ASX / Media Release
3 November 2025

Exenatide Shows Additional Positive Benefits in Alzheimer's Disease Brain Model

Key Points

- **Previous studies with Exenatide in the ADBrain™ model showed significant improvements in neuronal cell survival under conditions of oxidative stress and ferroptosis[1]**
- **New results indicate Exenatide confers a 40% reduction in neurofilament light chain expression, which is a well-accepted measure of neuronal cell damage**
- **Exenatide may mitigate early neurodegenerative processes driven by a reduction in neuroinflammation, a hallmark of Alzheimer's Disease (AD) progression**
- **Material uncertainty whether Tessara collaboration will continue if two of the existing Directors are removed at the upcoming General Meeting, where the Board of Invex recommends shareholders vote against all resolutions[2]**
- **Shareholders are urged to vote by attending the Meeting in person or by returning a completed proxy form no later than 4:00 pm AWST on Saturday 8 November 2025**

Invex Therapeutics Ltd (**Invex**, **ASX:IXC,** or **the Company**) a biopharmaceutical company focused on the development and commercialisation of Exenatide for neurological conditions relating to raised intracranial pressure (ICP), today provides the results of the additional research collaboration with Tessara Therapeutics (Tessara). The new analysis further assesses Exenatide in Tessara's ADBrain™ model, which has previously shown that Exenatide significantly improves neuronal cell survival under conditions mimicking Alzheimer's Disease (AD).

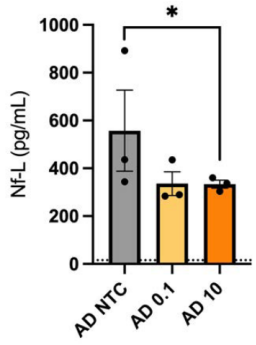
The use of GLP-1 receptor agonists (a class of drugs that includes Exenatide) in the treatment of AD offers significant promise, with two large Phase 3 clinical trials (EVOLVE/EVOLVE+) expected to report data later in 2025.[3] According to the Alzheimer's Association, GLP-1s may also have effects on the brain and nervous system by reducing brain inflammation, improve blood vessel health and neuroprotective effects.[4] These findings have opened the door to exploring whether GLP-1s could be repurposed to help treat or prevent AD. Invex believes the collaboration with Tessara will offer new and important insights into the role of Exenatide in AD, leading to new potential intellectual property and value accretion to the portfolio.

[1] See ASX announcement dated 16 December 2024
[2] See ASX announcement dated 8 November 2025
[3] https://pubmed.ncbi.nlm.nih.gov/39780249/
[4] Alzheimer's association analysis

In the recent set of analysis, the effects of Exenatide on neuroinflammatory cytokine release and glucose metabolism was assessed in the ADBrain™ model. In addition, the effect of Exenatide on neural networks such as network density, branch length, and number of branches was explored with the effect of Exenatide on AD biomarkers, namely amyloid-beta (AB) and phosphorylated Tau (PT).

Exenatide reduced secreted neurofilament light chain (Nf-L) levels in ADBrain™ microtissues by approximately 40%, as shown below at 0.1 µM and 10 µM concentrations. Elevated Nf-L is a marker of cell stress and a putative biomarker used in blood-based bioassays to detect AD early in its progression. This result is consistent with Exenatide having a potential therapeutic benefit early in the disease process. In addition, Exenatide showed reductions in pro-inflammatory cytokine expression, notably IL-6 and IL-8. Neuroinflammation is a hallmark of a number of neurodegenerative diseases, including AD.

