

MIINK THERAPEUTICS · NASDAQ: INKT

A Novel Cell Therapy for Acute Lung Failure

agenT-797 in severe pneumonia with moderate-to-severe ARDS — a one-time, off-the-shelf infusion of one billion iNKT cells

Corporate Deck

October 2026

Disclaimer and Forward-Looking Statements

This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on information currently available to management. All statements other than statements of historical fact contained in this presentation are forward-looking statements. Forward-looking statements include, but are not limited to, statements concerning: the therapeutic potential of agentT-797 and iNKT cells; the mechanism of action, potency and safety of agentT-797; interim or top-line data; future development plans and timelines, including preclinical, clinical, regulatory, manufacturing and commercial; estimated treatment costs, pricing and market opportunity; the sufficiency of the Company's capital resources; MiNK's ability to continue to successfully manufacture iNKT cells, including capacity and scalability; and any other statements containing the words "may," "believes," "expects," "anticipates," "hopes," "intends," "plans," "forecasts," "estimates," "will" and similar expressions.

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MiNK Therapeutics: Off-the-Shelf iNKT Cell Therapy for Immune Failure

Clinical-stage biotech (Nasdaq: INKT) developing allogeneic invariant natural killer T cells for life-threatening immune dysfunction

THE PLATFORM

1 Immune restoration, not suppression

iNKT cells sense lipid and stress signals and coordinate innate and adaptive immunity

2 Off-the-shelf by design

No HLA matching, no lymphodepletion; stored on site and infused once

3 Clinical validation

Phase 1/2 survival and safety data in severe viral ARDS published in Nature Communications (2024)

agentT-797 PROGRAMS

Severe pneumonia with ARDS

Randomized Phase 2 (C-1300-02) · lead indication

Graft-versus-host disease prevention

Phase 1 with UW–Madison · STTR and philanthropy grant-funded

Solid tumors (gastric, MSS colorectal)

Phase 2 combination studies (*funded by Scripps*)

Pediatric cancers (PRAME-TCR iNKT)

IND-enabling with C-Further

Partners and funders include ARPA-H, NIH/NIAID and C-Further

Lead goal: one agentT-797 infusion of 1×10^9 to keep patients with pneumonia and ARDS alive and off VV-ECMO

agentT-797 and all MiNK product candidates are investigational and have not been approved by the FDA or any other regulatory authority

Sources: MiNK Therapeutics Form 10-Q (Q2 2026) and company releases; Hammond TC, et al. Nat Commun. 2024. Program stages as of October 2026.

Partnerships Support Development, Clinical Execution and Patient Access

DEVELOPMENT FUNDING AND COLLABORATION



BoSS · agenT-797 as cell platform

Up to \$87M total BoSS funding across participants; Draper-led. Goal: room-temperature cell-therapy stabilization.



Pediatric PRAME-TCR iNKT

Reimbursed R&D + downstream participation.
>\$1M+ for IND-enabling work; double-digit share of downstream commercial revenue.



GvHD prevention · Phase 1

Investigator-sponsored trial at UW–Madison; supported by an NIH/NIAID STTR grant and the Mary Gooze award.

CLINICAL EXECUTION AND PATIENT ACCESS



Colorectal cancer with liver metastases

Phase 2 of agenT-797 in combination therapy for checkpoint-refractory MSS disease, at Scripps.



Randomized Phase 2 · ARDS

Clinical site for the C-1300-02 trial; first patients enrolled in Lviv, with US sites activating.

MiNK NAMED-PATIENT ACCESS

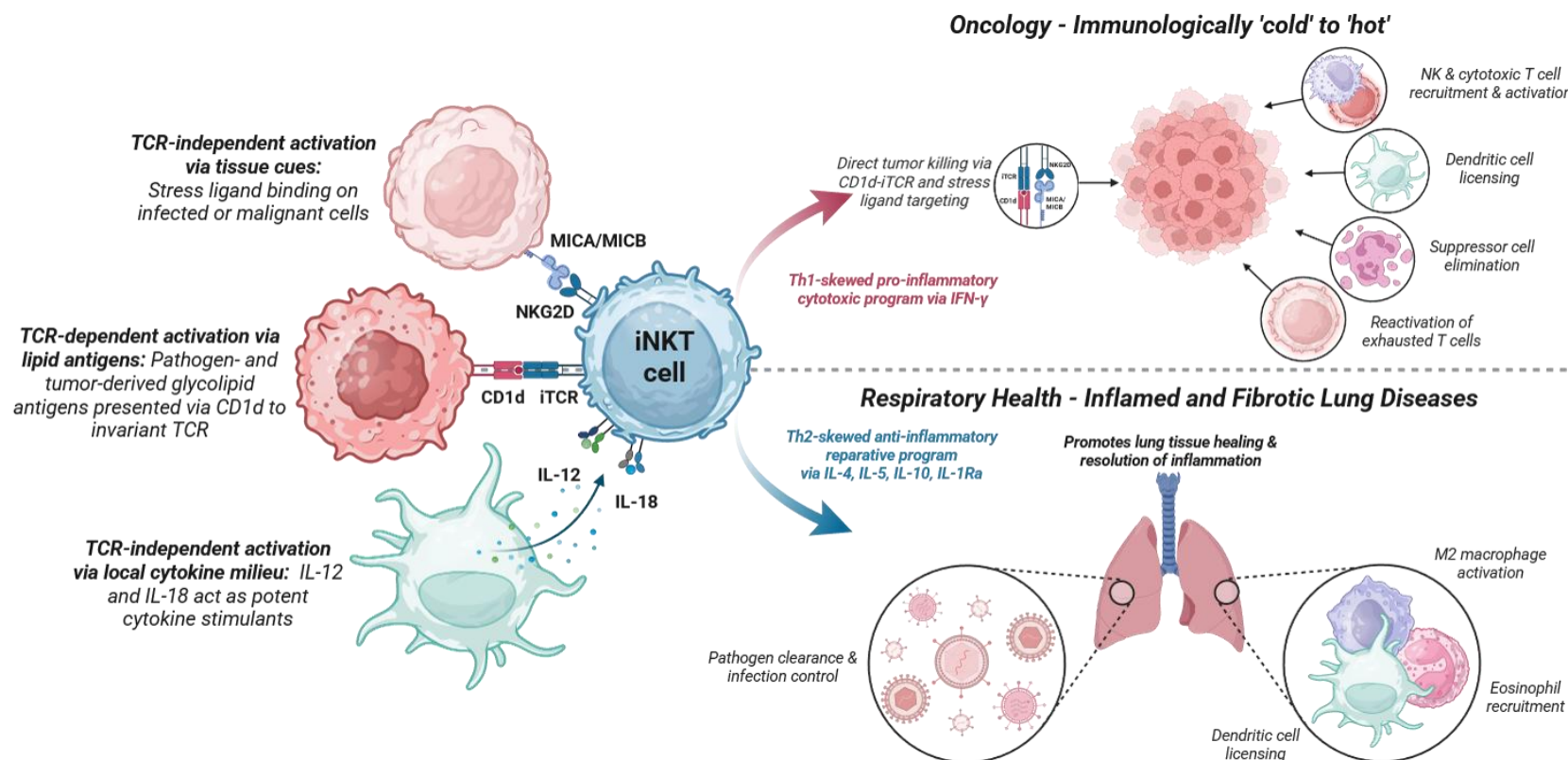
International named-patient access

Where authorized: physician-requested, paid per-patient supply with ODC and BAP; not a commercial approval.

Sources: MiNK Therapeutics releases: ARPA-H BoSS, 30 Sep 2026; C-Further, 10 Mar 2026; UW–Madison, 8 Jan 2026; Q2 2026 results (named-patient access, Lviv enrollment), 13 Aug 2026; Scripps CRC study, 2 Oct 2026. ARPA-H figure is total program funding shared across all participants, not a MiNK award. Full citations in the reference section.

iNKT SCIENCE – POISED, DYNAMIC EFFECTORS IN ONCOLOGY & RESPIRATORY HEALTH

iNKT cells inflame tumors and repair injured lungs



OFF-THE-SHELF BY DESIGN

No HLA matching

iNKT cells see lipids on CD1d, nearly identical in everyone, so one donor batch treats any patient

No lymphodepletion

Given without conditioning chemotherapy, so frail, ICU and transplant patients can be dosed

Stored on site

Cells wait frozen at the hospital, so patients are dosed when needed with no manufacturing wait or shipping

HOW iNKT CELLS RESPOND TO CONTEXT

iNKT cells recognize CD1d-presented lipids through their invariant TCR and stress ligands through NKG2D; cytokine release and CD40L-CD40 signaling then coordinate innate effectors and dendritic-cell priming of adaptive T-cell responses.

The Problem: ARDS Represents a Large and Entirely Unserved Population

~93,000 Annual US Patients With Moderate-to-Severe ARDS in the Initial Label Population

~286,000

US ARDS cases
annually

~200,000

Moderate-to-severe ARDS
70% of cases

~157,000

On invasive ventilation
78.6%

~93,000

Pneumonia-associated
trial population

FDA-APPROVED PHARMACOLOGIC THERAPIES FOR ARDS

None -Care is supportive only

- Care is highly concentrated in ICUs, so a focused key-account model can reach the highest-volume US centers
- Pneumonia is the largest single ARDS risk factor; extrapulmonary sepsis, aspiration and trauma represent the label-expansion path
- One healthy-donor leukopak yields hundreds to thousands of doses, so supply scales without a per-patient manufacturing constraint

Market build: 349M US population × 82 ARDS cases per 100,000 per year (Rubenfeld GD, et al. N Engl J Med. 2005) = ~286,000 cases; × 70% moderate-to-severe and × 78.6% invasive mechanical ventilation (LUNG SAFE, Bellani G, et al. JAMA. 2016;315(8):788-800) = ~157,000; × 59.4% with pneumonia as an ARDS risk factor (LUNG SAFE) = ~93,000. Note that LUNG SAFE risk factors are non-exclusive; 59.4% is the share of ARDS patients with pneumonia as a risk factor. Cross-check: ~1.59M annual US adult CAP hospitalizations, 17.1% requiring ICU admission (Ramirez JA, et al. Clin Infect Dis. 2017;65(11):1806-1812, population-based Louisville cohort extrapolated nationally), of which 2.7% of mechanically ventilated CAP patients go on to develop ARDS (Kasotakis G, et al. J Intensive Care Med. 2024;39(10), US Premier Healthcare Database).

