ProMIS Neurosciences: Selective targeting of pathogenic misfolded proteins, based on a proprietary discovery platform

Toronto Stock Exchange (TSX) ticker: PMN.TO
OTCQB ticker: ARFXF.

October 22, 2021

Forward looking statement: safe harbor

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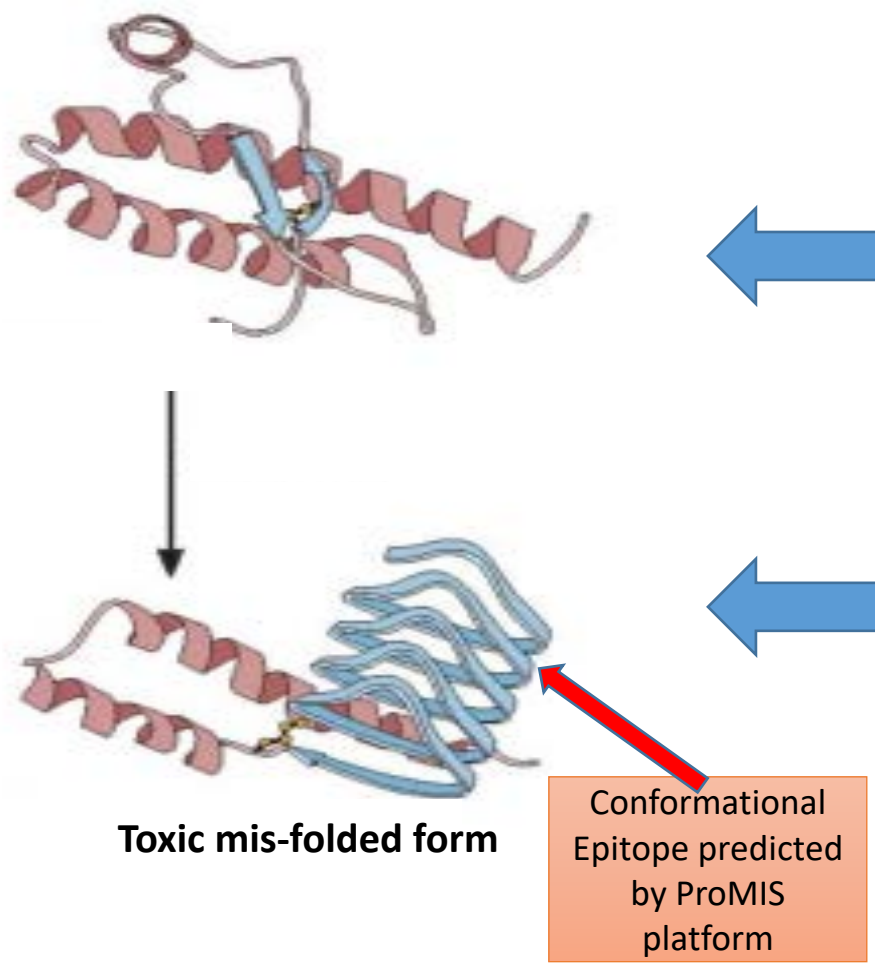
ProMIS Summary

- Differentiated ***technology platform*** – Computational approaches to rational design of selective antibodies, a unique ProMIS capability
- ***Growing portfolio*** of antibodies ***selective for mis-folded proteins*** implicated in neurodegenerative diseases
- High selectivity a ***ProMIS competitive advantage.*** Lack of selectivity for mis-folded proteins likely the primary source of failures or limited success in prior competitor programs in neurodegenerative diseases
- ***Lead program PMN310 potential "best of the next generation" antibody therapy in Alzheimer's disease:*** highly selective for toxic oligomer form of amyloid, differentiated from likely first generation products from Biogen, Eisai, and Lilly; PMN310 differentiated from first oligomer selective antibody from Acumen
- ***Growing portfolio*** contains differentiated, selective antibodies against toxic forms of TDP-43 (ALS, FTD); alpha synuclein (PD, MSA), tau, SOD1, and others
- Fluid-based biomarkers may enable ***rapid and capital efficient path to clinical readout*** and value inflection for all programs
- Multiple programs 21-36 months from clinical data – IND enabling work followed by SAD/MAD trial in patients

ProMIS Platform - Summary

- ProMIS is founded on a ***unique, revolutionary approach tailoring antibodies to defined targets***, using an interdisciplinary synthesis of physics and biology
- IBM's quantum computing group in April 2020 predicted that within five years [i.e. by 2025] quantum computing will enable ***"developing novel biologic products based on protein folding predictions"***
- ***ProMIS is 10+ years ahead*** of that prediction - by focusing on the problem of predicting conformational epitopes (amino acid sequence ***and shape***) exposed only on pathogenic, ***mis-folded proteins***
- Through sophisticated scaffolding, and other methods refined over several years, ProMIS creates ***peptide antigens*** that reproduce the amino acid sequence and conformation of the predicted epitopes to be used for immunization and generation of monoclonal antibodies.
- **ProMIS at AAIC 2021** – "*Conformational epitopes exposed on misfolded toxic forms of amyloid-beta, tau and alpha-synuclein directly contribute to their seeding activity*" - potential breakthrough in scientific understanding of disease, exposed epitopes play a direct role in disease progression, additional mechanism for ProMIS antibodies
- Continuous discussions with large pharma over the last five years have confirmed that as of now, to the best of our knowledge, ***no one has an antibody "rational design" capability like ProMIS'***

Mis-folded proteins have the same amino acid sequence as normal proteins…the only difference is the shape…ProMIS identifies conformational epitopes exposed only on mis-folded proteins



Normal protein – folds into a specific shape to perform its physiologic function

Mis-folded protein…improper folding exposes toxic portions of the protein…..in a particular shape or conformation…

ProMIS platform predicts conformational epitopes – both amino acid sequence and shape, only exposed on toxic mis-folded proteins….

Immunizations with those epitopes lead to selective antibodies

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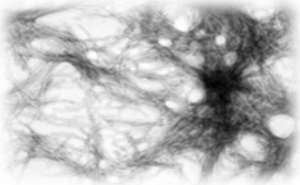
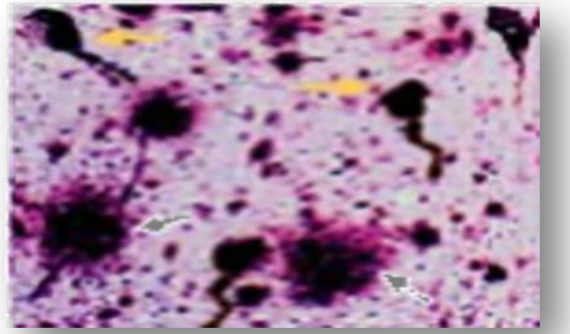
Image adapted from Racaniello V et al, virology.ws, 2016

Alzheimer's, Parkinson's and ALS are protein misfolding diseases, where the toxic mis-folded proteins propagate in a prion-like manner

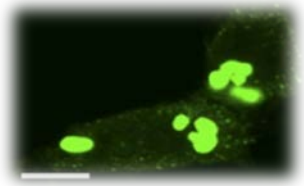
Huntington's disease
(huntingtin)



Alzheimer's disease
(amyloid-beta and tau)

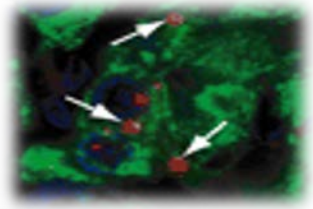


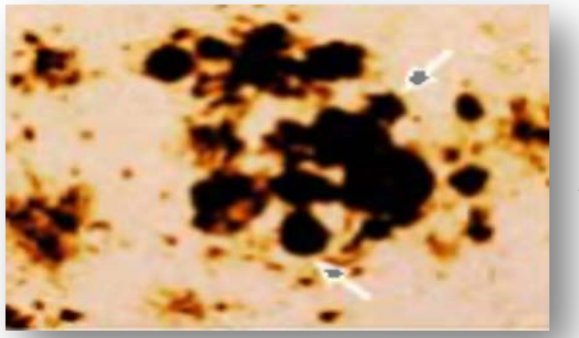
Schizophrenia
(DISC1)



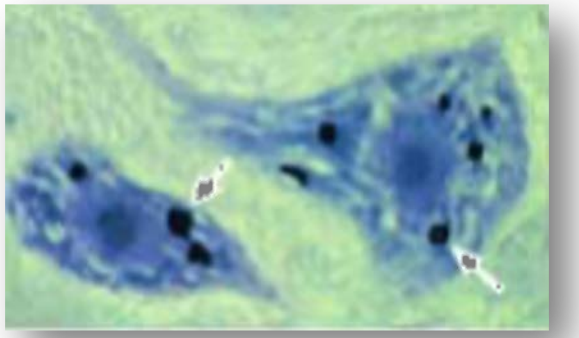
Senile amyloidosis
(transthyretin)

Type 2 diabetes
(amylin)

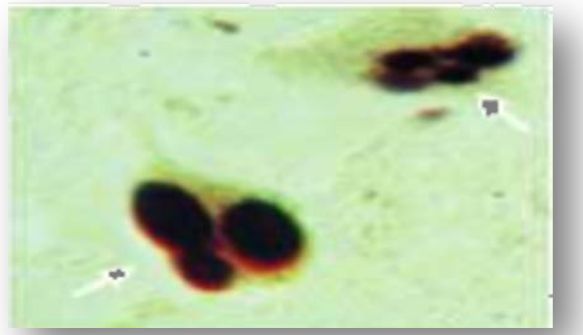




Prion diseases
(prion protein)



ALS
(TDP43, SOD1, FUS)



Parkinson-like diseases
(alpha-synuclein)

The ProMIS platform generates antibodies selective for the misfolded toxic forms of pathogenic proteins

Identification of epitopes selectively exposed on toxic misfolded form of the target protein using predictive computational algorithm

Immunization with disease-associated epitope and screening of monoclonal antibodies with desired binding profile and protective activity

Neutralize the toxic mis-folded form

Don't interfere with the normal form, critical for brain health

Successful track record thus far in generating antibodies selective for pathogenic forms of amyloid-beta, tau, alpha-synuclein, TDP-43, SOD1

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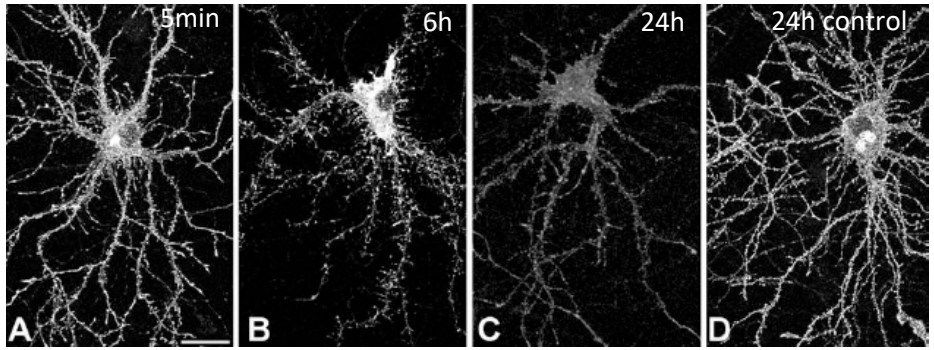
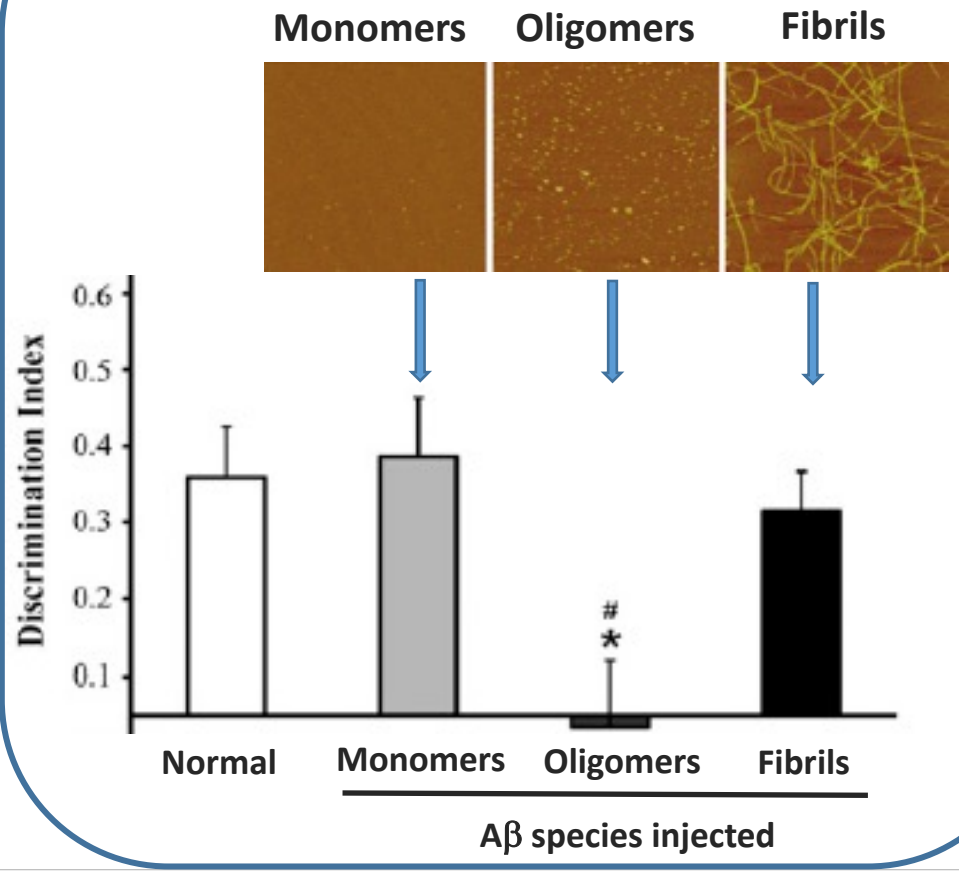
Alzheimer's disease: soluble toxic Amyloid-beta oligomers – not plaque or monomers – are the most neuropathogenic Aβ species

- Synapse abnormalities and memory impairment correlate poorly with plaque burden in human and mouse AD[1,2]
- Aβ monomers and Aβ insoluble fibrils (plaque) have little or no demonstrable toxicity in vitro or in vivo[3-5]
- Soluble Aβ oligomers show the highest degree of neurotoxicity[6]
  - Toxicity in primary neuron cultures and brain slices[3,5,7-9]
  - Induction of cognitive impairment in rodents[3,4,10]

Synaptotoxicity of human Aβ oligomers on hippocampal neurons in vitro[7]



In vivo impairment of recognition memory by Aβ oligomers, not monomers and not fibrils[10]



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[1]Jacobsen et al, 22006, PNAS; [2]Brier et al, 2016, Science Trans Med; [3]Shankar et al, 2008, Nature Med; [4]Cleary et al, 2005, Nature Neuroscience; [5]Hong et al, 2016, Science; [6]Benilova et al, 2012, Nature Neuroscience - Review; [7]Lacor et al, 2007, J Neuroscience; [8]Jin et al, 2011, PNAS; [9]Lauren et al, 2009, Nature; [10]Balducci et al, 2010, PNAS

Degree of selectivity for the correct (toxic) form of amyloid explains past clinical results

Forms of Amyloid	**Monomer** – Important for brain health	**Mis-folded Toxic oligomers** – thousands of scientific studies showing neurotoxicity	**Plaque** – usually present, but no significant role in disease
Amyloid Binding profile of clinical programs	**10** programs/**25** phase 2 and 3 clinical trials **targeting monomer** **All negative – 0/25**		
	4 programs/**13** phase 2 and 3 clinical trials **non-selectively targeting all forms of amyloid** **All negative – 0 / 13**		
		Three drugs: Biogen's aducanumab approved, EISAI's BAN2401, Lilly's donanemab positive phase 2– selective for *aggregated amyloid* **ALL POSITIVE**	
		PMN310 **potential best in class**, selective for toxic oligomers	

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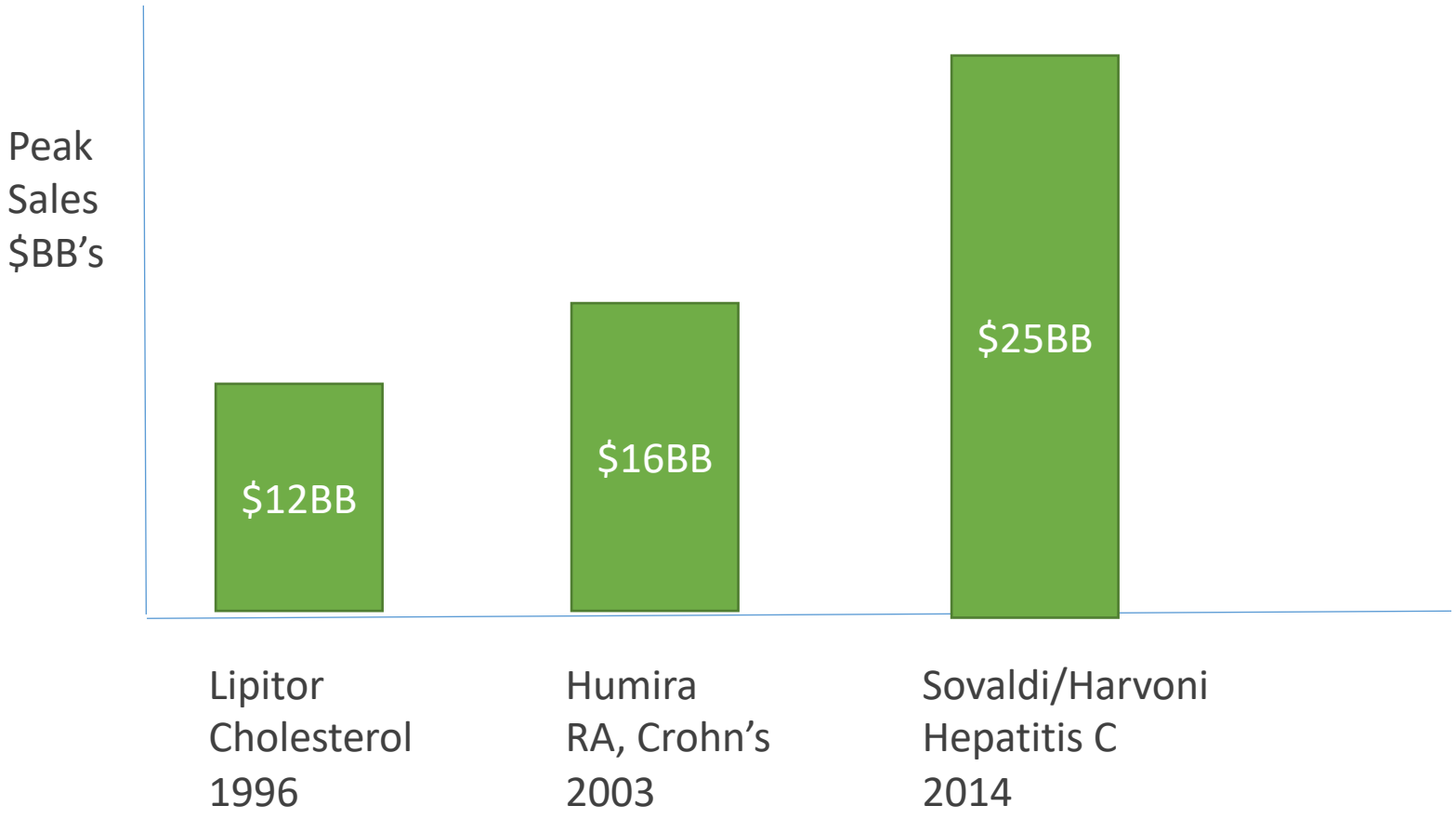
The three largest products in industry history were not first in class, but "best in class" – the inventors identified improvements to existing drugs



ProMIS following the "best in class" playbook:

- Took advantage of "proof of biology" developed by earlier products:
- Used ProMIS proprietary science platform to design an improved product, which may yield superior clinical results

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PMN310: an anti-Aβ-oligomer antibody with strong potential to demonstrate best-in-class characteristics in Alzheimer's treatment

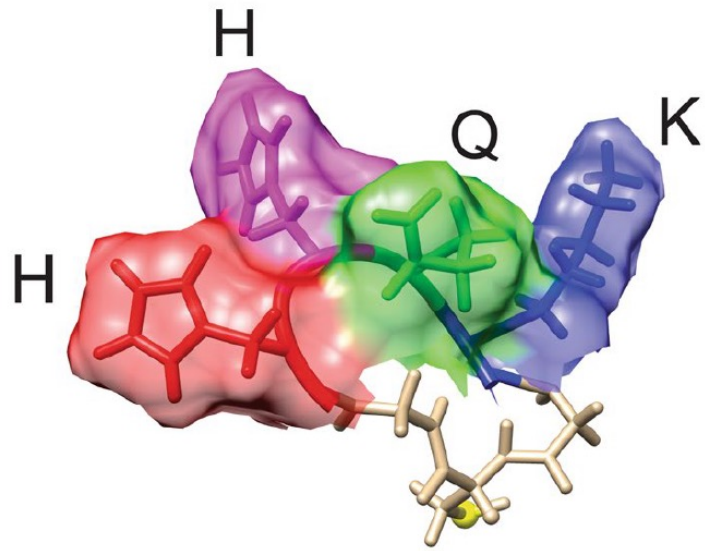
Aducanumab (Biogen), BAN2401 (Eisai), donanemab (Lilly) selective for all aggregated amyloid – plaque and oligomers

