Next generation drugs for

Chronic age-related diseases

Cancer
Dementia
Macular degeneration

Presentation – October 2025

Chronic inflammation-associated diseases of older people

- Heart disease
- Dementia
- Chronic diseases of lung, kidney and liver
- Type 2 diabetes
- Macular disease
- Age-related cancers *(eg, prostate, breast, lung, pancreas, large bowel)*

are already presenting major problems for national health systems in the face of an increasingly ageing global population

Chronic inflammatory diseases are complex illnesses, usually decades in the making, involving the abnormal behaviour of hundreds of different genes.

Current anti-inflammatory drugs deliberately target as few genes as possible (so-called *'precision medicine'*). Filamon believes this narrowness of activity plays a major role in why the successful management of chronic age-related disease remains the medical challenge of our time.

Filamon believes it has taken an important step in meeting this challenge by developing three technology platforms deliberately designed to address the misbehaviour of multiple genes and their proteins involved in chronic inflammation.

Founders

Paul de Souza

Medical oncologist
Professor of Medicine
University of Sydney

Kieran Scott

Medical scientist
Assoc. Professor of Oncology
Western Sydney University

Graham Kelly

Medical scientist, biotech entrepreneur
Adjunct Professor, University of Sydney

Board

ROBERT EDGLEY
NON-EXECUTVE CHAIRMAN

Extensive career in International Finance and Investment Banking with the NatWest Group and Royal Bank of Scotland. 25+ years experience with a proven track record as a Non-Executive Director in high-growth ASX-listed and private companies.

GRAHAM KELLY
MANAGING DIRECTOR AND CEO

Medical scientist with over 55 years of experience in medical research and drug development. Founder, Chairman, and CEO of four publicly listed biotechnology companies: Noxopharm Ltd (NOX.ASX), Nyrada Inc (NYR.ASX), MEI Pharma Inc (MEIP.NAS), and Kazia Therapeutics Ltd (KZIA.NAS). Successfully raised > $350 million in funding

PAUL DE SOUZA
NON-EXECUTIVE DIRECTOR

PhD from the University of UNSW. Formerly with Eli Lilly Australia, followed by the role of Chair of Medical Oncology at the University of Sydney. A medical oncologist specialising in translational medicine,with extensive experience as Principal Investigator in multiple Phase 1, 2, and 3 clinical drug trials

STEPHEN MENZIES
NON-EXECUTIVE DIRECTOR

One of Australia's leading securities lawyer. Head of Ashurst China practice until 2018. Variously founder, seed investor and director of a range of biotechnology companies. Director of Platinum Asset Management 2014- 2022. Recently retired as Chair of Silicon Quantum Computing P/L

PHILIP MARSHALL
NON-EXECUTVE DIRECTOR

Medicinal chemist and senior executive with hands-on experience at Mayne Pharma Australia and Sigma. Extensive expertise in drug discovery, formulation, manufacturing, pharmaceutics and regulatory compliance.

Three technology platforms delivering a current pipeline targeting five major unmet needs

Cancer

- **Blocking cancer-generated inflammation with the aim of reducing or eliminating tumour growth and metastatic spread**
- **Overcoming the susceptibility of EGFR to mutate in many common forms of cancer**
- **Reversing T cell exhaustion to boost immune checkpoint inhibitor therapy response rates**

Eye

- **Developing a more effective, safer and patient-friendly treatment of wet AMD**

Brain

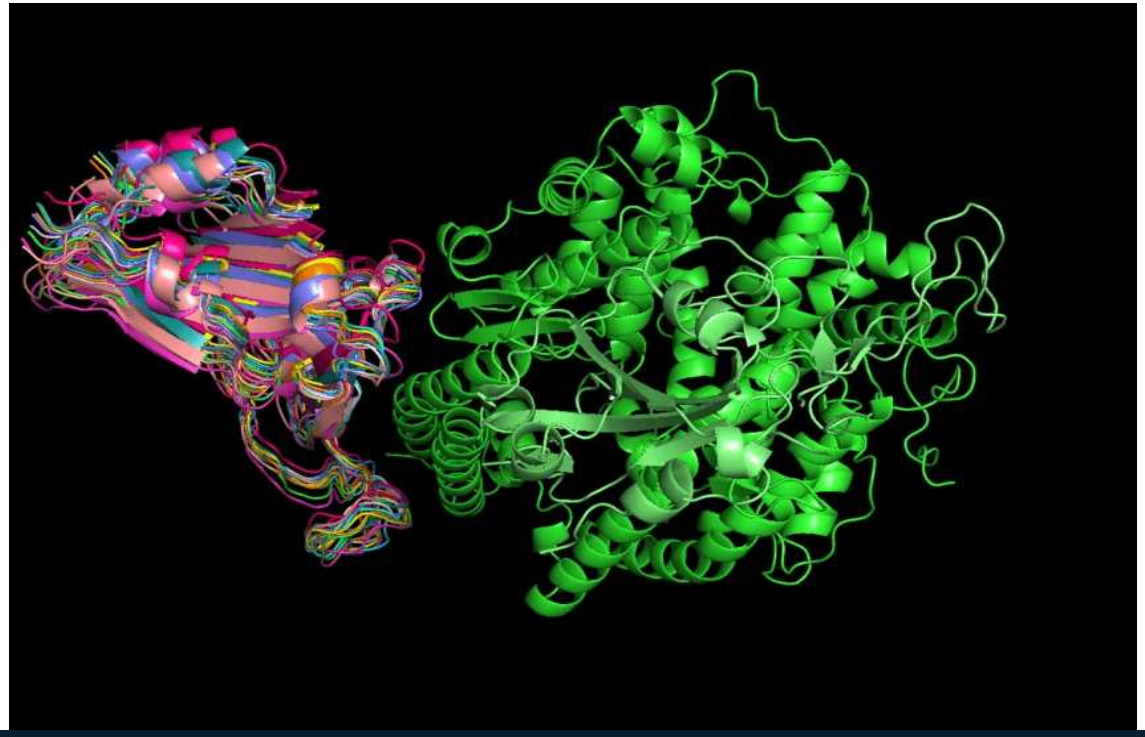
- **Developing an impactful early-stage dementia and Parkinson's Disease treatment via the disruption of small protein clump formations in the brain**

ALPHA Platform drugs

Protein-protein interaction modifiers

Filamon owns this key IP outright

ALPHA technology is clinical-stage



Protein-protein interaction or **PPI** (as shown here) is fundamental to life. The connection of one protein to another underpins the great majority of biological functions in the body.

Equally, the great majority of chronic diseases, notably cancer and dementia, are associated with abnormalities in PPI.

ALPHA technology is concerned with normalizing PPI behaviour.

ALPHA drugs are cyclic peptides that are dosed orally.

