease



Alzheimer's Disease (AD)

- AD is an irreversible neurodegenerative brain disease of the elderly, which, through the death of brain cells, leads to a progressive decline in memory and cognitive abilities, such as thinking, language, and learning capacity
- MCI (mild cognitive impairment) with a slight but noticeable and measurable decline in cognitive abilities, including memory and thinking skills, but no dementia
- Prodromal AD or MCI due to AD are MCI subjects with biomarkers showing AD disease pathology
- Early AD encompass mild AD and MCI due to AD
- 47 million people worldwide suffer from dementia and by 2050 expected to be 130 million. >50 % of dementia diagnosed as AD
- 25 million people worldwide suffer from Alzheimer’s disease today and the number is expected to double in 20 years
- Future disease modifying drugs will be used earlier in the disease and in combination with symptomatic drugs used today
- Huge market with demand for several products and expected to be used in combination
- New guidelines from FDA and EMA focus on the value of early diagnosis and treatment of Alzheimer’s disease patients and the importance of biomarkers and cognition data. This is well in line with the development program of BAN2401 and the upcoming analyses of the study

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BAN2401 – Innovative Phase 2b Study Design
Positive 18 Month Results Reported

Important parameters

Right target
- Address the soluble protofibrils – a toxic form of amyloid

Right patient population
- Early AD – MCI due to AD & Mild AD
- Identify right patients – biomarkers

Right dose & exposure
- Selecting doses with exposures above preclinical IC50
- Adaptive design testing several doses and dose regimens

Right measurements
- More sensitive cognition scales
- Biomarkers for disease progression and disease modification

Right safety
- Well tolerated with a 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 included
- **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:
- Δ from baseline in ADCOMS at 12 months
- Safety and tolerability

Treatment 18 months

Key analyses:
- Δ from baseline in ADCOMS, CDR-SB, ADAS-cog at 18 months
- Δ from baseline in brain amyloid as measured by amyloid PET
- Δ from baseline in CSF biomarker and MRI (total hippocampal volume)
- Safety and tolerability

BAN2401 18 months treatment demonstrated an effect on both cognition and biomarkers with a good tolerability profile

Completion of study after 18 months treatment and 3 months follow-up - Q4 2018

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

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BAN2401 Phase 2b Study Demonstrated Positive Results at 18 Months in Early Alzheimer's Disease

- BAN2401 Phase 2b study is the first late stage study demonstrating effects on both cognition and biomarkers

- In the final 18-month analyses of BAN2401 Phase 2b clinical study with 856 early Alzheimer patients BAN2401 demonstrated dose-dependent, clinically meaningful and statistically significant slowing of clinical decline and reduction of amyloid beta accumulated in the brain with a good tolerability profile

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BAN2401 Showed Effect on Clinical Parameters and Biomarkers with Good Tolerability in Early AD Patients at the Highest Dose at 18 Months - I

Clinical effect:
- ADCOMS
- ADAS-Cog
- CDR-SB

- **ADCOMS** cognition scale (the key efficacy parameter) showed statistically significant slower decline of 30% (p=0.034) with 10 mg/kg twice a month (highest dose)
  - ADCOMS showed effect already at 6 months – as well as after 12 and 18 months of treatment
  - Slowing of disease progression observed across sub-groups*
  - The clinical effect increased over time
- **ADAS-Cog** (well-established cognition scale) showed statistically significant slower decline of 47% (p=0.017)
- **CDR-SB** (cognition and function scale) showed slower decline of 26% (p=0.125)

*MCI due to AD – mild AD, ApoE4 carriers – non-carriers, with or without symptomatic treatment

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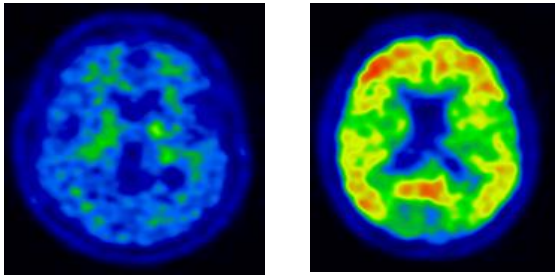
BAN2401 Showed Effect on Clinical Parameters and Biomarkers with Good Tolerability in Early AD Patients at the Highest Dose at 18 Months - II