- Aducanumab (phase 3), BAN2401 (phase 2) and donanemab (phase 2) showed modest but meaningful efficacy in reducing cognitive worsening
- ***Aducanumab approved by FDA – June 7,2021***
- None bind monomer (the physiologic amyloid species)
- All bind amyloid plaque → ARIA-E (brain swelling)

PMN310 is a next-generation, best-in-class anti-amyloid therapy

- Highly selective for only toxic oligomers
- Dose expected not to be limited by off-target binding or side effects
  - Does not bind monomer
  - Does not bind plaque → ***likely no ARIA-E side effect***
- All dosed PMN310 will be focused on neutralizing toxic oligomers → ***potentially greater clinical efficacy***

PMN310 will utilize blood-based biomarkers in initial trials, enabling rapid timeframe and limited capital needs to value-creating clinical data



Aβ amino acids 13-16 (HHQK) form a unique, Aβ oligomer specific conformational epitope targeted by PMN310

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There are three forms of amyloid, PMN310 is differentiated by selective binding of the toxic form (oligomers)

Bapineuzumab (Pfizer)
- Phase 2 failure
- Phase 3 failure
- ARIA-E side effect

Solanezumab (Eli Lilly)
- Phase 2 failure
- Phase 3 failure

Aducanumab (Biogen)
- Phase 2 & 3 success
- ARIA-E side effect

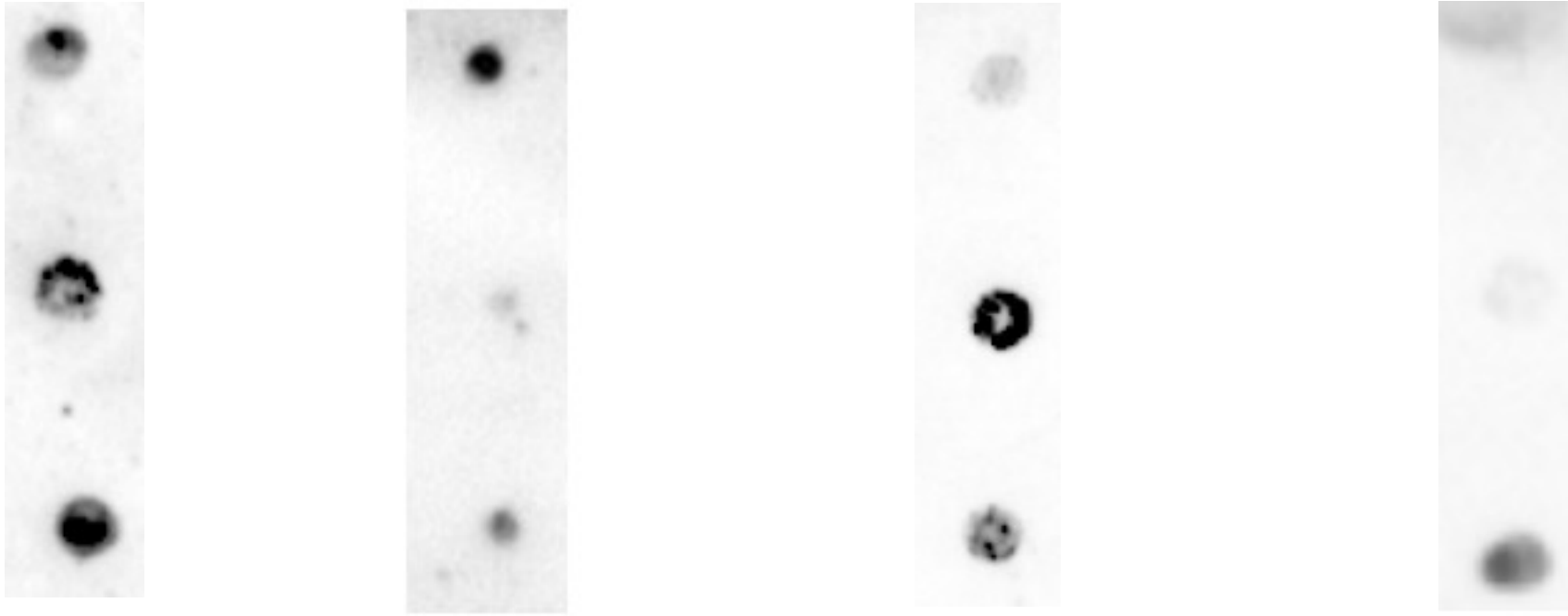
PMN310
- Selective binding to oligomers

-> Expected improvement in efficacy & safety

MONOMERS
- binding wastes therapeutic ammunition

FIBRILS (Plaque)
- binding wastes therapeutic ammunition
- contributes to ARIA-E side effect

OLIGOMERS*
- the right target



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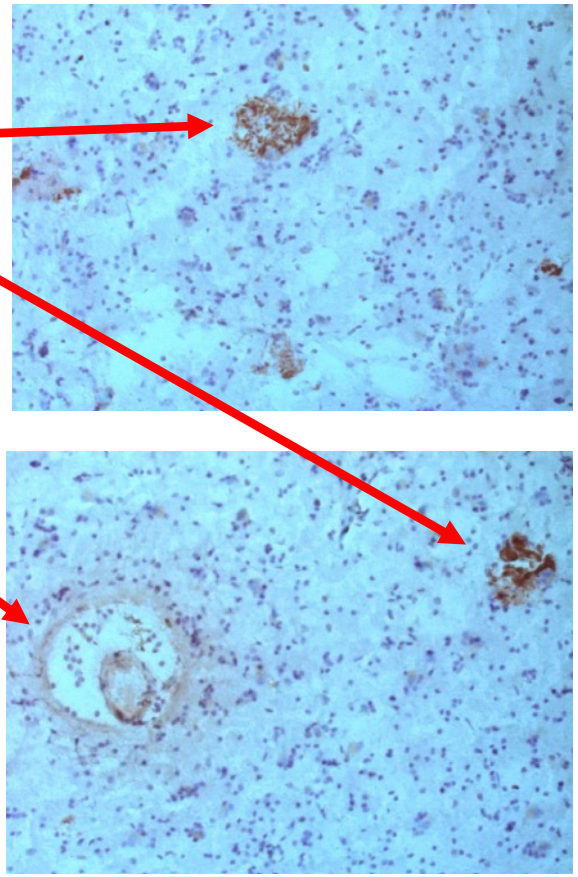
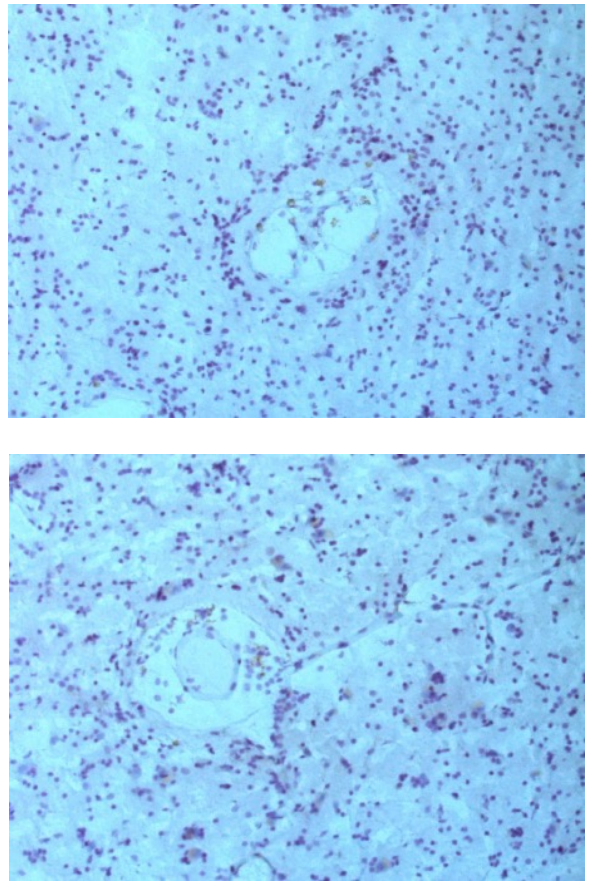
* Synthetic oligomers

ARIA-E associated with aducanumab, BAN2401 & bapineuzumab; PMN310 lack of binding to Aβ plaque strongly suggests a *potential safety advantage - no ARIA-E*

Aducanumab



PMN310



No binding to plaque or vascular deposits

Most likely no ARIA-E*

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* ARIA-E (Amyloid Related Imaging Abnormality-Edema) also known as brain swelling

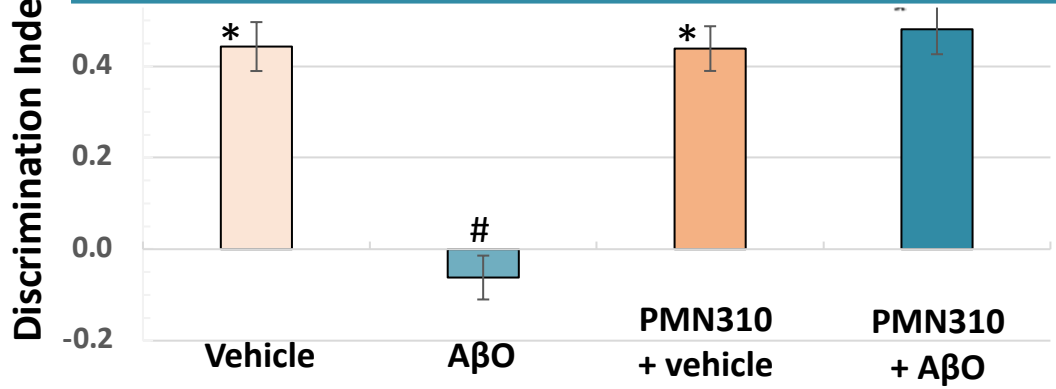
Administration of PMN310 to mice prevents loss of short-term memory formation caused by toxic oligomers, by saving mouse neurons

THE EXPERIMENT

- Mice are tested for discriminating objects after brain injection of:
  - Buffer (vehicle) - normal response
  - Toxic Aβ oligomer
  - PMN310 and buffer (vehicle)
  - PMN310 and Aβ Oligomer

7 days

THE RESULTS



N=12 per arm, *different from AβO ($p < 0.05$), #different from vehicle ($p < 0.05$)

Novel Object Recognition Assay

- **Control mice remember a familiar object when re-exposed to it and spend more time exploring a new object**
- **Oligomer-injected mice lose the ability to discriminate between known and novel objects and spend equivalent amounts of time exploring both**

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Discrimination index = (Time exploring new object – time exploring familiar object) / total exploration time
Results press released January 9, 2017, www.promisneurosciences.com

PMN310 has the potential to be the "best of the next generation" therapy in Alzheimer's

Amyloid beta-targeted therapies

All BACE inhibitors

Non-selective antibodies

Monomer-targeted antibodies

Aduhelm (Biogen, Approved)

BAN2401 (Eisai, Positive Ph2)

Donanemab (Lilly, Positive Ph2)

ACU193 (Acumen, Ph1 start June 2021 Recent NASDAQ IPO)

PMN310 – best in class:

- ***High selectivity for toxic oligomers***
- ***Effector Function (IgG1)***
- ***Potential subcutaneous delivery***

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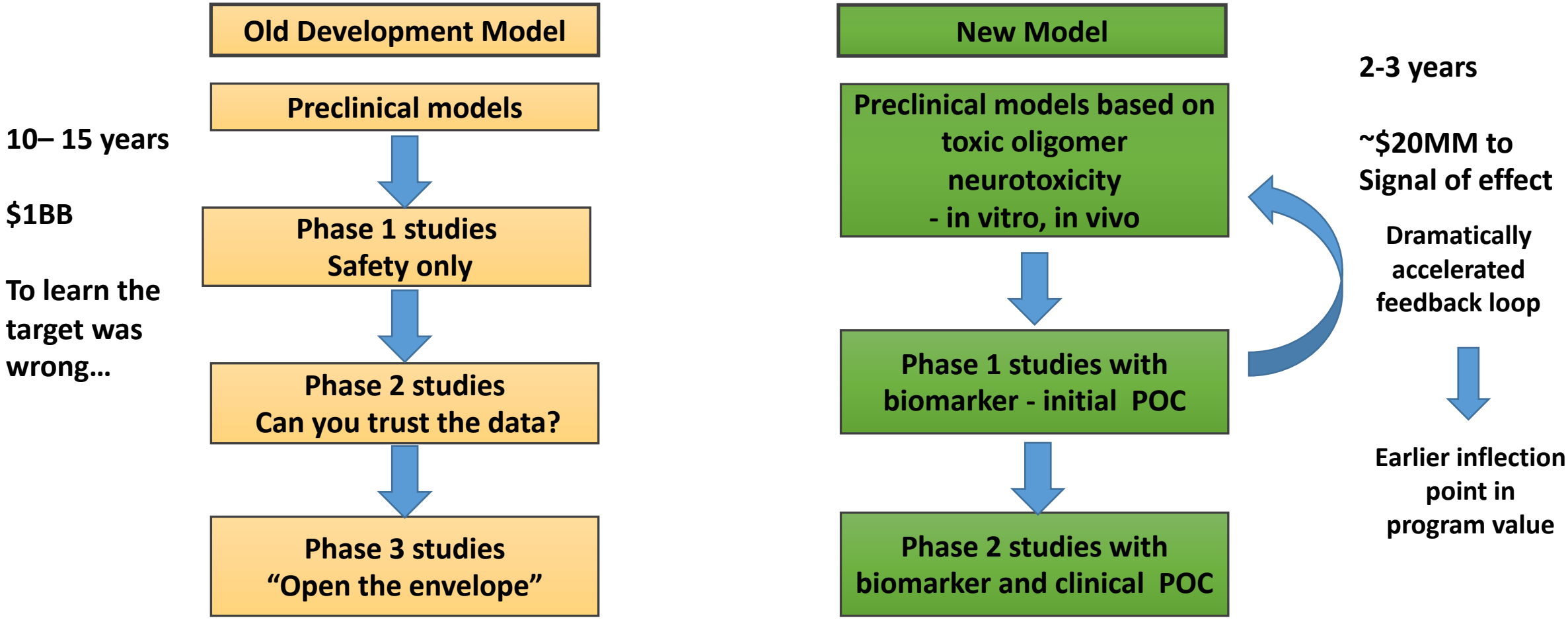
A new development paradigm for Alzheimer's and other neurodegenerative diseases using biomarkers will dramatically improve cost, risk….and time to success



Alzheimer's drug R&D: we're in the midst of a biomarker revolution!

Early Alzheimer's Disease: Developing Drug

Guid

This guidance docu

Comments and suggestions
publication in the *Federal*
guidance. Submit electroni
comments to the Dockets M
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For questions regarding thi
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Center for Biologics Evaluation and Research (CBER)

February 2018
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Alzheimers Dement. Author manuscript; available in PMC 2018 May 18.

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NIA-AA Research Framework: To
Alzheimer's disease

Clifford R. Jack Jr.[a,*], David A. Bennett[b], Kaj E
Samantha Budd Haeberlein[f], David M. Holtzm
Karlawish[j], Enchi Liu[k], Jose Luis Molinuevo[l],
Katherine P. Rankin[o], Christopher C. Rowe[p],
Snyder[d], and Reisa Sperling[s]

Almost there

The unexpected way we might one day diagnose Alzheimer's

By Bill Gates | April 2, 2019

Jeff Bezos teams with Bill Gates to fund new and better ways to diagnose Alzheimer's

by john carroll — on April 4, 2019 10:10 AM EDT

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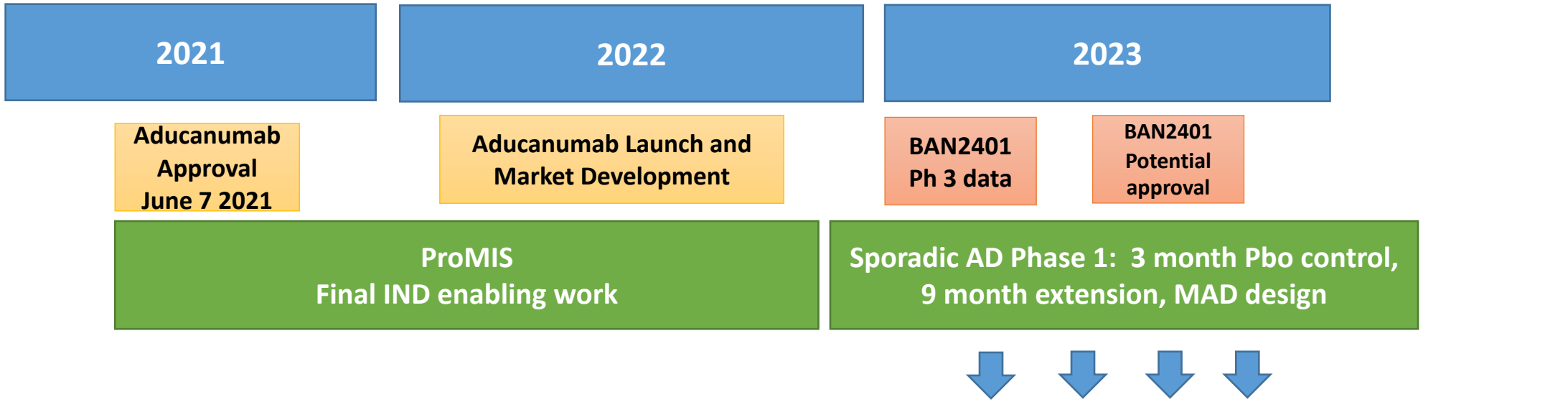
PMN310: potential for value-creating clinical data in the near term

- likely positive market developments could amplify PMN value



Open label after 3 months of treatment
Ongoing biomarker readouts could provide signal

- Recent advances in blood-based biomarkers may allow ProMIS to detect an objective treatment signal as early as Phase 1, potentially providing rapid & cost-effective proof-of-concept

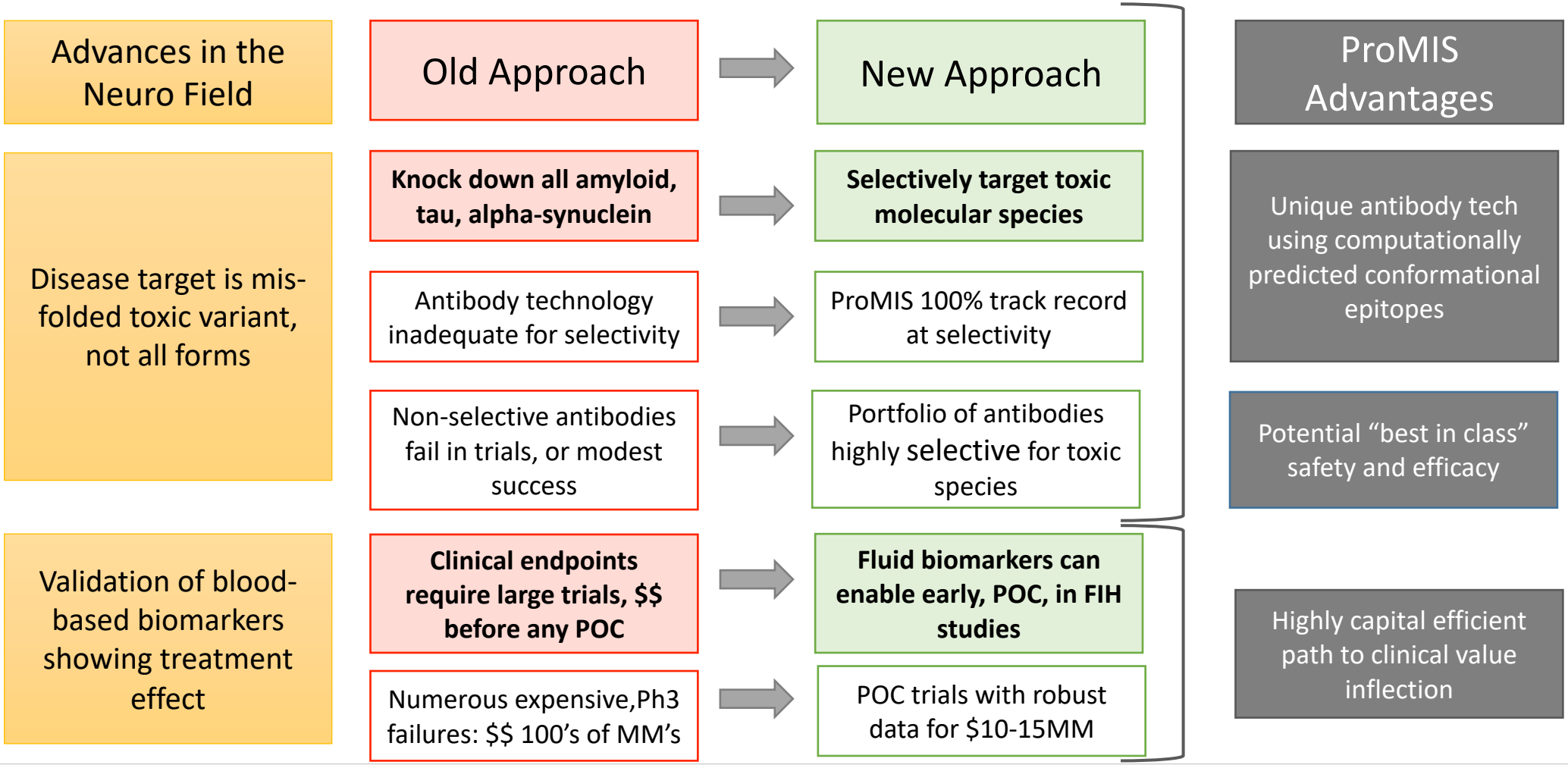
ProMIS: a broad differentiated portfolio; a unique technology platform

- *Potential "best of the next generation" for all of neurodegenerative disease*

Misfolded protein target	Lead indication	Other Indications	Status
Amyloid beta	Alzheimer's		IND enabling work ongoing
TDP-43	ALS	FTD, LATE	Lead antibodies
Alpha synuclein	Multiple System Atrophy	Parkinson's, LBD	Lead antibodies
tau	Alzheimer's	PSP, other tauopathies	Lead selection
SOD1	ALS		Lead antibodies
RACK1	ALS	HD, cancers	Immunizations
Ataxin2	ALS		Computational modeling
Disc1	ALS	Schizophrenia	Computational modeling
Amylin	T2Diabetes		Computational modeling

DLB: Dementia with Lewy bodies, FTD: Frontotemporal dementia, LATE: Limbic-predominant age-related TDP-43 encephalopathy, ALS: Amyotrophic lateral sclerosis, PSP: Progressive supranuclear palsy, AD: Alzheimer's disease, HD: Huntington's disease

Advances in the neurodegeneration field + ProMIS unique capabilities = value creation potential



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Thank You

Please feel free to contact us with any additional questions.