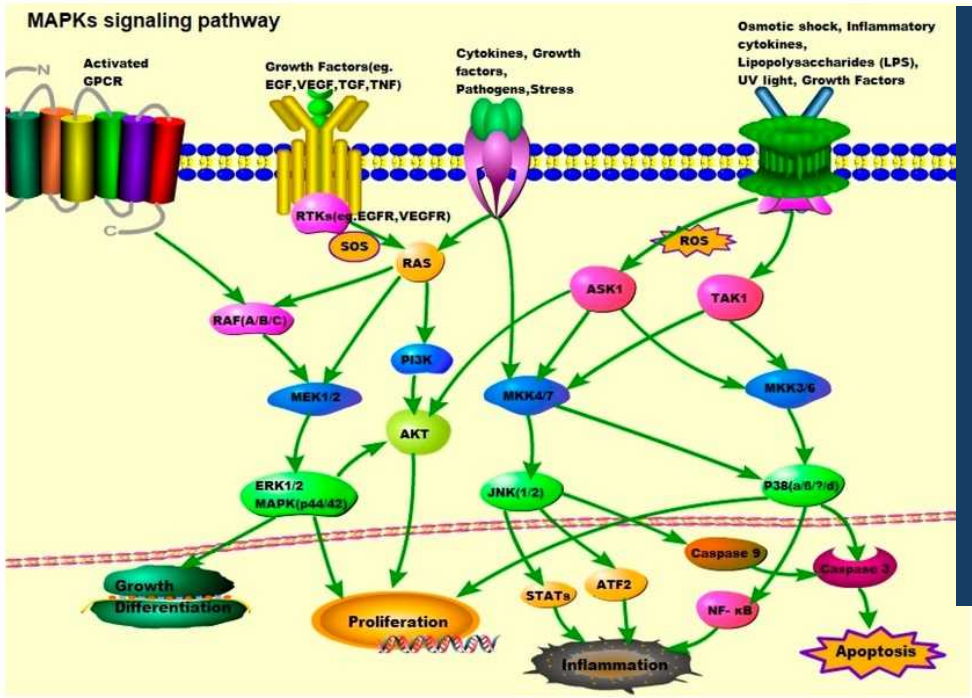
BETA platform drugs

The MAPK enzyme signaling pathway is one the cell's most important control mechanisms playing a central role in maintaining the health of all tissues. Abnormal MAPK signaling also is linked to many chronic inflammatory and degenerative diseases. It is a major and validated drug target for many chronic diseases.

It is also one of the most complex signaling pathways, comprising 3 main sub-families called ERK (7 forms), JNK (3 forms) and p38 (4 forms) plus multiple minor sub-families comprising an as yet unidentified total number of kinases (assumed to exceed 30 kinases)

This considerable complexity, compounded by extensive cross-talk between all members of this family, has to date blocked the development of broadly effective and safe MAPK inhibitors.

Type 4 kinase inhibitors of select MAPK kinases



Filamon believes BETA drugs to be uniquely acting inhibitors of MAPK proteins.

1. They are selective to which MAPK proteins they bind
2. They are Type 4 inhibitors, binding to an allosteric site neighboring the kinase-binding site, providing them with the ability to modify downstream signaling activity resulting in both down- and up-regulation of genes.

Filamon in-licenced the original BETA IP from UNSW and owns outright all IP improvements

BETA technology is pre-clinical

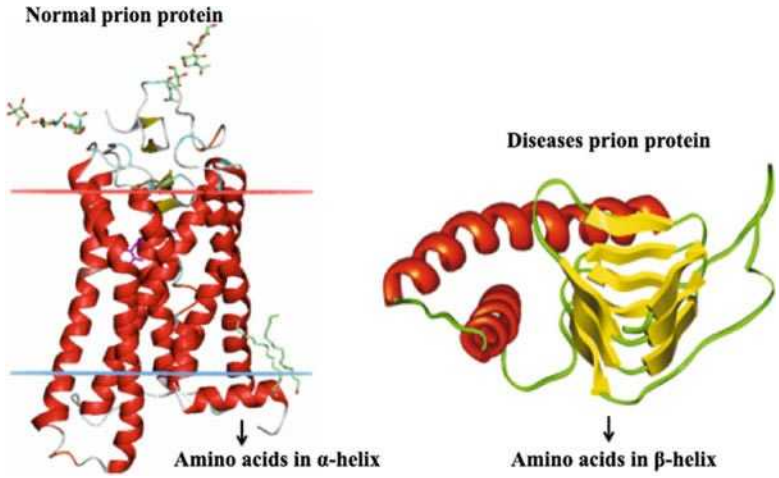
BETA platform drugs currently are the recipients of $7.3M in non-dilutionary grant funding.

SIGMA platform drugs

Inhibitor of proteins with prion-like behaviour

In-licenced from a private biotech with Filamon to own outright all IP improvements

SIGMA technology is pre-clinical



Prions are normal proteins that mis-fold and assume a dysfunctional shape causing them to behave abnormally. Instead of being instantly degraded, prions resist degradation (protease resistant), accumulate and spread throughout a tissue by transforming all other like proteins to the same prion form.

SIGMA drugs bind to and inactivate a range of proteins displaying prion behaviour.

Spongioform encephalopathies (Mad Cow Disease of cattle and Creutzfeld-Jacob Disease (CJD) in humans are examples of severe prion protein behaviour involving the master prion protein (PrP).

However, prion behaviour is now known to involve a range of other proteins including the proteins that misfold and form aggregates in Alzheimer's Disease and Parkinson's Disease, in eye diseases including macular degeneration, and in some cancers.

The field remains speculative, but evidence is mounting for a key role of prion protein behaviour in a growing list of degenerative conditions marked by rapid spread.

SIGMA drugs may be the first class of drug able to distinguish between normal and prion forms of the same protein.

Filamon Oncology

KESONOTIDE
ALPHA drug

Oral comprehensive inhibitor of both cancer cells and cancer-associated cells making up the tumour micro-environment