SUMMARY

Ongoing Phase 2/3 Program Testing agentT-797 in Moderate-to-Severe ARDS

Target seamless Phase 2/3 protocol; Plan to have Phase 2 patients count toward approval; Targeting potential FDA approval in 4Q 2029*

THE THERAPY

1 infusion

Off-the-shelf iNKT cells in a single 1B-cell dose

THE EVIDENCE

Higher observed survival

Well tolerated, no dose-limiting toxicities; longer survival in ventilated patients vs non-randomized in-hospital controls¹

THE ENDPOINT

ECMO-free survival

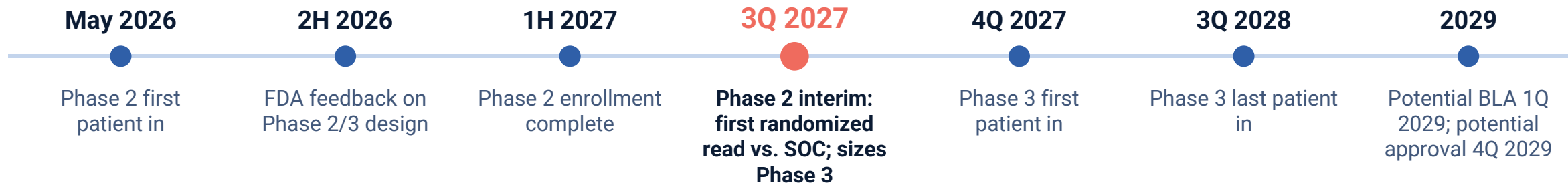
Free of VV-ECMO at day 14, then 28-day mortality; $\geq 80\%$ power in Phase 2b, up to 95% in Phase 3

THE NEED

~93,000

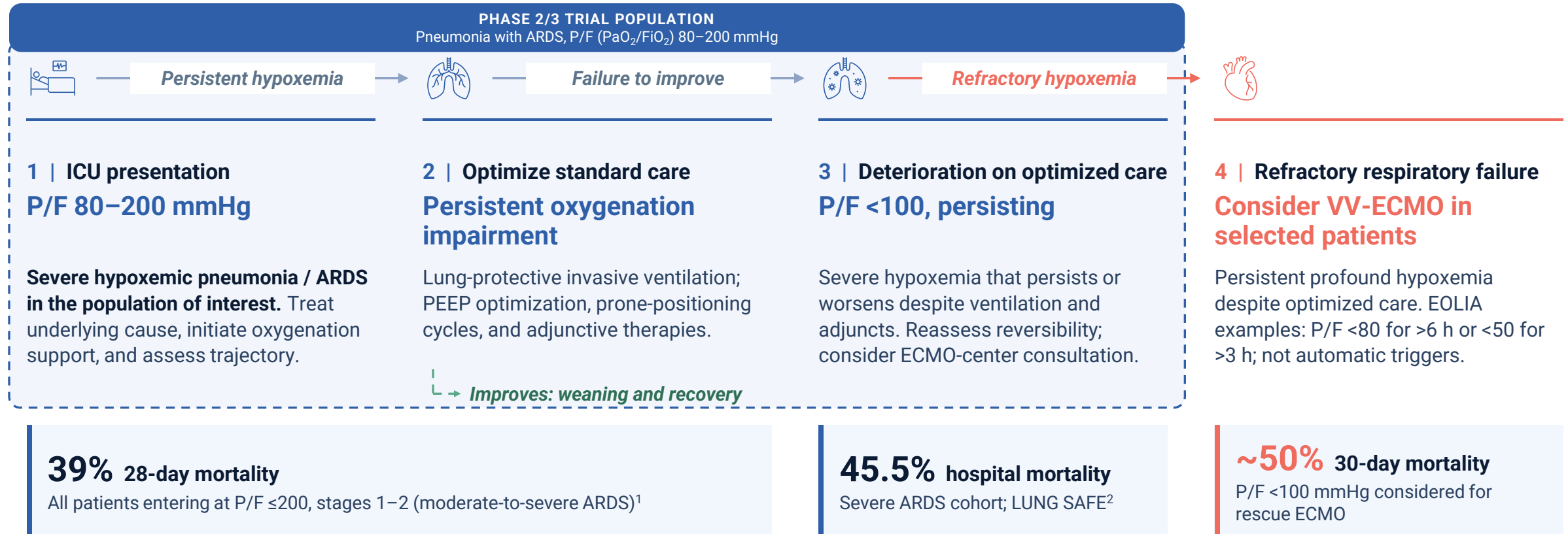
US patients a year; no FDA-approved drug

KEY UPCOMING CATALYSTS



Power: win ratio, two-sided $\alpha=0.05$, by simulation against published control-arm benchmarks. 1. Hammond TC, et al. Nat Commun. 2024;15:974 (Phase 1/2, NCT04582201); open-label, single-arm (n=21). Survival is compared with in-hospital controls, not a randomized arm; this is not a head-to-head comparison and may not predict randomized results. *Timelines are estimates and depend on enrollment, data and FDA review; FDA has not agreed that Phase 2 patients will count toward approval or that the program is registrational.

Worsening Hypoxemia Drives Escalation Toward Rescue ECMO



Separate published cohorts and endpoints; not sequential risk estimates or MiNK trial outcomes.

Investigational opportunity: address immune dysfunction before rescue escalation

Can earlier immune-directed treatment reduce progression to refractory respiratory failure?

¹ Villar J, et al. Sci Rep. 2023;13:1543 (38.6%). ² Bellani G, et al. JAMA. 2016;315:788-800 (LUNG SAFE, 46.1%). ³ Combes A, et al. N Engl J Med. 2018;378:1965-75 (EOLIA). ATS ARDS guideline 2024.

THE PATIENT

Our Patient: Hypoxemic Pneumonia With Moderate-to-Severe ARDS

The Phase 2/3 population: ICU patients whose lungs are failing, enrolled within 7 days of onset

WHO QUALIFIES

- 1 ICU admission with severe pneumonia**
Any cause: viral, bacterial, fungal or mixed, with or without trauma
- 2 Acute hypoxemic respiratory failure**
The lungs can no longer oxygenate the blood without intensive support
- 3 Moderate-to-severe ARDS**
Meets Global ARDS criteria, with a P/F ratio ≤ 200 mmHg
- 4 Enrolled within 7 days of onset**
Treated early, while the lung injury is still evolving

Excluded: shock needing more than two vasopressors, pregnancy, prior cytokine release syndrome

WHY THIS GROUP

~93,000 US patients a year: the 59% of ventilated ARDS cases caused by pneumonia

39% 28-day mortality in moderate-to-severe ARDS on today's care

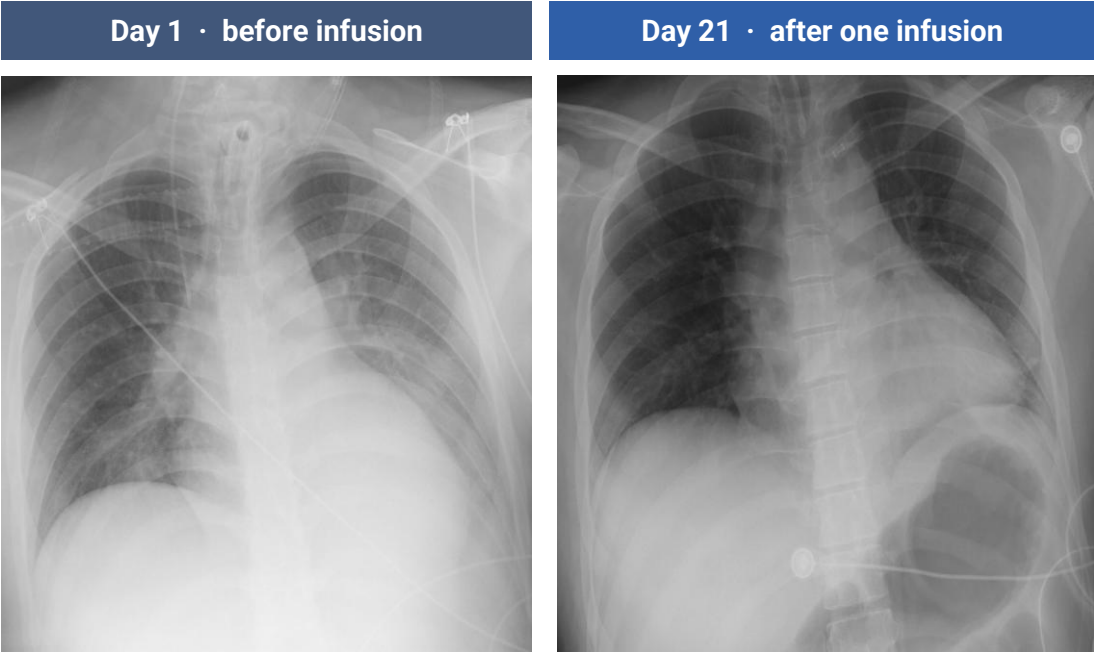
~50% 30-day mortality once hypoxemia is severe enough to consider rescue VV-ECMO

0 FDA-approved drugs for ARDS

Eligibility: ClinicalTrials.gov NCT07615010 (agenT-797 plus standard of care in severe pneumonia with moderate-to-severe acute hypoxemic respiratory failure); other protocol criteria may apply. Villar J, et al. Sci Rep. 2023;13:1543; pneumonia share from LUNG SAFE, Bellani G, et al. JAMA. 2016;315(8):788-800. ECMO-eligible mortality: Combes A, et al. N Engl J Med. 2018;378:1965-75 (EOLIA).

Representative Case(s) Treated with agent-797

agent-797 Is Designed to Functionally Replace the iNKT Compartment That ARDS Depletes



Pt-001 at ICU admission: multilobar *S. pneumoniae* pneumonia with bacteremia, invasive ventilation, P/F 136

Day 21: off pressors, breathing trials. **Day 28:** alive, afebrile, off pressors

REPRESENTATIVE AFTER ONE INFUSION · DAY 1 → DAY 28

	Day 1	Day 28	Improvement
Oxygenation PaO ₂ /FiO ₂ , mmHg	136.5 →	399	2.9×
Organ dysfunction SOFA-2 score	8 →	1	-7 pts
Systemic inflammation CRP, mg/L	186 →	70.3	-62%
Infection marker Procalcitonin, ng/mL	2.5 →	1.7	-32%
Coagulation D-dimer, mg/L	2.1 →	0.76	-64%

BAL: IMMUNE AND LUNG-REPAIR SIGNALS RISE BY DAY 7-14

Epithelial repair EGF, LIF	Angiogenesis FGF-2, VEGF-A	Inflammation control IL-10, IL-1RA
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ACROSS THE PROGRAM

70% Survival vs 10% (non-randomized)	2 of 2 Alive at day 28, first two Phase 2 patients	>91 Patients treated to date	0 Severe CRS or ICANS cases observed
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Pt-001 X-ray, laboratory and BAL data as presented at MHSRS 2026 (single uncontrolled case); full Pt-001 and Pt-002 case detail in the appendix. Program figures: Hammond TC, et al. Nat Commun. 2024 (Phase 1/2, open-label, single-arm, n=21; 10% survival in in-hospital controls, not a randomized arm or head-to-head comparison); MiNK Corporate Presentation, June 2026 (91 patients); Phase 2 Pt-001 and Pt-002 as presented at MHSRS 2026.

