MiNK Therapeutics New Data Highlights Its Off-the-Shelf iNKT Cell Therapy Targets Two Key Mechanisms of Tumor Resistance Presented at the Society of Immunotherapy of Cancer (SITC 2026)

November 6, 2026

- ***Colorectal cance**r: in a human liver-metastasis organoid model resistant to botensilimab and balstilimab alone, the addition of agenT-797 supported immune reprogramming and tumor-cell killing, reinforcing the rationale for an ongoing Phase 2 combination study*
- ***Senolytic activity**: agenT-797 and FAP-targeted MiNK-215 cleared senescent cells left behind after chemotherapy; cells linked to relapse and to fibrotic disease, and FAP targeting extended MiNK-215's killing into the tumor's supporting stroma*
- ***Pulmonary development:** findings highlight immune mechanisms relevant to acute inflammation, cellular senescence and tissue remodeling, supporting further investigation in MiNK's randomized Phase 2 trial in acute hypoxemic respiratory failure/ARDS*
- ***Off-the-shelf platform:** MiNK's allogeneic iNKT platform combines a clinical-stage unmodified cell therapy with next-generation engineered candidates, designed for scalable, cryopreserved delivery without patient-specific manufacturing*

NEW YORK, Nov. 06, 2026 (GLOBE NEWSWIRE) -- MiNK Therapeutics, Inc. (NASDAQ: INKT), a clinical-stage biopharmaceutical company pioneering allogeneic invariant natural killer T (iNKT) cell therapies, today announced preclinical findings from two studies presented at the 41st Annual Meeting of the Society for Immunotherapy of Cancer (SITC) in Phoenix, Arizona. The studies address two distinct mechanisms by which tumors resist treatment: immune-suppressive metastatic environments that limit checkpoint blockade, and senescent cells that persist after therapy. Together they show how one foundational iNKT biology, in unmodified and engineered form, can be directed against both through tumor-cell killing, immune modulation and engineered targeting.

In one study, agenT-797 shifted suppressive immune populations toward an immune-stimulatory phenotype in a human organoid model of colorectal cancer liver metastases. Further, agenT-797, in combination with antigen-specific T cells and BOT+BAL, also increased tumor-cell killing.

In a second study, conducted in collaboration with Dr. Jesús Gil and his laboratory at the MRC London Institute of Medical Sciences, Imperial College London, both unmodified agenT-797 and MiNK-215, MiNK's FAP-targeted, IL-15-armored CAR-iNKT candidate, killed senescent tumor cells. FAP-CAR engineering additionally enabled MiNK-215 to kill FAP-expressing fibroblasts, extending the platform's reach into the tumor stroma. These concordant findings reinforce the scientific foundation of MiNK's pipeline and add to the growing body of evidence supporting iNKT cell therapy across cancer indications.

"These findings demonstrate the breadth of our iNKT platform and its potential to address distinct mechanisms of immune dysfunction. In colorectal cancer, agenT-797 reprograms the tumor environment to support T-cell-mediated killing, providing a mechanistic rationale for our ongoing Phase 2 combination study. In parallel, our engineered iNKT platform expands our ability to target senescent and stromal cells implicated in persistent inflammation and tissue remodeling. This biology is also relevant to acute lung injury, where dysregulated inflammation can lead to lasting pulmonary damage and fibrosis. With agenT-797 advancing in a randomized Phase 2 study in acute respiratory failure, we are focused on translating these insights into clinical outcomes across serious diseases," said ***Jennifer Buell, Ph.D., President and Chief Executive Officer of MiNK Therapeutics.***

SITC 2026 Poster Presentations

1. Poster #270: "AgenT-797, An Allogeneic iNKT Cell Therapy, Enables Tumor-cell Killing In A Human Organoid Model of Immune Checkpoint Inhibitor-refractory Colorectal Cancer Liver Metastases."
   Presenter: Shanmugarajan Krishnan, Ph.D.; Saturday, November 7, 2026; 10:00 a.m.–2:00 p.m. and 6:00–8:10 p.m. MST.