LYTAP drug
ALPHA drug

Oral EGFR degrader

BETA –TT17
BETA drug

Oral cytotoxic, VEGF-inhibitor, PD-1 inhibitor

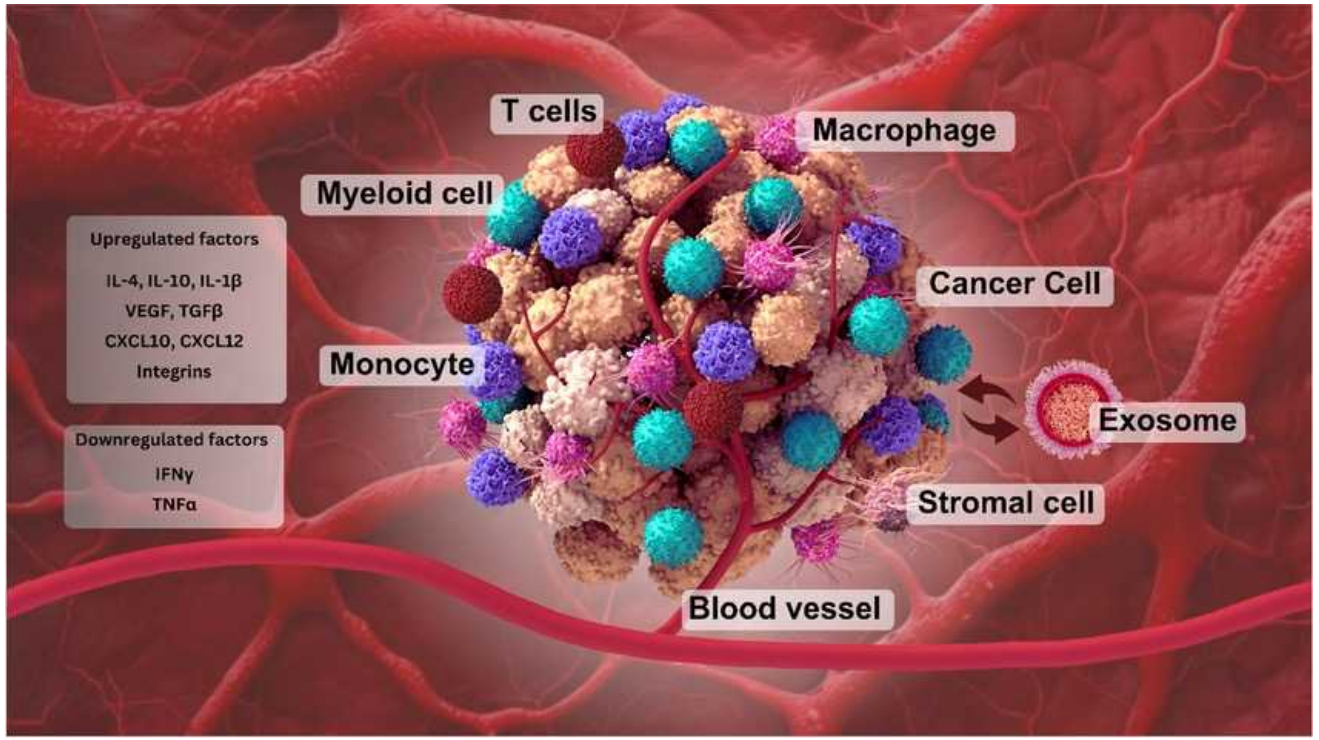
KESONOTIDE

Inflammation is one of the hallmarks of cancer.

The inflammation is coming from the cancer cells themselves which are highly plastic, able to develop inflammatory cell-like functions.

This inflammation creates the cancer support cells known as the tumor micro-environment (TME) that:

- drives tumour growth, aggression and invasiveness
- suppresses the immune cells
- helps create drug resistance.



Kesonotide – aiming to be the first drug approved that blocks the inflammatory forces that cancer cells use to generate the TME cells driving cancer cell growth and spread.

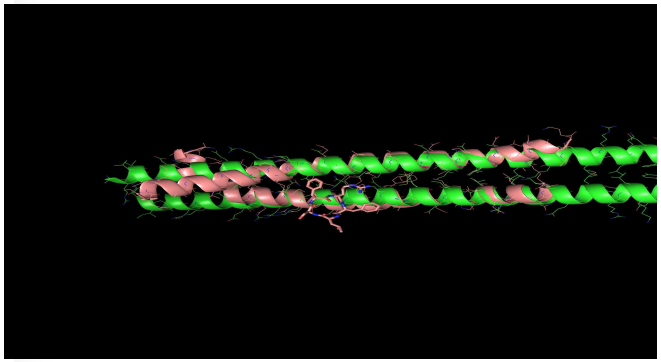
KESONOTIDE

A new class of anti-inflammatory drug designed to block **multiple structural proteins** from responding to **multiple inflammatory proteins (ligands).**

Kesonotide is a cyclic peptide designed to block major inflammatory proteins such as hGIIA from activating structural proteins including vimentin, integrins etc and growth receptors such as EGFR.

Kesonotide is able to be multi-functional because it has been designed to bind to so-called *promiscuous ligand-binding sites* on a range of proteins, blocking their ability to respond to multiple inflammatory proteins (ligands).

Kesonotide is intended to deprive cancer cells of their ability to grow, to be aggressive and to invade.



Kesonotide binds to vimentin, the component in Type 3 intermediate filaments, at the site where the protein is activated by various signals, effectively blocking vimentin's ability to create the TME.

KESONOTIDE

A new class of anti-inflammatory drug being tested first in cancer.

Kesonotide is the subject of multiple PCT patent applications

A preliminary first-in-human study has already been completed

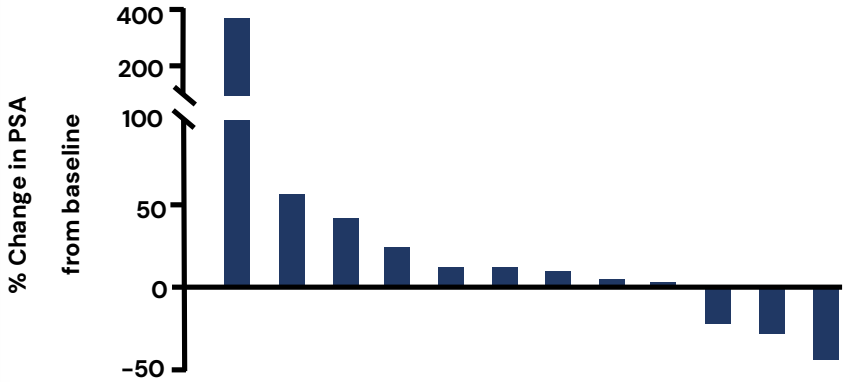
Location: Liverpool Hospital, Sydney

Approval: Based on potent anti-cancer effect against human prostate cancer cells in xenografted mice and no demonstrable toxicity in mice, rats and dogs

Funding: Prostate Cancer Foundation of Australia

Participants: 12 patients with late-stage prostate cancer

Treatment: Oral capsules. 10 mg/day for 6 weeks



Results

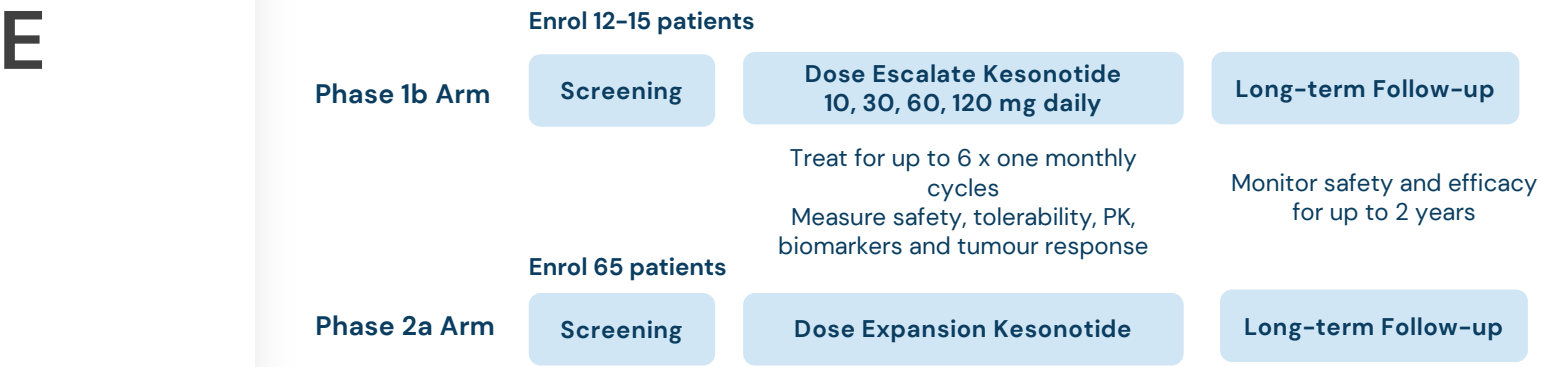
Confirmed:

- Oral bioavailability
- Safety/tolerance over 2 treatment cycles
- Notable PSA outcomes in some patients despite limited treatment

KESONOTIDE

ADVICE Phase 1b/2a clinical study

(status: commenced)



The clinical trial targets patients with advanced disease who are progressing on standard of care (SoC) treatments.