Phase 1 Results: Encouraging Survival Observations in Severe Viral ARDS

70% 30-day survival
with agenT-797

10% 30-day survival, in-
hospital control*

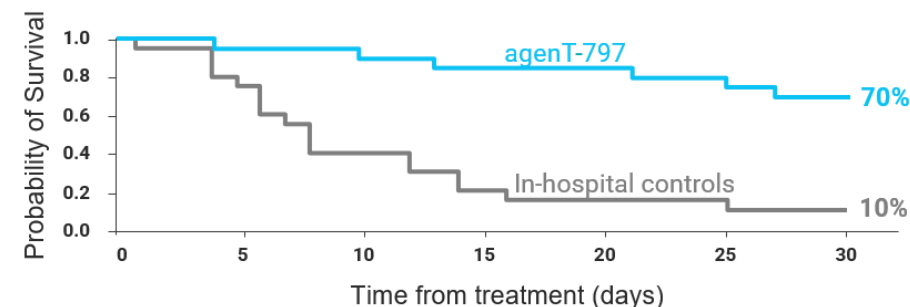
Phase 1/2 Trial — Moderate-to-Severe ARDS (n=21)*

- 30-day survival of 70% in AgenT-797 treated patients (n=21) vs. 10% in-hospital control
- 75% survival in patients on VV-ECMO vs 30% in in-hospital ECMO controls (non-randomized)
- Secondary bacterial pneumonia in 15% of patients at the recommended dose; published rates in comparable ventilated populations range up to 84%†
- Only 5% of patients experienced a treatment-related adverse event ≥ Grade 3

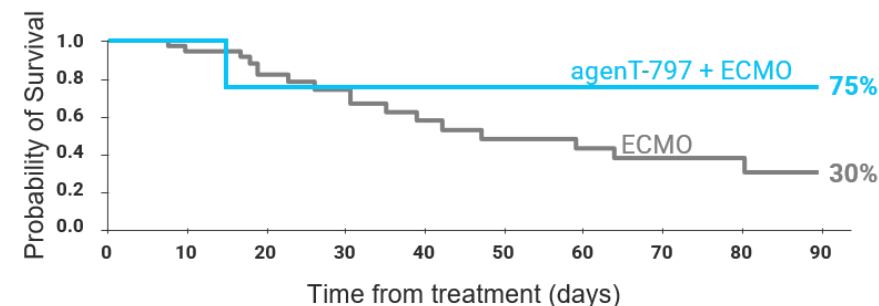
**Severely ill ICU patients on mechanical ventilation, including a cohort on ECMO*

Hammond TC et al., Nature Communications 2024; ATS 2024; the Phase 1/2 study was open-label, single-arm and conducted in the early start of the pandemic 2020. *Survival is compared with in-hospital controls, not a randomized arm. †Cross-study comparison with published literature.

Increased survival vs case control



Increased survival on VV-ECMO



agenT-797 Is Well Tolerated Across Indications

91 Patients Treated to Date, with No Severe CRS and No ICANS Observed

91

PATIENTS TREATED

oncology, ARDS and GvHD

1.5%

SERIOUS TRAE

1 of 67 in completed trials

0%

TRAE LEADING TO DEATH

none observed

0

SEVERE CRS OR ICANS

no cases across programs

Treatment-related adverse events (n=67)	n (%)
Any TRAE	23 (34.3%)
Any TRAE ≥ Grade 3	2 (3.0%)
Any TRAE leading to discontinuation	2 (3.0%)
Serious TRAE	1 (1.5%)
TRAE leading to death	0 (0%)

WHY THIS PROFILE MATTERS IN THE ICU

- agenT-797 is delivered by standard 30-minute IV infusion with no cell laboratory, no lymphodepleting conditioning and no apheresis — nothing that adds ICU burden or delay.
- In the Phase 1/2 ARDS study, only 5% of patients experienced a treatment-related adverse event of Grade 3 or higher, and secondary bacterial pneumonia occurred in 15% of patients at the recommended dose (published rates in ventilated patients range up to 84%†).

Sources: MiNK Therapeutics Corporate Presentation, June 2026 (91 patients treated as of June 2026; TRAE table reflects completed trials, n=67); Hammond TC, et al. ATS 2023, Poster P1172; Hammond TC, et al. Nat Commun. 2024. TRAE = treatment-related adverse event; CRS = cytokine release syndrome; ICANS = immune effector cell-associated neurotoxicity syndrome. Grade ≥3 TRAEs comprised one Grade 3 anemia and one Grade 4 dyspnea. †Cross-study comparison with published literature, not head-to-head; populations, definitions and standard of care differ, and no conclusions on relative efficacy should be drawn.

Adaptive Phase 2b/3 Protocol for a Potentially Registrational Readout

C-1300-02 · Seeking FDA feedback on the clinical development plan, including trial protocol; FDA response expected 2H 2026

Population

- Severe ARDS on guideline-concordant SOC, including sCAP complicated by ARDS
- Stratified by hyper- / hypo-inflammatory phenotype
- Enriched for severity: P/F ratio $80 < 200$ mmHg

Endpoints

Primary: hierarchical win ratio — tier 1: alive and free of VV-ECMO at day 14; tier 2: all-cause mortality through day 28

Key secondary: time to VV-ECMO escalation; 28-day all-cause mortality; ECMO-free survival through day 28

Secondary: ventilator- and vasopressor-free days, ICU-free days, time to pathogen clearance, SOFA trajectory

Why the design is efficient

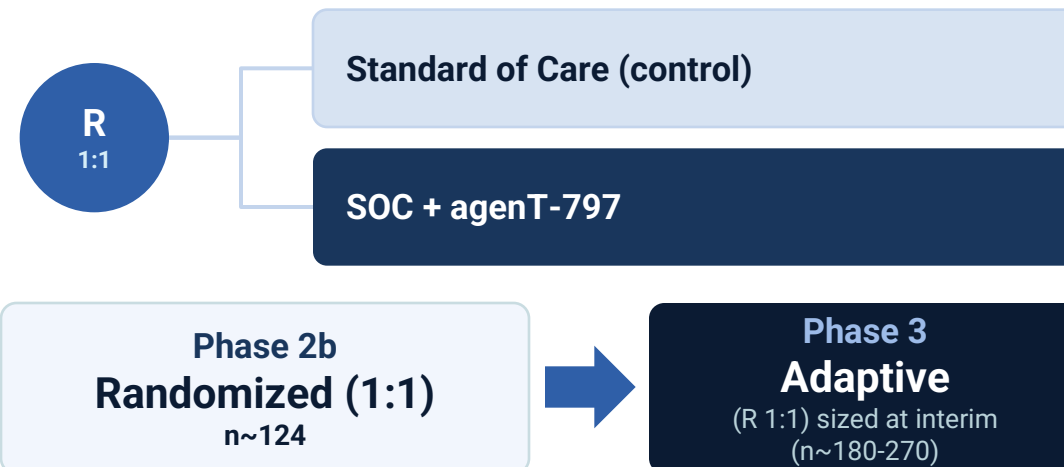
Efficient endpoint: scoring ECMO prevention, then survival, requires far fewer patients than a mortality-only design

Robust power: $\geq 80\%$ in Phase 2b; up to 95% in Phase 3

Protected integrity: interim rules fixed before unblinding; Type I error preserved

C-1300-02 protocol; proposed adaptive Phase 2b/3 design. The design is subject to FDA review; FDA has not agreed that this study will be registrational or that Phase 2b patients will count toward approval.

Phase 2b/3 Trial Schema — C-1300-02



SCIENTIFIC ADVISORY BOARD — CRITICAL ILLNESS & TRAUMA



Michael Matthey



Michael Harhay



Shibani Pati



John Holcomb



Terese Hammond

First Two Phase 2 Patients Alive and Recovering at Day 28

One infusion added to standard of care; oxygenation, organ function and inflammation improved (MHSRS 2026)

Phase 2 (C-1300-02)

- Randomized, controlled study of agentT-797 plus standard of care versus standard of care alone in severe pneumonia with moderate-to-severe ARDS
- Initiated in Lviv, Ukraine with First Lviv Medical and the UNBROKEN Foundation, with U.S. sites in parallel
- Hierarchical primary endpoint: alive and VV-ECMO-free at day 14, then 28-day all-cause mortality
- Expansion to US actively underway; FDA safe to proceed completed and site activation launched



Enrollment initiated May
2026

Observations

- Both mechanically ventilated with moderate-to-severe hypoxemic respiratory failure and resistant co-infections
- Each received one infusion of agentT-797 (1×10^9 iNKT cells) plus guideline-directed standard of care
- Day 28: both alive; Pt-001 afebrile and off pressors; Pt-002 off the ventilator, later discharged to rehab
- No infusion reactions; one SAE (CoNS bacteremia, Pt-002) judged unlikely related
- Uncontrolled cases; the randomized interim in 3Q 2027 tests against standard of care

Source: Pt-001 and Pt-002 as presented at MHSRS 2026; full case detail in the appendix. Trial: C-1300-02, ClinicalTrials.gov NCT07615010. Individual patient outcomes are not intended to predict results in the randomized study.

Pricing Reflects Cost Efficiency, Including Manufacturing

>\$9.5Bn total annual US opportunity

Target population · THE UNMET NEED

~93,000

US patients per year with
**ventilated, pneumonia-
associated moderate-to-
severe ARDS**

~39%

die within 28 days on
current care¹

PRICE IN CONTEXT · COST OF ILLNESS PER PATIENT (2026 USD)

~\$498,000

Prolonged ventilation,
first year

~\$346,000

VV-ECMO admission

\$108–158K

ARDS index admission,
US range (2021 USD)

~\$70,000

8 fewer ventilated ICU
days at ~\$8,200/day

THREE VALUE LEVERS, IN ORDER OF WEIGHT

1 Mortality, the primary driver

A clinically important absolute survival gain in a syndrome with no approved therapy.

2 Hospital margin under DRG payment

~\$8,200 per ventilated ICU day fully allocated (\$1,200–\$1,500 direct-variable). Payment is per discharge, so avoided days add margin.

3 Avoided catastrophic tails

VV-ECMO admissions cost \$229,000–\$346,000, above payment. Avoiding prolonged ventilation saves ~\$498,000 in year one.

No approved pharmacologic therapy competes for this population today

Market estimate from the company's most conservative pricing model; population build on slide 6. ¹28-day mortality in moderate-to-severe ARDS, Villar J, et al. Sci Rep. 2023;13:1543. Cost of illness: Boucher PE, et al. Chest. 2022 (systematic review, US index admission \$107,886–\$157,800); Siuba MT, et al. PLoS One. 2022;17(1):e0263000; Dasta JF, et al. Crit Care Med. 2005;33(6):1266-71; Kahn JM, et al. Med Care. 2008;46(12):1226-33; Ratiu A, et al. J Extra Corpor Technol. 2025;57:218-24; Mazzeffi M, et al. J Cardiothorac Vasc Anesth. 2023;37(8):1449-55; Unroe M, et al. Ann Intern Med. 2010;153(3):167-75. Inflated to 2026 USD (BLS CPIMEDSL).



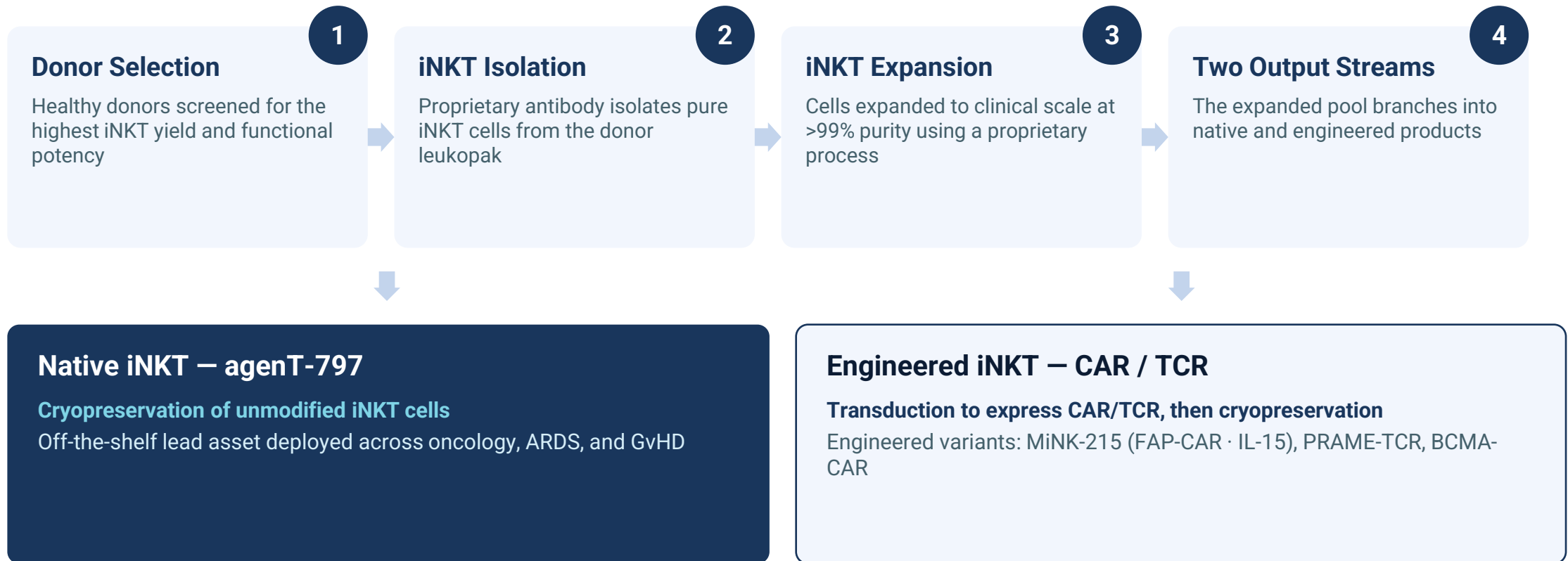
APPENDIX

Scalable Manufacturing

One donor platform, manufactured once and deployed off-the-shelf across every indication

