Transformative Therapy for Neurodegenerative Diseases

Corporate Overview

January 2026

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Forward-Looking Statements

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Important factors that may affect actual results or outcomes include, among others, the risk that regulatory approvals for the Company's product development are not obtained, are delayed or are subject to unanticipated conditions that could adversely affect NKGen; the Company's ability to manage future growth; the Company's ability to manage clinical trials or studies, including any compassionate use programs and product pipeline; the risk associated with the use and reliance on the initial or preliminary results and data from the compassionate use programs and the ongoing Phase I/2a clinical trial; the dependence on the success of the Company's SNK natural killer cell technology platform; the complexity of numerous regulatory and legal requirements that the Company needs to comply with to operate its business; the concentrated ownership of New NKGen common stock among the Company's existing executive officers, directors and principal stockholders; the Company's ability to raise financing in the future; the Company's ability to service its operations and expenses and other liquidity needs and to address its ability to continue as a going concern; the ability to regain and maintain a listing of NKGen Common Stock on a national securities exchange such as the Nasdaq Global Market, and the potential liquidity and trading of the Company's securities, including while trading on the OTC Markets; changes in applicable laws or regulations; the Company's ability to successfully commercialize any product candidates that it successfully develops and that are approved by applicable regulatory authorities; the Company's expectations for the timing and results of data from clinical trials and regulatory approval applications; statements regarding the Company's anticipated timing and content of its filings with the U.S. Securities and Exchange Commission (the "SEC") and other public disclosures; the Company's business, operations and financial performance including: the Company's history of operating losses and expectations of significant expenses and continuing losses for the foreseeable future, the Company's ability to execute its business strategy and the Company's ability to develop and maintain its brand and reputation; the Company's ability to partner with other companies; the size of the addressable markets for the Company's product candidates; the Company's expectations regarding its ability to obtain and maintain intellectual property protection and not infringe on the rights of others; the outcome of any legal proceedings that may be instituted against the Company; the Company's ability to successfully integrate the business, operations, technologies, and personnel of NKGen Biotech Korea; the future performance of NKGen Biotech Korea's business; potential challenges in realizing expected synergies, cost savings, and other anticipated benefits from the acquisition of NKGen Biotech Korea, including the risk that such benefits may not be achieved on the expected timeline or at all; risks associated with managing a more complex and expanded corporate structure and operational footprint post-acquisition; unfavorable conditions in the Company's industry, the global economy or global supply chain, including financial and credit market fluctuations, international trade relations, pandemics, political turmoil, natural catastrophes, warfare (such as the war between Russia and Ukraine and the armed conflict in Israel and the Gaza Strip and Israel's declaration of war against Hamas), and terrorist attacks; and those factors discussed under the heading "Risk Factors" in the Company's Annual Report on Form 10-K filed on October 8, 2025, and the Company's other filings with the U.S. Securities and Exchange Commission, as available. New risk factors emerge from time to time, and it is not possible to predict all such risk factors, nor can NKGen assess the impact of all such risk factors on the business, or the extent to which any factor or combination of factors may cause actual results to differ materially from those contained in any forward-looking statements.

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Corporate Summary – Strategic Pivot from Oncology to Alzheimer's Disease due to Serendipitous Human Data in Advanced AD

Unprecedented improvement in advanced AD patient positions NKGen's **pivot to AD**

Troculeucel (SNK01), autologous "Super" NK cells in **early Phase 2a for moderate AD**

2017

NKGen Biotech established, focusing on NK cell therapy for **oncology**

2019

2023

NKGen merged with Graf Acquisition Corp. IV, listed on NASDAQ as **NKGN**; moved to OTC market in 2024

2025

SNK01 receives **Fast Track status** after showing early signs of clinical benefit: **company prioritizes neurodegeneration**

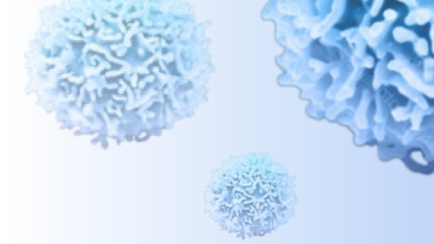
Investment-to-date

$145M

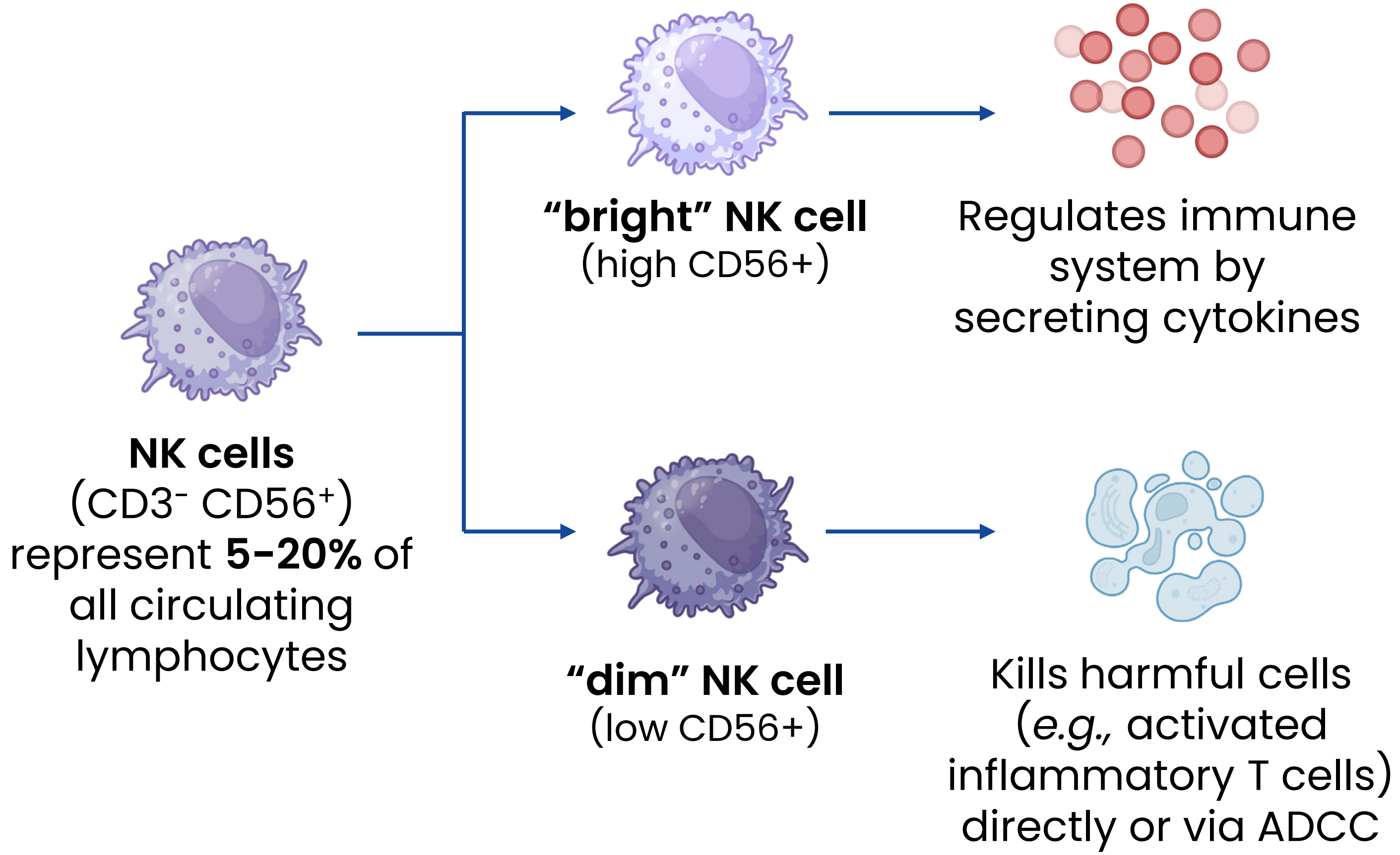
Funded legacy oncology programs, build out of ***two GMP Facilities*** *in South Korea and Orange County, California*

AD = Alzheimer's Disease

Different Populations of NK Cells Play Critical Roles in Modulating Inflammation



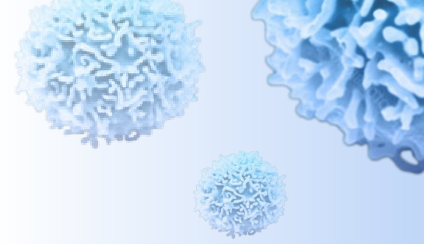
NK cells can recognize harmful foreign cells via 40+ receptors, **migrating** to target region to mediate immune defense



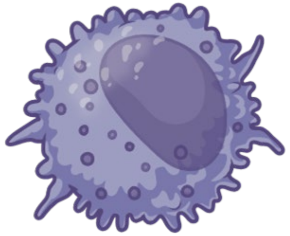
NK cell dysregulation typically occurs with immune senescence (aging) and chronic inflammation

- Quality and number of NK cells go down in aging and diseased population

Super NK Autologous Cells: Dual-Function NK Power to Maximize Immune Integrity



*We uniquely **combine the functional strengths** of both bright and dim NK cell subtypes into one*



"dim" NK cell
CD56^{dim} CD16^{+}
Cytotoxic activity

"Infantry"