- **Phase 1b arm** will recruit patients with lung, prostate, breast, ovarian, pancreatic, large bowel and brain cancers.
- **Phase 2a** will focus on selecting the 2-3 cancer types showing the best responses.
- The **adaptive trial design** allows for flexible expansion based on **clinical responses,** facilitating the most effective application of the treatment and the fastest path to regulatory approval.

ADVICE is the first known clinical trial of a drug specifically designed to kill both cancer cells and their supportive cells, potentially overcoming resistance and enhancing therapeutic outcomes.

LYTAP Drug

Novel Targeted Protein Degradation

LYTAPs (Lysosomal Targeting Peptides) are a promising new class of anti-cancer drug being developed by Filamon and that are proprietary to Filamon.

LYTAP drugs are part of a new wave of drugs under development called ***targeted protein degraders.*** These drugs hijack the cell's natural protein-disposal system to destroy specific target proteins that drive cancer growth.

Targeted protein degraders are a response to the ready ability of cancer cells to mutate drug targets within the cancer cell, rendering the cancer cell resistant to the drug.

One such drug target is the **Epithelial Growth Factor Receptors (EGFR),** a potent driver of inflammation and growth of epithelial cancers (breast, lung, large bowel, prostate, brain etc).

EGFR inhibitors are a major form of anti-cancer therapy for such cancers but enjoy limited success because of the ability of the cancer cell to mutate its **EGFRs**, typically rendering EGFR inhibitors ineffective after ~12-15 months.

Targeted protein degradation offers a way to eradicate the target protein regardless of its mutational status.

LYTAP Drug

Lysosome Targeting Peptides

LYTAP drugs are derived from **kesonotide.**

The two drugs share an ability to block inflammatory proteins binding to proteins involved in cell growth, including **EGFR.**

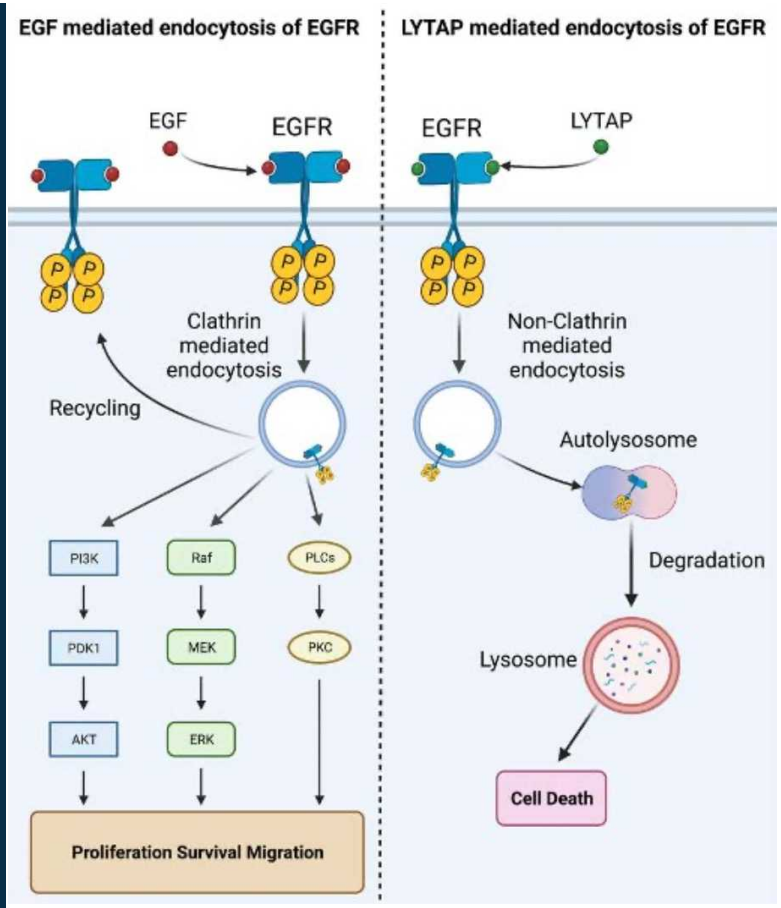
LYTAPs were created by modifying **kesonotide** so that instead of just blocking EGFR, the EGFR is driven inside the cancer cell to be degraded the the lysosomes.

This action gives **LYTAP** drugs the ability:

- to work independently of EGFR mutations
- to lower EGFR recycling by ~99%.

LYTAPs are small molecules dosed orally.

The **LYTAP program** is at the stage of lead candidate optimization.



BETA-TT17

Designed to boost the effectiveness of immune checkpoint inhibitor treatment

Immune checkpoint inhibitors, despite their early promise, remain disappointing for most cancer patients. Good response rates of 25-40% in some cancers such as lung cancer and melanoma are offset by response rates of up to 15% in the majority of cancer types.

Exhaustion of the cytotoxic T cells (CD8+ T cells) in tumours is considered a primary reason why market-leading PD-1 inhibitors, Keytruda and Opdivo, fail to provide a durable response in the majority of cancer patients. Reversing this exhaustion is a major area of current research activity with the aim of boosting the effectiveness of these and other forms of immunotherapy.

One area of major interest in the field in the last 2 years is so-called **bispecific antibody drugs** where a VEGF inhibitor is combined to a PD-1 inhibitor. The rationale being that the endothelial cells lining the blood vessels inside a tumour secrete inflammatory proteins that suppress T cell function. With endothelial cell function driven by the protein, VEGF, a VEGF inhibitor offers the prospect of reversing T cell exhaustion.

The promise of bispecific antibodies has led to significant M&A activity over the last 12 months:

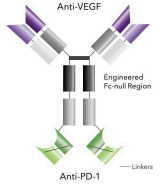
Bristol Myers Squibb `	USD 11 billion
Pfizer	USD 6 billion
Merck	USD 3.3 billion

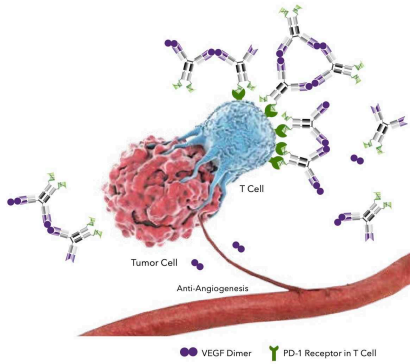
BETA-TT17

A unique oral **cytotoxic/ anti-VEGF/ anti-PD-1** drug

Bispecific antibodies

- Require 2-weekly IV injections
- Have significant side-effects
- Expensive to manufacture





BETA-TT17

Multiple advantages over bispecific antibody drugs

Filamon believes **BETA drugs** are a breakthrough in:

- reducing PD-1 expression by T cells
- re-activating $CD69^{+}$ $CD8^{+}$ cytotoxic T cells
- inhibiting all major genes associated with angiogenesis including VEGF, VCAM-1 and ICAM-1
- being directly cytotoxic to cancer cells via MAPK inhibition.