GMP Manufacture of Healthy Donor-Derived iNKT Cells

One healthy-donor leukopak yields billions of cells — a single manufacturing run deployed broadly across patients
One leukopak -> 20-30 days -> hundreds of doses



No requirement for a cell laboratory; cells administered by standard IV infusion in ~30 minutes and are currently in community centers globally

Experienced Leadership, Board and Scientific Advisors

Cell-therapy development leadership and an experienced board, with critical-care, trauma and iNKT expertise guiding the program

MANAGEMENT	SAB — CRITICAL ILLNESS AND TRAUMA	SAB — iNKT AND CELL THERAPY
Jennifer S. Buell, PhD President and Chief Executive Officer Terese C. Hammond, MD Head of Inflammatory and Pulmonary Diseases Marco Purbhoo, PhD Translational Research Nils-Petter Rudqvist, PhD Research Operations Thiago Favano Portfolio Operations Hayley Cullen Manufacturing	Michael Matthay, MD UCSF NHLBI ARDS Network and PETAL investigator; STAT MSC trial Michael Harhay, PhD University of Pennsylvania Methodologist for ICU trial endpoints and design John Holcomb, MD UAB/Trauma/ARDS Led PROPPR trauma trial; former Army trauma surgeon Shibani Pati, MD, PhD UCSF Trauma-induced lung injury and cell-therapy biology Lauren Baker, PhD INSIGHT Regulatory Expert	Yelena Janjigian, MD Chief, GI Oncology, Memorial Sloan Kettering Marcela Maus, MD, PhD Director, Cellular Immunotherapy, Mass General Manuel Hidalgo, MD, PhD Professor of Medicine, Weill Cornell Jenny Gumperz, PhD Professor, Microbiology and Immunology, UW–Madison Lydia Lynch, PhD Professor, Molecular Biology, Princeton Mark Exley, PhD CSO, LiFT BioSciences Robert Stein, MD, PhD Venture Partner, Samsara BioCapital

Cell-therapy builders run the program; ARDS and trauma trial leaders advise it.

Titles as of October 2026. SAB = Scientific Advisory Board. PETAL = NHLBI Prevention and Early Treatment of Acute Lung Injury Network; PROPPR = Pragmatic, Randomized Optimal Platelet and Plasma Ratios trial.

MiNK THERAPEUTICS · NASDAQ: INKT

One immune cell. Cancer. Lung failure. Inflammation.

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APPENDIX

Additional Detail

Mechanism, regulatory detail, platform optionality, manufacturing, and full source references



APPENDIX

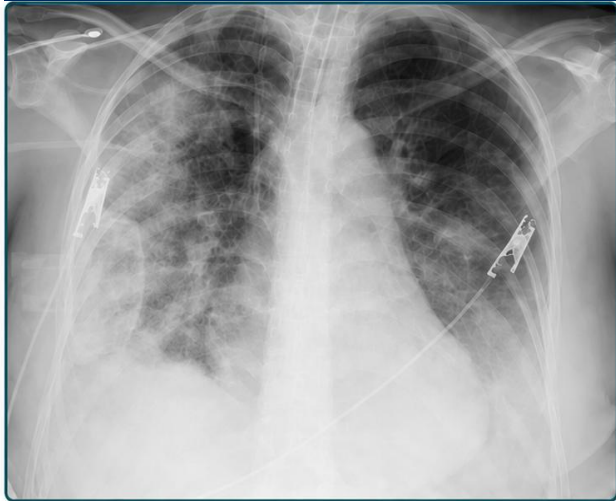
Phase 2 Preliminary Data

Pt-001 and Pt-002 imaging, laboratory and BAL biomarker data, as presented at MHSRS 2026

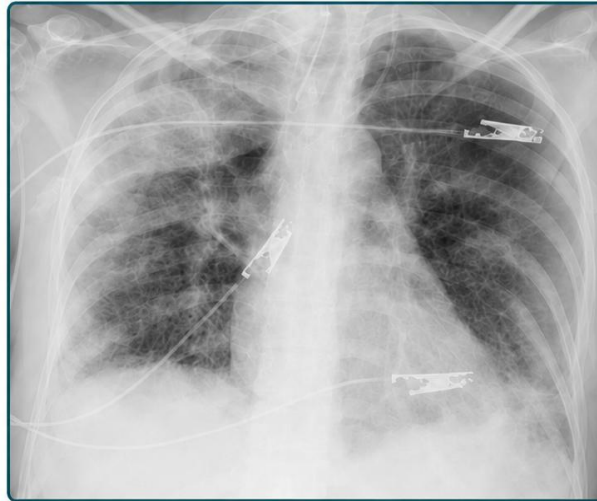
First Treated Patient at First Lviv Territorial Medical Union

**41 y/o F with poorly controlled diabetes,
Pneumococcal Sepsis, 38.6°C**

Day 1



Day 21



ICU admission: Blood and sputum + *S. pneumonia*, MDRO *Klebsiella*, *Acinetobacter* and *pseudomonas* within 24 hours of intubation; Ventilator PEEP 12, PaO2 109, 80% FiO2 P/F ratio 136

Day 21: Off pressors, afebrile, minimal sedation, starting spontaneous breathing trials

Day 28: Alive, afebrile, off pressors, P/F ratio 388

Antibiotic	<i>S. mitis</i> / <i>S. oralis</i>	<i>S. pneumoniae</i>	<i>A. baumannii</i>	<i>P. aeruginosa</i>	<i>K. pneumoniae</i>	<i>S. mitis</i>
Ampicillin	S	-	-	-	-	I/R
Ampicillin/Sulbactam	-	-	R	-	-	-
Amoxicillin/Clavulanate	-	-	-	-	R	-
Benzylpenicillin	-	S	-	-	-	I/R
Piperacillin/Tazobactam	-	-	-	R	R	-
Aztreonam	-	-	-	I	R	-
Cefepime	-	-	-	R	R	-
Cefotaxime	-	-	-	-	R	-
Ceftazidime	-	-	-	R	R	-
Ceftazidime/Avibactam	-	-	-	R	R	-
Ceftriaxone	S	-	-	-	R	S
Clindamycin	S	R	-	-	-	S
Ciprofloxacin	-	-	R	R	R	-
Erythromycin	-	R	-	-	-	-
Amikacin	-	-	R	R	R	-
Gentamicin	-	-	R	-	R	-
Tobramycin	-	-	S	R	R	-
Trimethoprim/Sulfamethoxazole	-	S	R	-	R	-
Vancomycin	S	S	-	-	-	S
Imipenem	-	-	R	R	R	-
Meropenem	-	-	R	R	R	-
Levofloxacin	-	I	R	R	R	-
Linezolid	-	S	-	-	-	-
Moxifloxacin	-	S	-	-	-	-
Oxacillin	-	R	-	-	-	-
Rifampin	-	S	-	-	-	-
Tetracycline	-	R	-	-	-	-

Antibiotics administered:

Pre-Tx→D2 Vancomycin, Meropenem, Doxycycline

D2→D6 Benzylpenicillin, Azithromycin

D6→D20 Ampicillin/Subactam, Polymyxin B, Colistin, Gentamicin (D10)

D21→D28 Tigecycline, Colistin, Co-trimoxazole, Metronidazole (D28)

Pt-001 Clinical Activity: Physiologic and Immune Recovery Through Day 28

Gas exchange and organ function

agenT-797 1×10⁹ cells + SOC · Dosed 14 Jul 2026

Assessment	Day 1	Day 7	Day 14	Day 21	Day 28
PaO ₂ /FiO ₂ , mmHg	136.5	172.1	244	232.5	399
Respiratory support	Invasive MV	Invasive MV	Invasive MV	Invasive MV	Invasive MV
SOFA-2 score	8	5	2	1	1

Inflammatory markers

Marker	D1	D7	D14	D21	D28
CRP, mg/L	186	122	126	61.5	70.3
Procalcitonin, ng/mL	2.5	2.23	1.22	0.53	1.7
Ferritin, µg/L	736	1108	922	571	752
D-dimer, mg/L	2.1	1.57	2.1	1.52	0.76

Hematology

Parameter	D1	D7	D14	D21	D28
WBC, ×10 ⁹ /L	10	8.3	2.91	6.56	7.21
Lymphocytes, ×10 ⁹ /L	0.56	0.95	0.2	0.79	0.63
Hemoglobin, g/L	95	77	84	91	81
Platelets, ×10 ⁹ /L	188	142	135	225	178

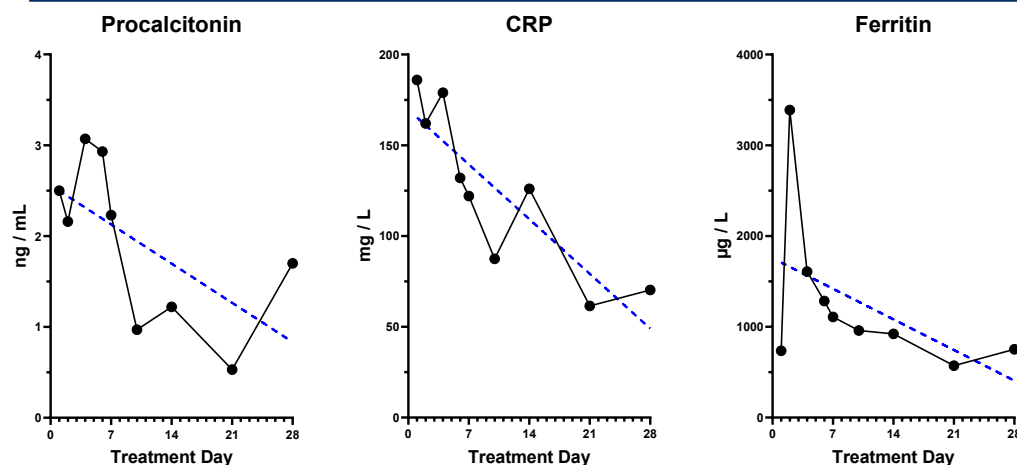
ICU Admission: Multi-lobar pneumonia, severe hypoxemia and respiratory failure due to *Streptococcus pneumoniae*, bacteremia. Febrile (38.6 °C). Ventilator PEEP 12, PaO₂ 109, 80% FiO₂ P/F ratio 136

Day 21: Off pressors, afebrile, minimal sedation, starting spontaneous breathing trials

