

Tiziana Life Sciences Announces Podium Presentation at AD/PD of Nasal Anti-CD3 in Alzheimer’s Disease

* **Data shows reduction of microglia activation and improvement in behavior in rodent models of Alzheimer’s disease (AD) and Parkinson’s disease (PD)**
* **Nasal anti-CD3 reduced hemorrhage and edema that occurs with ARIA based on animal studies**
* **Neuroinflammation modulation may be synergistic to approved treatments in Alzheimer’s Disease**

NEW YORK, March 5, 2024 -- Tiziana Life Sciences Ltd. (Nasdaq: [TLSA](https://www.nasdaq.com/market-activity/stocks/tlsa/real-time)) (“Tiziana” or the “Company”), a biotechnology company developing breakthrough neuro-immunomodulation therapies, today announced that Dr. Howard Weiner will present positive data of intranasal anti-CD3 monoclonal antibody in models of Alzheimer’s and Parkinson’s disease at AD/PD, March 5-9, 2024. AD/PD is the annual International Conference on Alzheimer’s and Parkinson’s Diseases and Related Neurological Disorders in Lisbon, Portugal.

**Presentation Information**

**Presenter:** Howard L Weiner, M.D.

**Title:** Nasal immunotherapy of the Monocyte-Microglial axis to treat Alzheimer’s **Disease**

**Day:** Friday, March 8th

**Time:** 15:05 UTC

**Location:** Auditorium V

Howard L. Weiner, M.D., Co-Director of the Ann Romney Center for Neurologic Diseases at Brigham and Women’s Hospital, a founding member of Mass General Brigham, and Chairman of Tiziana's Scientific Advisory Board, stated, “I believe that the modulation of innate immunity via targeting microglia will play a synergistic role with the currently approved anti-amyloid Alzheimer’s treatments. Our research has demonstrated that intranasal rodent anti-CD3 mAb and intranasal fully human anti-CD3 mAb (foralumab) will decrease microglia activation in rodents and humans, respectively. Nasal anti-CD3 has also shown to reduce hemorrhage and edema that occurs with ARIA”

“With the focus on effective Alzheimer’s disease treatments and we have the potential to be a novel, first in class neuroinflammatory modulator,” commented Gabriele Cerrone, Chairman, acting CEO and founder of Tiziana Life Sciences. “Our intranasal approach has large potential as it does not target beta-amyloid or other proteins, but focuses on the neuroinflammatory process itself, which may be complementary or synergistic with existing FDA approved treatments. The data also excitingly shows a reduction of hemorrhage and edema that occurs with ARIA.”

**About Foralumab**

Activated T cells play an important role in the inflammatory process. Foralumab, the only fully human anti-CD3 monoclonal antibody (mAb), binds to the T cell receptor and dampens inflammation by modulating T cell function, thereby suppressing effector features in multiple immune cell subsets. This effect has been demonstrated in patients with COVID and with multiple sclerosis, as well as in healthy normal subjects. The non-active SPMS intranasal foralumab Phase 2 trial dosed its first patient in December of 2023. Immunomodulation by nasal anti-CD3 mAb represents a novel avenue for treatment of neuroinflammatory and neurodegenerative human diseases.[[1]](#footnote-2),[[2]](#footnote-3)

**About Tiziana Life Sciences**

Tiziana Life Sciences is a clinical-stage biopharmaceutical company developing breakthrough therapies using transformational drug delivery technologies to enable alternative routes of immunotherapy. Tiziana’s innovative nasal approach has the potential to provide an improvement in efficacy as well as safety and tolerability compared to intravenous (IV) delivery. Tiziana’s lead candidate, intranasal foralumab, which is the only fully human anti-CD3 mAb, has demonstrated a favorable safety profile and clinical response in patients in studies to date. Tiziana’s technology for alternative routes of immunotherapy has been patented with several applications pending and is expected to allow for broad pipeline applications.

For further inquiries:

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1. https://www.pnas.org/doi/10.1073/pnas.2220272120 [↑](#footnote-ref-2)
2. https://www.pnas.org/doi/10.1073/pnas.2309221120 [↑](#footnote-ref-3)