Biomarkers:
- Amyloid PET

- **Amyloid PET:** BAN2401 reduced brain amyloid-beta dose-dependent and statistically significant, amyloid decreased ~70 units (from 74.5 at baseline to 5.5 at 18 months for the top dose) with Centiloid scale ($p<0.0001$)
  - Amyloid PET visual read showed dose-dependent and statistically significant improvements and 81% of the patients in the BAN2401 top dose converted from amyloid positive to amyloid negative ($p<0.0001$)
  - Amyloid PET demonstrated consistent and pronounced reduction of amyloid in the brain across all clinical sub-groups*



Brain images provided by PET-Centre, Uppsala University Hospital, Sweden, showing a normal brain (left) and an Alzheimer brain (right).
The images are illustrative examples of PET scans and are not images from the BAN2401 Phase 2b study.

* MCI due to AD – mild AD, ApoE4 carriers – non-carriers, with or without symptomatic treatment

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BAN2401 Showed Effects on Amyloid and CSF Markers of Neurodegeneration Consistent with Impact on Underlying Disease Pathophysiology - III

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

- Abeta increase shows target engagement
- Neurodegenerative markers show effect of BAN2401 on underlying pathophysiology
  - Reduction in t-tau (downstream tau pathway)
  - Reduction in p-tau (downstream tau pathway)
  - Reduction in neurogranin (synaptic damage)
  - Reduction in increase of Neurofilament Light (NfL) (axonal degeneration)

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BAN2401 Showed Effect on Clinical Parameters and Biomarkers with Good Tolerability in Early AD Patients - IV

Safety & tolerability

- BAN2401 was **well-tolerated** with infusion reactions and ARIA as the most common side effects (mostly mild to moderate)
- ARIA-E incidence:
  - <10% at any dose
  - <15% in APOE4 carriers at the highest dose
  - ~90% of ARIA-E cases were asymptomatic
  - Generally occurred within the first 3 months of treatment

ARIA-E, Alzheimer's Related Imaging Abnormality-Edema

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Positive Phase 2b Study Results Support BAN2401 as a Potential Treatment for a Broad Population of Early Alzheimer Patients

BAN2401
Treatment Effect in Early AD

Clinical Outcome Measures	Brain Amyloid PET	CSF Biomarkers
• Slowing of disease progression observed across clinical outcome measures at the highest dose, including 30% on ADCOMS • Slowing of disease progression observed across sub-groups	• Pronounced dose-dependent amyloid clearance across the dose range • 81% of subjects converted to amyloid negative state • Consistent and pronounced amyloid clearance across all sub-groups	• Elevated Abeta demonstrates target engagement • Impact on AD pathophysiology with benefits on neuro-degeneration markers: t-tau, p-tau, neurogranin and NfL

BAN2401 was well tolerated with < 10% ARIA-E at any dose

Selectively targeting Abeta protofibrils with low affinity to monomers confer an advantegous benefit risk profile

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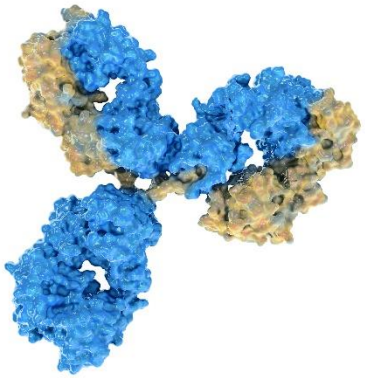
BAN2401 – Next Steps

- Eisai is currently conducting interactions with regulatory agencies regarding the future BAN2401 program
- The study will be completed in Q4 2018 and includes a further 3 months follow-up after completion of 18 months of treatment (at 21 months)
- Open-label extension study with BAN2401, without placebo, for patients from the Phase 2b study will be initiated Q4 2018



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About Parkinson's Disease

Network of Neurons in Parkinson's Disease

Parkinson's Disease (PD)

- Tremor is the best known sign of the disease. The disease develops gradually and can start with hardly noticeable tremor in one hand or symptoms related to disturbances in the REM sleep, smell and bowel function. The disease often also leads to stiffness and slow movements
- PD is the second most common neurodegenerative disease
- Compared to AD it affects a younger patient group, which means that many who fall ill are still at working age
- From 1990 to 2015 the prevalence of PD has doubled
- In 2015 it was estimated that 6.2 million people suffered from PD worldwide
- There is currently no disease modifying treatment for PD