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Appendices

1) ProMIS unique antibody design platform	- Computational modeling approaches combining Physics and Biology - 100% track record at achieving target molecular species selectivity
2) PMN310 – lead program in Alzheimer's	- Comparator data support "best in class" potential - In vivo data: blocks cognitive deficit caused by amyloid-beta oligomers - Competitor clinical data support science – oligomer is target
3) Alpha Synuclein program	- Several antibody candidates with selectivity and functional benefit - Illustrates ProMIS' competitive advantage in achieving selectivity
4) TDP-43 program	- Multiple antibodies with target selectivity - Functional benefit both as extracellular antibody and intracellular intrabody

ProMIS Platform

The ProMIS platform generates antibodies selective for the misfolded toxic forms of pathogenic proteins

Identification of epitopes selectively exposed on toxic misfolded form of the target protein using predictive computational algorithm

Immunization with disease-associated epitope and screening of monoclonal antibodies with desired binding profile and protective activity

Neutralize the toxic mis-folded form

Don't interfere with the normal form, critical for brain health

Successful track record thus far in generating antibodies selective for pathogenic forms of amyloid-beta, tau, alpha-synuclein, TDP-43, SOD1

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Image adapted from Racaniello V et al, virology.ws, 2016

Therapies must be selective for the mis-folded form – ProMIS has a unique advantage

Protein	Role of physiologic monomer	Disease Caused by Mis-Folded Form
Amyloid Beta	Synaptic remodeling	Alzheimer's
Alpha Synuclein	DNA repair	Parkinson's Lewy Body Dementia MSA
TDP43	RNA transcription	ALS FTD

- ProMIS has a portfolio of antibodies with high selectivity for the mis-folded form of each of these proteins

- Differentiated from first generation and competitor products

- ProMIS unique capability

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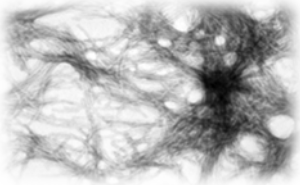
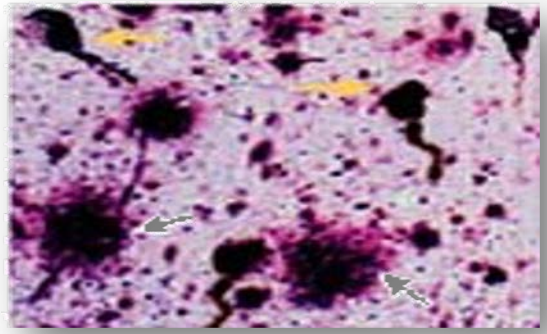
The same proteins perform essential cellular functions when in their normal, physiological form

Huntington's disease
(huntingtin)



Synaptic Remodeling

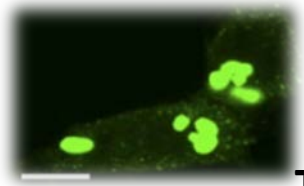
Alzheimer's disease
(**amyloid-beta** and **tau**)



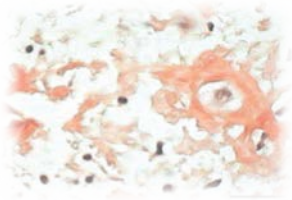
Neuron Stability

DNA Repair

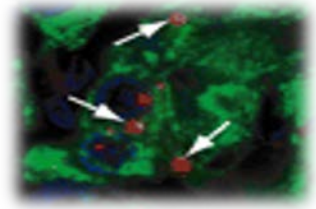
Schizophrenia
(DISC1)



Senile amyloidosis
(transthyretin)

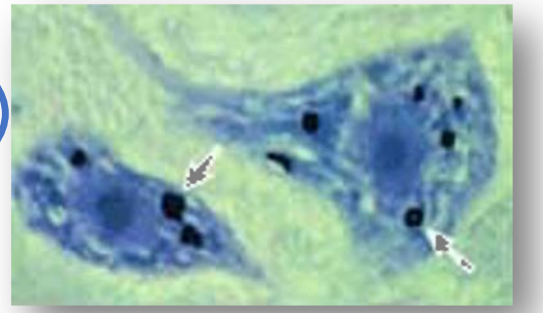


Type 2 diabetes
(amylin)



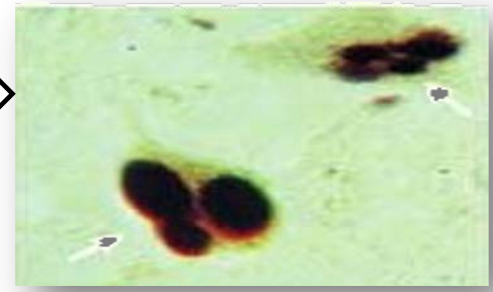
Normal forms play critical role in brain health

RNA Splicing, Transport



DNA Repair

ALS
(**TDP43**, **FUS**, **SOD1**)



Synaptic Remodeling

DNA repair

Parkinson-like diseases
(**alpha-synuclein**)

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Collective Coordinates: Algorithm to predict misfolding-specific epitopes

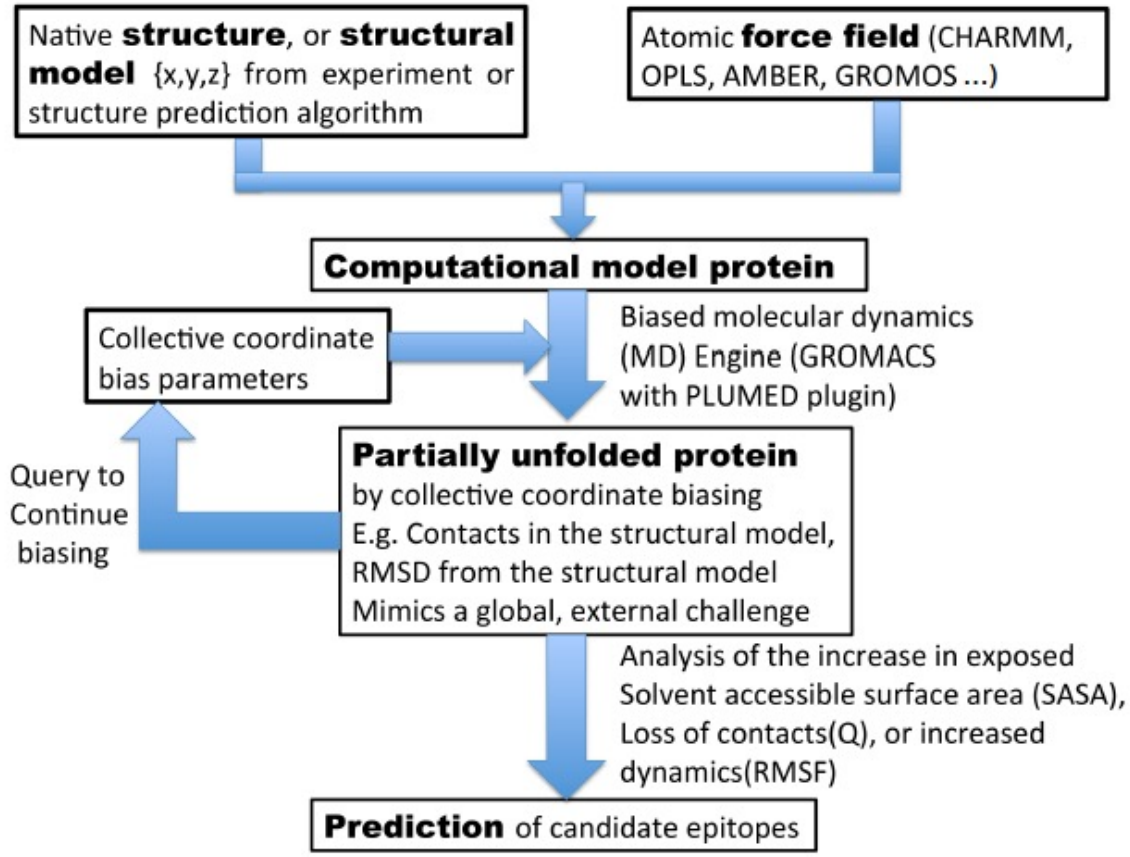
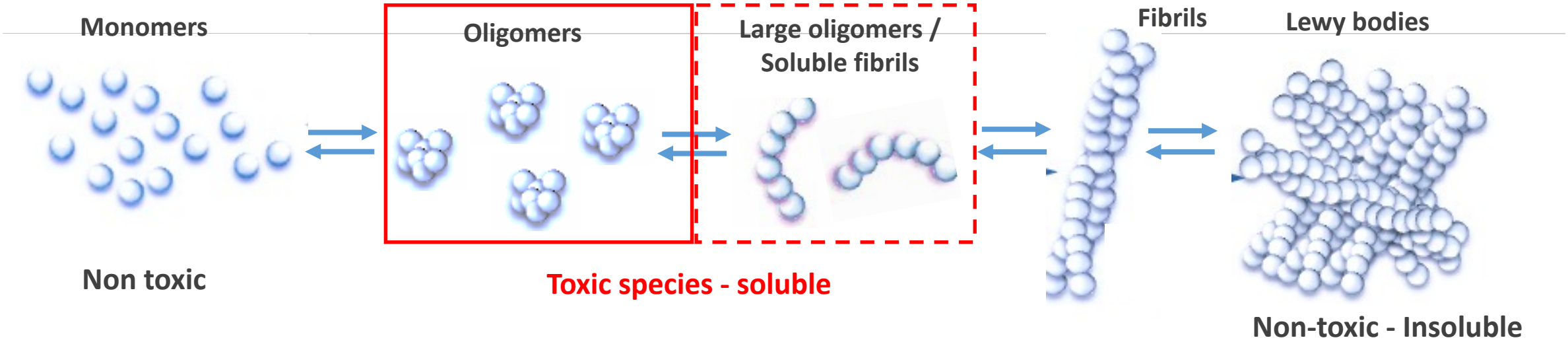


Figure 1: Flowchart of the steps in the prediction algorithm

J Phys Chem 2018

- General principles:
  - Weakly stable regions in the native fold may constitute target epitopes that are preferentially exposed in misfolded species.
  - These misfolding-specific epitopes can be distinguished from the same regions in the native fold by their conformational properties
  - By properly scaffolding the epitopes, antibodies may be raised to selectively bind to misfolded species
- Molecular dynamics simulations partially unfold a protein or fibril in order to predict misfolding-specific epitopes generated by regions that are most likely to locally unfold in response to global stress
- Several metrics used to measure local disorder: solvent exposed surface area, native contacts, root mean squared fluctuations
- Patent filing: Plotkin SS. Systems and methods for predicting misfolded protein epitopes by collective coordinate biasing. International application No: PCT/CA2016/051306. 2016
- Manuscript on Collective Coordinates applied to SOD1: Peng X, Cashman NR & Plotkin SS, J Phys Chem, 2018

ProMIS discovery platform applied to alpha-synuclein: Selectivity advantage over other antibodies



- Generation of monoclonal antibodies through immunization with peptide/monomer or synthetic oligomers gives rise to antibodies that cross-react to varying degrees with non-toxic monomers and/or insoluble fibrils
  - Binding to non-toxic species is likely to reduce the effective dose and diminish efficacy
  - Binding to monomers/physiological tetramers may interfere with normal alpha-synuclein function
  - Interaction with circulating alpha-synuclein may cause infusion reactions (observed with Prothena antibody at 60 mg/kg)
- The ProMIS platform allows for identification of conformational epitopes predicted to be exposed solely on toxic species -> Immunization with these epitopes allows for generation of antibodies selective for pathogenic α-syn
  - The selectivity and activity of the antibodies is validated empirically in vitro and in vivo
  - Reactivity with patient-derived material (native alpha-syn species) is an essential component of the validation process

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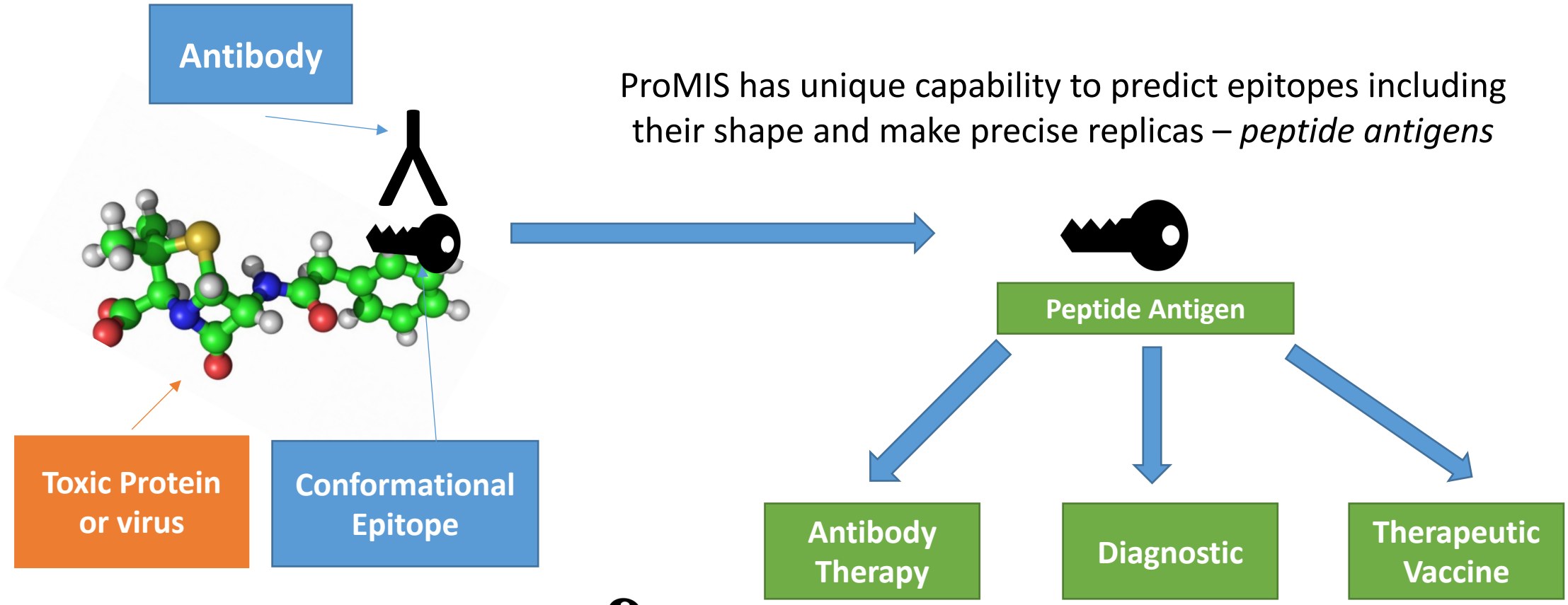
Image adapted from BioArctic Healthcare Day 2017

FAQs

- What is unique about ProMIS technology platform?	- ProMIS combines physics and biology to predict portions of mis-folded proteins exposed as epitopes; it requires physics to predict ***shape,*** and shape is the only difference between normal and toxic mis-folded proteins
- If the science is clear about toxic, mis-folded proteins, why are large companies not targeting selectivity?	- Many large pharma programs failing/working modestly now were designed before the science was as clear - Without ProMIS unique approach to antibody design it is impossible to systematically create selective antibodies
- Will others eventually catch up, and compete?	- Eventually, advances in computing power (like IBM quantum computing) will enable others to predict shape and create selective antibodies - ProMIS is building a large IP estate in the mis-folded protein area; there are only so many conformational epitopes exposed - ProMIS identifies a few for every target protein, creates antibodies, and files for IP

ProMIS unique technology platform: computation of conformational epitopes exposed only on mis-folded proteins, led to PMN310, and a therapeutic vaccine



Antibodies bind to targets called *EPITOPES* ( ) which have a specific conformation or shape

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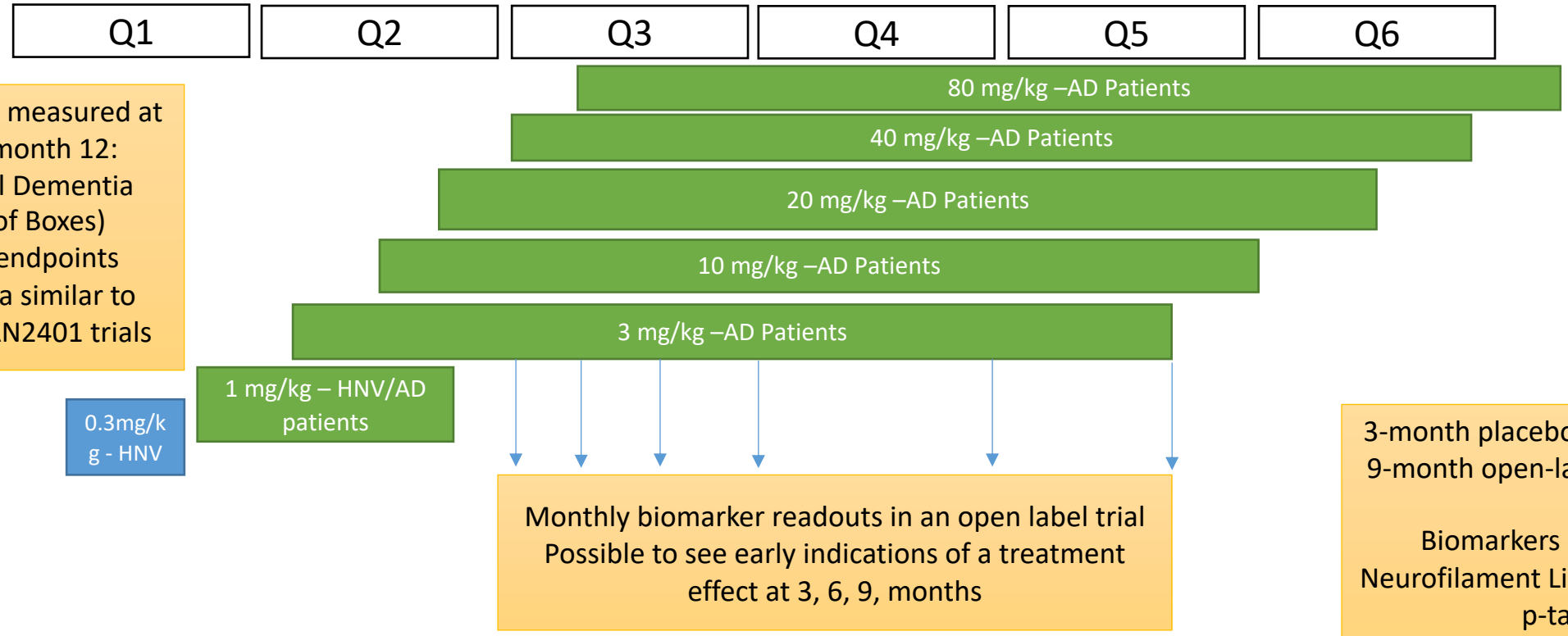
ProMIS Template Development Program- Summary

