BioSpectrum ASIA EDITION

Can Precision Antibodies Deliver a Safer Future for Alzheimer's Patients?

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With Phase 1b data on the horizon, ProMIS Neurosciences is advancing a new generation of precision therapies aimed at neutralising the toxic protein species believed to drive neurodegeneration.

Alzheimer's disease research has entered a pivotal new phase following the arrival of the first disease-modifying therapies targeting amyloid. Yet significant challenges remain, particularly around safety, efficacy, and the need for earlier intervention. Increasingly, researchers are turning their attention to toxic amyloid-beta oligomers—small, misfolded protein aggregates believed to trigger neurodegeneration long before plaques appear. **At BIO International Convention 2026 in San Diego, BioSpectrum Asia spoke with Neil Warma, CEO of ProMIS Neurosciences,** about the company's precision-targeting strategy, the promise of next-generation antibodies, and why selective engagement of toxic protein species could transform the treatment of Alzheimer's disease and other neurodegenerative disorders

Alzheimer’s research is entering a new era. What opportunities remain unexplored?

We are indeed in an exciting new era for Alzheimer’s disease research. The recent approvals of amyloid-targeting antibodies, following decades with no disease-modifying treatment options, have provided important clinical validation of the amyloid pathway. That said, new treatments are still needed to meaningfully improve patient outcomes, and the door is now open for a next generation of more precise therapies, ones with both improved efficacy and a better safety profile.

The most important opportunity ahead is selectivity: the ability to target specific drivers of disease while avoiding off-target binding that can pose serious safety risks. Current approved therapies bind amyloid plaques, but plaque binding has been associated with ARIA, a serious side effect encompassing brain swelling and microhemorrhages, which has meaningfully constrained real-world use. The evidence increasingly points to upstream, toxic forms of amyloid pathology as a primary disease driver, specifically amyloid-beta oligomers. These are small, soluble, misfolded aggregates of amyloid-beta believed to trigger synaptic dysfunction, neuroinflammation, and tau phosphorylation long before plaques accumulate. While other programs have attempted to target oligomers, few therapies have been designed with the degree of selectivity that ProMIS delivers to avoid off-target binding, and that remains a significant gap.

Greater precision also opens the door to earlier intervention through different modalities. Vaccine-based approaches, for example, that generate an immune response selective for toxic oligomers rather than broadly targeting amyloid species, could potentially allow treatment before significant neuronal damage has occurred, transforming AD from a disease we slow to one we may one day prevent.

Finally, Alzheimer's is a multifactorial disease. As the field continues to uncover the interconnected roles of amyloid, tau, and neuroinflammation, combination therapy approaches will likely be explored. The progress we are seeing today is laying the groundwork for that next chapter.

Why focus specifically on toxic misfolded proteins?

As previously mentioned, small, misfolded protein clusters of amyloid-beta form long before plaques accumulate in AD patients, and before symptoms appear. They behave like seeds, aggregating into toxic forms, spreading through the brain, and recruiting more amyloid-beta protein to misfold, driving neurodegeneration. The goal of ProMIS is to selectively target these key drivers and slow disease progression while minimizing off-target effects associated with plaque clearing, such as brain edema and microbleeding (ARIA). This is the rationale behind our approach at ProMIS and the basis of our lead candidate, PMN310, a humanized monoclonal antibody currently being studied in PRECISE-AD, our Phase 1b clinical trial in patients with early Alzheimer's disease.

Toxic misfolded proteins, in Alzheimer's and other neurodegenerative diseases, are structurally distinct from their healthy counterparts. They expose unique, disease-specific surface regions called epitopes that do not exist on the properly folded form of the same proteins. That distinction is very important therapeutically, because it means you can, in principle, design an antibody that binds to the toxic form while leaving the healthy protein intact.

ProMIS's EpiSelect™ platform integrates physics, biology, and computational modeling to predict and identify those disease-specific epitopes with precision. We then generate therapeutic antibodies against these epitopes to selectively target the toxic misfolded species. This selectivity is what we believe may ultimately differentiate next-generation therapies from those that came before, in both efficacy and safety.

How could next-generation antibodies improve patient outcomes?

The most meaningful improvements will come from antibodies that are more precise, safer, and potentially deployable earlier in the disease course.