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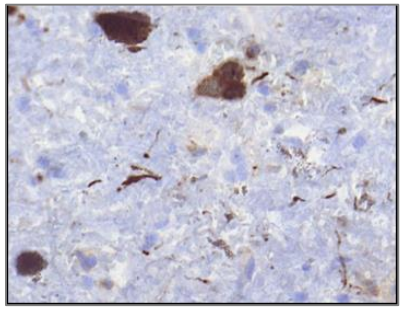
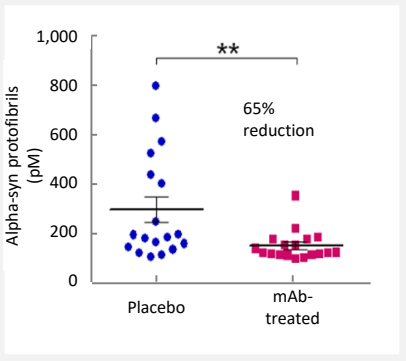
BAN0805 – Groundbreaking Disease Modifying Drug in PD with Rationale for Selective Targeting of Alpha-synuclein Oligomers/ Protofibrils

Rationale for targeting alpha-synuclein

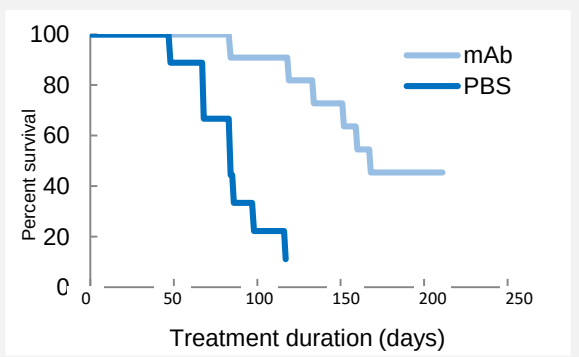
Human genetics → Pathology → Pre-clinical proof of concept



Reduction of neurotoxic alpha-synuclein oligomers/protofibrils



Increases lifespan



Alpha-synuclein mutations lead to PD or Dementia with Lewy Bodies and are associated with increased oligomer/protofibril formation

Alpha-synuclein deposition is a hallmark of PD pathophysiology and alpha-synuclein oligomers/protofibrils are elevated in PD

Oligomer/protofibril selective antibody reduces neurotoxic alpha-synuclein oligomer/protofibril levels, delays disease progression and increases life-span in a PD mice model

BAN0805 in preparation for IND to start clinical trials in the US 2019

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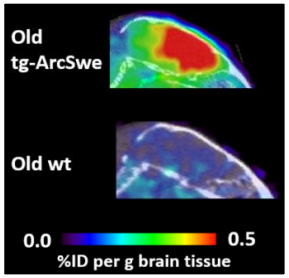
Several Novel Approaches to Improve Diagnostics and Treatment

Imaging and biochemical biomarkers in AD

Aβ protofibril PET tracer

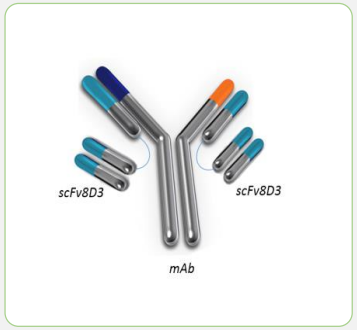


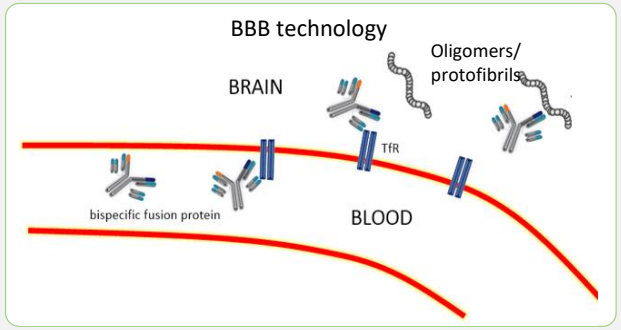
- PET with an antibody-based ligand
- Binding to Aβ oligomers/protofibrils
- Has short half-life
- Improved BBB penetration