- First step is computational predictions of epitopes, creating peptide antigens, immunizing mice and/or rabbits to create initial antibody candidates
- Lead candidates selected through assays to screen for selectivity, including selective binding to patient bio-samples vs healthy controls, in vitro assays
- Lead candidates humanized (Vendor – Abzena), CHO cell line (Selexis), GMP manufacturing (KBI), and GLP tox conducted to enable IND
- All programs will treat patients in the first in human study (after healthy normal in low dose arms)
- All programs targeting disease populations with elevated biomarkers (like NfL – neurofilament light, an indicator of the rate of neuronal death; or p-tau, phosphorylated tau, a measure of the toxic tau created by toxic amyloid-beta oligomers), enabling a dose-dependent treatment response to be seen in monthly blood draws, quantitative assays, that could correlate with clinical endpoints
- Extremely capital efficient relative to past programs in neurodegenerative disease. ProMIS estimates:
  - Discovery - <6 months, <$100k
  - IND enabling work: $8MM - $10MM
  - Clinical program to initial POC: $10MM - $15MM

Phase 1 trial design concept: biomarkers in addition to clinical endpoints can give a signal suggesting therapeutic benefit early in clinical development



Clinical endpoints measured at baseline and month 12:
CDR-SB (Clinical Dementia Rating, Sum of Boxes)
Other clinical endpoints
Inclusion criteria similar to aducanumab, BAN2401 trials

Monthly biomarker readouts in an open label trial
Possible to see early indications of a treatment effect at 3, 6, 9, months

3-month placebo-control, then 9-month open-label extension

Biomarkers including:
Neurofilament Light chain (NfL)
p-tau

HNV = healthy normal volunteers

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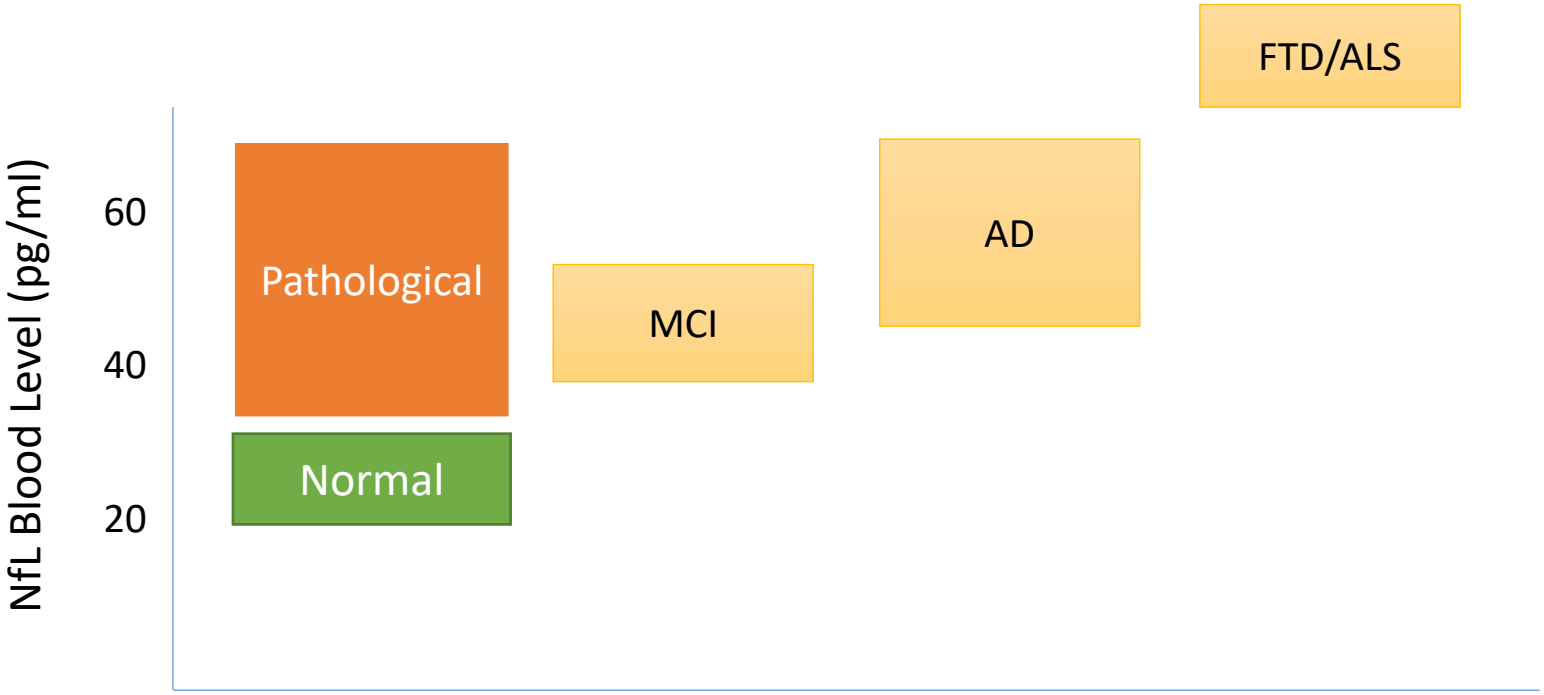
Neurofilament light (NfL) is a measure of the rate of neuronal death…..



Sources: from AAIC 2018 ABBVIE, Wash U, U Sorbonnne;
Rohrer et al, 2016 AAN; Mattson et al, JAMA Neurology 2017

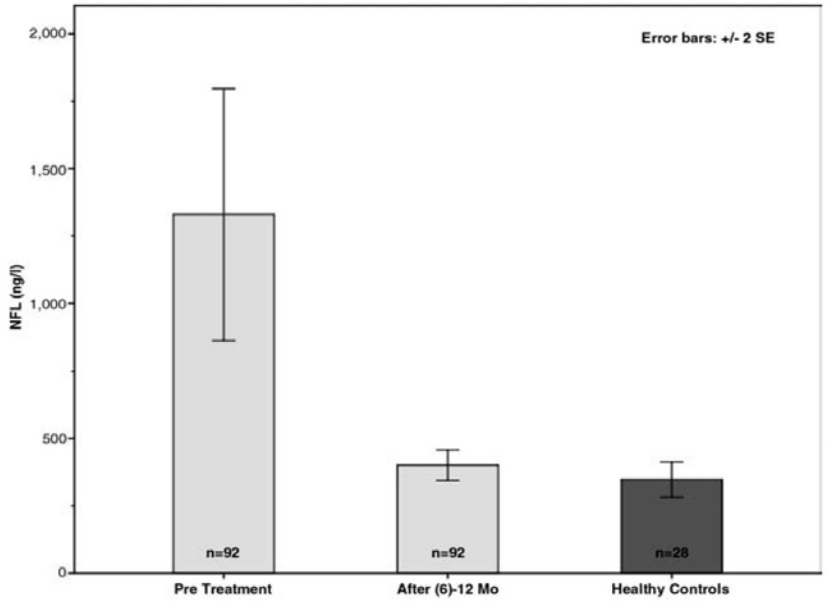
From *JAMA* Neurology, April 22 2019

" The findings suggest that plasma NfL can be used as a noninvasive biomarker associated with neurodegeneration in patients with AD and may be useful to monitor effects in trials of disease-modifying drugs"

Mattsson, et al, JAMA Neurology

NfL has proven to be a valuable pharmacodynamic marker in multiple sclerosis and SMA (spinal muscular atrophy)

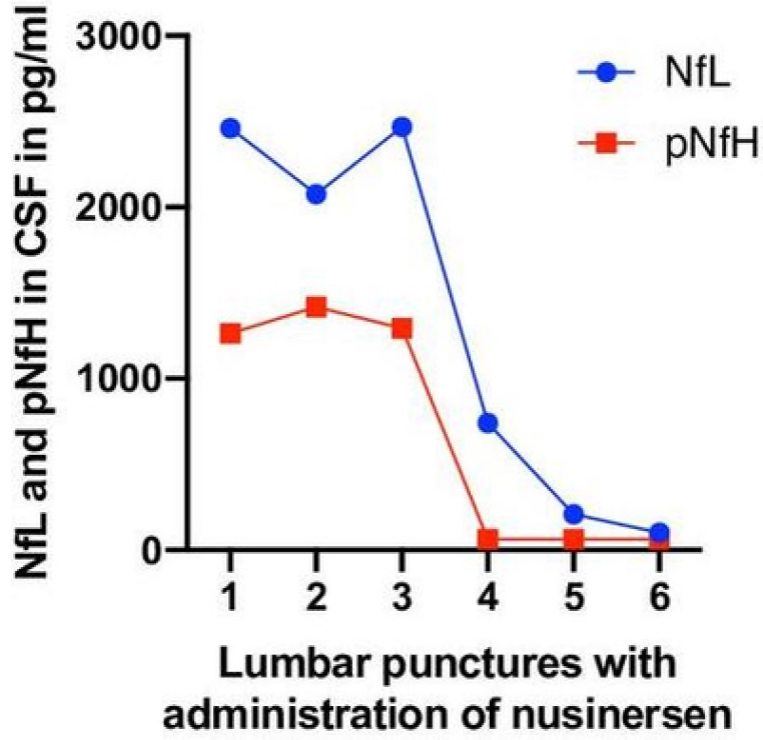
Tysabri in multiple sclerosis



Neurofilament light in cerebrospinal fluid following monthly administration of natalizumab (300 mg) in 92 patients with multiple sclerosis

Gunnarsson, et al Annals of Neuro 2011

Spinraza in spinal muscular atrophy



Winter B. et al, J Neurol Neurosurg Psychiatry 2019

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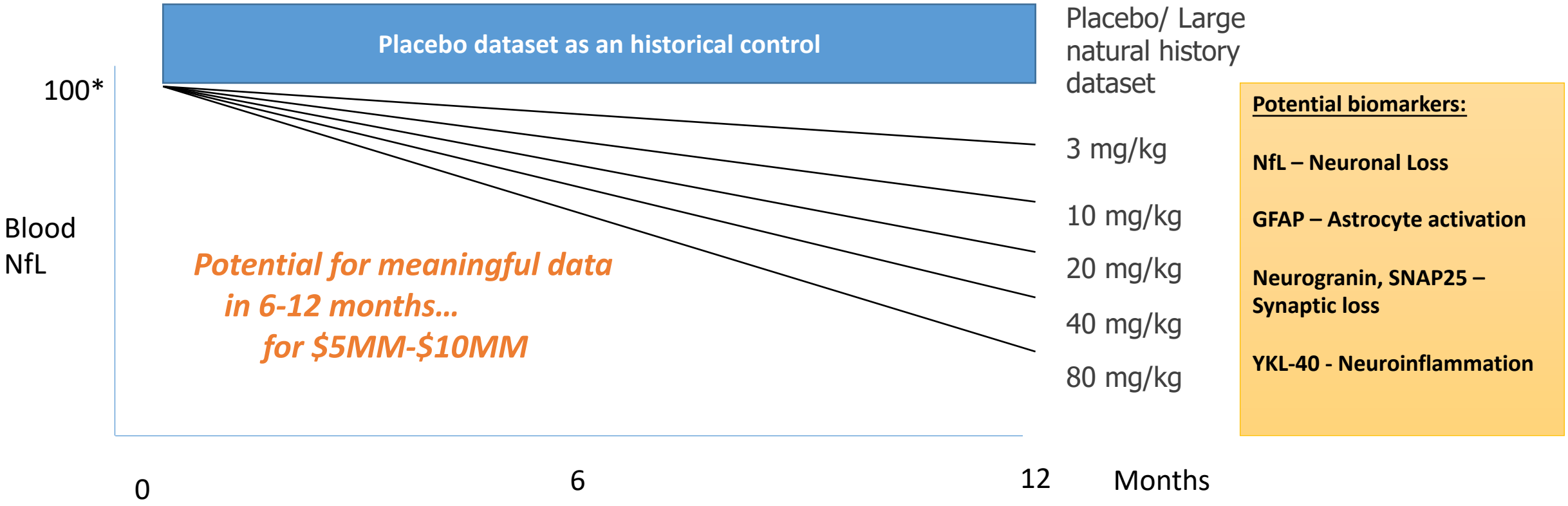
Hypothetical example – Phase 1 biomarker readout could show a disease-modifying treatment effect in a dose-dependent fashion *at a cost of ~$10MM*



Potential biomarkers:

NfL – Neuronal Loss

GFAP – Astrocyte activation

Neurogranin, SNAP25 – Synaptic loss

YKL-40 - Neuroinflammation

* 100 = patient baseline value

Dose escalation design: 3-month placebo-control, then 9-month open-label extension

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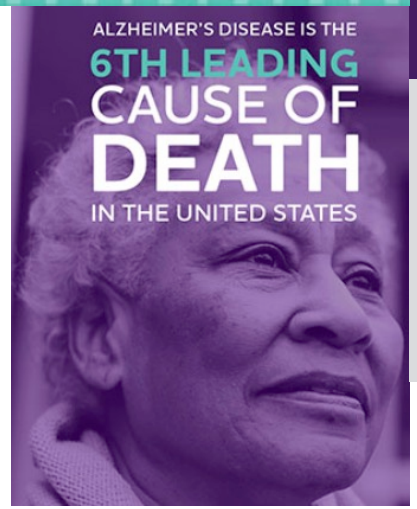
ProMIS Alzheimer's Lead Program: PMN310

Alzheimer's Disease: the epidemic of this century

"Will Bankrupt Medicare
if Therapy is not developed"

Direct and Indirect Costs
Today in the US $500BB….
…. and the number
is tripling by 2030….



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Alzheimer's is at an inflection point like immuno-oncology ten years ago

"Neuroscience has the potential to be in the '20s what oncology has been in the last decade."

– Bill Anderson, CEO of Roche Pharmaceuticals (JPM 2020)

Immuno-Oncology

~2000's

- Numerous failures
- Skepticism, lack of capital

2010 -inflection

- First cancer vaccine approved (sipuleucel-T)
- First positive clinical data for PD-1 (nivolumab)

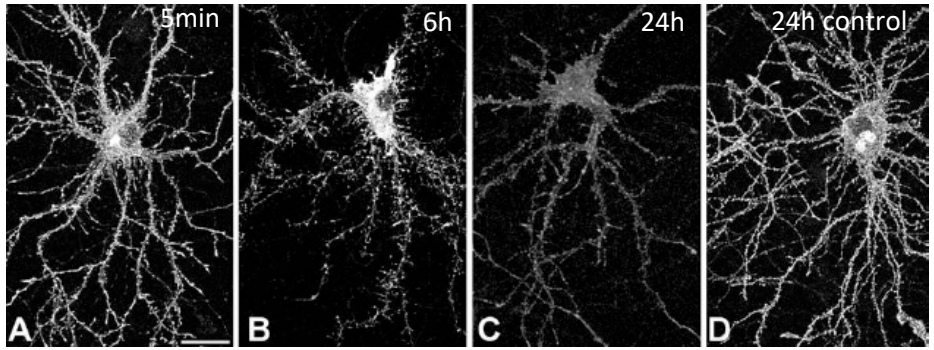
2017/2018 - validation

- Gilead acquires Kite (2009 start up) $11.9BB
- Celgene acquires Juno (2013 start up) $9BB

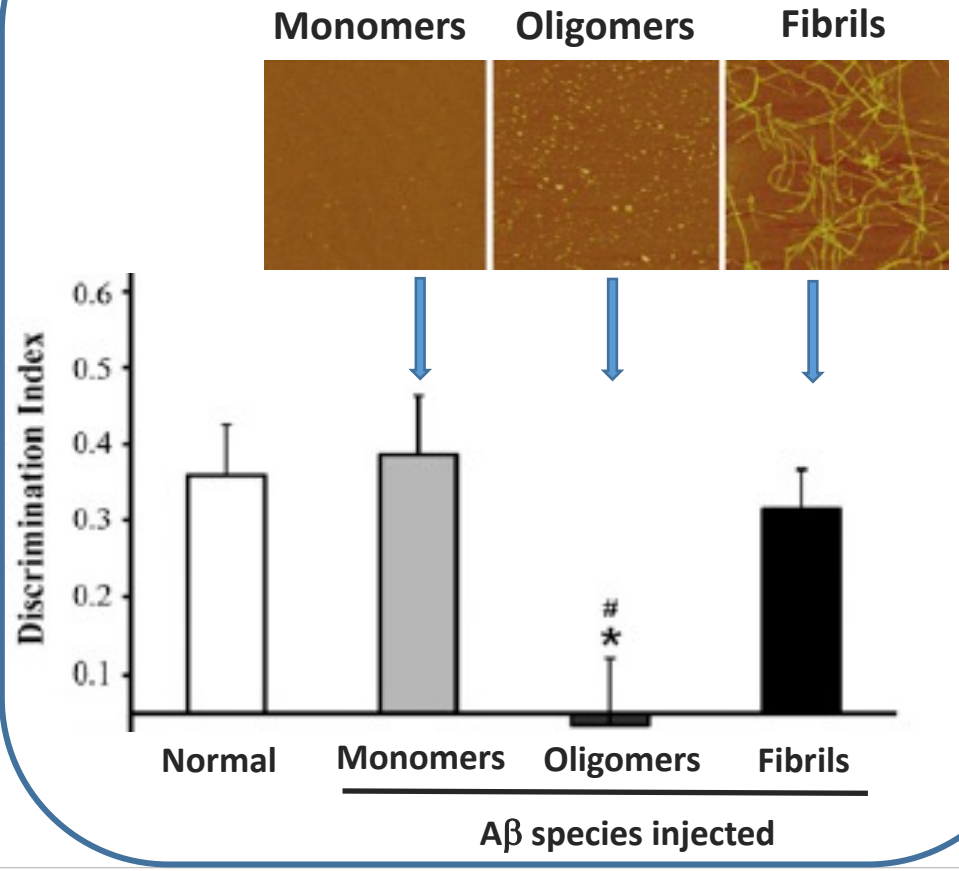
Alzheimer's disease: soluble toxic Aβ oligomers – not plaque or monomers – are the most neuropathogenic Aβ species

- Synapse abnormalities and memory impairment correlate poorly with plaque burden in human and mouse AD[1,2]
- Aβ monomers and Aβ insoluble fibrils (plaque) have little or no demonstrable toxicity in vitro or in vivo[3-5]
- Soluble Aβ oligomers show the highest degree of neurotoxicity[6]
  - Toxicity in primary neuron cultures and brain slices[3,5,7-9]
  - Induction of cognitive impairment in rodents[3,4,10]

Synaptotoxicity of human Aβ oligomers on hippocampal neurons in vitro[7]



In vivo impairment of recognition memory by Aβ oligomers, not monomers and not fibrils[10]



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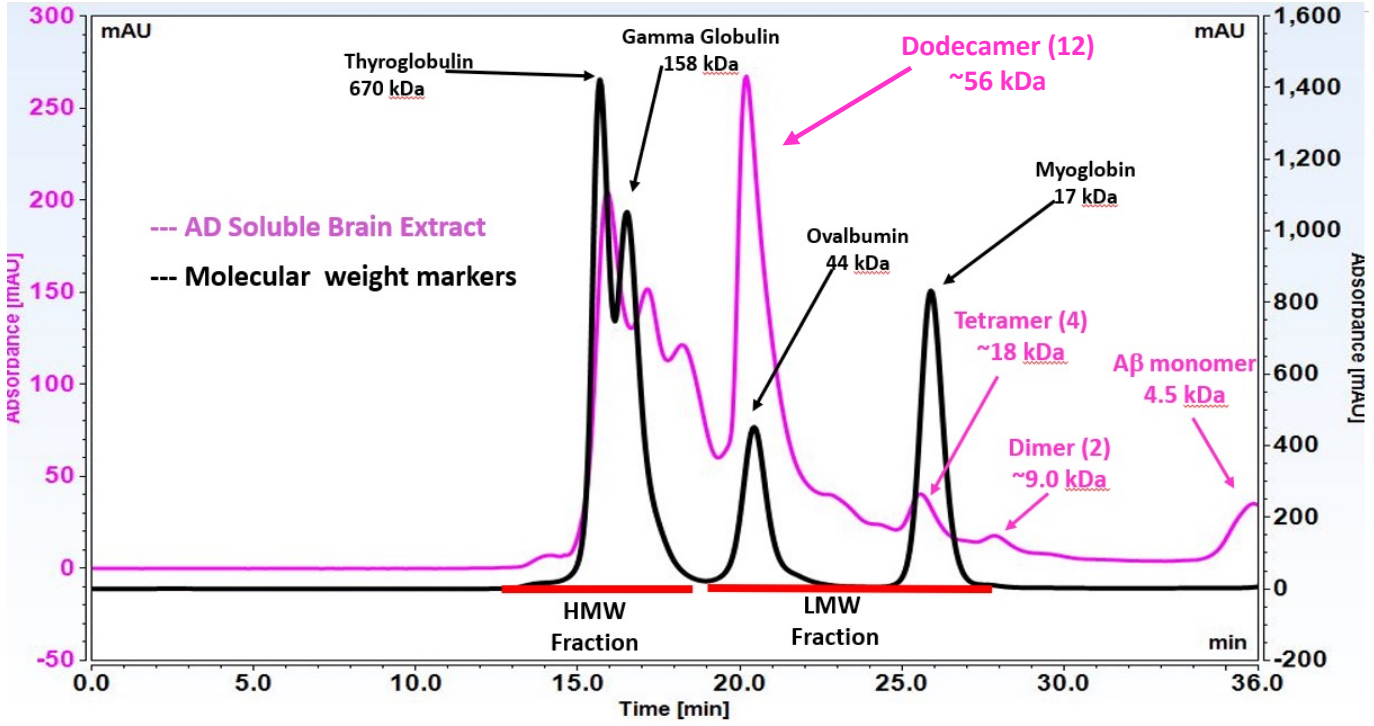
[1]Jacobsen et al, 22006, PNAS; [2]Brier et al, 2016, Science Trans Med; [3]Shankar et al, 2008, Nature Med; [4]Cleary et al, 2005, Nature Neuroscience; [5]Hong et al, 2016, Science; [6]Benilova et al, 2012, Nature Neuroscience - Review; [7]Lacor et al, 2007, J Neuroscience; [8]Jin et al, 2011, PNAS; [9]Lauren et al, 2009, Nature; [10]Balducci et al, 2010, PNAS

