White Paper

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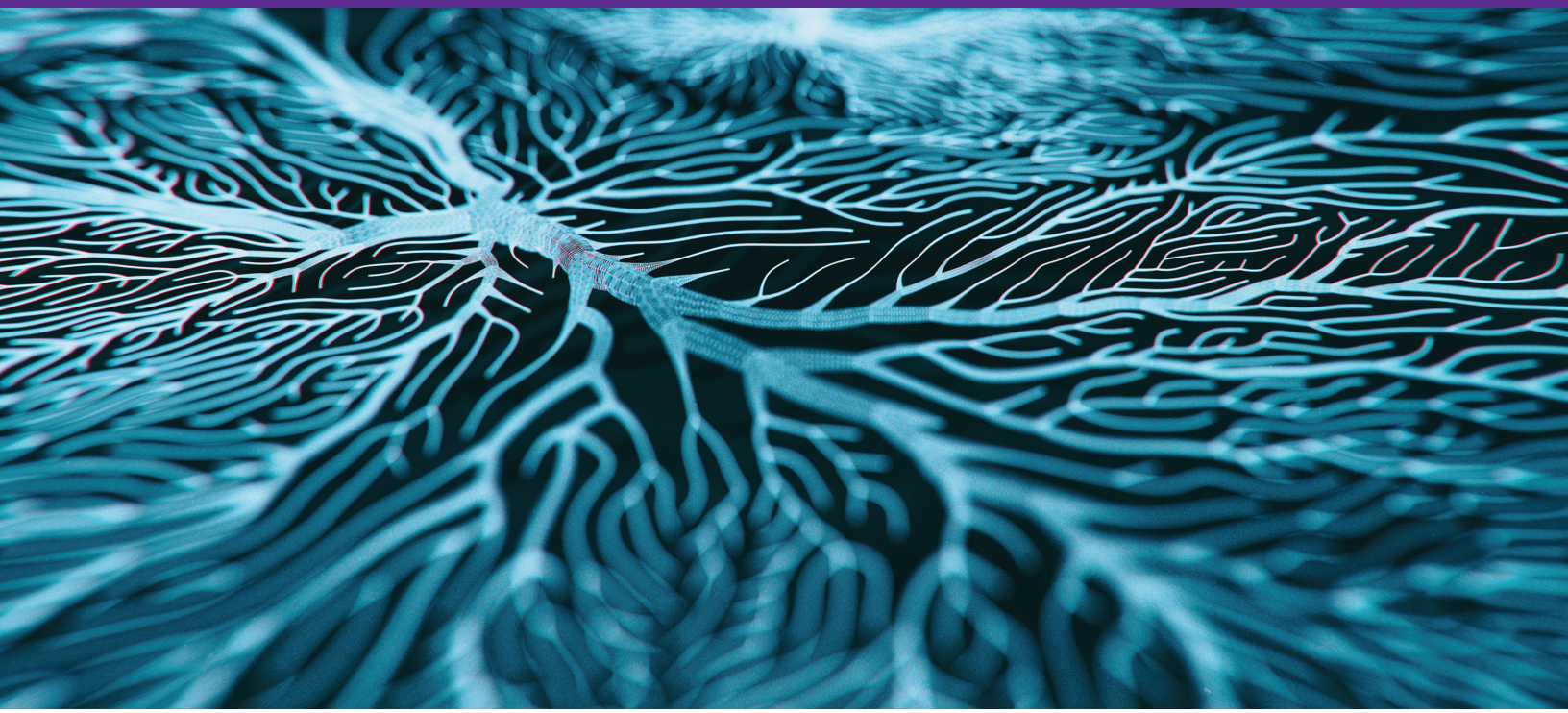

Neuroinflammation

Introduction

Neuroinflammation involves the activation of CNS-resident immune cells and infiltration of peripheral immune populations across the blood-brain barrier (BBB). Over the past few years, perception has shifted from neuroinflammation being a secondary phenomenon associated with disease to a recognized pathological driver in its own right. Yet only a few programs against neuroinflammatory targets have advanced far into clinical development. Most have yet to generate definitive positive efficacy data, complete target validation in human disease, and bridge the translational gap between preclinical models and patient biology.

Neuroinflammation itself is a broad term. It can initiate from acute CNS injury, autoimmune demyelination, aging, and chronic neurodegeneration. These contexts differ fundamentally in cellular drivers and therapeutic logic. The focus of this whitepaper is neuroinflammation in neurodegenerative disease, mainly Alzheimer's disease (AD), Parkinson's disease (PD), multiple sclerosis (MS), and amyotrophic lateral sclerosis (ALS). Rather than a comprehensive review, we present a reference point for those tracking the field, offering a structured perspective on the biological trends, select target classes, and therapeutic approaches that have gained meaningful traction.

As in our previous whitepapers, we complement the scientific perspective with a look at the commercial landscape, where interest is beginning to reflect the field's momentum. Sanofi's $470 million acquisition of Vigil Neuroscience and Lilly's commitment of up to $1.2 billion for Ventyx Biosciences are early signals of that shift.[1,2] Early-stage financing is picking up as well, spanning target-focused bets such as LRRK2 (Neuron23) and CSF1R (Elixiron) to broader, more exploratory plays on neuroinflammation in neurodegeneration.

