AMPORIN Pharmaceuticals

Repairing Membranes to Rescue Mitochondria in Degenerative Diseases

Seed investment opportunity

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Problem: The Social and Economic Cost and Unmet Need of Degenerative Diseases is Huge and Growing Rapidly with the Aging Population

- Alzheimer's
- Parkinson's
- Diabetes II
- ALS
- Huntington's
- >50 rare diseases

- 540 million people
- 3.6 million deaths
- $3 TRILLION costs
- $100B drug market
- 5% annual growth
- **No effective cures**

Sources: www.who.int, www.cdc.gov, www.healthdata.org, www.alz.org, www.parkinson.org, www.idf.org

Many Degenerative Diseases Are Now Associated With Protein Misfolding; Existing Treatments Do Not Stop Disease Progression

Protein misfolding diseases	Protein/peptide(s)	Approved symptomatic treatments	Approved disease-modifying treatments
Alzheimer's disease (AD) Other β-amyloid diseases: CAA	β-amyloid (Aβ) Tau protein (Tau)	Cholinesterase inhibitors (donepezil, rivastigmine, galantamine), memantine	**Modest slowing of disease, no reversal, significant safety risks (ARIA):** Leqembi (lecanemab), Kisunla (donanemab)
Parkinson's disease (PD) Synucleinopathies: PDD, DLB, MSA	α-Synuclein (αSyn)	Levodopa/carbidopa, dopamine agonists, COMT inhibitors, MAO-B inhibitors, anticholinergics	None
Type II diabetes (T2D)	Amylin (IAPP)	Metformin, insulin analogs, thiazolidinediones, sulfonylureas, SGLT2 inhibitors, GLP-1 agonists, meglitinides, DPP-4 inhibitors, pramlintide	None that prevent progressive β-cell loss
Amyotrophic lateral sclerosis (ALS) Other TDP-43 diseases: FTLD, LATE	SOD1, TDP-43	Off-label: Baclofen, tizanidine	**Modest slowing of disease, no reversal:** Riluzole, edaravone, Qalsody (tofersen) for SOD1-ALS only (~2% of ALS cases)
Frontotemporal Dementia (FTD) Other tauopathies: PSP, CBD, CTE	Tau protein (Tau)	Off-label: SSRIs, amantadine, trazodone	None
Huntington's disease (HD) Other CAG repeat diseases: SBMA, Spinocerebellar ataxias (SCA1-3, 6, 7)	Huntingtin (mHtt) Other PolyQ proteins	Tetrabenazine/deutetrabenazine, valbenazine Off-label: antidepressants, benzodiazepines	None
TTR amyloidosis (ATTR) Other TTR diseases: FAP, FAC	Transthyretin (TTR)	Off-label: Diflunisal	**Significant slowing, no reversal:** Vyndaqel (tafamidis), Attruby (acoramidis), Onpattro (patisiran), Amvuttra (vutrisiran), Tegsedi (inotersen), Wainzua (eplontersen)
Creutzfeldt-Jakob disease (CJD) Other prion diseases: vCJD, GSS, FFI	Prions (PrP)	Off-label: Sedatives, antidepressants	None
> 40 other (rare) diseases Dialysis-related amyloidosis (DRA), AL amyloidosis, AA amyloidosis, etc.	Various	Various (off-label only)	None

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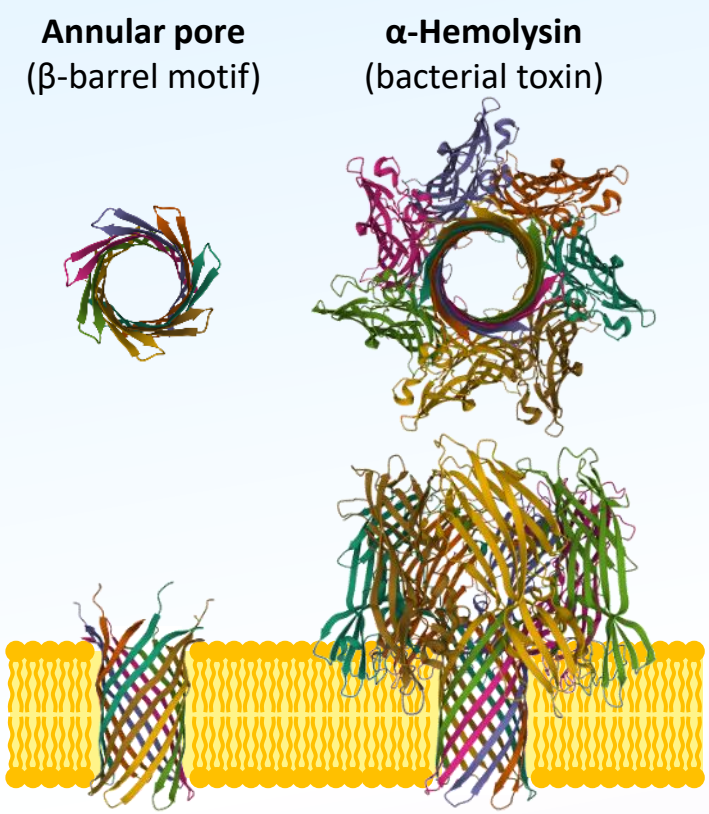
Amporin Is Uniquely Targeting Membrane Pores to Enable Rapid Functional Recovery in These Diseases, Starting With Parkinson's Disease (PD)

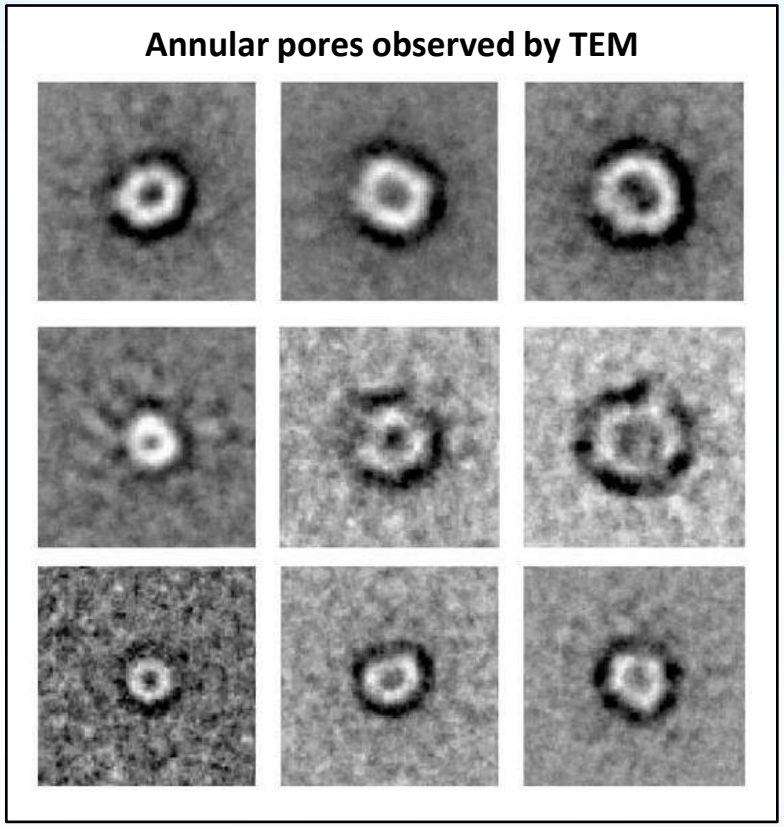
Membrane perforation as a common pathogenic mechanism

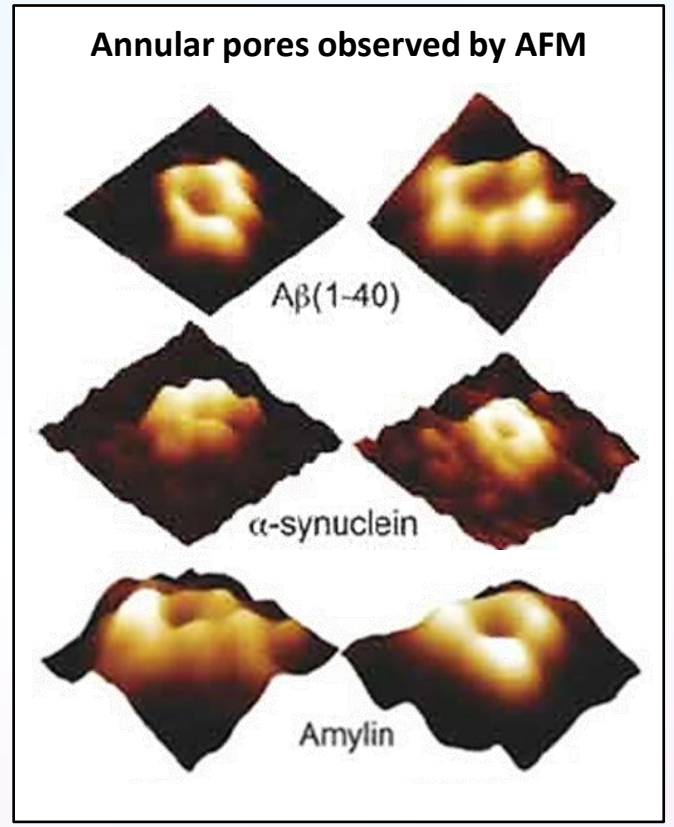
- Misfolded proteins form **toxic pores** that perforate different membranes in different cells and tissues to cause different diseases
- Membrane perforation allows **uncontrolled calcium influx**, causing cellular stress and initial disruption of cellular function and homeostasis
- Sustained calcium influx overloads and damages **mitochondria** with chronic oxidative stress, causing mitochondrial failure, energy depletion, and loss of homeostasis, leading to progressive cell death and disease

Amporin's first-in-class oral drug platform uniquely targets the toxic pores to restore membrane integrity, mitochondrial function and cellular homeostasis, with initial focus on PD and early out-licensing of AD and Type II Diabetes

Misfolded Protein Oligomers Form Annular Pore-like Structures in Cell and Mitochondrial Membranes, Similar to Bacterial Pore-Forming Toxins







1. Nutini A. Amyloid oligomers and their membrane toxicity - A perspective study. Prog Biophys Mol Biol. 2024 Jan 9: S0079-6107(24)00002-6.
2. Viles J.H. Imaging Amyloid-β Membrane Interactions: Ion-Channel Pores and Lipid-Bilayer Permeability in Alzheimer's Disease. Angew Chem Int Ed Engl. 2023; 62(25): e202215785.
3. Budvytyte R., et al. The interactions of amyloid β aggregates with phospholipid membranes and the implications for neurodegeneration. Biochem Soc Trans. 2023; 51(1): 147-159.
4. Diociaiuti M., et al. Amyloid prefibrillar oligomers: The surprising commonalities in their structure and activity. Int J Mol Sci. 2021 Jun 16; 22(12): 6435.
5. Gonzalez-Garcia M., et al. Membrane interactions and toxicity by misfolded protein oligomers. Front Cell Dev Biol. 2021 Mar 11; 9: 642623.
6. Vassallo N. Amyloid pores in mitochondrial membranes. Neural Regen Res. 2021 Nov; 16(11): 2225-2226.