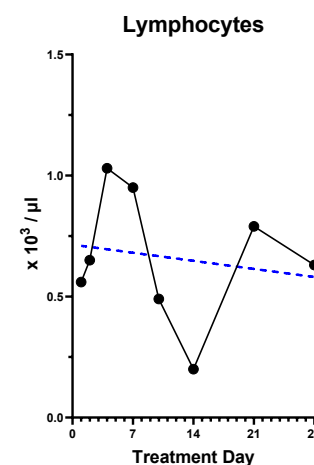
Day 28 outcome: Alive, afebrile, off pressors, P/F ratio 388

Clinical and BAL Biomarkers Show Coordinated Immune Recovery and Lung Repair Following agentT-797

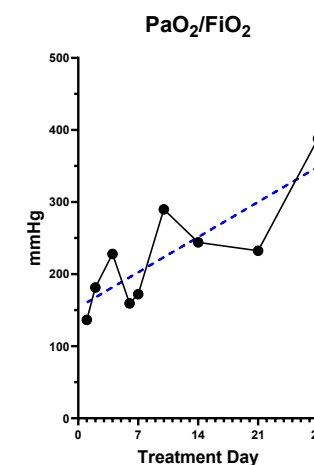
Inflammation resolution



Immune reconstitution

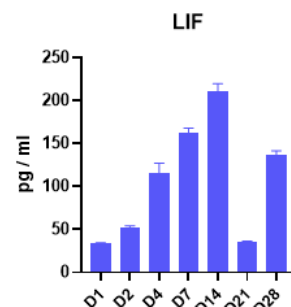
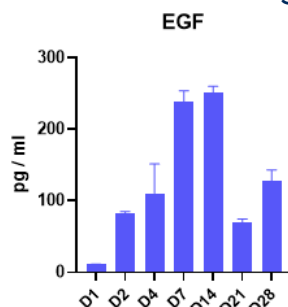


Lung reconstitution

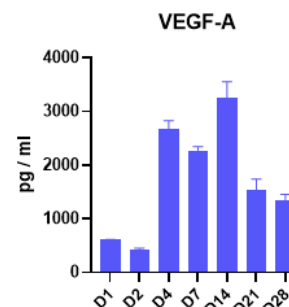
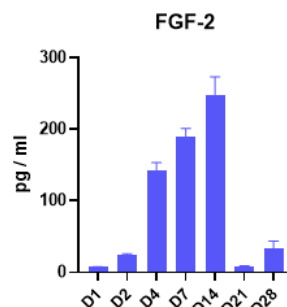


BAL Biomarker Analysis indicates activation of multiple tissue repair pathways

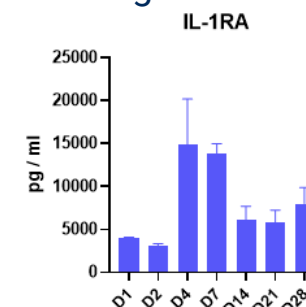
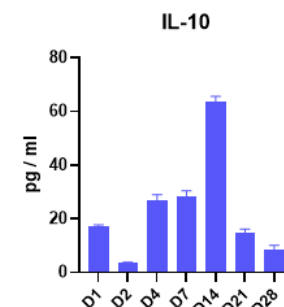
Epithelial protection / regeneration



Angiogenesis / Post-injury vascularization



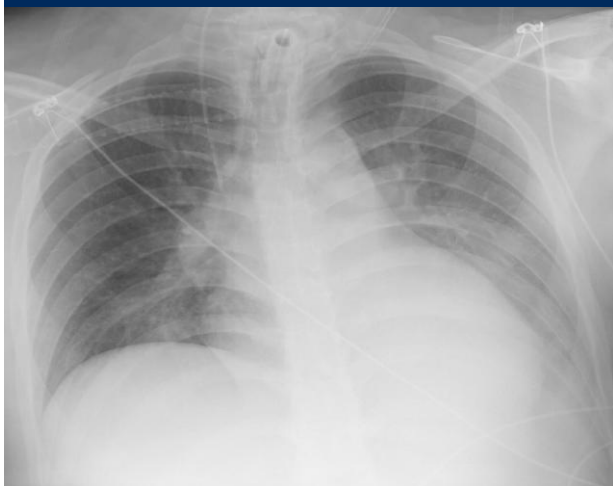
Inflammation control / Limiting tissue damage



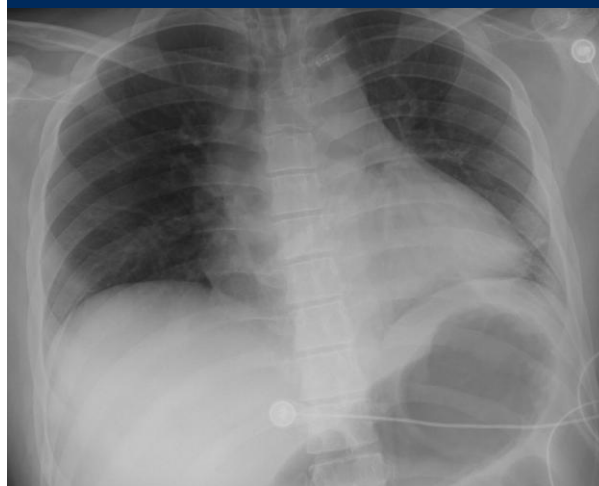
Second Treated Patient at First Lviv Territorial Medical Union

18 y/o M with EtOH associated MVA, polytrauma
(TBI, chest, long bone), 40.1 °C

Day 2



Day 26



ICU admission: Suspected: MDRO *Klebsiella* + *E. coli* prior to treatment; Ventilator PEEP 10, PaO₂ 86, 50% FiO₂ P/F ratio 172

Day 28: Alive, afebrile, P/F ratio 467, off ventilator, no new infections

Post Day 28: Decannulated, surgical fixation of long bone fractures, discharged to rehab

Antibiotic	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>	<i>Elizabethkingia</i> <i>meningoseptica</i>
Amikacin	R	R
Amoxicillin-clavulanic acid	R	—
Gentamicin	R	—
Trimethoprim-sulfamethoxazole	R	—
Imipenem	R	—
Meropenem	R	—
Piperacillin-tazobactam	R	I
Tobramycin	R	R
Cefepime	R	R
Cefotaxime	R	—
Ceftazidime	R	—
Ciprofloxacin - non-meningitis	R	—
Ciprofloxacin	—	R
Aztreonam	R	—
Levofloxacin	R	—
Ceftazidime-avibactam	S	—
Ceftriaxone	R	—

Antibiotics administered:

Pre-Tx→D16 Colistin, Meropenem
D17→D26 Tigecycline, Piperacillin, Tazobactam

Pt-002 Clinical Activity: Physiologic and Immune Recovery Through Day 28

Gas exchange and organ function

Assessment	Day 1	Day 7	Day 14	Day 21	Day 28
PaO ₂ /FiO ₂	172	171.6	169.2	359.5	467
Respiratory support	Invasive MV	Invasive MV	Invasive MV	Invasive MV	Room air
SOFA-2 score	4	2	3	1	0

Inflammatory markers

Marker	D1	D7	D14	D21	D28
CRP, mg/L	217	38.8	NC	10.7	3.59
Procalcitonin, ng/mL	0.74	0.95	0.25	<0.1	0.49
Ferritin, µg/L	786	1,180	597	313	295
D-dimer, mg/L	3.2	9.9	10.7	6.5	8.77

Hematology

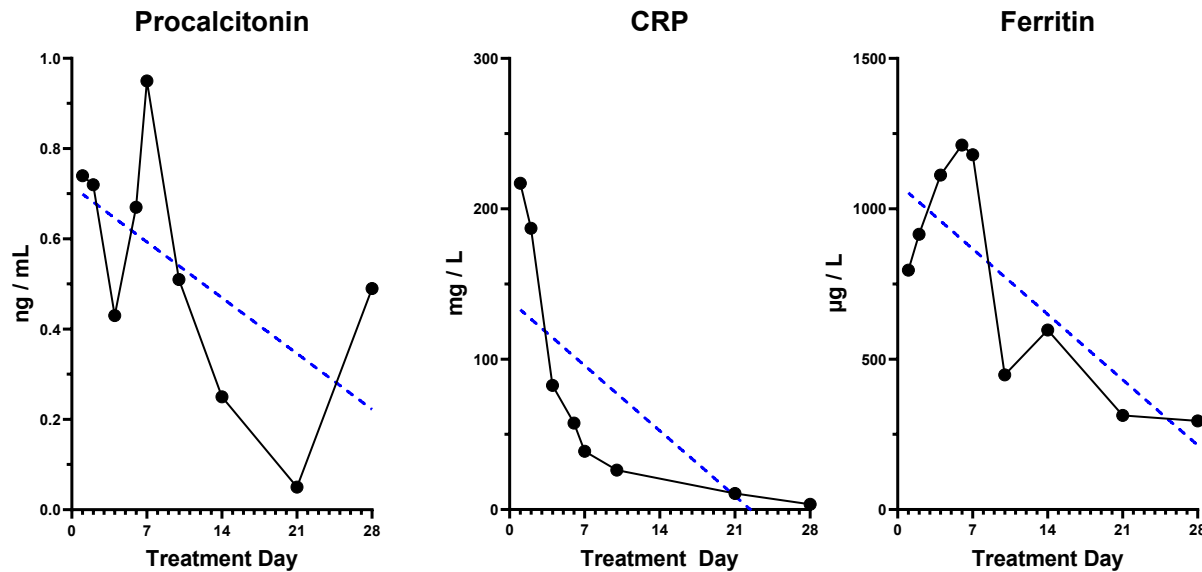
Parameter	D1	D7	D14	D21	D28
WBC, ×10 ⁹ /L	4.06	6.82	4.69	5.61	8.43
Lymphocytes, ×10 ⁹ /L	0.38	0.83	1.25	1.15	2.16
Hemoglobin, g/L	92	81	89	105	124
Platelets, ×10 ⁹ /L	194	309	137	196	317

Day 28 outcome: alive on room air, SpO₂ 98%, SOFA-2 of 0; hypoxemia resolved 504 h after infusion, with 6 ventilator-free days. ICU care ended 15 Jul; total ICU stay 39 days including the pre-enrollment period.

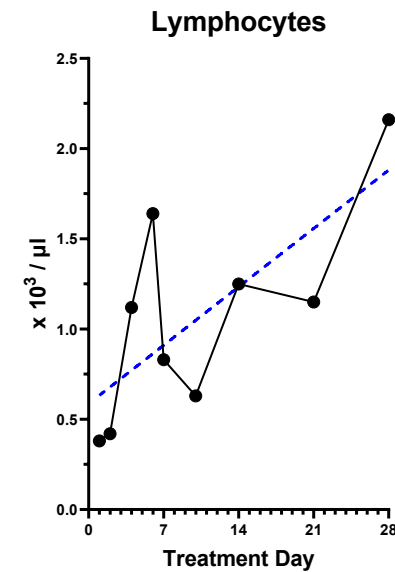
cSAE: coagulase-negative staphylococcal bacteremia (1 and 8 Jul) judged unlikely related to agenT-797 — no infusion reaction, concurrent inflammatory and immune recovery; blood cultures negative by 15 Jul.