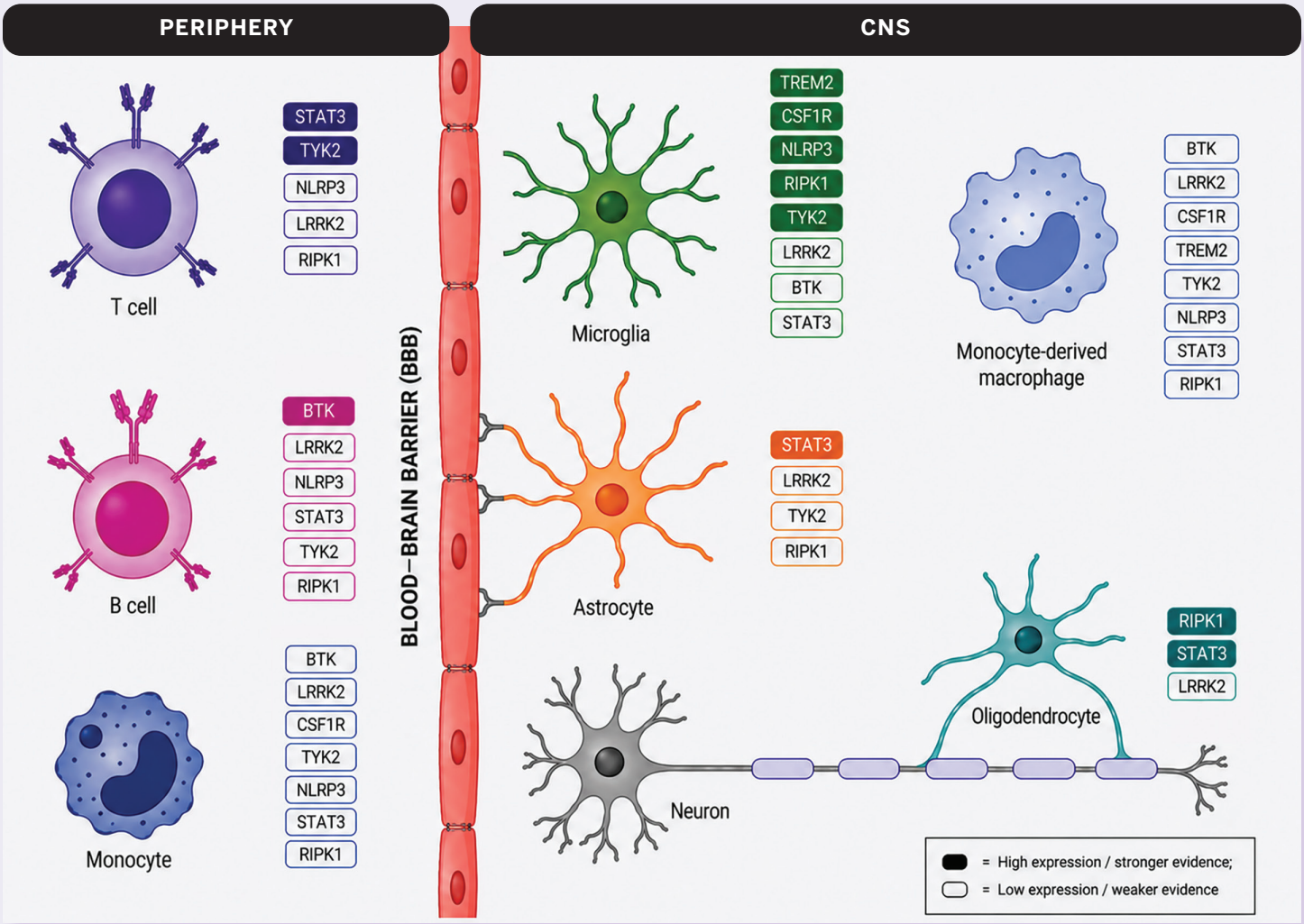

Figure 1. Neuroinflammation Targets Expression Across CNS-Resident Cells and Peripheral Compartment
The figure maps the cellular landscape of neuroinflammation across two anatomical compartments divided by the blood-brain barrier (BBB): the CNS (right), encompassing microglia, astrocytes, oligodendrocytes, monocyte-derived macrophages (MdMs), and neurons; and the periphery (left), comprising T cells, monocytes, and B cells. For each population, a curated subset of therapeutically relevant targets is displayed. The figure is not intended as a comprehensive expression atlas, but rather as a directional overview of targets with current drug development relevance in neuroinflammation. Filled labels denote high expression or stronger supporting evidence; outlined labels indicate moderate expression or more limited data. Expression tier assignments should be read as a qualitative guide and interpreted with appropriate caution given that the evidence base varies across targets and cell types.

BBB, blood-brain barrier; MdM, monocyte-derived macrophages; CNS, central nervous system.

Cell Types in the CNS

Throughout this analysis, neuroinflammation is treated as a two-compartment process: CNS-resident cell activity on one side, peripheral immune engagement on the other. We begin with the CNS, where the mechanistic picture is most developed, the bulk of current drug development is concentrated, and where glial cells sit at the center of the inflammatory response **(Figure 1)**.

Microglia

Microglia are the resident immune cells of the CNS and the long-thought primary drivers of neuroinflammation, functioning as the brain's first line of defense against pathogens, injury, and accumulating pathological protein aggregates. During neuroinflammation, microglia transition into reactive states and secrete pro-inflammatory cytokines to manage the insult. Typically, glia then return to a homeostatic state, allowing them to perform neuroprotective functions, including amyloid-beta (Aβ) clearance via phagocytosis, trophic support, and myelin debris removal essential for remyelination.[3] However, when glial activation goes unresolved, due to the constant presence of, for example, pathological proteins or cellular changes within the glia themselves, it can create a neurotoxic environment that exacerbates pathology across neurodegenerative diseases. This functional duality has direct implications for therapeutic strategy and for the question of timing, addressed later.

Given their principal role in neuroinflammation, microglia are also the most clinically tractable cell type in this space. Most advanced CNS-specific assets are predominantly linked to microglial function: TREM2 agonists and CSF1R inhibitors all act primarily through microglial modulation and represent the bulk of active programs in neurodegeneration, though other cell types also play pivotal functions in neuroinflammatory processes.

Astrocytes

Astrocytes are essential for neuronal survival and normal CNS homeostasis. Under neuroinflammatory conditions, they also undergo reactive transformation into functionally distinct states, neuroprotective or neurotoxic, with the balance between these states shaping disease trajectory. Microglia are involved in this process. In AD, for example, microglial release of IL-1α, TNF, and C1q drives astrocyte conversion toward neurotoxic states. In cyclical turns, reactive astrocytes reciprocally activate microglia, creating a self-reinforcing loop that amplifies Aβ deposition and tau propagation.[4,5]

Astrocytes are a compelling therapeutic target, but a difficult one. The population is highly heterogeneous, and intervening in the wrong subpopulation risks suppressing neuroprotective functions essential for tissue repair. Preclinical models compound the challenge. Rodent astrocytes differ from their human counterparts in morphology and gene expression, limiting translational confidence.[6] As a result, the astrocyte field remains largely preclinical, with target selection, disease context, and intervention timing having yet to be defined. Whether the lag in therapeutic targeting relative to microglia reflects greater intrinsic heterogeneity, technical barriers, or simply less research remains an open question.