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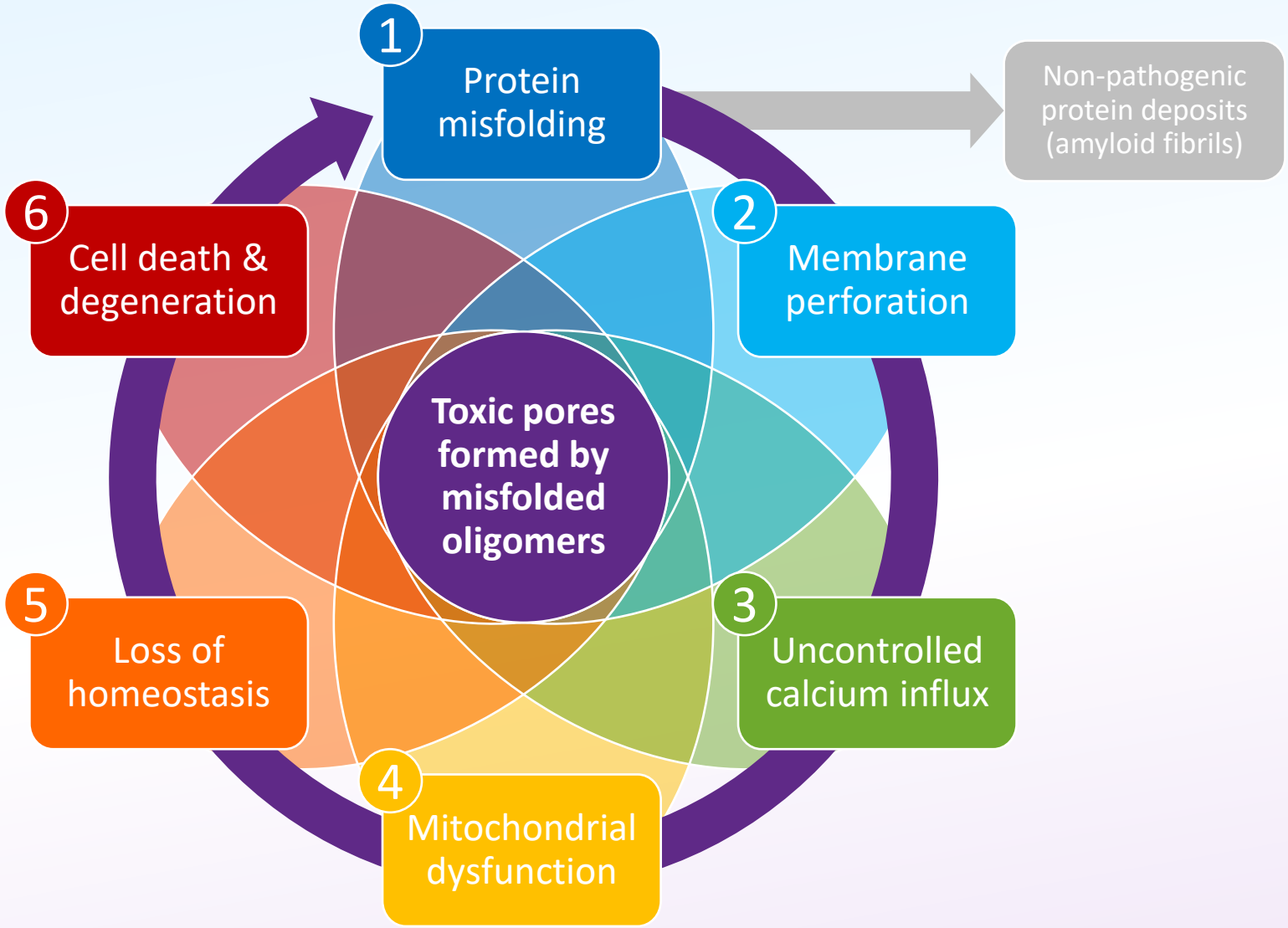
Misfolded Proteins Form Toxic Pores That Perforate Cell and Mitochondrial Membranes, Causing Mitochondrial Dysfunction, Cell Death and Disease



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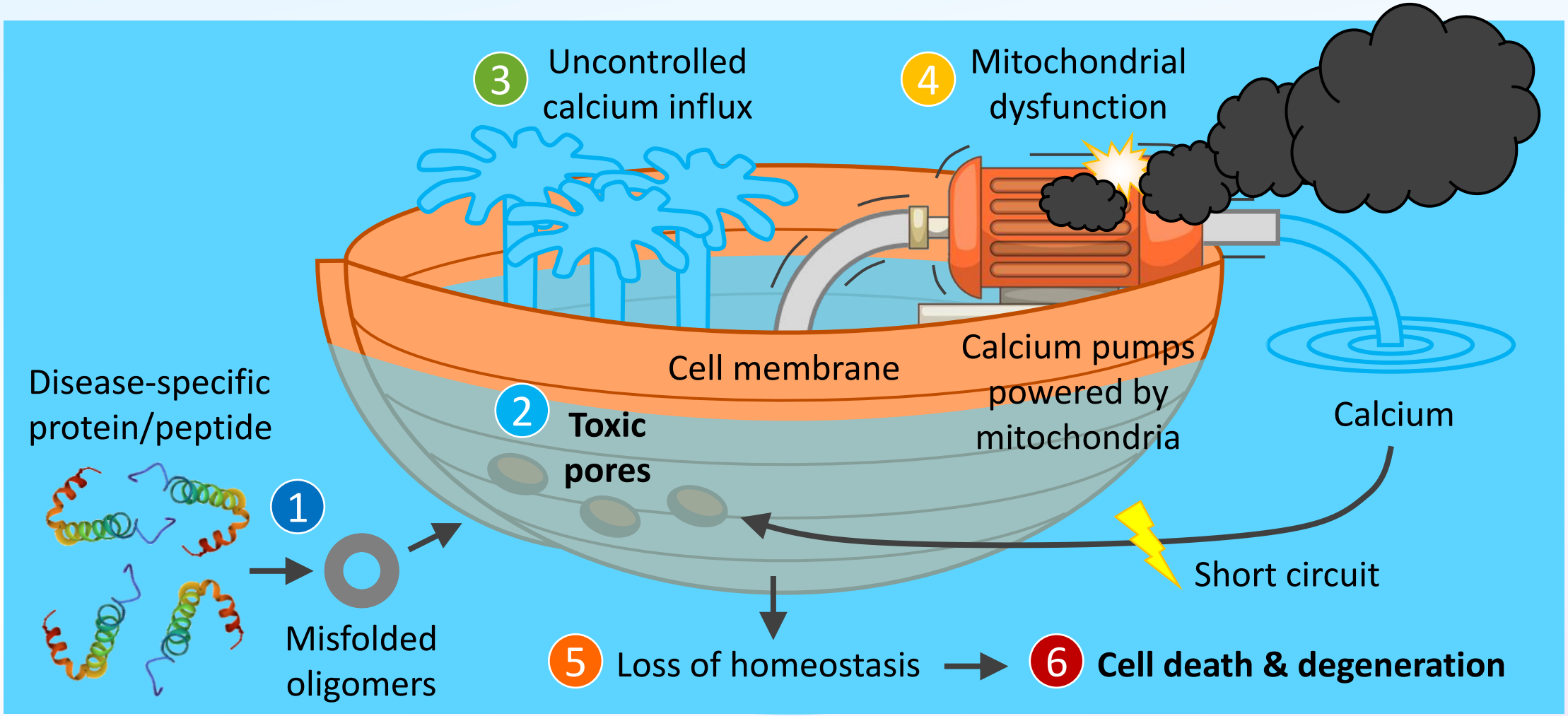

Misfolded Proteins Form Toxic Pores That Perforate Cell and Mitochondrial Membranes, Causing Mitochondrial Dysfunction, Cell Death and Disease

Pathogenic step		Mechanistic details
1	**Protein misfolding**	• Disease-associated proteins misfold and self-associate into soluble oligomers • Soluble oligomers accumulate before large insoluble fibrils and plaques form • Examples include Aβ, tau, αSyn, IAPP, SOD1, TDP-43, mHtt, TTR, PrP, etc.
2	**Membrane perforation**	• Soluble oligomers associate with cell and/or mitochondrial membranes • Membrane-bound oligomers rearrange to form toxic annular pore-like structures • **Toxic pores perforate membranes and increase ion conductance**
3	**Uncontrolled calcium influx**	• Toxic pores allow uncontrolled calcium influx from the extracellular space • Calcium influx causes initial reversible disruption of cellular function/homeostasis • Excess calcium accumulates in mitochondria via toxic pores and/or the MCU
4	**Mitochondrial dysfunction**	• Mitochondrial calcium overload disrupts the ETC and increases ROS production • ETC disruption and increased ROS production impair oxidative phosphorylation • Impaired oxidative phosphorylation reduces ATP synthesis, causing ATP depletion
5	**Loss of homeostasis**	• ATP depletion impairs calcium export and causes loss of ionic homeostasis • Sustained calcium influx and oxidative stress trigger persistent mPTP opening • Persistent mPTP opening causes ΔΨm collapse and mitochondrial failure
6	**Cell death & degeneration**	• ΔΨm collapse and mitochondrial failure cause mitochondrial swelling and rupture • Mitochondrial rupture causes progressive cell death by apoptosis or necrosis • Progressive cell death causes DAMP release, inflammation, and degeneration

Abbreviations: Aβ = Amyloid-β; αSyn = α-Synuclein; IAPP = Islet Amyloid Polypeptide; SOD1 = Superoxide Dismutase 1; TDP-43 = TAR DNA-binding Protein 43; mHtt = Mutant Huntingtin; TTR = Transthyretin; PrP = Prion Protein; MCU = Mitochondrial Calcium Uniporter; ETC = Electron Transport Chain; ROS = Reactive Oxygen Species; ATP = Adenosine Triphosphate; mPTP = Mitochondrial Permeability Transition Pore; ΔΨm = Mitochondrial Membrane Potential; DAMP = Damage-Associated Molecular Patterns

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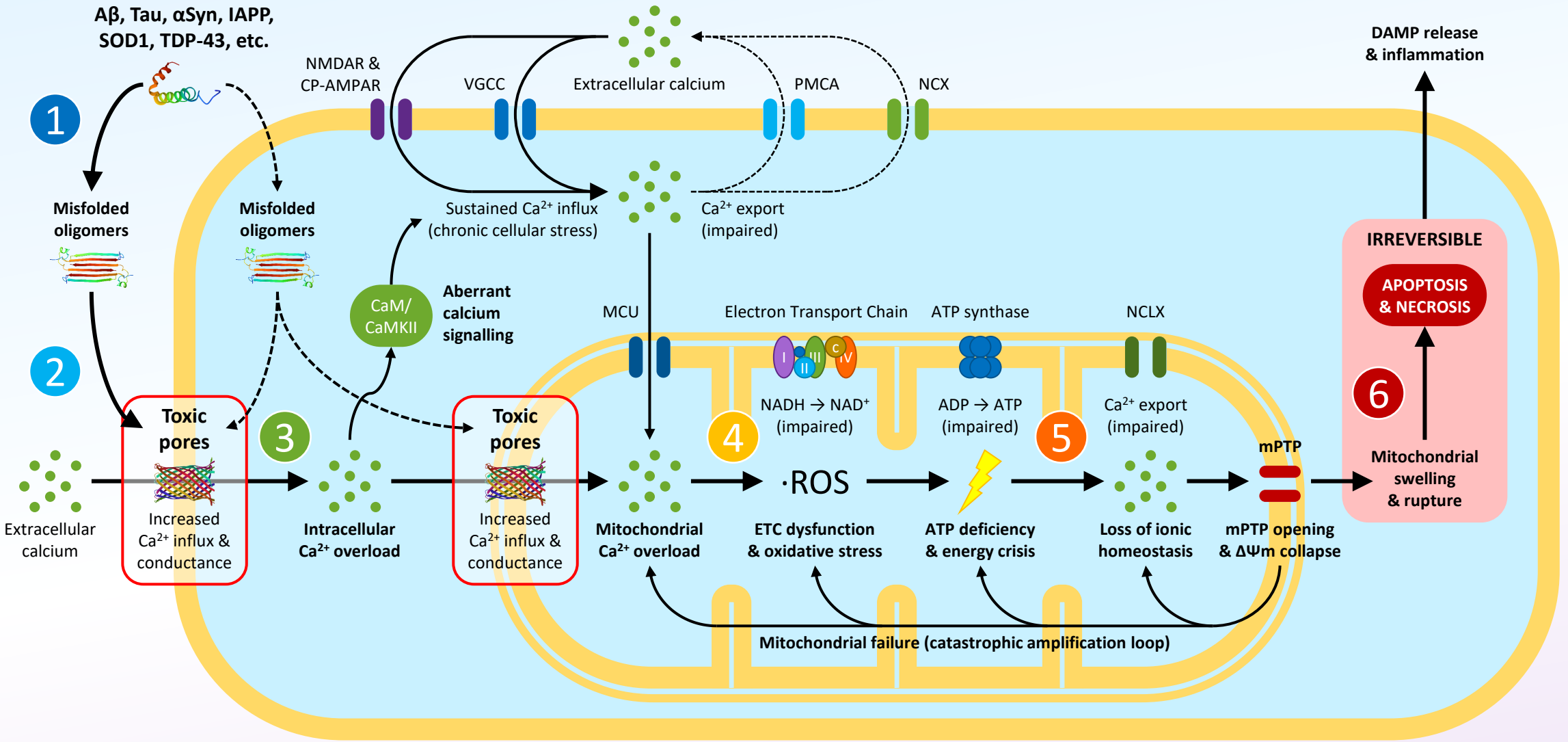
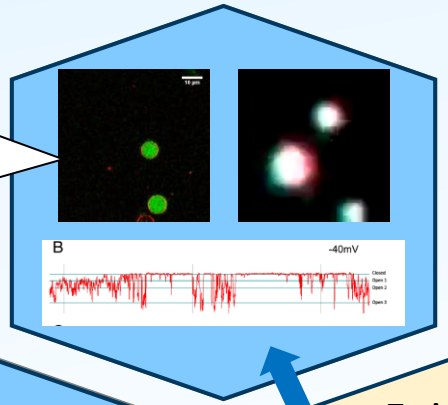
Misfolded Proteins Form Toxic Pores That Perforate Cell and Mitochondrial Membranes, Causing Mitochondrial Dysfunction, Cell Death and Disease