Current approved antibodies in Alzheimer's have demonstrated that you can slow cognitive decline by targeting amyloid, and that is an important proof of concept. However, the benefit has been modest, and the safety profile has been a challenge. ARIA, amyloid-related imaging abnormalities including brain swelling and microhemorrhages, occurs at significantly higher rates in patients treated with plaque-binding antibodies. This requires intensive MRI monitoring, creates practical barriers to treating broader patient populations, and is a particularly meaningful concern for patients with APOE4 genetic variants.

Next-generation antibodies designed to selectively target upstream pathological drivers, such as toxic oligomers, aim to improve safety profiles and potentially enable broader patient access. Our lead candidate PMN310, for instance, is engineered to selectively target amyloid-beta oligomers while avoiding binding to amyloid plaques and vascular deposits, a design choice we believe may meaningfully reduce ARIA liability.

Beyond safety, precision targeting offers the prospect of intervening earlier in the disease cascade, neutralizing the believed drivers of neuroinflammation and synaptic dysfunction before irreversible damage accumulates. That is the path toward meaningfully modifying disease, rather than slowing its later-stage progression.

What is needed to accelerate innovation in neurodegenerative diseases?

First, we need better knowledge of the biology. For too long, the field operated on hypotheses about which protein species were most pathologically relevant. While the evidence base for soluble toxic oligomers as primary drivers is now much stronger, we need to continue building the science, particularly around early disease mechanisms and patient stratification.

Second, in parallel with treatment development, we need better biomarkers to enable earlier detection, specifically the ability to identify individuals in the presymptomatic phase of disease before cognitive decline sets in. Advances in plasma-based biomarkers, including p-tau217, are promising in this respect, and several are being incorporated directly into our PRECISE-AD trial.

Third, we need capital and partnership models that are willing to fund the long arc of neurodegeneration research. The timelines are longer than in other fields, but the unmet need is enormous, with millions of patients affected by these conditions worldwide. At ProMIS, we are encouraged by the investment community's growing appetite for differentiated science, and we believe our upcoming Phase 1b data readout will be an important signal for the field.

Finally, we need regulatory frameworks that can keep pace with scientific innovation, enabling adaptive trial designs, accepting surrogate biomarker endpoints where appropriate, and supporting earlier access pathways for patients with no meaningful alternatives.

What milestone would most significantly advance the field?

A key milestone would be clinical results from any approach that demonstrates meaningful clinical improvement with a positive safety profile. That would fundamentally change the risk-benefit calculus for treating Alzheimer's disease, enabling earlier intervention and access to broader patient populations.

For us at ProMIS, data from our PRECISE-AD Phase 1b trial could validate the oligomer-selective approach as a biological strategy for the field to pursue. The first reporting on this data will be in early Q3 2026, when we present an interim analysis of blinded safety and biomarker trends, with top-line data to follow in early 2027. We are monitoring a panel of pharmacodynamic biomarkers associated with clinical outcomes, alongside rigorous ARIA monitoring. If we see biomarker signals consistent with target engagement and a clean safety profile, that would represent a preliminary proof of concept, not just for PMN310, but for the broader principle that precision targeting of toxic amyloid-beta oligomers is a relevant path forward.

Beyond Alzheimer's, demonstrating platform transferability across misfolded proteins would also be a major milestone. Validating the same precision approach across multiple disease targets would signal a fundamentally new paradigm for tackling neurodegeneration as a class.

What is the single biggest takeaway from BIO 2026 that will influence your company's strategy over the next 12 months?

What struck me most at BIO this year was the renewed conviction among investors and partners that differentiated science, not just fast science, is what the market is looking for right now. After a challenging few years for biotech capital markets, there is genuine excitement around companies with compelling data, clear mechanisms, and assets that address real unmet needs rather than offering incremental improvements on existing therapies.

That resonates deeply with where ProMIS sits today. We are at an inflection point, heading into a blinded interim readout in early Q3 and top-line data in early 2027, and the conversations at BIO reinforced that the investment community and potential strategic partners are paying close attention to the Alzheimer's space, particularly as the limitations of first-generation plaque-targeting antibodies have become clearer in real-world use.

The takeaway for our strategy is to stay focused on the science and the data, communicate with clarity and transparency, and continue building relationships with potential partners and investors who understand the differentiated opportunity in front of us. The appetite is there, and the next 6-9 months will be an important opportunity for ProMIS to demonstrate why precision targeting of toxic oligomers represents the next chapter in Alzheimer's treatment.