BETA-TT17 also is a small molecule:

- readily absorbed orally
- offering very low manufacturing costs.

STATUS. BETA-TT17 currently is undergoing pre-clinical studies to make the drug clinic-ready in early-2027.

Filamon Ophthalmology

BETA eyedropper and oral dosage forms for the treatment of age-related macular degeneration (AMD)

Presentation – October 2025

Age-related macular degeneration (AMD) is the major cause of loss of vision in older people, affecting an estimated 200 M people globally.

AMD is a neurodegenerative disease affecting the nerve fibres in the macula region of the retina, the macula being responsible for central vision and fine detail vision.

AMD is linked to an increased risk of developing Alzheimer's Disease and the two conditions share common pathological features including misfolded proteins, the accumulation of protein aggregates, and protein prion behaviour.

AMD begins as a degenerative condition that in its later stages can become inflammatory involving the growth of abnormally leaky blood vessels that result in oedema.

The outcome of these degenerative and inflammatory disease processes is disruption of the photoreceptor cells (nerve cells).

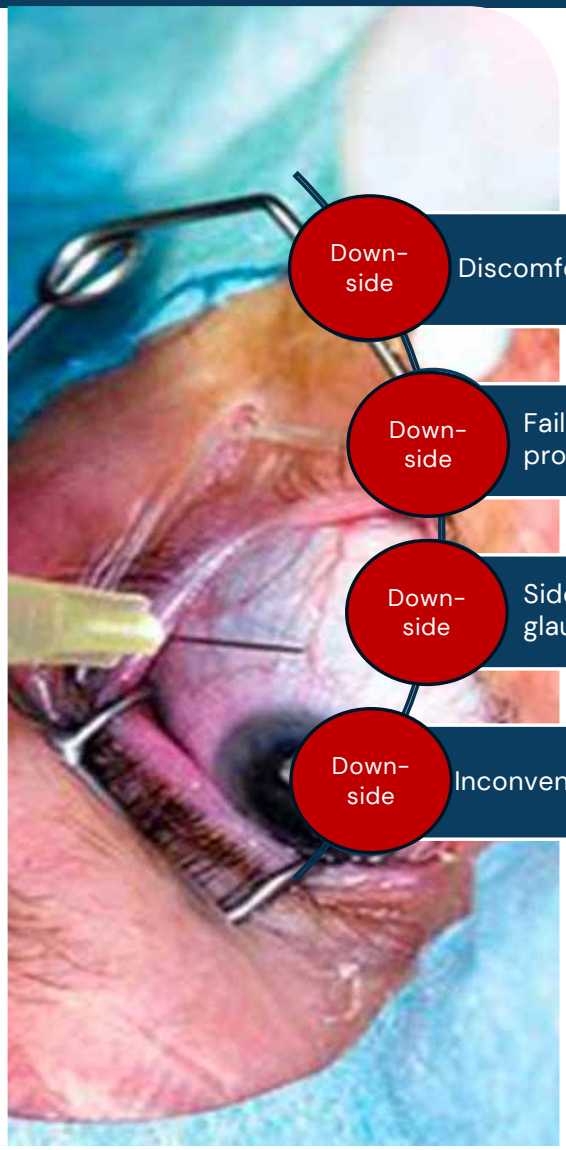
Four stages of the disease are described.

- **Mild dry AMD** - mild blurriness and some reading difficulties
- **Intermediate dry AMD -** greater blurriness and difficulty with low lighting
- **Advanced dry AMD** (also known as geographic atrophy) – permanent loss of central vision
- **Wet AMD** – the most advanced stage of AMD leading to permanent loss of central vision. Approximately 15% of AMD cases advance to this stage.

The rationale of current treatments of wet AMD is the use of antibodies that block the action of VEGF proteins.

The limitations of this approach are:

1. VEGF proteins are an important driver of abnormal angiogenesis (growth of blood vessels), but they are not the only ones. Proteins such as VCAM-1 and ICAM-1 are equally important and also need to be blocked.
2. Angiogenesis is not the only function taking place in the disease process. A wide range of inflammatory and immune responses involving dozens, possibly hundreds of inflammatory genes are thought to be involved and need to be addressed.



Down-side: Discomfort and anxiety associated with eyeball injection – high dropout rate

Down-side: Fail to work in one-third patients and slows down, not stops, disease progression in the others

Down-side: Side-effects include pain, bleeding into the eyeball and increased risk of glaucoma

Down-side: Inconvenience and significant cost of 1-2 monthly visits to an ophthalmologist

BETA-TT8

Aim is to develop a drug that compared to current treatment offers the potential for

- a clinical response in more patients
- a more potent and more durable response
- a safer, better tolerated treatment
- a more patient-friendly treatment
- a more cost-effective treatment.

BETA-TT drug family

- Unique allosteric modulators of the MAPK/ERK/JNK signaling pathway
- Confirmed inhibitors of 80 of estimated 300 pro-inflammatory genes believed involved in the AMD process
- Inhibit the primary genes believed involved in the wet AMD disease process
  VEGF-A, VEGF-B, VCAM-1, ICAM-1, IL-1b, IL-6, CCL20, CXCL-1, -3, -8)
  Note: Aflibercept (Eylea) inhibits three gene products only: VEGF-A, VEGF-B, placental growth factor
- Outperformed Eylea in animal model of wet AMD by a factor of 3*.
  (* Data on file)

BETA-TT8

- Small molecule
- Readily (45%) orally bioavailable
- Not susceptible to metabolism; extended half-life of >9 hours
- Readily crosses both blood-brain barrier and blood-eye barrier
- Well-tolerated over 14-days testing using estimated therapeutic dose.

Oral BETA-TT8

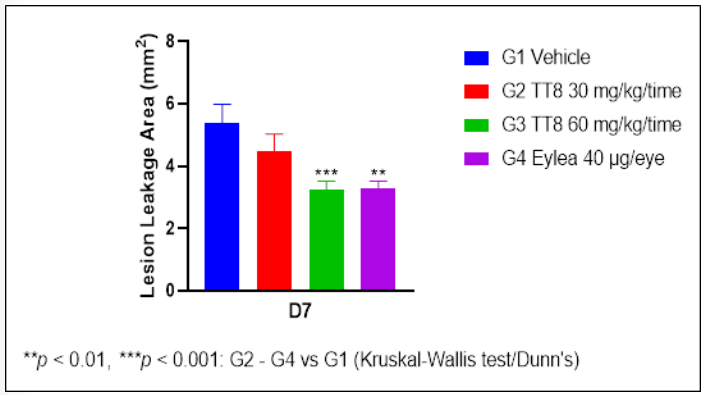
A small molecule offering a self-administered treatment that:

- avoids the anxiety, discomfort and side-effects associated with eyeball (intravitreal) injections
- avoids the inconvenience and cost of regular, rest-of-life clinic visits
- raises the likelihood of greater treatment compliance with better long-term outcomes for both the patient and the insurer

