BIOTECHNOLOGY EQUITY RESEARCH

Tiziana Life Sciences (TLSA, Buy, $7 PT)

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Validated Approach to Modulate CNS Inflammation Puts Tiziana in the Race for the Next Important Treatment of Neurodegeneration. Initiating at BUY and a $7 PT.

WHAT YOU SHOULD KNOW: Tiziana is a clinical-stage biotechnology company focused on using the anti-CD3 antibody foralumab to stimulate regulatory T-Cell (Treg) production to cool the inflammatory drivers of neurodegeneration. Foralumab is delivered through a nasal spray to the NALT lymphoid tissue. NALT Tregs cross paths with the CNS lymphatic system - so the stimulated Tregs are naturally primed to reduce inflammation due to CNS antigens and **to reprogram the overactive microglial cells** at the center of neurodegenerative disease. Definitive clinical data are being developed in real time, but the company has used TSPO imaging to show that foralumab reduces the CNS inflammation seen in secondary progressive MS (SPMS), multiple system atrophy (MSA) and Alzheimer's disease (AD) patients. The SPMS and MSA indications are both significant commercial applications due to unmet medical need, with approved SPMS drugs only active very early in the disease (soon after progressing from relapsing remitting MS) and the MSA indication currently having no approved disease-modifying drugs. The biggest catalyst is the INFORM-MS trial to be presented at ACTRIMS/ECTRIMS in Toronto in 10/26. This readout could de-risk the entire pipeline if the reductions in TSPO imaging signals (the biomarker for reduced inflammation) correlate with clinical symptoms such as fatigue. It's only a 12-week trial and, based on the significant validation of the TSPO signal, we would see reduction in this biomarker in a large majority of patients (~75%) as a positive result. Additional supportive clinical data are expected from the ongoing trial in MSA patients and a readout for AD in 1H27.

- **The central thesis seems clear, and the approach is relatively simple.** The ability of Tregs to reduce inflammation is as documented as anything in neuroscience and immunology. The ability to stimulate NALT Tregs to fight neurodegeneration has been shown by others and foralumab is certainly easier to deliver than cellular therapies. As a result, the major unknown is whether the Tiziana approach will develop enough Tregs to drive clinical effects. The imaging data seen for SPMS, MSA and AD suggest the approach drives measurable immunomodulation and confidence with the approach should build with the next readouts.
- **Ideal for combinations and mechanistically cleaner.** The nasal delivery and tolerance approaches are both attractive candidates for future combinations as both are distinct from approved therapies for neurodegeneration. Rather than depleting or blocking immune cells, foralumab reprograms the immune system toward tolerance by generating Tregs. Crucially, by stimulating tolerance through the nasal route, foralumab avoids systemic Tcell activation and associated AEs. To date, Tiziana reports no serious AEs in 37 patient years of exposure.
- **Entire pipeline de-risked by early applications.** A reduction of aberrant microglial activity and reductions in Treg activity are seen in each neurodegenerative disease that has been tested. So a simple treatment approach like foralumab could be used in many places. The use of immune modulators in these diseases is an active area of research as evidenced by the high activity around developing NLRP3 inhibitors. Time will tell what the winning approach will be, but using Tregs - an approach with a relatively high antigen specific mechanism - seems to have a very good chance of reducing inflammation without leaving patients open to opportunistic infections.
- **Valuation:** We value TLSA using a DCF (17.0% discount rate and a 1.0% TG rate).

Company Data	
Closing Price	$0.96
Price Target	$7.00
Market Cap (M)	$116.12
Shares Out (M)	120.51
Avg Daily Vol-3 Months (M)	0.54

Revisions	Previous	Current
Rating	-	Buy
Price Target	-	$7.00
FY26E EPS	-	(0.18)
FY27E EPS	-	(0.18)
FY26E NI	-	(21.66)
FY27E NI	-	(22.20)

EPS (GAAP)			
FY Dec	2025A	2026E	2027E
Q1	-	-	-
Q2	(0.05)	(0.08)	(0.08)
Q3	-	-	-
Q4	(0.11)	(0.10)	(0.10)
FY EPS	**(0.16)**	**(0.18)**	**(0.18)**
FY P/E	-	-	-

Net Income			
FY Dec	2025A	2026E	2027E
Q1	-	-	-
Q2	(5.63)	(9.42)	(9.65)
Q3	-	-	-
Q4	(12.80)	(12.24)	(12.55)
FY NI	**(18.43)**	**(21.66)**	**(22.20)**

Source: FactSet, BTIG Estimates and Company Documents reported as $ currency.
FY = Fiscal Year **CY** = Calendar Year

Investment Thesis

The foralumab approach seems a straightforward approach to stimulating Treg production and Tregs are an extremely well-validated modulator of the immune system. As a result, we see the Tiziana programs to be relatively de-risked and the degree and timeline for correction of patient's disease is the big readout. The company is continually generating additional clinical data with readouts in SPMS, MSA and AD all expected in the next 12 months. In each case we expect data sets that corroborate the biomarkers seen to date and continue to support the thesis that true clinical benefit will be seen in larger trials with longer treatment periods.

Upcoming Catalysts

- Topline Phase 2a Data in Non-Active Secondary Progressive Multiple Sclerosis - Late 3Q26 or early 4Q26
- Multiple System Atrophy (MSA) Phase 2a Readouts - YE2026
- Early Alzheimer's Disease Phase 2 Initial efficacy or biomarker data - 1H27

Base Case Assumptions: **$7 Price Target**

Both the SPMS P2a readout and the ongoing cohorts of MSA patients continue to show reductions in CNS inflammation in foralumab treated patients. Signs of clinical activity are reported from the early trials.

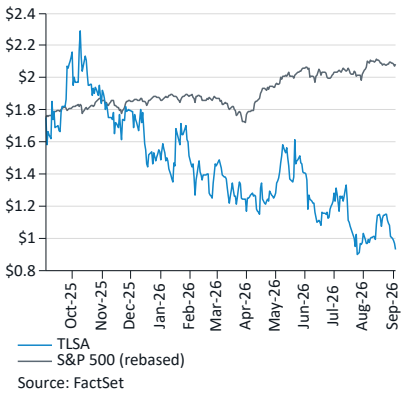
Upside Scenario

Strong signs of clinical activity that correlate with biomarker readouts like TSPO imaging are reported.

Downside Scenario

Biomarker readouts are less robust and only seen in a handful of patients. Any toxicity would be a negative surprise after the company has reported no serious AEs after 37-patient years of exposure to foralumab.

Price Performance



Source: FactSet

Company Description

Tiziana Life Sciences is a clinical-stage biotechnology company focused on developing therapies for neuroinflammatory and neurodegenerative diseases. The company's lead asset, foralumab — believed to be the only fully human anti-CD3 monoclonal antibody in clinical development — is being evaluated intranasally across multiple indications, including non-active secondary progressive multiple sclerosis (na-SPMS), Alzheimer's Disease, amyotrophic lateral sclerosis (ALS), and Multiple System Atrophy (MSA).

Key Investment Considerations

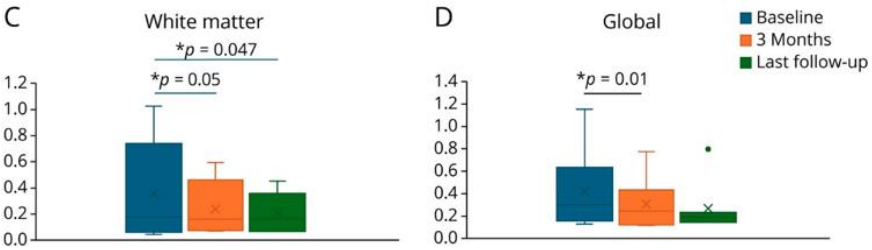
The central thesis: inflammation is the key driver. The connection between chronic inflammation and neurodegeneration has been building for decades and has recently moved from background scientific consensus to actionable therapeutic thesis. Progressive neurological conditions — multiple sclerosis (MS), Alzheimer's disease (AD), multiple system atrophy (MSA), amyotrophic lateral sclerosis (ALS), Parkinson's disease (PD) — share a common inflammatory substrate: chronically activated microglia, infiltrating CD4+ T cells, elevated CNS levels of IL-1β, TNF-α, and IL-6, and a progressive failure of the mechanisms that normally resolve inflammation and clear cellular debris. These processes are not incidental to neurodegeneration — they are increasingly understood to be causal. Activated microglia release reactive oxygen species and pro-inflammatory cytokines that directly damage oligodendrocytes and neurons; dysfunctional regulatory T cells (Tregs) fail to suppress this activity; and the resulting cycle of inflammation and cell death perpetuates itself even in the absence of overt immune attacks or new lesion formation. The **analogy to smoldering cardiovascular inflammation is instructive** — just as chronically elevated hsCRP signals ongoing arterial wall inflammation that predicts future cardiovascular (CV) events, chronically elevated TSPO PET signal in the brain indicates a low-grade inflammatory process that correlates with long-term neurological decline.

Foralumab has shown significant proof of biology. Foralumab is a fully human anti-CD3 monoclonal antibody administered once daily as a nasal spray, designed to engage the nasal-associated lymphoid tissue (NALT) and induce Tregs that subsequently traffic to the central nervous system and suppress neuroinflammation. The mechanistic lineage draws directly on decades of mucosal tolerance science pioneered by Dr. Howard Weiner's laboratory at Harvard's Brigham and Women's Hospital. Several data sets directly imaging the effects of foralumab on the brains of patients with neurodegenerative disease have been reported. The best data are probably from patients with secondary progressive multiple sclerosis (SPMS), where the mechanistic picture is clearest. A peer-reviewed publication in *Neurology: Neuroimmunology & Neuroinflammation* reported results in non-active SPMS (na-SPMS) patients with progression independent of relapses (PIRA), which is currently the hardest-to-treat form of MS because there's no active inflammation visible on conventional MRI, yet patients keep declining.

Ideal for combinations and mechanistically cleaner. The nasal delivery and tolerance approaches are both distinct from approved MS therapies suggesting combinations could be interesting going forward. Rather than depleting (CD20 approaches) or blocking immune cell trafficking, foralumab works to reprogram the immune system toward tolerance by generating Tregs that actively suppress the inflammatory cascade driving neurodegeneration rather than T effector cells that cause the disease. Crucially, by stimulating

tolerance through the nasal route rather than intravenously, foralumab also avoids the systemic T cell activation. This negative aspect of CD3 binding remains at the heart of any CD3-directed approach after the extreme cytokine storm that made early-generation IV anti-CD3 antibodies (like OKT3) clinically untenable. **By contrast, Tiziana has reported no serios AEs in 37 patient-years of exposure to foralumab.**

Exhibit 1. Foralumab reduces activated microglia in SPMS patients



Source: Tiziana company reports

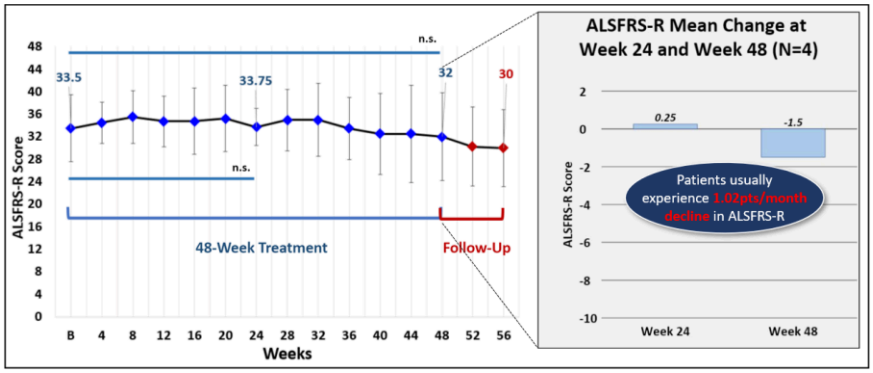
Mechanism seems established – TSPO imaging is relevant. Inflammation involves a broad set of biological pathways and clinical surprises relative to prior biomarker readouts remain possible. The recent ZEUS trial in CV disease was a good example where an IL6 modulator reduced a general marker of inflammation (hsCRP) but did not protect against CV disease (our review HERE). This situation seems less likely for TSPO imaging and its correlation with neurodegenerative disease as TSPO is a mitochondrial translocator protein expressed on activated microglia, and [18F]PBR06 PET imaging provides a quantitative, spatially resolved, in-vivo readout of neuroinflammation as defined by microglial activation (numbers of activated cells rather than degree of activation). Most importantly, post-mortem validation studies have confirmed that disease severity correlates with histological microglial activation.

Hints of clinical benefit. In addition to all the imaging data suggesting foralumab cools disease-relevant inflammation across the CNS, a handful of patients have been followed for signs of clinical benefit. Foralumab stabilized disability and improved fatigue in a meaningful proportion of patients. Preliminary unblinded data from 10 treated patients offered early encouragement: all demonstrated EDSS stabilization, three of four patients receiving continuous treatment showed improvement, and six of ten reported fatigue reduction. In a parallel expanded-access program for MSA three consecutive patients treated with foralumab have each demonstrated consistent reductions in brain neuroinflammation on PET imaging (up to 34% SUV reduction, 26% SUVR reduction in the most recent patient).

Supportive data from others. Coya Therapeutics (COYA, Buy, $16 TP) is probably the most direct competitor to Tiziana but also has generated some of the best supportive data for the ability of activated Tregs to fight neurodegeneration. As reviewed in more detail in this note (HERE), COYA 302 froze ALS patient's disability in its first clinical experience. It was a small

trial, but in a P1b trial at Houston Methodist Hospital, four ALS patients (one familial + three sporadic) were treated with COYA 302 for 48 weeks with an 8-week follow-up period. Each of these patients was in the symptomatic stages of the disease and would be expected to progress ~1 point on the ALSFRS-R per month. Rather, all four patients appeared stable over the course of the trial (48 weeks). This treatment effect needs to be repeated in a larger trial, but it's worth noting that none of the approved ALS drugs has done more than slow the progression on the ALSFRS-R scale and the most recent approval showed only trends toward any clinical benefit (QALSODY was approved on a biomarker signal).

Exhibit 2. ALSFRS-R Scores Stabilized During the Treatment Period



Source: Coya company reports

Direct Treg effects. The foundational work for Tregs in neurodegenerative disease also came from Stanley Appel and David Beers at Houston Methodist - the scientific founders of Coya. Firstly, this group established that Tregs in ALS patients are numerically reduced and functionally impaired, and that the degree of dysfunction correlates with rate of disease progression. These data were considered compelling enough to support a P1 trial involving Treg cellular therapy. Their Phase 1 study (published in *Neurology: Neuroimmunology & Neuroinflammation*) expanded autologous Tregs ex vivo and re-infused them into ALS patients. The results were encouraging: the infusions were safe and well-tolerated, and ALSFRS-R decline rates slowed during the infusion period compared to pre-infusion trajectory. It was a small proof-of-concept study but it established feasibility and gave the field a clinical signal to build on.

Tiziana's clinical programs

A unique approach in each indication. There is no other clinical-stage intranasal anti-CD3 program competing for any of the indications targeted by Tiziana. The most-watched class in progressive MS is the BTK inhibitors (tolebrutinib, fenebrutinib, evobrutinib). These drugs act by modulating B cell and microglial signaling rather than through Treg induction. In addition, the BTK programs have seen liver toxicity signals (fenebrutinib's Phase 3 was

stopped; tolebrutinib has an ongoing regulatory review). This creates a genuine opening for a tolerogenic approach with a clean safety profile. MSA is an indication with no approved therapeutics that modify the disease course and AD therapeutics are currently focused more on reducing toxic aggregates than directly cooling inflammation (although recent approaches based on TREM2 and reducing CNS TNFa have recently failed). Finally, research to control the raging inflammation in ALS patients is a hotbed of activity, but the need is clear and the Treg approach seems to have a good chance of walking the tightrope of cooling inflammation without resulting in increased infections.

SPMS – the lead indication. The therapeutic justification for developing foralumab in na-SPMS rests on a well-established clinical and biological problem: there is little else to help these patients. The disease is a significant opportunity as approximately 25%-30% of the ~850,000 people with relapsing-remitting MS (the T-Cell phase) will transition to secondary progressive disease over their lifetime (the MG phase). Based on multiple clinical trials, once MS patients become non-active (no new relapses, no gadolinium-enhancing lesions) they lose benefits from current disease-modifying therapies and face an uninterrupted trajectory of disability accumulation. Mitoxantrone is approved in this setting but is rarely used due to cardiac toxicity and leukemia risk. Siponimod received approval in 2019 on the back of EXPAND trial data showing a 21% reduction in 3-month confirmed disability progression. However, detailed analysis suggests this benefit was concentrated in patients with recent relapses or active MRI lesions, not in the truly non-active population foralumab is targeting.

Short wait for key data. The INFORM-MS trial — a randomized, double-blind, placebo-controlled study, with 48 patients, 12 weeks of treatment — completed enrollment in May 2026 and dosed its last patient on June 25, 2026. Topline data are expected in October, with results set to be presented at ACTRIMS/ECTRIMS in Toronto in October 2026. This readout has the potential to de-risk the entire pipeline if the reductions in TSPO imaging signals (the biomarker for reduced inflammation) correlate with clinical symptoms such as fatigue. It's a lot to ask for in a 12-week treatment experience and, based on the significant validation of the TSPO signal, we would see reduction in this biomarker in a large majority of patients (~75%) as a positive result.

FDA seems to be interested. In the recent CRL for the BTK inhibitor Tolebrutinib, the FDA expressed its willingness to bend the rules slightly based on the unmet need in SPMS. For example, the FDA stated: "We acknowledge that this subpopulation was not predefined in the enrolled population; however, the Division was willing to show flexibility to consider narrowing the indication to a population for which there are no FDA-approved therapies, and for which there is a significant unmet medical need." The letter goes on to detail the modest evidence for efficacy (the P3 HERCULES

trial was considered positive) in any subpopulation and the risk of significant liver toxicity seen with the drug and concludes the risk - reward profile doesn't support approval. Foralumab still needs to show clear clinical efficacy but the drug is very safe, and the FDA is interested - two out of three isn't bad going into the next round of data.

Strong unmet medical need in SPMS. There is no other clinical-stage intranasal anti-CD3 program in later-staged development for the na-SPMS indication. The most promising class of drugs in progressive MS are BTK inhibitors (tolebrutinib, fenebrutinib, evobrutinib). Again, these drugs inhibit B-Cell and microglial signaling (block bad actors) rather than through Treg induction (re-train bad actors). The BTK programs have also been plagued by liver toxicity signals. For example, fenebrutinib's P3 trial was stopped and tolebrutinib received the CRL that was very positive on the indication but found the drug just too toxic. The NLRP3 inhibitors could play in the space eventually, but none are in clinical trials.

The MSA program – high need, decent validation and no competition. MSA represents a smaller commercial opportunity but arguably even more scientific urgency. MSA is uniformly fatal, typically kills within six to ten years of diagnosis, and has no approved disease-modifying treatment. The consistent PET signal across three consecutive treated patients — in an uncontrolled setting, yes, but with a validated imaging endpoint and no precedent for spontaneous improvement of this magnitude — is the most provocative clinical data in the Tiziana story and the reason the MSA program warrants attention disproportionate to its current commercial modeling.

AD and ALS make sense. These two programs are both interesting but probably less central to the investment thesis. AD is clearly an inflammatory disease with the TSPO imaging of others showing overlap in the regions of heavy Ab plaque and inflammation (activated MG surround plaque). However, the next trial would likely be large, and a partner is probably needed. So, we expect advances in this program and await further data. There is an ongoing P2 foralumab trial with data expected 1H27 which we expect to show reductions in inflammation. ALS is a highly inflammatory disease and is currently being tackled with several approaches to reduce inflammation. For example, Coya is in P2/3 with a Treg approach that forms some of the basis for our interest in Tiziana but is also a direct competitor in this indication.