2. Poster #177: "Native and engineered allogenic invariant natural killer T (iNKT) cells eliminate senescent tumor cells and fibroblasts in oncogenic in vitro models."
   Presenter: Jing Fang, Ph.D.; Friday, November 6, 2026; 12:15–1:45 p.m. and 6:40–8:10 p.m. MST.

Location: Phoenix Convention Center, North Building, Lower Level, Exhibition Halls 4–6, Phoenix, Arizona.

Reprogramming Immune Resistance in Colorectal Cancer Liver Metastases

Patients with microsatellite-stable (MSS) colorectal cancer and liver metastases represent a substantial unmet need with no approved immunotherapy option: the immune-suppressive metastatic environment limits the activity of existing therapies, and liver involvement is associated with resistance to checkpoint blockade.

In a human organoid model incorporating colorectal cancer, liver-associated and immune cells, agenT-797 shifted suppressive immune populations

toward a more immune-stimulatory phenotype, depleting CD206+ M2-like macrophages and increasing CD86+/HLA-DR+ antigen-presenting cells. When combined with antigen-specific T cells and botensilimab (BOT), an Fc-enhanced anti-CTLA-4 antibody, plus balstilimab (BAL), an anti-PD-1 antibody, the triple combination increased CD8 T-cell density, raised granzyme B, IFN-γ, TNF-α, CXCL-9, CCL3 and IL-8, reduced EpCAM+ tumor-cell content and increased tumor-cell apoptosis. Critically, the model was refractory to BOT+BAL alone, which produced no statistically significant tumor-cell killing. Adding agenT-797 unlocked killing, and the full effect required all three components.

These findings provide a mechanistic rationale for the ongoing Phase 2 study of agenT-797 with BOT+BAL in previously treated microsatellite stable (MSS) colorectal cancer with liver metastases (CRC LM) (NCT07550088; *funded by Scripps Institute, PI Darren Sigal*).

Eliminating Senescent Cells with Unmodified and Engineered iNKT Therapies

Senescent cells can accumulate after cancer treatment or other cellular stress. Although they stop dividing, they can release signals that contribute to inflammation, tissue remodeling and immune escape. Dr. Jesús Gil and his team evaluated whether unmodified and engineered iNKT cell therapies could recognize and eliminate these cells and identified the cellular factors and resistance mechanisms that determine whether iNKT killing succeeds.

MiNK-215 is an allogeneic CAR-iNKT cell engineered to target fibroblast activation protein (FAP), which is expressed on cancer-associated and senescent fibroblasts — they support cells that surround tumors — and armored with IL-15 to support persistence and immune activation.

In this model, three findings emerged. First, both unmodified agenT-797 and MiNK-215 killed therapy-induced senescent breast cancer cells, which carried high levels of the NKG2D stress ligands MICA/B and ULBP2/5/6 and low checkpoint-ligand expression. Second, FAP-CAR engineering enabled MiNK-215 to kill non-senescent, FAP-high lung fibroblasts that unmodified agenT-797 did not; a targeting capability conferred by the CAR. Third, senescent lung fibroblasts resisted both therapies and carried elevated PD-L1, PD-L2 and CD155 —***making checkpoint-ligand upregulation a defined, addressable obstacle that the next generation of engineered iNKT therapies can be built to overcome.***

Loading the lipid antigen α-galactosylceramide (α-GalCer) restored unmodified agenT-797 killing of the less-sensitive senescent fibroblasts, indicating that this resistance can be overcome through antigen presentation and informing engineering and combination strategies. Because cellular senescence and fibroblast activation contribute to persistent tissue remodeling in cancer and fibrotic diseases, these findings provide a rationale for further investigation of engineered iNKT therapies in fibroinflammatory conditions.

“The different responses across these models show that senescence does not create a single uniform immune target. Observing the signals associated with sensitivity and resistance will help us define where iNKT-based approaches are most useful,” said **Jesús Gil, Professor of Cell Proliferation, MRC London Institute of Medical Sciences, Imperial College London**, and corresponding author of the study.