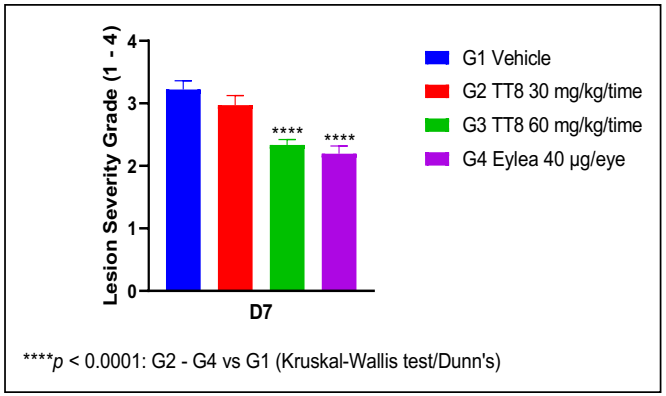
- **AND POTENTIALLY WORK AT LEAST AS WELL AS CURRENT TREATMENTS**

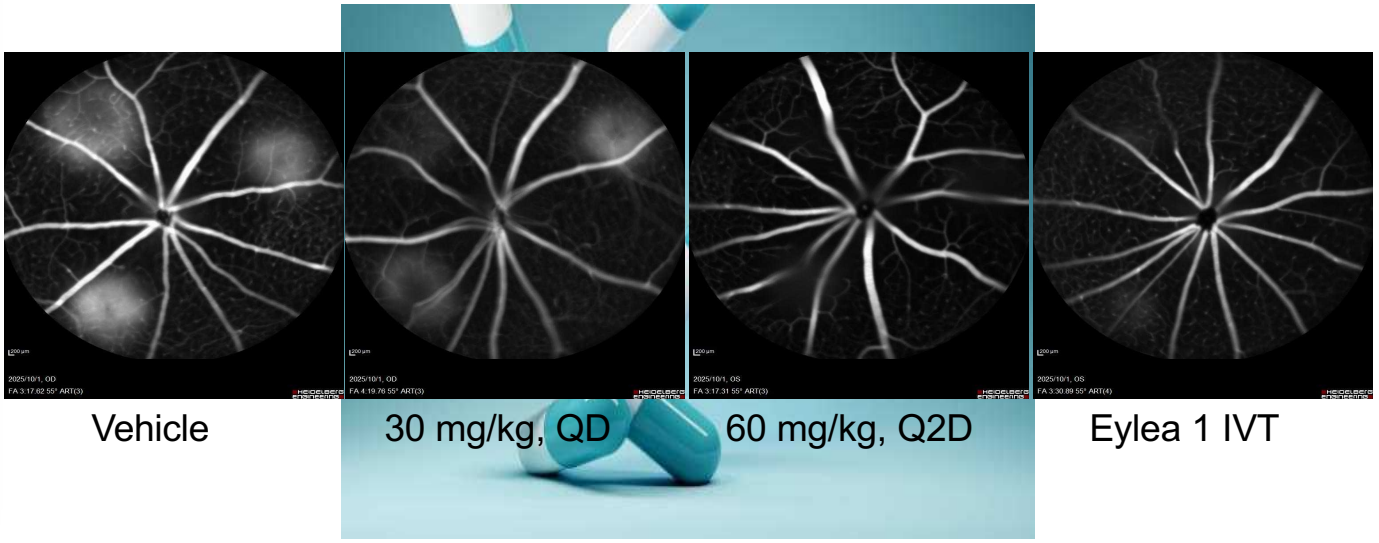
Leakage measurement - Fundus Fluorescent Angiography

Area



Score (see table)





Vehicle | 30 mg/kg, QD | 60 mg/kg, Q2D | Eylea 1 IVT

Filamon Neurology

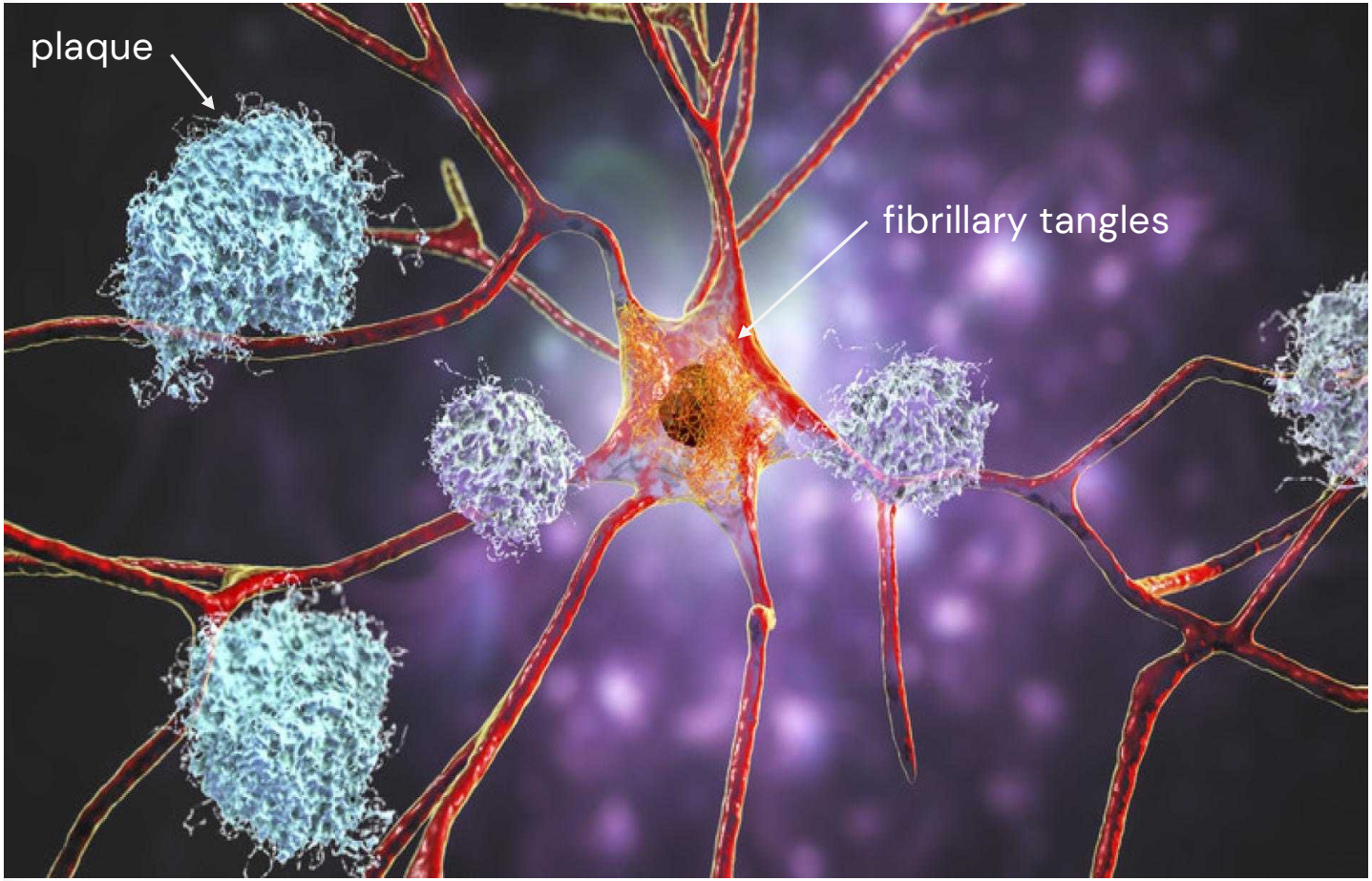
ALPHA -D Series A potential answer to the problem of toxic oligomers in neurodegenerative diseases

Presentation – October 2025

Most neurodegenerative diseases are characterized by particular proteins in the brain becoming ‘sticky’ and clumping together, leading to large aggregates inside cells known as **fibrils (**forming **fibrillary tangles)** or large aggregates between cells (known as **plaques**).