The front-line defense: killing harmful cells but low proliferation

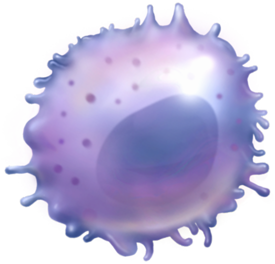
+

"bright" NK cell
CD56^{bright} CD16$^{low/-}$
Immunoregulation

"Scouts"

Intelligence: release cytokines to orchestrate immune response

→



Super NK cell (SNK™)
CD56^{bright} CD16^{+}
Cytotoxic + Immunoregulation

"Special Forces"

High expression of activating receptors* and elevated levels of the chemokine receptor CXCR3

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*NKG2D, DNAM-1, NKp30, NKp44, NKp46

A Serendipitous Discovery Guides NKGen's Pivot: Remarkable Symptom Improvement in Advanced AD Patient

Cultural context: boosting immunity with cell therapy

In Japan, physician-led, non-GMO cell therapies can be administered as medical procedures under ASRM[1]

Wellness clinics offer expansion and reinfusion of autologous immune cells for **anti-aging or longevity**

NKGen's GMP facility in Korea, NKMAX, is registered under ASRM[1] and **can export SNK01 to Japan** for use in regenerative cell therapies

SNK01 infusion shows notable improvement in family member AD symptoms

Compassionate case study: family member of an NKGen executive with cancer and advanced AD

In 2019, **infused with SNK01**, intended as immune "boost" for infection protection in a senior residence

Profound improvement in AD symptoms: **regained ability to get out of bed and speak short phrases**

[1] ASRM = the Act on the Safety of Regenerative Medicine (Japan), similar to FDA oversight in the USA

Compassionate Case Study:
Daniel, 38 Y.O. Korean Man with *PSEN1* Mutation + Advanced AD

Pre-Treatment Advanced AD with strikingly decreased parietal activity with moderate diffuse temporal, occipital and posterior fossa activity

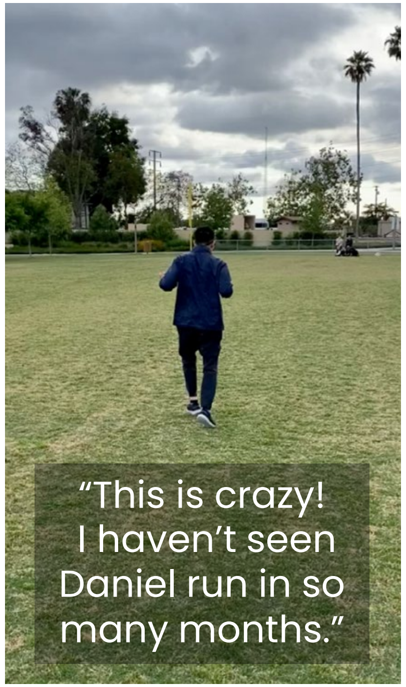
Treatment: 19 doses SNK01, 4~8B cells per dose (once every 2 weeks), over 9 months

Baseline:
- Can't talk
- Can't walk
- Can't get out of car unassisted
- Can't feed himself

After SNK:
- Can talk
- Can walk
- Can get out of the car
- Can eat by himself


10/13/20

1/5/21



5/19/21

Compassionate Case Study:
Janice, 70 Y.O. Woman with Advanced Alzheimer's

Improvement after resuming treatment even after 2y pause

Pre-treatment	2020	2021	2022	2023	2024

treatment — *paused treatment* — *treatment*

Pre-treatment

- Difficulty naming family members
- Can't remember her hometown
- Can't find things in a room
- Can't draw or copy simple drawing

Initial treatment:
7 doses SNK01, 2-8B cells/dose (1x every 3 or 4 weeks), over 6 months

- ➢ Significant improvement in cognition after 7 doses for 6 months at the end of initial treatment
- ➢ **The improvement sustained for 6 months** before showing sign of decline

No treatment for nearly 2 years

- ➢ Cognitive function and motility decreased
- ➢ Cannot make eye contact, less verbalization, memory decrease,

Slow movement, **needs assistance to stand up** and walk

Subsequent treatment:
23 doses, 4B cells/dose, over 1 yr and 6 months (1x every 3 weeks)

- ➢ Stand up, walk, run without assistance
- ➢ Natural eye contact
- ➢ Remembers her immediate family members' names

Janice **exercising** on step machine

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Initial treatment in Mexico, subsequent treatment in USA

These Three Advanced AD Patients Demonstrate Unprecedented Improvement in Symptoms

2021 — 2025

Prior to 2021, no disease-modifying therapies existed	**Paradigm shift:** Anti-amyloids (*e.g.*, lecanemab)	**Next-generation:** NKGen Biotech's SNK™ autologous cell therapy
Drugs only offered short-lived symptomatic management	**Modestly slows decline only in <u>mild AD</u>**, in studies performed over 18 mos.	*For the <u>first</u> time ever*, **can slow decline AND improve AD symptoms in <u>moderate-to-advanced AD</u>**
	Reduces amyloid plaque burden	*Improves neuroimmune health & reduces neuroinflammation*

Our SNK™ cell therapy has restored lost cognitive function in three advanced AD patients in compassionate use cases, which is NOT POSSIBLE with current anti-amyloid paradigm

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Advancing from Remarkable Anecdotal Data to Small Ph1 in Mexico

Serendipitous observation of improved symptoms in advanced AD (NKGen executive's family member)

Compassionate use in advanced AD patients also showing similar effects

Phase 1 study in Mexico, IRB, GCP (MX04)

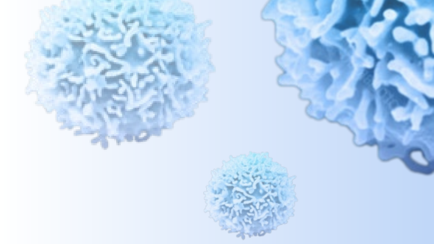
US IND Filing

How could we convince the FDA to allow a Phase 1/2a study in AD with SNK01?

- Needed to provide scientific justification
- Making autologous mouse SNK cells to conduct preclinical studies is not feasible

NKGen ran a small human dose escalation study in Mexico to generate data to support US IND filing

SNK01-MX04 Phase 1: Study Design to Support IND in US



Phase I Dose Escalation: 3+3 Design

Cohort 3 $4.0\text{x}10^9$ cells

Cohort 2 $2.0\text{x}10^9$ cells

Cohort 1 $1.0\text{x}10^9$ cells

Patients received **4 doses,** dosed **Q3W**

Objective: Establish safety in AD patients and capture biomarker data to provide mechanistic rationale to support IND filing

Single Site in MX

- Location: Angeles Tijuana Hospital, 10 patients (11 enrolled), all AD:
    - 5 mild, 3 moderate, and 2 severe AD

I/E Criteria

- Age 55 to 85 years old, Stage 3,4,5, and 6 AD*
- MRI and PET-FDG for a diagnosis of MCI or AD in the past six months
- No other medical or neurological diseases that could be a contributing cause of cognitive impairment
- No history of cardiovascular, psychiatric, seizure, endocrine, kidney, liver, infectious disorders
- No current treatment with anticoagulants

Endpoints

- **Primary:** safety – DLT, other AEs
- **Secondary:** preliminary efficacy – ADAS-Cog, MMSE, CDR-SB, ADCOMS
- **Biomarkers**: amyloid beta, pTau, tTau, GFAP

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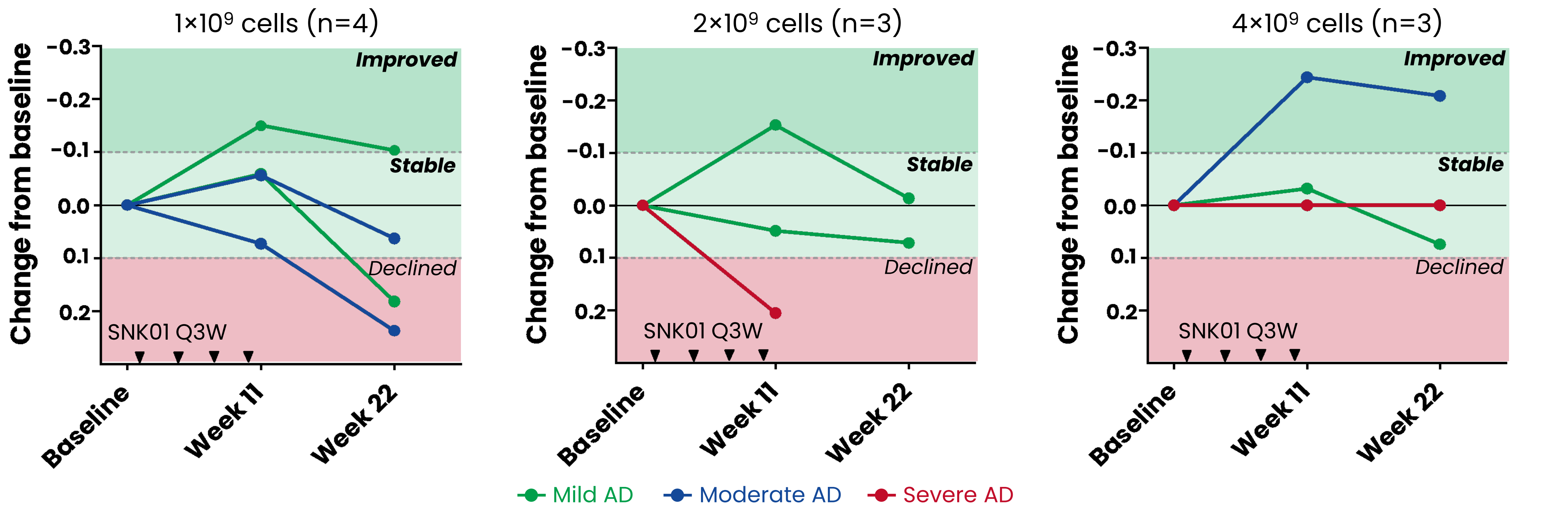
*Stages of AD defined by the Global Deterioration Scale (GDS)

SNK01-MX04 Phase 1:
Efficacy in Dose Escalation Study by Dose

ADCOMS



At Week 11, **90%** of participants showed either stable or improved composite ADCOMS scores (±0.1)[1] after only 4 doses, including a remarkable example of **moderate AD patient improving and a severe AD patient stabilizing** (with the highest dose) – a trend towards dose response was observed.