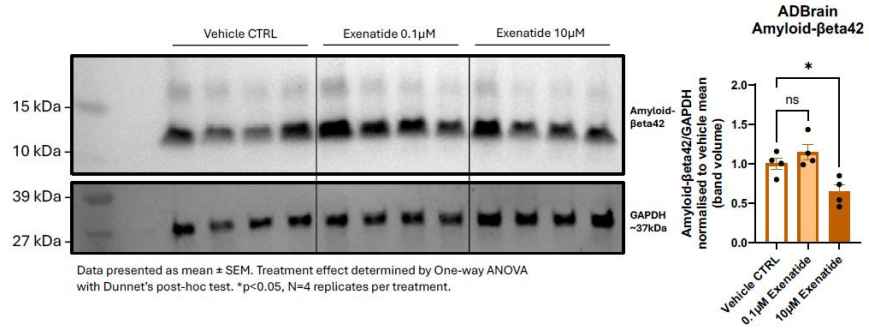
Effect of exenatide on Nf-L release into conditioned media of ADBrain



Levels of cell secreted Nf-L in ADBrain micro-tissues following 7-day exposure to 0.1 µM exenatide, 10 µM exenatide or basal non treated control (NTC). *p<0.05

Although 10µM Exenatide was associated with a 35% decrease in AB burden, needs to be further interrogated as the decrease in AB in micro-tissues was concomitant with an increase in GAPDH, the housekeeping protein used to normalise the experiment, as shown below. Previous studies have indicated that Exenatide can reduce AB deposition, accumulation, and toxicity in animal models. This finding will be investigated further.

Effect of exenatide on amyloid beta (Aβ) biomarkers in ADBrain

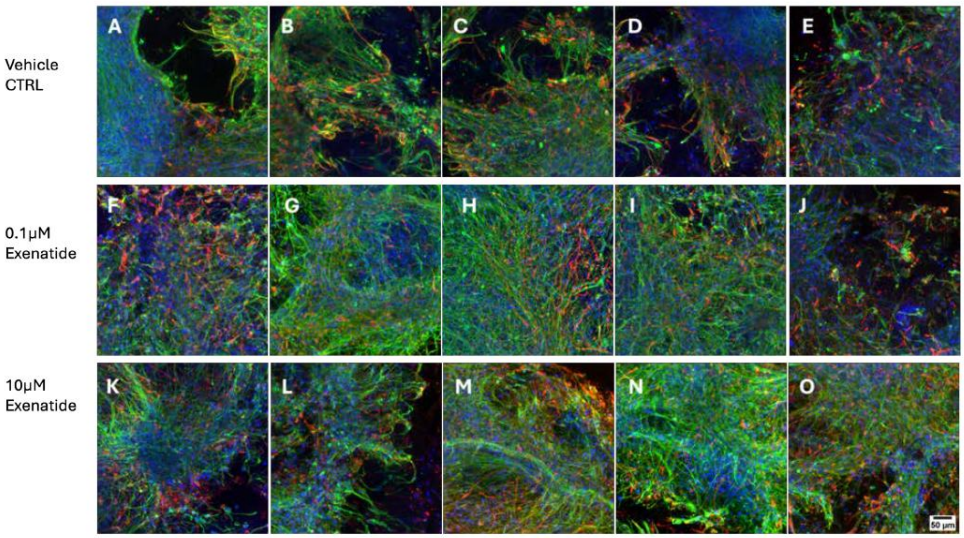


Measurement of Aβ in ADBrain following exposure to exenatide. Western blot and matching graph of densiometric quantification are shown. Data is expressed relative to β-actin. Data shown are mean values +/- SEM.

Exenatide did not affect PT levels in ADBrain™ under the conditions tested. Exenatide did not significantly influence insulin-dependent glucose uptake in the ADBrain™ model under the tested conditions.

Although the analysis algorithm used by Tessara did not find any overall increase in the number of networks associated with Exenatide treatment, increases in network complexity measures such as neural branching, number of junctions and average branch length all suggest that Exenatide may have a positive effect on neuronal health in Tessara's ADBrain™ model, even at the relatively low dose of 0.1 µM of Exenatide and 7 days exposure, as shown below.