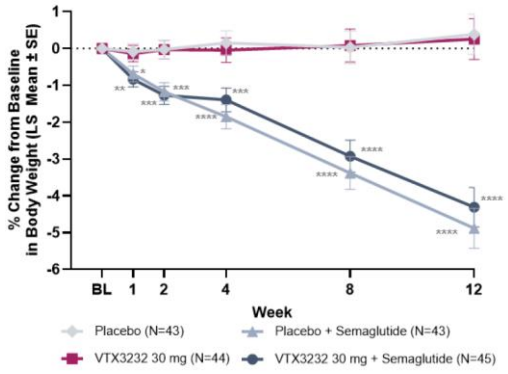
TZLS-501: The Forgotten Asset. Tiziana's second program is TZLS-501, an anti-IL-6 receptor antibody targeting a validated pathway and is likely to be advanced in a partnership in our view. We believe that activity seems likely as tocilizumab, sarilumab, and siltuximab have all demonstrated that IL-6 inhibition works across multiple inflammatory diseases.

Risks to Our Investment Thesis

Neuroscience is inherently complex. Drug discovery is inherently risky. The current stance of the FDA seems mixed with the agency increasingly willing to look at the totality of data and to consider molecular endpoints as supportive evidence of activity. However, the agency also seems more focused on larger datasets that are well-controlled. For a smaller company like Tiziana, these issues will raise the cost of clinical trials and for attractive markets like SPMS, MSA and AD, we expect the agency will eventually want two well-controlled trials for full approval, and that accelerated approval may lead to very spotty reimbursement (based on Aduhelm). In addition, **Causality detection in humans is still imperfect.** Most human data is observational. We know inflammation predicts bad outcomes, but whether reducing immune activity in otherwise healthy older adults will meaningfully extend a healthy lifespan is not yet proven in RCTs.

Mice aren't men: Ventyx's weightloss readout. The P2 readout for Ventyx Biosciences' (acquired by Eli Lilly [LLY, Not Rated]) VTX-3232 was about as good as it could be hoped on the CV front. The drug reduced hsCRP and IL6 below the levels associated with 25% reductions in MACE endpoints in the majority of patients. However, contrary to animal models, NLRP3 inhibition did not reduce patient's weight as monotherapy and did not increase the weight loss experienced with concomitant incretins. This result had management extolling the fact that GLP-1 associated weight loss was not reduced.

Exhibit 3. VTX3232 weight loss readout was somewhat unexpected



Source: Ventyx Company reports

Biology risk and later-stage patients. The biology around stimulating Treg production is complex as is the general role of the brain's innate immune system in neurodegeneration. Similarly, the idea that the Tregs generated by foralumab will focus on antigens draining from the CNS seems mechanistically reasonable and is backed by animal model data, but we are aware of no drug that has been approved that depends strictly on this mechanism. In addition, there remains significant risk for any program

attempting to back patients out of their disease after years to decades of destructive inflammation. In other words, even drugs that might be very effective at stopping a disease from progressing at very early stages might not have the same effects in later-stage patients. This type of efficacy is seen with the Ab plaque busting mAbs approved in AD. Large studies make it clear that patients do better if they are treated earlier in their disease.

General execution and competitive risk. As a clinical-stage biopharmaceutical company with no approved products and a limited operating history, Tiziana faces meaningful execution risk across its pipeline indications. The company has incurred significant operating losses since inception and will require substantial additional capital to fund ongoing and planned clinical programs; an inability to raise capital on acceptable terms could force delays or reductions in development activity. Foralumab faces a competitive risk from other approaches that target neuroinflammation. This risk has become greater with the **rapid advances in the NLRP3 inhibitor landscape** that includes programs from Ventyx Biosciences, NodThera (Private), BioAge (BIOA, Buy, $40 TP), AC Immune (ACIU, Buy, $8 TP), Roche (RHHBY, Not Rated), Merck (MRK, Not Rated), Novo Nordisk (NVO, Not Rated), AstraZeneca (AZN, Not Rated), and others, as well as established injectable anti-IL-6 agents such as ziltivekimab and canakinumab.

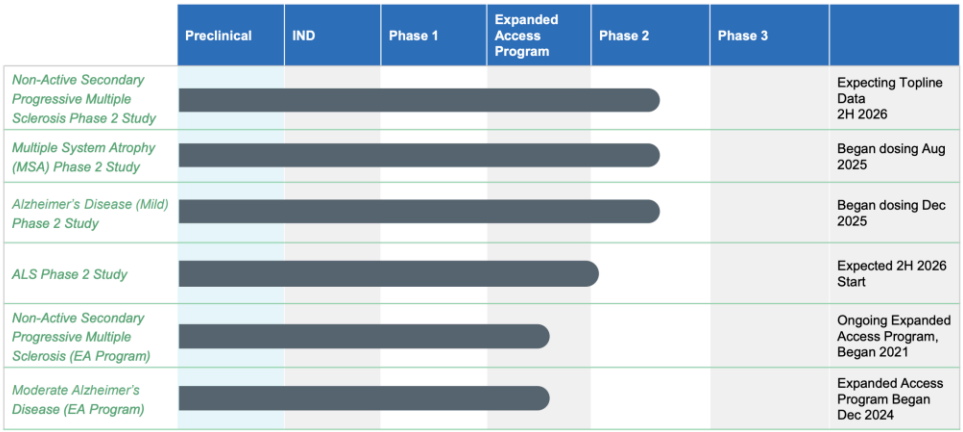
Direct competition in the Treg space. The important roles of regulatory T Cells in avoiding autoimmunity have been understood for decades and data showing decreased Treg levels in diseases like ALS are similarly well grounded. Some of the key work that underpins our confidence in the Tiziana approach is from potential competitors like Coya Therapeutics . The lead therapeutic programs have only modest overlap as Coya is focused on ALS and its companion disease FTD while Tiziana seems most focused on SPMS and MSA. However, we expect the optimal approach to developing Tregs may eventually be the commercial winner across many diseases. Both approaches seem commercially tractable and involve therapeutic antibodies rather than cellular therapies (Coya started with cellular approaches but has switched to ld-IL2 based approaches). In the shorter term, these two approaches could reinforce one another, but longer-term, we expect some competition.

Commercial and pricing risk. Even if foralumab achieves regulatory approval, commercial success will depend heavily on pricing, reimbursement coverage, and the ability to penetrate a primary care prescribing channel that has historically been slow to adopt novel anti-inflammatory therapies. To our eyes, this issue for Tiziana will likely depend on the clinical data. If foralumab shows only a modest slowing of progression, a price of $70,000 per year might be expected. However, if the larger trials repeat the early experience and foralumab freezes the disease, a price 2-2.5 times higher would seem reasonable to us.

Pipeline and upcoming events

The Tiziana pipeline is built around intranasal foralumab and its ability to **increase Treg production in a manner that seems poised to reduce the inflammation in the brain** that both potentiates and drives neurodegeneration. The approach could be active across neurology, and the company has chosen four lead indications that reflect both large market opportunities (AD) and strong unmet need (MSA). Importantly, all four indications share a common mechanism involving peripheral Treg induction via NALT stimulation that leads to reduced neuroinflammation. As a result, **each upcoming dataset reinforces the platform in addition to de-risking the specific indication**. The MS readout is the pivotal event; if INFORM-MS shows a significant effect on TSPO-measured inflammation along with some hints of clinical benefit, it would substantially de-risk the ALS and AD programs as well.

Exhibit 4. The Tiziana Pipeline



Source: Tiziana company reports

We see two steps to firm proof of concept for this basic approach.

- Reducing inflammation. This step is likely to be shown in the shorter term via biomarker readouts of inflammatory cytokines and direct imaging of inflammation in affected brain regions. We have already seen supportive readouts in three of the four Tiziana programs and the role in ALS is well established by others.
- Correlation with clinical benefit. Demonstrating that reducing inflammation will have clinical benefits in diseases like Alzheimer's and Multiple System Atrophy is the true catalyst. We have seen hints in both SPMS and MSA but corroborative data should build investor confidence.

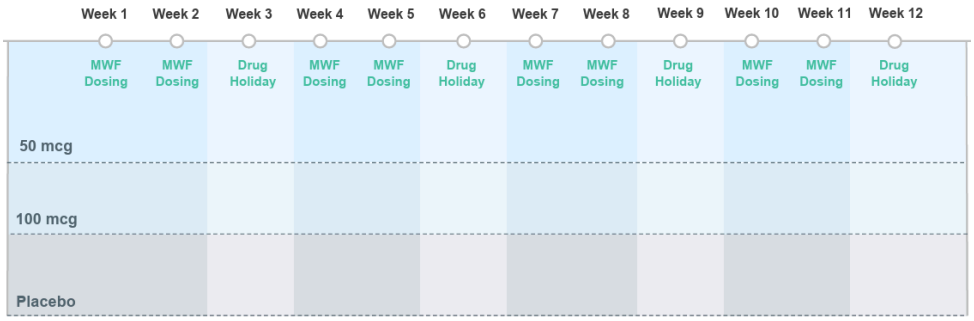
Key Trials

SPMS-non active (INFORM-MS). This P2 placebo-controlled trial completed enrollment in May 2026, the last patient was dosed on June 25, 2026 (n=48 at two doses, historical comparator). Data are expected in October. Detailed data are likely at the ACTRIMS/ECTRIMS meeting in Toronto, but topline data might be released

earlier. Earlier open-label data (May 2026) showed fatigue scores improved in 64% of non-active secondary progressive MS patients. The key measurements for the trial include:

- **Baseline screening:** PET scans, MRI, biomarkers, EDSS and MFIS
- **3-month endpoint:** PET scans, MRI, biomarkers, EDSS and MFIS

Exhibit 5. The SPMS trial seems relatively straightforward.



Source: Tiziana company reports

Multiple System Atrophy (MSA). This trial is more of a Compassionate Use / Case Series but the data to date have been interesting, and the medical need is huge. In the most recent update (July 2026) Tiziana reported reduction in brain inflammation in a third MSA patient treated with intranasal foralumab. The company expects to report a final patient from this first cohort and then follow up with a second cohort of patients in the steep part of the decline curve for the disease. This first set of ~10 patients is expected to form the basis of an FDA discussion around a pivotal trial design and the company will likely seek BTD.

ALS (BANYAN). A P2a trial is underway as part of the HEALEY framework (shared placebo arm), and data were presented at a poster at The ALS Nexus conference in August. This program is an early, POC effort. No human clinical data in ALS for foralumab have been reported.

The BANYAN trial is 3:1 randomized, placebo-controlled, multicenter study enrolling 24 patients across sites within the Network of Excellence for ALS (NEALS) Consortium. Inclusion in the Healey ALS MyMatch infrastructure is operationally meaningful: successful investigational products from this platform may advance to future regimens of the HEALEY ALS Platform Trial, a perpetual, multi-regimen, late-phase clinical efficacy study, or transition directly to standalone Phase 3 trials, providing a potentially accelerated pathway to late-phase development. The BANYAN trial is currently in operational startup.

Alzheimer's Disease. The P2 trial began enrollment in late 2025 and the first patient was dosed in December 2025. The trial includes two complementary arms:

- as a monotherapy targeting neuroinflammation, and
- in combination with lecanemab or donanemab.

The combination rationale is directly grounded in the lecanemab TSPO-PET observation: given that anti-amyloid agents clear plaques but leave microglial activation unresolved, layering a Treg-inducing agent has the potential to address the residual neuroinflammatory burden that persists after amyloid reduction. The dual-arm design positions foralumab both as a standalone option for patients ineligible for anti-amyloid therapy, including those with moderate disease or APOE4 homozygosity, and as a rational combination partner to the emerging anti-amyloid standard of care. Primary endpoints include microglial activation changes on TSPO-PET, cognitive assessments, and biomarker measures of amyloid, tau, NfL, and GFAP, with a primary readout at three months.

The AD trial is again supported by decent mechanistic data as Tiziana previously reported a significant reduction in microglial activation on PET scans in a moderate AD patient. Data are expected in 1H27 and larger trials are expected to be part of a partnership (n>1,000).

Tiziana Valuation and Market Models

We value TLSA using a DCF analysis and assuming a 17% discount rate and a 1% terminal growth rate. Our DCF analysis captures revenue projections out to 2040 from four key programs: SPMS, MSA, AD, and ALS.

The discount rate of 17% reflects the relatively high risk of clinically developing a CNS drug in a competitive market and the fact that the main programs are in the early stages of development.

The positive terminal growth rate reflects the broad applicability of TZLS-401 across inflammatory neurological disorders, including the four main programs and Parkinson's.

Exhibit 6. CNS Inflammation Comps Table 9/1/2026

Company Name	Ticker	Price	Market cap (M)	EV (M)	Shares out (M)	Shares out diluted (M)	BTIG Rating/TP
Tiziana Life Sciences	TLSA	$ 0.99	$ 132.30	$ 128.30	128.3	118.3	Buy, $7 TP
Coya Therapeutics	COYA	$ 4.56	$ 107.00	$ 63.80	23.5	23.5	Buy, $16 TP
Alector	ALEC	$ 2.26	$ 258.30	$ 95.40	111.7	111.2	Buy, $6 TP
AC Immune	ACIU	$ 2.64	$ 270.70	$ 188.20	101.8	101.8	Buy, $8 TP
BioAge Labs	BIOA	$ 9.50	$ 436.20	$ 121.60	45.9	44.8	Buy, $40 TP
Inmune Bio	INMB	$ 2.30	$ 64.30	$ 45.90	27.8	26.7	Not Rated
Immunic Therapeutics	IMUX	$ 15.80	$ 879.00	$ 723.90	13.6	42.5	Not Rated

Source: BTIG Research, Factset

Exhibit 7. TLSA Valuation

DCF Valuation	
Discount rate	17.0%
Terminal growth rate	1.0%
PV of FCF (MM)	$ 895
Cash and equivalents 4Q27E (MM)	$ 8
Debt 4Q27E (MM)	$ -
Market value (MM)	$ 903
Shares outstanding 4Q27E (MM)	121
Price per share	$ 7.43

Discount Rate \ Terminal Growth Rate	-0.5%	0.0%	0.5%	1.0%	1.5%	2.0%	2.5%
15.5%	9.14	9.29	9.46	9.63	9.82	10.02	10.24
16.0%	8.40	8.53	8.68	8.83	8.99	9.17	9.35
16.5%	7.72	7.84	7.96	8.10	8.24	8.39	8.56
17.0%	7.10	7.20	7.31	7.43	7.56	7.69	7.83
17.5%	6.53	6.62	6.72	6.82	6.93	7.05	7.17
18.0%	6.00	6.08	6.17	6.26	6.36	6.46	6.57
18.5%	5.52	5.59	5.67	5.75	5.84	5.93	6.03

Source: BTIG Research estimates

TZLS-401 (Foralumab) for SPMS Market Model

- We assume a 50% probability of success.
- We estimate the current prevalence of MS in the US is approximately 1 million patients in 2026 (Source: National Multiple Sclerosis Society Website).
- We estimate that the percentage of patients that are diagnosed and currently seeking treatment to be 80%.
- Of all MS patients, we estimate that the percentage of relapsing remitting MS (RRMS) patients to be 85% while the percentage of primary progressive MS (PPMS) patients to be the other 15%.
- Of the RRMS patients, we estimate that 25% of these patients progress to secondary progressive MS (SPMS).
- We estimate that 70% of SPMS patients are non active (naSPMS).
- We assume market entry of TZLS-401 for naSPMS to be in 2030.
- We assume net prices per year to be $70,000 with an annual growth rate of 1.5%.
- We assume a peak market penetration of 15% of naSPMS patients.

Exhibit 8. TZLS-401 (Foralumab) for SPMS Market Model

	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045
US Market																				
Multiple Sclerosis Prevalence	1,000,000	1,007,000	1,014,049	1,021,147	1,028,295	1,035,493	1,042,742	1,050,041	1,057,391	1,064,793	1,072,247	1,079,752	1,087,311	1,094,922	1,102,586	1,110,304	1,118,077	1,125,903	1,133,784	1,141,721
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Diagnosed and seeking treatment	800,000	805,600	811,239	816,918	822,636	828,395	834,194	840,033	845,913	851,834	857,797	863,802	869,849	875,937	882,069	888,244	894,461	900,722	907,028	913,377
% diagnosed and seeking treatment	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
RRMS Patients	680,000	684,760	689,553	694,380	699,241	704,136	709,064	714,028	719,026	724,059	729,128	734,232	739,371	744,547	749,759	755,007	760,292	765,614	770,973	776,370
% of MS patients with RRMS	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
PPMS Patients	120,000	120,840	121,686	122,538	123,395	124,259	125,129	126,005	126,887	127,775	128,670	129,570	130,477	131,391	132,310	133,237	134,169	135,108	136,054	137,007
% of MS patients with PPMS	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*
SPMS patients	170,000	171,190	172,388	173,595	174,810	176,034	177,266	178,507	179,757	181,015	182,282	183,558	184,843	186,137	187,440	188,752	190,073	191,404	192,743	194,093
% of RRMS patients progressing to SPMS	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*
naSPMS patients	119,000	119,833	120,672	121,517	122,367	123,224	124,086	124,955	125,830	126,710	127,597	128,491	129,390	130,296	131,208	132,126	133,051	133,982	134,920	135,865
% of SPMS patients who are non-active	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*
Price per patient annually	-	-	-	-	$ 70,000	$ 71,050	$ 72,116	$ 73,197	$ 74,295	$ 75,410	$ 76,541	$ 77,689	$ 78,854	$ 80,037	$ 81,238	$ 82,456	$ 83,693	$ 84,949	$ 86,223	$ 87,516
% growth	-	-	-	-	-	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-	*2%*	*5%*	*8%*	*11%*	*13%*	*14%*	*15%*	*14%*	*12%*	*11%*	*9%*	*8%*	*8%*	*8%*	*8%*	*4%*
					0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5	0.5	0.5
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
SPMS Revenue (MM) (U.S.)	**-**	**-**	**-**	**-**	**$ 116**	**$ 507**	**$ 863**	**$ 1,323**	**$ 1,532**	**$ 1,751**	**$ 1,884**	**$ 1,733**	**$ 1,574**	**$ 1,408**	**$ 1,233**	**$ 1,051**	**$ 1,074**	**$ 1,098**	**$ 1,122**	**$ 573**
EU Market																				
Multiple Sclerosis Prevalence	1,200,000	1,208,400	1,216,859	1,225,377	1,233,954	1,242,592	1,251,290	1,260,049	1,268,870	1,277,752	1,286,696	1,295,703	1,304,773	1,313,906	1,323,104	1,332,365	1,341,692	1,351,084	1,360,541	1,370,065
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Diagnosed and seeking treatment	960,000	966,720	973,487	980,301	987,164	994,074	1,001,032	1,008,039	1,015,096	1,022,201	1,029,357	1,036,562	1,043,818	1,051,125	1,058,483	1,065,892	1,073,353	1,080,867	1,088,433	1,096,052
% diagnosed and seeking treatment	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
RRMS Patients	816,000	821,712	827,464	833,256	839,089	844,963	850,877	856,834	862,831	868,871	874,953	881,078	887,246	893,456	899,710	906,008	912,350	918,737	925,168	931,644
% of MS patients with RRMS	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
PPMS Patients	144,000	145,008	146,023	147,045	148,075	149,111	150,155	151,206	152,264	153,330	154,404	155,484	156,573	157,669	158,772	159,884	161,003	162,130	163,265	164,408
% of MS patients with PPMS	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*
SPMS patients	204,000	205,428	206,866	208,314	209,772	211,241	212,719	214,208	215,708	217,218	218,738	220,269	221,811	223,364	224,928	226,502	228,088	229,684	231,292	232,911
% of RRMS patients progressing to SPMS	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*
naSPMS patients	142,800	143,800	144,806	145,820	146,841	147,868	148,904	149,946	150,995	152,052	153,117	154,189	155,268	156,355	157,449	158,551	159,661	160,779	161,904	163,038
% of SPMS patients who are non-active	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*	*70%*
Price per patient annually	-	-	-	-	$ 40,000	$ 40,600	$ 41,209	$ 41,827	$ 42,455	$ 43,091	$ 43,738	$ 44,394	$ 45,060	$ 45,736	$ 46,422	$ 47,118	$ 47,825	$ 48,542	$ 49,270	$ 50,009
% growth	-	-	-	-	-	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-	*2%*	*5%*	*8%*	*11%*	*13%*	*14%*	*15%*	*14%*	*12%*	*11%*	*9%*	*8%*	*8%*	*8%*	*8%*	*4%*
					0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5	0.5	0.5
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
SPMS Revenue (MM) (E.U.)	**-**	**-**	**-**	**-**	**$ 79**	**$ 347**	**$ 592**	**$ 907**	**$ 1,051**	**$ 1,200**	**$ 1,292**	**$ 1,188**	**$ 1,079**	**$ 965**	**$ 846**	**$ 720**	**$ 736**	**$ 753**	**$ 769**	**$ 393**
Total SPMS Revenue (MM)	-	-	-	-	**$ 195**	**$ 854**	**$ 1,455**	**$ 2,230**	**$ 2,583**	**$ 2,951**	**$ 3,175**	**$ 2,921**	**$ 2,654**	**$ 2,373**	**$ 2,079**	**$ 1,771**	**$ 1,810**	**$ 1,850**	**$ 1,891**	**$ 966**

Source: BTIG Research Estimates

TZLS-401 (Foralumab) for MSA Market Model

- We assume a 30% probability of success.
- We estimate the current prevalence of MSA in the US is approximately 0.005% or 17,130 patients in 2026 (Source: NINDS NIH Website).
- We assume that 85% of patients are diagnosed and currently seeking treatment.
- We assume market entry of TZLS-401 for MSA to be in 2031.
- We assume net prices per year to be $70,000 with an annual growth rate of 1.5%.
- We assume a peak market penetration of 25% of diagnosed patients with MSA.

