Harnessing the Power of Naturally Occurring NK Cells to Fight Cancer & Autoimmune Disease

Investor Presentation

Forward Looking Statement

This presentation contains forward-looking statements that are based on the company ’s current expectations, assumptions, estimates and projections about the company and the pharmaceutical industry. The company makes no representations about the accuracy of such statements estimates or projections. Forward-looking statements are indicated by words such as: may, will, should, predict, continue, plan, expect, anticipate, estimate, intend, believe, could, goal objectives and similar expressions. Forward-looking statements may include, but are not limited to, statements concerning the company ’s anticipated performance, including revenue and profit expectations; development and implementation of collaborations; benefits provided to collaboration partners by our technology; business mix; revenues and growth in our partner base; market opportunities; competing technologies, industry conditions and trends; and regulatory developments. Actual results may differ materially from the anticipated results due to substantial risks and uncertainties related to the company and the biopharmaceutical industry in which the company operates.

Our Team

Deep Development Experience & Track Record of Execution

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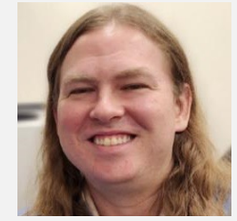

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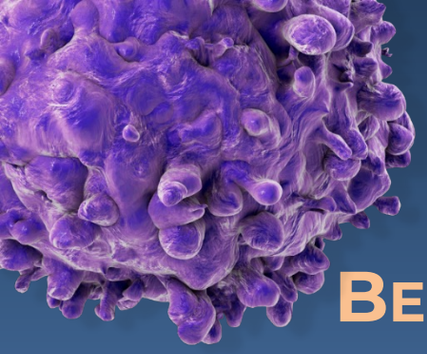
INDAPTA THERAPEUTICS



INDAPTA THERAPEUTICS

Best-in-Class on-demand NK Cell Therapy Platform for Cancer & Autoimmune Disease

Clearly differentiated from

- Conventional NK cells
- CAR-T approaches

Phase 1 trial in hematologic malignancies enrolling

- FastTrack designation
- 6 mo data 2H 2025

Robust manufacturing process & Strong IP position

Autoimmune Disease

- Two IND filings in 2024
- POC in MS & Autoimmune kidney diseases

g-NK Cells

Uniquely Positioned to Deliver Superior Responses in Cancer and Autoimmunity

Subset of NK cells that undergo epigenetic modifications in response to CMV exposure

- Arise in 25% of CMV exposed individuals
- Long lasting epigenetic modifications result in modulated gene expression and unique, stable phenotype
- Highly potent compared to conventional NK cells with multiple differentiating MOAs
  - Superior antibody-dependent cellular cytotoxicity (ADCC)
  - g-NK cells are negative for NK checkpoint NKG2A
  - Potent killing of HLA-E expressing cancer or autoimmune reactive cells via NKG2C
  - Primed by HCMV for potent killing of virally infected cells (irrespective of virus)
- Indapta has proprietary high-yield process to preferentially expand g-NK cells from CMV seropositive donors screened for increased g-NK number & function

MOA=Mechanism of Action

g-NK Cells

Highly Differentiated from Other Cell Therapy Approaches for Cancer & Autoimmune Disease

Property		G-NK	cNK	CAR-NK	Auto CAR-T	Allo CAR-T
Mechanism: **Cancer**	Targeting mechanism	mAb	mAb	CAR	CAR	CAR
	HLA-E targeting	YES	NO	NO	NO	NO
	Anti-viral (HPV*)	YES	LESS	NO	NO	NO
Mechanism: **Autoimmune**	B cell depletion	YES	YES	YES	YES	YES
	Killing *autoreactive* T & B cells	YES	NO	NO	NO	NO
	Anti-viral (EBV*)	YES	LESS	NO	NO	NO
Safety	Output treatment (low tox)	YES	YES	YES	NO	NO
	Vector malignancy risk	NONE	NONE	YES	YES	YES
Treatment	Off-the-shelf	YES	YES	YES	NO	YES
	Multiple cycles feasible	YES	YES	YES	DIFFICULT	DIFFICULT
	Targeting multiple Ags by combining with mAbs	SUPERIOR	YES	NO	NO	NO
	COGS	LOW-MOD	VARIABLE	MODERATE	HIGH	MODERATE