ADBrain microtissues with exenatide treatment



β3-Tub/GFAP/DAPI **immunofluorescent staining.**
5 replicate representative images

Confocal images of ADBrain micro-tissues treated with exenatide and stained with specific antibodies to β(III)-tubulin (green), GFAP (red) and nuclei (blue). Vehicle Control treated micro-tissues **(A-E)**, 0.1µM exenatide treated microtissues **(F-J)**, and 10 µM exenatide treated microtissues **(K-O)**. Imaged on a Leica SP8 confocal microscope with 20x air objective. Max. projection images from 175µM 3D stacks (2.5 µM step size, 70 images per stack), 1024 x 1024 resolution (581 x 581 microns), n=5 biological replicates per treatment.

Next Steps

Gene expression analysis remains pending, but the planned comparison between ADBrain™ and healthy ArtiBrain tissues will be critical in understanding the broader transcriptomic impact of Exenatide, potentially unveiling pathways involved in neuroprotection and synaptic plasticity.

The research collaboration with Tessara is being undertaken under a Research Collaboration Agreement between the Company and Tessara, which was entered into by the parties in August 2024 (Tessara Collaboration Agreement). The Board is pleased that the collaboration relationship, which was established and continues to be managed by Directors David McAuliffe and Thomas Duthy, has produced encouraging results to date and potential new mechanisms of the effect of Exenatide in AD.

Based on those results, and the potential for the collaboration relationship to yield new and significant intellectual property for the Company, the Directors intend to continue advancing the

research collaboration relationship with Tessara, on the basis that it would be in the best interests for Invex to continue developing and driving value in its intellectual property portfolio relating to Exenatide.

However, the Board is cognisant that, should the composition of the Board materially change following the general meeting on 10 November 2025 (at which shareholder requisitioned resolutions[5] to remove Directors David McAuliffe and Thomas Duthy will be put to shareholders), there is a material risk that the Tessara Collaboration Agreement will be terminated and the research collaboration relationship will not proceed. This is because:

- the relationship with Tessara has been managed by Directors David McAuliffe and Thomas Duthy and, if they are removed as Directors, there is no available successor to step into their roles to continue management of the Tessara relationship (it being noted that the requisitioning shareholder has not nominated any replacement directors and accordingly it is not clear who will be appointed to the Board to make up the minimum 3 directors, should Directors David McAuliffe and Thomas Duthy be removed); and

- for the same reasons above, it is not clear what the strategy of the Company will be following the Board changes, and in particular whether or not the new management team and Board will seek to generate value from the Company's intellectual property portfolio relating to Exenatide and/or the Tessara Collaboration Agreement, or if the Company will be steered in a different direction altogether.

Accordingly, shareholders should note that, notwithstanding the potential for the Tessara Collaboration Agreement to create value for shareholders through enhancement of the Exenatide intellectual property portfolio, there is no certainty that the ongoing research collaboration relationship with Tessara will continue, or that if it does continue, that the Company will be able to effectively harness and monetise the new intellectual property generated from it.

Shareholders are reminded that the deadline for returning completed proxy forms for the shareholder requisitioned meeting is **4:00 pm AWST on Saturday 8 November 2025**. The meeting will be held in person at **4:00 pm AWST on Monday 10 November 2025 at** Level 29, Central Park Tower, 152-158 St Georges Terrace, Perth WA 6000 Australia.

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This release dated 3 November 2025 has been authorised for lodgement to ASX by the Board of Directors of Invex Therapeutics.

For more information, please contact:

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[5] The shareholder meeting has been requestioned by Celtic Capital Pte Ltd <Investment 1 A/C>

About Invex Therapeutics Ltd

Invex is a biopharmaceutical company focused on the repurposing of an already approved drug, Exenatide, for efficacious treatment of neurological conditions derived from or involving raised intracranial pressure. Invex has trademarked its repurposed Exenatide as Presendin™. www.invextherapeutics.com.