



MiNK Therapeutics Reports New Data on agenT-797, an allo-iNKT Cell Therapy at ATS 2026; Simultaneously Published in Clinical Immunology Communications

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- agenT-797 demonstrates pathogen suppression, lung immune restoration and tissue repair activation, supporting Ongoing Phase 2 trial
- Sequential immunotherapy with agenT-797 and N-803, an FDA-approved IL-15 superagonist, Anktiva® may provide impactful clinical combination targeting *Coccidioides immitis*

NEW YORK, May 20, 2026 (GLOBE NEWSWIRE) -- [MiNK Therapeutics](#), Inc. (NASDAQ: INKT), a clinical-stage biopharmaceutical company developing allogeneic invariant natural killer T (allo-iNKT) cell therapies to restore immune balance and treat immune-mediated diseases and cancer, today announced new clinical and translational data presented at the American Thoracic Society (ATS) International Conference 2026 and the simultaneous publication in [Clinical Immunology Communications](#)¹ describing the use of sequential immunotherapy with MiNK's off-the-shelf iNKT cell therapy, agenT-797 and N-803, an IL-15 superagonist, in a critically ill patient with unresolving disseminated *Coccidioides immitis* infection, severe acute respiratory distress syndrome, and concurrent hospital-acquired *Pseudomonas aeruginosa pneumonia*.

The poster, titled '**Novel Invariant Natural Killer T Cell (agenT-797) and Interleukin-15 Superagonist (N-803) Combination Immunotherapy for Unresolving *Coccidioides immitis* Infection with Concurrent ARDS**,' was presented by Terese C. Hammond, M.D., Head of Inflammatory and Pulmonary Diseases at MiNK Therapeutics, and describes a sequential immunotherapy regimen pairing agenT-797, MiNK's investigational off-the-shelf iNKT cell therapy, with Anktiva® (N-803), an FDA-approved IL-15 superagonist developed by ImmunityBio, reflecting a combination strategy built on two clinically validated immune mechanisms. The patient was critically ill, facing a convergence of severe threats: disseminated *Coccidioides immitis* (valley fever) fungal infection, severe acute respiratory distress syndrome (ARDS), and concurrent hospital-acquired *Pseudomonas aeruginosa* bacterial pneumonia, all after standard-of-care antifungal, antibacterial, and supportive therapies had failed to halt clinical decline.

Key Clinical and Translational Findings

Following sequential treatment with Anktiva (N-803) and agenT-797, investigators observed:

- Suppression of both fungal and bacterial pathogens
- Active immune cell recruitment into the lung
- Reduced early inflammatory signaling
- Activation of tissue repair and immune regulatory pathways

Immune profiling revealed a coherent biological sequence: Anktiva triggered early immune activation, followed by agenT-797-driven changes consistent with iNKT cell engagement, pathogen control, and a transition from inflammation toward resolution and repair. The pattern suggests the treatment sequence may have helped restore coordinated immune function in a patient whose infection and respiratory failure had continued to progress despite intensive intervention.

"These observations are clinically and biologically important because they point to a pattern we are increasingly seeing across severe lung injury where immune failure itself may become a dominant driver of poor outcomes," said **Terese Hammond, M.D., Head of Inflammatory and Pulmonary Diseases at MiNK Therapeutics**. "As a practicing pulmonary critical care physician, what stands out in this case is not only the reduction in inflammatory signaling, but the evidence of immune recovery and pathogen control in a patient whose infection had persisted despite intensive antifungal, antibacterial and supportive therapy. Many critically ill patients do not decline from early inflammation alone. They deteriorate later from prolonged respiratory failure, secondary infections, immune exhaustion and multi-organ dysfunction. While this is a single case and must be interpreted with appropriate caution, the findings support the clinical question we are now studying prospectively, whether restoring immune competence can change the trajectory for patients with severe pneumonia, respiratory failure and critical illness."

The patient's family ultimately pivoted to comfort care due to recurrent cardiac decompensation in the setting of pre-existing mitral valve disease, acute mitral regurgitation and cardiogenic shock. The authors further supported this rationale in the ATS poster presentation, when chest imaging, clinical data including real time pulmonary arterial catheter and cardiac echocardiography data augmented post-mortem cytokine and biomarker results to prioritize acute heart failure rather than cytokine release or infectious contributions as the most likely mechanism.

The ATS presentation and *Clinical Immunology Communications* publication build on MiNK's prior clinical experience with agenT-797 in moderate to severe ARDS, where the therapy was associated with a favorable safety profile, improved survival rate and decreased secondary infection rates. The new findings also support MiNK's broader platform thesis that iNKT cells may respond dynamically to the immune environment they encounter, with potential relevance across oncology, severe lung injury, persistent infection and immune dysfunction.

Expanding on these findings, MiNK recently [announced the initiation](#) of a randomized Phase 2 clinical trial evaluating agenT-797 plus standard of care compared with placebo plus standard of care in adults with severe acute lung injury and critical illness, including moderate to severe acute hypoxemic respiratory failure due to severe pneumonia, who meet Global ARDS criteria and are admitted to the ICU. The study is designed to prospectively evaluate agenT-797 in a defined critical care population where respiratory recovery, ventilator-free days, secondary infection and survival can be assessed using clinically meaningful endpoints.

The full ATS poster will be available on the publications section of MiNK Therapeutics' website following the presentation.

References

¹ Hammond TC, et al. Rapid suppression of unresolving disseminated coccidioidomycosis infection with combined IL-15 super-agonist (N-803) and invariant natural killer T cell therapy. *Clinical Immunology Communications*. 2026. doi:10.1016/j.clicom.2026.04.002

About MiNK Therapeutics

MiNK Therapeutics is a clinical-stage biopharmaceutical company pioneering the development of allogeneic invariant natural killer T (iNKT) cell therapies and precision immune modulators designed to restore immune balance and drive durable cytotoxic responses. MiNK's proprietary iNKT platform bridges innate and adaptive immunity to address cancer, autoimmune disease, and immune collapse.

Its lead candidate, agenT-797, is an off-the-shelf, cryopreserved iNKT cell therapy currently in clinical trials for solid tumors, graft-versus-host disease (GvHD), and critical pulmonary immune failure. MiNK's pipeline also includes TCR-based and neoantigen-targeted iNKT programs that enable tissue-specific immune activation. With a scalable manufacturing process and broad therapeutic potential, MiNK is advancing a new class of immune reconstitution therapies designed to deliver durable, accessible, and globally deployable treatments.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the federal securities laws, including statements regarding the potential, safety, clinical benefit, and development plans for agenT-797 and other iNKT-based therapies. These statements involve risks and uncertainties, including those described under "Risk Factors" in MiNK's most recent SEC filings. MiNK undertakes no obligation to update these statements except as required by law.

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