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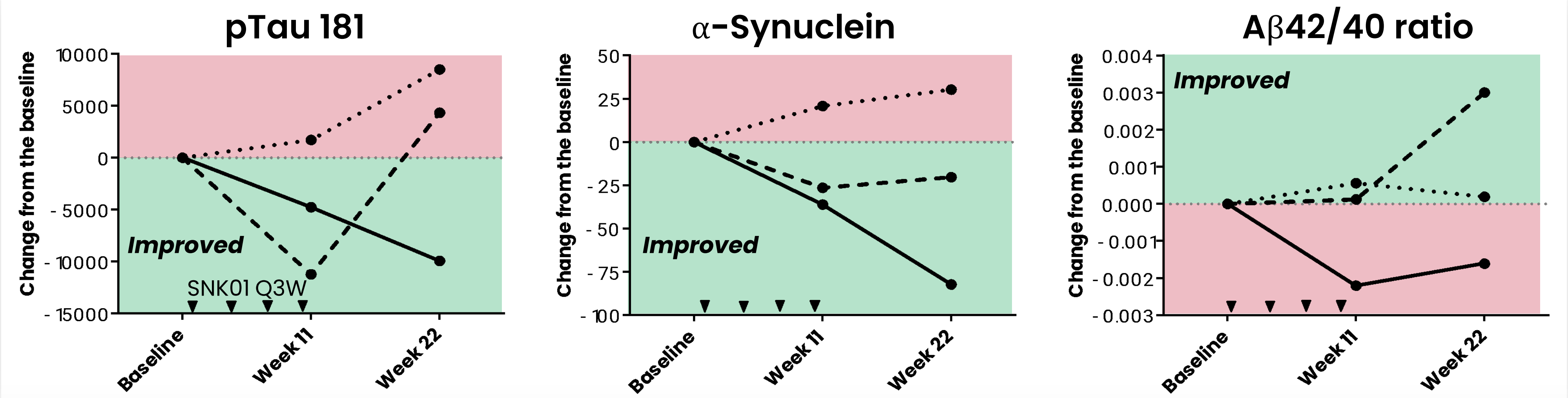
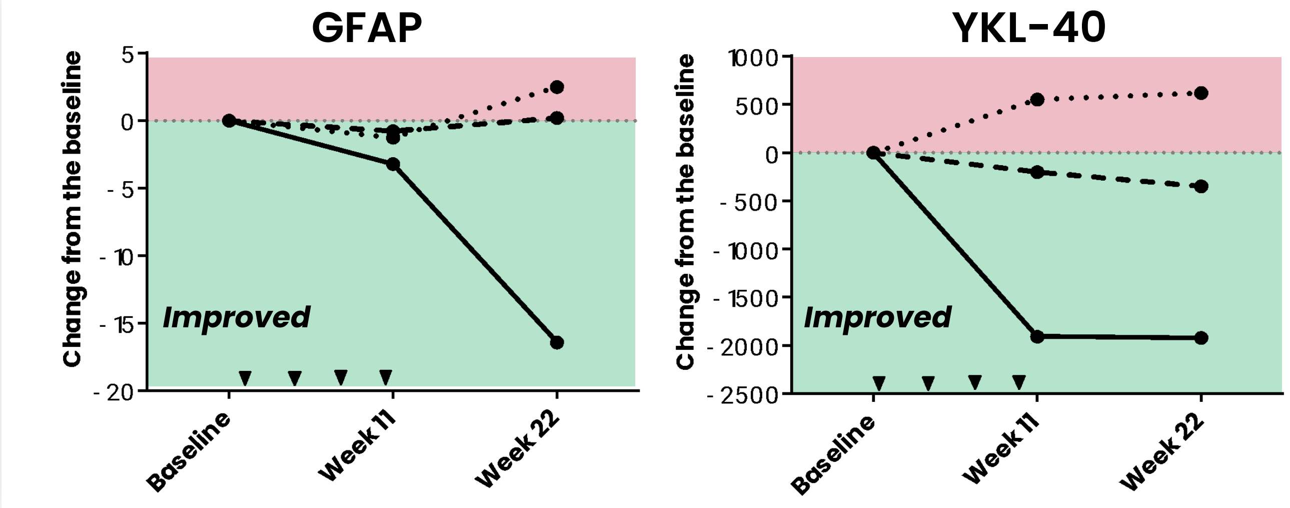
[1] ADCOMS = Alzheimer's Disease Composite Score. A change of 0.1 in the ADCOMS score is considered clinically meaningful and represents the **Minimal Clinically Important Difference** (**MCID**) with negative change from baseline representing improvement.

SNK01-MX04 Phase 1:
CSF Biomarkers Data

Abnormal proteins



Neuroinflammatory markers



1×10^9 cells *N=4*

2×10^9 cells *N=3*

4×10^9 cells *N=3*

CSF biomarker changes correlated with improvement in both abnormal proteins and neuroinflammatory markers

SNK01-AD01 Study:
Abbreviated Phase 1 in Moderate AD Patients in the US

- **MX04 Mexico dose ranging study supported FDA IND filing**
- NKGen wanted to move to higher dose for 1 year of dosing Q3W of 6B cell/dose and needed to bridge from "fresh" cells used in MX04 study to "frozen" for Phase 2
- **FDA required an abbreviated Phase 1 SNK01-AD01 study:**
  - Evaluate safety 3 weeks after first dose for each patient, in sequence
  - No safety issues after last patient reaches day 21 post-dose would trigger **randomized placebo controlled double blind Phase 2 study**
- Phase 1 patients could continue to be dosed at Q3W for one year (2 patients completed 1 year : 16-17 doses each, 1 patient completed 10 doses)

SNK01-AD01

Abbreviated Phase 1

- 3 Moderate AD patients
- Dose 6B cells, one dose (could continue at Q3W for 1 yr)

No DLT observed

SNK01-AD01

Randomized, Double Blind PBO controlled Phase 2a

- 30 Patients (20 SNK01, 10 Placebo)
- Dose 6B cells, Q3W for 1 yr
- Placebo drug, Q3W for 1 yr