Oligodendrocytes

Oligodendrocytes are the myelin-producing cells of the CNS. They are increasingly recognized as active participants in neuroinflammation. In several diseases, demyelination and oligodendrocyte loss precede overt neuronal dysfunction. Oligodendrocytes exhibit repair capacity through remyelination, driven primarily by the differentiation of oligodendrocyte precursor cells (OPCs).[7]

However, the field is moving away from treating any single glial cell type as a standalone target toward a more integrated view—one where microglia, astrocytes, and oligodendrocytes function as a co-regulatory system. The scientific evidence for this framework is building. In PD, all three glial cell types undergo reactive transformation that reinforces an inflammatory loop driving neurodegeneration.[8] In MS, reactive astrocytes that lose cholesterol-synthesizing capacity directly compromise oligodendrocyte function and remyelination,[9] while microglial state determines whether OPC differentiation is suppressed or promoted.[10]

Multi-cellular strategies that simultaneously modulate all three populations represent a compelling future direction, one that may prove essential in diseases where pathology is distributed across the entire glial network. Which disease contexts would benefit most is yet to be established.

CNS-Centric Targets

TREM2

TREM2 has emerged as one of the most established targets in neuroinflammation, underpinned by robust genetic evidence and a compelling mechanistic rationale. TREM2 is a sensor for damage-associated molecular patterns (DAMPs), such as Aβ. Its activation attenuates neuroinflammation by promoting clearance of aggregates and misfolded proteins, inhibiting tau hyperphosphorylation, and modulating microglial and macrophage polarization toward reparative phenotypes. However, its clinical development has been defined as much by high-profile failure as by forward momentum. Of 12 total assets across all stages, half are now inactive **(Figure 2)**. Six discontinued programs, split equally between preclinical and clinical discontinuations, reflect a target whose biology has proven harder to translate.

AL002, the first TREM2 agonist antibody to advance to phase II, failed in Alzheimer's trials in November 2024.[11] Despite confirmed target engagement, AL002 failed to meet the primary endpoint, showing no measurable impact on cognitive decline, functional outcomes, neuroinflammatory biomarkers, or brain amyloid load. ARIA-E (edema, 13.4%) and ARIA-H (microhemorrhages, 8.4%) added a safety liability that compounds the efficacy failure.[12]

Several things could explain AL002's failure. Its stalk-binding mechanism drove receptor internalization, removing surface TREM2 at the very moment of engagement. The subsequent reduction in soluble TREM2 (sTREM2) may have eliminated a species with independent neuroprotective function. Moreover, questions remain regarding the timing: early microglial activation is likely beneficial for the removal of Aβ plaques, possibly requiring patient stratification based on disease course and degree, whereas prolonged or excessive activation can aggravate neurodegenerative processes. Together, this represents a target with a likely narrow therapeutic window and highly complex biology that might require both industry and academia to revisit core assumptions before moving forward.