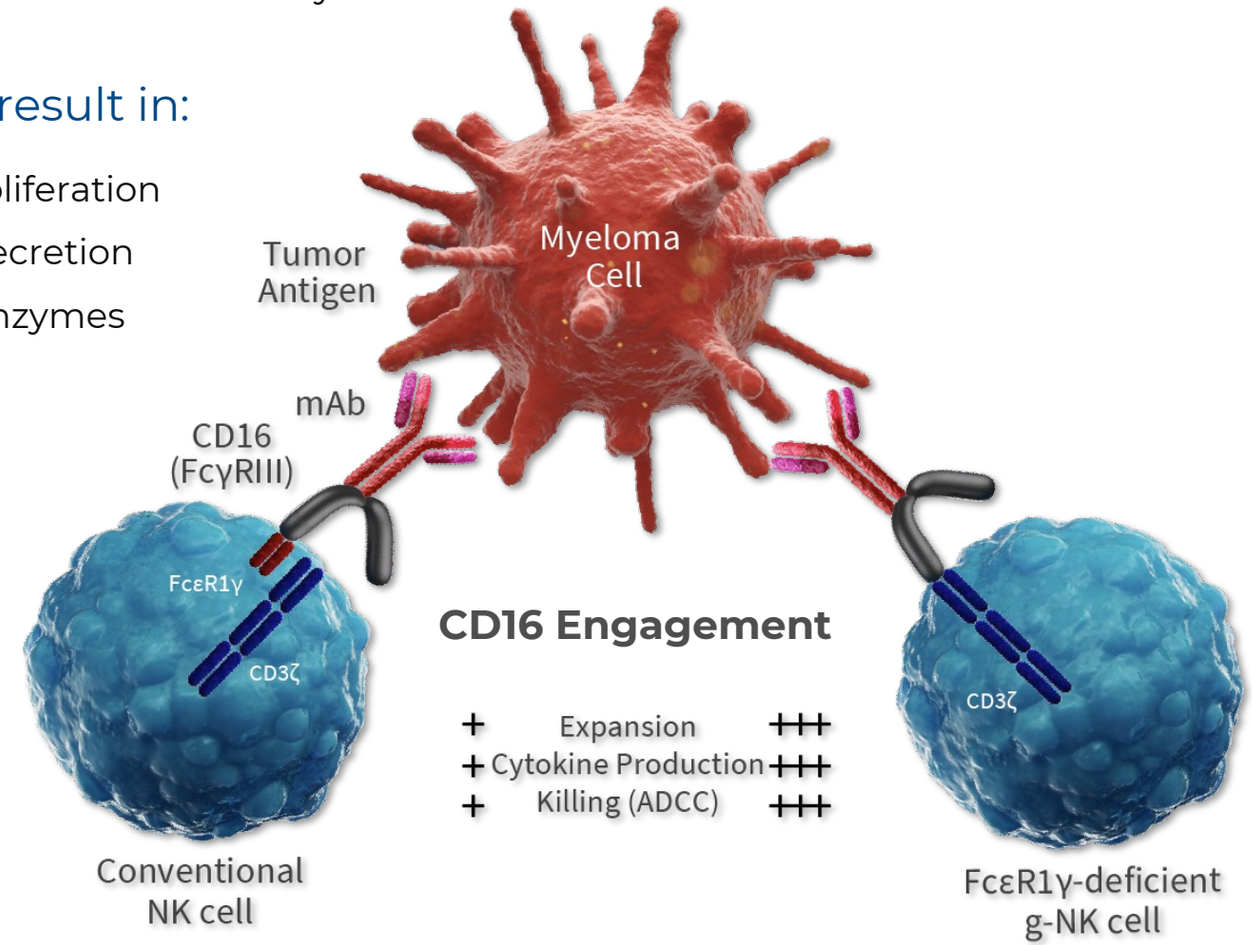
* Mechanism relevant example

G-NK Cells Have Robust ADCC Activity

Signaling exclusively through CD3ζ because FcεR1γ is downmodulated

Ideal to Combine with mAbs, ADCs, Innate Immune Engagers (IIE)

- Called ”G minus NK cell” or ‘g-NK’ because lack FceR1g[1,2]
  - When CD16 is engaged by Fc, all signaling goes through CD3z (3 ITAM motifs vs 1 ITAM for FceR1g)
  - In addition, g-NK are deficient in Syk
- These changes result in:
  - Stronger cell proliferation
  - More cytokine secretion
  - More cytolytic enzymes
  - Better ADCC



ADCC=antibody-dependent cytotoxicity
[1]*Bigley et al., Blood 2021* ; [2]*Dahlvang et al., 2023*