Exhibit 9. TZLS-401 (Foralumab) for MSA Market Model

	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045
US Market																				
US Population	342,600,000	344,998,200	347,413,187	349,845,080	352,293,995	354,760,053	357,243,374	359,744,077	362,262,286	364,798,122	367,351,709	369,923,171	372,512,633	375,120,221	377,746,063	380,390,285	383,053,017	385,734,388	388,434,529	391,153,571
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
MSA patients	17,130	17,250	17,371	17,492	17,615	17,738	17,862	17,987	18,113	18,240	18,368	18,496	18,626	18,756	18,887	19,020	19,153	19,287	19,422	19,558
% with MSA	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*
Diagnosed MSA patients	14,561	14,662	14,765	14,868	14,972	15,077	15,183	15,289	15,396	15,504	15,612	15,722	15,832	15,943	16,054	16,167	16,280	16,394	16,508	16,624
% of MSA patients seeking treatment	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
Price per patient annually	-	-	-	-	-	$ 70,000	$ 71,050	$ 72,116	$ 73,197	$ 74,295	$ 75,410	$ 76,541	$ 77,689	$ 78,854	$ 80,037	$ 81,238	$ 82,456	$ 83,693	$ 84,949	$ 86,223
% growth	-	-	-	-	-	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-		*3%*	*8%*	*13%*	*19%*	*21%*	*24%*	*25%*	*23%*	*20%*	*18%*	*15%*	*13%*	*13%*	*13%*	*6%*
						0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5	0.5
Gross-to-net adjustment						*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
MSA Revenue (MM) (U.S.)	-	-	-	-	$ -	$ 26	$ 81	$ 138	$ 211	$ 245	$ 280	$ 301	$ 277	$ 251	$ 225	$ 197	$ 168	$ 172	$ 175	$ 90
EU Market																				
EU Population	452,000,000	455,164,000	458,350,148	461,558,599	464,789,509	468,043,036	471,319,337	474,618,572	477,940,902	481,286,489	484,655,494	488,048,083	491,464,419	494,904,670	498,369,003	501,857,586	505,370,589	508,908,183	512,470,540	516,057,834
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
MSA patients	22,600	22,758	22,918	23,078	23,239	23,402	23,566	23,731	23,897	24,064	24,233	24,402	24,573	24,745	24,918	25,093	25,269	25,445	25,624	25,803
% with MSA	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*	*0.005%*
Diagnosed MSA patients	19,210	19,344	19,480	19,616	19,754	19,892	20,031	20,171	20,312	20,455	20,598	20,742	20,887	21,033	21,181	21,329	21,478	21,629	21,780	21,932
% of MSA patients seeking treatment	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
Price per patient annually	-	-	-	-	-	$ 40,000	$ 40,600	$ 41,209	$ 41,827	$ 42,455	$ 43,091	$ 43,738	$ 44,394	$ 45,060	$ 45,736	$ 46,422	$ 47,118	$ 47,825	$ 48,542	$ 49,270
% growth	-	-	-	-	-	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-		*3%*	*8%*	*13%*	*19%*	*21%*	*24%*	*25%*	*23%*	*20%*	*18%*	*15%*	*13%*	*13%*	*13%*	*6%*
						0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5	0.5
Gross-to-net adjustment						*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
MSA Revenue (MM) (E.U.)	-	-	-	-	$ -	$ 20	$ 61	$ 104	$ 159	$ 185	$ 211	$ 227	$ 209	$ 190	$ 170	$ 149	$ 127	$ 129	$ 132	$ 68
Total MSA Revenue (MM)	-	-	-	-	-	46	142	242	371	429	490	528	485	441	394	346	294	301	307	157

Source: BTIG Research Estimates

TZLS-401 (Foralumab) for AD Market Model

- We assume a 15% probability of success.
- We estimate the current prevalence of Alzheimer's Disease in the US is approximately 7.4 million patients in 2026 (Source: Alzheimer's Association website).
- We assume that the percentage of patients that are diagnosed and currently seeking treatment is 80%.
- We estimate that 50% of diagnosed patients fall under the early symptomatic and moderate disease bucket.
- We assume market entry of TZLS-401 for AD to be in 2032.
- We assume net prices per year to be $70,000 with an annual growth rate of 1.5%.
- We assume a peak market penetration of 5% of early symptomatic and moderate patients.

Exhibit 10. TZLS-401 (Foralumab) for AD Market Model

	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045
US Market																				
Alzheimer's Prevalence	7,400,000	7,451,800	7,503,963	7,556,490	7,609,386	7,662,651	7,716,290	7,770,304	7,824,696	7,879,469	7,934,625	7,990,168	8,046,099	8,102,422	8,159,139	8,216,253	8,273,766	8,331,683	8,390,004	8,448,734
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Diagnosed and seeking treatment	5,920,000	5,961,440	6,003,170	6,045,192	6,087,509	6,130,121	6,173,032	6,216,243	6,259,757	6,303,575	6,347,700	6,392,134	6,436,879	6,481,937	6,527,311	6,573,002	6,619,013	6,665,346	6,712,004	6,758,988
% diagnosed and seeking treatment	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Early Symptomatic & Moderate Disease	2,960,000	2,980,720	3,001,585	3,022,596	3,043,754	3,065,061	3,086,516	3,108,122	3,129,878	3,151,788	3,173,850	3,196,067	3,218,440	3,240,969	3,263,655	3,286,501	3,309,507	3,332,673	3,356,002	3,379,494
% early symptomatic and moderate	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*
Price per patient annually	-	-	-	-			$ 70,000	$ 71,050	$ 72,116	$ 73,197	$ 74,295	$ 75,410	$ 76,541	$ 77,689	$ 78,854	$ 80,037	$ 81,238	$ 82,456	$ 83,693	$ 84,949
% growth	-	-	-	-	-		*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-	*0%*	*0%*	*1%*	*2%*	*3%*	*4%*	*4%*	*5%*	*5%*	*5%*	*4%*	*4%*	*3%*	*3%*	*3%*	*1%*
							0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
AD Revenue (MM) (U.S.)	-	-	-	-	$ -	$ -	$ 972	$ 2,981	$ 5,079	$ 7,786	$ 9,019	$ 10,303	$ 11,085	$ 10,197	$ 9,265	$ 8,286	$ 7,259	$ 6,183	$ 6,320	$ 3,230

Source: BTIG Research Estimates

TZLS-401 (Foralumab) for ALS Market Model

- We assume a 15% probability of success.
- We estimate the current prevalence of ALS in the US is approximately 35,000 patients in 2026 (Source: American Medical Association Website).
- We assume that 80% of patients are diagnosed and currently seeking treatment.
- We assume market entry of TZLS-401 for ALS to be in 2032.
- We assume net prices per year to be $70,000 with an annual growth rate of 1.5%.
- We assume a peak market penetration of 15% of diagnosed ALS patients seeking treatment.

Exhibit 11. TZLS-401 (Foralumab) for ALS Market Model

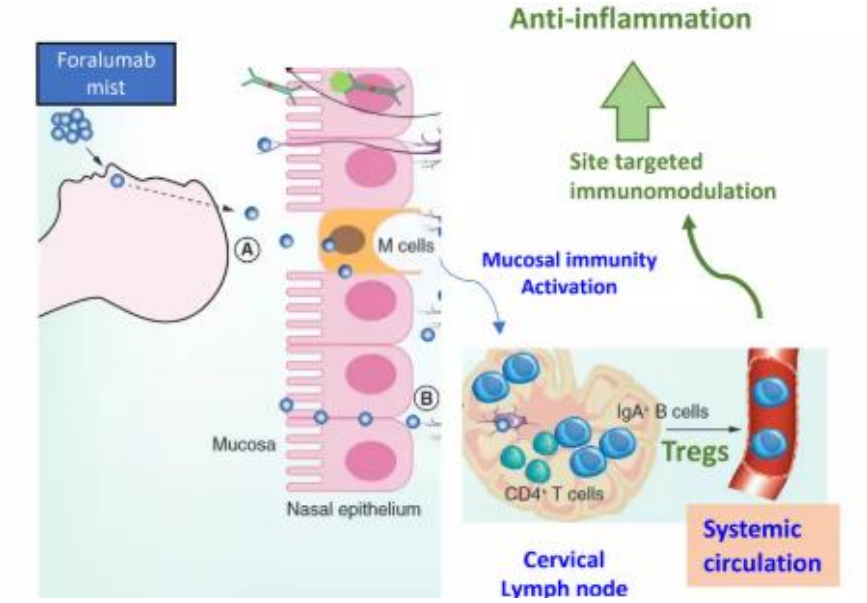
	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045
US Market																				
ALS Prevalence	35,000	35,245	35,492	35,740	35,990	36,242	36,496	36,751	37,009	37,268	37,529	37,791	38,056	38,322	38,591	38,861	39,133	39,407	39,682	39,960
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Diagnosed and seeking treatment	28,000	28,196	28,393	28,592	28,792	28,994	29,197	29,401	29,607	29,814	30,023	30,233	30,445	30,658	30,872	31,089	31,306	31,525	31,746	31,968
% diagnosed and seeking treatment	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Price per patient annually	-	-	-	-			$ 70,000	$ 71,050	$ 72,116	$ 73,197	$ 74,295	$ 75,410	$ 76,541	$ 77,689	$ 78,854	$ 80,037	$ 81,238	$ 82,456	$ 83,693	$ 84,949
% growth	-	-	-	-	-		*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
% penetration	-	-	-	-	*0%*	*0%*	*2%*	*5%*	*8%*	*11%*	*13%*	*14%*	*15%*	*14%*	*12%*	*11%*	*9%*	*8%*	*8%*	*4%*
							0.1	0.3	0.5	0.75	0.85	0.95	1	0.9	0.8	0.7	0.6	0.5	0.5	0.5
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
ALS Revenue (MM) (U.S.)	-	-	-	-	$ -	$ -	$ 28	$ 85	$ 144	$ 221	$ 256	$ 292	$ 315	$ 289	$ 263	$ 235	$ 206	$ 175	$ 179	$ 92

Source: BTIG Research Estimates

TIZIANA Secret Sauce.

The NALT default. One of the key features of the foralumab story is that nasal delivery results in delivery to cells that are biased toward Treg stimulation rather than inflammatory Teff production. In more detail, the nasal epithelial cells, under non-inflammatory conditions are constantly exposed to innocuous inhaled antigens. Most of these antigens must be ignored to avoid chronic inflammation. This situation is similar to the immune system in the intestine that also must ignore huge numbers of antigens to avoid triggering immune responses to food. As a result, the NALT is biased toward tolerance rather than immunity. This bias is enforced partly by the fact that in the nasal mucosa, antigens are delivered in low amounts (many antigens, each relatively rare) and the antigens are delivered without adjuvant (no danger signals). This type of immune stimulation generally results in a tolerogenic (Treg) over an inflammatory or effector (Teff) response.

Exhibit 12. Treg Production is Enriched in NALT.



Source: Tiziana company reports

NALT Tregs drive systemic and CNS effects. Tregs induced in NALT can exit NALT through the deep cervical lymph nodes and exert systemic suppressive effects (Exhibit 12). This trafficking has been seen in RA animal models where intranasal tolerance can suppress responses at peripheral joints far from the nasal mucosa.

NALT-derived Tregs meet CNS antigens. More relevant to the foralumab and Tiziana story, Tregs induced in NALT also traffic to deep cervical lymph nodes where they sit and wait while positioned to modulate CNS immune responses. This key positioning reflects the critical anatomy that these nodes that see NALT Tregs also receive meningeal lymphatic drainage from the CNS. This crossing is the key therapeutic loop that drove the development of foralumab: intranasal antigen delivery primes Tregs in NALT that migrate to the specific draining lymph nodes where CNS antigens are

presented. The approach still attracts definitive human clinical data but makes sense mechanistically.

Tolerogenic DC subsets are enriched in NALT. There are many kinds of dendritic cells - the great primers of specific immune responses. But the NALT is enriched in the key subset required for Treg induction. These Plasmacytoid DCs drive the production of IDO (indoleamine 2,3-dioxygenase) which suppress effector T cell proliferation and favor Foxp3 upregulation. For reference, IDO is a target in immune-oncology that was blocked in attempts to stimulate Teff production. TGF-β levels are also relatively high in NALT and this very pleiotropic cytokine is a central driver of Treg responses (the core molecular pathway for Foxp3 induction in naive CD4+ T cells in the periphery). In the absence of strong pro-inflammatory signals (IL-6, IL-12, IL-4), TGF-β drives conversion to Foxp3+ induced Tregs (pTregs/iTregs) rather than to Th17 or other effector lineages. IL-10 produced locally further reinforces this.

Nasal Delivery - No Drop in the Bucket

Nasal delivery is increasingly interesting as a route to target both lymphoid tissues (the goal of Tiziana) and to reach the CNS (goal of NeOnc's [NTHI, Buy, $15 TP] 212).

Immune stimulation. In this case, the target is the Nasal-Associated Lymphoid Tissue (NALT). This approach is probably the simplest as NALT tissue is the most directly accessible lymphoid structure in the body. In humans, the NALT is distributed across the nasal mucosa and overlapping with Waldeyer's ring (the sets of tonsils just after the nasopharynx). The power of immunizing via this route is demonstrated by intranasal vaccines that elicit strong mucosal IgA responses and are thought to generate some of the best protective vaccines (rather than systemic vaccines that reduce sequelae of infections).

Direct CNS delivery. Material small enough to traverse the olfactory epithelium can travel along the olfactory nerve through the cribriform plate and reach the subarachnoid space. From there, cerebrospinal fluid and interstitial fluid drain via meningeal lymphatics (dural lymphatic vessels running alongside dural sinuses) to the deep cervical lymph nodes particularly the nodes near the jugular foramen. This CNS-to-cervical-node axis has implications for neuroinflammation, brain tumor immunotherapy, and CNS vaccine strategies. Intranasal delivery of antigens or adjuvants can tap into this pathway and prime immune responses in the deep cervical nodes that are spatially associated with CNS drainage.

Control of pathways via particle sizes. The key variables that determine which nodes you reach intranasally are particle/droplet size (smaller particles penetrate deeper toward the olfactory epithelium; larger droplets deposit in the anterior turbinate region), formulation (mucoadhesive carriers extend residence time in NALT), volume (too large a volume clears quickly to the nasopharynx and is swallowed), and the use of transcytosis-enabling vehicles for crossing mucosal barriers. Formulations targeting the olfactory region specifically tend to use low volumes (< 10–15 µL in mice) delivered with the animal inverted.

Key Clinical Programs for Foralumab

The Tiziana clinical pipeline is built around intranasal foralumab and its ability to **increase Treg production and reduce the inflammation** in the brain that both potentiates and drives neurodegeneration. The approach could be active across neurology, and the company has chosen four lead indications that reflect both large market opportunities (AD) and strong unmet need (MSA). Importantly, all four indications share a common mechanism involving peripheral Treg induction via NALT stimulation that leads to reduced neuroinflammation. As a result, **each upcoming dataset reinforces the platform in addition to de-risking the specific indication**. The MS readout is the pivotal event; if INFORM-MS shows a significant effect on TSPO-measured inflammation along with some hints of clinical benefit, it would substantially de-risk the ALS and AD programs as well.

After SPMS. MSA is probably the next biggest source of future investor interest based on almost completely unmet medical need. The AD and ALS programs both face competition as inflammation is a well-known driver of both diseases. However, the sophistication of the Treg approach seems to increase the hope that destructive inflammation can be turned down without allowing opportunistic infections to occur. Below, we review each of the four programs with an eye to the data supporting inflammation as a driver of the disease, the data to date suggest foralumab is a reasonable option to try, and finally, the unmet medical need for the target patient populations.

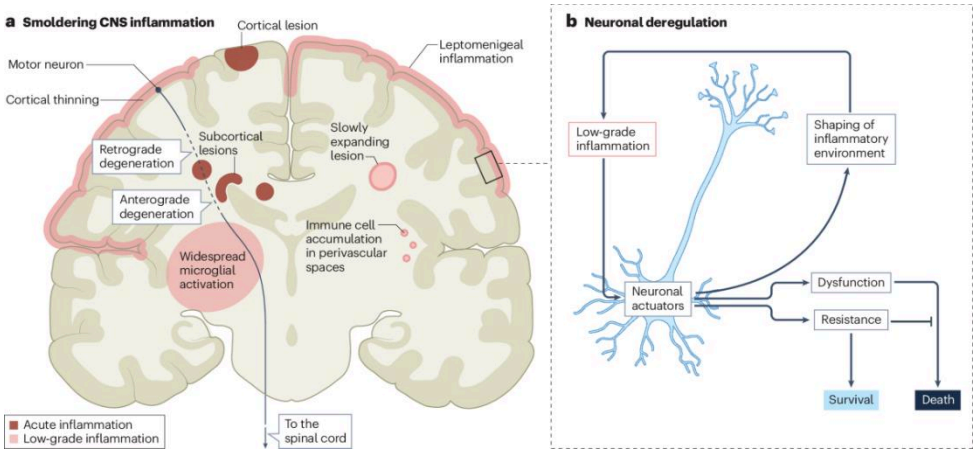
Lead program: SPMS – strong rationale and good supporting data

SPMS – the lead indication. The therapeutic justification for developing foralumab in na-SPMS makes good commercial and medical sense as there is little else to help these patients. The disease is a significant opportunity as approximately 25%-30% of the ~850,000 people with relapsing-remitting MS (the T-Cell phase) will transition to secondary progressive disease over their lifetime (the MG phase). Based on multiple clinical trials, once MS patients become non-active (no new relapses, no gadolinium-enhancing lesions) they lose benefits from current disease-modifying therapies and face an uninterrupted trajectory of disability accumulation.

Key potential de-risking of entire story in October. The INFORM-MS trial — a randomized, double-blind, placebo-controlled study, with 48 patients, 12 weeks of treatment — completed enrollment in May 2026 and dosed its last patient on June 25, 2026. Topline data are expected in October, with results likely to be presented at ACTRIMS/ECTRIMS in Toronto in October 2026. This readout has the potential to de-risk the entire pipeline if the reductions in TSPO imaging signals (the biomarker for reduced inflammation) correlate with clinical symptoms such as fatigue. It's a lot to ask for in a 12-week treatment experience and, based on the significant validation of the TSPO signal, we would see reduction in this biomarker in a large majority of patients (~75%) as a positive result.