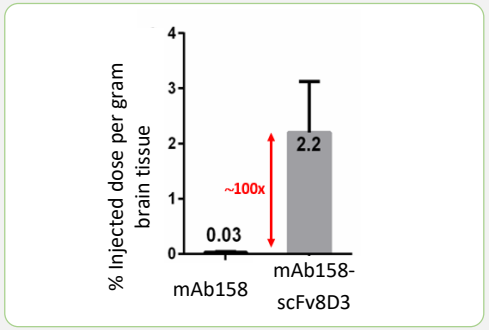
Aβ protofibril biochemical biomarker

- Sensitive biochemical method for Aβ measurement in development is of great clinical importance

Blood-brain barrier technology







Source: Sehlin et al 2016 Nature Communications. Hultqvist et al 2017, Theranostics.

Substantially increased antibody brain uptake by BioArctic´s brain shuttle technology

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About Complete Spinal Cord Injury

Spinal Cord Injuries (SCI)

- A complete spinal cord injury is defined as an injury where the patient can accomplish no voluntary movement or sensory feedback below the injury
- A spinal cord injury causes degeneration of the nerve fibers below the site of the injury as nerve cells do not regenerate
- Besides paralysis patients with complete spinal cord injury suffer from other serious symptoms, including neuropathic pain, bowel and bladder incontinence, sensory loss, pressure sores, infertility and sexual dysfunction
- Increasing stability, restoring bowel and bladder control, reducing pain or enabling sexual functionality would constitute a major improvement of the patient's quality of life
- 40 percent have complete spinal cord injuries
- Complete spinal cord injuries are more common among younger persons, primarily men injured in accidents
- 2.5 million people live with paralysis
- Today there is no effective treatment for complete spinal cord injury
- The patients require life-long treatment and care, which means high costs for healthcare systems and societies

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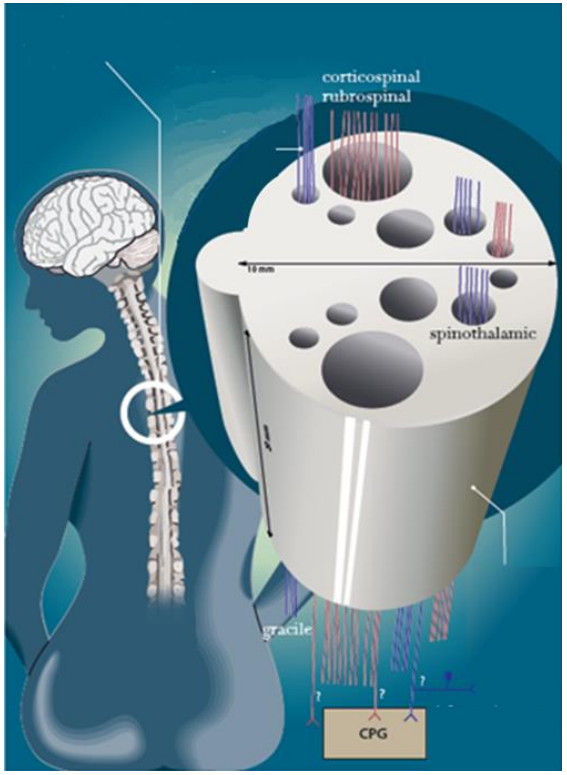
SC0806 – Unique Regenerative Treatment of Complete SCI

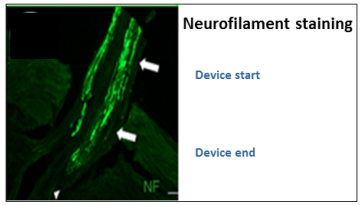
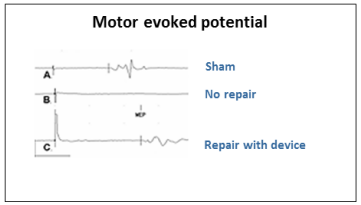
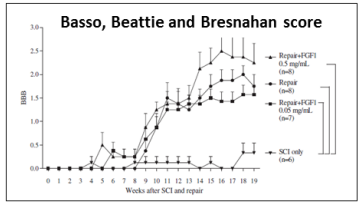
SC0806 – Regenerative Treatment of Complete SCI