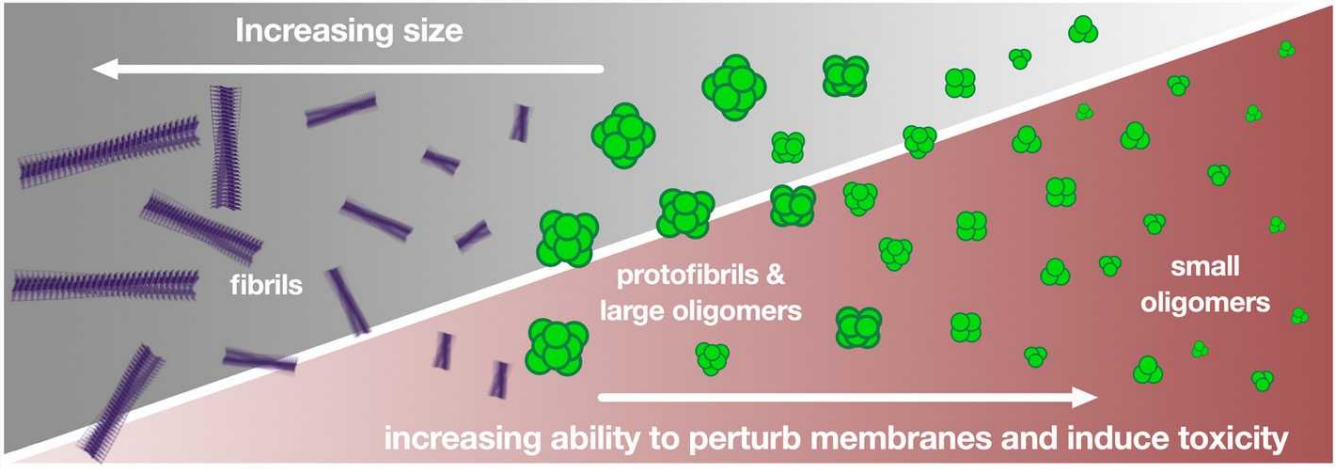
Protein aggregates are toxic to brain cells, physically blocking synaptic transmission leading to loss of memory and motor function before damaging cell structure sufficiently leading to cell death.

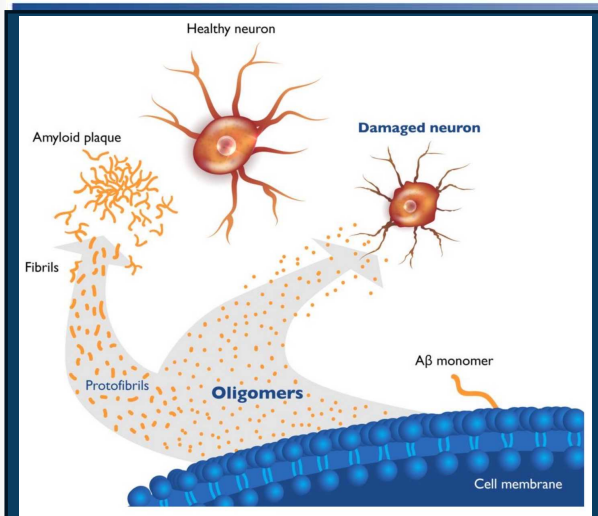
Drug discovery in the field to date has focused on the large, readily visible protein aggregates, a strategy increasingly coming to be accepted as misplaced.



Evidence now points to brain toxicity being more associated with sub-microscopic aggregates known as oligomers rather than the larger, more visible aggregates. Large aggregates such as plaques are increasingly now regarded as stable structures causing little toxicity. The smaller the aggregate, the more destructive it is because of its greater solubility and ability to penetrate cell membranes.

Aggregation is a step-wise process. It begins with single molecules of protein (known as monomers) clumping together to form structures known as oligomers. Oligomers begin with 2-4 monomers, gradually accumulating more monomers (up to ~ 50) until they starting re-organizing into protofibrils and then fibrils and then into very large aggregates such as plaque.





Oligomers drill holes in the cell membranes of brain cells, initially interfering with cell function, but eventually killing the cell.

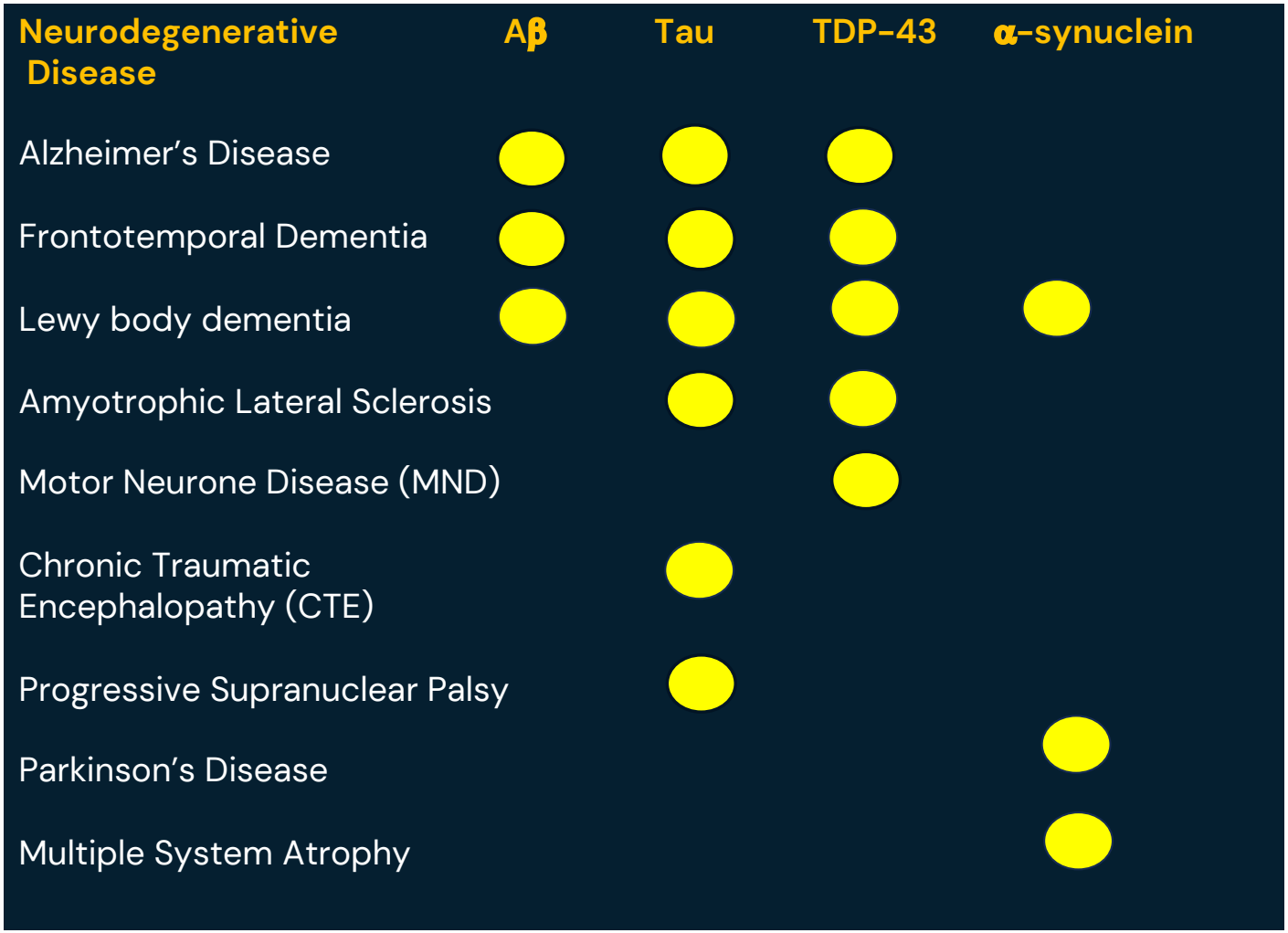
The smaller the size of the oligomer, the more able it is to penetrate the cell membrane, and therefore the greater its toxicity.