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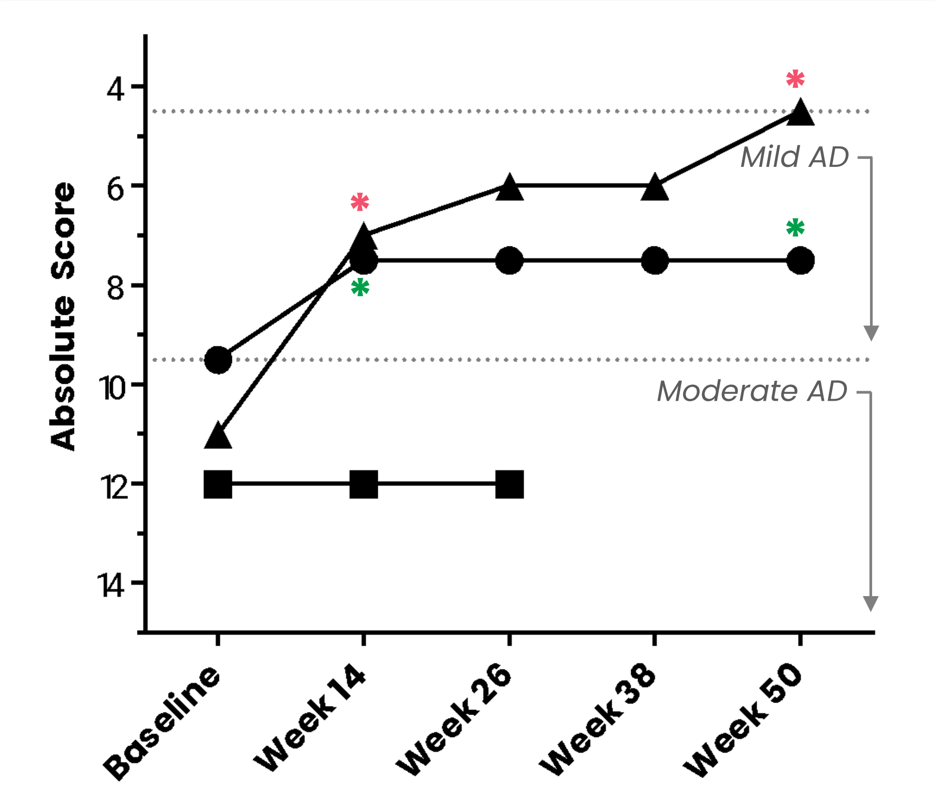
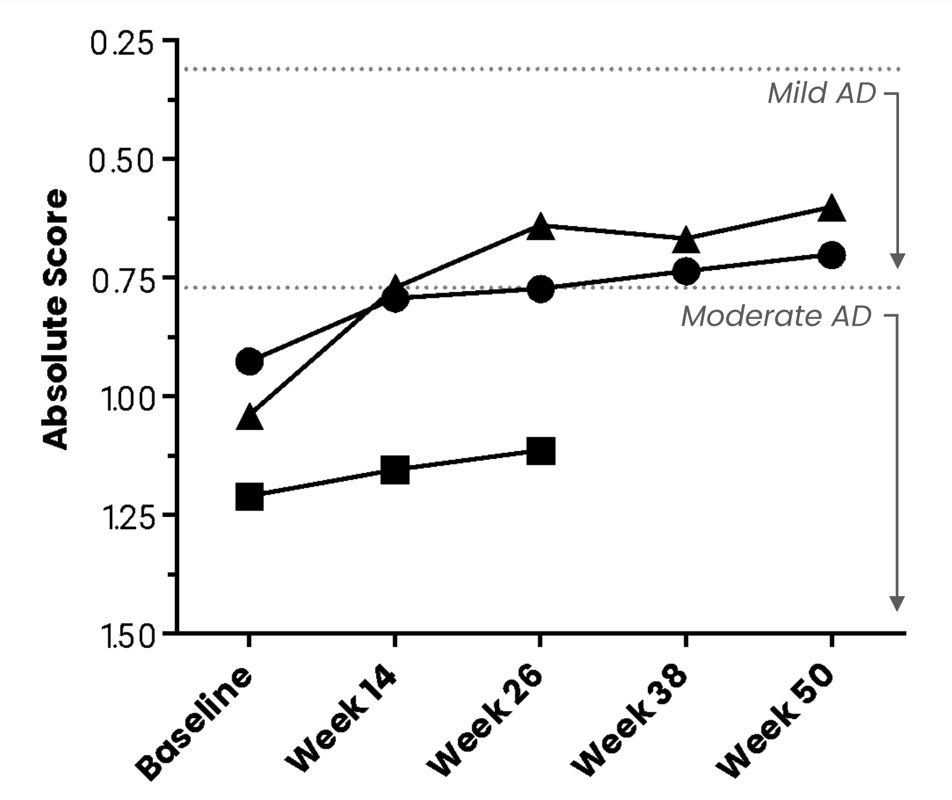
SNK01-AD01 Study Phase 1:
2/3 Moderate AD Patients Show Improvement in Cognition

SNK01 6x10^9 cells Q3W (17 doses); n = 3 moderate AD patients: **extraordinary improvement in <u>moderate AD</u>**
No drug-related adverse events.

CDR-SB



ADCOMS

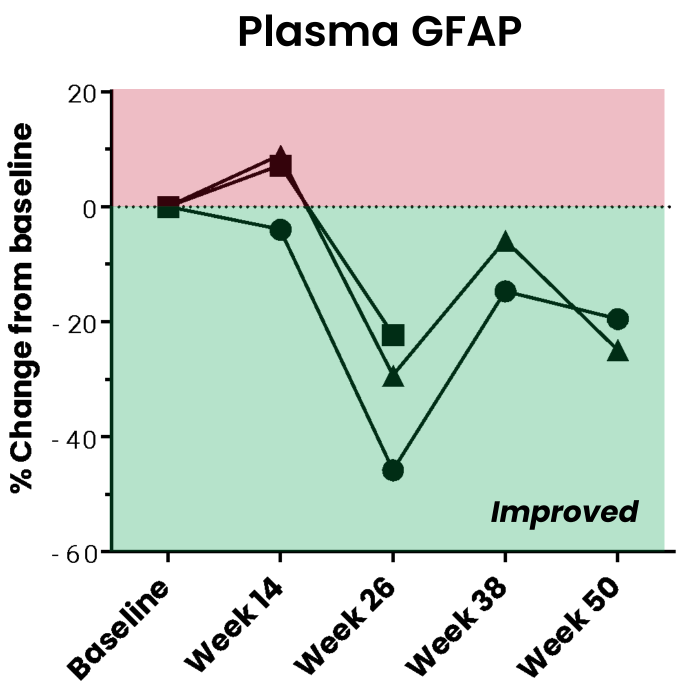


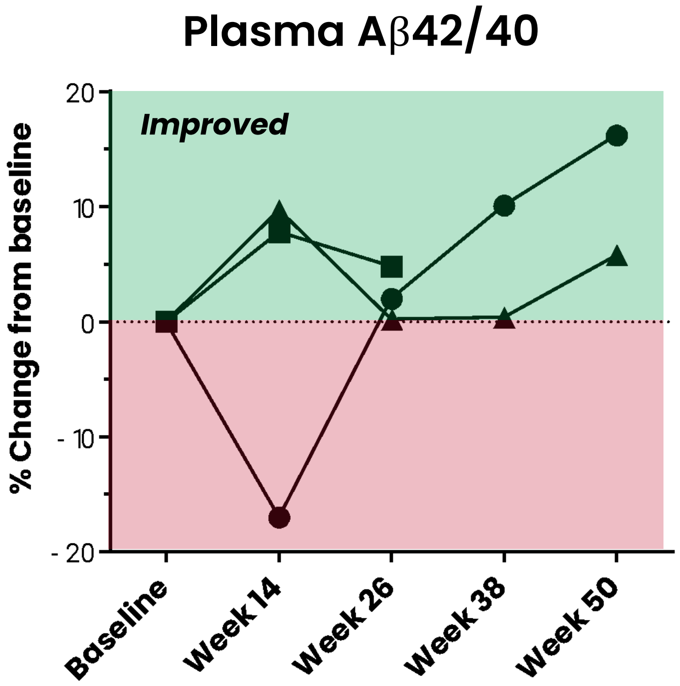
CDR-SB = Clinical Dementia Rating Scale Sum of Boxes; lower score is better. ADCOMS = Alzheimer's Disease Composite Score; lower score is better.
** *emphasizes baseline moderate AD patient improving to mild AD and continually improving or stabilizing at wk 14 and 50*

SNK01-AD01 Study Phase 1:
Preliminary Biomarker Data

SNK01 6x10^9 cells Q3W (17 doses); n = 3 moderate AD patients: **improvement in biomarkers observed as well**