Treatment Rationale

SC0806 makes nerve regeneration possible

FGF1 activated by heparin	• Stimulation of central axon outgrowth • Decreases gliosis
Peripheral nerve autografts	• Optimal regeneration environment
Biodegradable device	• Provides sustained release of FGF1 • Positioning of nerve grafts from white to gray matter

Nerve regeneration	Electrophysiology restored	Motor function improved
		

Preclinical Proof of Concept shown in rats

- Rat experiments demonstrate nerve regeneration, restored electrophysiology and motor function
- The motor evoked potential (MEP) has been restored in rats with resected spinal cords

Source: Nordblom et al. Restorative Neurology and Neuroscience 30 (2012) 91–102

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SC0806 – Unique Regenerative Treatment of Complete SCI

The Lokomat™ used in the Rehabilitation

Project Status

- Clinical Phase 1/2 trial ongoing with SC0806 in patients with Complete Spinal Cord Injury
  - Surgery in Sweden
  - Rehabilitation 18 months with Lokomat™ in Sweden, Estonia, Finland and Norway
  - Patients receiving SC0806 treatment are given the option of 12 months additional participation in an extension study
  - 9 patients included in Panel A (6 treated with SC0806 and 3 control patients)
  - Screening of patients for Panel B on-going
  - Interim analysis planned Q4 2019/Q1 2020
- Orphan Drug designation in US and EU – granting 7 and 10 years exclusivity, respectively
- EU Horizon 2020 research and innovative program Grant Agreement No. 643853 of MEUR 6.4

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BioArctic has a Strong Financial Position

- BioArctic has a successful business-model with research collaborations and license agreements with big pharma – Eisai and AbbVie
- Close collaboration with universities
- Grants from Vinnova and EU Horizon 2020
- External validation of high quality deliverables
- Positive results last 5 years and all years but 3 since start 15 years ago
- Solid cash position with more than SEK 1 billion

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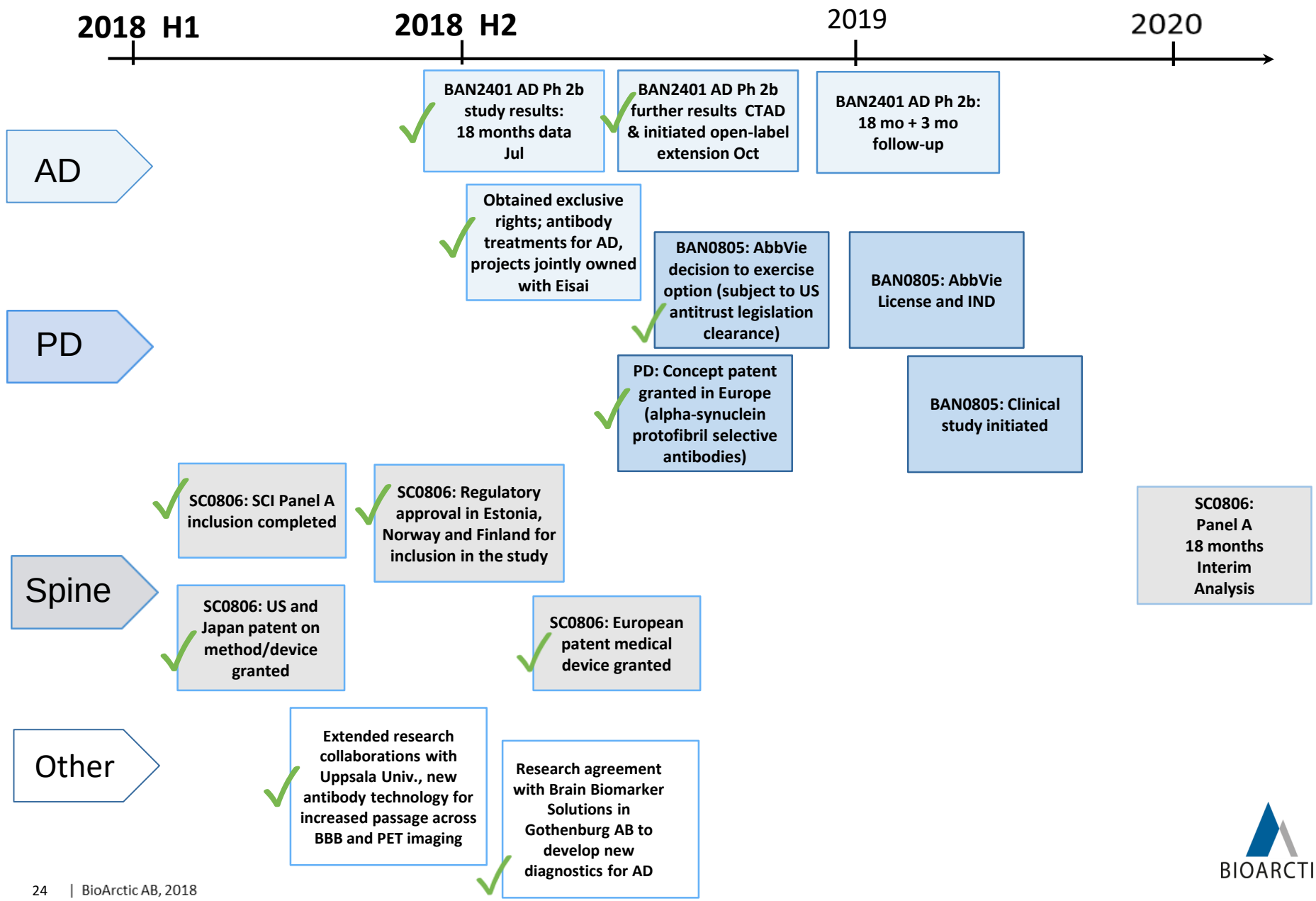
Recent & Anticipated News Flow