Neurodegenerative disease is marked by the persistence of the highly toxic oligomers in the brain, with their progression to the less toxic, larger aggregates delayed.

Each form of neurodegenerative disease is characterized by the aggregation of four different types of proteins

- Aβ
- tau
- TDP-43
- α-synuclein.

The involvement of multiple types of aggregates in the three main forms of dementia and ALS suggest that successful treatment of these conditions is going to require a multi-pronged approach to the aggregation of up to 4 different protein types.

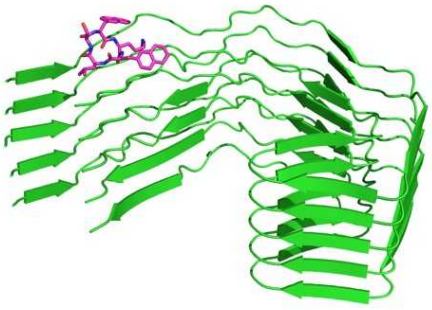


ALPHA-D series

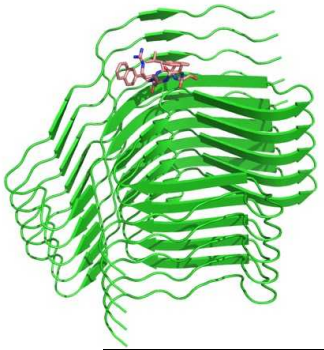
Introducing a new concept in the treatment of neurodegenerative disease.

A drug with a blend of two unique mechanisms of action:

- accelerating the progression of small oligomers to the fibrillar state
- Only known drug to bind to oligomers of Aβ, tau, TDP-43 and α-synuclein, providing the potential for a single drug effective across a wide range of neurodegenerative diseases.



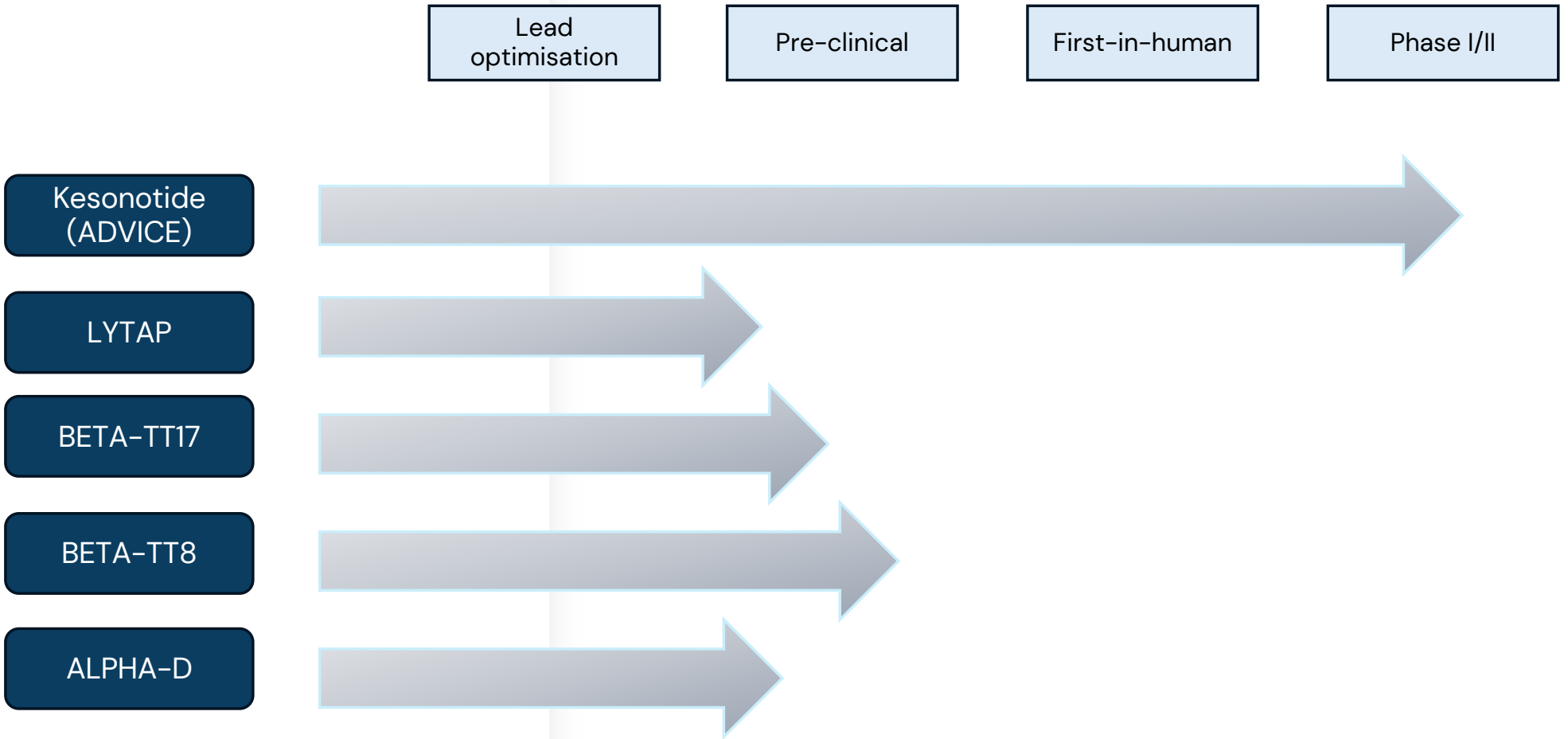
ALPHA-D1 drug binding to tau



ALPHA-D1 binding to TDP-43

ALPHA-D series drugs physically promote the aggregation process, accelerating the transition of misfolded, 'sticky' proteins to the fibrillar state within minutes, an action designed to deplete the brain of the highly toxic oligomers and convert them into considerably less toxic, more stable large aggregates.

ALPHA-D series drugs are designed to protect brain cells against toxic oligomers of the four main protein varieties involved in neurodegenerative disease.


Lead optimisation
Pre-clinical
First-in-human
Phase I/II
Kesonotide (ADVICE)
LYTAP
BETA-TT17
BETA-TT8
ALPHA-D

Filamon

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