Clinical Biomarkers Show Coordinated Immune Recovery Following agentT-797

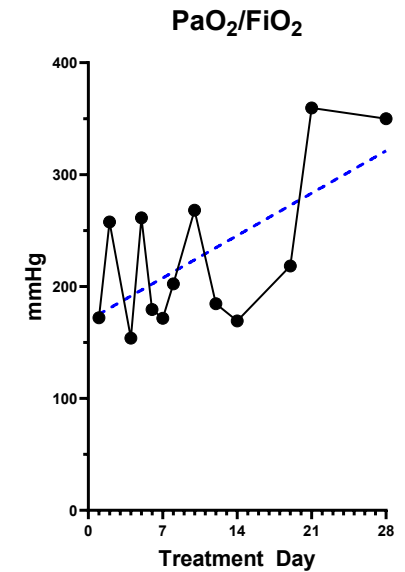
Inflammation resolution



Immune reconstitution



Lung reconstitution



Conclusions: Both Patients Alive and Recovering at Day 28

Pt-001

41 y/o F · pneumococcal sepsis with bacteremia · MDRO co-infection

Oxygenation, P/F (mmHg) **136 → 399**

Organ dysfunction, SOFA-2 **8 → 1**

CRP (mg/L) **186 → 70.3**

Day 28 **Alive, afebrile, off pressors**

Pt-002

18 y/o M · polytrauma (TBI, chest, long bone) · suspected MDRO infection

Oxygenation, P/F (mmHg) **172 → 467**

Organ dysfunction, SOFA-2 **0 at day 28**

Day 28 **Alive, off ventilator, room air**

After day 28 **Decannulated; to rehab**

WHAT THE CASES SHOW

- **Survival:** both patients, each with a resistant co-infection, were alive at day 28 after a single 1×10^9 -cell dose added to standard of care
- **Recovery across systems:** oxygenation, organ function and inflammatory markers improved together; Pt-001 BAL showed repair (EGF, LIF, FGF-2, VEGF-A) and anti-inflammatory (IL-10, IL-1RA) signals rising by day 7–14
- **Safety:** no infusion reactions; the one serious adverse event (CoNS bacteremia, Pt-002) was judged unlikely related to agent T-797
- **Next:** two uncontrolled cases; the randomized Phase 2 interim in Q3 2027 tests whether this holds against standard of care



APPENDIX

iNKT Cells in Acute Inflammation

Recalibrating, not suppressing, the immune system in severe lung injury and GvHD

One Donor Platform: ARDS Now, GvHD, Fibrosis and Solid Tumors Next

Every program below runs on the same donor-derived iNKT cells and the same manufacturing and release process.

CRITICAL CARE	INFLAMMATION AND FIBROSIS	ONCOLOGY
STATUS agenT-797 on a potentially registrational Phase 2/3 path in moderate-to-severe ARDS; Phase 1 in acute GvHD activated for dosing. OBSERVATIONS 70% 30-day survival in severe viral ARDS (single-arm, n=21) against 10% in non-randomized in-hospital controls, and no severe CRS or ICANS in any indication studied. NEXT READOUT Randomized Phase 2 survival readout; first GvHD safety cohort.	STATUS MiNK-215 in preclinical development across the acute-to-fibrotic lung continuum. OBSERVATIONS iNKT-derived IFN-γ restrains the TGF-β myofibroblast axis and reprograms profibrotic macrophages, the same biology that resolves ARDS. NEXT READOUT IND-enabling package; fibrosis model readouts.	STATUS Gastric/GEJ: agenT-797 + BOT/BAL, Phase 2 ALL: agenT-797 + blinatumomab with allo-HSCT Liver-met MSS CRC: agenT-797 + BOT/BAL OBSERVATIONS Complete remission in a refractory patient, and cold tumors converted hot with CD8 T-cell infiltration. NEXT READOUT Phase 2 combination data; engineered candidate selection.

One donor, one process, one release: the ARDS program pays for the manufacturing and safety base that every other program inherits.

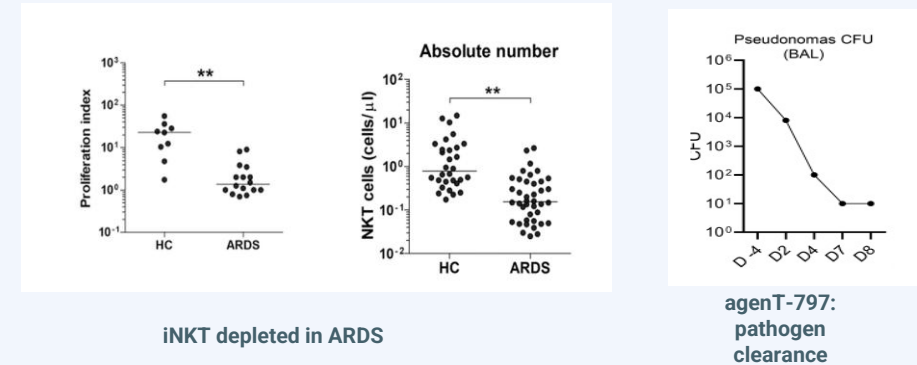
BOT/BAL = botensilimab and balstilimab; ALL = acute lymphoblastic leukemia. Data on clinical and appendix slides. Registrational status is subject to FDA agreement.

The Lung Is the Largest Immune Interface – and a Target for iNKT Therapy

A Critical Balance Between Tolerance and Immunity

- Continuously exposed to pathogens, particulates, and environmental insults – yet must avoid chronic, tissue-damaging inflammation
- iNKT cells are tissue-resident sentinels uniquely positioned to sense infection and injury rapidly, then orchestrate either pathogen clearance or repair
- In severe viral or bacterial ARDS, intrinsic iNKT cells are depleted – agenT-797 is designed to functionally replace them with a single infusion
- In Pulmonary Fibrosis, decreased pulmonary iNKTs reveal poorer prognosis (Hammond et al. Keystone 2026)

iNKT biology governs lung infection control



Left: reduced iNKT proliferation and number, ARDS vs. controls (Park KJ et al., Front Immunol 2024). Right: BAL Pseudomonas clearance after agenT-797 + N-803 (MiNK case; ASGCT 2026).

agenT-797 Resets the Cytokine Program in Viral ARDS: Down on Inflammation, Up on Repair



Suppresses Hyperinflammation

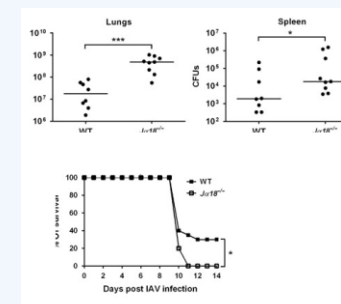
- IL-18 reduced — a cytokine linked to decreased ARDS survival and a validated biomarker of severe acute kidney injury
- VEGF-D reduced — an angiogenic factor implicated in detrimental alloreactive immune responses
- CRS-associated markers (IL-6, IP-10, TNF α , IFN γ , IL-17A, CRP, ferritin, D-dimer) trend down from baseline through Days 7–28
- TNF α , IL-1 β and neutrophil counts fall in bronchoalveolar lavage, correlating with resolution of infection



Promotes Repair and Resolution

- IL-1RA and VEGF-D rise over time — signaling a shift toward tissue recovery and vascular repair
- IL-4 and IL-5 increase in the lung — driving macrophage-mediated remodeling and eosinophil-supported repair
- Monocyte/macrophage influx in BAL tracks with infection clearance as neutrophil counts fall
- Net effect: immune recalibration — not blanket activation or suppression

iNKT loss worsens infection control

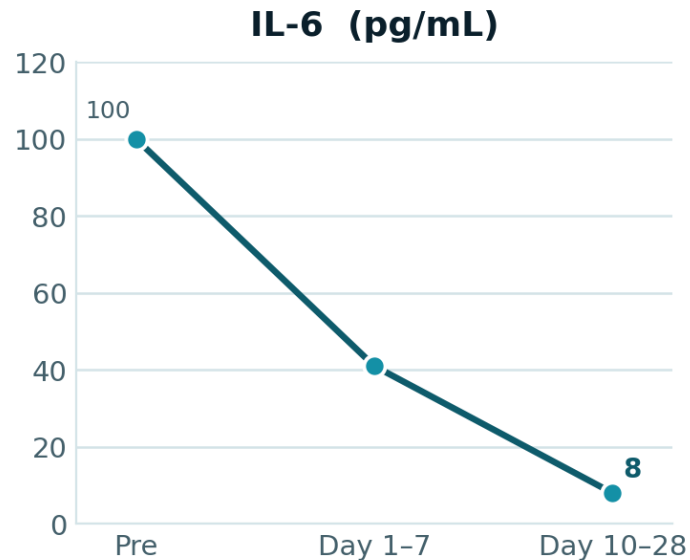


iNKT-deficient mice (Ja18^{-/-}) show higher bacterial burden and worse survival after secondary pneumococcal infection post-influenza.

Barthelemy A et al., *Mucosal Immunol* 2017.

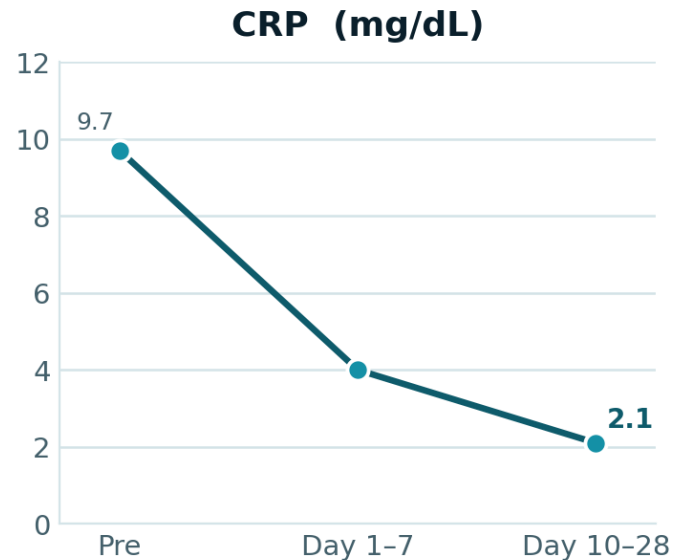
Reversing the Hyper-Inflammatory Profile

A single agentT-797 infusion drives a rapid, observable decline in the systemic biomarkers that drive mortality in hyper-inflammatory ARDS.



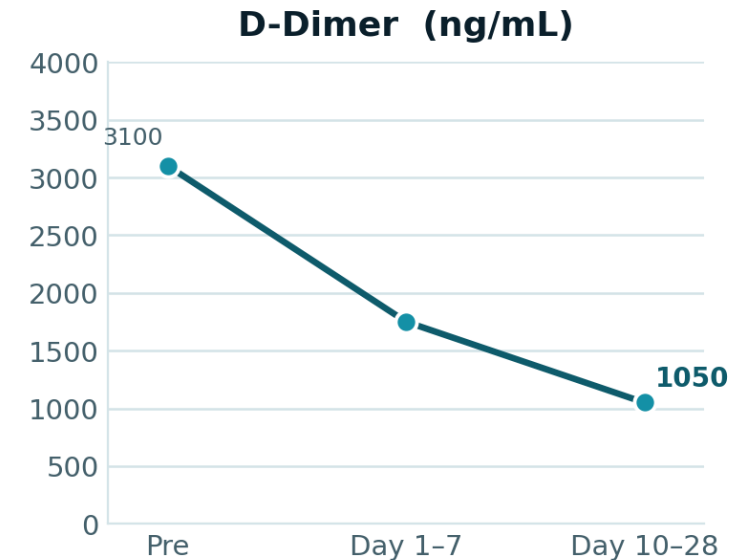
Systemic dampening

Rapid reduction in the biomarkers (IL-6, CRP, D-Dimer) that drive mortality in the hyper-inflammatory subphenotype



Targeted shift, not blanket suppression

Promotes an anti-inflammatory systemic environment while preventing secondary multi-organ dysfunction



In vivo persistence

Infused allogeneic iNKT cells remain detectable during follow-up, providing sustained, active immunomodulation

Representative systemic biomarker trajectories. Hammond TC et al., Nature Communications 2024; ATS 2024.