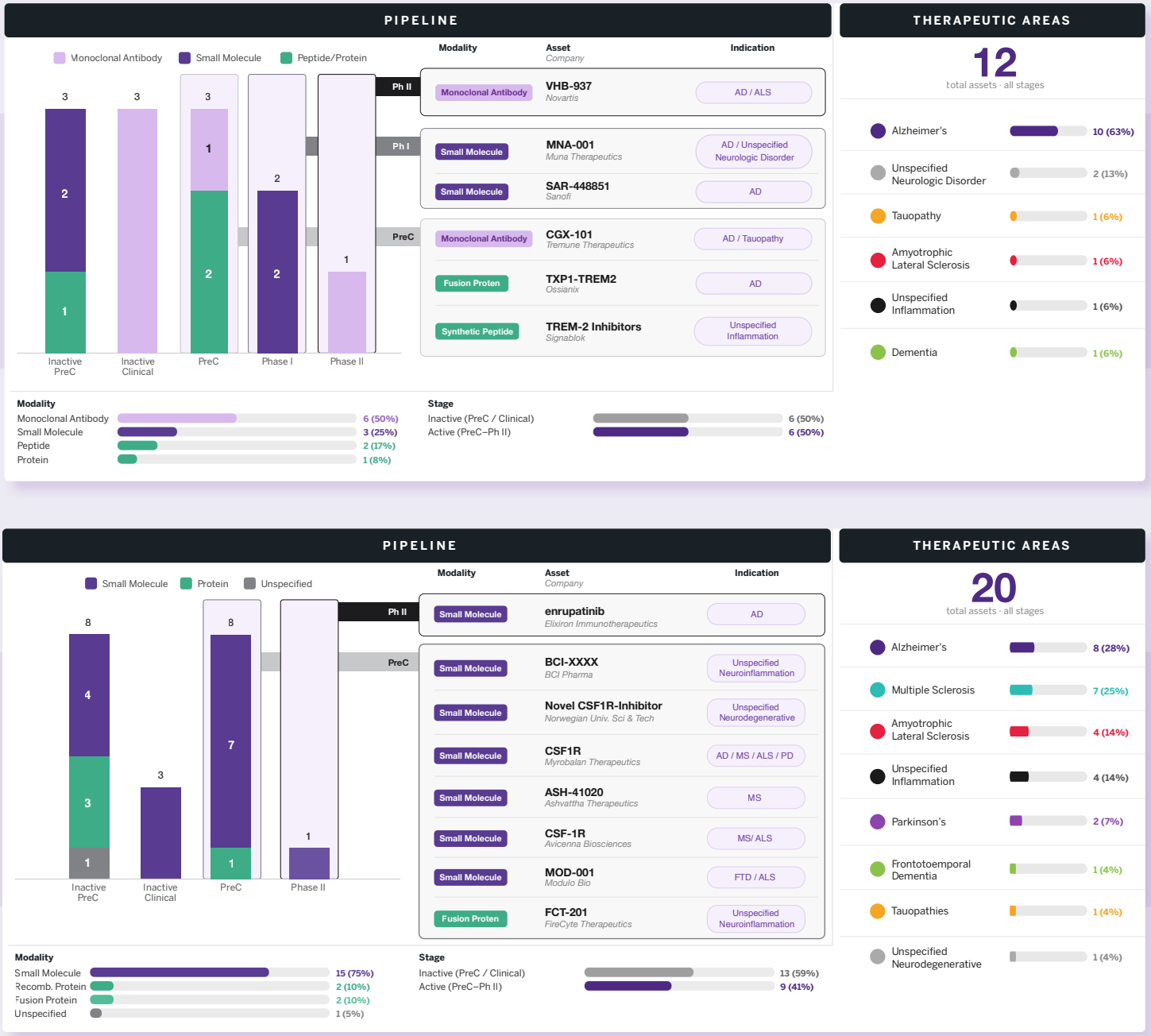

Figure 2. Drug Development Pipelines—CNS-centric Targets
The figure shows the current drug development landscape for TREM2 (top) and CSF1R (bottom) across pipeline stage, modality, and therapeutic indication. For each target, assets are stratified from inactive preclinical through the latest clinical stage, with individual programs annotated by modality, company, and primary indication. The proportion of active versus inactive programs and various modalities are summarized alongside each pipeline view. Indication frequency is displayed as an annotated bar chart in the right panel. Assets may span multiple indications; total indication counts may therefore exceed total asset counts. Pipeline is based on GlobalData information as of April 2026.

AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; ASO, antisense oligonucleotide; FTD, Frontotemporal Demetia; Gene Tx, gene therapy; MS, Multiple Sclerosis; PD, Parkinson's disease; PreC, preclinical.

Six active programs remain, covering AD, ALS, and unspecified neurological indications. The active pipeline reflects a deliberate pivot away from the mechanism that failed. Three programs are advancing with meaningfully differentiated approaches:

- **VHB-937 (Novartis, Phase II):** this is the most advanced program, targeting AD and ALS. Mechanistically, VHB-937 binds the IgSF domain rather than the stalk, stabilizing surface TREM2 without triggering internalization and uniquely producing dose-dependent increases in CSF sTREM2 alongside reductions in pro-inflammatory markers. This is the opposite pharmacodynamic profile to AL002 and the clinical attempt to test whether sTREM2 elevation is itself beneficial.[13,14]
- **SAR-448851/VG-3927 (Sanofi/Vigil, Phase I):** an oral small molecule acting as a molecular glue on membrane-bound TREM2, explicitly designed to bypass the sTREM2 sink.[15] phase I confirmed CNS penetration and ~50% sTREM2 reduction,[16] with phase II in AD planned. Sanofi's $470 million acquisition of Vigil in the summer of 2025, on the back of phase I data, signals strong internal conviction.[17]
- **MNA-001 (Muna Therapeutics/GSK, Phase I):** dimerizes TREM2 to promote activation without direct internalization. Topline Phase I data in early AD is expected in the summer of 2026,[18] with GSK's $35 million upfront and $147 million in milestones providing external validation of the program's differentiation hypothesis.[19]

The ALS readout of VHB-937 in H1 2026 will be the first test of whether the mechanistic corrections from AL002 hold in practice. The more consequential question of whether those corrections can restore efficacy in AD will take longer to answer. With VHB-937's AD trial only initiating enrolment in Q3 2025 and SAR-448851 yet to start phase II, a definitive read on the TREM2 hypothesis in AD may not arrive before 2027.

CSF1R

CSF1R is the master regulator of microglial survival and proliferation. Its dimerization after stimulation with CSF1 or IL-34 leads to activation of PI3K/Akt, JNK, and ERK1/2 pathways, promoting microglia proliferation and survival. CSF1R signaling also intersects with the TREM2/DAP12 complex, with both pathways converging on microglial functioning.[20] Consistent preclinical evidence indicates that downregulation of CSF1R is beneficial in suppressing chronically activated microglia across neurodegenerative disorders.[21] Of 22 pipeline assets, only one is in active clinical development, with three having been discontinued in the clinic **(Figure 2)**.

Sotuletinib (Novartis) was terminated in July 2024 after its phase II ALS trial, with the official reason stated as "assessment of potential benefit-risk from available data."[22] The Alzheimer's Drug Discovery Foundation, in their review, characterized Sotuletinib as a substrate for efflux transporters, potentially limiting its CNS utility.[23] Another asset, PLX-5622 (Plexxikon) did not advance beyond healthy volunteers. Instead, Plexxikon redirected its CSF1R efforts toward pexidartinib (PLX3397), a multi-kinase inhibitor for pigmented villonodular synovitis and additional oncology applications. Pexidartinib saw limited development in CNS indications, likely reflecting poor BBB penetrance, with CSF levels in non-human primates reported to be extremely low.[24] Edicotinib (JNJ-40346527, Janssen) targets the same kinases as pexidartinib but with greater selectivity toward CSF1R and better predicted BBB penetrance. Unfortunately, its phase Ib trial was terminated in 2021 due to the COVID-19 pandemic, and no results were published or further development announced.[25]

The field's last remaining clinical asset, enrupatinib (EI-1071, Elixiron) is in a phase II AD trial in Taiwan with primary completion registered for March 2026. Phase I in 58 healthy volunteers confirmed an acceptable safety profile, and preclinical data showed modulated activity in plaque-associated microglia while preserving homeostatic populations in 5xFAD and J20 mouse models.[26] Whether the prior clinical failures reflect poor drug design, insufficient CNS penetration, inadequate selectivity, or something more fundamental about CSF1R biology in the human brain remains unclear.

Brain Shuttle Technologies

The neuroinflammation pipeline is dominated by small molecules. This is strategically logical, as small molecules achieve CNS penetration through physicochemical design without active transport. Antibodies, oligonucleotides, peptides, and proteins are also advancing in neuroinflammation, but face an inherent BBB constraint. Their CSF-to-plasma ratios typically fall at or below 0.1 percent.[27] Brain shuttle technologies represent the path to closing this gap, but clinical experience in neuroinflammation remains limited.