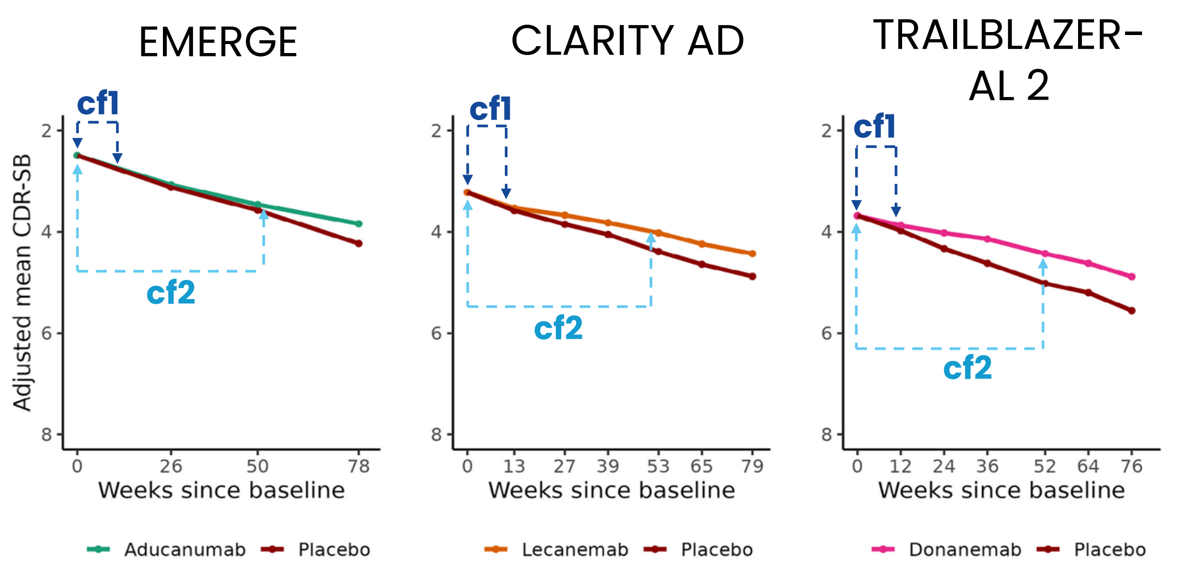
Biomarkers





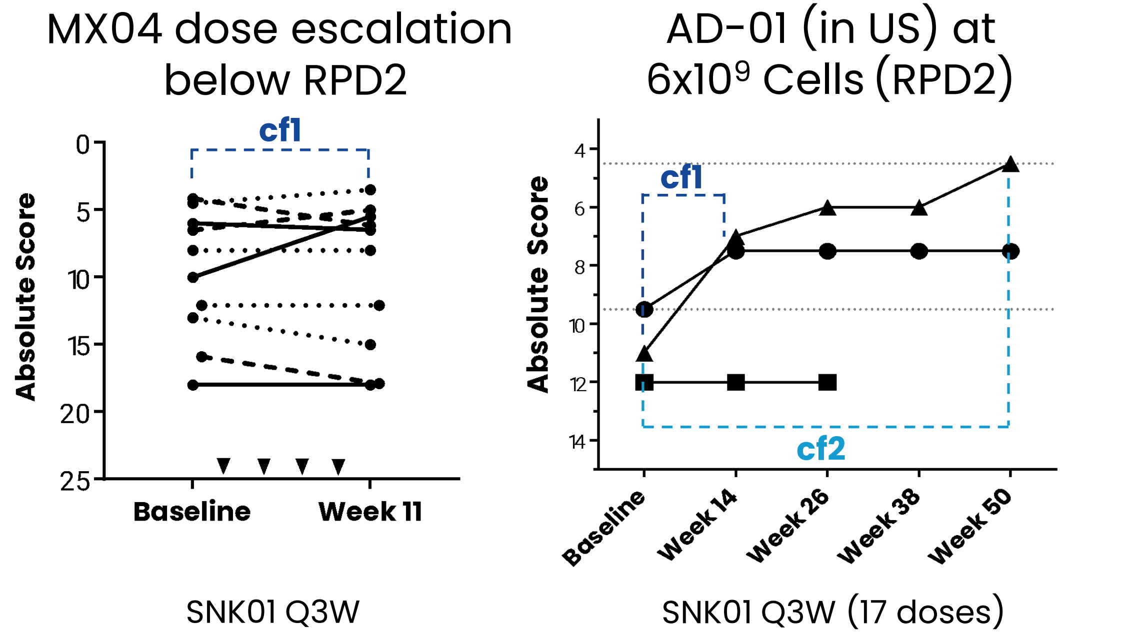
Anti-Aβ Drugs (Mild AD) Still Showed Decline, Most SNK01 Patients (Moderate AD) Stabilized or Improved

CDR-SB, Anti-Aβ Drugs



Raket, L. L. et al., Alzheimer's & Dementia (2024)

CDR-SB, SNK01



cf1 = compare 0 vs. 12 weeks; **cf2** = compare 0 vs. 52 weeks

Anti-Aβ: patients **declined**

SNK01: most patients **stable or improved**

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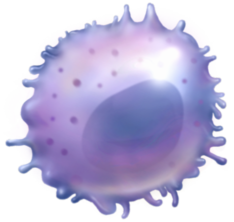
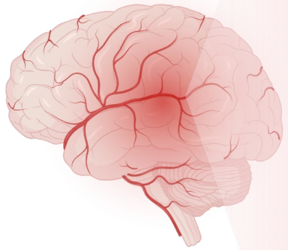
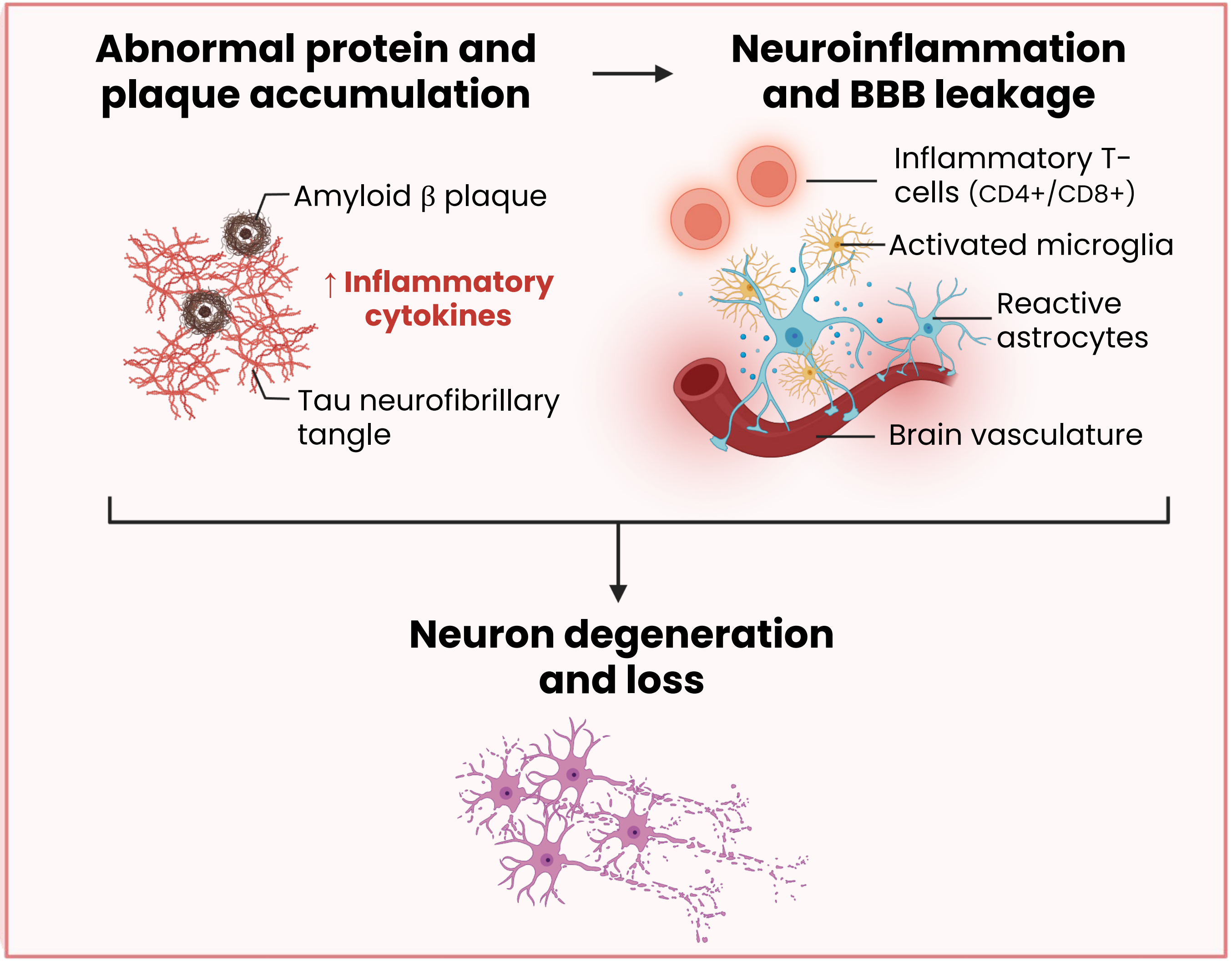
CDR-SB = Clinical Dementia Rating Scale Sum of Boxes; ; lower score is better

Therapeutic Mechanism-of-Action Hypothesis:
SNK Cells Reduce AD Pathogenesis via Anti-Neuroinflammation

Super NK cell (SNK™)
CD56^{bright} CD16^{+}







- Can enter brain via CXCR3 and leaky BBB
- Secretes anti-inflammatory cytokines
- Internalizes protein aggregates
- Kills inflammatory T-cells via NKG2D and DNAM-1

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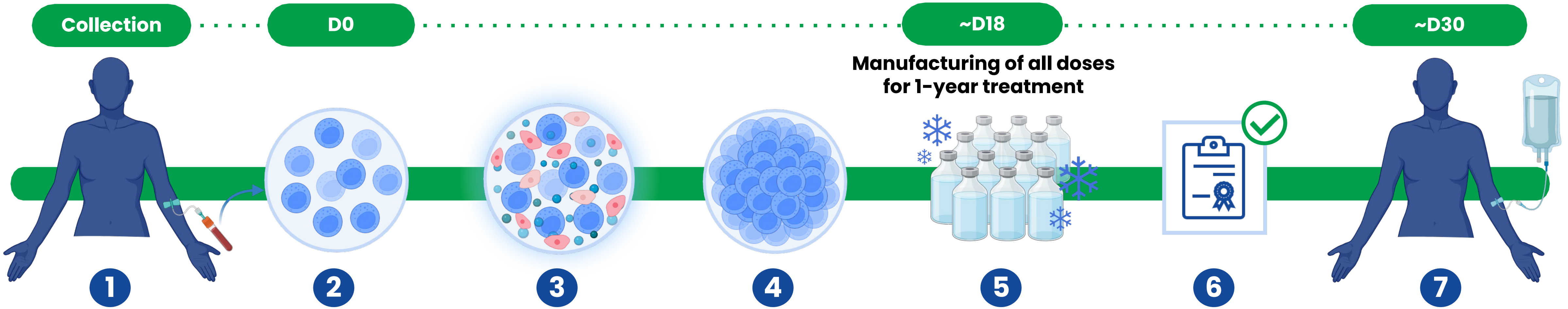
Autologous Next Generation Low-Cost Manufacturing
NKGen's "Off-the-Shelf" SNK^{TM} Cell Therapy