Not all oligomers are equivalent ….. soluble low molecular weight toxic oligomers drive neurotoxicity and disease progression

- AD brain homogenates contain heterogeneous Aβ aggregates dominated by high molecular weight species including protofibrils (>70kDa, or ~15+ "monomers")[1,2]
- Neurotoxic activity resides primarily in the **LMW (low molecular weight) fraction** (<70 kDa)[1]
- A subset of the LMW oligomers are the most toxic oligomers (**dimer, tetramer, dodecamer)**[1,3-7]

Size exclusion chromatography of AD brain extract



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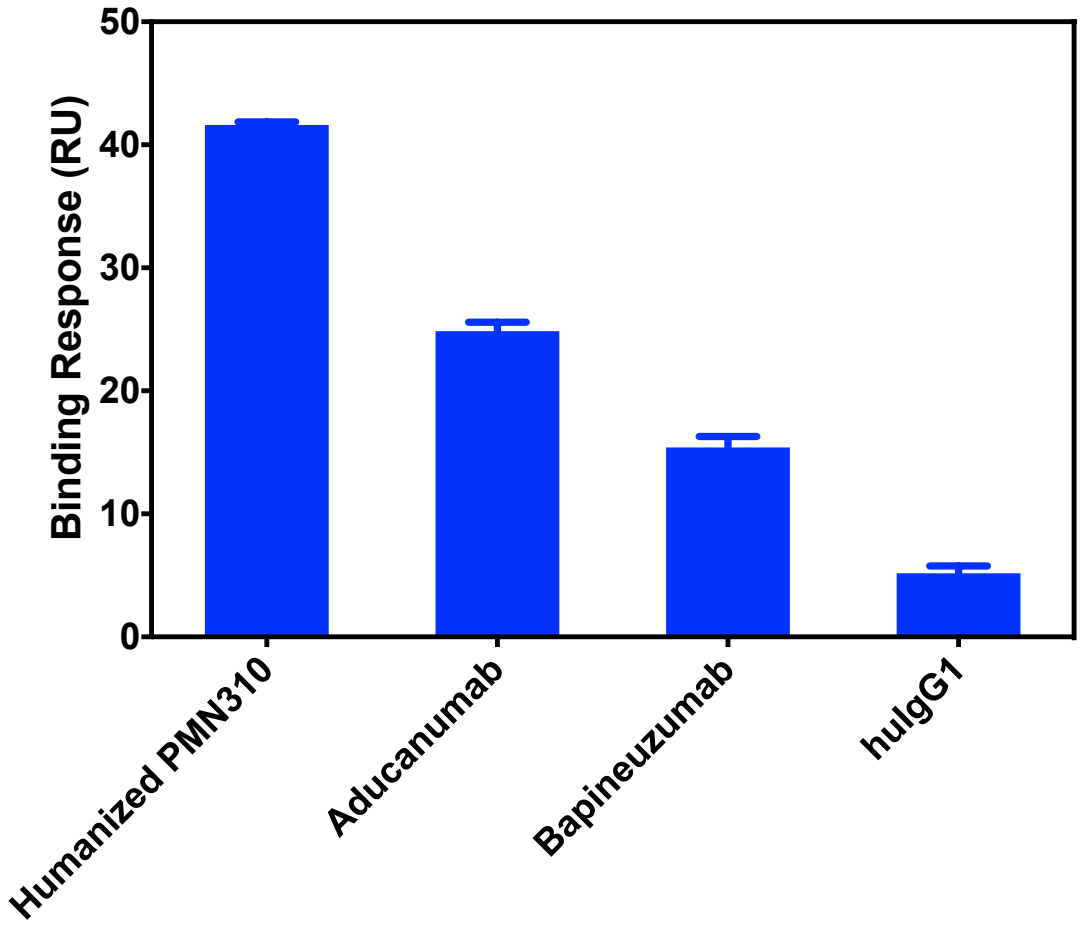
[1]Yang, T et al, 2017, J Neurosc; [2]Esparza TJ et al, 2016, Nature Scientific Reports; [3]Shankar, GM et al, 2008, Nat Med; [4]Jin, M et al, 2011, PNAS; [5]Cleary, JP et al, 2005, Nature Neurosc; [6]Lesne, SE et al, 2013, Brain; [7]Ferreira, ST et al, 2015, Frontiers in Cellular Neuroscience

PMN310 shows superior binding to toxic oligomers from human AD brains vs other antibodies directed against amyloid-beta

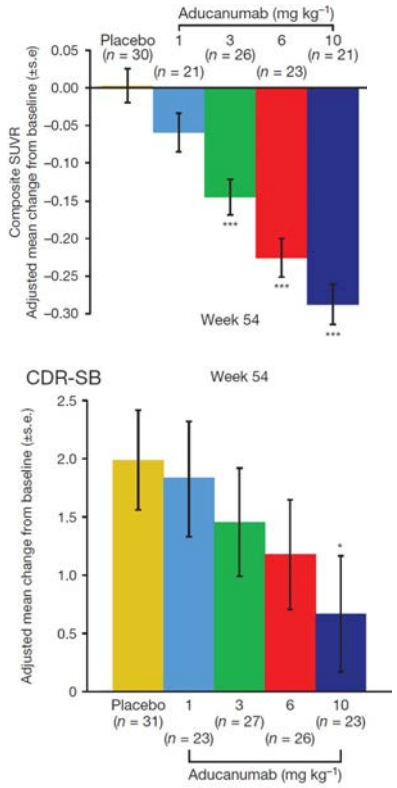


- Binding of antibodies to the toxic oligomer-enriched LMW fraction of soluble human AD brain extract was evaluated by surface plasmon resonance (SPR)
- Results representative of over 10 SPR runs with extracts from 11 different AD brains
- huIgG1 = Background control

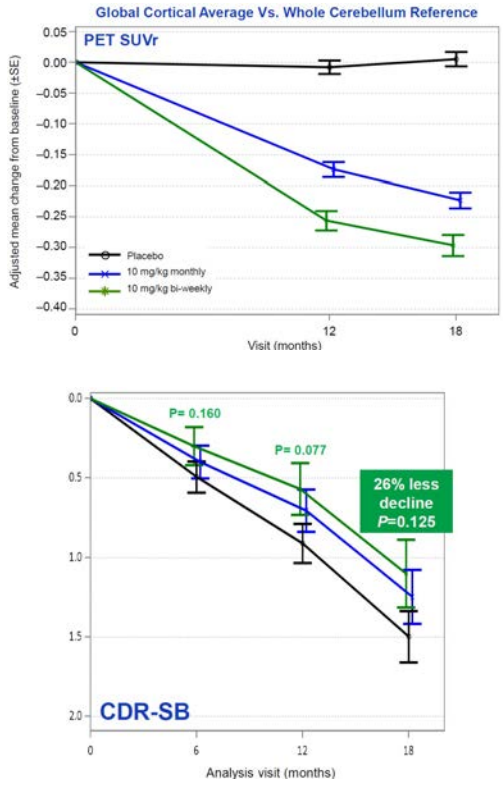
Source for comparative reagents: Creative Biolabs

Four positive trials…partially selective antibodies…higher dose over long duration leads to better outcomes, but all limited by brain swelling (ARIA-E)

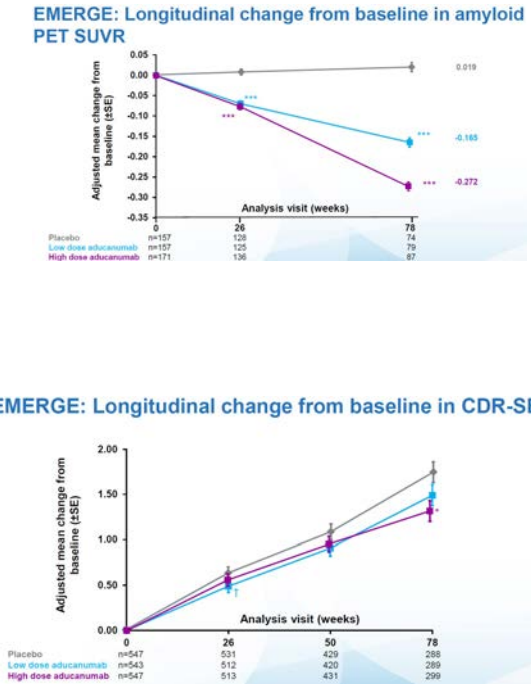
Biogen Ph1b "PRIME" December 2014



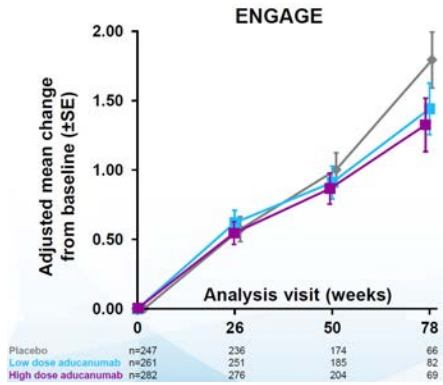
Eisai BAN2401 Phase 2 July 2018



Biogen pivotal EMERGE Dec 2019*



Biogen pivotal ENGAGE Dec 2019**



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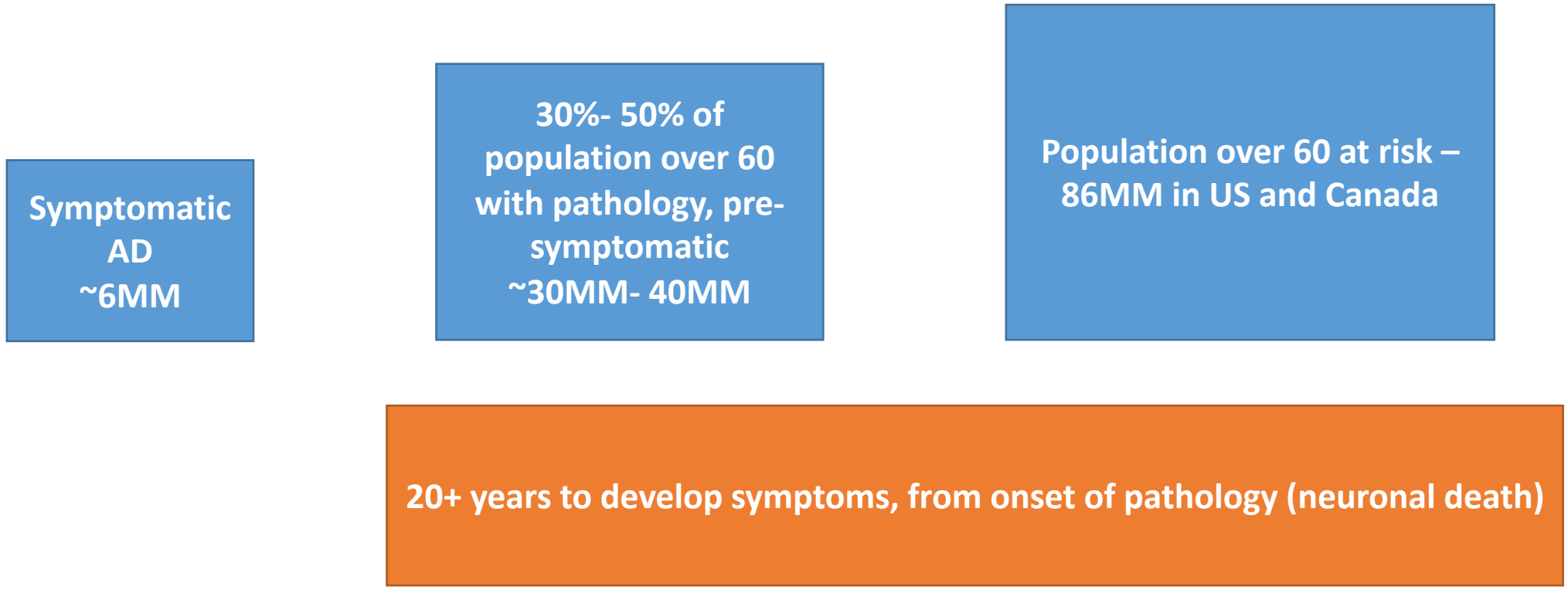
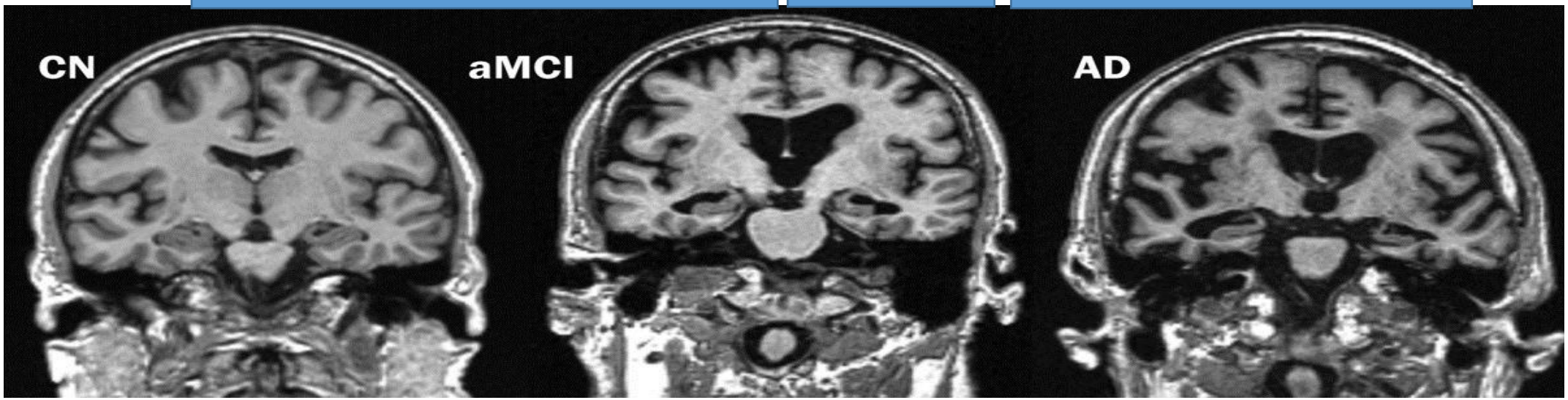
* Final Intent-to-treat data set ** Post-protocol version 4 data set

The goal in Alzheimer's is prevention – a large and growing market



Billions of neurons were killed leading to the Alzheimer's brain on the right……..toxic amyloid is a root cause

15 -20 years pre-symptomatic | 5 years MCI | 10 years Alzheimer's



Icelandic Genetic mutation:
No amyloid
No AD

Down Syndrome:
Excess amyloid
Early, High % AD

Healthy Brain

Brain with Mild Cognitive Impairment

Brain with AD[1]

Toxic, mis-folded amyloid plays a role throughout disease

Toxic, mis-folded amyloid hyper-phosphorylates tau

Disease detection and progression measurable in blood – NfL (neuronal death), p-tau – (phosphorylated tau)

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[1] Reviewed in Bloom 2014, JAMA Neurol

ProMIS therapeutic vaccine for Alzheimer's disease

- Large patient population with symptomatic AD in need of effective treatment (n≥5M, USA) –> **Therapeutic vaccination**
- Ever greater number of individuals in the pre-symptomatic phase of the disease (potentially 40% of the population over 65, ~50M) –> **Prophylactic preventative vaccine**
- Vaccine approach will benefit from recent progress in the development of blood-based biomarkers of neurodegeneration to diagnose AD and identify individuals at risk of developing disease
- **ProMIS discovery platform being applied to devise a safe and effective vaccine to induce a specific immune response against toxic Aβ oligomers**
  - Identified a set of 6 conformational peptide epitopes selectively exposed on toxic Aβ oligomers
  - All 6 peptide epitopes shown to be capable of inducing selective and protective antibodies against toxic Aβ oligomers
  - Successful proof of concept vaccination study conducted with one of the peptides (cSNK) in a mouse model of AD (APP/PS1 mice): Neuronal protection and improvement in cognitive deficits[2]
  - Concept: multivalent vaccine with some or all 6 Aβ peptides. Tau peptides recently identified could also be included.
- **Immediate aims: Construct and test multivalent Aβ vaccine for ability to induce a protective antibody response**

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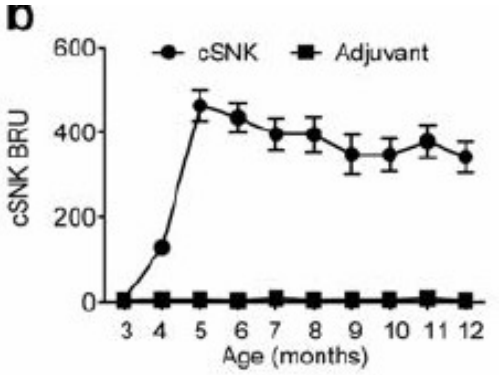
[1]Marciani DJ, 2019, AAAS Research, https://doi.org/10.34133/2019/5341375; [2]Silverman, JM et al, 2018, ACS Chem Neurosci

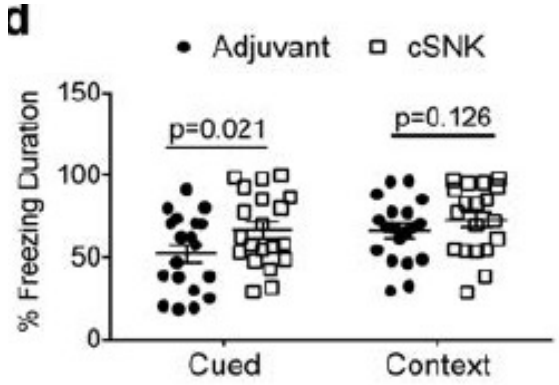
Benefit of vaccination with ProMIS Aβ oligomer (AβO) epitope

Mice vaccinated with conformational Aβ oligomer epitope (cSNK) coupled to KLH

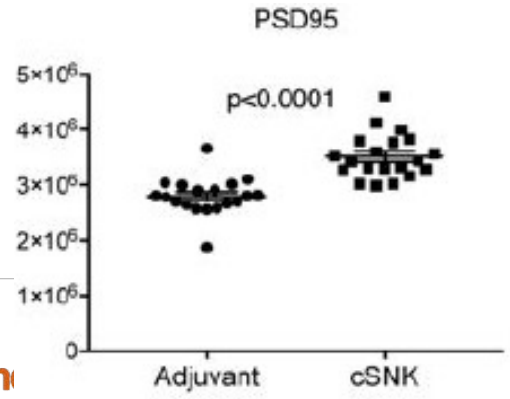
Robust, sustained antibody response (SPR)