The Role of Neuroinflammation in SPMS Progression. Contrary to older conceptualizations of progressive MS as a purely degenerative process, a substantial and growing body of neuropathological and imaging evidence has established that CNS-intrinsic neuroinflammation is a mechanistically active and central driver of SPMS progression. The primary cellular mediators of this process are microglia, the resident immune cells of the brain and spinal cord, along with dysfunctional astrocytes. In naSPMS, activated microglia are diffusely distributed throughout the white matter and cortex, perpetuating a state of smoldering, low-grade inflammation that promotes ongoing axonal damage and neuronal loss even in the complete absence of peripheral immune system activity. This compartmentalized inflammatory state is qualitatively distinct from the acute pathology of RRMS: it operates at lower intensity but with far greater chronicity, and it is not addressed by conventional immunosuppressive or anti-trafficking agents. Advanced PET imaging using radioligands targeting translocator protein (TSPO), a validated marker of microglial activation, has provided a critical in-vivo window into this process, with TSPO-PET burden correlating with both current disability severity and future disease progression in progressive MS patients. This biology has profound implications for drug development: any meaningful therapeutic strategy for naSPMS must engage compartmentalized CNS neuroinflammation directly, rather than simply reducing peripheral immune trafficking into the CNS.

Exhibit 13. CNS inflammation in MS disease pathology



Source: Woo, M.S., et al., *Nat. Rev. Neurosci.* **25**, 493–513 (2024).

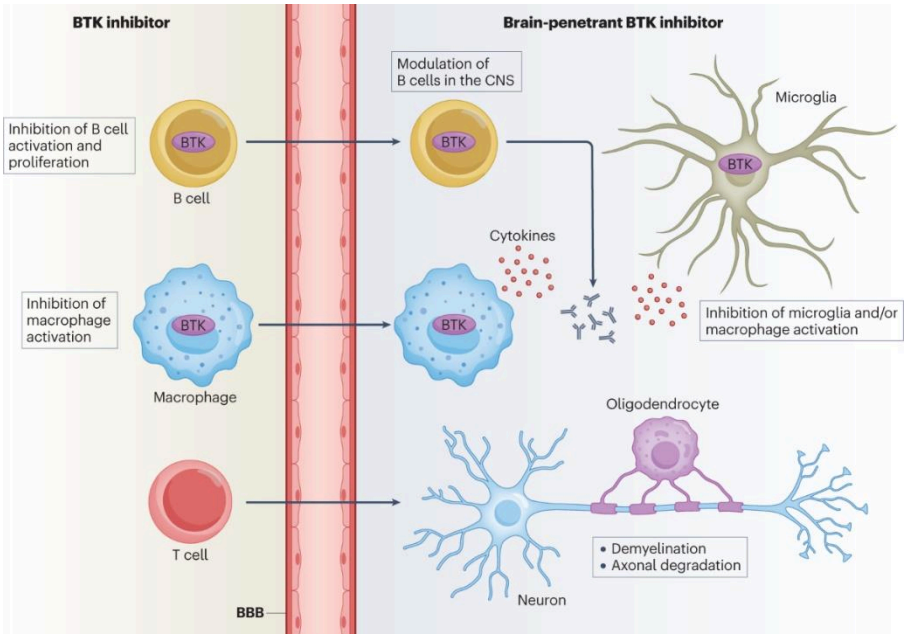
Standard of Care and the naSPMS Treatment Vacuum

The MS treatment landscape has been transformed over the past two decades, with more than 20 FDA-approved disease-modifying therapies (DMTs) available for relapsing forms of the disease. These span multiple drug classes, from injectable interferons and glatiramer acetate at the older, moderately efficacious end, to oral agents including dimethyl fumarate, teriflunomide, and sphingosine-1-phosphate modulators, to high-efficacy intravenous anti-CD20 B-cell depleting agents, primarily ocrelizumab and ofatumumab, which now anchor the treatment of active disease. For active SPMS specifically, siponimod (Mayzent, Novartis [NVS, Not Rated]) received FDA approval in 2019 and demonstrated the ability to reduce relapse

frequency and modestly slow disability progression, but its benefit is largely confined to patients with residual inflammatory activity, and its utility in naSPMS patients, where the inflammatory driver is wholly compartmentalized, is limited. For naSPMS, there are currently no FDA-approved therapies. This represents a significant unmet need affecting an estimated over a hundred thousand U.S. patients, for whom the standard of care amounts to symptom management and supportive care.

The closest the field has come to filling this gap is through Bruton's tyrosine kinase (BTK) inhibitors, a class of oral small molecules with CNS penetrance capable of targeting B cells and myeloid cells, including microglia, within the brain. Tolebrutinib (Sanofi, SNY, Not Rated) is the most advanced candidate in this class for naSPMS, and its Phase 3 HERCULES trial demonstrated a 31% reduction in time to confirmed disability progression, a clinically meaningful result. However, this benefit was accompanied by a hepatotoxicity signal, with 4.1% of patients experiencing liver function test abnormalities, including one death attributed to hepatic toxicity. Additionally, separate tolebrutinib RRMS trials, GEMINI 1 and 2, failed to demonstrate superiority over teriflunomide in reducing relapses, and the drug was associated with higher gadolinium-enhancing lesion accumulation. Taken together, even the most advanced naSPMS candidate to date carries a meaningful safety liability and fails to address the full disease picture, leaving the field in clear need of additional, mechanistically differentiated approaches.

Exhibit 14. Proposed mechanism of BTK inhibitors for MS



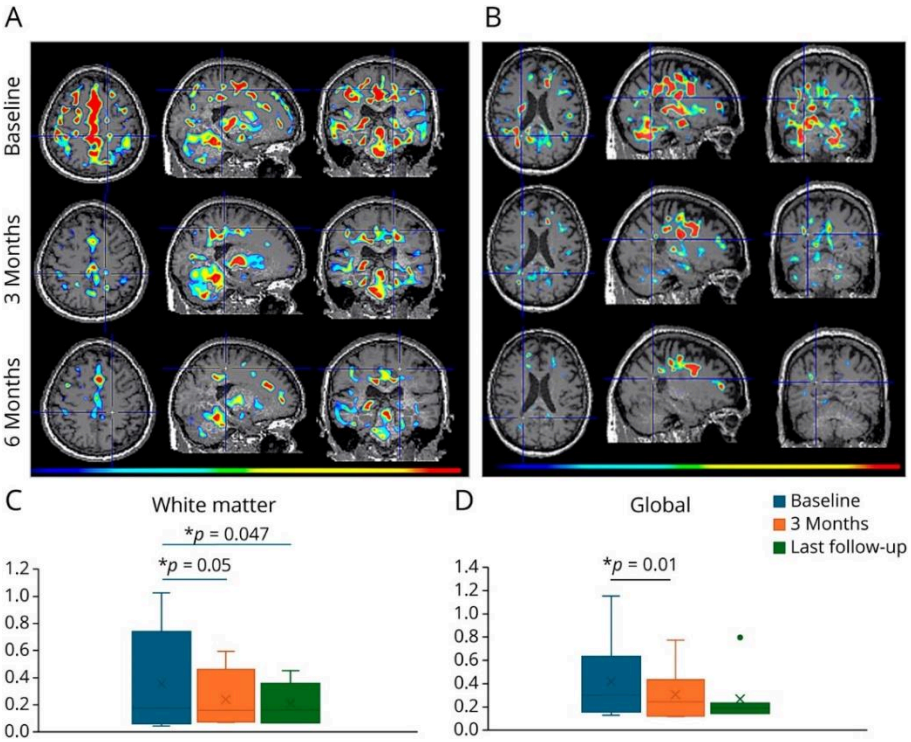
*Source: Krämer, J., et al. Nat Rev Neurol **19**, 289–304 (2023).*

Foralumab: A Novel Tolerogenic Strategy Targeting CNS Neuroinflammation

Against this backdrop, Tiziana Life Sciences is developing intranasal foralumab, the only fully human anti-CD3 monoclonal antibody currently in clinical development, as a novel immunomodulatory strategy for naSPMS. Rather than broadly suppressing peripheral immunity, foralumab is administered nasally to engage the mucosa-

associated lymphoid tissue of the nasal cavity, inducing a tolerogenic immune response characterized by the expansion of regulatory T cells (Tregs). These Tregs migrate to sites of CNS inflammation and suppress microglial activation, directly targeting the compartmentalized neuroinflammatory process that drives SPMS progression, without systemic T-cell depletion or the cytokine release syndromes associated with IV CD3-directed approaches. The nasal delivery route is a key mechanistic differentiator, enabling immune modulation through the mucosal immune system without CD3 receptor downregulation or systemic adverse effects, and exploiting a well-established principle of mucosal tolerance induction.

Exhibit 15. Nasal foralumab Reduced TSPO-PET signal



Source: Chitnis, T., et al. Neurol Neuroimmunol Neuroinflamm 2026; 13:e200543.

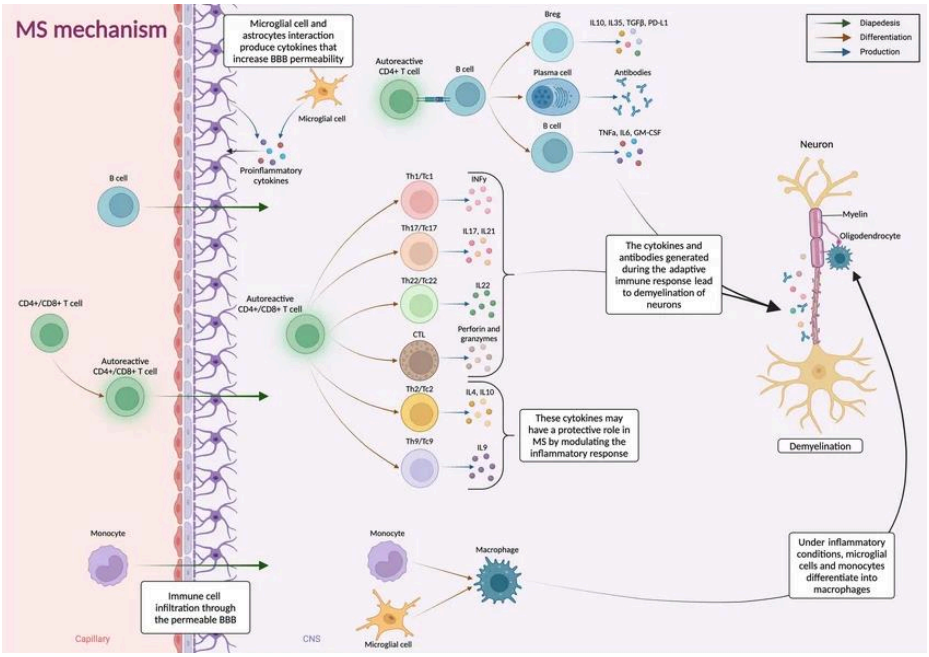
Initial clinical evidence from Tiziana's expanded access program, published in peer-reviewed form in Neurology: Neuroimmunology and Neuroinflammation, offers early proof-of-concept support for this approach in human naSPMS. In ten treatment-refractory naSPMS patients treated with nasal foralumab, the study reported EDSS stabilization, clinically meaningful improvements in fatigue scores, and, critically, direct in-vivo reductions in microglial TSPO-PET signal, with 80% of patients showing qualitative PET improvement after six months of treatment. Quantitative white matter Z-scores, a validated measure of microglial activation burden, improved by a median of 36% in early cohorts. Single-cell RNA sequencing of peripheral blood samples identified immune biomarker changes consistent with the proposed mechanism, including upregulation of FoxP3+ Tregs and modulation of TGF-beta pathways across multiple immune cell populations. These data are derived from a small, open-label, uncontrolled study and cannot establish clinical efficacy in isolation, but they provide mechanistic coherence, biological plausibility, and a compelling basis for the

ongoing Phase 2a randomized program. That study, a double-blind, placebo-controlled trial evaluating 50 µg and 100 µg intranasal doses in naSPMS patients continuing to progress despite standard of care, is currently enrolling at leading academic MS centers including Brigham and Women's Hospital, Yale, Johns Hopkins, Cornell, UMass, and Thomas Jefferson University, with topline data expected in 2026. In July 2024, the FDA granted Fast Track designation to intranasal foralumab for naSPMS, formal recognition of both the severity of the condition and the stark absence of approved alternatives.

Background: Multiple Sclerosis: Overview and Disease Biology

Multiple sclerosis (MS) is a chronic, immune-mediated disease of the central nervous system (CNS) characterized by demyelination, axonal injury, and progressive neurodegeneration. Affecting approximately 2.9 million people worldwide, with roughly one million in the United States, MS is the leading non-traumatic cause of neurological disability in young adults, with onset typically occurring between the ages of 20 and 50. The disease is more prevalent in women, at a ratio of approximately 3:1 in relapsing forms. While the precise etiology remains incompletely understood, MS is broadly characterized by a dysregulated immune response in genetically susceptible individuals, in which autoreactive T cells and B cells breach the blood-brain barrier and attack myelin sheaths, the insulating protein coat surrounding CNS axons, disrupting electrical signal conduction. The resulting lesions produce the hallmark clinical features of the disease: motor weakness, sensory disturbance, visual loss, fatigue, and cognitive impairment. Over time, as the disease evolves, neurodegeneration becomes an increasingly dominant pathological process, contributing to irreversible disability accumulation that standard anti-inflammatory therapies are poorly equipped to address.

Exhibit 16: Multiple Sclerosis is an immune related disease

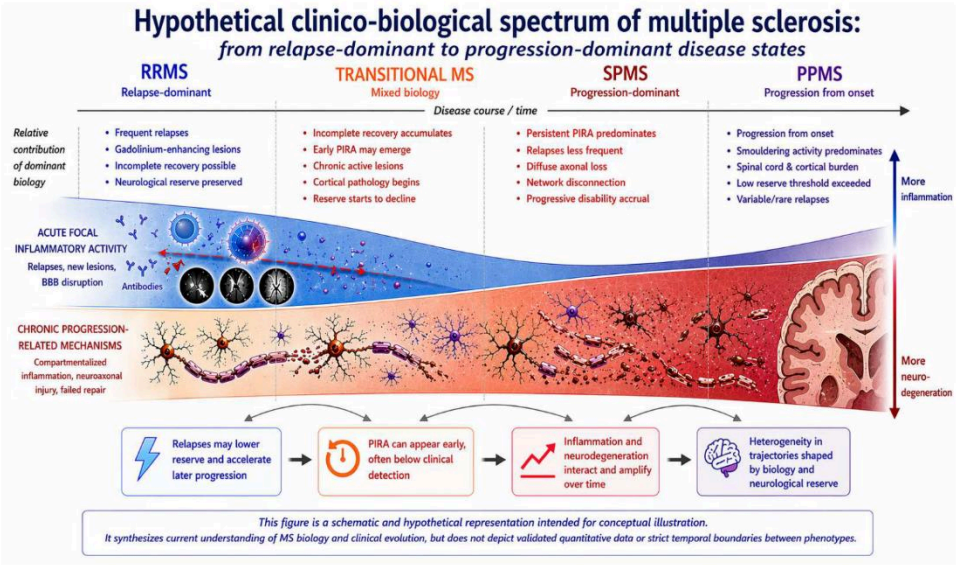


Source: Zabihi, M., et al., (2024). Molecular Neurobiology. 62. 3396-3411.

Disease Subtypes: The Relapsing-to-Progressive Continuum

MS is not a single disease entity but a spectrum of related conditions, broadly classified into relapsing and progressive subtypes. The most common form, relapsing-remitting MS (RRMS), accounts for approximately 85% of diagnoses at onset and is characterized by discrete clinical attacks, or relapses, separated by periods of partial or complete recovery. A distinct minority, roughly 15% of patients, present with primary progressive MS (PPMS), in which neurological decline is continuous from the outset without an antecedent relapsing phase. Over time, a substantial proportion of RRMS patients transition into secondary progressive MS (SPMS), in which steady neurological worsening becomes the dominant feature, with or without continued relapse activity. This transition typically occurs 10 to 20 years after initial diagnosis and is often difficult to identify in real time. Clinically, SPMS is further sub-classified as "active," when new relapses or new and enlarging MRI lesions remain present, or "non-active" (naSPMS), in which patients no longer exhibit signs of acute peripheral inflammation but continue to experience relentless neurological worsening. It is this non-active phenotype that represents the most underserved and pharmacologically neglected stage of the disease.

Exhibit 17: MS disease subtypes



Source: Vasilev, G. V., et al., Neurology International, 18(5), 86 (2026).

Secondary Progressive MS: The Silent Phase of Disability Accumulation

In naSPMS, patients have moved beyond the reach of nearly every tool that defines modern MS management. Without the visible footprints of gadolinium-enhancing lesions or clinical relapses, these patients are effectively invisible to conventional MRI surveillance and are excluded from the treatment paradigms built around anti-inflammatory suppression of peripheral immune activity. Yet they continue to accumulate disability steadily, losing mobility, dexterity, and cognitive function over years, driven by a process that operates largely independent of the acute inflammatory mechanisms targeted by existing therapies. This phenomenon, now

formally characterized as progression independent of relapse activity (PIRA), has emerged as one of the most important concepts in modern MS research and represents the central therapeutic challenge in naSPMS. PIRA is driven not by blood-brain barrier breakdown and peripheral lymphocyte infiltration, but by chronic, compartmentalized CNS-intrinsic inflammation that persists behind an intact or reconstituted barrier, largely inaccessible to the agents that dominate the MS formulary.

MSA. Good rationale, some corroborative data, unmet need.

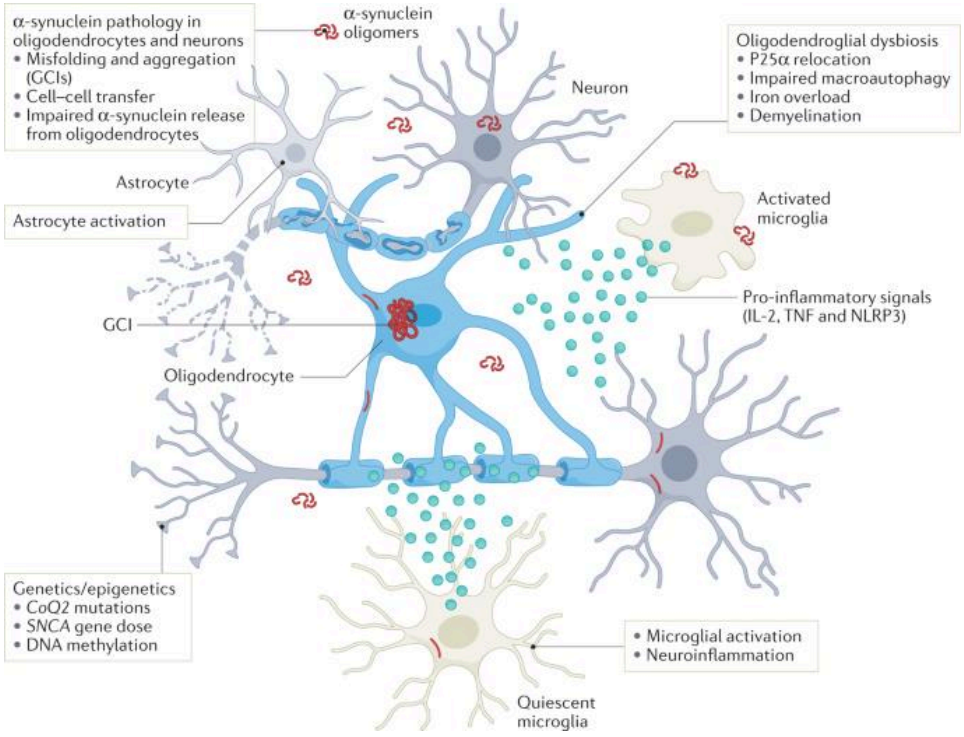
The MSA program – high need, decent validation and no competition. MSA represents a smaller commercial opportunity than SPMS but arguably even more scientific urgency. MSA is uniformly fatal, typically kills within six to ten years of diagnosis, and has no approved disease-modifying treatment. The consistent TSPO PET signal across three consecutive treated patients is a provocative finding and should be scrutinized more closely if the SPMS data in October support the overall approach.