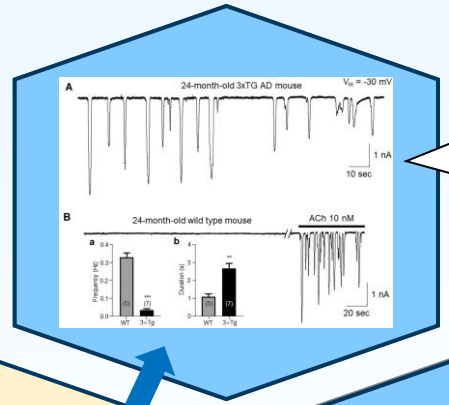


Over 400 Scientific Papers (1993-2026) Now Support the Key Pathogenic Role of Toxic Pores in Degenerative Diseases

1

Oligomers permeabilize liposomes, lipid vesicles, and planar lipid bilayers
- Van Rooijen *et al.,* 2010
- Brøchner *et al.,* 2025
- Ghio *et al.,* 2019



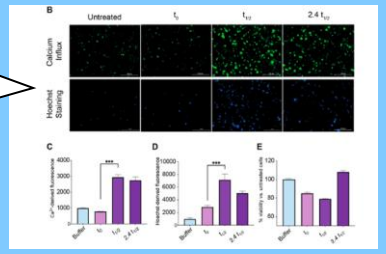


4

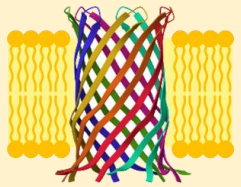
Annular pores/channels are observed in animals and *ex vivo* brain tissues
- Inoue *et al.,* 2008
- Lasagna-Reeves *et al.,* 2011
- Li *et al.,* 2023

2

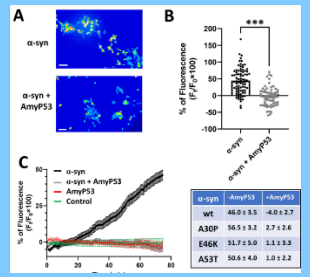
Oligomers permeabilize cell membranes, causing calcium influx & toxicity
- Camilleri *et al.,* 2013
- Ghio *et al.,* 2019
- Ludtmann *et al.,* 2018



Toxic pores



Aβ, Tau, αSyn, IAPP, TDP-43, etc.

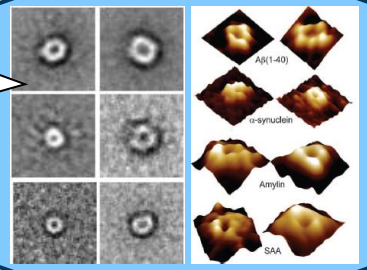


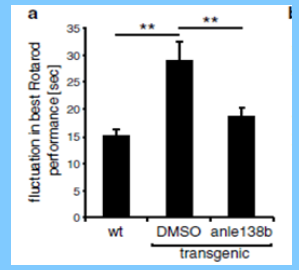
5

Oligomer/pore inhibitors protect and restore cell viability *in vitro*
- Saravan *et al.,* 2019
- Wrasidlo *et al.,* 2016
- Fantini *et al.,* 2025

3

Annular pores/channels are observed by EM/AFM and in MD simulations
- Lashuel *et al.,* 2002
- Quist *et al.,* 2005
- Sepehri *et al.,* 2023





6

Oligomer/pore inhibitors protect and rescue animal models of disease
- Wagner *et al.,* 2013
- Price *et al.,* 2018
- Fantini *et al.,* 2025

Many Review Papers Support the Key Pathogenic Role of Pores in Disease

Are amyloid diseases caused by protein aggregates that mimic bacterial pore-forming toxins?

Lashuel HA, Lansbury PT Jr. Q Rev Biophys. 2006 May;39(2):167-201.

Membrane pores in the pathogenesis of neurodegenerative disease

Kagan BL. Prog Mol Biol Transl Sci. 2012;107:295-325.

Membrane permeabilization: a common mechanism in protein-misfolding diseases

Lashuel HA. Sci Aging Knowledge Environ. 2005 Sep 21;2005(38):pe28.

The channel hypothesis of Alzheimer's disease: current status

Kagan BL, Hirakura Y, Azimov R, Azimova R, Lin MC. Peptides. 2002 Jul;23(7):1311-5.

Molecular mechanism of neurodegeneration induced by Alzheimer's beta-amyloid protein: channel formation and disruption of calcium homeostasis

Kawahara M, Kuroda Y. Brain Res Bull. 2000 Nov 1;53(4):389-97.

Interaction of Alzheimer's β-amyloid peptides with cholesterol: mechanistic insights into amyloid pore formation

Di Scala C, Chahinian H, Yahi N, et al. Biochemistry. 2014 Jul 22;53(28):4489-502.

Poration of mitochondrial membranes by amyloidogenic peptides and other biological toxins

Vassallo N. J Neurochem. 2025 Jan;169(1):e16213.

Amyloid-beta Alzheimer targets - protein processing, lipid rafts, and amyloid-beta pores

Arbor SC, LaFontaine M, Cumbay M. Yale J Biol Med. 2016 Mar 24;89(1):5-21.

Amyloid peptide channels

Kagan BL, Azimov R, Azimova R. J Membr Biol. 2004 Nov;202(1):1-10.

Amyloid beta ion channel: 3D structure and relevance to amyloid channel paradigm

Lal R, Lin H, Quist AP. Biochim Biophys Acta. 2007 Aug;1768(8):1966-75.

Imaging amyloid-β membrane interactions: ion-channel pores and lipid-bilayer permeability in Alzheimer's disease

Viles JH. Angew Chem Int Ed Engl. 2023 Jun 19;62(25):e202215785.

The interactions of amyloid β aggregates with phospholipid membranes and the implications for neurodegeneration

Budvytyte R, Valincius G. Biochem Soc Trans. 2023 Feb 27;51(1):147-159.

Membrane interactions and toxicity by misfolded protein oligomers

Gonzalez-Garcia M, Fusco G, De Simone A. Front Cell Dev Biol. 2021 Mar 11;9:642623.

Interactions between misfolded protein oligomers and membranes: A central topic in neurodegenerative diseases?

Andreasen M, Lorenzen N, Otzen D. Biochim Biophys Acta. 2015 Sep;1848(9):1897-907.

Membrane permeabilization by Islet Amyloid Polypeptide

Engel MF. Chem Phys Lipids. 2009 Jul;160(1):1-10.

Interactions of alpha-synuclein with membranes in Parkinson's disease: Mechanisms and therapeutic strategies

Li B, Dettmer U. Neurobiol Dis. 2024 Oct 15;201:106646.

Amyloid oligomers and their membrane toxicity - A perspective study

Nutini A. Prog Biophys Mol Biol. 2024 Mar;187:9-20.

Neurotoxicity of β-amyloid protein: oligomerization, channel formation and calcium dyshomeostasis

Kawahara M. Curr Pharm Des. 2010;16(25):2779-89.

Abeta ion channels. Prospects for treating Alzheimer's disease with Abeta channel blockers

Arispe N, Diaz JC, Simakova O. Biochim Biophys Acta. 2007 Aug;1768(8):1952-65.

The amyloid beta ion channel hypothesis of Alzheimer's disease

Shirwany NA, Payette D, Xie J, Guo Q. Neuropsychiatr Dis Treat. 2007;3(5):597-612.

Interaction of Alzheimer's β-amyloid peptides with cholesterol: mechanistic insights into amyloid pore formation

Di Scala C, Chahinian H, Yahi N, et al. Biochemistry. 2014 Jul 22;53(28):4489-502.

Amyloid peptide pores and the beta sheet conformation

Kagan BL, Thundimadathil J. Adv Exp Med Biol. 2010;677:150-67.

Ion channel formation and membrane pathologies of misfolded proteins: the role of dangerous unchaperoned molecules

Kourie JI, Henry CL. Clin Exp Pharmacol Physiol. 2002 Sep;29(9):741-53.

Membrane interactions of oligomeric alpha-synuclein: potential role in Parkinson's disease

van Rooijen BD, Claessens MM, Subramaniam V. Curr Protein Pept Sci. 2010;11(5):334-42.

Heterogeneous amyloid-formed ion channels as a common cytotoxic mechanism: implications for therapeutic strategies

Kourie JI, Culverson AL, Farrelly PV, et al. Cell Biochem Biophys. 2002;36(2-3):191-207.

Ion channel hypothesis for Alzheimer amyloid peptide neurotoxicity

Pollard HB, Arispe N, Rojas E. Cell Mol Neurobiol. 1995 Oct;15(5):513-26.

Beta-Amyloid Ca(2+)-channel hypothesis for neuronal death in Alzheimer disease

Arispe N, Pollard HB, Rojas E. Mol Cell Biochem. 1994 Nov 23;140(2):119-25.

Why Have Toxic Pores in Membranes Been Overlooked Until Now?

Basic reason	Detailed explanation	Typical push-backs	Amporin's counter-argument	Amporin
Lack of awareness	Scientists work in disease biology silos with > 100K papers published per year, but < 20 p.a. (0.02%) focus on pores	*"I've never heard of these toxic pores before, so they can't be very important"*	The pore hypothesis is already supported by hundreds of papers, but key papers have been widely scattered and buried in the noise until now (needle in a haystack)	✓ **Resolved**
Challenging target	Toxic pores are dynamic, metastable, disordered, heterogeneous, and buried in membranes, so difficult to target	*"Targeting pores will be difficult because they are not well defined"*	We have already identified potent pore inhibitors that are effective at just 1 nM, while we already see strong SAR for further optimization	✓ **Resolved**
Efficacy concerns	Acute disease reversal does not seem possible as cells can't be revived once they are dead	*"Preliminary data showing acute disease reversal are too good to be true"*	Acute disease reversal can be explained and expected since affected cells are stressed and dysfunctional, but still alive and recoverable in the early stages of disease	✓ **Resolved**
Selectivity concerns	Toxic pores act like non-specific ion channels, so targeting them may also block natural calcium channels	*"Targeting pores may also block natural ion channels"*	Toxic pores have a unique molecular architecture that is very different from natural gated ion channels, while our inhibitors show no acute toxicity in cell assays	✓ **Resolved**
Cost & time concerns	Targeting neurodegenerative diseases has required big, long, expensive trials with a high failure rate	*"Targeting these diseases is too risky, and it will take years to get any read-out"*	By targeting acute disease reversal, we can get a fast biomarker-based read-out in smaller, shorter, cheaper clinical trials, just like testing symptomatic treatments	✓ **Resolved**
Risk avoidance	There are still many unknowns about toxic pores and the potential effects of targeting them in disease	*"We're still not sure about targeting pores, so we will wait for more data"*	The pore hypothesis is already well supported, while the remaining unknowns can be quickly, cheaply, and easily addressed to reduce risk and create value	✓ **Resolved**
Herd mentality	No other company or research group has tried to target pores, raising doubts that the strategy is viable	*"If targeting pores is such a good idea, why has nobody else tried it?"*	Every innovation requires somebody with the courage to lead, otherwise everyone is just waiting for others to lead: No risk, no return!	✓ **Resolved**

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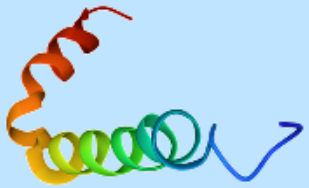
Amporin is Uniquely Targeting Toxic Pores Formed by Misfolded Proteins in Cell and Mitochondrial Membranes

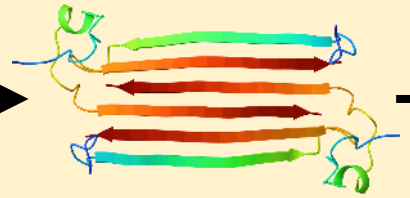
Disease-related gene expression

Disease-specific protein/peptide

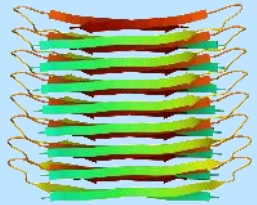


Aβ, Tau, αSyn, IAPP, SOD1, TDP43, mHtt, TTR, PrP, etc.