Improvement in behavioral deficits of APP/PS1 mice

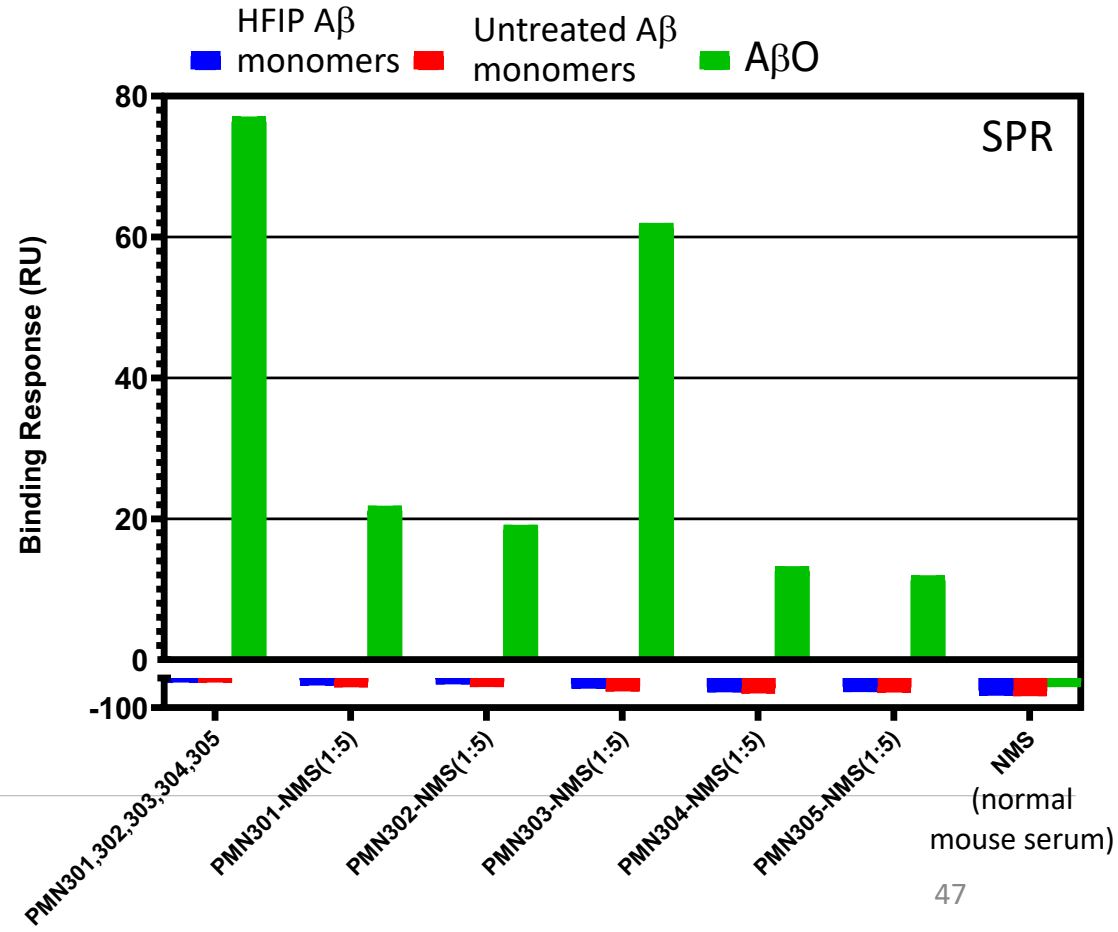


Protection against synaptic damage



Silverman et al. 2018. ACS Chem Neurosci 9: 1591-1606

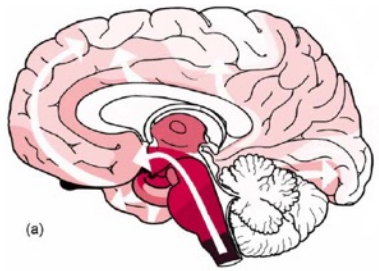
Biological support for multivalent vaccine approach: Greater binding with mixture of sera from mice immunized with different AβO epitopes vs immune serum against individual peptides



Alpha-Synuclein Program

Alpha-synuclein is the major driver of Parkinson's disease and other synucleinopathies

- Synucleinopathies: Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by loss of dopaminergic neurons located in the midbrain and the presence of intraneuronal inclusions (Lewy bodies/Lewy neurites) consisting mainly of aggregates of α-synuclein (α-syn). Accumulation of insoluble α-syn fibrils in the brain is also observed in dementia with Lewy bodies (LBD) and multiple system atrophy (MSA).
- Genetic mutations (A53T, A30P, E46K, H50Q, G51D) and duplications/triplications of the α-syn gene cause inherited familial PD[1]
- Polymorphisms in the α-syn gene and its promoter are associated with an increased risk of sporadic PD[1]
- Mice injected with aggregated α-syn from diseased patient brains develop synucleinopathy-like pathology and symptoms[2]
- Aggregated α-syn propagates from cell to cell in a prion-like manner in cell culture[3] and in animals[4,5] mimicking disease progression in patients



Braak, H et al, Neurobiol of Aging, 2003

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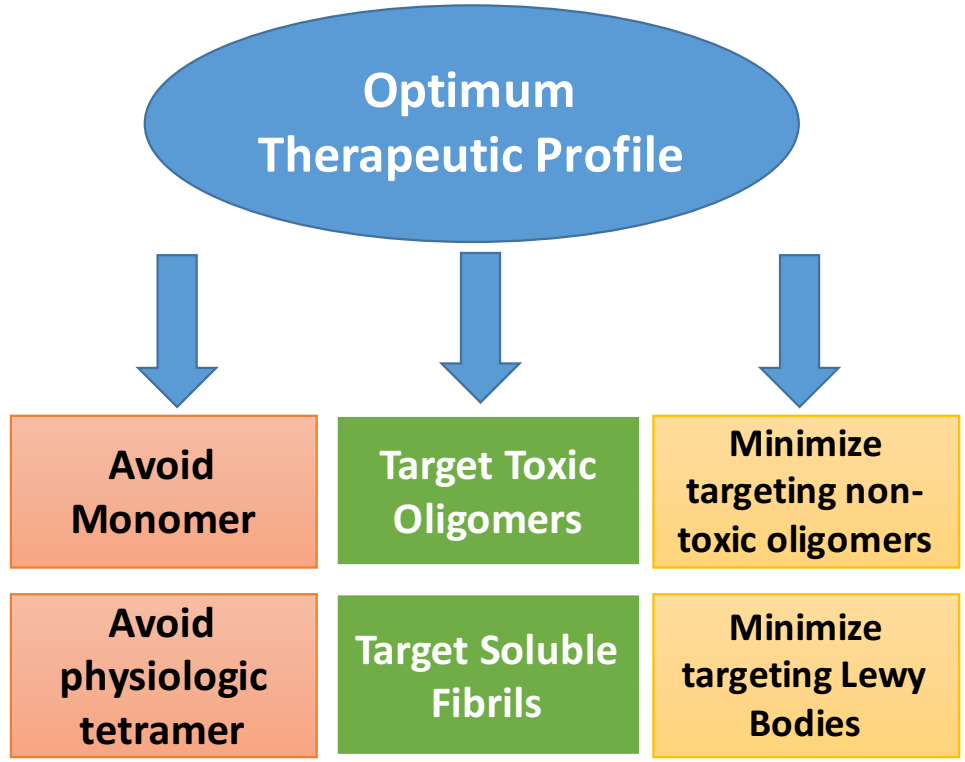
[1]Reviewed in Vaikath, NN et al, J Neurochem 2019; [2]Reviewed in Karpowicz Jr, RJ et al, Lab Invest 2019; [3]Choi et al, Cell Reports 2018; [4]Peelaerts et al, Nature, 2015; [5]Froula JM et al, JBC 2019

Therapeutic imperative: selectively target only the toxic α-synuclein aggregates

- **Alpha-synuclein exists in different forms including normal, physiologically important conformations and toxic forms:**
  - Monomers play an important role in the regulation of synaptic vesicle trafficking and release as well as neuronal survival[1]
  - Physiological α-syn tetramers inhibit aggregation and must be preserved for healthy α-syn homeostasis[2-4]
  - Lewy bodies consisting of insoluble α-syn are a marker of disease but unlikely to have direct neurotoxicity[5]
  - Recent evidence indicates that α-syn toxicity resides primarily with soluble oligomers[6,7]
  - Progression of disease is likely mediated by oligomers and small soluble fibrils of α-syn that have been shown to propagate from cell to cell in a prion-like manner in vitro[8] and in vivo[9]
- **Maximal efficacy and safety is expected to require selectivity for the toxic forms of α-syn, oligomers and/or small soluble fibrils, while avoiding physiologic forms of α-syn**



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[1]Lashuel HA et al, Nat Rev Neurosci, 2013; [2]Nuber et al, Neuron, 2018; [3]Dettmer et al, PNAS, 2015; [4]Foulds et al, Scientific Reports, 2013; [5]Vaikath NN et al, J Neurochem 2019; [6]Fusco et al, Science, 2017; Desplats P et al, PNAS, 2009; [8]Choi et al, Cell Reports, 2018; [9]Peelaerts et al, Nature, 2015

ProMIS' technology platform has created antibodies with greater selectivity than other α-synuclein-directed antibodies

Target Properties	PMN Antibodies	Prothena/ Roche	BioArctic/ ABBVIE	Neurimmune/ Biogen
No binding to monomers	Yes	X	+/-	X
No binding to physiological tetramers	Yes	X	+/-	X
Binding to oligomers/small soluble fibrils	Yes	Yes	Yes	Yes
Binding to native toxic α-syn in PD/LBD brain extract	Yes	Yes	Yes	Yes
Little or no binding to insoluble fibrils (Lewy bodies)	Yes	X	X	X

Limited benefit in PASADENA trial may be due to lack of selectivity

Discontinued: poor results may be due to lack of selectivity

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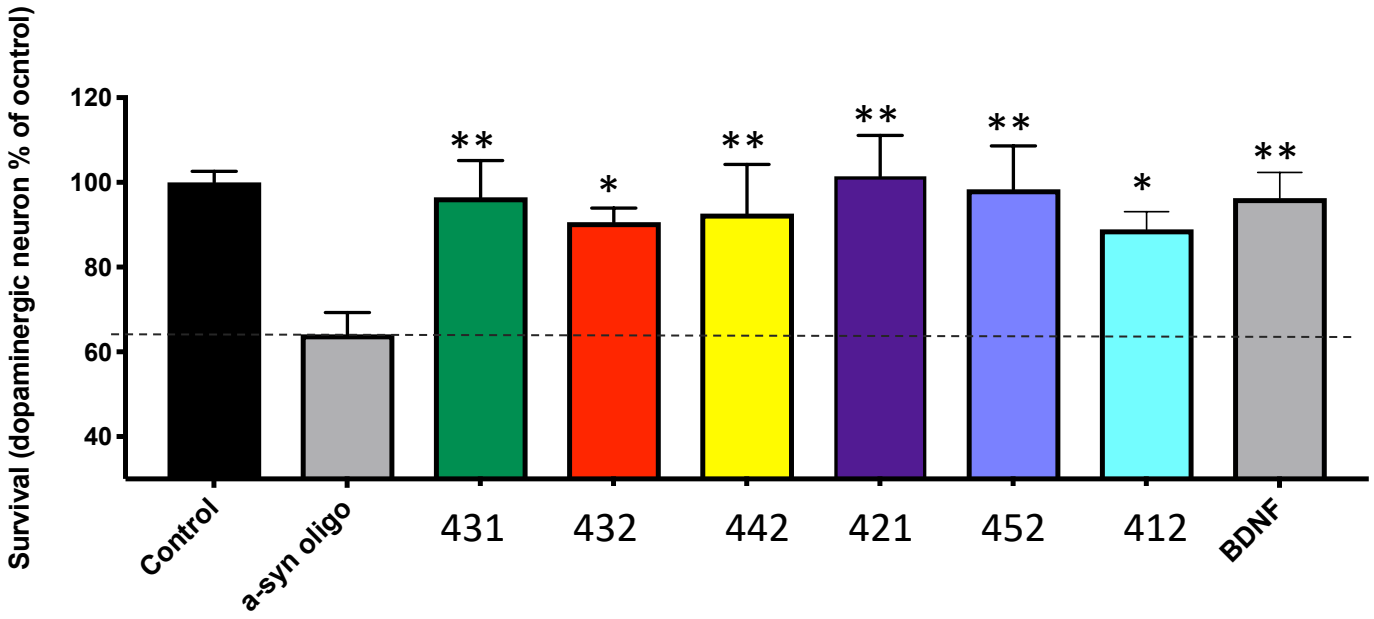
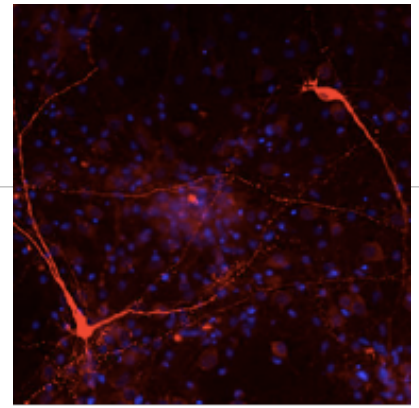
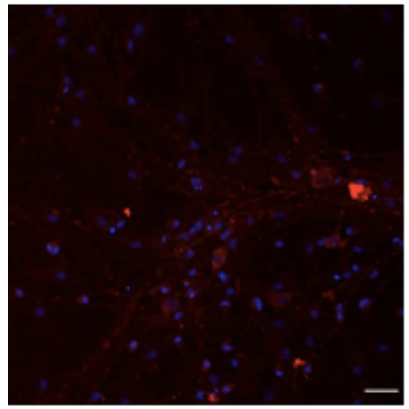
ProMIS antibodies protect dopaminergic neurons against α-synuclein oligomer toxicity *in vitro*





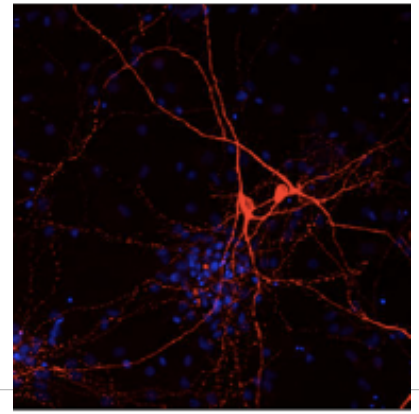
CONTROL

Normal neurons in bright red



α-SYN OLIGOMERS

Neurons killed by toxic oligomers



PMN ANTIBODY + α-SYN OLIGOMERS

Neuronal death blocked by PMN antibody

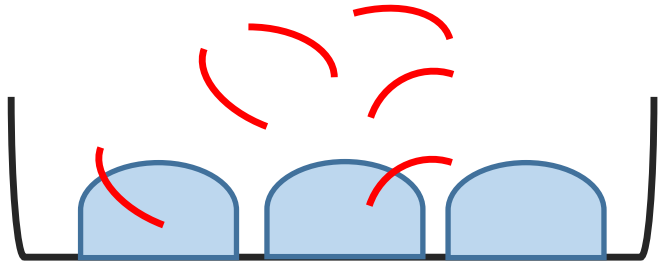
- Multiple antibodies provide neuroprotection in the same range as the brain-derived neurotrophic factor (BDNF) positive control
- As expected, antibodies alone had no effect on viability (not shown)

*p<0.05, **p<0.01 vs a-syn oligomers alone

ProMIS™ Neurosciences

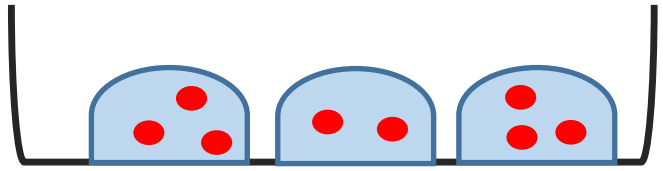
ProMIS antibodies inhibit α-syn propagation: Reduced PFF uptake and formation of aggregates

Human soluble α-syn fibrils +/- PMN antibody

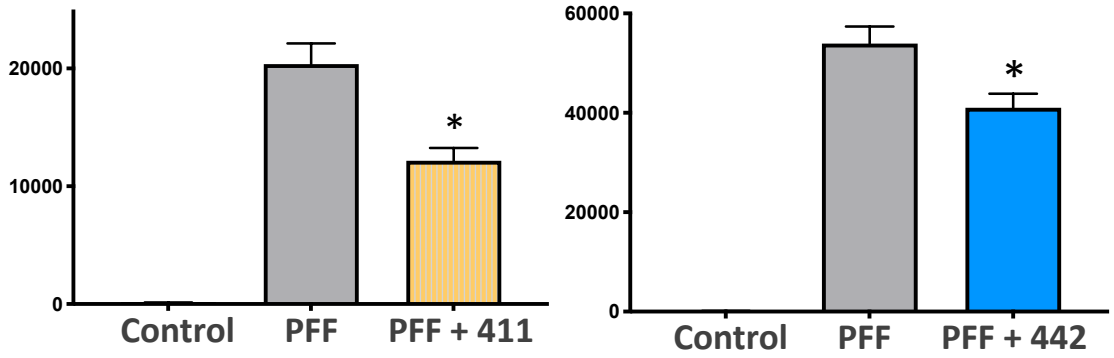


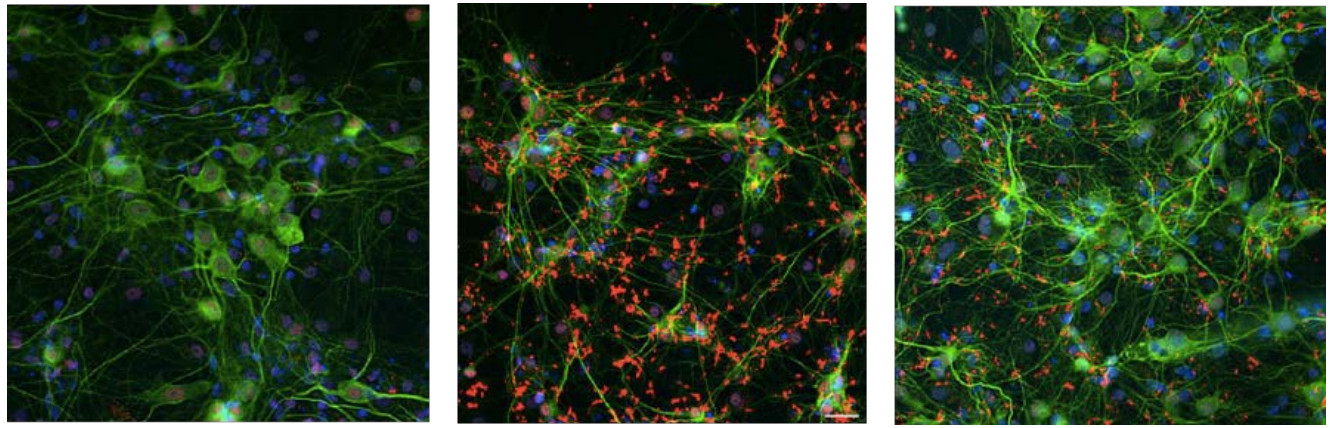
14 days

Staining for **human α-syn aggregates**



ProMIS antibodies significantly decrease formation of α-syn aggregates





CONTROL | **α-SYN SOLUBLE FIBRILS** | **α-SYN SOLUBLE FIBRILS + PMN ANTIBODY 411**

Human α-syn aggregates stained red

*p<0.05 vs fibrils alone (PFF)

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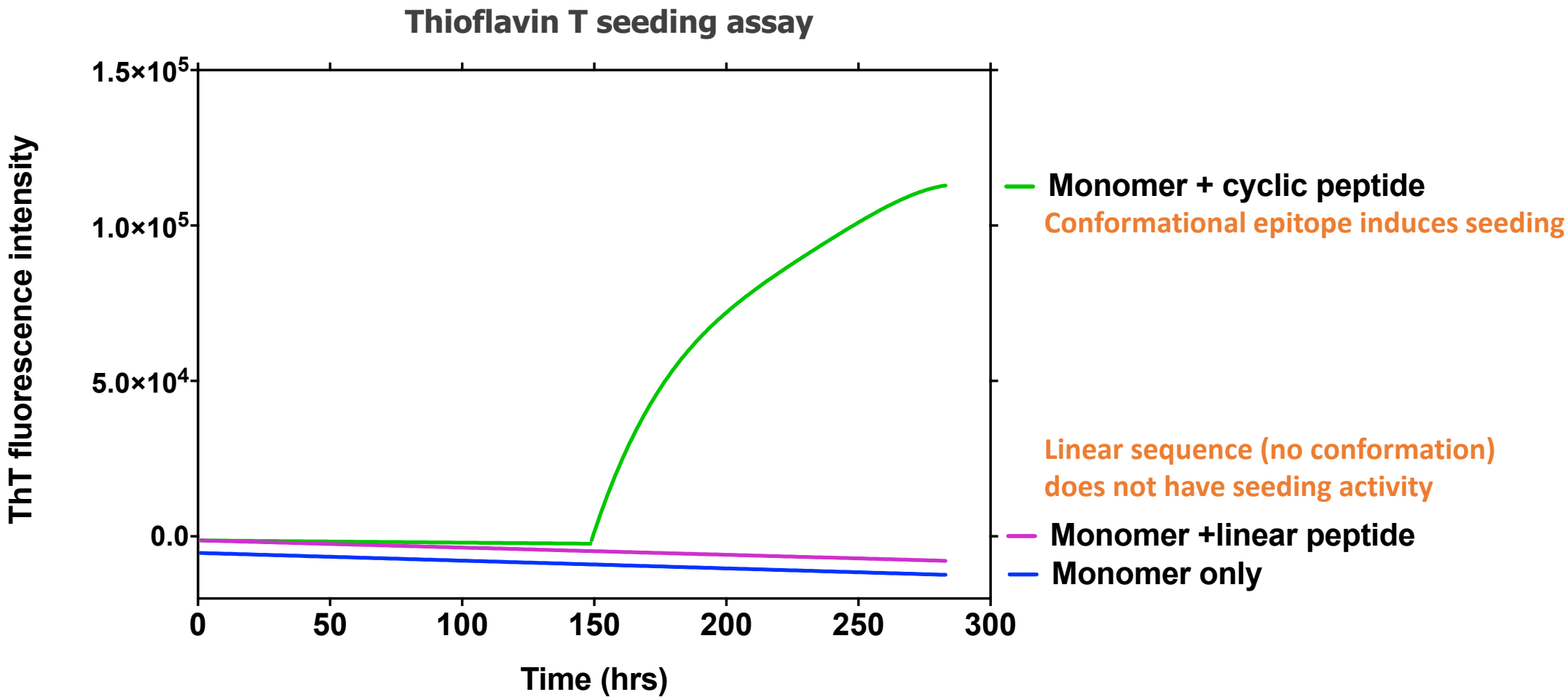
The misfolded portion of α-syn (conformational epitope) acts as a seed for toxic, prion-like propagation…confirming epitope prediction…explaining biology of disease