APPENDIX

Engineering the Next Wave with IL-15-FARP-CAR-iNKT

From acute lung injury to fibrotic disease: extending the iNKT platform with armored, tissue-directed designs

MiNK-215: Extending iNKT Biology Across the Acute-to-Fibrotic Lung Disease Continuum

Acute Injury

ARDS / acute lung injury

Resolving Inflammation

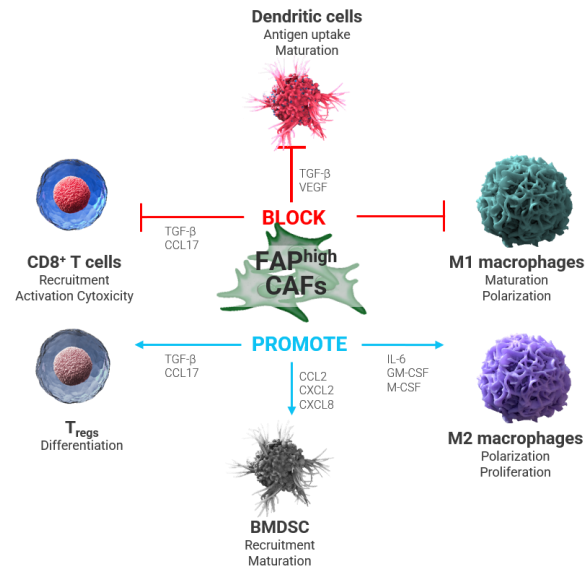
Cytokine recalibration, repair signaling

Chronic Fibrosis

IPF — aberrant repair, progressive scarring

agenT-797 acts upstream in acute injury — MiNK-215 is purpose-engineered for the downstream fibrotic endpoint

How FAP^{high} fibroblasts shape immunity



FAP-CAR: Targeting the Fibroblasts That Suppress Immunity

- FAP^{high} fibroblasts block CD8⁺ T cells, dendritic cells and M1 macrophages, and promote Tregs, M2 macrophages and MDSCs (TGF- β , VEGF, CCL17, IL-6)
- FAP^{high} CAFs occur in **>90% of epithelial-derived tumors**; FAP is also upregulated on activated fibroblasts in fibrotic lung
- FAP-CAR directs iNKTs to these cells rather than relying on native trafficking alone



IL-15 Armoring: Persistence in Hostile Tissue

- IL-15 co-expression supports iNKT persistence in fibroblast-rich, immunosuppressive tissue
- In an orthotopic lung tumor model, MiNK-215 with host T cells kept tumor burden near baseline and **~90% of mice alive at day 100** (slide 36)

Source: MiNK, CAR-TCR Summit, Feb 2024; Boi S, et al. ASGCT 2023, Poster 1488. In vivo data to date are from solid-tumor models.

Armoring the Chassis: How MiNK Engineers Native iNKT Biology for Solid Tumor Precision



Targeting Module (CAR or TCR)

Adds antigen-specific recognition — FAP, PRAME, NY-ESO-1, BCMA — on top of native CD1d/NK-receptor sensing, widening the addressable tumor and tissue set



Cytokine Payload (IL-15)

Co-expressed IL-15 supports persistence and proliferative fitness in immunosuppressive tumor or fibrotic microenvironments without systemic IL-15 toxicity



Preserved Innate Layer

Engineering adds a targeting module without deleting native iTCR and NK-receptor cytotoxicity — engineered iNKTs retain dual-pathway killing



Same Manufacturing Backbone

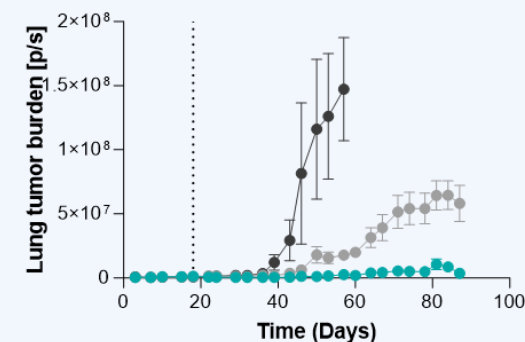
Engineered products are produced on the same donor-derived, GMP, cryopreservation platform as native agent-797 — no new manufacturing paradigm required

MiNK-215 in vivo: orthotopic lung tumor model

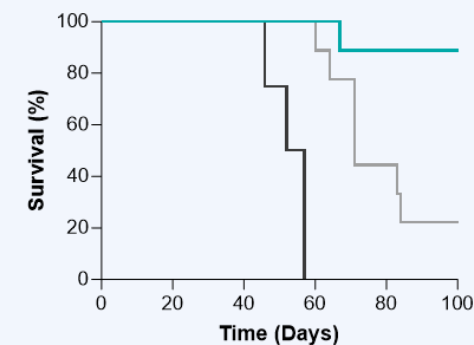
A-549 NY-ESO-1+ tumors in immunodeficient mice; NY-ESO-1 TCR T cells on day 18, MiNK-215 on day 25

● Untreated ● Host T cells only ● MiNK-215 + host T cells

Lung tumor burden



Survival



Tumor burden stayed near baseline through day 87; about 90% of mice survived to day 100, vs about 20% with T cells alone and none untreated

Source: Boi S, et al. ASGCT 2023, Poster 1488. Preclinical; not predictive of clinical benefit.

PIPELINE

A Focused Pipeline of Native and Engineered iNKT Cell Therapies

Product	Target / Approach	Indication	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3
agenT-797	Native iNKT	Severe acute lung injury / ARDS (viral & bacterial)					
agenT-797	Native iNKT	Graft-versus-host disease (GvHD)					
agenT-797	Native iNKT	Gastric cancer (2L) + SoC ± BOT/BAL					
agenT-797	Native iNKT	iNKts+ CD3 engagers					
MiNK-215	Engineered FAP-CAR · IL-15	Solid tumors and fibrotic / inflammatory lung disease (IPF)					
PRAME-TCR	Engineered TCR	Solid tumors (pediatric focus, with C-FURTHER)					
BCMA-CAR	Engineered CAR · IL-15	Myeloma and autoimmune disease					
pMLL-B07 TCR	Engineered TCR	Acute myeloid leukemia					

Bar length indicates the most advanced stage reached for that indication / program.

NCT04582201; NCT06251973; NCT05108623

MiNK THERAPEUTICS · NASDAQ: INKT

One immune cell. Cancer. Lung failure. Inflammation.

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APPENDIX

Supporting Data & References

Reference tables, study identifiers, and citations for data presented in this deck

Human and Mouse Data Link iNKT Cells to Infection Control

Three independent lines of evidence point the same way; none alone establishes clinical benefit

HUMAN · ARDS

Patients with viral ARDS have fewer, less functional iNKT cells

- Circulating iNKT counts significantly lower than in healthy controls
- Remaining iNKT cells proliferate significantly less

Evidence level: *observational; association, not cause*

Park KJ, et al. Front Immunol. 2024

HUMAN · ON TREATMENT

Infection cleared as inflammation fell after agentT-797

- BAL Pseudomonas cleared; TNF α , IL-1 β and neutrophils fell in step
- Phase 1/2: secondary bacterial pneumonia in 15%; published rates up to 84% (cross-study)

Evidence level: *uncontrolled cases and single-arm study*

MiNK cases, ASGCT 2026; Hammond TC, Nat Commun. 2024

PRECLINICAL

Removing iNKT cells worsens infection in mice; restoring them helps

- iNKT-deficient mice: higher *S. pneumoniae* burden, worse survival after influenza
- iNKT transfer protected mice from lethal influenza

Evidence level: *mouse models; plausibility, not efficacy*

Barthelemy A, Mucosal Immunol. 2017; De Santo C, J Clin Invest. 2008

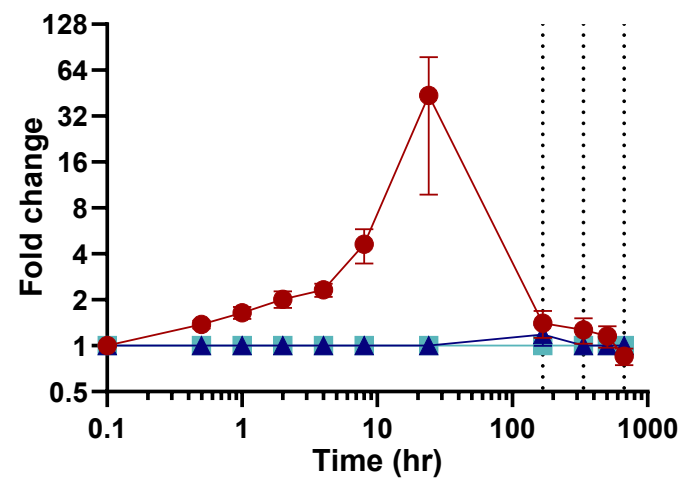
Taken together: iNKT cells are reduced in ARDS, their absence worsens infection in mice, and restoring them improves outcomes in mice. That supports testing agentT-797 as replacement of a missing cell population rather than another anti-inflammatory. Whether it improves patient survival is the question the randomized Phase 2b is designed to answer.

Preclinical findings support biological plausibility and do not predict clinical benefit. Human findings in ARDS are observational associations. The on-treatment pathogen-clearance observation is drawn from an uncontrolled case series. Comparisons with published rates are cross-study and not head-to-head.

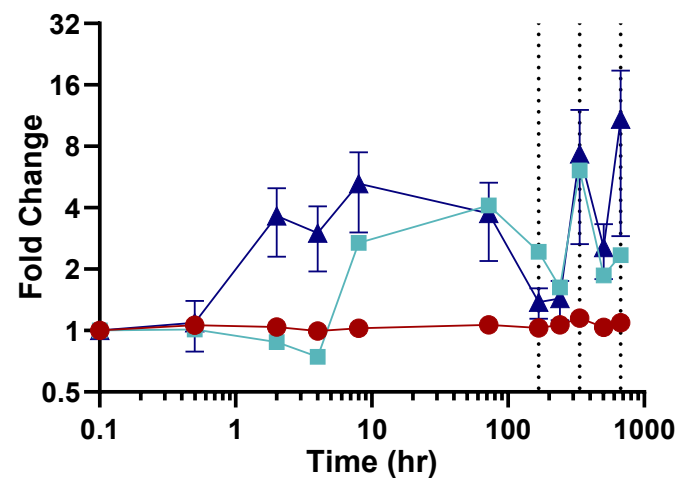
The Same agentT-797 Batch Switched On Th1 in Cancer and Th2 in ARDS

ASGCT 2026: Cytokine Responses in 34 Solid-Tumor and 20 ARDS Patients Given Unmodified iNKT Cells

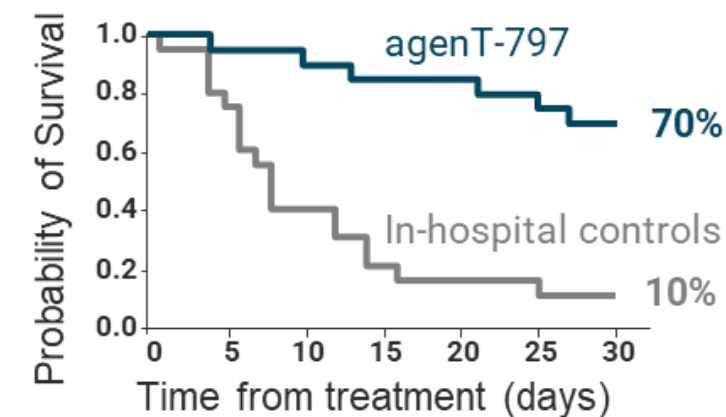
Solid tumors, n=34 · cytokine fold change



ARDS, n=20 · cytokine fold change



ARDS survival · agentT-797 vs in-hospital controls



● IFN-γ (Th1) ▲ IL-4 (Th2) ■ IL-13 (Th2) Dotted lines: weeks 1, 2 and 4

Same cells, opposite programs

The disease setting, not genetic engineering, set the response; it held across donors and batches.