Thank you for your attention
Q&A

Gunilla Osswald, CEO

Next Report & IR Contact

- **Next report:**
  Full Year Report 2018
  Feb 14, 2019
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  ir@bioarctic.se

For subscription of financial reports/press releases and more information, please visit www.bioarctic.com

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Snapshot of BioArctic

Company overview

- **Research oriented biopharma company** focusing on development of drugs in areas with a large unmet medical need, such as Alzheimer's and Parkinson's Disease, and Complete Spinal Cord Injury
- **Founded in 2003** by Prof. Lars Lannfelt and Dr. Pär Gellerfors
- **Flexible organization** with approx. 30 FTEs complemented with consultants and close collaborations with external partners
- **Headquartered** in Stockholm, Sweden
- **Listed on Nasdaq Stockholm Mid Cap** since October 2017

Investment highlights

- **Highly educated organization** with proven track record of bringing drugs from idea to market
- **Innovative portfolio** of differentiated first-generation disease modifying agents in Alzheimer's and Parkinson's Disease, diagnostics and pioneering Complete Spinal Cord Injury treatment
- **Strategic collaborations** with Eisai and AbbVie validating highly innovative research organization and unique product candidates
- **Attractive combination** of fully financed partner projects and cutting-edge, well funded, proprietary R&D pipeline with substantial market and out-licensing potential

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Highly Educated and Research-oriented Organization with Vast Experience of Bringing Drugs from Idea to Market

GUNILLA OSSWALD

PhD, CEO, former VP AstraZeneca (Portfolio, Projects, Clinical, Marketing)

30 years of relevant experience

HANS BASUN

Professor, MD, VP CMO, Geriatrician at Memory Clin in Uppsala, former AstraZeneca (Clinical Development)

35 years of relevant experience

CHRISTER MÖLLER

PhD, VP CSO, extensive experience from small biotech (Research & Development)

20 years of relevant experience

JOHANNA FÄLTING

PhD, VP Translational Science & Pharmacology, former AstraZeneca R&D (Discovery & Drug Projects)

15 years of relevant experience

MIKAEL MOGE

PhD, VP CMC and Protein chemistry, former AstraZeneca (Pharmaceutical Development), Syntagon (Head Development & Pilot Plant)

20 years of relevant experience

NORA SJÖDIN

VP Regulatory Affairs, former Pharmalink, NDA Regulatory Service, AstraZeneca

25 years of relevant experience

LARS LANNFELT

Professor, MD, Co-founder, Senior VP University Collaborations, Senior Professor, Uppsala University, discovered the Swedish and the Arctic mutations in Alzheimer's Disease