Extending iNKT Biology from Acute Inflammation to Tissue Remodeling and Fibrosis

MiNK is advancing agenT-797 in C-1300-02, a randomized, placebo-controlled Phase 2 trial in acute hypoxemic respiratory failure, including ARDS (NCT07615010), evaluating acute clinical outcomes. Severe acute lung injury involves dysregulated immune responses, epithelial and endothelial damage, and persistent inflammation that, in some patients, progresses to abnormal tissue repair and pulmonary fibrosis.

The SITC findings highlight complementary mechanisms relevant to this continuum: agenT-797's ability to reprogram suppressive immune environments, and the capacity of unmodified and engineered iNKT cells to recognize selected senescent and FAP-expressing cells involved in tissue remodeling. *These observations extend the potential of MiNK's pulmonary strategy, from intervening during acute inflammatory injury to exploring engineered iNKT approaches for persistent fibroinflammatory disease.*

About MiNK Therapeutics

MiNK Therapeutics is a clinical-stage biopharmaceutical company pioneering allogeneic invariant natural killer T (iNKT) cell therapies and precision-targeted immune technologies. MiNK’s proprietary platform is designed to restore immune balance and drive cytotoxic responses across cancer, immune-mediated diseases, and pulmonary immune failure. MiNK’s lead candidate, agenT-797, is an off-the-shelf iNKT cell therapy currently in clinical development for GvHD, solid tumors, and severe pulmonary inflammation. With a scalable cryopreserved manufacturing process and differentiated biology bridging innate and adaptive immunity, MiNK is committed to developing next-generation immune reconstitution therapies. For more information, visit www.minktherapeutics.com or follow us on X @MiNK_iNKT.

About agenT-797

agenT-797 is an allogeneic invariant natural killer T (iNKT) cell therapy that harnesses the dual power of innate and adaptive immunity. iNKTs function as “master regulators,” combining the cytotoxic capabilities of NK cells with T-cell–like antigen recognition and memory. This unique biology enables a robust, pathogen-agnostic immune response that can be directed against hard-to-treat tumors. Manufactured by MiNK Therapeutics in Lexington, MA, agenT-797 is a scalable, off-the-shelf product designed to provide accessible, transformative treatment options. In clinical trials, agenT-797 can bolster peripheral memory T-cell activation, enhance tumor infiltration, and potentially improve outcomes for patients with solid cancers (Cytryn et al. AACR IO 2024, Oncogene. 2024) and to combat inflammation in critically ill patients with severe respiratory pathology (Nature Communications. 2024).

About MiNK-215

MiNK-215 is an investigational, allogeneic, off-the-shelf CAR-iNKT cell therapy engineered to target fibroblast activation protein (FAP) expressed on stromal cells within the tumor microenvironment. MiNK-215 also incorporates soluble IL-15, designed to enhance cell persistence and immune activation. (Cancer Immunology Research)

Forward-Looking Statements

This press release contains forward-looking statements made pursuant to the safe harbor provisions of the federal securities laws, including statements regarding the therapeutic potential, safety, anticipated benefits and clinical development of agenT-797 and MiNK-215; the potential relevance of preclinical findings to cancer, senescence and fibrotic diseases; possible combination strategies; future studies; the design, enrollment and timing of data from the Phase 2 study of agenT-797 with botensilimab and balstilimab and from the C-1300-02 trial; the initiation and funding of IND-enabling studies of MiNK-215; the scope, operation and continuation of MiNK’s named-patient access program; and MiNK’s development plans. Preclinical findings may not be predictive of results in patients. Such statements involve risks and uncertainties detailed in MiNK’s most recent filings

with the Securities and Exchange Commission. MiNK cautions investors not to place undue reliance on these statements, which speak only as of the date of this release.

Contacts

Investor Contact: 917-362-1370 | investor@minktherapeutics.com

Media Contact: 781-674-4428 | communications@minktherapeutics.com

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