Activity in both diseases

Complete remission of a metastatic germ cell tumor with anti-PD-1; 30-day ARDS survival 70% vs 10% for controls.

No cytokine release syndrome

Immune activation without uncontrolled CRS. The data support two randomized Phase 2 trials.

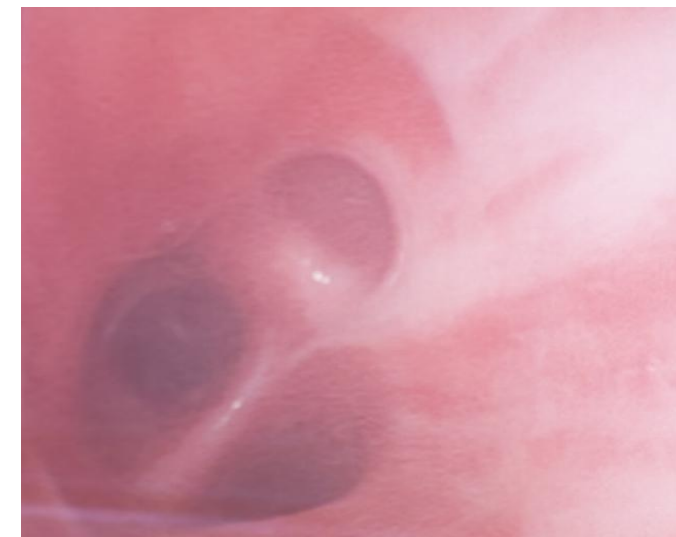
Source: Purbhoo MA, Hammond TC, et al. ASGCT 2026, Poster 3371; Hammond TC, et al. Nat Commun. 2024; Garmez B, et al. Oncogene. 2025. Cancer: NCT05108623; ARDS: NCT04582201. ARDS survival is compared with in-hospital controls, not a randomized arm; cytokine results are group means.

DEEP SCIENCE — CASE: agentT-797 + IL-15 (N-803)

N-803 Then agentT-797 in Refractory Fungal Lung Infection

ATS 2026 Case: Coccidioides and Pseudomonas Pneumonia With Severe ARDS, Treated Under EUA After Three Weeks of Failed Therapy

4/24	5/7	5/11	5/15	5/17	5/18	5/20	5/21	5/22-24	5/25
HD1	HD14	HD18	HD 22, TD1 N-803	HD25, TD3 agentT-797	HD26, TD4	HD27, TD6	HD26, TD7	HD 29, TD8-10	HD31, TD11
Admitted with Afib with RVR, multi-lobar pneumonia and acute hypoxemic resp failure BAL = + C. Immitis	Worsening despite escalation to fluconazole, steroids and meropenem	Reintubated, HD cath placed, Chest Tube placed for post thora PTX	600 mcg SQ N-803 (IL-15 super agonist)	Extubated am, dosed with 1 x 10 ⁹ iNKts mid-day	Reintubated No fevers; ferritin declining; BP stable. BNP 22,000, MR worse on echo FOB with pink airways	Worsening infiltrates, no fevers, neuro changes fevers or other signs of CRS	Attempted extubation but failed due to secretions weak cough	Cardiac cath and swan placed LVEF 65%, mod MR	Tolerating extubation until early am, reintubated Swan c/w cardiogenic shock PAM > 40 mmHg, PCWP > 20 mmHg CI < 1 (L/min/m ²)
Ampho B, Azithro CTX Solu med	Added Fluconazole Increased Solu med	Added meropenem P.Aeru > 10 ⁵ CFU + cocci	Light rash at site, BP stable, no AEs P.Aeru 10 ⁴	No fevers or neuro changes, BP stable No AEs day	FOB Pink Airways, no purulent secretions P.Aeru 10 ² No Fungi Mod to sev MR, +SLED	BNP elevated, cont SLED Toci x 2 abundance of caution, increased steroids to 1 mg/kg IV +SLED	CXR with presumed acute cardiogenic edema P.Aeru < 1000 CFU No fungi +SLED	Trach discussed family reluctant and requested additional trial of extubation, done 5/24	Bedside POCUS with severe MR, Afib then VF/VT Family elected to pursue comfort



Bronchoscopy, treatment day 4: pink airways, no purulence

Infection cleared

Pseudomonas fell from >10⁵ to <10³ CFU and BAL showed no fungi by treatment day 7; airways pink with no purulence on day 4.

Two-step immune response

N-803 drove a Th1 response (IFN-γ, IL-8). agentT-797 then raised IL-10 and IL-1RA and lowered TNF-α, IL-1β and IL-18.

No cytokine release syndrome

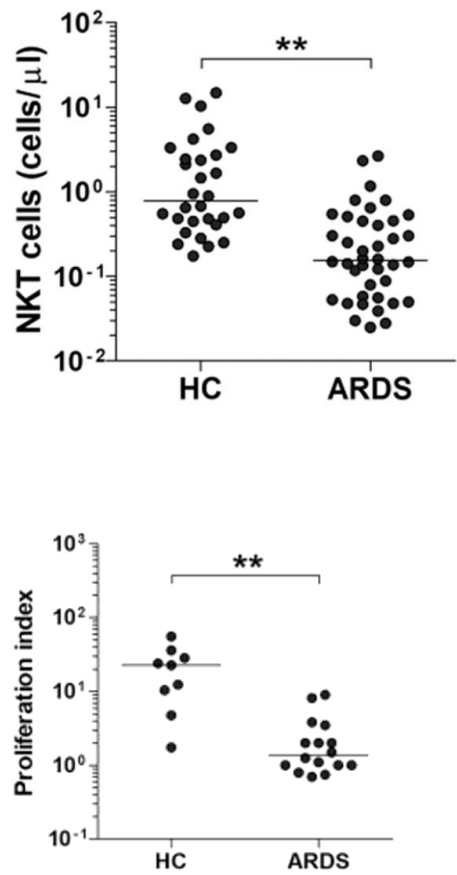
IL-6 peaked transiently at 3 hours; the profile never met CRS criteria. The case informed the randomized Phase 2 design.

Source: Hammond TC, et al. ATS 2026, Poster 103. Single patient (70-year-old man; COPD, CKD, atrial fibrillation) treated under EUA; no control. Outcome: the patient later developed severe mitral regurgitation and cardiogenic shock, and the family chose comfort care on hospital day 31. HD = hospital day; TD = treatment day; BAL = bronchoalveolar lavage.

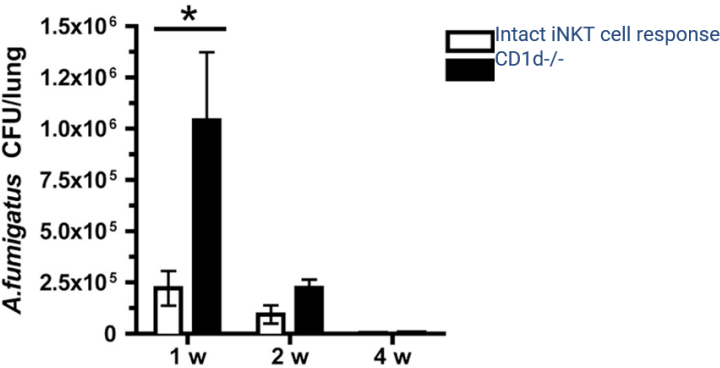
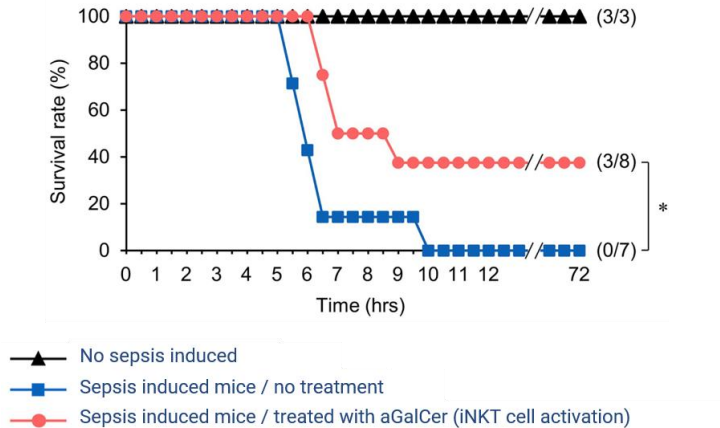
CLINICAL AND PRECLINICAL EVIDENCE - VIRAL ARDS, SEPSIS AND FUNGAL INFECTION

iNKT Loss Tracks With Worse Infection Outcomes in Patients and Mice

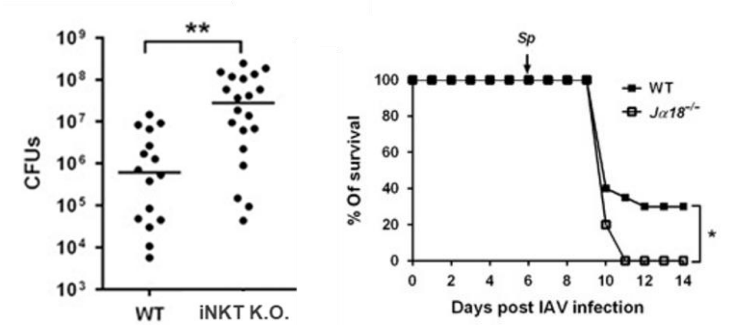
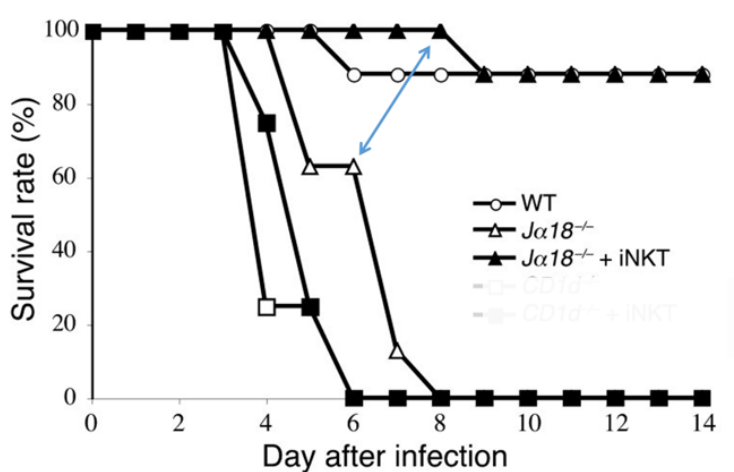
Patients with viral ARDS have fewer, impaired iNKT cells



iNKT activity raises sepsis survival and speeds fungal clearance in mice



iNKT cells protect mice from influenza and secondary S. pneumoniae



Source: Park KJ, et al. Front Immunol. 2024

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One immune cell.

Cancer. Lung failure. Inflammation.

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