The most advanced example is DNL919 (ATV: TREM2, Denali/Takeda), a TREM2 agonist antibody incorporating a transferrin receptor (TfR)-binding sequence into its Fc region, enabling active BBB transcytosis. TfR is an iron transporter broadly expressed across tissues, including at the luminal surface of BBB endothelial cells.[28] Phase I confirmed CNS target engagement via reductions in microglial CSF biomarkers (CSF1R, SPP1, IL-1RA), but Denali discontinued the program in August 2023 following moderate reversible hematological toxicity,[29] which is an inherent risk of the TfR targeting arising from its endogenous role in iron homeostasis.[30]

Beyond BBB crossing, distribution within the CNS presents an equally critical challenge. Getting into the brain is only half the problem: a therapeutic must reach the disease-relevant region at pharmacologically active concentrations—the substantia nigra in PD, the entorhinal cortex and hippocampus in early AD, the cortex and white matter in progressive MS. For larger modalities, achieving adequate regional targeting remains an underexplored frontier that could meaningfully improve both efficacy and therapeutic window for next-generation neuroinflammation assets. For a more comprehensive review of BBB shuttle technologies across CNS indications, refer to Scitaris' recent analysis: Transporting Macromolecules Across the Blood-Brain Barrier: The Next Frontier in CNS Disease Treatment.[31]

Targets in the Periphery

Immune cells in the peripheral compartment are inherently more accessible than CNS-resident populations, and the pharmacological toolkit for modulating them is more mature. It is perhaps unsurprising that some of the most clinically advanced neuroinflammation strategies target the periphery first. In diseases like MS, the immune assault on the CNS originates largely from the periphery. In relapsing MS, B cells and T cells activated in lymphoid tissue breach the BBB, triggering focal inflammatory lesions. The success of peripheral immunotherapies, like natalizumab, ocrelizumab, and cladribine, confirms that intercepting the peripheral immune response is a clinically validated strategy for relapsing disease.[32] The challenge is progressive MS, where the inflammatory burden shifts to the CNS, rendering peripheral-only approaches insufficient.

This creates a strategic case for targets expressed in both compartments simultaneously. Here, just a few select targets have gained attention. BTK is expressed on B cells and monocytes in the periphery and on microglia in the CNS.[33] TYK2 follows a similar logic: peripheral expression drives pathogenic Th17 differentiation, while microglial expression mediates interferon and IL-23 signaling. LRRK2 and NLRP3 extend the pattern further. LRRK2 is expressed on peripheral monocytes and neutrophils as well as microglia, with gain-of-function mutations among the most common genetic risk factors for PD. Similarly, NLRP3 is active on peripheral macrophages and monocytes, as well as microglia, with systemic activation contributing to CNS pathology through peripheral IL-1β production and monocyte infiltration.

BTK

Being expressed on peripheral B cells and monocytes and on CNS microglia, makes BTK one of the most strategically compelling dual-compartment targets in neuroinflammation.[33] The clinical story, however, is complex. Tolebrutinib (Sanofi), an oral brain-penetrant BTK inhibitor, earned FDA Breakthrough Therapy designation for non-relapsing secondary progressive MS (nrSPMS) in December 2024[34] and later demonstrated a 31% reduction in 6-month confirmed disability progression in nrSPMS in the phase III HERCULES trial.[35] In relapsing MS (RMS), the phase III GEMINI trials showed a comparable 29% risk reduction in disability accumulation. However, in the PERSEUS phase III trial in primary progressive MS (PPMS), tolebrutinib failed its primary endpoint of time to six-month composite confirmed disability progression. Secondary MRI outcomes showed reduced lesion accrual and brain volume loss, but were insufficient to translate into clinical.[36] On the regulatory front, the FDA issued a Complete Response Letter in December 2025 declining to approve tolebrutinib for nrSPMS, with reasons not publicly disclosed.[37]

The PPMS picture, however, has shifted with fenebrutinib (Roche). In February 2026, Roche announced that fenebrutinib met its primary endpoint of non-inferiority to ocrelizumab (OCREVUS) in reducing disability progression in PPMS—achieving a 12% reduction in the risk of disability progression compared to ocrelizumab, with curves separating as early as 24 weeks.[38] A separate phase III trial in RMS also met its primary endpoint, showing clinically meaningful and statistically significant results versus teriflunomide.[39] Together, these readouts directly challenge the interpretation that tolebrutinib's failure reflected a target-level limitation rather than a compound-specific one. Scitaris' internal analysis, based on clinical CSF data and in vitro IC_{50}s, suggests a potential pharmacological explanation: tolebrutinib's CSF concentrations likely fall well below its cellular IC_{50}, indicating insufficient CNS target coverage, while fenebrutinib reaches CSF concentrations approximately at its IC_{90}, suggesting meaningful CNS BTK occupancy was achieved. Compound design, not target biology, may have been the deciding factor all along. Therefore, the BTK hypothesis in progressive MS is very much alive.

LRRK2

LRRK2 is a large multidomain kinase expressed predominantly in microglia and monocytes, with additional expression in astrocytes, T cells, and B cells.[40] The neuroinflammatory rationale operates on two levels. In LRRK2 mutation carriers, such as the gain-of-function G2019S, which accounts for ~4–5 percent of familial PD, constitutively hyperactivate LRRK2, driving microglial activation and accelerating neuronal loss.[41] More broadly, LRRK2 kinase activity has been reported to play a role in non-mutation carrier PD patients and other neuroinflammatory diseases.[42,43]

The LRRK2 pipeline has approximately 60 assets, unsurprisingly dominated by PD (86 percent) and small molecules (74 percent) **(Figure 3)**. DNL151 (Denali/Biogen) is

one of the most advanced assets, running parallel strategies with the phase IIa BEACON study in LRRK2-mutation PD patients, complemented by the phase IIb LUMA trial enrolling early-stage PD patients to test the broader activation hypothesis, with topline data expected soon.[44] ARV-102 (Arvinas) is mechanistically distinct as a PROTAC degrader eliminating LRRK2 protein (for more information on the modality see our recent whitepaper "Induced-Proximity Therapeutics"). A recent update from the phase I trial in PD reported ≥50% LRRK2 reduction in CSF and a generally safe profile.[45]

HT-4253 (Halia Therapeutics) aimed for AD rather than PD, based on evidence that LRRK2 is activated in Aβ-associated microglia and that its inhibition reduces plaque burden and preserves synaptic structure in AD models.[43] No data have been disclosed, but if successful, it would be the first clinical test of whether LRRK2's neuroinflammatory biology extends beyond PD. Whether LRRK2 targeting will prove most valuable as a precision therapy in genetically defined mutation carriers, or whether the broader neuroinflammatory rationale supports use across other neurodegenerative diseases, remains to be seen.