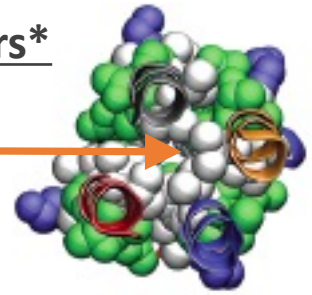


ProMIS™ Neurosciences

Only ProMIS antibodies bind toxic oligomers (tetramers) but not physiologic tetramers (SPR) – unrivalled ability to create multiple selective antibodies based on computational predictions of conformational epitopes

Physiologic tetramers*

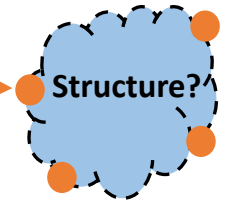
Target epitope of PMN antibodies is buried in the hydrophobic core

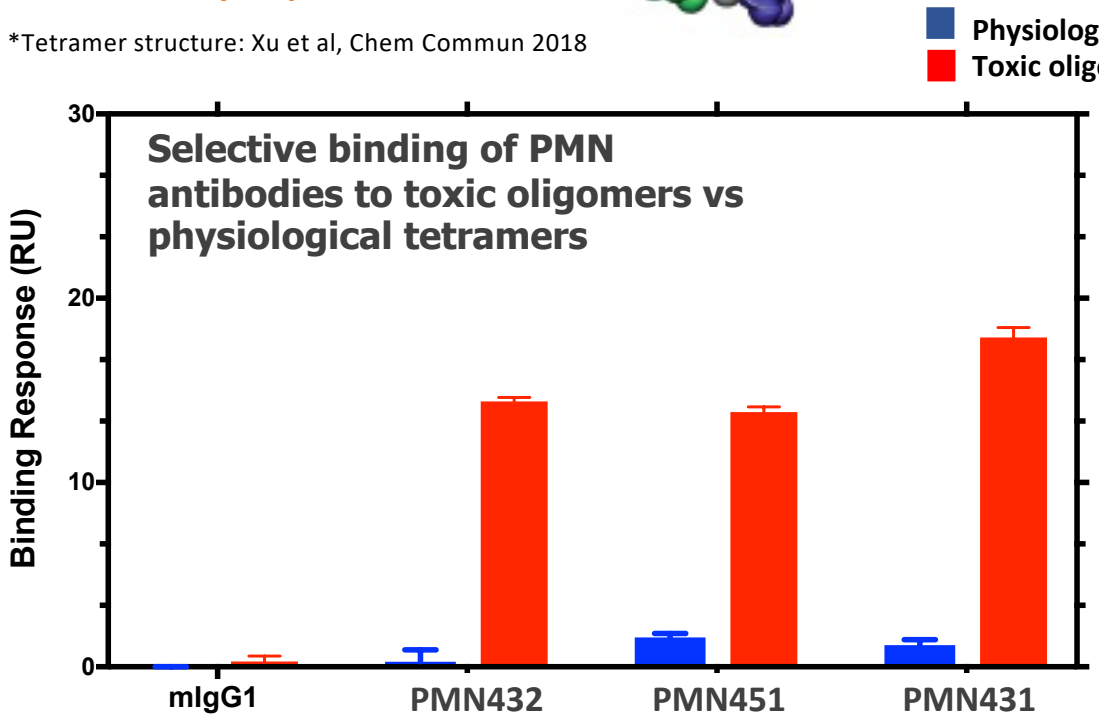


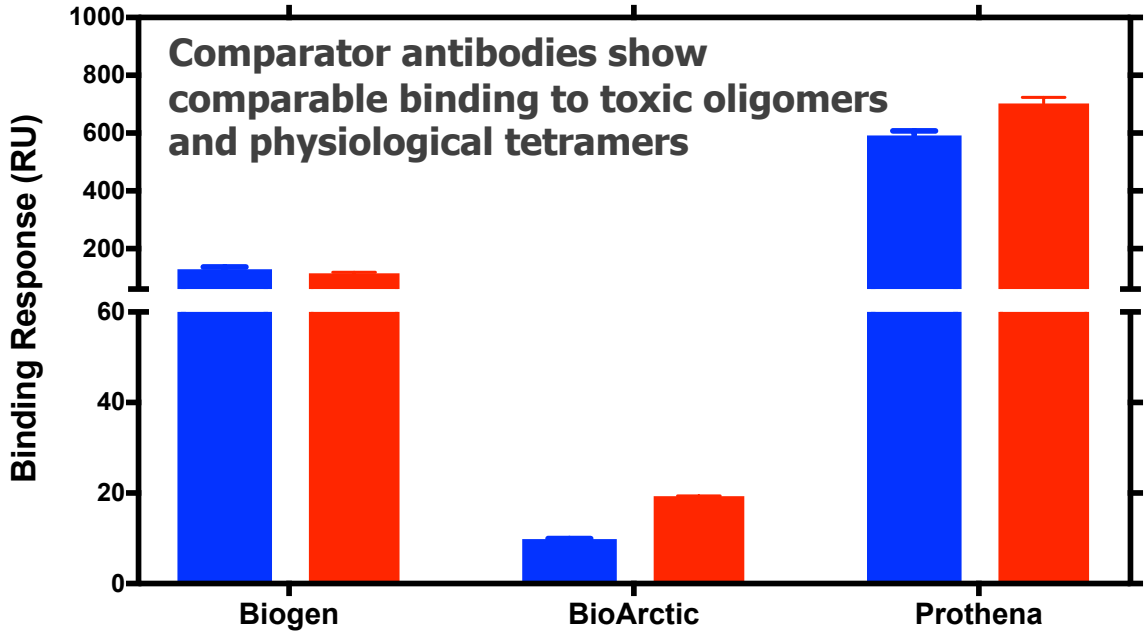
*Tetramer structure: Xu et al, Chem Commun 2018

Misfolded Toxic oligomers

Target epitope of PMN antibodies is exposed and available for binding







ProMIS™ Neurosciences

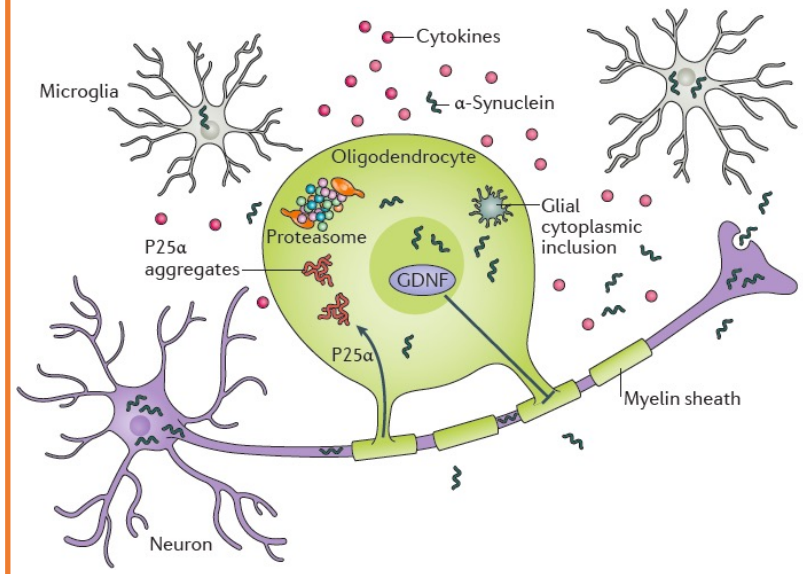
Physiological tetramers isolated by SEC following spontaneous monomer aggregation

Toxic oligomers prepared by SynAging, neurotoxicity confirmed in in vitro toxicity assay

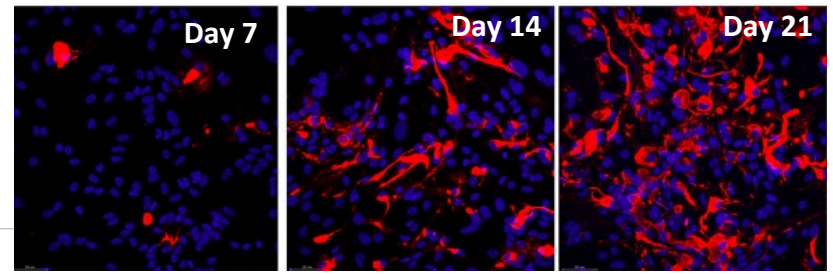
Multiple system atrophy (MSA) as a disease indication for therapeutic antibodies targeting toxic species of α-synuclein

- MSA is a rare neurodegenerative disease with an estimated prevalence of 3.4-4.9 cases per 100,000 population[1]
- MSA is characterized by rapidly progressive autonomic failure and motor symptoms with predominant parkinsonian features (MSA-P) or dominant cerebellar features (MSA-C)[1,2]
- There is no effective treatment and the mean survival from the onset of symptoms is 6-10 years[1,2]
- Histologically characterized by α-synuclein aggregates in the cytoplasm of oligodendrocytes and to a lesser extent in neurons and other glial cells[1,2]
- Alpha-synuclein aggregates from MSA brain homogenates propagate in a prion-like manner in vitro[3] and in vivo[4] and cause MSA-like neurodegeneration in mice[5]
- Suitability for clinical testing:
  - Rapid progression allows for earlier efficacy signal
  - High levels of NfL in serum represent a potential biomarker for effect on neuronal damage – early clinical PoC
  - No placebo effect observed in clinical trials to date
  - Rare disease but easy recruitment due to unmet need and existence of a global MSA Registry (GLOMAR) and supporting organizations[1,2]

α-syn glial cytoplasmic inclusions (GCI)[2]



In vitro propagation of MSA α-syn aggregates[3]



[1]Fanciulli A & Wenning GK, 2015, NEJM; [2]Krismer F & Wenning GK, 2017, Nature Neurol; [3]Krejciova Z et al, 2019, Acta Neuropathol Comm; [4]Watts JC et al, 2013, PNAS; [5]Bernis ME et al, 2015, Acta Neuropathol Comm

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ProMIS Alpha-Synuclein Program- Summary

- ***Several antibody candidates*** show desired selectivity and in vitro benefit, blocking neurotoxicity and propagation
- Alpha-synuclein protein a difficult challenge in molecular species selectivity – six molecular species of alpha-synuclein, two physiologically important that should be avoided, two toxic that should be targeted
- ***ProMIS has better selectivity than competitor antibodies*** in the clinic: Roche/Prothena antibody completely non-selective, mixed results in Phase 2 but proceeding; Biogen/Neurimmune antibody completely non-selective, discontinued.
- Only ProMIS antibodies can selectively target ***toxic*** tetramers of alpha synuclein, but not ***physiologic*** tetramers. ProMIS has achieved this with multiple antibodies, created with different conformational epitope predictions, illustrating the power of its unique platform
- Lead antibodies are undergoing humanization, next step is IND enabling work
- Target indications are MSA (multiple system atrophy, rare disease, elevated biomarkers) and Parkinson's disease

TDP-43 Program

Selective targeting of pathogenic TDP-43 for optimal safety and efficacy

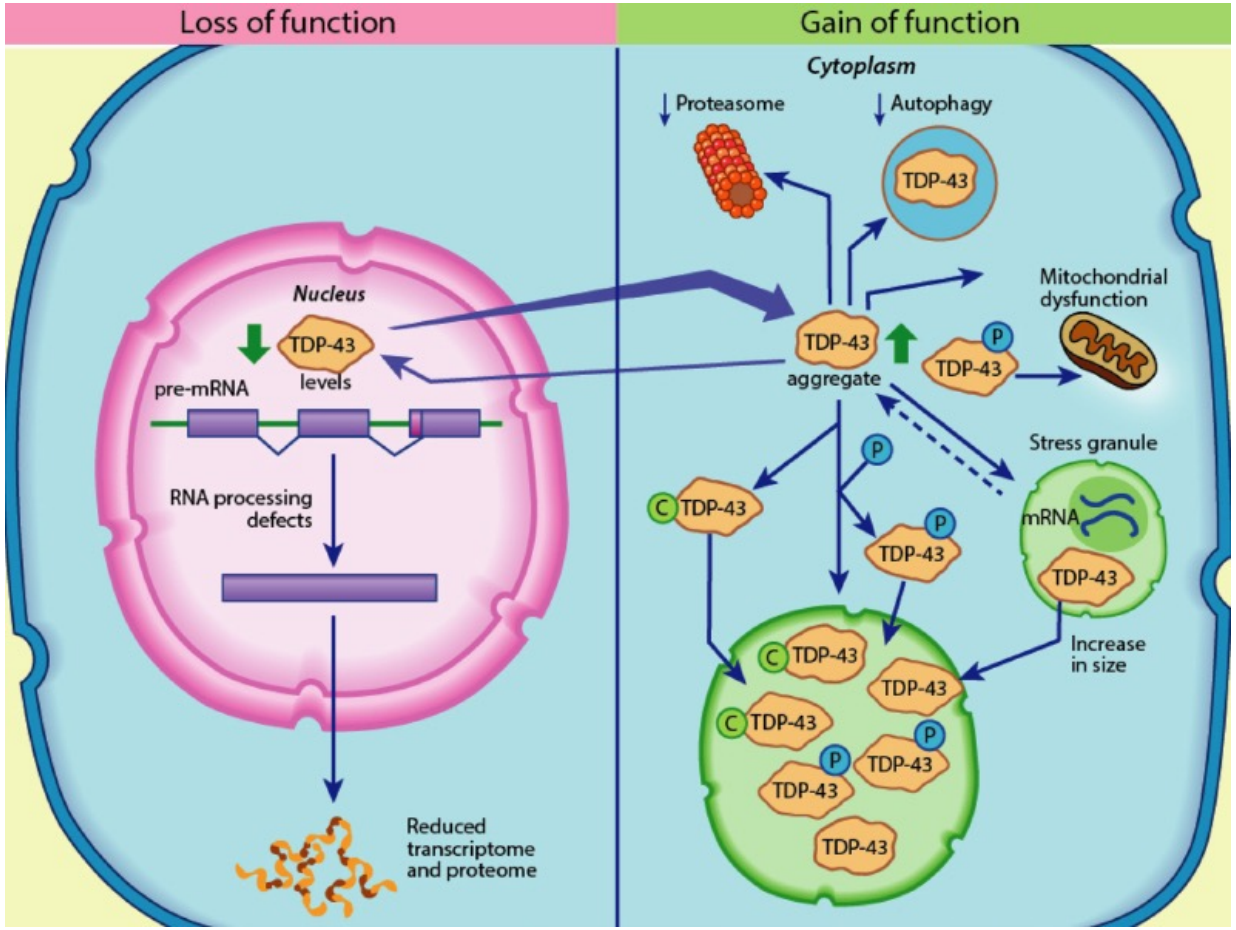

Figure from de Boer et al[1]

TDP-43 is essential to neuronal cell survival[1]

- TDP-43 is normally present in the nucleus of all cells and performs an essential role in RNA splicing, transport and stability
- Under stress conditions (e.g. oxidative stress) normal TDP-43 also forms protective stress granules in the cytoplasm

Misfolded TDP-43 gives rise to both loss of function and toxic gain of function[1]

- Loss of function: Under disease conditions, misfolding of TDP-43 causes formation of mislocalized cytoplasmic aggregates. Nuclear depletion leads to defective splicing and transport of mRNA.
- Toxic gain of function: Cytoplasmic aggregates of misfolded TDP-43 are toxic and interfere with physiologic stress granule function. They also induce misfolding of other proteins into pathogenic aggregates[2-4] - "TDP-43 Pathological Interactome"

Targeting of pathogenic TDP-43 requires stringent selectivity for the misfolded form of the protein to avoid safety concerns

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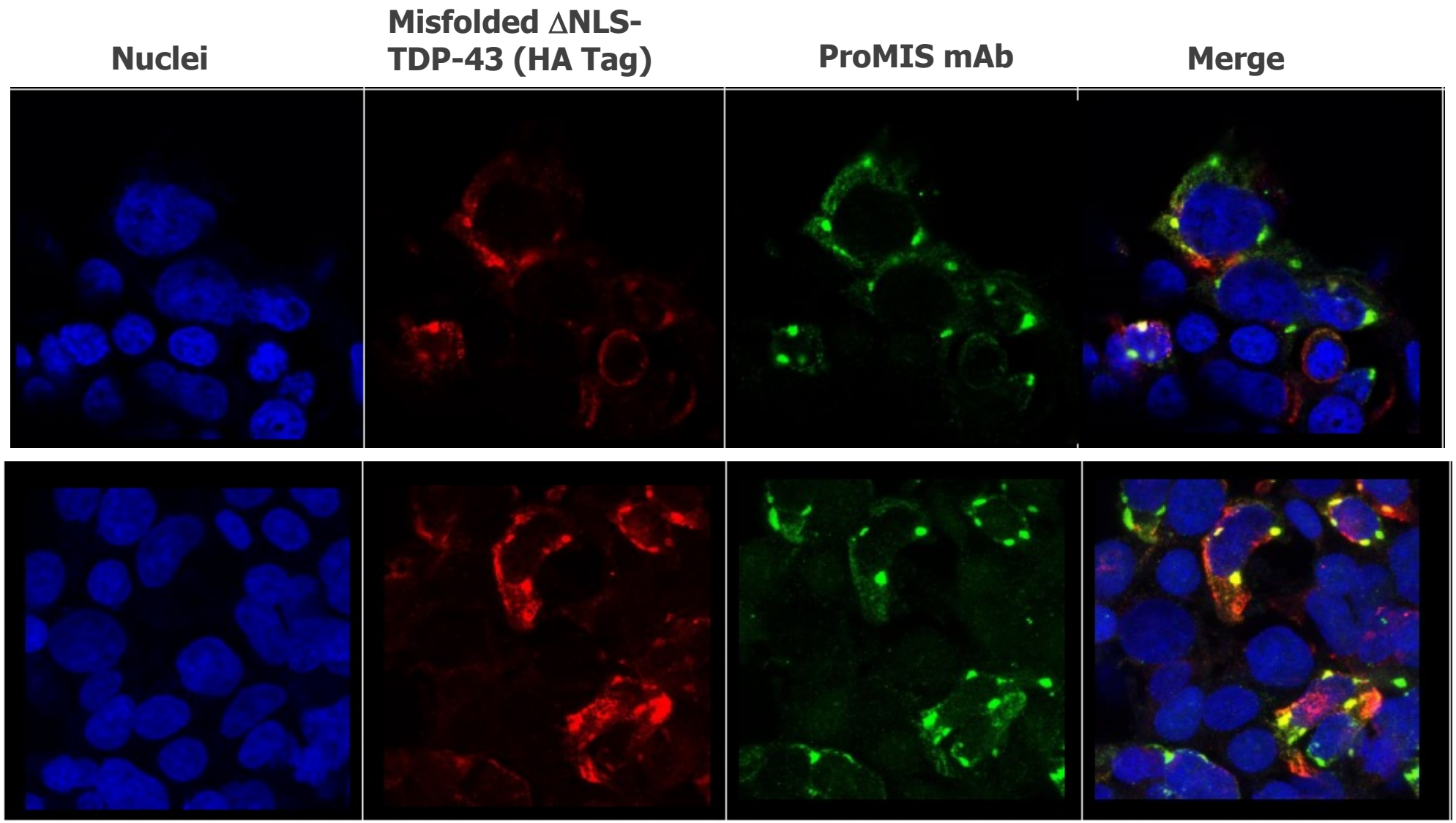
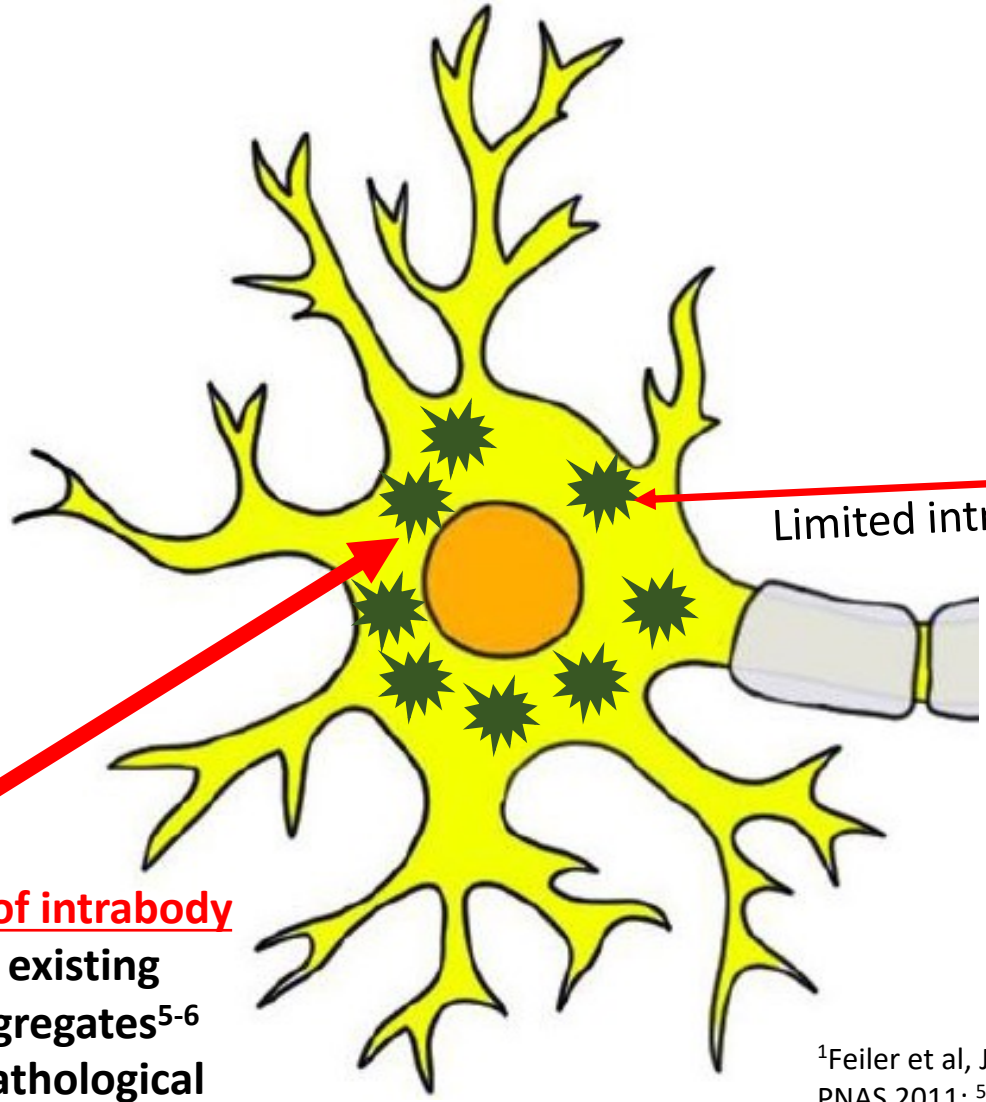
[1]de Boer, EMJ et al, 2020, J Neurol Neurosurg Psychiatry; [2]Pokrishevsky et al, 2016, Scientific Reports; [3]Chou et al, 2018, Nat Neurosci; [4]Endo et al, 2018, Biological Psych