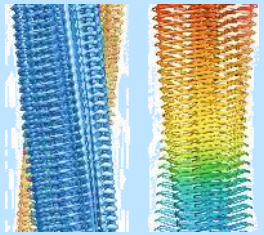
Misfolded oligomers (soluble and toxic)



Protofibrils (soluble, non-toxic)



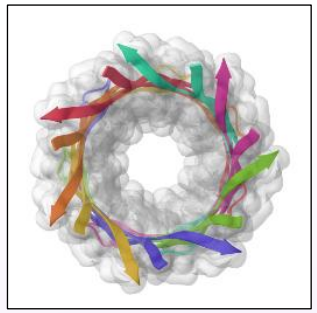
Amyloid fibrils (insoluble, non-toxic)



Upstream targets (chronic prevention)

Downstream targets (non-pathogenic)

Amporin targets (acute treatments)



Toxic annular pores (dynamic, metastable)

Acute effects (reversible)

- Calcium influx
- Aberrant signalling
- Loss of function
- Mitochon. overload
- Oxidative stress
- ATP depletion

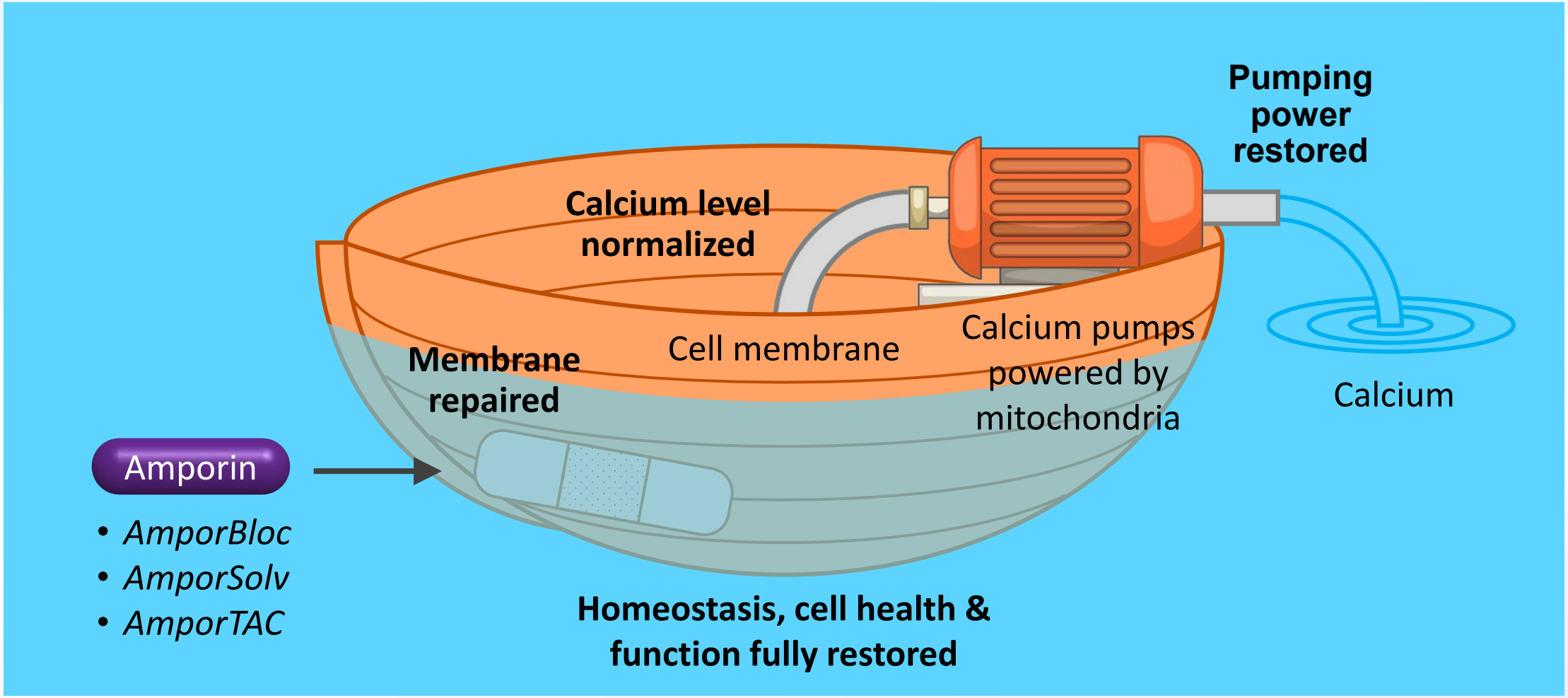
Chronic effects (irreversible)

- mPTP opening
- Mitochon. collapse
- Loss of homeostasis
- Cell death
- Degeneration
- Tissue atrophy

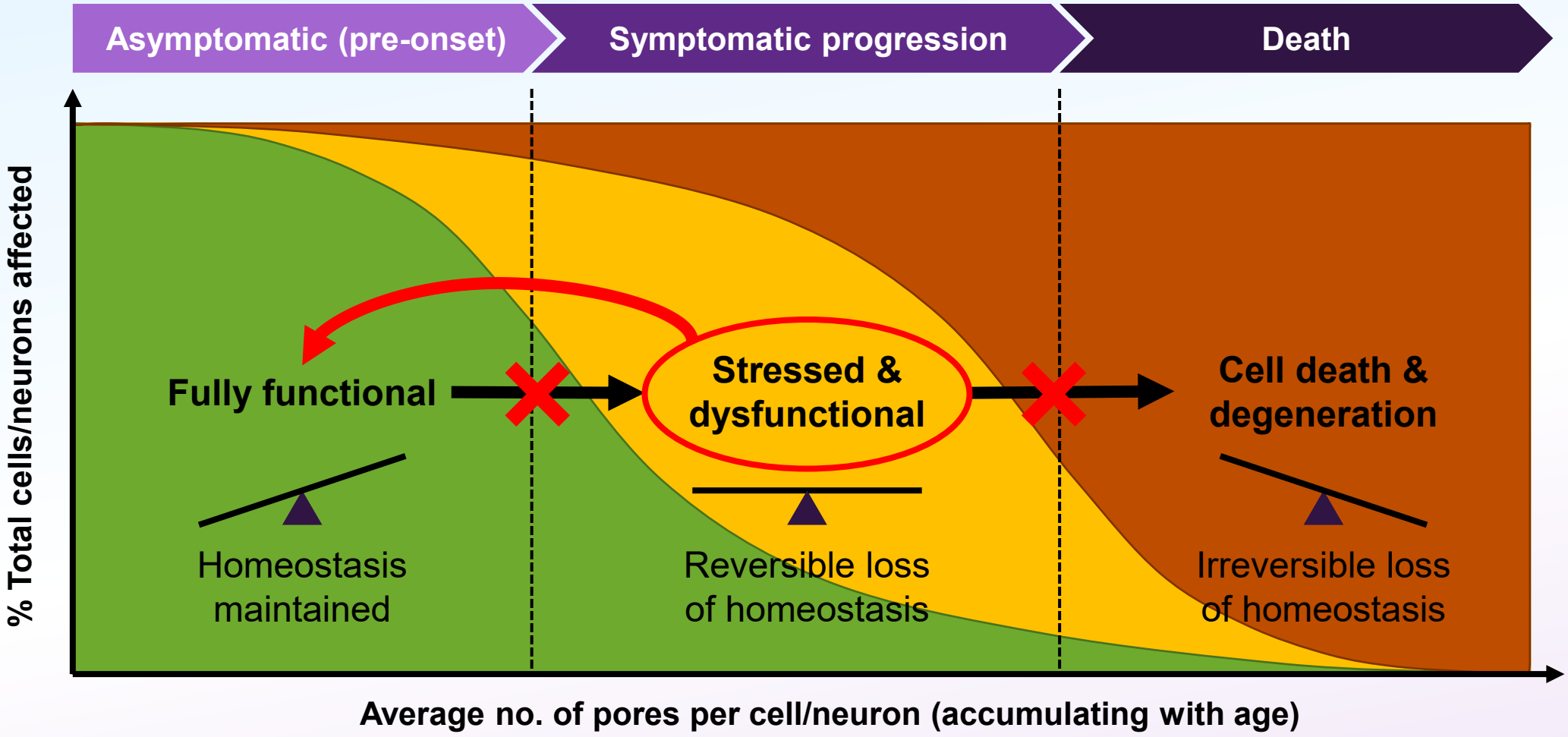
Amporin Aims to Stop the Death Spiral by Blocking Toxic Pores to Restore Membrane Integrity, Mitochondrial Function and Cellular Homeostasis

Amporin aims to restore membrane integrity and stop uncontrolled calcium influx, allowing surviving neurons to restore mitochondrial function and cellular homeostasis

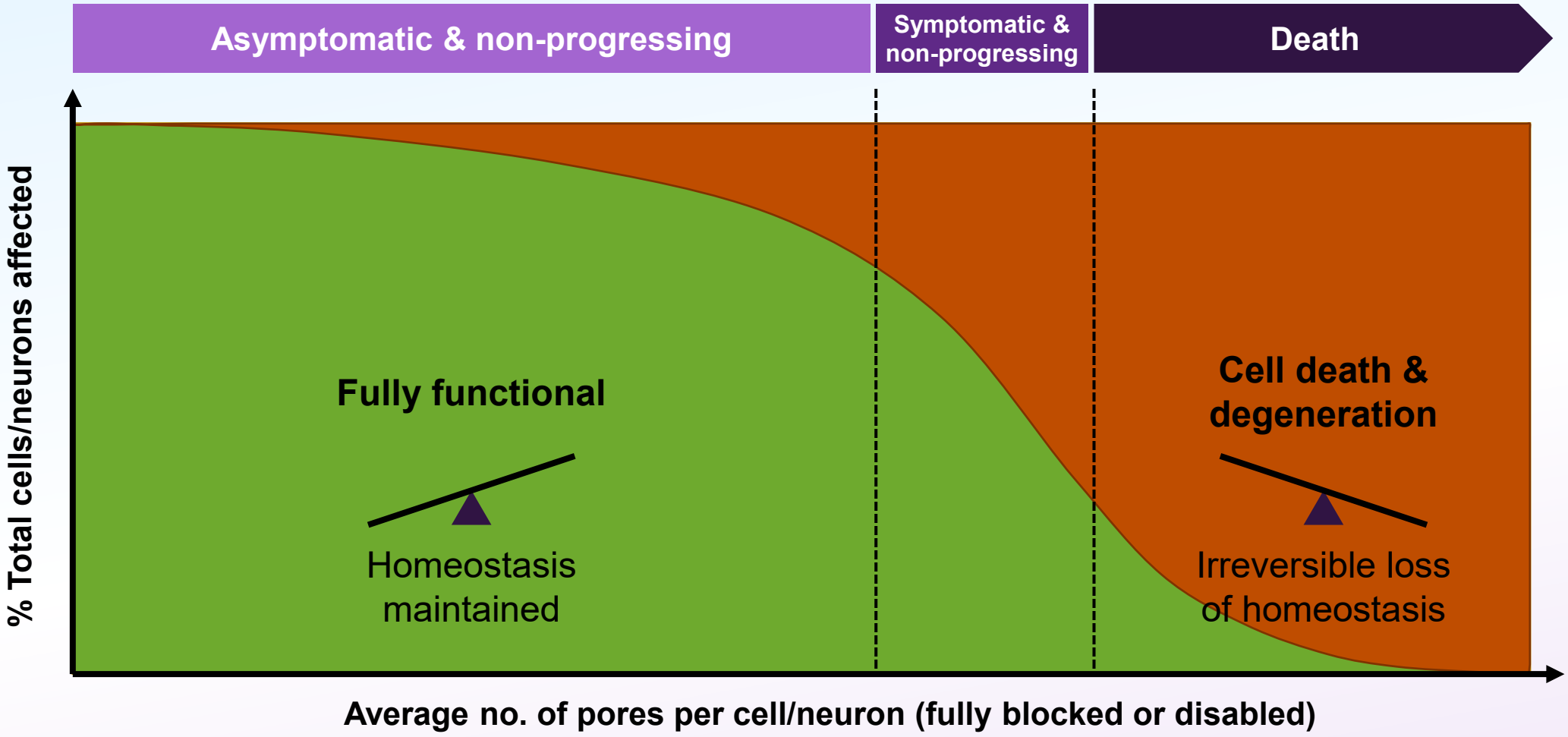
Amporin's 3 New Drug Classes Uniquely Block, Dissolve, and Degrade Toxic Pores to Restore Membrane Integrity, Cellular Homeostasis and Function