The ongoing MSA trial is more of a Compassionate Use / Case Series but the data to date have been interesting, and the medical need is huge. In the most recent update (July 2026) Tiziana reported reduction in brain inflammation in a third MSA patient treated with intranasal foralumab. The company expects to report a final patient from this first cohort and then follow up with a second cohort of patients in the steep part of the decline curve for the disease. This first set of ~10 patients is expected to form the basis of an FDA discussion around a pivotal trial design and the company will likely seek BTD.

The Role of Neuroinflammation in MSA Progression. Neuroinflammation, mediated primarily by microglial activation, is now recognized as a central and mechanistically active driver of MSA progression rather than a secondary consequence of oligodendroglial and neuronal loss. Post-mortem neuropathological studies have documented extensive microglial activation in the striatum, substantia nigra, pons, inferior olivary nuclei, and cerebellar white matter, regions that correspond precisely to areas of maximal GCI burden and neurodegeneration in MSA. Activated microglia in this context adopt a pro-inflammatory phenotype, releasing reactive oxygen species, pro-inflammatory cytokines, and complement factors that amplify oligodendrocyte dysfunction, disrupt myelin integrity, and accelerate neuronal loss in a self-sustaining cycle that operates in parallel with, and is potentiated by, ongoing alpha-synuclein aggregation. The mechanistic relationship between alpha-synuclein and microglial activation is bidirectional: extracellular alpha-synuclein fibrils are potent microglial activators through TLR4 and NLRP3 inflammasome pathways, while chronically activated microglia impair the phagocytic clearance of alpha-synuclein, establishing a feed-forward loop that accelerates both pathological burden and clinical decline. TSPO-PET imaging has provided in-vivo evidence of this microglial burden in MSA patients, with elevated TSPO signal consistently localizing to the basal ganglia and cerebellar white matter, the two regions most directly implicated in functional disability across both MSA

subtypes and establishing a neuroimaging framework directly applicable to clinical trial endpoint design. This biology positions microglial suppression as a rational and tractable therapeutic target in MSA, independent of whether alpha-synuclein aggregation can be directly reversed.

Exhibit 18. Pathophysiological mechanisms in MSA



*Source: Poewe, W., et al. Multiple system atrophy. Nat Rev Dis Primers **8**, 56 (2022).*

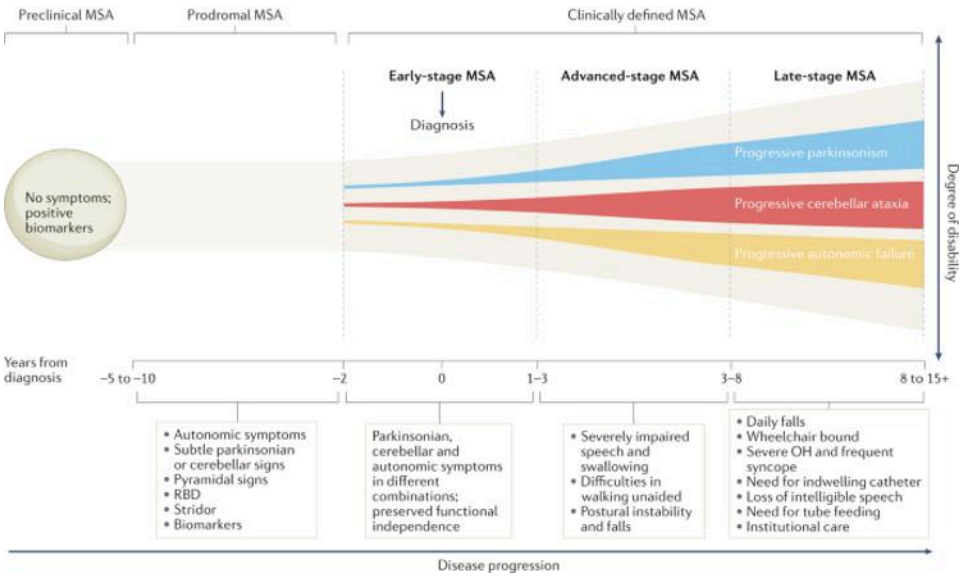
Foralumab: Early Signal Generation in MSA

Against this background, Tiziana initiated a Phase 2a, open-label, six-month clinical trial of intranasal foralumab in MSA patients in August 2025, with the first participant enrolled and dosed at Brigham and Women's Hospital in Boston. The trial carries FDA orphan disease designation and evaluates foralumab's ability to reduce neuroinflammation through Treg-mediated microglial suppression, applying the same mechanism of action established across the naSPMS and AD programs to a third, distinct neuroinflammatory CNS context. The program is at a very early stage, and no clinical outcome data have been reported. Tiziana has, however, released quantitative TSPO-PET imaging results from the first three MSA patients to complete dosing, and the consistency of the signal across all three provides an early and encouraging biological read on foralumab's target engagement in this indication. In the first two patients, treatment was associated with up to approximately 35% reduction in standardized uptake value (SUV) and 24% reduction in SUVR in the basal ganglia and cerebellar white matter, the brain regions most clinically relevant to MSA. In the third patient, results were comparable, with up to 34% reduction in SUV and 26% reduction in SUVR in the same disease-relevant regions. Principal investigator Dr. Tarun Singhal of the Ann Romney Center for Neurologic Diseases at Brigham and

Women's Hospital noted the consistency of findings across patients as encouraging, while appropriately calling for correlation with clinical features and further validation with additional quantitative techniques.

These data are preliminary by design and must be interpreted accordingly. This is a small, open-label, signal-generating program, and the PET reductions reported reflect imaging biomarker changes rather than clinical endpoints. The absence of a placebo control, very limited patient numbers, and lack of reported functional or quality-of-life data mean that efficacy conclusions cannot be drawn at this stage. However, the reproducibility of the neuroinflammatory signal reduction across three consecutive patients in a disease with no approved therapies, combined with the mechanistic coherence of foralumab's tolerogenic approach to MSA's microglial-driven pathology, provides a credible rationale for continued development, in our view. Tiziana plans to enroll additional patients in the ongoing Phase 2a program, with further PET imaging data and clinical outcome measures expected as the trial matures.

Exhibit 19. Progression of MSA



*Source: Poewe, W., et al. Multiple system atrophy. Nat Rev Dis Primers **8**, 56 (2022).*

Background: Multiple System Atrophy: Overview, Pathophysiology, and Unmet Need

Multiple system atrophy (MSA) is a rare, rapidly progressive neurodegenerative disorder characterized by a clinical triad of autonomic dysfunction, parkinsonism, and cerebellar ataxia, in variable combinations depending on disease subtype. MSA-P, the parkinsonian-predominant subtype, is defined by bradykinesia, rigidity, and postural instability with limited or transient levodopa responsiveness, while MSA-C, the cerebellar-predominant subtype, presents with gait ataxia, limb incoordination, and dysarthria. Autonomic failure, manifesting as orthostatic hypotension, urogenital dysfunction, and sudomotor impairment, is a universal and defining feature across

both subtypes. The disease is relentlessly progressive, with a median survival of only 6 to 9 years from symptom onset, and the pace of functional decline substantially exceeds that seen in Parkinson's disease. MSA is classified as an orphan disease, affecting an estimated 15,000 to 50,000 patients in the United States. Pathologically, MSA is distinguished from other synucleinopathies by the accumulation of alpha-synuclein into glial cytoplasmic inclusions (GCIs) within oligodendrocytes rather than in neurons, with GCI burden concentrated in the striatonigral and olivopontocerebellar systems, producing the pattern of selective oligodendroglial and neuronal loss that underlies the clinical phenotype. There are currently no FDA-approved disease-modifying therapies for MSA. Available care consists entirely of symptomatic management, including levodopa, which provides only modest and transient benefit in a minority of MSA-P patients, fludrocortisone and midodrine for autonomic dysfunction, and supportive physical and speech therapy. The absence of any approved agent capable of altering disease course represents one of the most profound unmet needs in movement disorder neurology.

Alzheimer's Disease – huge market that needs another drug

Alzheimer's Disease. The P2 trial began enrollment in late 2025 and the first patient was dosed in December 2025. The trial includes two complementary arms:

- as a monotherapy targeting neuroinflammation, and
- in combination with lecanemab or donanemab.

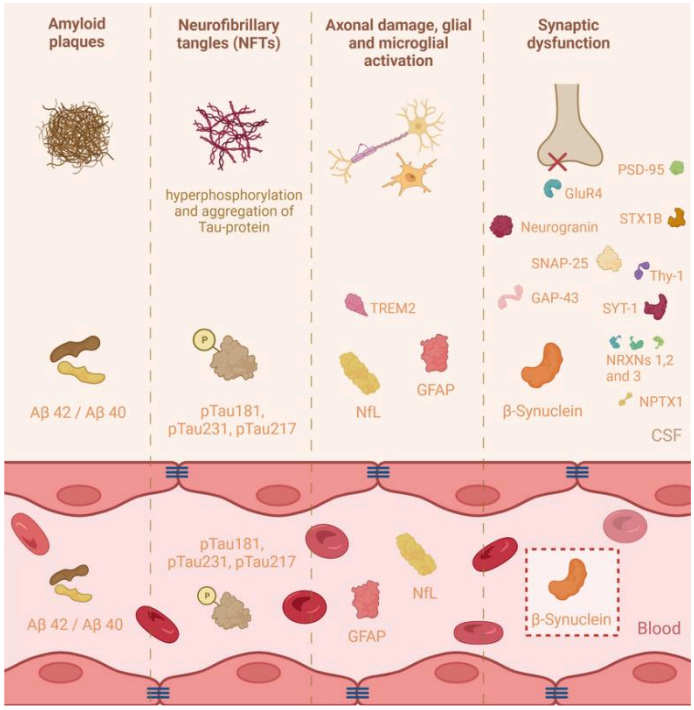
The combination rationale is directly grounded in the lecanemab TSPO-PET observation: given that anti-amyloid agents clear plaques but leave microglial activation unresolved, layering a Treg-inducing agent has the potential to address the residual neuroinflammatory burden that persists after amyloid reduction. The dual-arm design positions foralumab both as a standalone option for patients ineligible for anti-amyloid therapy, including those with moderate disease or APOE4 homozygosity, and as a rational combination partner to the emerging anti-amyloid standard of care. Primary endpoints include microglial activation changes on TSPO-PET, cognitive assessments, and biomarker measures of amyloid, tau, NfL, and GFAP, with a primary readout at three months.

The AD trial is again supported by decent mechanistic data as Tiziana previously reported a significant reduction in microglial activation on PET scans in a moderate AD patient. Data are expected in 1H27 and larger trials are expected to be part of a partnership (n>1,000). Tiziana's Phase 2 clinical trial, which enrolled its first patient in December 2025, evaluates intranasal foralumab in early AD.

The Role of Neuroinflammation in AD Progression. Neuroinflammation has emerged as a third core pathological axis in AD, now widely recognized as an active driver of progression rather than a secondary consequence. Microglia respond to Abeta plaques, dystrophic axons, and phosphorylated tau by transitioning from a homeostatic to a disease-associated microglial (DAM) state, characterized by upregulation of TREM2, APOE, and complement pathways. While initially serving a plaque-clearing function, chronically activated microglia shift toward a pro-

inflammatory, neurodegenerative phenotype that amplifies synaptic pruning, promotes tau propagation across Braak stages, and accelerates neuronal loss. TSPO-PET imaging, an established in-vivo measure of microglial activation density discussed in the MS section, has confirmed that microglial burden increases with disease stage, co-localizes with high amyloid burden regions including the precuneus and posterior cingulate, and independently predicts cognitive decline. Critically, a TSPO-PET study in a patient on active lecanemab treatment demonstrated persistent, widespread microglial activation despite effective amyloid plaque reduction, confirming that clearing amyloid does not extinguish the underlying neuroinflammatory response and pointing directly to the therapeutic rationale for agents capable of modulating microglial activation independently.

Exhibit 20. Pathophysiological hallmarks of AD and its associated biomarkers



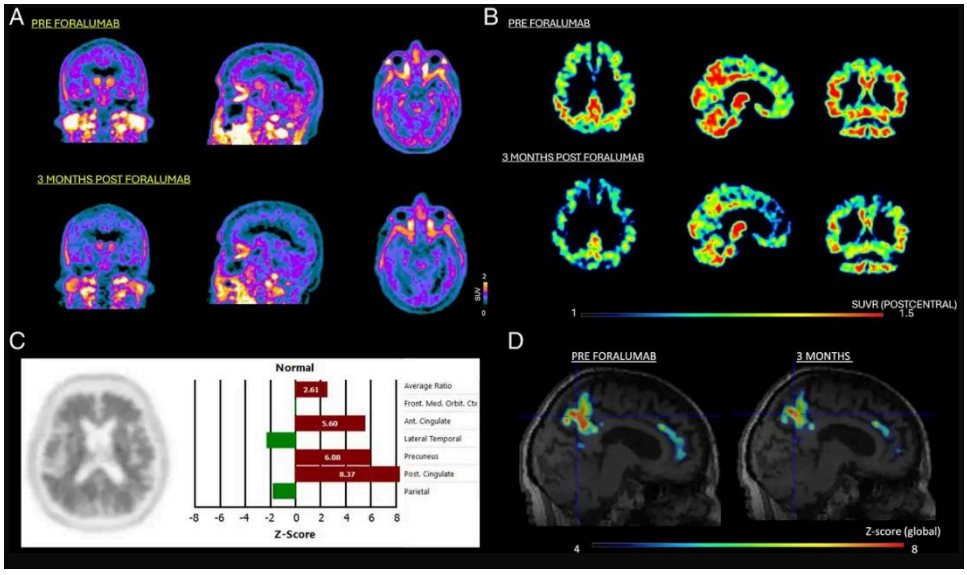
Source: Mohaupt, P., et al. (2022). Alzheimer's Research & Therapy. 14. 179.

Foralumab: Targeting Neuroinflammation as Monotherapy and in Combination

Tiziana is developing intranasal foralumab as a mechanistically differentiated approach to AD, targeting the neuroinflammatory axis through Treg-mediated suppression of microglial activation. As in naSPMS, nasal delivery engages mucosal lymphoid tissue to induce tolerogenic Tregs that migrate to the CNS and dampen microglial activity without systemic immunosuppression. Preclinical validation in the 3xTg AD mouse model demonstrated that nasal anti-CD3 reduced microglial activation and improved cognition independently of Abeta deposition, with gene expression analysis confirming decreased oxidative stress, increased axogenesis and synaptic organization, and hippocampal and cortical metabolic improvements.

The first human evidence, published by Singhal et al. in Clinical Nuclear Medicine (2025), reported on a 78-year-old patient with moderate AD treated under FDA expanded access. 18F-PBR06 TSPO-PET demonstrated diffuse reduction in radiotracer uptake throughout the brain at three months, with particularly notable decreases in the precuneus, posterior cingulate, and anterior cingulate gyri — regions of high baseline amyloid burden on 18F-Florbetapir-PET. Peripheral blood transcriptional analysis confirmed broad immunomodulatory effects across CD4, CD8, and monocyte populations, and unexpectedly identified upregulation of phagocytosis markers in classical monocytes, raising the hypothesis of enhanced peripheral amyloid clearance as a secondary mechanism. These findings are consistent with the microglial dampening effects observed in the naSPMS expanded access program, suggesting mechanistic generalizability across neuroinflammatory CNS conditions.

Exhibit 21. Dampening of Microglial Activation With Nasal Foralumab

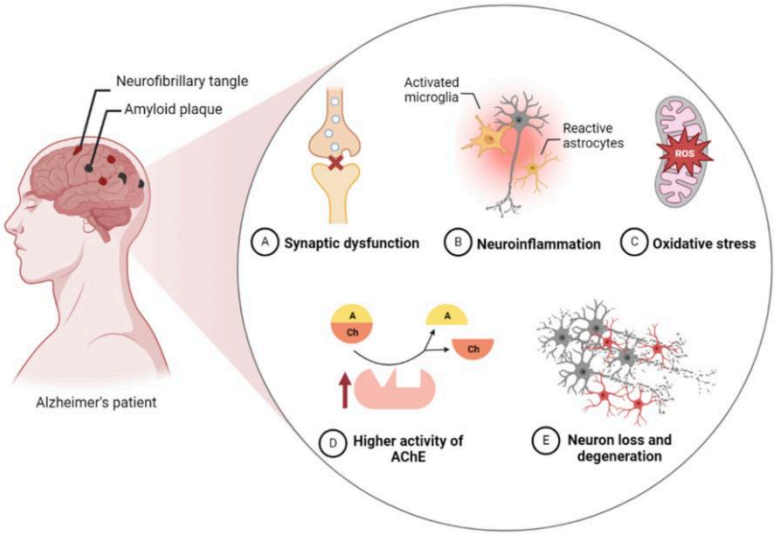


Source: Singhal, T., et al. Clinical Nuclear Medicine 50(8):p 756-757 (2025).

Tiziana's Phase 2 clinical trial, which enrolled its first patient in December 2025, evaluates intranasal foralumab in early AD across two complementary arms: as a monotherapy targeting neuroinflammation, and in combination with lecanemab or donanemab. The combination rationale is directly grounded in the lecanemab TSPO-PET observation: given that anti-amyloid agents clear plaques but leave microglial activation unresolved, layering a Treg-inducing agent has the potential to address the residual neuroinflammatory burden that persists after amyloid reduction. The dual-arm design positions foralumab both as a standalone option for patients ineligible for anti-amyloid therapy, including those with moderate disease or APOE4 homozygosity, and as a rational combination partner to the emerging anti-amyloid standard of care. Primary endpoints include microglial activation changes on TSPO-PET, cognitive assessments, and biomarker measures of amyloid, tau, NfL, and GFAP, with a primary readout at three months.

Alzheimer's Disease: Overview and Pathophysiology. Alzheimer's disease (AD) is a progressive, irreversible neurodegenerative disorder and the leading cause of dementia, accounting for 60% to 80% of all dementia cases. Approximately 7.4 million Americans currently live with AD, a figure projected to nearly double by 2050. Pathologically, AD is defined by two hallmark proteinopathies: extracellular amyloid-beta (Abeta) deposition into senile plaques and intraneuronal accumulation of hyperphosphorylated tau into neurofibrillary tangles, both of which disrupt synaptic integrity and propagate neurodegeneration along established anatomical networks. The amyloid cascade hypothesis, which posits Abeta accumulation as the initiating pathological event upstream of tau propagation and synaptic loss, has anchored AD drug development for three decades. The revised 2024 NIA-AA biological staging framework, supported by amyloid-PET, tau-PET, CSF and plasma biomarkers including Abeta42/40, phosphorylated tau 181 and 217, NfL, and GFAP, now defines AD along a continuous biological spectrum from preclinical amyloid accumulation through MCI to overt dementia.

Exhibit 22. Alzheimer's disease hallmarks and neuropathological features



Source: Ribeiro, J., et al. *Molecules* **2023**, *28*(16), 6015.

Standard of Care and Remaining Unmet Need. Two anti-amyloid monoclonal antibodies have achieved FDA approval with demonstrated clinical benefit: lecanemab/Leqembi (Eisai, ESAIY, Not Rated and Biogen, BIIB, Neutral), granted traditional approval in June 2023 for early AD, and donanemab/Kisunla (Eli Lilly), approved in July 2024 for early symptomatic AD with confirmed amyloid pathology, notably the first anti-amyloid therapy to support treatment discontinuation once plaques are eliminated. Both agents demonstrated statistically significant slowing of clinical progression in Phase 3 trials, representing the first disease-modifying approvals in AD. However, both carry a class-wide risk of amyloid-related imaging abnormalities (ARIA), require MRI monitoring, and restrict use in APOE4 homozygotes given substantially elevated ARIA risk. Critically, both are approved only for early symptomatic disease, and neither has demonstrated benefit in moderate or advanced AD, where neuroinflammatory and neurodegenerative cascades are well

established. For moderate AD, no FDA-approved disease-modifying therapy exists. Symptomatic agents, primarily acetylcholinesterase inhibitors and the NMDA receptor antagonist memantine, offer modest, temporary benefit with no impact on underlying disease biology, and the neuroinflammatory axis remains entirely unaddressed across all approved paradigms.