TYK2

TYK2 mediates signaling downstream of type I interferons, IL-12, and IL-23, with functional expression in the CNS and peripherally.[46] Several pipeline assets co-target JAK1 alongside TYK2, based on the rationale that JAK1 mediates a complementary set of pro-inflammatory cytokines **(Figure 3)**. The trade-off is selectivity and safety: JAK1 co-inhibition carries increased risk, whereas selective allosteric TYK2 inhibition has a cleaner profile. BHV-8000 (Biohaven), a dual TYK2/JAK1 inhibitor, is the most advanced asset, currently in phase II/III in early PD (primary completion August 2027), with phase I confirming CNS penetration and reduced downstream cytokines.[47] Among the three selective TYK2 assets in phase I, A-005 (Alumis) has the most complete data, demonstrating BBB penetration with a CSF:plasma ratio 1:1 and maximal target inhibition in both compartments in healthy volunteers,[48,49] with phase II in MS planned but not yet disclosed. NEU-627 (Neuron23) and SUDO-550 (Sudo Biosciences) are also in phase I with no data reported. Whether selective TYK2 inhibition alone provides sufficient anti-inflammatory breadth or whether JAK1 co-targeting is needed for efficacy will only become clear once the first disease-specific clinical datasets emerge.

NLRP3

NLRP3 is the most investigated and clinically pursued member of the inflammasome family comprising of multi-protein cytosolic complexes that sense danger signals and drive sterile inflammation. The pipeline has 37 assets, 70 percent active, with 10 clinical assets, all small molecules **(Figure 3)**.

NLRP3 inhibitors have yet to demonstrate efficacy in a neurological indication, but the early clinical data are building. Selnoflast (Roche) completed phase I in early idiopathic PD, reporting brain penetration and an acceptable safety profile.[50] Usnoflast (Zydus) also completed phase I, but in ALS, with reported CNS target engagement and early efficacy signals, including improvement on the ALS Functional Rating Scale (ALSFRS-R) and Slow Vital Capacity.[51] Most notably, VTX-3232 (Ventyx) presented phase IIa data in early PD in October 2025, showing an acceptable safety profile alongside improvements on both motor and non-motor domains of the MDS-UPDRS.[52] The signal was sufficiently compelling for Lilly to acquire Ventyx Biosciences in January 2026. This is a clear signal that big pharma views the target as credible ahead of definitive efficacy readouts.[53] The Sanofi and Lilly deals make clear that large-cap pharma interest in neuroinflammation is more than exploratory. Both were willing to commit significant capital on phase I data alone, before efficacy was established.

Timing and Cellular Compartment

A critical dimension of neuroinflammation drug development is not simply what to target, but when and where. Identifying the precise disease stage at which a given inflammatory function switches from beneficial to pathological remains one of the hardest problems in the field.

The clearest example comes from microglia. In AD and PD, early microglial activation is considered protective through Aβ clearance and synaptic maintenance, but sustained stimulation can become deleterious with excess cytokine release propagating Aβ pathology and accelerating neuronal loss.[54] Broad microglial suppressors deployed too early, therefore risk abolishing a phagocytic response, but still do useful work. The INVOKE-2 trial for the TREM2 agonist tried to account for this by enrolling early-stage patients, where further activation may promote beneficial microglial activity. But its clinical failure suggests a more nuanced relationship between activation and benefit, which remains unresolved.

The compartment question is equally stage dependent. In RMS, peripheral adaptive immunity drives focal lesion formation, making peripherally acting agents the appropriate first intervention. But as disease progresses, inflammation moves into the CNS, and peripheral depletion alone no longer reaches the relevant cells, thereby necessitating dual peripheral-CNS targeting. In early AD, the logic inverts. Where peripheral macrophages are still clearing, CNS-selective neuroprotective agents that spare peripheral myeloid function may be preferable.[55] The central challenge across all indications is the same: capturing these changes. Developing biomarkers capable of this will ultimately enable stage-appropriate intervention and transform this conceptual framework into clinical practice.