Amporin Aims to Stop Disease Progression and Enable Functional Recovery by Allowing Surviving Cells to Fully Recover and Restore Homeostasis



Thus, Amporin's Goal is to Fully Restore Cellular Function in Early Disease, and Stop or Slow Progression in Advanced Disease



Amporin Is Uniquely Positioned to Enable Rapid Functional Recovery and Stop Progression of Many Degenerative Diseases with a Simple Oral Pill

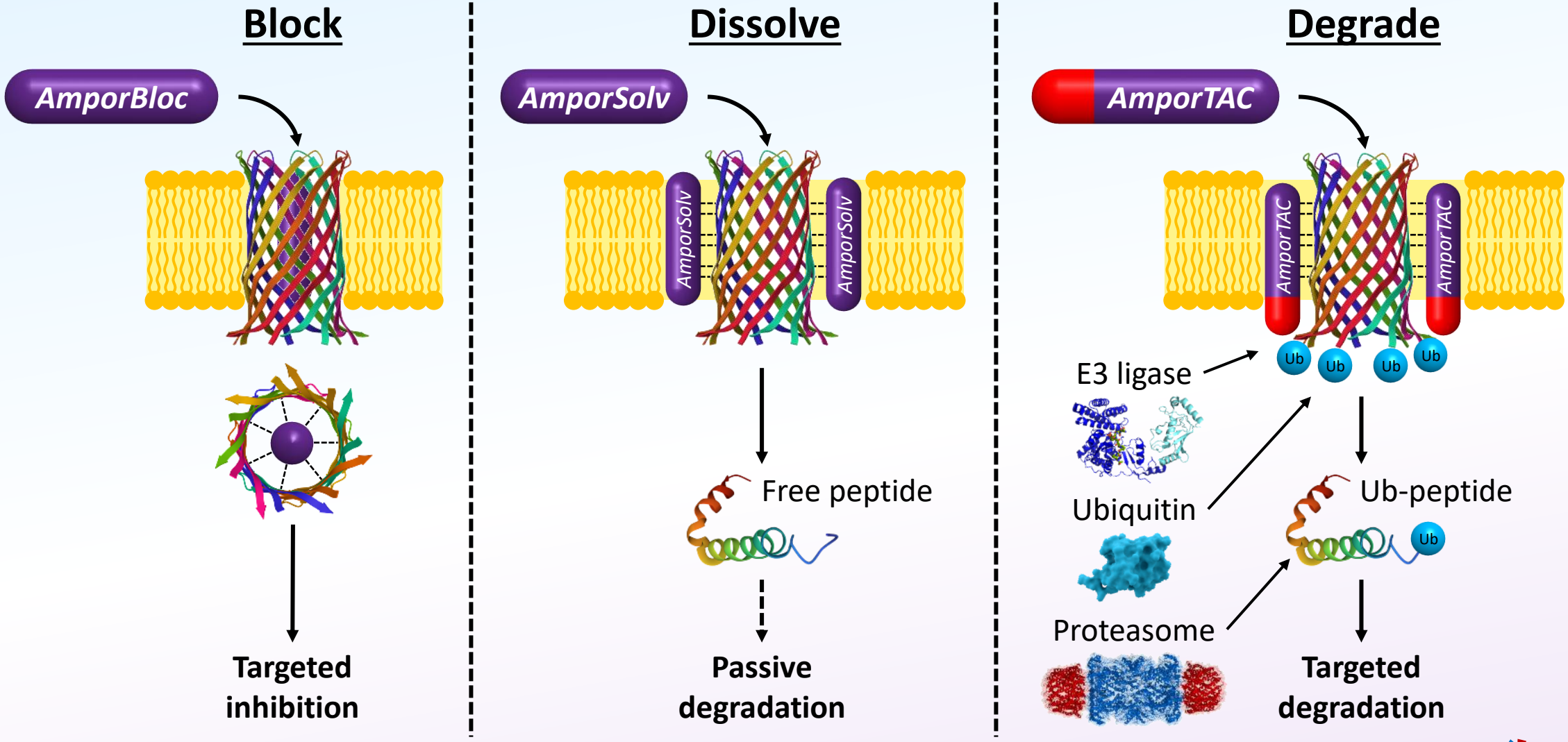
	Chronic preventive treatments Targeting oligomers to delay disease	**Rapid functional recovery** Targeting pores to restore function
Oral drug molecules	NeuroPore Therapies, MODAG Neuroscience Solutions, ALZHEON preserving future memories, TRIMTECH THERAPEUTICS, TauRx Pharmaceuticals Innovation in neurodegeneration, COGNITION Therapeutics, TREVENTIS CORPORATION, Alterity THERAPEUTICS, Oligomerix, WaveBreak	AMPORIN Pharmaceuticals • Restore membrane integrity • Stop uncontrolled Ca^{2+} influx • Eliminate metabolic stress • Restore mitochondrial function • Restore cellular homeostasis • Recover full cellular function
Antibodies & peptides	Biogen, Lilly, BIOARCTIC, ucb, Eisai, Roche, AC Immune, ACUMEN, ProMIS™ Neurosciences, PRIAVOID	AmyPore

Why Have Other Therapeutic Strategies Failed to Stop Disease Progression?

- **Targeting the wrong protein:** Many proteins are affected in degenerative diseases, but they are not ultimately responsible for causing disease (correlation vs causation)
- **Targeting the wrong form of protein:** Insoluble amyloid fibrils, plaques and other protein deposits are a non-pathogenic result of protein misfolding, rather than a direct cause of disease (smoke vs fire)
- **Targeting the right protein too early:** Blocking protein expression, misfolding or aggregation might prevent the formation of new toxic pores to slow disease progression over time, but will not stop the progressive effects of existing toxic pores once they have already formed
- **Wrong therapeutic platform:** Antibodies and peptides are unable to cross the BBB, enter neurons, and target toxic pores buried deep within cell and mitochondrial membranes
- **Targeting the effects of disease:** Targeting the downstream effects of toxic pores (intracellular calcium overload, mitochondrial dysfunction, oxidative stress, inflammation, etc.) may provide short-term symptomatic relief, but does not address the underlying cause of disease by restoring membrane integrity
- **Summary:** No therapy will stop disease progression unless and until membrane integrity is restored, otherwise sustained calcium influx will continue to cause chronic cellular stress, mitochondrial dysfunction, ATP depletion, loss of homeostasis, and progressive cell death and degeneration

Clearing glass/debris from the road or inflating your tyres will not help if you already have a puncture!

Amporin is Developing 3 New Drug Classes to Block, Dissolve and Degrade Toxic Pores in Cell and Mitochondrial Membranes



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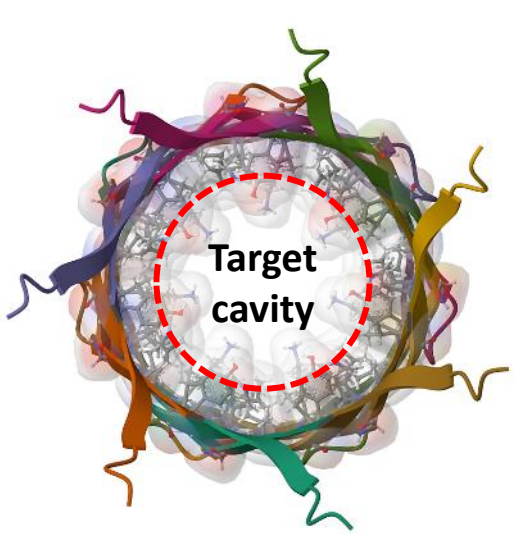
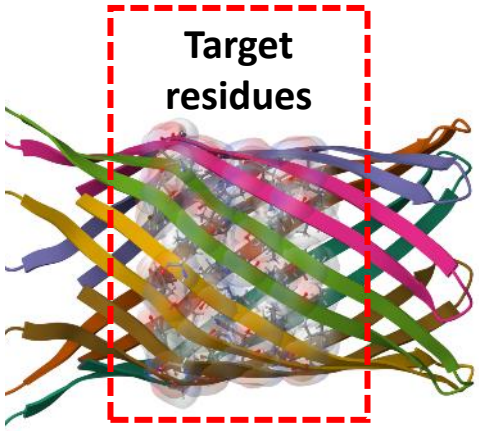
AmporBlocs are Designed to Block Pores More Tightly and Specifically than Reported Protein Aggregation Inhibitors and Other Known Compounds