1	2	3	4	5	6	7
Collection	**PBMC Isolation /NK Cell Selection**	**Activation**	**Ex-vivo Expansion**	**Harvesting, Packaging, Cryo-preservation**	**Drug Product QC Testing**	**Administration**
Leukapheresis or Whole Blood Collection	$CD3^-CD56^+$ NK cell enrichment	Co-cultivation & reaction with $CD3^-CD56^+$ NK cells (2 irradiated feeder cells + 2 cytokines)	High purified SNK (≥95%) production for 17-18 days (~10,000 fold expansion)	Cryopreserving formulation technique for long term storage (~2 years)	Developed in-house testing method	Cold chain logistics

CD3-CD56+ NK cell
Feeder cell
Cytokine

Automation Process Implementation - Manufacturing Cost Reduction

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PBMC = peripheral blood mononuclear cells

NKGen's Strong, Multi-layered IP Strategy

Major/original technology
Core NK cell activation and expansion technology with feeder cells

Manufacturing technology
Manufacturing process of the product candidate and formulation for the drug product

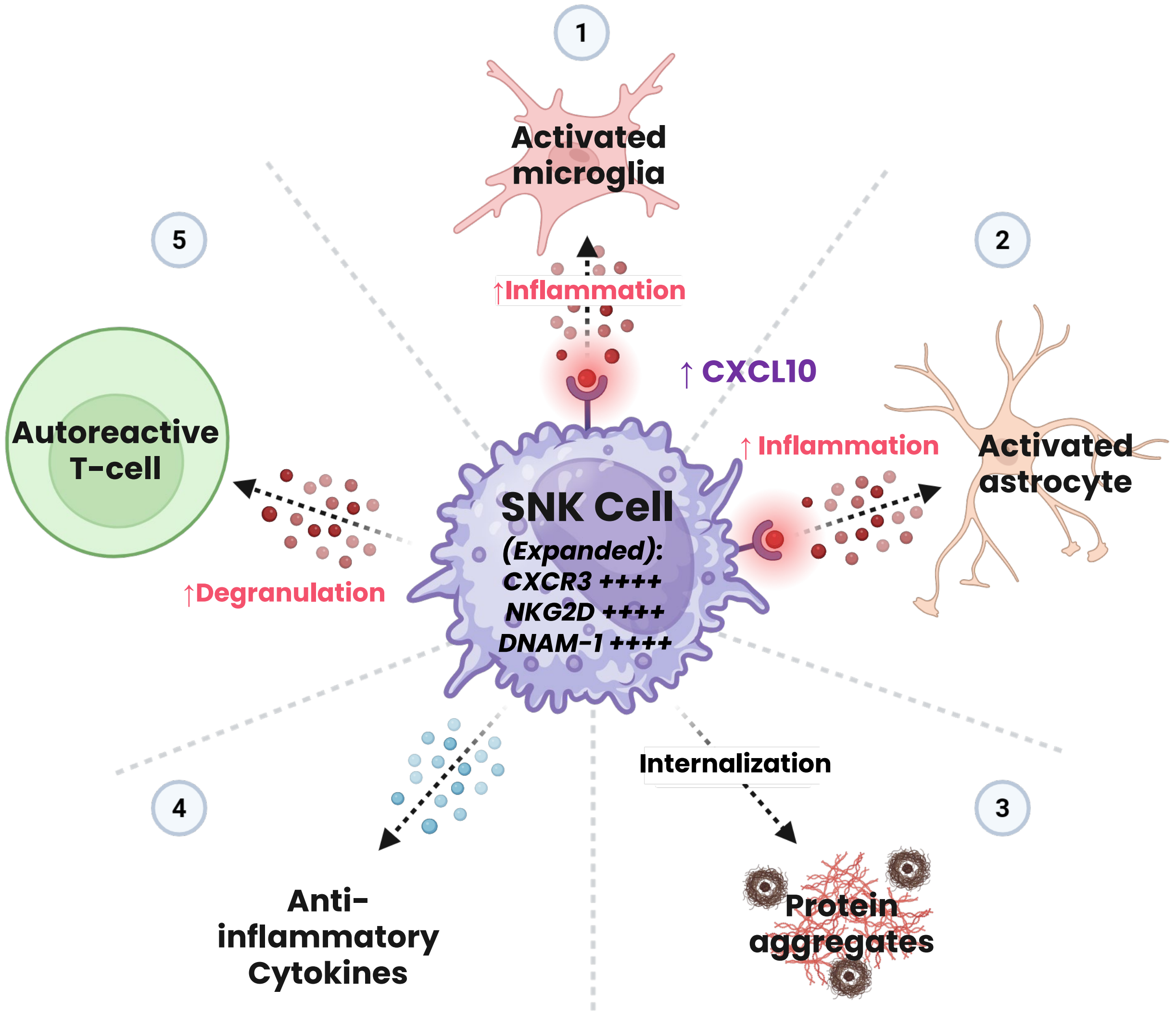
Therapeutic applications
Invention of the product candidate for **Parkinson's disease**, **Alzheimer's disease**, and **Autism**

Thank you!

Contact: investors@nkgenbiotech.com

Appendix

SNK01 MOA (Expanded)



Abnormal protein aggregation (Aβ / Tau /α-Synuclein), leading to inflammation

1, 2.) Disruption of immune homeostasis (astrocytes, microglia) chronically activated by proteinopathy

↑CXCL10

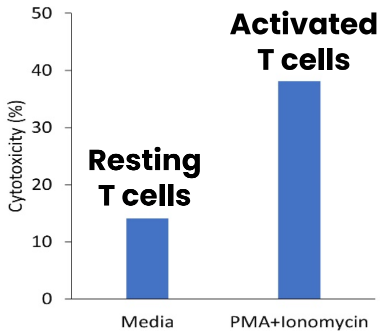
Chemotactic migration to brain via CXCR3

SNK cells can potentially…

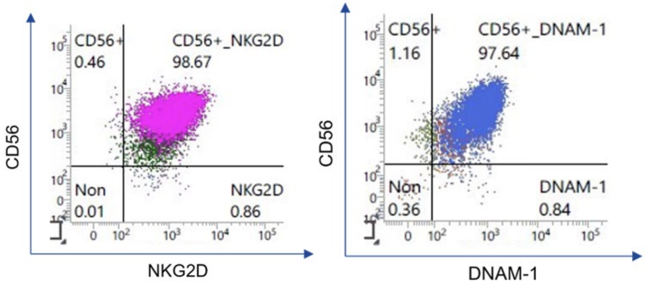
3.) Internalize protein aggregates
4.) Secrete anti-inflammatory cytokines
5.) Kill activated T-cells via innate immune mechanism
6.) Kill autoreactive T-cells via NKG2D and DNAM-1 signaling

Mechanistic Investigation of SNK01:
Functional Activity

SNK01 kill activated inflammatory T cells through engagement of highly expressed NKG2D & DNAM-1

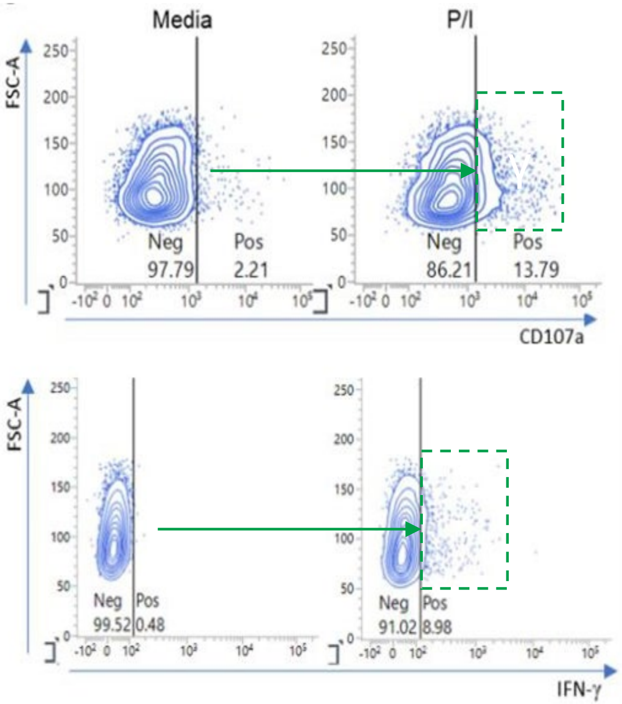


Increased SNK01 **cytotoxic killing activity** against activated T cells



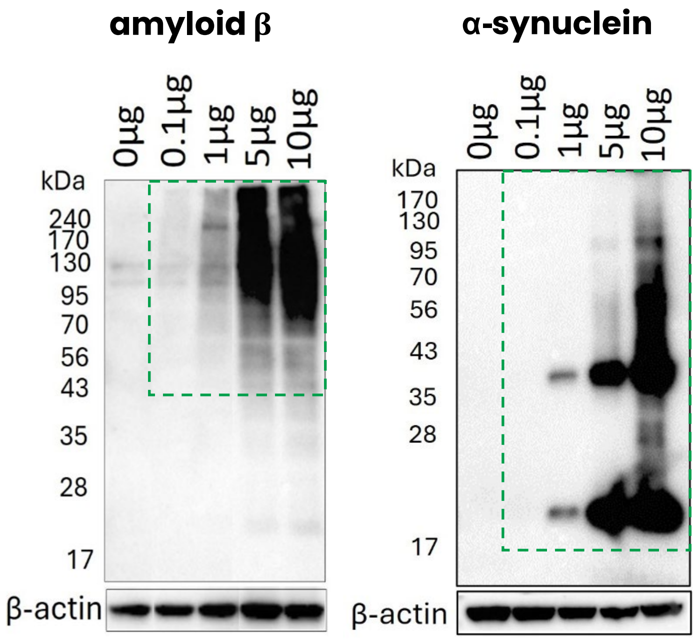
SNK01 has high expression of **NKG2D** + **DNAM-1**