ProMIS mAbs to misfolded TDP-43 show excellent selectivity: react with mislocalized, aggregated ΔNLS-TDP-43 but not nuclear wild type TDP-43



- HEK-293 cells transfected with ΔNLS TDP-43 lacking a functional nuclear localization signal
- Cells stained for HA tag (red) of overexpressed ΔNLS TDP-43 or with rabbit mAb to misfolded TDP-43 epitope at 2 μg/ml (green).
- Nuclei stained with DAPI (blue)
- Images analyzed by confocal microscopy (Z-stacks)

Antibody-Based Targeting of Pathogenic TDP-43 - Two approaches; extra cellular antibody, vectorized intrabody (gene therapy – with a partner)



Extracellular Antibody Delivery

Neutralization of cell-to-cell propagation

Limited intracellular uptake

Cell to cell transmission of pathogenic species via exosomes and free protein[1-4]

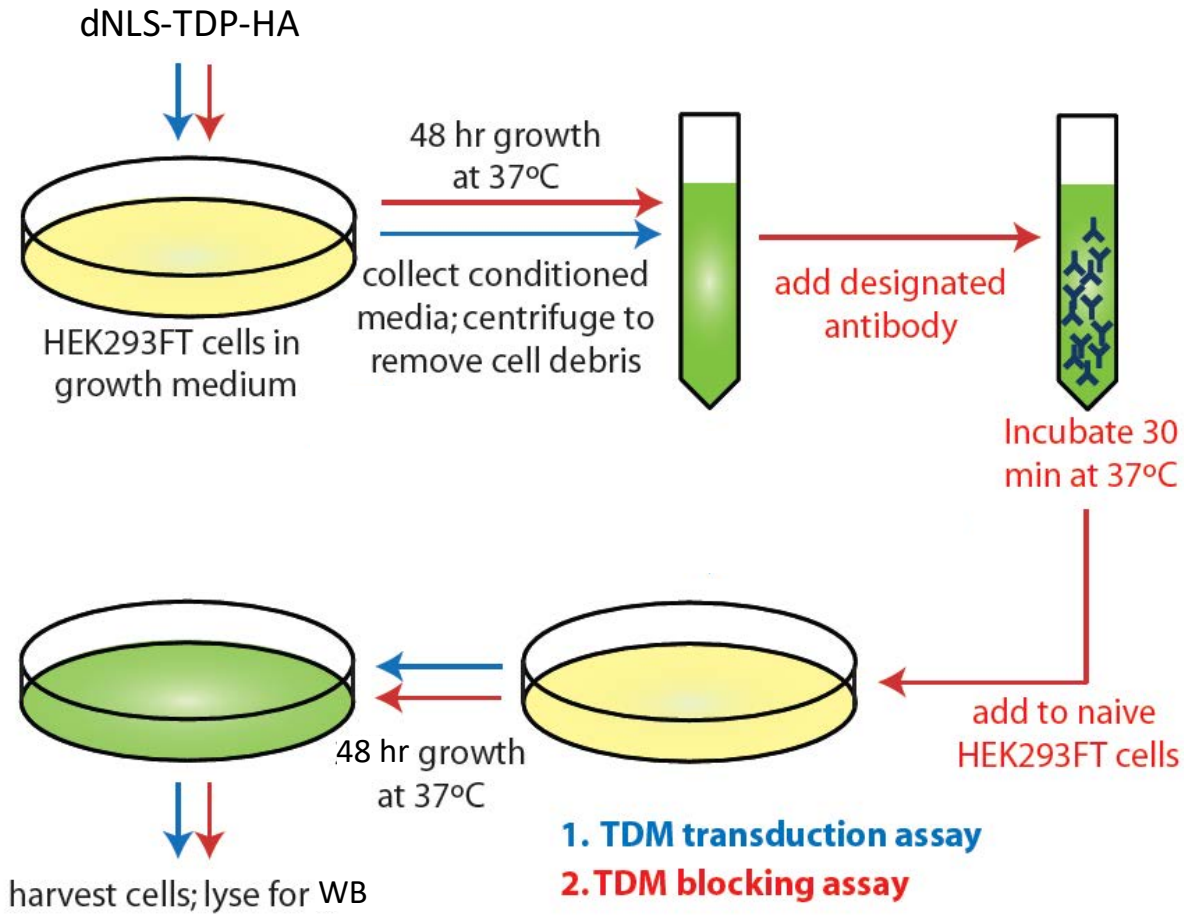
Vector delivery of intrabody

Clearance of existing pathogenic aggregates[5-6]

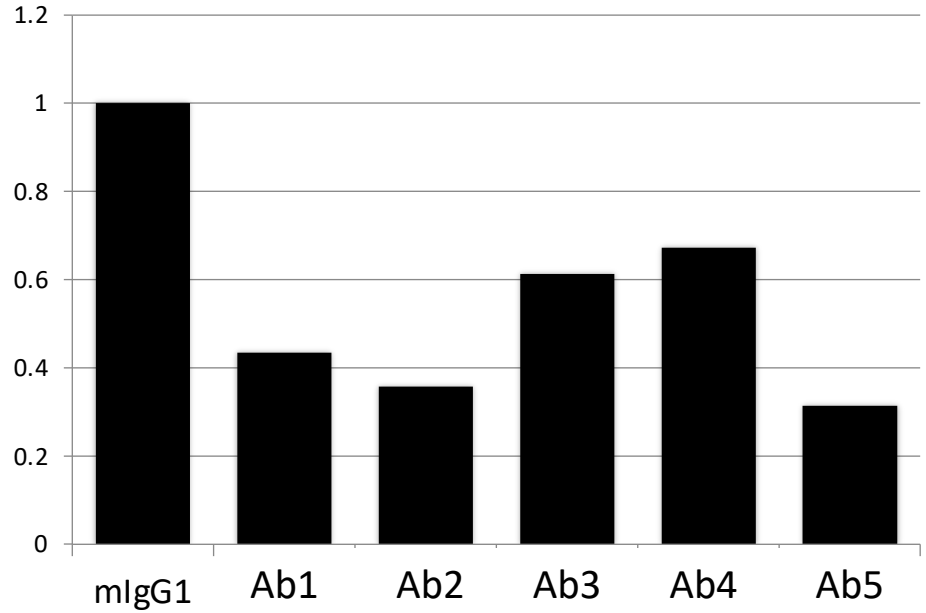
Inhibition of pathological interactome and further propagation[7-9]

[1]Feiler et al, JCB 2015: [2]Porta et al, Nature Commun 2018; [3]Munch et al, PNAS 2011; [4]Grad et al, PNAS 2011; [5]Tamaki et al, Scientific Reports 2018; [6]Ganashyam et al, Neurobiol of Dis 2018; [7] Pokrishevsky et al, PLoS-One 2012; [8]Pokrishevsky et al, Sci Rep 2016; [9]Chou et al Nat Neurosci 2018

Functional Assay: ProMIS *antibodies* inhibit cell-to-cell transmission of misfolding ΔNLS-TDP43 – suggests potential as extracellular antibody



HA-ΔNLS-TDP-43 transmission relative to mIgG1 control



ProMIS mAbs inhibit transmission of misfolding TDP-43 from the conditioned medium of donor HEK293 cells transfected with ΔNLS-TDP-43 to naïve recipient cells

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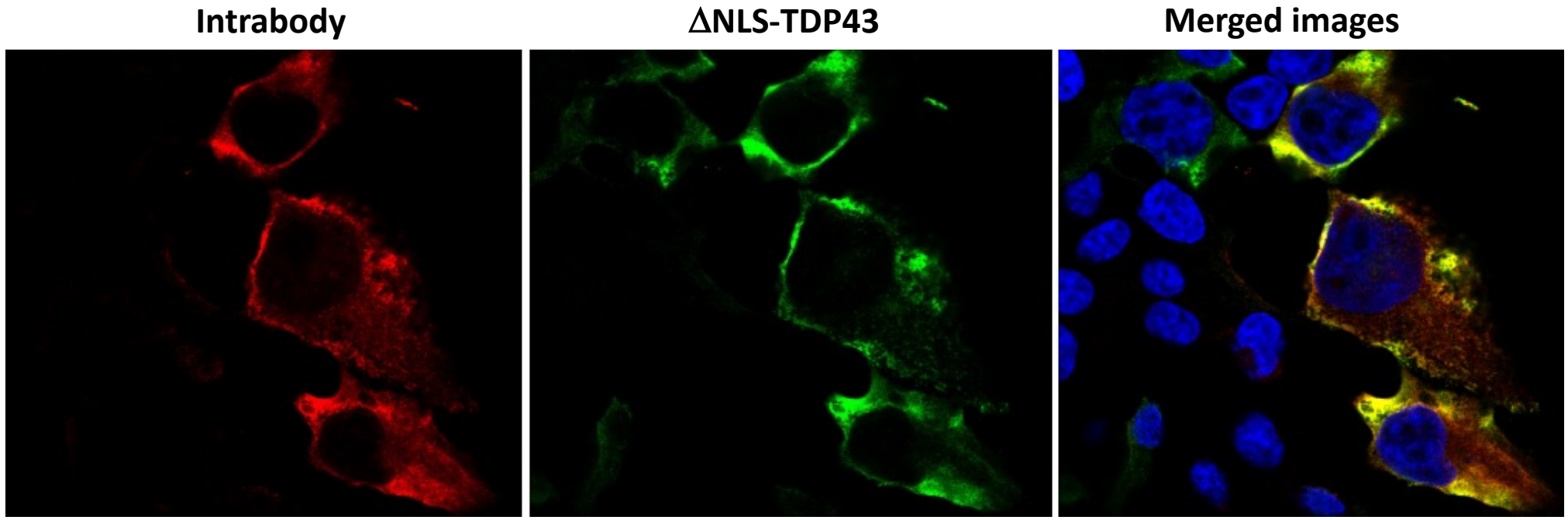
ProMIS *intrabodies* highly selective: interact only with cytoplasmic ΔNLS-TDP43 aggregates and not normal nuclear TDP43

HEK293 cells transfected with ΔNLS-TDP43 and single chain ProMIS intrabody



- Expression of ProMIS TDP43 intrabody is not toxic to cells
- Intrabody co-localizes with mislocalized, aggregated cytoplasmic ΔNLS-TDP43
- Intrabody does not interact with endogenous normal TDP43 in the nucleus

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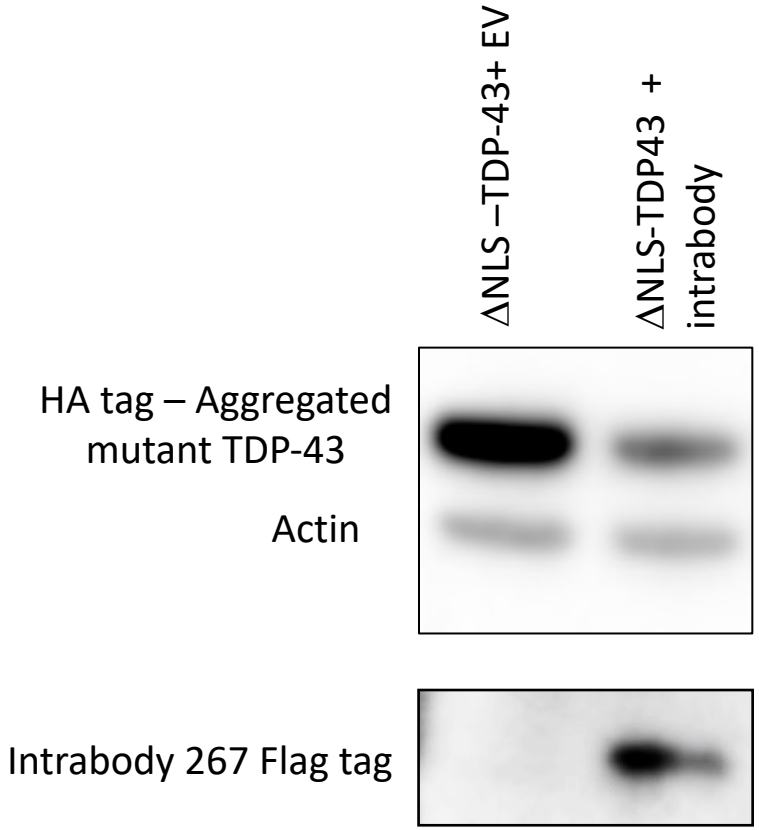
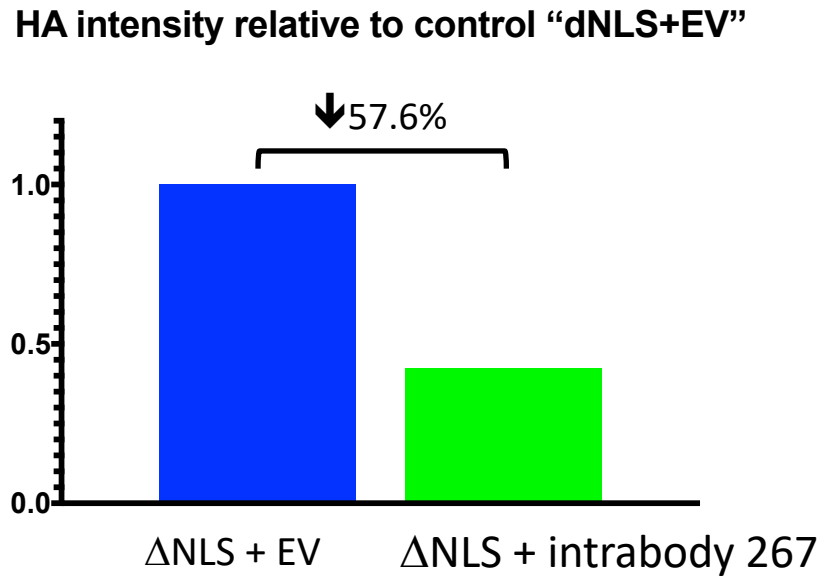
TDP-43 intrabody promotes clearance of misfolded TDP-43 aggregates inside the cell – functional benefit



Misfolded TDP43-selective intrabody with lysosomal targeting signal promotes degradation of TDP-43 aggregates without cellular toxicity



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EV = empty vector

ProMIS TDP-43 program: Summary

- Several antibody candidates show molecular species selectivity: binding to toxic mis-folded TDP-43, not normal physiologic TDP-43
- One candidate a lead priority for use as an extracellular antibody, shows functional benefit in blocking prion-like propagation in vitro
- Other candidates prioritized as "intrabodies", targeting intracellular mis-folded TDP-43; high affinity binding, selectivity for toxic TDP-43, no detrimental impact on cell viability, clearance of pathogenic aggregates from inside cells
- ProMIS unlikely to enter gene therapy space directly, pursuing partnering discussions with large "peer" organizations with gene therapy/vector capability
- TDP-43 toxic "interactome" with other ProMIS targets – RACK1, SOD1, ataxin2 – potential synergistic portfolio for ALS disease-modifying treatment
- All candidates preclinical stage, initiating humanization, next step is IND enabling work

Experienced Leadership Team

Name	Title	Years of Experience	Prior Experience
Gene Williams	Chairman & CEO	25+	▪ Former SVP at Genzyme, with senior roles integrating commercialization, drug development, and deal making ▪ Recently the CEO of Dart Therapeutics, an Orphan Disease drug development company ▪ Founder and director of Adheris, which became the largest company in the patient adherence/compliance area
Elliot Goldstein	President	25+	▪ Held positions as SVP of Strategic Product Development at SmithKline Beecham (now GSK) ▪ Chief Operating Officer and Chief Medical Officer of Maxygen ▪ Chief Operating Officer at DART Therapeutics
Neil Cashman	Chief Science Officer	25+	▪ Holds the Canada Research Chair in Neurodegeneration and Protein Misfolding Diseases, ▪ Serves as the Director of the University of British Columbia ALS Centre, ▪ Awarded the Jonas Salk Prize for biomedical research in 2000
David Wishart	Chief Physics Officer	25+	▪ Distinguished University Professor in the Departments of Biological Sciences and Computing Science at the University of Alberta ▪ Co-Director of The Metabolomics Innovation Centre ▪ Bristol-Myers Squibb Research Chair in Pharmaceutical Sciences 1995-2005 ▪ Fellow of the Royal Society of Canada
Dan Geffken	CFO	25+	▪ Founding Managing Director of Danforth Advisors ▪ Served as the Chief financial officer of Homology, Inc, GenePeeks, Inc., Transkaryotic Therapies, Inc., Cidara, Inc., Apellis, Inc. and Stealth BioTherapeutics, Inc.
Johanne Kaplan	Chief Development Officer	25+	▪ Former VP of Research at Genzyme ▪ Associate Immunopathologist at SmithKline Beecham where she established an Immunotoxicology program ▪ Her work has resulted in over 60 scientific publications and multiple patents
Gavin Malenfant	Chief Operating Officer	25+	▪ Head of Operations at Sarepta, in support of approval/ launch of EXONDYS 51 ▪ At Genzyme, head of operations for research and development ▪ At Genzyme, ead rare disease program management organization

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Scientific Advisory Board

Name	Years of Experience	Prior Experience	Affiliations
Sharon Cohen, MD	20+	▪ Medical Director & Principal Investigator of Toronto Memory Program ▪ FRCPC in neurology from Royal College of Physicians of Canada and a fellowship in Behavioural Neurology from the University of Toronto	Toronto Memory Program
Rudy Tanzi, PhD (Chairman)	20+	▪ Professor of Neurology at Harvard University, Vice Chair of Neurology, Director of Genetics & Aging Research Unit, Co-Director McCance Center for Brain Health at Mass General Hospital	Harvard University; Massachusetts General Hospital
Bill Mobley, MD, PhD	25+	▪ Dean for Neurosciences Initiatives, Distinguished Professor of Neurosciences, and Florence Riford Chair for Alzheimer Disease at the University of California, San Diego	UC San Diego School of Medicine
James Kupiec, MD	20+	▪ Former VP, Global Clinical Leader for Parkinson's disease, and Clinical Head of the Neuroscience Research Unit for Pfizer, Inc. ▪ Clinical focus on development of therapies for neurodegenerative disorders	Ciba; Pfizer; sanofi~synthelabo
C. Warren Olanow, MD	25+	▪ Previous Henry P & Georgette Goldschmidt Professor & Chairman, Department of Neurology at Mount Sinai School of Medicine, presently Professor Emeritus Department of Neurology & Department of Neuroscience, CEO of CLINTREX	MSSM; Mount Sinai School of Medicine
Andre Strydom, MD, PhD	25+	▪ Professor Institute of Psychiatry, Psychology and Neuroscience at King's College London ▪ Honorary Consultant psychiatrist, South London and the Maudsley NHS Foundation Trust	King's College London; NHS South London and Maudsley NHS Foundation Trust
Michelle Hastings, PhD	20+	▪ Professor and Director, Center for Genetic Diseases, Rosalind Franklin University of Medicine and Science ▪ Faculty Member at the Chicago Medical School	Rosalind Franklin University of Medicine and Science