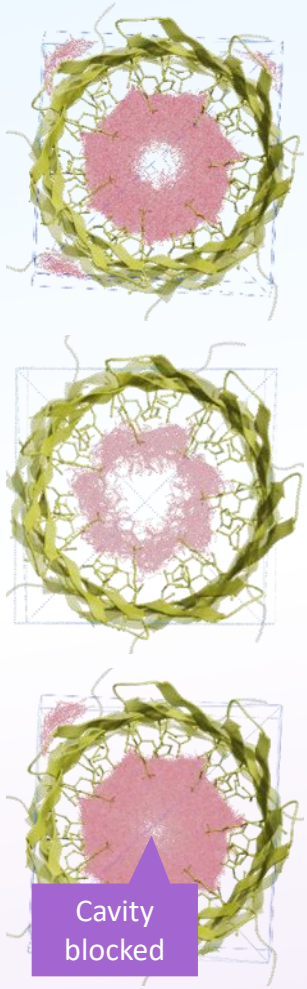


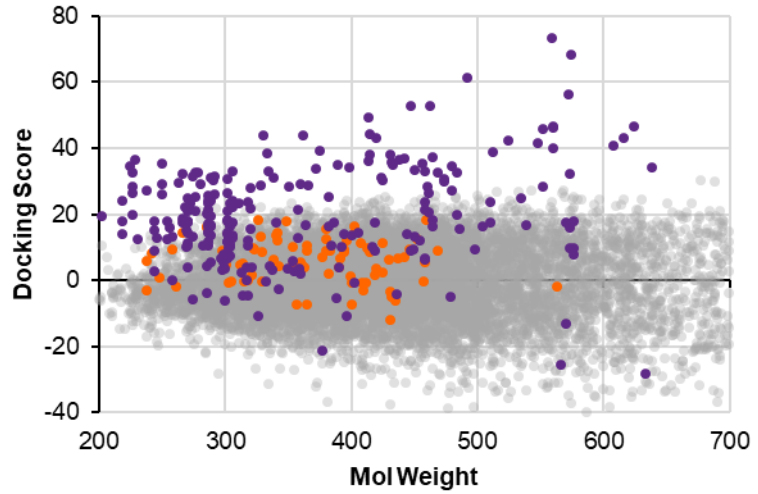
Molecular docking

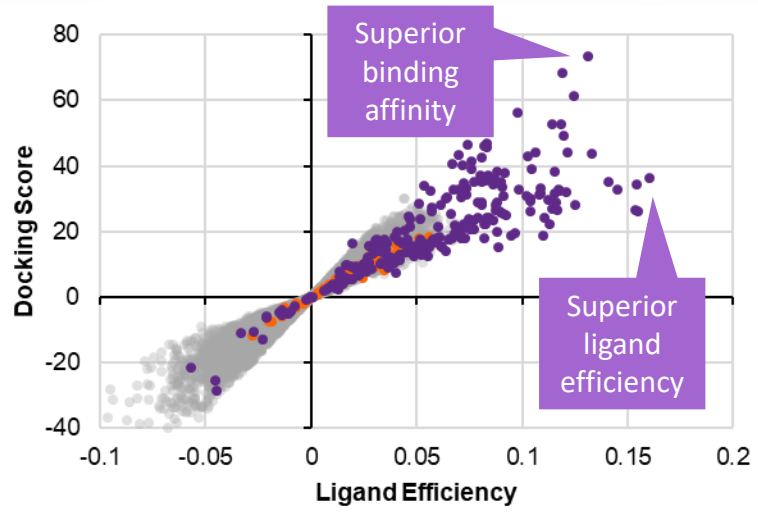
ChEMBL library

Reported inhibitors

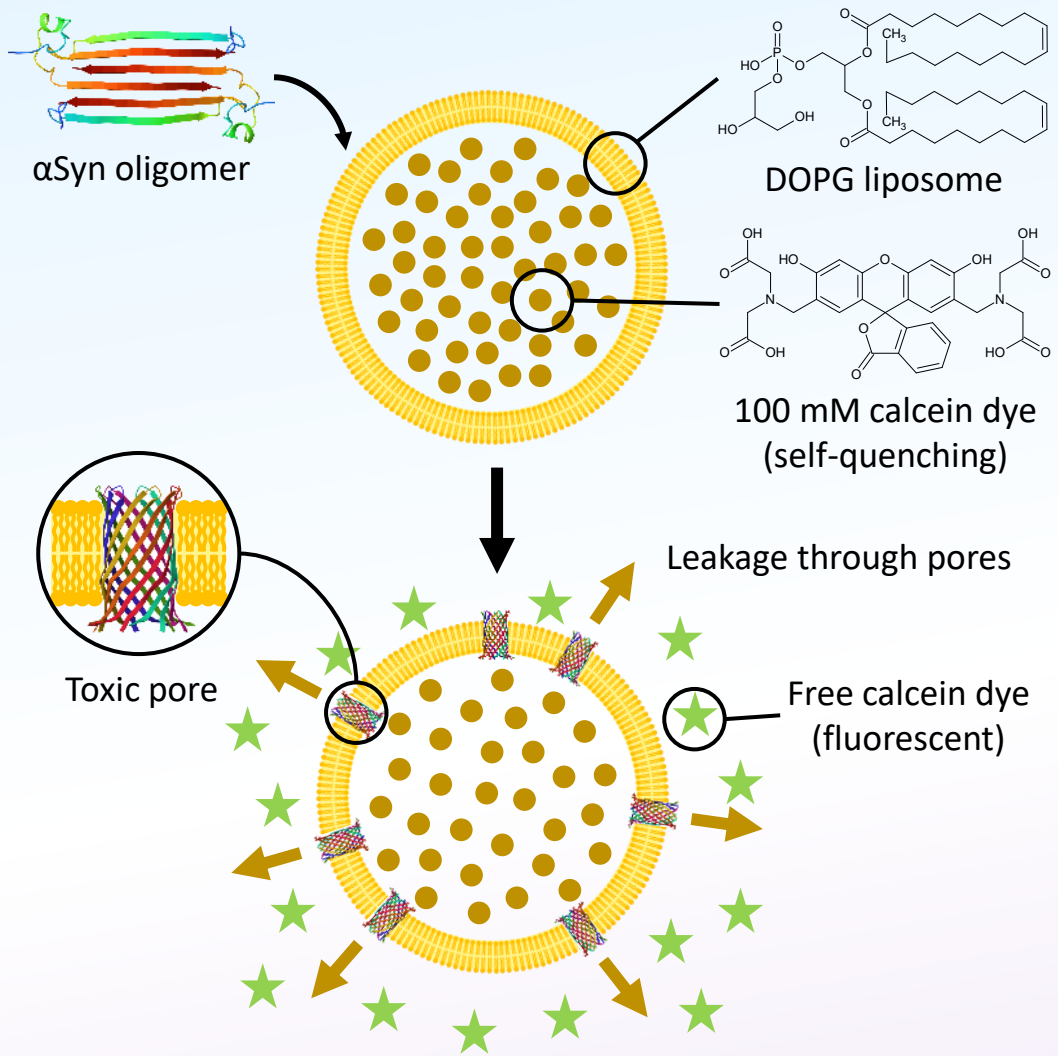
AmporBloc library

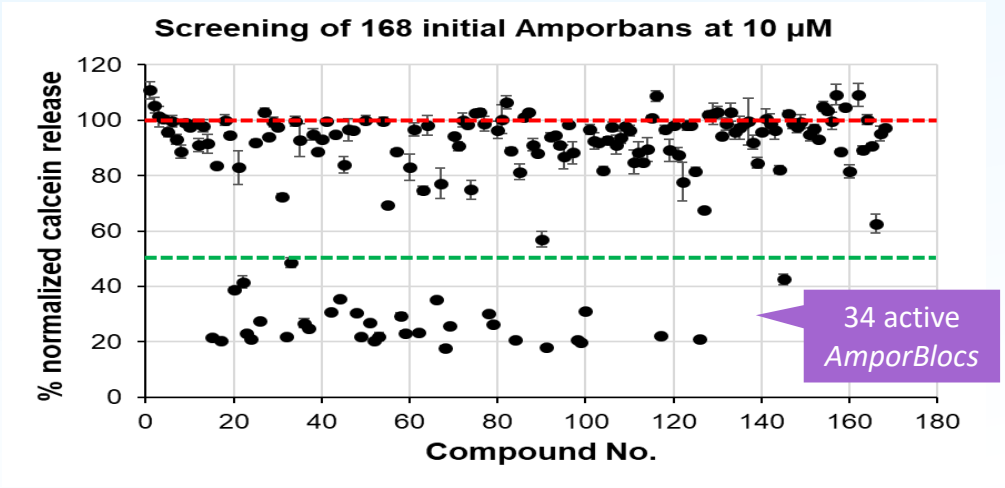


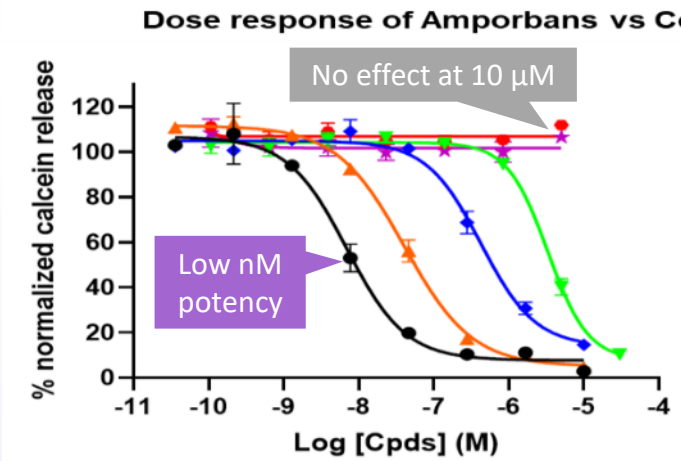




AmporBlocs Stop α-Synuclein-Induced Leakage of Calcein from Liposomes (via Pores or Otherwise) at Low Nanomolar Concentrations







Compound	IC50 (nM)
AMP072	7
AMP078	40
AMP069	424
AMP063	3,282
Anle138b	>10,000
Minzasolmin	>10,000

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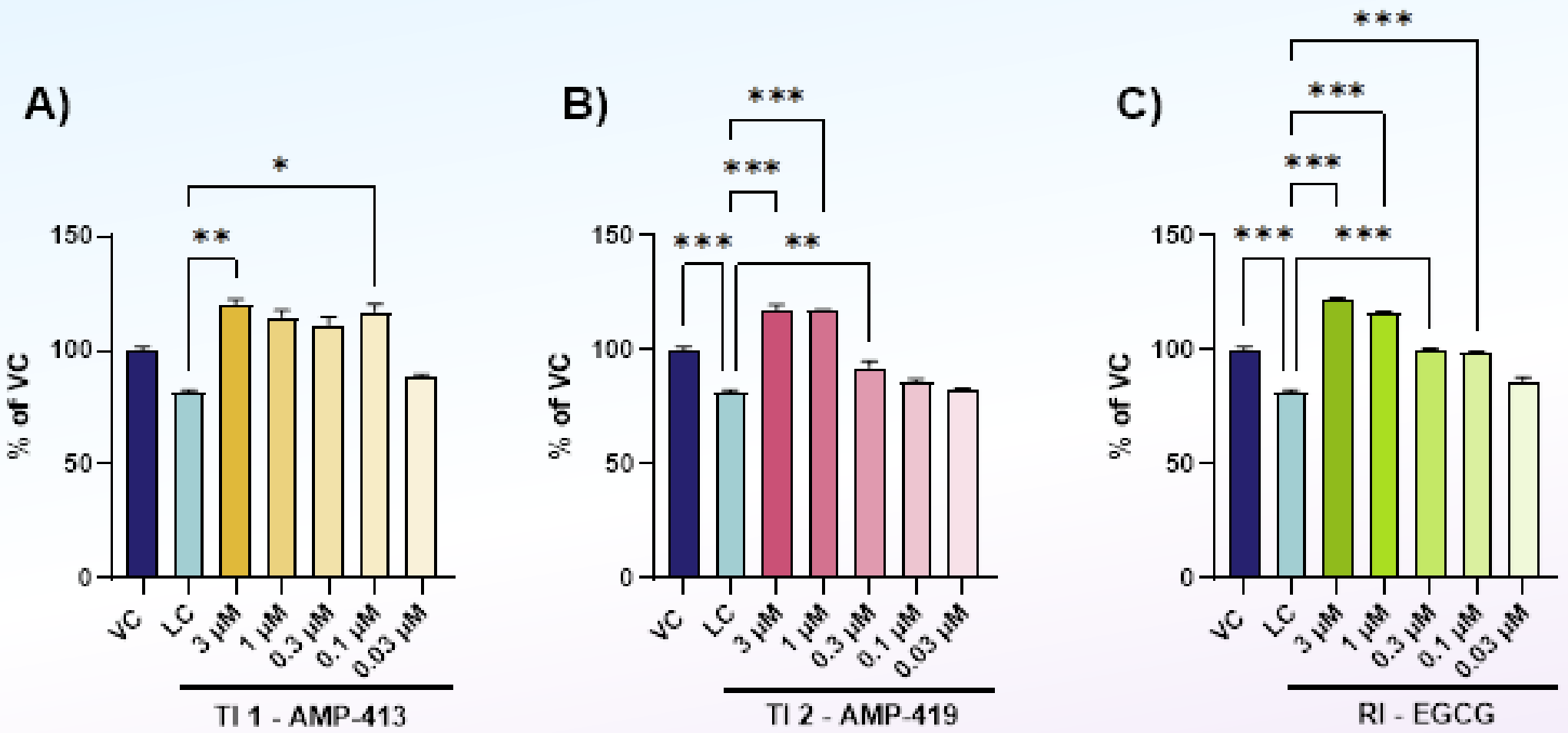
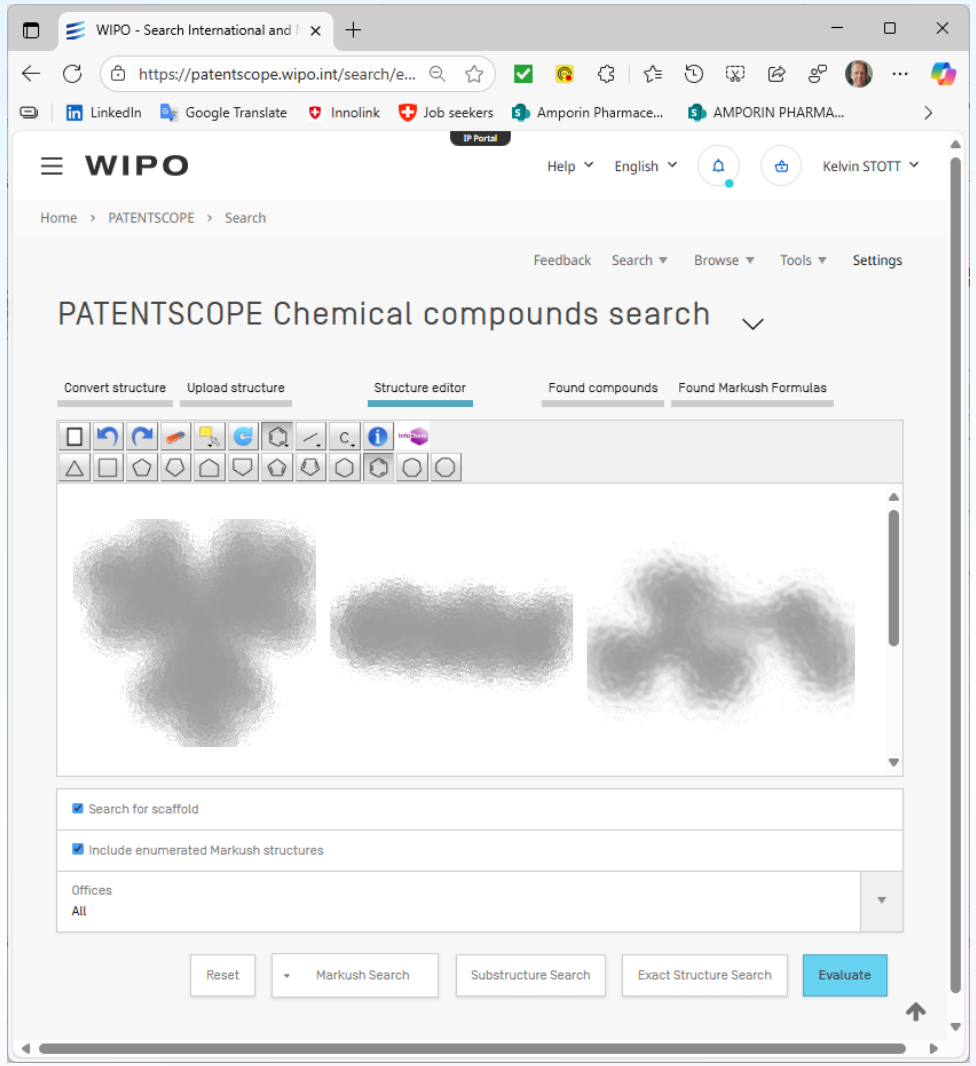
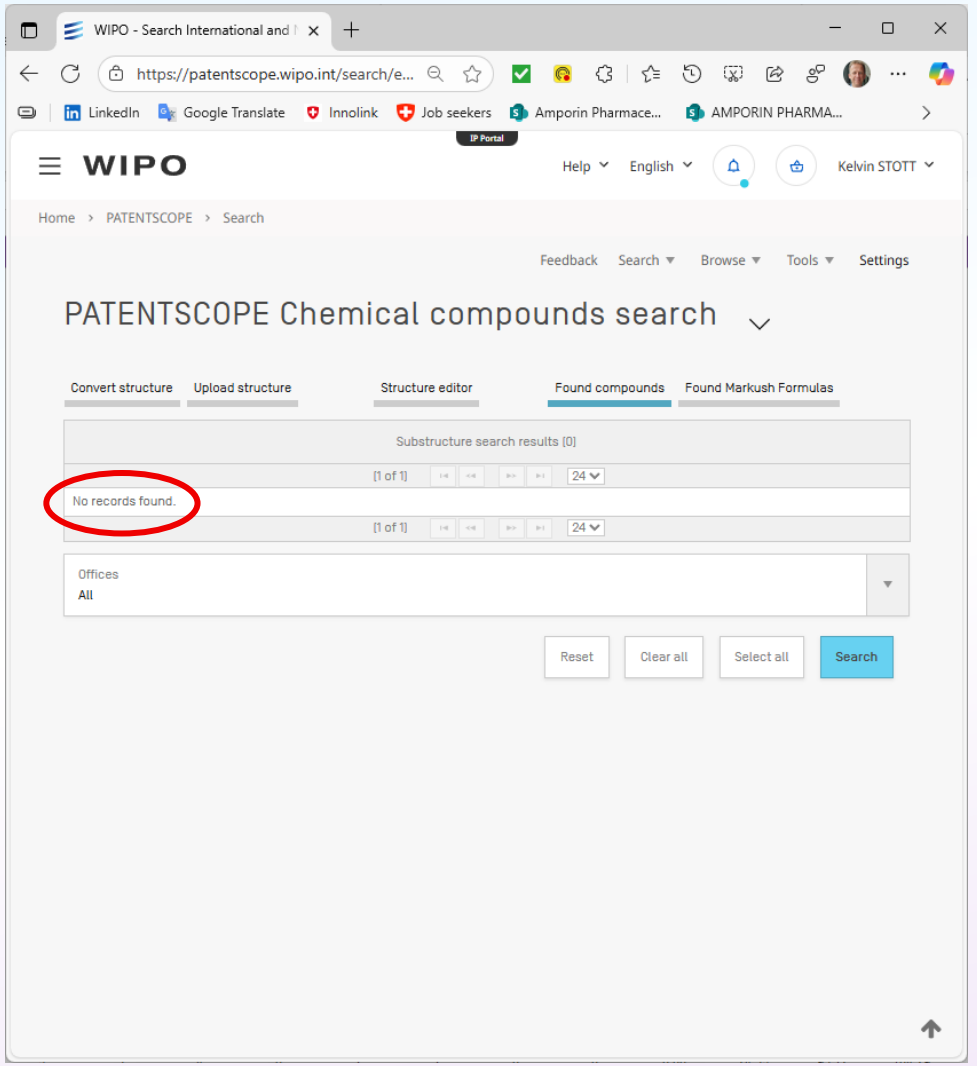
AmporBlocs Demonstrate Potent, Dose-Dependent Protection of Neuronal SH-SY5Y Cells Against α-Synuclein Oligomer-Induced Cell Toxicity