ALS – Waiting for foralumab data to date but need and rationale are strong

ALS is the final clinical target for foralumab and is not yet backed by imaging data from Tiziana. The disease is highly inflammatory in nature and the unmet need is high. In addition, there are networks that are willing to help with clinical trials (described below). Overall, we expect most investor interest in the SPMS and MSA programs, but if those trials readout positively, we would expect foralumab to move to the front of the line for promising ALS therapies.

The Role of Neuroinflammation and Treg Dysfunction in ALS Pathogenesis. Neuroinflammation is one of the most extensively characterized and mechanistically compelling pathological features of ALS, and the Treg axis in particular has emerged as a quantitatively important and clinically actionable dimension of disease biology. Post-mortem neuropathological studies have documented robust microglial activation throughout the motor cortex, corticospinal tracts, and anterior horn of the spinal cord in ALS patients, with microglial phenotype shifting from homeostatic to a chronically activated, pro-inflammatory state that releases reactive oxygen species, glutamate, and pro-inflammatory cytokines including IL-1beta, TNF-alpha, and IL-6, compounding motor neuron injury and accelerating disease spread. Activated microglia in ALS also impair the phagocytic clearance of misfolded TDP-43 and SOD1 aggregates, creating a feed-forward loop in which protein aggregation and neuroinflammation mutually amplify one another. This inflammatory cascade is not confined to the CNS: ALS patients exhibit a well-characterized and quantified systemic immune imbalance characterized by a significant reduction in both the number and suppressive function of peripheral Tregs, with Treg deficiency correlating independently with faster rates of ALSFRS-R decline and shorter survival.

Firm Treg stomping ground. The mechanistic link between peripheral Treg dysfunction and CNS neuroinflammatory burden is direct: Tregs normally traffic to the CNS and restrain microglial activation, promote a neuroprotective microglial phenotype, and suppress the infiltration and pathogenic activity of effector T cell populations at sites of motor neuron injury. When Treg numbers are reduced or their function is impaired, as is consistently observed in ALS, this regulatory brake is lifted, and unchecked neuroinflammation and microglial activation contribute materially to motor neuron death and disease progression. Importantly, the Treg deficiency in ALS is not simply a late-stage epiphenomenon: longitudinal studies have demonstrated that Treg dysfunction tracks with disease tempo across the full clinical course, making it both a prognostic biomarker and a rational pharmacological target at multiple stages of the disease. High levels of glutamate-driven excitotoxicity provide an additional and intersecting source of chronic neurotoxicity that further compounds microglial-mediated damage, underscoring the importance of

approaches capable of simultaneously addressing multiple converging injury mechanisms. The **well-characterized, quantitative relationship between Treg function, microglial activation, and the rate of clinical progression** establishes the Treg-microglial axis as among the most compelling and biologically supported intervention points in the current ALS drug development landscape.

Foralumab: Mechanistic Rationale and the BANYAN Trial. The convergence of Treg deficiency and microglial activation as active, quantifiable drivers of ALS progression provides a direct mechanistic rationale for intranasal foralumab in this indication. As established across the naSPMS and AD programs, nasal delivery of foralumab engages cervical lymph nodes via mucosal immune pathways, expanding and activating Tregs that subsequently migrate to the CNS to suppress pathogenic microglial activity and restore immune homeostasis, without systemic immunosuppression. In ALS, this mechanism directly addresses the documented Treg functional deficit and the downstream neuroinflammatory cascade it permits, offering a biologically coherent strategy for slowing motor neuron injury that is orthogonal to, and potentially complementary with, all currently approved agents. Preclinical support for this approach includes evidence from the SOD1 mouse model of ALS, which recapitulates the T cell abnormalities and immune dysregulation observed in human disease, confirming that the neuroinflammatory and Treg-deficient biology of the clinical condition is amenable to study and intervention in relevant animal systems.

Tiziana's Phase 2a BANYAN trial, conducted within the Healey ALS MyMatch program at the Sean M. Healey and AMG Center for ALS at Mass General Brigham, represents the clinical translation of this rationale. The trial is a 3:1 randomized, placebo-controlled, multicenter study enrolling 24 patients across sites within the Network of Excellence for ALS (NEALS) Consortium, led by principal investigators Suma Babu and James Berry at Mass General Brigham, and is supported by a grant from the ALS Association as part of the Hoffman ALS Clinical Trial Awards Program. The study is designed to assess safety, biomarkers of immune modulation and neuroinflammation including TSPO-PET imaging of microglial activation, and early signals of clinical effect in people living with ALS. The trial's inclusion in the Healey ALS MyMatch infrastructure is operationally meaningful: successful investigational products from this platform may advance to future regimens of the HEALEY ALS Platform Trial, a perpetual, multi-regimen, late-phase clinical efficacy study, or transition directly to standalone Phase 3 trials, providing a potentially accelerated pathway to late-phase development. The BANYAN trial is currently in operational startup, with enrollment timing and site locations to be confirmed in forthcoming communications.

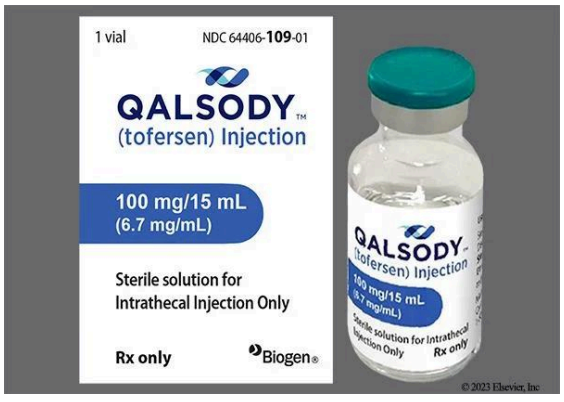
This program is best considered a POC effort. No human clinical data in ALS for foralumab have been reported, and the BANYAN trial is the first controlled evaluation of the agent in this indication. The program's scientific foundation, the Healey platform infrastructure, and the non-dilutive funding secured from the ALS Association collectively strengthen the credibility and resource efficiency of the development strategy, while the biological coherence of foralumab's Treg-based

mechanism with the established Treg-deficiency pathology of ALS provides a more direct and quantitatively grounded mechanistic hypothesis than is available for most investigational neuroinflammation approaches in this space.

Amyotrophic Lateral Sclerosis: Overview, Pathophysiology, and Standard of Care. Amyotrophic lateral sclerosis (ALS), commonly known as Lou Gehrig's disease, is a progressive, invariably fatal neurodegenerative disorder characterized by the selective degeneration of upper and lower motor neurons in the motor cortex, brainstem, and spinal cord. The clinical consequence of this dual motor neuron loss is a relentless progression from focal muscle weakness and fasciculations to global paralysis, dysarthria, dysphagia, and ultimately respiratory failure, which is the primary cause of death. Median survival from symptom onset is two to five years, though a minority of patients survive beyond a decade. ALS affects an estimated 35,000 Americans at any given time, with approximately 5,000 new diagnoses annually, and onset typically occurs in mid- to late life.

The disease is heterogeneous in its genetic architecture: approximately 90 to 95% of cases are sporadic, arising without a clear family history, while the remaining 5 to 10% are familial, with identified pathogenic mutations most commonly in SOD1, C9orf72, FUS, and TARDBP. The genetic subtype has meaningful implications for drug development, as mutation-specific targeting has proven feasible in SOD1-ALS and is being actively explored in C9orf72-ALS, while the vast majority of sporadic patients remain without a targetable genetic driver. The molecular pathology of ALS converges on several interconnected processes: misfolded protein aggregation, including TDP-43 proteinopathy present in approximately 97% of all ALS cases regardless of genetic etiology, mitochondrial dysfunction, excitotoxicity driven by impaired glutamate clearance, and, critically, neuroinflammation mediated by microglial and T cell dysregulation.

Exhibit 23. QALSODY was approved based on Nf-L biomarker data.



Source: Biogen company reports

The ALS treatment landscape includes three FDA-approved agents: riluzole, a glutamate release inhibitor that extends median survival modestly; edaravone, a free radical scavenger that modestly slows functional decline on the ALSFRS-R scale in a selected patient population; and tofersen (Qalsody, Biogen), an antisense

oligonucleotide approved in 2023 for SOD1-ALS, which lowers SOD1 protein levels and reduces neurofilament light chain (NfL), a validated biomarker of neuroaxonal injury, but is applicable only to the small minority of patients harboring SOD1 mutations. Relyvrio, a combination of sodium phenylbutyrate and tauroursodeoxycholic acid approved in 2022, was voluntarily withdrawn from the market in 2024 following a confirmatory Phase 3 trial failure. None of the available agents address the neuroinflammatory axis, none have demonstrated meaningful disease modification in the broad sporadic ALS population, and no approved therapy substantially alters the disease's fatal trajectory. The unmet need in ALS is profound and largely unaddressed.

Background: Tregs generated in the periphery control microglia in the CNS.

Several converging lines of evidence point to the CD4+ T cell axis — and specifically to the Treg/effector T cell balance — as a central regulator of neuroinflammation. These cells have long been implicated in the control of autoimmunity in the periphery and are increasingly implicated in the control of the activated microglia that drive much of neurodegeneration.

Foralumab. The basic idea driving the development of foralumab was that nasal delivery of particles reaches immune-sensitive tissues (NALT) that can modulate the responses to antigens produced in the CNS. This targeting of the immune system in the brain through therapeutics delivered in the nose is the key idea behind Tiziana. It's also a slightly counterintuitive idea as Tiziana's antibody therapeutic foralumab doesn't enter the CNS. Below, we review some of the broader themes that form the basis for the Tiziana approach to treating its target diseases.

Human genetics. Human genetic studies in MS identify variants in genes controlling T cell differentiation and Treg function as among the strongest genetic risk factors for disease. In both MS and Alzheimer's disease, Tregs from patients show impaired suppressive function relative to healthy controls, suggesting that restoring Treg activity is a physiologically grounded intervention.

Tregs: One Cell to Rule Them All. An adaptive immune response generating antigen-specific B-Cells and T-Cells is best thought of as an immune cell eruption with a handful of cells dividing into billions of progeny. As such, stopping these responses from recognizing self-antigens is a major goal of the immune system. The first line of defense is the negative selection that goes on early in immune system development where immune cells that recognize self-antigens are self-removed by apoptosis. This process is important but incomplete, leaving the need for additional lines of defense against autoimmunity – and the **final line of defense is a special T-Cell driven by the FoxP3 protein to form a regulatory T-Cell or Treg**. These cells are essential for life and animal models lacking FoxP3 don't form these cells and suffer catastrophic autoimmunity.

Microglia are CNS specific immune cells. Like most immune cells whose job is to react to differing situations, MGs are highly dynamic. A simple, but useful model is that MGs continuously sample their environment and shift between distinct states based on what they find. These "states", that include M1, M2 and DAM states are best thought of as an early approximation of the MG landscape and MGs are currently thought of as occupying a continuous landscape as indicated by specific gene expression that is shaped by their local microenvironment, developmental history, and infection triggers.

Species differences (mouse vs. human) are also significant — some states defined in mouse models don't map cleanly onto human microglia, and human-specific states (like the "intermediate" state enriched in AD patients) have been identified in postmortem tissue atlases.

- **Homeostatic microglia** - are the baseline state and best thought of as "looking for trouble". These MGs extend and retract feelers covered with various receptors designed to identify reasons to get excited and clear debris. More sophisticated functions include the pruning of synapses - the connections between neurons that must be continually refreshed. These two functions, along with keeping cytokine levels within tight bands all fall under the heading of neuronal health.
- **Inflammatory / M1-like microglia** are the "I found something state of MGs". They are classically activated by LPS or IFN-γ. They produce pro-inflammatory cytokines (IL-1β, TNF-α, IL-6), reactive oxygen species, and nitric oxide. This state is important for fighting infection but becomes damaging when sustained.
- **Disease-associated microglia (DAM)** - are the "I can't even remember why I found this interesting" state of MGs. They were defined largely through single-cell RNA sequencing studies of Alzheimer's mouse models. They downregulate homeostatic genes and upregulate *Trem2*, *ApoE*, *Lpl*, and *Cst7*. They cluster around amyloid plaques and are thought to represent a phagocytic, neuroprotective response — though *Trem2* loss-of-function studies show they also have pathological complexity.
- **Anti-inflammatory MGs.** This form was once called M2-like microglia in analogy to systemic M2 macrophages (also an early simplification) are induced by IL-4 or IL-13 and are associated with tissue repair.

Broad Treg stimulation vs. antigen specific autoimmunity.

This seeming mismatch remains one of the puzzles in the field — **how does a non-specific intervention produce what looks like antigen-specific suppression**? The short answer is who cares, it's pretty well established that it does in mice and there are hints it works in humans as well. However, the underlying mechanisms are complicated, but most of the pieces are well defined.

- **The disease attracts its cure.** Non-specific Treg stimulation works in autoimmunity because the disease itself solves the targeting problem. The site of autoimmune damage concentrates the self-antigen, recruits effector T cells, and provides the inflammatory signals that attract activated Tregs — then bystander suppression, linked suppression, and infectious tolerance do the rest. The drug doesn't need to know what the target antigen is; the immune architecture at the disease site funnels the suppression to where it's needed. This is also why intranasal anti-CD3 can work in mechanistically diverse diseases like MS, ALS, and MSA — the common thread is neuroinflammation driven by dysregulated T cells and microglia, not the specific antigens involved.

- **Bystander suppression.** The most important concept. Once a Treg is activated by *any* antigen, its suppressive effector functions — IL-10 secretion, TGF-β secretion, CTLA-4-mediated stripping of CD80/CD86 from APCs — operate on surrounding cells regardless of their antigen specificity. So a Treg that recognizes an innocuous food peptide can, once activated, suppress an autoreactive T cell attacking myelin in the same microenvironment. The suppression is local and contact- or short-range cytokine-dependent, so what matters is geography: Tregs need to be at or near the inflammatory site. The inflammation itself provides the beacon, since chemokines (CXCL9, CXCL10 via CXCR3; CCL17/22 via CCR4) and the cytokine milieu at sites of autoimmune activity recruit activated Tregs there preferentially.
- **Enrichment for disease-relevant Tregs.** Within the polyclonal pool of Tregs you expand or induce, some fraction will happen to have TCRs that recognize the self-antigen driving the disease. These Tregs get selectively retained and re-stimulated at the disease site by that antigen. So even if you start with non-specific expansion, the disease site acts as a filter — Tregs that recognize the relevant antigen accumulate there while others traffic through and leave. The result is local enrichment of disease-relevant specificity without any engineering.
- **Tolerance spread in inflamed tissue.** Tregs can convert neighboring conventional T cells into regulatory cells through TGF-β — a process called infectious tolerance or Treg propagation. A relatively small number of Tregs arriving at the disease site can trigger a cascade of local tolerance induction, amplifying the suppressive effect well beyond what the initial expansion would predict. This is partly how low-dose IL-2 or intranasal anti-CD3 can have disproportionately large downstream effects.
- **Preferential deletion of autoreactive T cells (anti-CD3 specific).** This is a mechanism specific to anti-CD3 antibodies and explains part of why foralumab works in an antigen-specific-looking way. T cells that are actively engaged in an autoimmune response are in a state of high activation and ongoing TCR signaling. When anti-CD3 delivers a partial signal (incomplete, without costimulation, as happens in the tolerogenic NALT environment), cells in this highly activated state are disproportionately susceptible to restimulation-induced cell death via Fas/FasL and mitochondrial apoptosis pathways. Naive or resting T cells are relatively resistant. So the cells most aggressively attacking self are paradoxically the ones most vulnerable to being deleted — creating a de facto antigen-specific effect from a non-antigen-specific drug.
- **Linked suppression.** When a Treg suppresses an APC — for example by CTLA-4 competing away CD80/CD86 — that APC can no longer effectively activate any T cell it presents antigen to, regardless of which antigen. This creates suppression that links across specificities: a Treg specific for antigen A, by tolerizing the APC that presents both A and B, prevents activation of T cells specific for B as well. In a tissue where a limited set of APCs are

presenting multiple self-peptides, linked suppression can dampen responses to many epitopes simultaneously.

- **Prevention of epitope spreading.** Autoimmune diseases typically start with responses to a small number of epitopes and then spread — as inflammation releases more self-antigen, the immune response diversifies to target new peptides on the same protein and eventually other proteins. Tregs that suppress the initial inflammatory response short-circuit this amplification loop, preventing the diversification that would otherwise make the disease progressively harder to treat. In some sense, early Treg-mediated suppression can contain what would otherwise become a broader polyclonal autoimmune attack.

Tiziana Intellectual Property

Tiziana Life Sciences maintains a multi-program intellectual property portfolio spanning three core therapeutic areas: foralumab (TZLS-401), an anti-IL-6/IL-6R antibody (TZLS-501), and Actinomycin D (ActD).

The foralumab portfolio is the most expansive, comprising thirteen patent families covering formulations, routes of administration, and a growing array of disease indications. The foundational family — covering formulations and dosing regimens — carries granted status across major commercial markets including the United States, Japan, China, and key European jurisdictions, with expiry in 2037. Several families are co-owned or licensed with Brigham and Women's Hospital, Inc., reflecting a collaborative development relationship, and span applications in CNS disorders, microglial activation, neurological conditions, Alzheimer's Disease, Multiple System Atrophy, spinal cord injury, and intracerebral hemorrhage. The more recently filed families, many of which remain at the PCT or provisional stage, carry potential expiries ranging from 2042 to 2046, reflecting the pipeline's ongoing expansion into new indications.

TZLS-501 is supported by two licensed patent families from NovImmune S.A (Private). The first — a composition of matter family with broad multi-jurisdictional granted coverage including six U.S. patents — expires in May 2029, representing the portfolio's most proximate patent cliff. A second family covering coronavirus-related methods extends protection to March 2041, though it remains pending across all jurisdictions.

Actinomycin D is covered by two proprietary families through 2036 and 2037, with granted status in the U.S., Japan, and Australia, and pending applications in Europe and Canada for the nanoparticle formulation family.

The company relies on a combination of patents, trade secrets, and confidentiality and invention assignment agreements to protect its proprietary position. Notably, patent prosecution for licensed technology is controlled solely by the respective licensors — NovImmune and Brigham and Women's Hospital — which introduces third-party dependency risk with respect to maintaining exclusivity.