SNK01 secrete anti-inflammatory cytokines



Increased degranulation activity (**CD107** expression) and **INF-γ production** by NK cells in response to activated T cells

SNK01 internalizes and degrade protein aggregates



SNK cells efficiently **internalize both amyloid β and α-synuclein** aggregates

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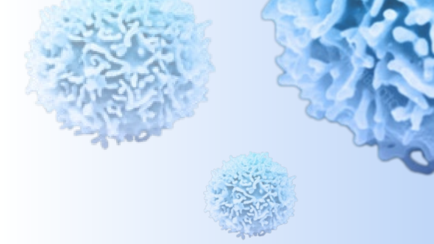
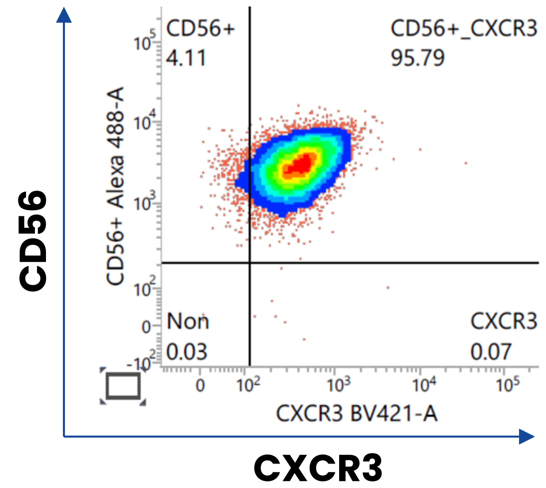
Mechanistic Investigation of SNK01:
Brain Migration

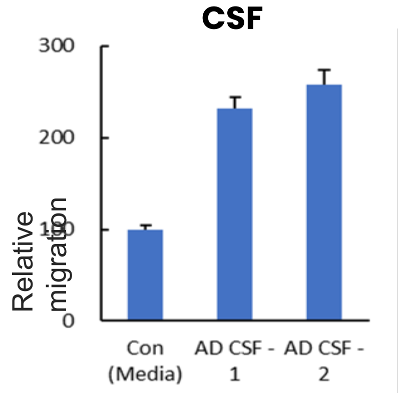


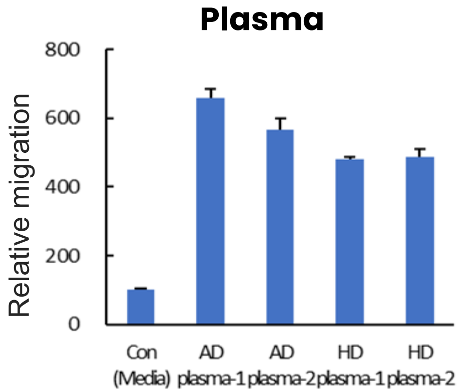
SNK01 expresses high level of CXCR3, also found in AD patients



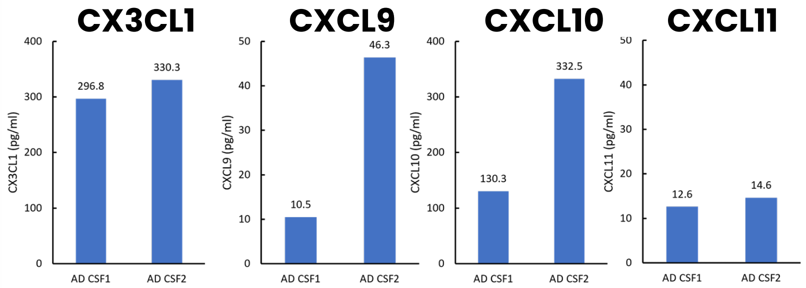
CXCR3 expression level of SNK01 from the AD patient, the same chemokine receptor as found on autoreactive T cells of AD patients

SNK01 migrates toward CSF and plasma of AD patients

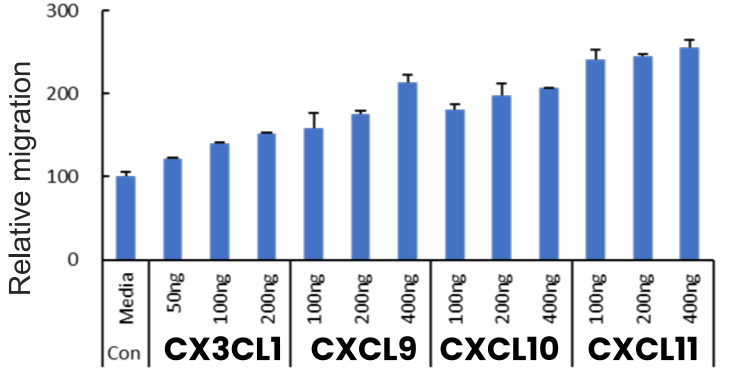




SNK01 has migratory potential toward chemokine ligands



Specific chemokines linked to neuroinflammation are present in the CSF of AD patients

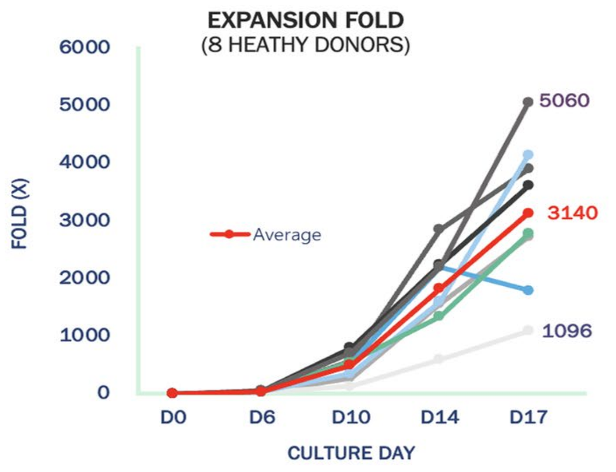


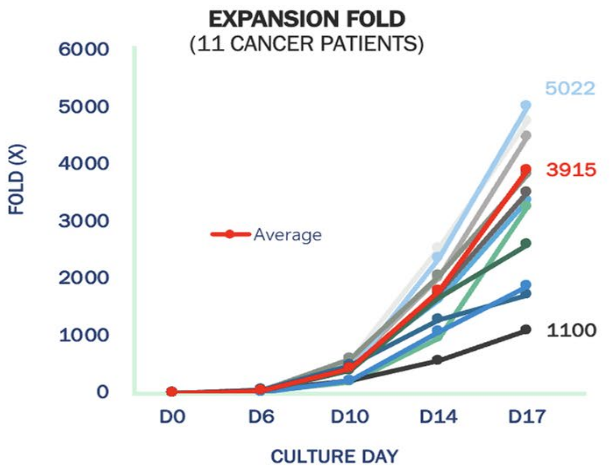
SNK01 migrates toward the chemokine ligands

NKGen's Value and IP Position is Driven by Manufacturing Process

NKGen's proprietary manufacturing process can **produce multiple doses of highly pure SNK cells** from a single collection of patient's whole blood (WB) or leukapheresis product (LP), even when derived **from patients that present with low NK cell counts**.

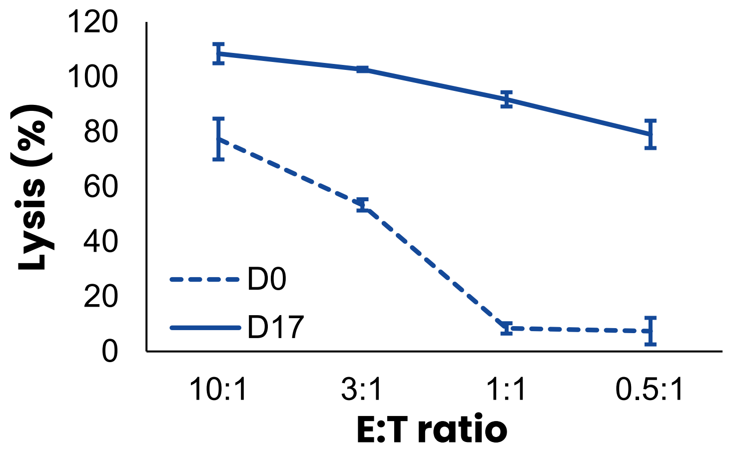
NKGen can expand NK cells from any donor





CD56+ NK cells, derived from healthy donors (n=8) and cancer patients (n=11), were expanded to up to ~5000-fold from Day 0 to Day 17

NKGen can increase NK cell killing potential



The lysis efficiency of CD56^{+} NK cells increased from Day 0 to Day 17 across all E:T ratios: enhanced cytotoxic activity