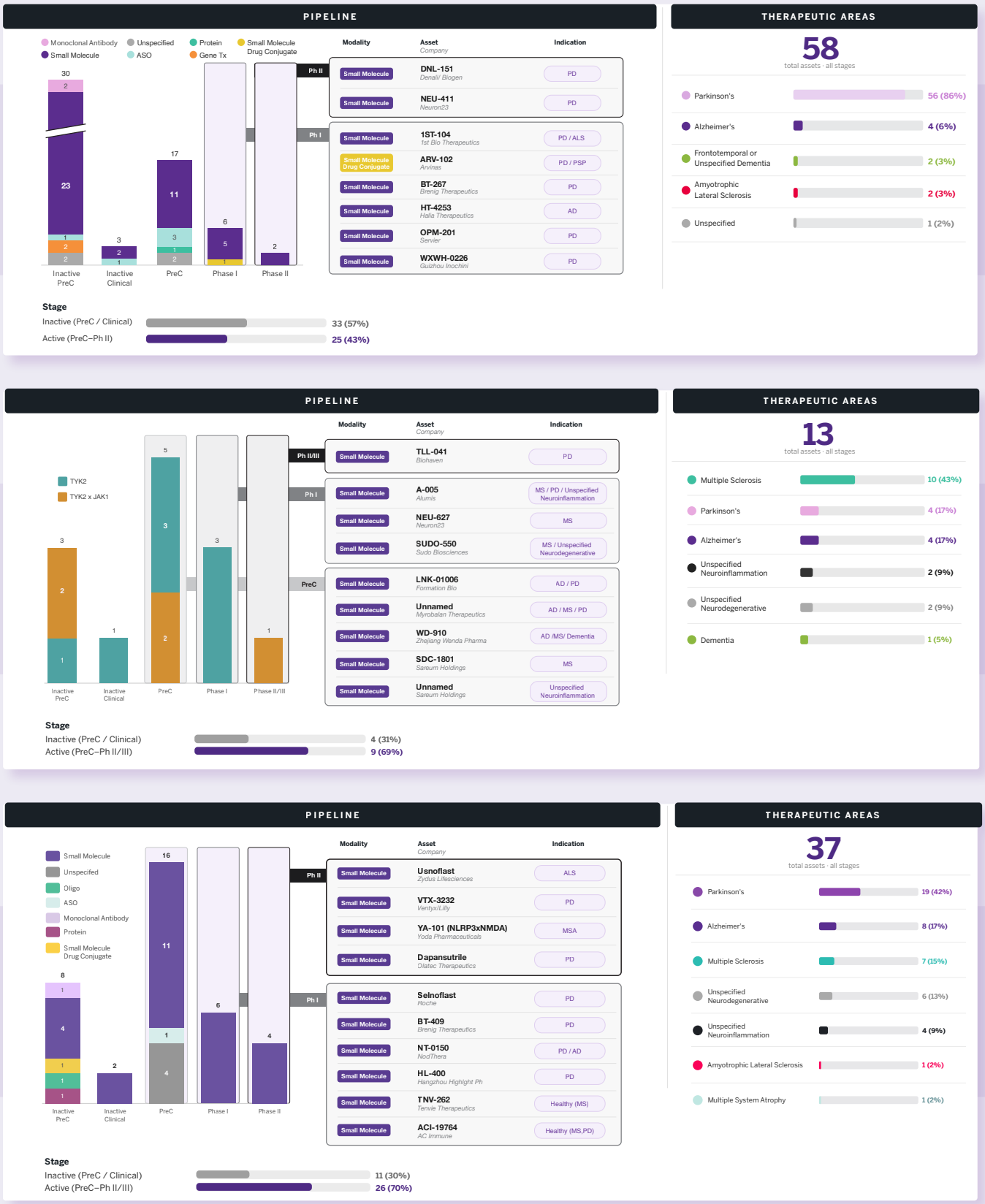

Figure 3. Drug Development Pipeline—Neuroinflammation Targets with CNS and Peripheral Immune Cell Components.
The figure depicts the current drug development landscape for LRRK2 (top), TYK2 (middle), NLRP3 (bottom) across pipeline stage, modality, and therapeutic indication. For each target, assets are stratified from inactive preclinical through the latest clinical stage, with individual programs annotated by modality, company, primary indication, and target. The proportion of active versus inactive programs are summarized alongside each pipeline view. Indication frequency is displayed as an annotated bar chart in the right panel. Assets may span multiple indications; total indication counts may therefore exceed total asset counts. Assets with dual targeting are noted. Pipeline is based on GlobalData information as of April 2026.

AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; ASO, antisense oligonucleotide; Gene Tx, gene therapy; MS, Multiple Sclerosis; MSA, multiple system atrophy; PD, Parkinson's disease; PreC, preclinical; PSP, progressive supranuclear palsy.

Deal Landscape

Deal activity in the neuroinflammation space reflects a field gaining momentum, though at an early transactional maturity. The deals highlighted in **Figure 4** are illustrative rather than exhaustive, as neuroinflammation lacks a universal definition, and relevant deals are frequently classified under broader neurodegeneration or CNS categories.

The two headline deals are Lilly's acquisition of Ventyx Biosciences, valued at up to $1.2 billion, anchored by emerging VTX-3232's NLRP3 clinical signal in PD,[54] and Sanofi's $470 million acquisition of Vigil Neuroscience, consolidating its TREM2 position following phase I data for SAR-44885.[56] The highest-value licensing agreement on the map is InnoCare/Zenas BioPharma, valued at over $2 billion. This deal granted Zenas global rights to orelabrutinib, a CNS-penetrant BTK inhibitor now in phase III for progressive MS, alongside two additional assets, including a TYK2 inhibitor.[57] The TransThera/Neurocrine and Formation Bio/Lynk Pharma partnerships signal continued mid-tier deal flow. A cluster of earlier-stage financing rounds, like Tenvie, Neuron23, and NRG Therapeutics, points to sustained VC activity in novel mechanisms.

Relative to in vivo CARs and induced-proximity therapeutics, neuroinflammation deal values remain more modest. Induced-proximity therapeutics saw several multibillion-dollar commitments from AbbVie, Novartis, and other big pharmas. The investment in the in vivo CAR space jumped from six to seven major acquisitions valued between $1 and $7 billion within 18 months, with Lilly's acquisition of Kelonia announced this April.[58] Both fields benefit from clearer mechanistic proof-of-concept and more tractable development paths. It is also worth noting that, unlike our previous whitepapers, which were focused on therapeutic modalities, this analysis is structured