AmporBlocs, AmporSolvs and AmporTACs Are Novel, With No Prior Art

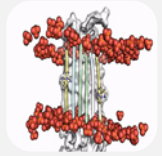




Amporin is Now Developing a Complete Screening Platform to Optimize, Select and Validate Specific Drug Candidates for Each Disease

Optional supporting studies

AI-based drug design
- AI-based QSAR
- Virtual libraries



Modeling/simulation
- Target modeling
- Molecular dynamics

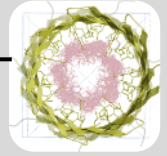
Mechanistic studies
- Conductance
- Calcium influx

Cellular biomarkers
- Oxidative stress
- Immunostaining

Other ADME assays
- Transporter assays
- Off-target assays

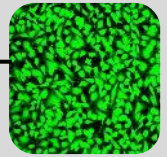
In silico & in vitro screening

Med chem expertise
- QSAR analysis
- Drug library design



In silico screening
- Molecular docking
- ADME prediction

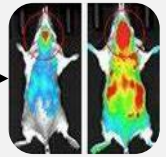
Calcein leakage assays
- Fixed dose
- Dose response



Cell toxicity assays
- SH-SY5Y cells
- Primary neurons

In vitro ADME assays
- LogD, Caco-2, P-gp
- CYP, microsomes, …

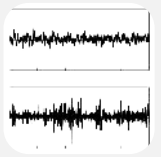
In vivo testing & validation



In vivo DMPK testing
- Drug metabolism
- Pharmacokinetics

Functional testing
- Motor function
- Cognitive tests

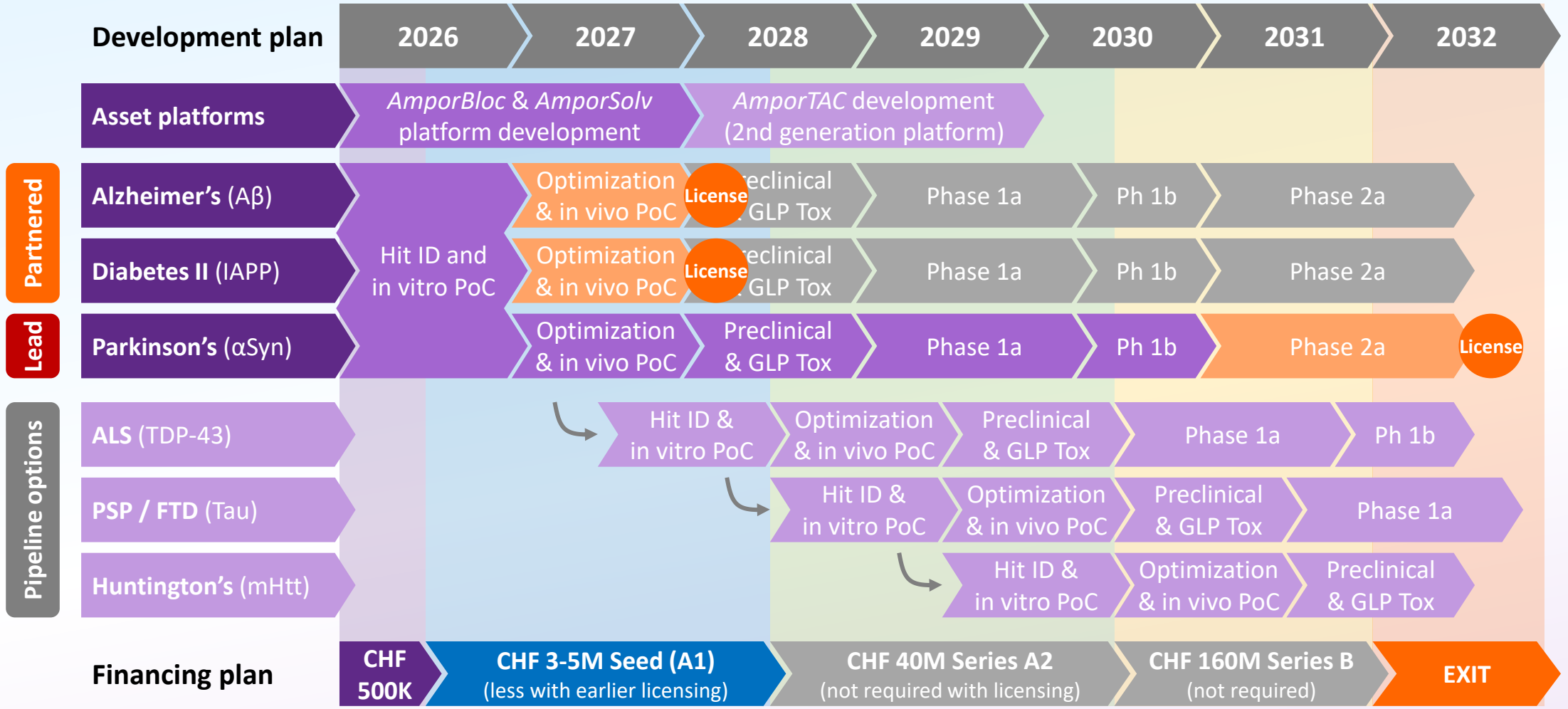
Behavioural testing
- Sleep structure
- Apathy/anxiety



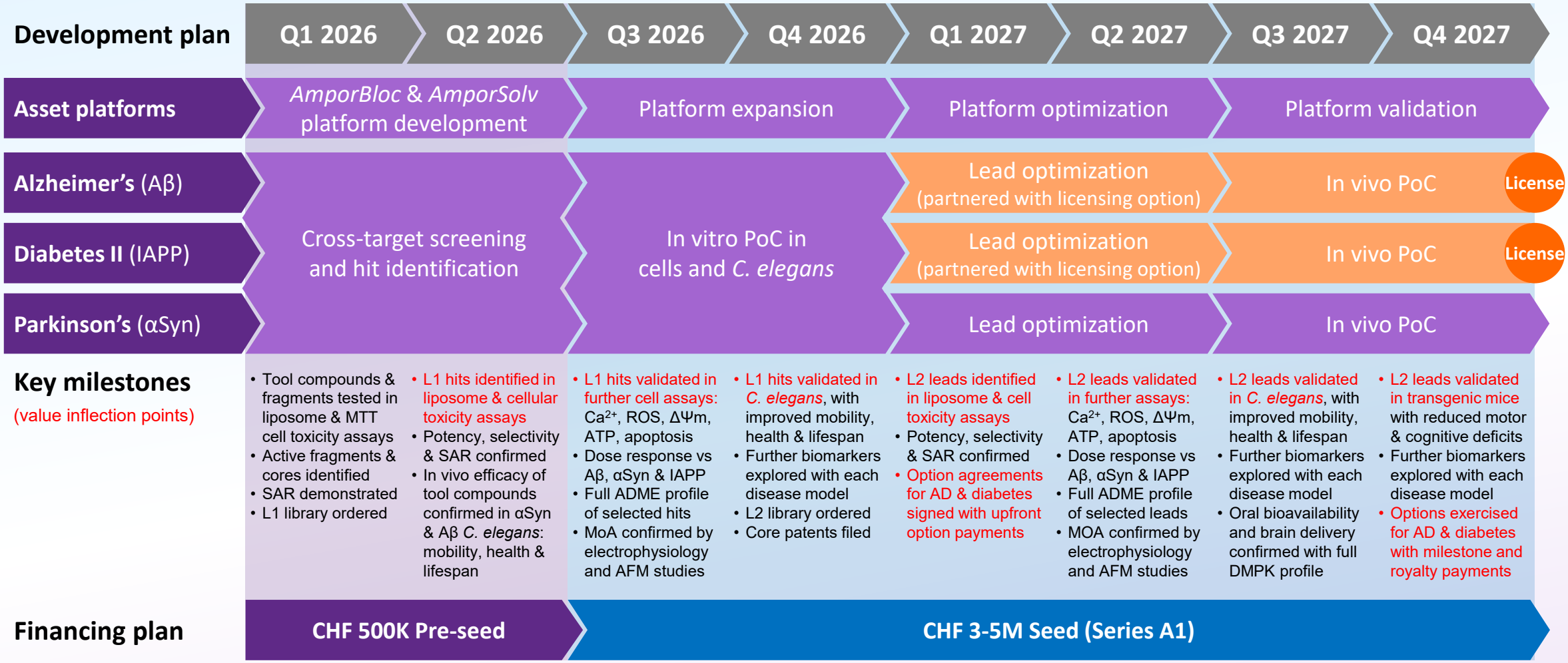
Electrophysiology
- EEG, EMG
- LTP, LFP

Biochemical markers
- Oxidative stress
- Inflammation

Amporin Plans to Develop and License a Full Pipeline of Drug Candidates, Supported by 2 Highly Leveraged Early Licensing Deals with Big Pharma