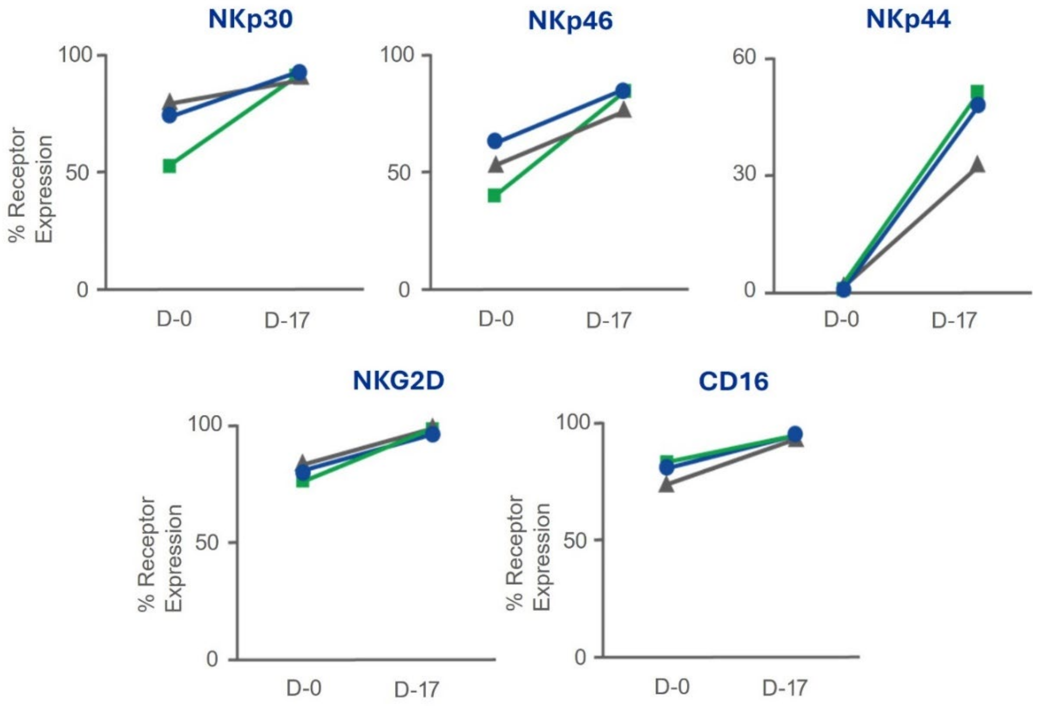
Superior Cell Expansion & Increased Cytotoxicity

NKGEN BIOTECH

Reference: in-house report; E:T ratio = effector cell (SNK) : target cell ratio

NKGen Can Reliably Produce Homogenous, Robust, Pure SNK Product

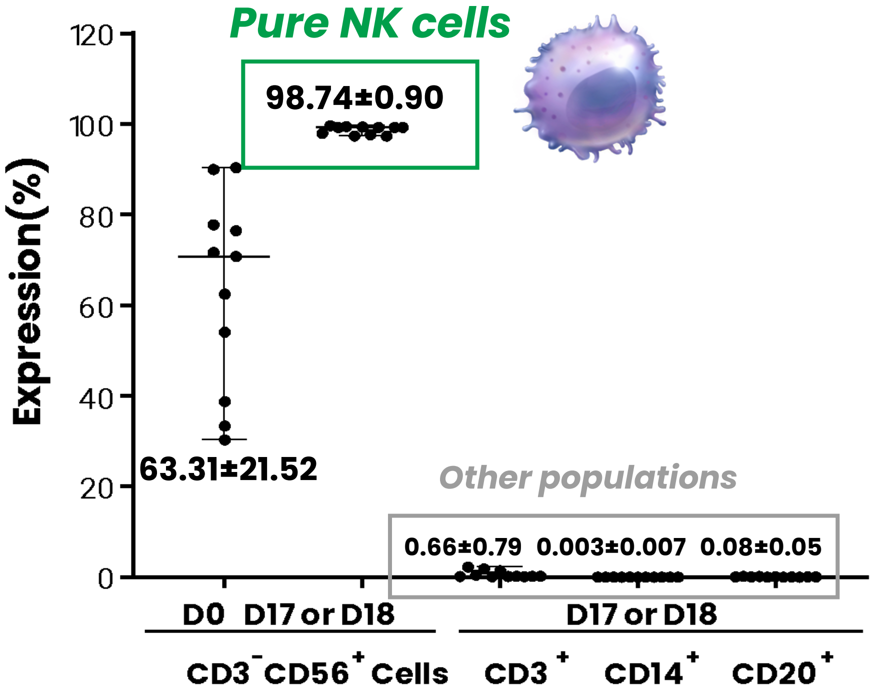
NKGen can increase activating receptor expression that are markers of NK cell effector Function



Elevated expression of activating receptors enhances NK cell cytotoxicity, degranulation, cytokine production (IFN-γ, TNF-α), and ADCC. NKG2D and NKp46 regulate neuroinflammation by eliminating autoimmune T cells and damaged neurons

C Rodriguez-Mogeda et al (2024), Z Ning et al (2023)

NKGen can produce high purity (≥95%) NK cell product

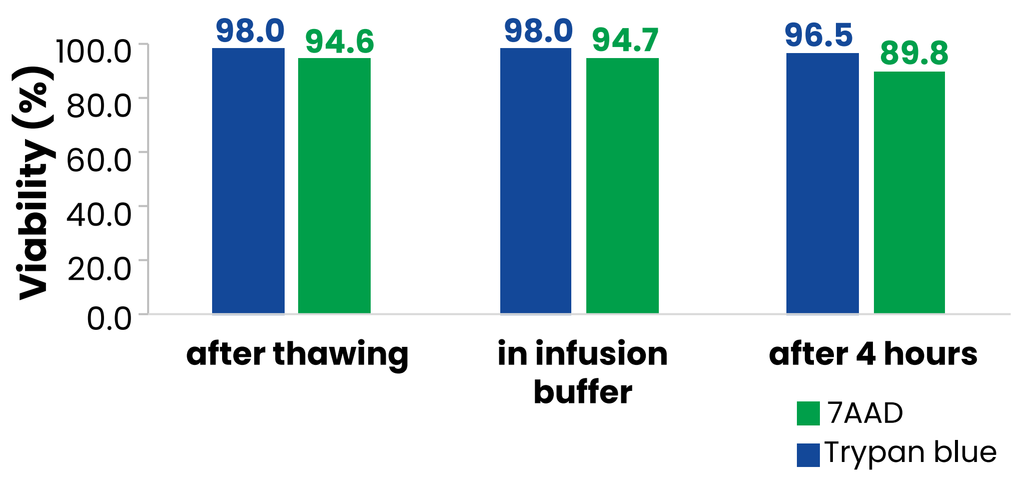


Highly pure NK cells (>95%) are generated from 11 AD patients

In-house report

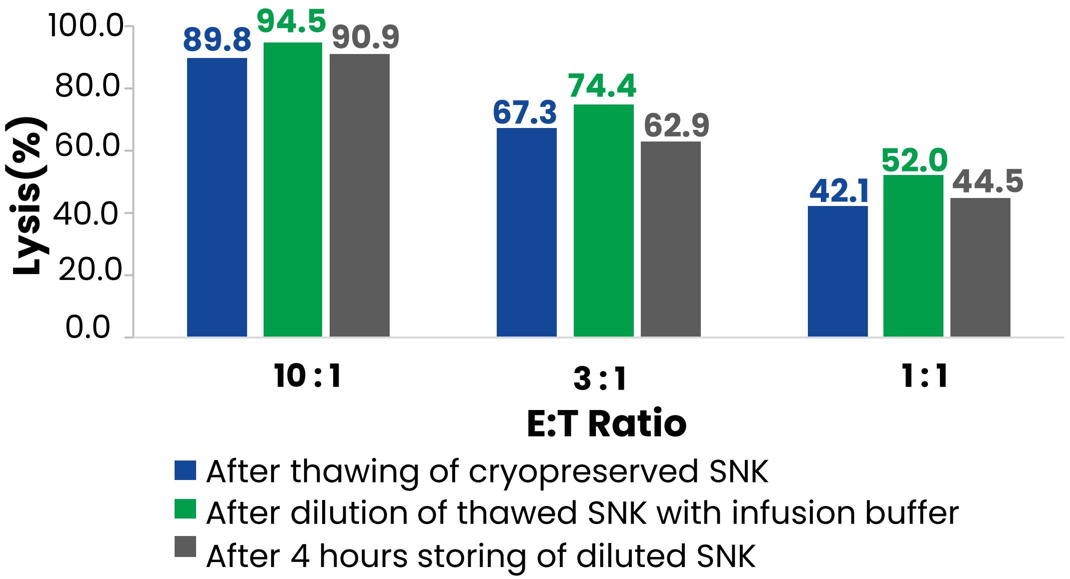
Preserved Cell Viability and Cytotoxic Function Following Cryopreservation

Cell Viability



*Cryopreserved SNK cell viability remained **≥90%** after thawing and up to 4 hours post-dilution, indicating excellent stability.*

Cytotoxicity Function



Cryopreserved SNK cell retained enhanced cytotoxic function upon thawing and up to 4 hours post-dilution.

Reference: in-house report; E:T ratio = effector (NK) : target cell ratio