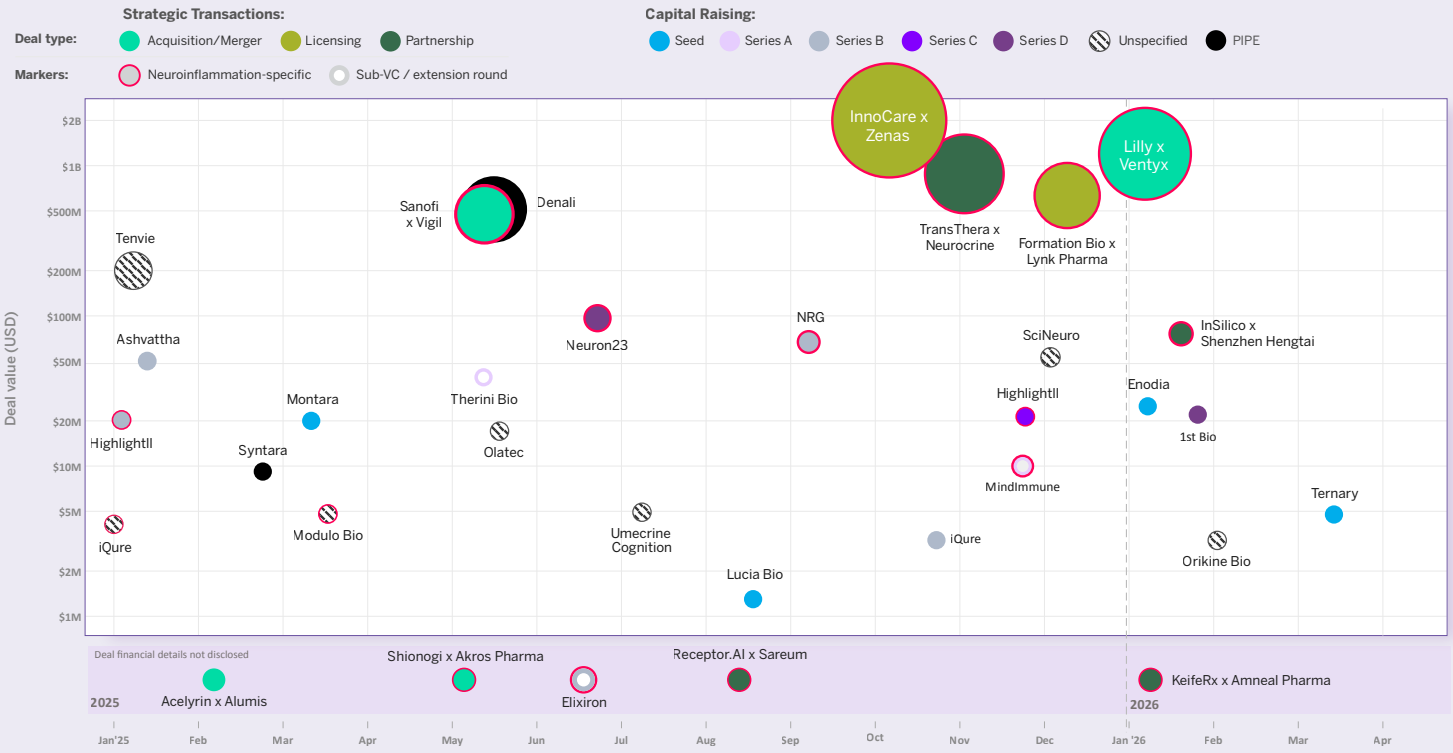

Figure 4. Select Deals in the Neuroinflammation Space
The figure maps deal activity across the neuroinflammation landscape from January 2025 through April 2026, plotting individual transactions by date (x-axis) and disclosed deal value (y-axis, log scale), with bubble size proportional to deal value. Data were curated from GlobalData, Google News, PitchBook, and publicly available press releases. This figure should be interpreted as an indicative overview of deal activity and transaction types rather than an exhaustive accounting of the space. Data reflect publicly available information as of April 2026.

Deal type is encoded by color across two distinct palettes: strategic transactions: acquisitions/mergers, licensing agreements, and partnerships are represented in green tones, while capital raising rounds (Seed through Series D) are displayed in blue and purple tones with Private Investment in Public Equity (PIPE) in black. Deals with undisclosed values are positioned according to timeline on the bottom. Neuroinflammation-specific deals are indicated by red border line; sub-VC or extension rounds are indicated by an open circle marker.

VC, venture capital; PIPE, Private Investment in Public Equity.

around a disease-associated pathomechanism and the target classes emerging from it. Could this be one of the reasons for a more modest deal activity in the neuroinflammation space or does it simply reflect a field that is earlier in its development? Modality-driven fields may generate broader platform excitement, but whether that reflects genuine differentiation or simply the appeal of a technology narrative is hard to say.

Neuroinflammation builds its case target by target and readout by readout, and most capital entering the space remains a bet on biology rather than a validated clinical signal. If readouts accumulate as expected through 2026 and 2027, it may mark the beginning of a more competitive acquisition environment.

Closing Remarks

Neuroinflammation has moved past early speculation. The biological rationale is more established, the pipeline is active, and the first efficacy signals are beginning to emerge. Yet the field is still searching for its first unambiguous efficacy readout in a neurodegenerative indication. These current data leave us with a humbling reminder that compelling biology and confirmed target engagement are not always sufficient. The translational challenges are real and not yet resolved: preclinical models remain a persistent limitation, robust biomarkers for disease staging are largely absent, and biology itself adds another layer of complexity with the same pathway harmful at one disease stage being potentially protective at another. Taken together, these are not minor obstacles, and they likely contribute to why the field has yet to deliver a definitive efficacy readout.

Several readouts over the next 12–18 months will begin to define the field's trajectory: VHB-937's (TREM2) ALS data in late 2026, fenebrutinib's (BTK) full PPMS dataset, and LUMA's (LRRK2) topline in unselected early PD. The Lilly/Ventyx acquisition marks one of the first major neuroinflammation deals anchored to emerging efficacy data, signaling that a more competitive consolidation environment is beginning to take shape. For early-stage companies, the field would reward clarity of hypothesis. Companies that will attract attention are those that have thought carefully about patient population, intervention timing, and what they expect to see as an early clinical signal. A lesson the field learned most visibly with AL002, where confirmed target engagement was not sufficient to translate into clinical benefit. In targets with prior clinical failures, the bar is likely higher: a credible mechanistic explanation for why the program is differentiated from what did not work is increasingly a prerequisite for serious engagement.

The deeper question running through this analysis remains open: which targets in which disease, at which disease stage, will prove clinically relevant. The next year will not answer that fully, but the readouts ahead will begin to separate strong hypotheses from weak ones.

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Abbreviations

AD: Alzheimer's disease

ADDF: Alzheimer's Drug Discovery Foundation

ALS: Amyotrophic lateral sclerosis

ALSFRS-R: ALS Functional Rating Scale: Revised

ARIA-E: Amyloid-related imaging abnormalities: oedema

ARIA-H: Amyloid-related imaging abnormalities: haemosiderosis

ATV: Antibody transport vehicle

Aβ: Amyloid beta

BBB: Blood-brain barrier

BTK: Bruton's tyrosine kinase

CSF: Cerebrospinal fluid

FTD: Frontotemporal dementia

mAb: Monoclonal antibody

MDS-UPDRS: Movement Disorder Society Unified Parkinson's Disease Rating Scale

MRI: Magnetic resonance imaging

MS: Multiple sclerosis

nrSPMS: Non-relapsing secondary progressive multiple sclerosis

OPC: Oligodendrocyte precursor cell

PD: Parkinson's disease

PPMS: Primary progressive multiple sclerosis

PROTAC: Proteolysis targeting chimera

RMS: Relapsing multiple sclerosis

SPP1: Secreted phosphoprotein 1

sTREM2: Soluble TREM2

TfR: Transferrin receptor

VC: Venture capital

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