Exhibit 24. Intellectual Property Portfolio of Tiziana

Program	IP Family	Type	Key Coverage	Status	Expiry	Key Jurisdictions	Licensor / Co-Owner
Foralumab (TZLS-401)	Formulations & Dosing Regimen	Composition / Method	Formulations and dosing regimens for treating various disorders	Issued / Pending	2037	Issued: US, AU, CN, JP, IL, BE, CH, DE, DK, ES, FR, GB, IE, IT, LU, NL, NO, SE, HK. Pending: AU, CA, CN, EU, US	Proprietary
	Methods of Use – CNS Disorders	Method	Use of foralumab in treating CNS disorders	Issued / Pending	2038	Issued: JP. Pending: CA, EU, JP, US	Licensed – Brigham and Women's Hospital
	Subcutaneous Administration	Method	Subcutaneous injection administration of foralumab	Pending	2042	Pending: US	Proprietary
	Nasal Formulations	Composition / Method	Nasal formulations of foralumab for various diseases	Pending	2043	Pending: AU, CA, EU, JP, US	Proprietary
	Microglial Activation Suppression	Method	Methods of suppressing microglial activation	Pending	2042	Pending: US, AU, CA, CN, EU, JP, IL	Co-owned – Brigham and Women's Hospital
	Combination w/ GLP-1 / SGLT2 Inhibitors (Obesity)	Method	Foralumab + GLP-1 agonists or SGLT2 inhibitors for obesity	Pending	2044	Pending: PCT	Co-owned – Brigham and Women's Hospital
	Combination w/ GLP-1 (Neurological)	Method	Foralumab + GLP-1 agonists for neurological conditions	Pending	2045	Pending: PCT	Co-owned – Brigham and Women's Hospital
	Combination w/ Anti-CD20 Therapies	Method	Foralumab + anti-CD20 combination therapies	Pending	2046	Pending: Provisional	Co-owned – Brigham and Women's Hospital
	Combination w/ Anti-Amyloid (Alzheimer's)	Method	Foralumab + anti-amyloid therapies for Alzheimer's Disease	Pending	2046	Pending: PCT	Co-owned – Brigham and Women's Hospital
	Multiple Systems Atrophy	Method	Treatment of Multiple Systems Atrophy	Pending	2046	Pending: Provisional	Proprietary
	Intracerebral Hemorrhage	Method	Nasal foralumab for intracerebral hemorrhage	Pending	2045	Pending: PCT	Licensed – Brigham and Women's Hospital
	Spinal Cord Injury	Method	Nasal foralumab for spinal cord injury	Pending	2045	Pending: PCT	Licensed – Brigham and Women's Hospital
	Biomarkers – Nasal Foralumab Treatment Response (Progressive MS)	Diagnostic / Method	Biomarkers for nasal foralumab treatment response in progressive MS	Pending	2046	Pending: PCT	Proprietary
TZLS-501 (Anti-IL-6/IL-6R)	Composition of Matter & Methods of Use	Composition / Method	Compositions and methods for treating various disorders	Issued	2029	Issued: US (x6), EU, AU, CA, CN, IN, IL, JP, MX, AT, BE, DK, FR, DE, IE, IT, LU, NL, ES, SE, CH, UK	Licensed – NovImmune S.A.
	Coronavirus Treatment (incl. Actinomycin D Combo)	Method	TZLS-501 for coronavirus, alone and in combo with Actinomycin D	Pending	2041	Pending: US, AU, CA, CN, EU, IL, JP	Licensed – NovImmune S.A.
Actinomycin D (ActD)	AML Treatment	Method	Use of ActD in treatment of acute myeloid leukemia	Issued	2036	Issued: US, EU, JP, AU, CA	Proprietary
	Nanoparticle Formulations – AML & MDS	Composition / Method	Nanoparticle formulations of ActD for AML and myelodysplastic syndrome	Issued / Pending	2037	Issued: US (x2), JP (x2), AU. Pending: EU, CA	Proprietary

Source: BTIG Research, Tiziana Company Reports

Tiziana Management Team

Gabriele Marco Antonio Cerrone — Executive Chairman

Gabriele Cerrone is the Founder and Executive Chairman of Tiziana Life Sciences, a role he has held since April 2014. Over his career, he has founded ten biotechnology companies across oncology, infectious diseases, ophthalmology, and molecular diagnostics, listing seven on Nasdaq and two on the London Stock Exchange, resulting in three FDA drug approvals. Notable prior roles include Co-Founder and Chairman of FermaVir Pharmaceuticals (merged with Inhibitex, sold to Bristol Myers Squibb for $2.5 billion in 2012), Co-Chairman of Cardiff Oncology, and Chairman of Synergy Pharmaceuticals. He currently holds concurrent roles as Executive Chairman of OKYO Pharma Ltd. and Gensignia Life Sciences, Non-Executive Chairman of Accustem Sciences Limited, and Co-Founder of Rasna Therapeutics and ContraVir Pharmaceuticals. Mr. Cerrone holds a Finance degree, Magna Cum Laude, from NYU's Stern School of Business.

Willy Simon — Non-Executive Director

Willy Jules Simon has served as Non-Executive Director of Tiziana since November 2015. A career banker, he held senior roles at Kredietbank N.V., Citibank London, Generale Bank NL (board member, 1997–1999), and Fortis Investment Management (CEO, 1999–2002), followed by the chairmanship of Bank Oyens & van Eeghen (2002–2004). He has been Chairman of Amsterdam-listed Bever Holdings since 2006 and Chairman of Ducat Maritime since 2015, and also serves as Non-Executive Director of OKYO Pharma Ltd.

John Brancaccio — Non-Executive Director

John Brancaccio, a retired CPA, has served as a director of Tiziana since July 2020. He served as CFO of Accelerated Technologies, Inc. from 2004 to 2017 and has held director roles across multiple biotech and pharma companies, including Callisto Pharmaceuticals, Tamir Biotechnology, Hepion Pharmaceuticals, Cardiff Oncology, Synergy Pharmaceuticals, Actavia Life Sciences, and OKYO Pharma Ltd.

Ivor Elrifi — Executive Director and Chief Executive Officer

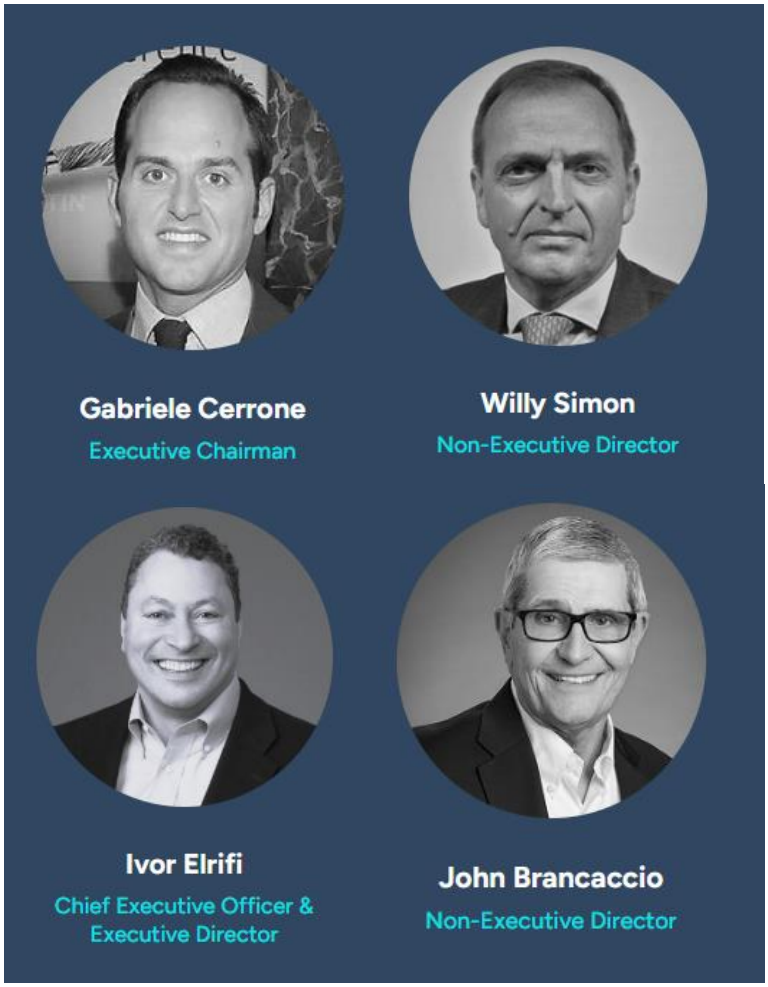
Ivor Elrifi serves as CEO and Executive Director of Tiziana. He brings deep IP expertise from a distinguished legal career, having served as Global Head of the Patent Group at Cooley LLP (2014 onward) and Global Head of Patents at Mintz Levin (1999–2014). He has advised leading life sciences companies on IP strategy, licensing, patent prosecution, and M&A, with transactions involving Novartis, Eli Lilly, Biogen, and Astellas. Mr. Elrifi has been ranked in Chambers USA since 2007 and named an "LMG Life Sciences: Life Science Star." He holds a B.S. and Ph.D. in Biology from Queen's University and a J.D. from Osgoode Hall Law School.

Keeren Shah — Chief Operating and Financial Officer

Keeren Shah serves as CFO and COO of Tiziana, and concurrently holds CFO roles at OKYO Pharma Ltd., Accustem Sciences Limited, and Actavia Life Sciences Inc. She

joined Tiziana in 2016 as Group Financial Controller before being promoted in 2020. Prior to Tiziana, she spent a decade at Visa, Inc. in senior finance roles, contributing to the company's landmark IPO and overseeing global FP&A and controllership functions. She began her career at Arthur Andersen and BBC Worldwide. Ms. Shah holds a BA (Hons) in Economics from the University of Manchester and is a member of the Chartered Institute of Management Accountants.

Exhibit 25. Tiziana Management Team



Source: Tiziana company reports

Tiziana Income Statement

Exhibit 26. TLSA Income Statement

Thomas Shrader 212.527.3551
Jinnie Kim 212.882.2344

Tiziana Life Sciences (TLSA) Income Statement

Period Ending $USD ('000s)	Dec-21 2021A	Dec-22 2022A	Dec-23 2023A	Dec-24 2024A	Jun-25 1H25A	Dec-25 2H25A	Dec-25 2025A	Jun-26 1H26E	Dec-26 2H26E	Dec-26 2026E	Dec-27 2027E	Dec-28 2028E	Dec-29 2029E	Dec-30 2030E	Dec-31 2031E	Dec-32 2032E	Dec-33 2033E	Dec-34 2034E	Dec-35 2035E	Dec-36 2036E	Dec-37 2037E	Dec-38 2038E	Dec-39 2039E	Dec-40 2040E
Product Revenue:																								
TZLS-401 for SPMS (50% risk adjusted)							-	-	-	-	-	-	-	97,465	426,942	727,300	1,115,066	1,291,676	1,475,549	1,587,544	1,460,373	1,326,804	1,186,616	1,039,583
TZLS-401 for MSA (30% risk adjusted)							-	-	-	-	-	-	-	-	37,484	63,855	97,900	113,406	129,549	139,382	128,217	116,490	104,182	91,273
TZLS-401 for AD (15% risk adjusted)							-	-	-	-	-	-	-	-	-	145,838	447,185	761,783	1,167,934	1,352,918	1,545,509	1,662,813	1,529,613	1,389,711
TZLS-401 for ALS (15% risk adjusted)								-	-	-	-	-	-	-	-	4,139	12,690	21,618	33,144	38,394	43,859	47,188	43,408	39,438
Total Revenue	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**97,465**	**464,427**	**941,132**	**1,672,841**	**2,188,483**	**2,806,177**	**3,118,237**	**3,177,958**	**3,153,295**	**2,863,818**	**2,560,004**
Operating Expenses:																								
Cost of goods sold	-	-	-	-		-	-	-	-	-	-	-	-	14,620	69,664	141,170	250,926	328,272	420,926	467,736	476,694	472,994	429,573	384,001
Research and development	13,208	12,955	8,113	5,229	3,243	7,036	10,279	3,324	7,212	10,536	10,799	11,069	11,346	48,733	232,213	470,566	669,136	656,545	701,544	623,647	572,032	504,527	429,573	384,001
General and administrative	13,311	1,631	9,871	10,565	5,943	4,910	10,853	6,092	5,033	11,124	11,402	11,687	11,980	194,931	464,427	470,566	752,778	875,393	841,853	779,559	730,930	662,192	572,764	512,001
Other	855																							
Total Operating Expenses	**27,374**	**14,586**	**17,984**	**15,794**	**9,186**	**11,946**	**21,132**	**9,416**	**12,245**	**21,660**	**22,202**	**22,757**	**23,326**	**258,283**	**766,304**	**1,082,301**	**1,672,841**	**1,860,211**	**1,964,324**	**1,870,942**	**1,779,656**	**1,639,713**	**1,431,909**	**1,280,002**
Operating Income (Loss) EBIT	**(27,374)**	**(14,586)**	**(17,984)**	**(15,794)**	**(9,186)**	**(11,946)**	**(21,132)**	**(9,416)**	**(12,245)**	**(21,660)**	**(22,202)**	**(22,757)**	**(23,326)**	**(160,818)**	**(301,877)**	**(141,170)**	**-**	**328,272**	**841,853**	**1,247,295**	**1,398,301**	**1,513,581**	**1,431,909**	**1,280,002**
Other Income (expense) Net	717	(811)	742	(952)	3,554	(853)	2,701																	
Income (Loss) before Taxes	**(26,657)**	**(15,397)**	**(17,242)**	**(16,746)**	**(5,632)**	**(12,799)**	**(18,431)**	**(9,416)**	**(12,245)**	**(21,660)**	**(22,202)**	**(22,757)**	**(23,326)**	**(160,818)**	**(301,877)**	**(141,170)**	**-**	**328,272**	**841,853**	**1,247,295**	**1,398,301**	**1,513,581**	**1,431,909**	**1,280,002**
Tax provision	3,240	-	**(449)**	4,883	0	0	0																	
Net Income (Loss)	**(23,417)**	**(15,397)**	**(17,691)**	**(11,863)**	**(5,632)**	**(12,799)**	**(18,431)**	**(9,416)**	**(12,245)**	**(21,660)**	**(22,202)**	**(22,757)**	**(23,326)**	**(160,818)**	**(301,877)**	**(141,170)**	**-**	**328,272**	**841,853**	**1,247,295**	**1,398,301**	**1,513,581**	**1,431,909**	**1,280,002**
Foreign currency translation adjustment	(4,478)	(3,582)	1,492	(72)	339	(427)	(88)																	
Total comprehensive income (Loss)	**(27,895)**	**(18,979)**	**(16,199)**	**(11,935)**	**(5,293)**	**(13,226)**	**(18,519)**	**(9,416)**	**(12,245)**	**(21,660)**	**(22,202)**	**(22,757)**	**(23,326)**	**(160,818)**	**(301,877)**	**(141,170)**	**-**	**328,272**	**841,853**	**1,247,295**	**1,398,301**	**1,513,581**	**1,431,909**	**1,280,002**
Basic EPS, GAAP	**(0.24)**	**(0.15)**	**(0.17)**	**(0.11)**	**(0.05)**	**(0.11)**	**(0.16)**	**(0.08)**	**(0.10)**	**(0.18)**	**(0.18)**	**(0.19)**	**(0.19)**	**(1.29)**	**(2.41)**	**(1.12)**	**-**	**2.56**	**6.51**	**9.58**	**10.65**	**11.45**	**10.75**	**9.54**
Diluted EPS, GAAP	**(0.24)**	**(0.15)**	**(0.17)**	**(0.11)**	**(0.05)**	**(0.11)**	**(0.16)**	**(0.08)**	**(0.10)**	**(0.18)**	**(0.18)**	**(0.19)**	**(0.19)**	**(1.29)**	**(2.41)**	**(1.12)**	**-**	**2.56**	**6.51**	**9.58**	**10.65**	**11.45**	**10.75**	**9.54**
Weighted average shares outstanding, basic	97,932	101,526	102,471	106,672	116,827	119,739	118,283	119,989	120,489	120,239	121,239	122,239	123,239	124,239	125,239	126,239	127,239	128,239	129,239	130,239	131,239	132,239	133,239	134,239
Weighted average shares outstanding, diluted	97,932	101,526	102,471	106,672	116,827	119,739	118,283	119,989	120,489	120,239	121,239	122,239	123,239	124,239	125,239	126,239	127,239	128,239	129,239	130,239	131,239	132,239	133,239	134,239

Source: BTIG Research, Tiziana Company reports

Tiziana Balance Sheet

Exhibit 27. TLSA Balance Sheet

Thomas Shrader 212.527.3551
Jinnie Kim 212.882.2344

Tiziana Life Sciences (TLSA) Balance Sheet

Period Ending $USD ('000s)	Dec-21 2021A	Dec-22 2022A	Dec-23 2023A	Dec-24 2024A	Dec-25 2025A	Jun-26 1H26E	Dec-26 2H26E	Dec-26 2026E	Dec-27 2027E	Dec-28 2028E	Dec-29 2029E	Dec-30 2030E	Dec-31 2031E	Dec-32 2032E	Dec-33 2033E	Dec-34 2034E	Dec-35 2035E	Dec-36 2036E	Dec-37 2037E	Dec-38 2038E	Dec-39 2039E	Dec-40 2040E
				-																		
Cash and cash equivalents	42,186	18,122	1,183	3,724	4,010	(8,878)	(21,533)	(21,533)	7,934	(13,118)	(34,700)	(247,419)	(670,010)	(875,073)	(998,611)	(645,687)	234,651	1,608,177	3,162,732	4,846,192	6,488,041	7,954,006
Accounts receivable	-	-	-	-	-	-	-	-	-	-	-	14,620	69,664	141,170	250,926	328,272	420,926	467,736	476,694	472,994	429,573	384,001
Prepayments and other receivables	1,301	300	223	145	459	4,708	6,122	6,122	6,275	6,432	6,593	64,571	191,576	270,575	418,210	465,053	491,081	467,736	444,914	409,928	357,977	320,000
Taxation receivable	4,736	4,246	3,793	32	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99
Related party receivables	456	1,614	2,138	3607	3984	3984	3984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984	3,984
Other current assets	-	-		-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Current Assets	**48,679**	**24,282**	**7,337**	**7,508**	**8,552**	**(87)**	**(11,328)**	**(11,328)**	**18,292**	**(2,602)**	**(24,024)**	**(164,145)**	**(404,687)**	**(459,245)**	**(325,392)**	**151,721**	**1,150,741**	**2,547,731**	**4,088,422**	**5,733,197**	**7,279,674**	**8,662,090**
Property and equipment, net	17	17	10	16	26	25	23	23	21	19	17	16	14	13	11	10	9	8	8	7	6	6
Right-of-use asset	-	372	283	171	473	449	427	427	385	348	314	283	256	231	208	188	170	153	138	125	112	102
Investment in related party		1,806	4,554	3,589	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444	2,444
Other non-current assets	130	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Assets	**48,826**	**26,477**	**12,184**	**11,284**	**11,495**	**2,831**	**(8,433)**	**(8,433)**	**21,143**	**209**	**(21,249)**	**(161,402)**	**(401,974)**	**(456,557)**	**(322,728)**	**154,363**	**1,153,364**	**2,550,336**	**4,091,012**	**5,735,773**	**7,282,237**	**8,664,641**
Accounts payable and accrued expenses	6,181	6,532	6,387	7,230	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460	10,460
Deferred income	-	-	-	-	530	530	530	530	530	530	530	530	530	530	530	530	530	530	530	530	530	530
Operating lease liabilities, current	-	122	138	106	222	222	222	222	222	222	222	222	222	222	222	222	222	222	222	222	222	222
Other current liability	1,365	9	14	12	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)	(3)
Total Current Liabilities	**7,546**	**6,663**	**6,539**	**7,348**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**	**11,209**
Lease liability, non-current	-	243	109	-	234	234	234	234	234	234	234	234	234	234	234	234	234	234	234	234	234	234
Other non-current liability	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Liabilities	**7,546**	**6,906**	**6,648**	**7,348**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**	**11,443**
Called up share captial	102	102	103	111	121	120	120	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134
Share Premium	15,596	15,596	16,492	23,105	34,663	35,415	36,395	36,395	88,172	89,994	91,861	112,525	173,830	260,415	394,243	543,061	700,208	849,885	992,258	1,123,436	1,237,990	1,340,391
Share based payment reserve - Options	13,797	5,190	6,905	7,626	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549	8,549
Share based payment reserve - Warrants	697	697	-	25	69	69	69	69	69	69	69	69	69	69	69	69	69	69	69	69	69	69
Merger relief reserve	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697	118,697
Treasury Shares	-	(1,320)	(1,574)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
RSU reserve, Shares to be issued reserve	-	-	225	910	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858	2,858
Translation service	454	(3,128)	(1,636)	(1,707)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)	(1,796)
Retained earnings	(108,063)	(116,263)	(133,676)	(144,831)	(163,109)	(172,525)	(184,769)	(184,769)	(206,971)	(229,728)	(253,054)	(413,872)	(715,749)	(856,919)	(856,919)	(528,646)	313,206	1,560,501	2,958,803	4,472,384	5,904,293	7,184,295
Total Stockholders' Equity (Deficit)	**41,280**	**19,571**	**5,536**	**3,936**	**52**	**(8,612)**	**(19,876)**	**(19,876)**	**9,700**	**(11,234)**	**(32,692)**	**(172,845)**	**(413,417)**	**(468,000)**	**(334,171)**	**142,920**	**1,141,921**	**2,538,893**	**4,079,569**	**5,724,330**	**7,270,794**	**8,653,198**
				-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Liabilities and Stockholders' Equity	**48,826**	**26,477**	**12,184**	**11,284**	**11,495**	**2,831**	**(8,433)**	**(8,433)**	**21,143**	**209**	**(21,249)**	**(161,402)**	**(401,974)**	**(456,557)**	**(322,728)**	**154,363**	**1,153,364**	**2,550,336**	**4,091,012**	**5,735,773**	**7,282,237**	**8,664,641**