Amporin Plans to Partner and License its AD and Diabetes Programs Early, and Establish In Vivo Proof-of-Concept for its Own In-house PD Program



Clinical Development Plan for Rapid Biomarker-based Proof-of-Mechanism (PoM) and Proof-of-Concept (PoC) With Rapid Functional Recovery in PD

Phase 1a

- Establish **safety/tolerability** with SAD/MAD studies in young, then elderly healthy volunteers
- Option to **evaluate biomarkers** with additional MAD study in αSyn-positive PD patients

Biomarkers (patients only)

- Diagnostic: SSAA, DAT-SPECT
- Digital: Motor, Speech
- Exploratory: Sleep, rsEEG

15 months; CHF 8-12M

Phase 1b

- Establish **Proof-of-Mechanism (PoM)** in αSyn+ PD patients
- Show **rapid functional recovery** with key biomarkers informed by preclinical and Phase 1a studies

Biomarkers

- Diagnostic: SSAA, DAT-SPECT
- Digital: Motor, Speech
- Behavioural: Sleep, Anhedonia, Reward functioning
- Exploratory: Cognitive functions, rsEEG, Olfaction, FDG-PET (PDRP)

9 months; CHF 8-12M

Phase 2a

- Establish **Proof-of-Concept (PoC)** in αSyn+ PD patients
- Confirm **rapid functional recovery** with consistent effect on selected biomarkers

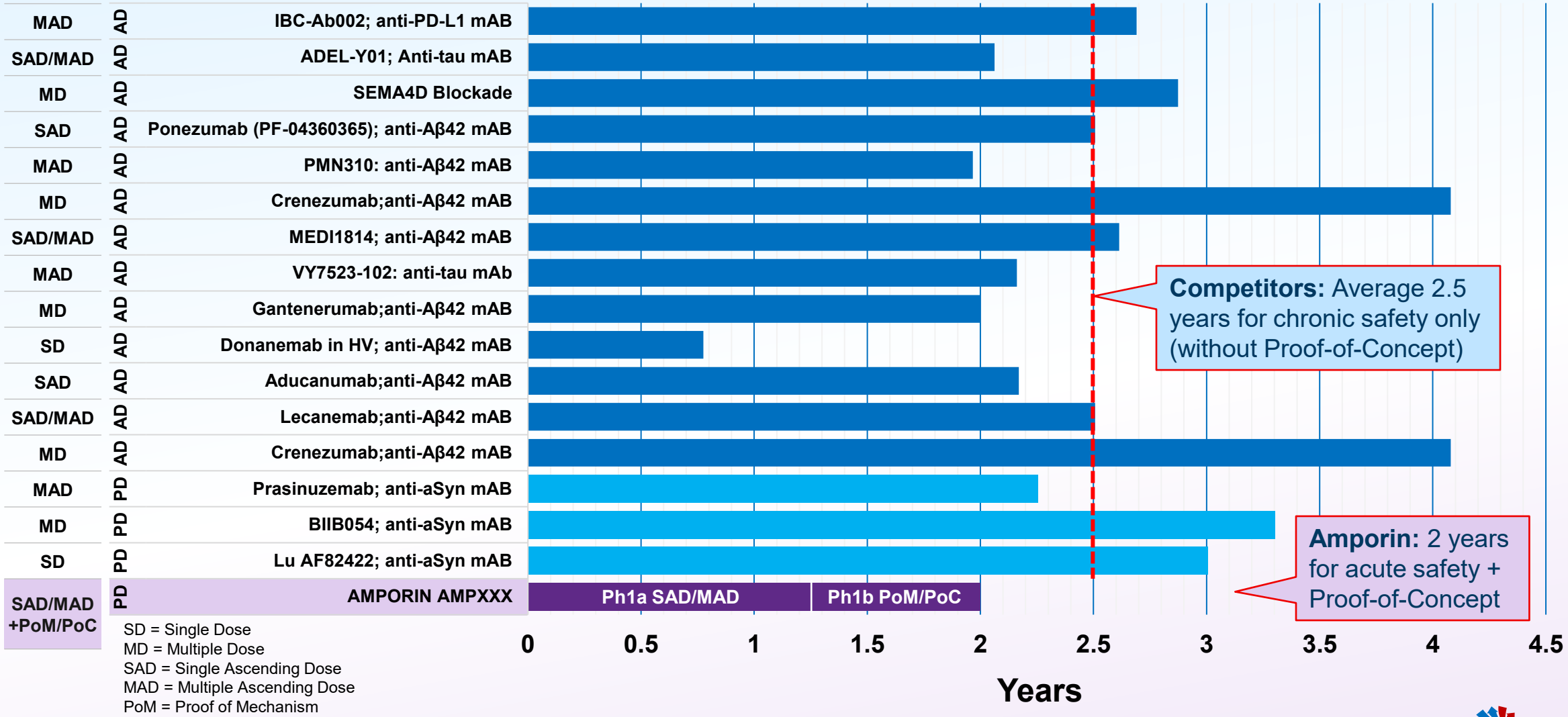
Biomarkers

- Diagnostic: SSAA, DAT-SPECT
- Digital: Motor, Speech
- Behavioural: Sleep, Anhedonia, Reward functioning
- Cognitive functions
- Exploratory: rsEEG, Olfaction

18 months; CHF 20-25M

SSAA = αSynuclein Seed Amplification Assay; DAT-SPECT = Dopamine transporter single-photon emission computed tomography; rsEEG = resting state electroencephalogram (EEG)

Acute Safety and Proof-of-Mechanism (PoM) for Rapid Functional Recovery can be Demonstrated Faster Than Chronic Safety of Competitor Programs



SD = Single Dose
MD = Multiple Dose
SAD = Single Ascending Dose
MAD = Multiple Ascending Dose
PoM = Proof of Mechanism

Amporin is Led by an Experienced Management Team (1/2)

Kelvin Stott PhD – CEO & Cofounder

- Senior R&D portfolio executive, scientist, inventor, visionary, serial entrepreneur
- >25 years in Pharma (Novartis, Merck Serono), biotech (CSL, Sensyne, Senexis), consulting (McKinsey, PwC, Quantelium) and venture capital (Inventages)
- Former founder & CSO at Senexis (also targeting degenerative protein misfolding diseases)
- Former Research Fellow and PhD at Cambridge with Nobel laureate Max Perutz

Hervé Schaffhauser PhD – CSO & Cofounder

- Experienced in vitro neuropharmacologist, translational neuroscience leader
- >25 years in Pharma (Roche, Merck) and biotech (Coave, Versameb, Preciphos, Cephalon)
- Expert in CNS drug discovery, assay and preclinical drug development
- Led discovery and development of CNS drugs from target validation into Phase 1 trials
- Former Director Translational Neuroscience at Coave, PhD at Louis Pasteur, Post-doc at Emory

Prof Daniel Umbricht MD – CMO & Cofounder

- Senior translational medicine expert, clinical development leader in neuroscience
- >30 years in Pharma (Roche, Novartis), biotech (Autifony, Gilgamesh), clinical practice (Univ. Zurich, Long Island JMC, Albert Einstein College of Medicine, etc.) and consulting (Xperimed)
- Former Global Head of Translational Medicine in Neuroscience at Roche
- Former Associate Professor of Psychiatry and MD at University of Zurich

Amporin is Led by an Experienced Management Team (2/2)

RJ Tesi MD – Board Member

- Specialist in CNS, inflammation, immunology and longevity
- >25 years in surgery, research, teaching and strategic development roles in Pharma & Biotech
- Former President and CEO at INmune Bio, focused on neuro-inflammation in Alzheimer's
- Former SVP at Sangstat Medical and EVP Clinical Development and Medical Affairs at Cellerant
- Founded several biotechs, with senior development and commercial leadership roles in each

Torben Skarsfeldt PhD – Board Advisor

- Specialist in CNS and Biopharmaceuticals
- 17 years in research & discovery + 13 years in international business development
- Former CEO of multiple biotech ventures, including Pharma Guidance and Phlogo
- Former CNS Project Director, BD&L leader, and Supervisory Board Member at Lundbeck
- Co-inventor of antipsychotic drug Serdolect (sertindole), marketed in 15 countries

Amporin Requires CHF 3-5M to Start IND-enabling Studies in Parkinson's Disease

Company	AG incorporated in Basel, April 2024
Funding to date	**CHF 500K total** • CHF 100K: 3 founders (equity) • CHF 150K: Venture Kick (loan) • CHF 100K: Kickfund (loan) • CHF 150K: 7 angels (loans)
Funding required	**CHF 3-5M Seed/Series A1 (equity)**
Closing	**End Q4 2026**
Use of funds	• Develop and validate platforms • Complete lead optimization • Select 3 drug leads with in vivo PoC • Partner/out-license AD & Diabetes • Start IND-enabling studies in PD
Exit	• Sale to Big Pharma by 2032

Recent Billion-Dollar Licensing/M&A Deals in Neurodegenerative Diseases

Year	Companies	Deal Type	Focus Area	Deal Value
2026	Novartis & SciNeuro	Licensing / Collaboration	AD	Up to $1.7B
2025	Novartis & Arrowhead	Licensing	PD (Synucleinopathies)	Up to $2.0B
	Sanofi & ADEL	Licensing	AD	Up to $1.1B
2024	AbbVie & Aliada	Acquisition	AD	$1.4B
	Takeda & AC Immune	Licensing	AD	Up to $2.2B
	Roche & Sangamo	Licensing	Neurodegenerative	Up to $1.9B
	BMS & BioArctic	Licensing	AD	Up to $1.35B
2023	Biogen & Reata	Acquisition	Rare Neurodegenerative	$7.3B
	BMS & Evotec	Collaboration	Neurodegenerative	Up to $4.0B
	Neurocrine & Voyager	Licensing / Collaboration	PD	Up to $1.5B
2022	Sanofi & ABL Bio	Licensing / Collaboration	PD (Synucleinopathies)	Up to $1.1B
2021	UCB & Novartis	Collaboration	PD	Up to $1.5B
2020	Biogen & Denali	Licensing / Collaboration	PD	$2.2B
	Biogen & Sangamo	Licensing / Collaboration	AD, PD	Up to $2.4B
	Roche & UCB	Licensing / Collaboration	AD	Up to $2.0B
	Eli Lilly & Prevail	Acquisition	PD, ALS	$1.0B
	Biogen & Sage	Collaboration	Neurological (CNS)	Up to $3.1B

AMPORIN Pharmaceuticals

Opportunity: A First-in-Class Platform with Multi-Blockbuster Potential

Investment rationale	Supporting details
Huge unmet need	• At least 50 deadly degenerative diseases affecting over 500 million people • $3 trillion associated annual cost, growing rapidly with the ageing population • $100 billion current drug market comprising only symptomatic treatments
Disruptive potential	• First-in-Class drug platform targeting novel mechanism in multiple diseases • First potential oral drugs to stop progression with rapid functional recovery • Opportunity to dominate market with a new class of disease-modifying drugs
Strong science & IP	• The toxic pore/channel hypothesis is now supported by hundreds of papers • Strong in vitro data, highly potent, clean dose response and SAR, no prior art • Preliminary evidence for rapid functional recovery in vivo (to be confirmed)
De-risking strategy	• Rapid lead optimization with established assays via CROs and collaborations • Rapid validation with established disease-specific preclinical/clinical biomarkers • Potential for early partnering and out-licensing of AD and Diabetes programs
Experienced team	• Founders/Board have >25 yrs R&D leadership experience in Pharma/Biotech • Proven track record of developing CNS drugs from the lab to the clinic/market
Huge exit potential	• Pharma desperate to fill its pipeline with novel disease-modifying treatments • Multiple recent billion-dollar Pharma deals in neurodegenerative diseases • Several Pharma companies have already initiated discussions with Amporin

Beyond investing in proprietary First-in-Class drug programs, investors will gain substantial valuation leverage from the planned early out-licensing of Alzheimer's and Diabetes programs in return for significant in-flow of cash

AMPORIN Pharmaceuticals

AMPORIN Pharmaceuticals

Repairing Membranes to Rescue Mitochondria in Degenerative Diseases

Thank you!

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