35 years of relevant experience

Highly educated staff with 80% PhDs with background from

UPPSALA UNIVERSITET · Karolinska Institutet · Stockholm University · KTH · The Rockefeller University · HARVARD MEDICAL SCHOOL · KU MEDICAL CENTER · Inserm

Significant in-house experience combined with collaborations with universities, hospitals, consultants, CDMOs/CROs and pharmaceutical industry

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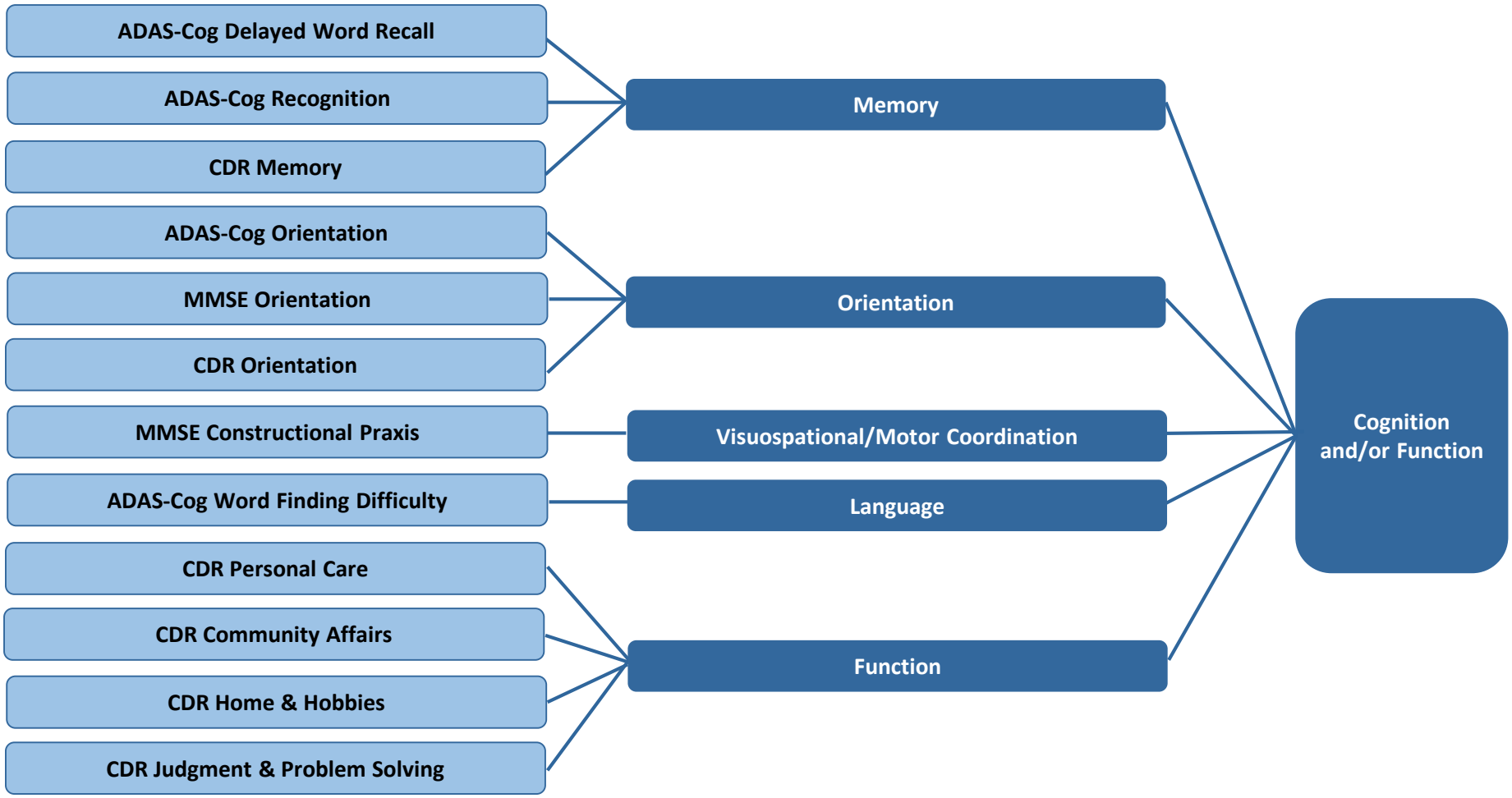
The ADCOMS End-point – a Sensitive Composite Cognition Score



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