Source: BTIG Research, Tiziana Company reports

Tiziana Cash Flow Statement

Exhibit 28. TLSA Cash Flow Statement

Thomas Shrader 212.527.3551
Jinnie Kim 212.882.2344

Tiziana Life Sciences (TLSA) Cash Flow Statement

Period Ending $USD ('000s)	Dec-21 2021A	Dec-22 2022A	Dec-23 2023A	Dec-24 2024A	Dec-25 2025A	Jun-26 1H26E	Dec-26 2H26E	Dec-26 2026E	Dec-27 2027E	Dec-28 2028E	Dec-29 2029E	Dec-30 2030E	Dec-31 2031E	Dec-32 2032E	Dec-33 2033E	Dec-34 2034E	Dec-35 2035E	Dec-36 2036E	Dec-37 2037E	Dec-38 2038E	Dec-39 2039E	Dec-40 2040E
Loss from operations before income taxes	(26,657)	(15,397)	(17,242)	(16,746)	(18,431)	(9,416)	(12,245)	(21,660)	(22,202)	(22,757)	(23,326)	(160,818)	(286,537)	(137,977)	-	327,939	841,626	1,250,392	1,411,535	1,527,560	1,445,966	1,293,524
Adjustments to reconcile net loss to net cash used in operating activities:																						
Shares issued in lieu of fees	-	-	425	3,401	1,344			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Share based payment - restricted stock, options, warrants	5,173	1,811	1,773	2,566	3,198	753	980	1,733	1,776	1,821	1,866	20,663	58,189	84,626	131,797	148,666	157,103	150,047	143,720	132,389	115,677	103,482
Options forfeited during the year	-	(3,221)	(39)	(227)	(109)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Fair value (gain)/loss on investment, net	-	869	402	1,760	(1,752)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Depreciation	8	1	7	12	12	1	1	3	2	2	2	2	2	1	1	1	1	1	1	1	1	1
Depreciation of right-of-use asset	133	50	89	112	222	24	22	46	42	38	34	31	28	25	22	20	18	17	15	13	12	11
(Gain)/loss on foreign exchange	(1,899)	(3,183)	1,519	158	(152)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cash (outflow)/ inflow from taxation	1,415	490	-	8,784	(75)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other	838	129	(1,050)	(794)	(166)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
				-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Changes in operating assets and liabilities:				-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net (increase) in related party receivables	(773)	(2,513)	(1,524)	(1,469)	(378)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net (increase)/decrease in operating assets/other receivables	516	1,002	80	78	(314)	(4,249)	(1,415)	(5,663)	(153)	(157)	(161)	(72,598)	(168,774)	(154,469)	(256,551)	(133,535)	(119,242)	(26,033)	7,465	39,253	95,796	83,843
Net increase/(decrease) in operating liabilities /other liabilities	(516)	347	(138)	839	3,224	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net increase/(decrease) in deferred revenue				-	530	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Cash Used in Operating Activities	**(21,762)**	**(19,615)**	**(15,698)**	**(1,526)**	**(12,847)**	**(12,886)**	**(12,656)**	**(25,542)**	**(20,535)**	**(21,054)**	**(21,585)**	**(212,721)**	**(397,092)**	**(207,793)**	**(124,730)**	**343,091**	**879,506**	**1,374,424**	**1,562,735**	**1,699,216**	**1,657,451**	**1,480,860**
Purchases of property and equipment	(22)	-	-	(19)	(21)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds from finance lease	152			-	-																	
Investment in Related Party	-	(2,676)	(1,000)	-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds on sale of investment shares, net	-			-	3,064			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Purchase of treasury shares	-	(1,320)	(253)	(52)	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other	-			-	-	-2	1	(1)	2	2	2	2	2	2	2	2	2	2	2	2	2	2
Net Cash Provided (Used in) by Investing Activities	**130**	**(3,996)**	**(1,253)**	**(71)**	**3,043**	**(2)**	**1**	**(1)**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**
Proceeds from issuance of ordinary shares - all	-	-	24	4,571	10,059			-	50,000	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds from issuance of warrants	129	-	135	-	48			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Repayment of leasing liabilities	(152)	(55)	(119)	(141)	(196)			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds of exercise of options	-	-	-	75	95			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
				-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Cash Provided by Financing Activities	**(23)**	**(55)**	**40**	**4,505**	**10,006**	**-**	**-**	**-**	**50,000**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**
Net Increase (Decrease) in Cash, Cash Equivalents	**(21,655)**	**(23,666)**	**(16,911)**	**2,908**	**202**	**(12,888)**	**(12,655)**	**(25,543)**	**29,467**	**(21,052)**	**(21,583)**	**(212,719)**	**(397,090)**	**(207,791)**	**(124,728)**	**343,093**	**879,508**	**1,374,426**	**1,562,737**	**1,699,218**	**1,657,453**	**1,480,862**
Exchange difference on cash and cash equivalents	(1,983)	(398)	(28)	(367)	84																	
Cash Beginning of Period	**65,824**	**42,186**	**18,122**	**1,183**	**3,724**	**4,010**	**(8,878)**	**4,010**	**(21,533)**	**7,934**	**(13,118)**	**(34,700)**	**(247,419)**	**(644,509)**	**(852,300)**	**(977,029)**	**(633,936)**	**245,572**	**1,619,998**	**3,182,735**	**4,881,952**	**6,539,406**
Cash End of Period	**42,186**	**18,122**	**1,183**	**3,724**	**4,010**	**(8,878)**	**(21,533)**	**(21,533)**	**7,934**	**(13,118)**	**(34,700)**	**(247,419)**	**(644,509)**	**(852,300)**	**(977,029)**	**(633,936)**	**245,572**	**1,619,998**	**3,182,735**	**4,881,952**	**6,539,406**	**8,020,268**

Source: BTIG Research, Tiziana Company reports

BTIG Covered Companies Mentioned in this Report

Tiziana Life Sciences (TLSA, Buy, $7 PT; Closing Price: $0.96)
AC Immune SA (ACIU, Buy, $8 PT; Closing Price: $2.68)
Alector Inc. (ALEC, Buy, $6 PT; Closing Price: $2.40)
BIOAGE Labs, Inc. (BIOA, Buy, $40 PT; Closing Price: $9.52)
Coya Therapeutics, Inc. (COYA, Buy, $16 PT; Closing Price: $4.51)
NeOnc Technologies (NTHI, Buy, $15 PT; Closing Price: $4.73)

Appendix: Analyst Certification and Other Important Disclosures

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I, Jinnie Kim, hereby certify that the views about the companies and securities discussed in this report are accurately expressed and that I have not received and will not receive direct or indirect compensation in exchange for expressing specific recommendations or views in this report.

I, Thomas Shrader, hereby certify that the views about the companies and securities discussed in this report are accurately expressed and that I have not received and will not receive direct or indirect compensation in exchange for expressing specific recommendations or views in this report.

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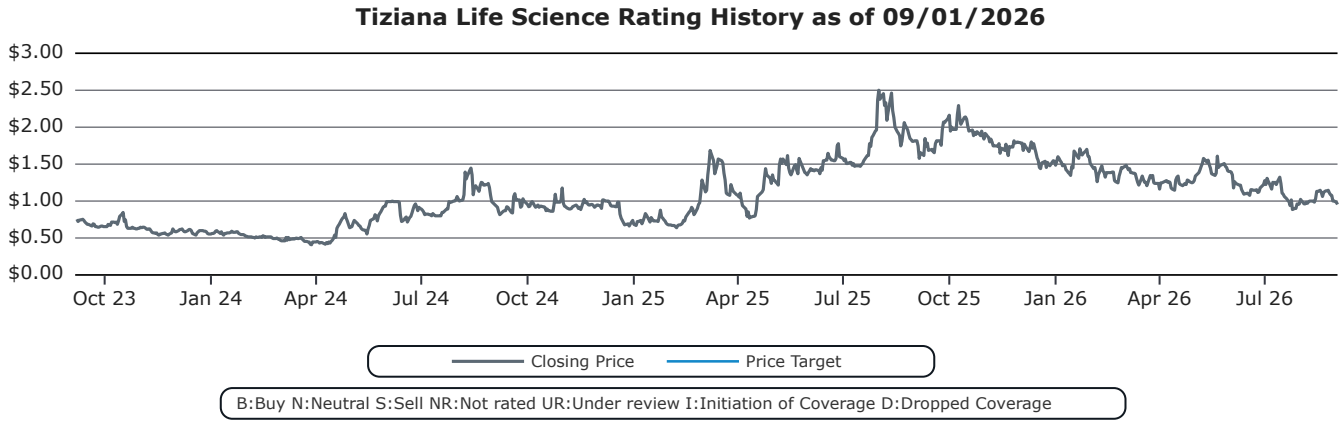
Coverage Universe	Count	Percent	Inv. Banking Relationships	Count	Percent
Buy	378	66.9%	Buy	129	34.1%
Neutral	183	32.4%	Neutral	46	25.1%
Sell	4	0.7%	Sell	0	0.0%

For purposes of FINRA ratings distribution rules, BTIG's stock ratings of Buy, Neutral and Sell fall into Buy, Hold and Sell categories, respectively.

Company Valuation and Risk Disclosures

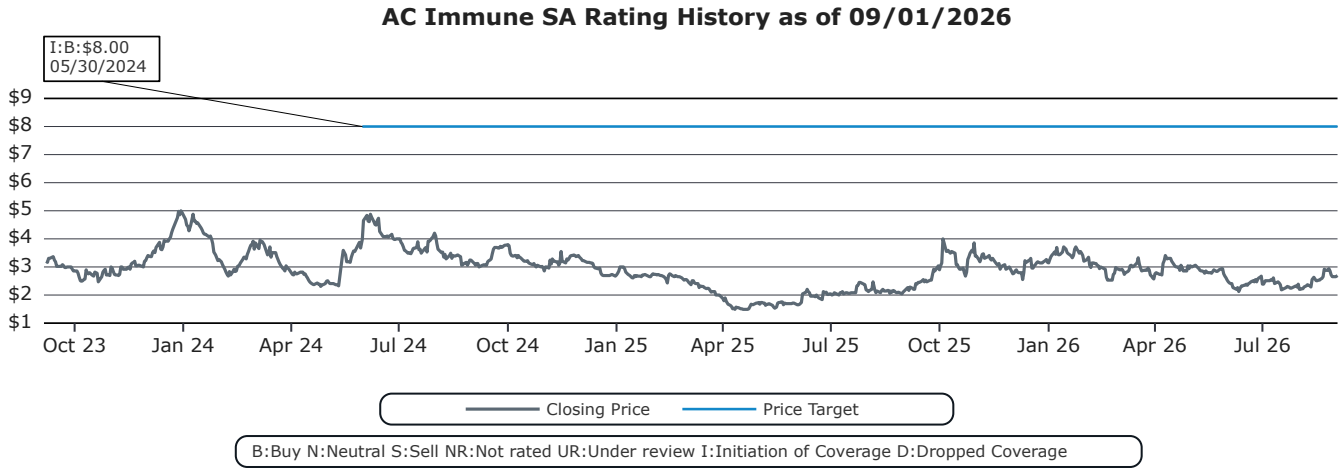
Tiziana Life Sciences (TLSA, Buy, $7 PT)

Valuation: We value TLSA using a DCF (17.0% discount rate and a 1.0% TG rate)
Risks: Tiziana is an established biotech company. It faces all the standard upside and downside risks for that industry, including unexpected outcomes or safety signals from clinical readouts, regulatory uncertainty, and increasingly complex and price-sensitive commercial markets for its product candidates.



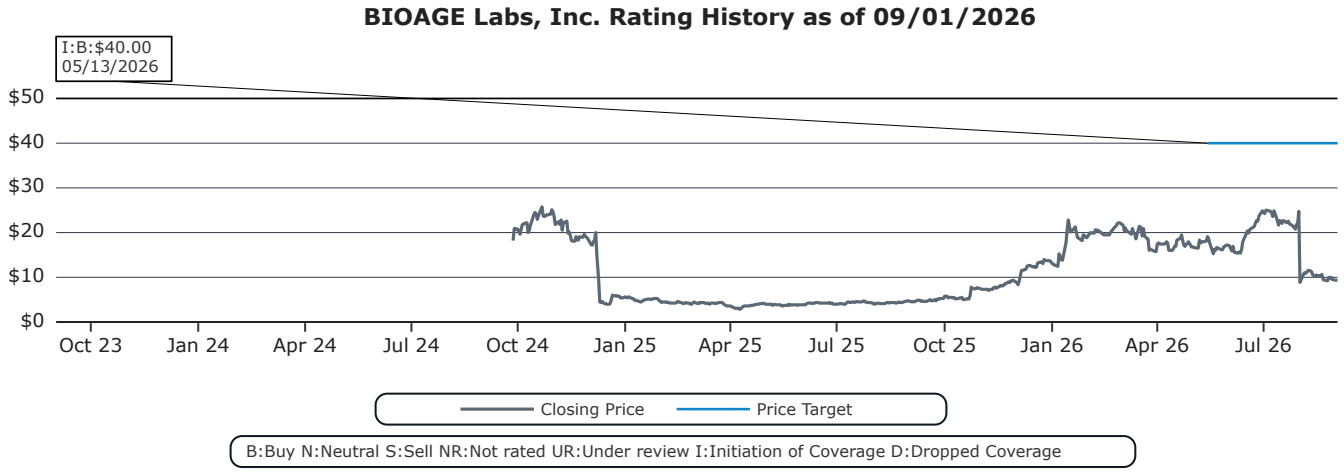
AC Immune SA (ACIU, Buy, $8 PT)

Valuation: We value ACIU using a DCF analysis (17.5% discount rate, 1.5% TG rate).
Risks: AC Immune SA is an established drug development company. It faces all the standard upside and downside risks for that industry, including unexpected outcomes or safety signals from clinical readouts, regulatory uncertainty, and increasingly complex and price-sensitive commercial markets for its product candidates.



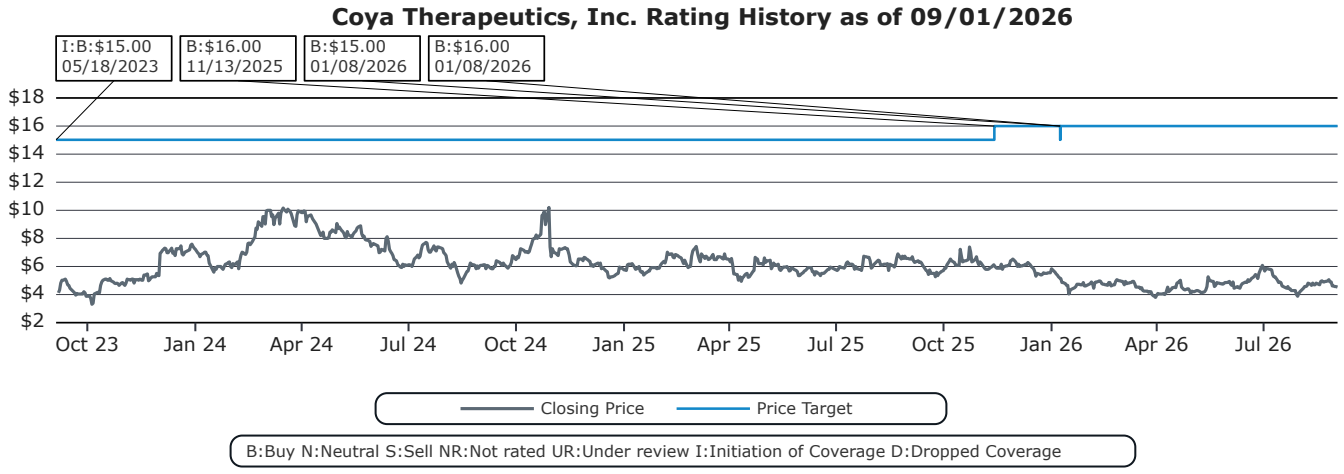
BIOAGE Labs, Inc. (BIOA, Buy, $40 PT)

Valuation: We value BIOA with a DCF analysis (15.5% discount rate, 1% TG).
Risks: BioAge Labs is an established biotechnology company. It faces all the standard risks for that industry, including unexpected outcomes from clinical readouts, regulatory uncertainty, and increasing complex and price-sensitive commercial markets for its product candidates.



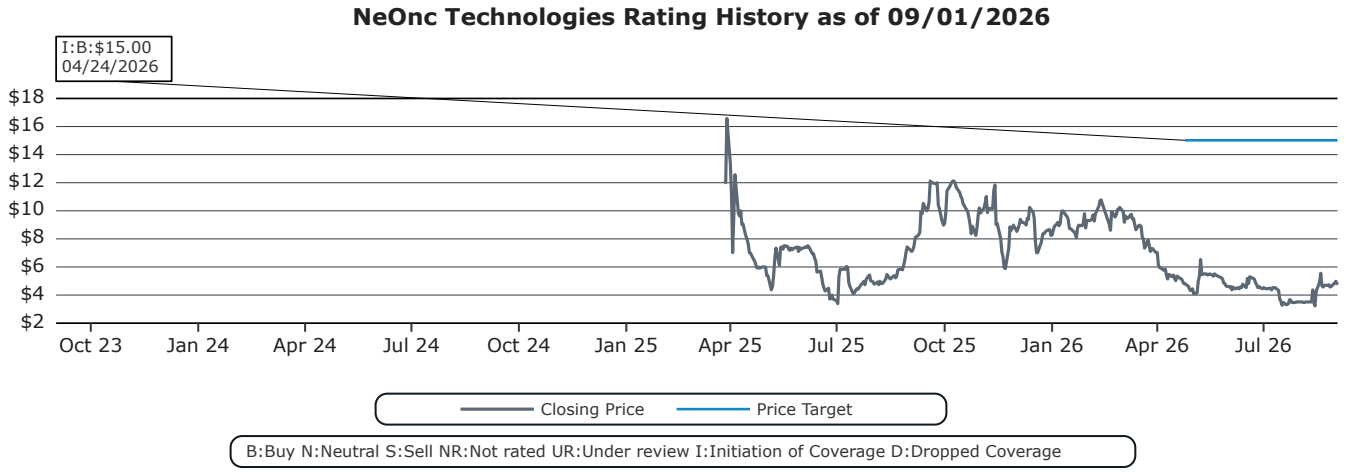
Coya Therapeutics, Inc. (COYA, Buy, $16 PT)

Valuation: We value COYA using a DCF (16.5% discount rate & 1.0% TG rate).
Risks: Coya is an established biotech company. It faces all the standard upside and downside risks for that industry, including unexpected outcomes or safety signals from clinical readouts, regulatory uncertainty, and increasingly complex and price-sensitive commercial markets for its product candidates.



NeOnc Technologies (NTHI, Buy, $15 PT)

Valuation: We value NTHI using a DCF analysis (16% discount rate, 0.5% TG).
Risks: NeOnc is a clinical-stage biotech company. It faces all the standard upside and downside risks for that industry, including unexpected outcomes or safety signals from clinical readouts, regulatory uncertainty, and increasingly complex and price sensitive commercial markets for its product candidates.



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