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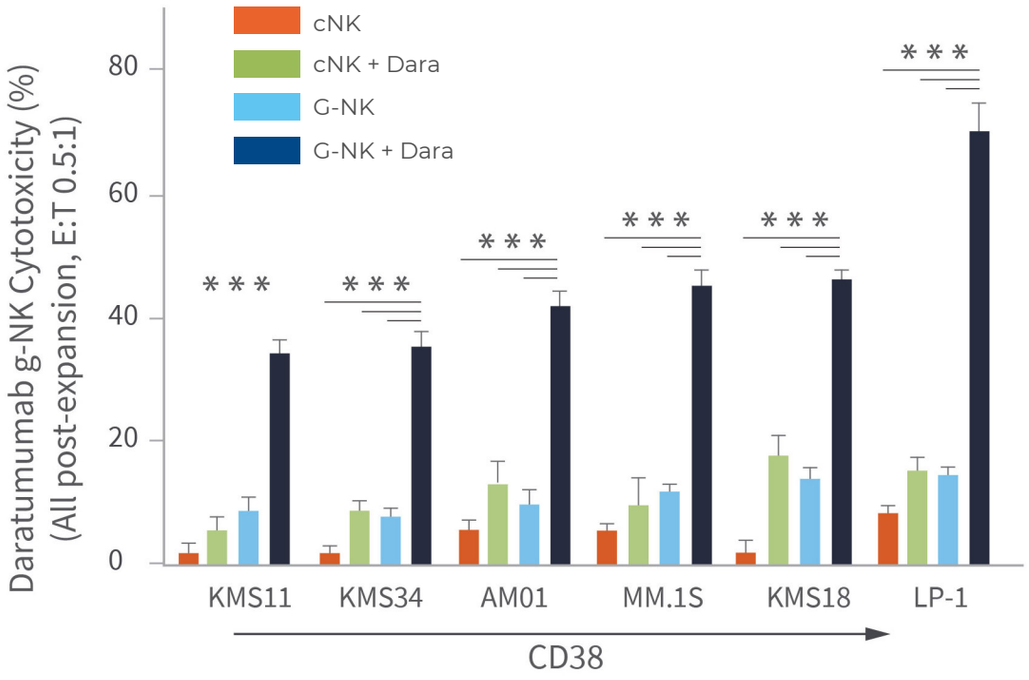
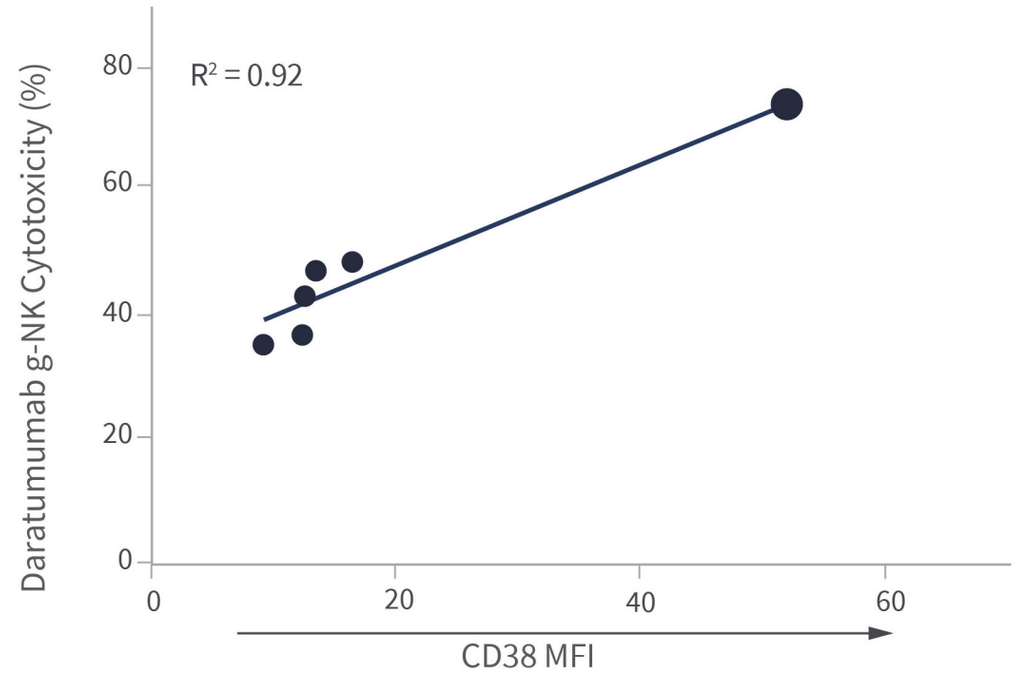
Expanded g-NK Cells Have Greater ADCC Than cNK

Highly Differentiated from Other Cell Therapy Approaches for Cancer & Autoimmune Disease



ADCC of g-NK (0.5:1 E:T) against 6 myeloma cell lines with increasing CD38 expression

Values are mean ± SE. *p<0.05, **p<0.01, and ***p<0.001 (one-way ANOVA with Bonferroni adjustment)



ADCC is proportionate to target antigen expression

g-NK ADCC is still markedly elevated against MM cell lines with dim expression of target antigen

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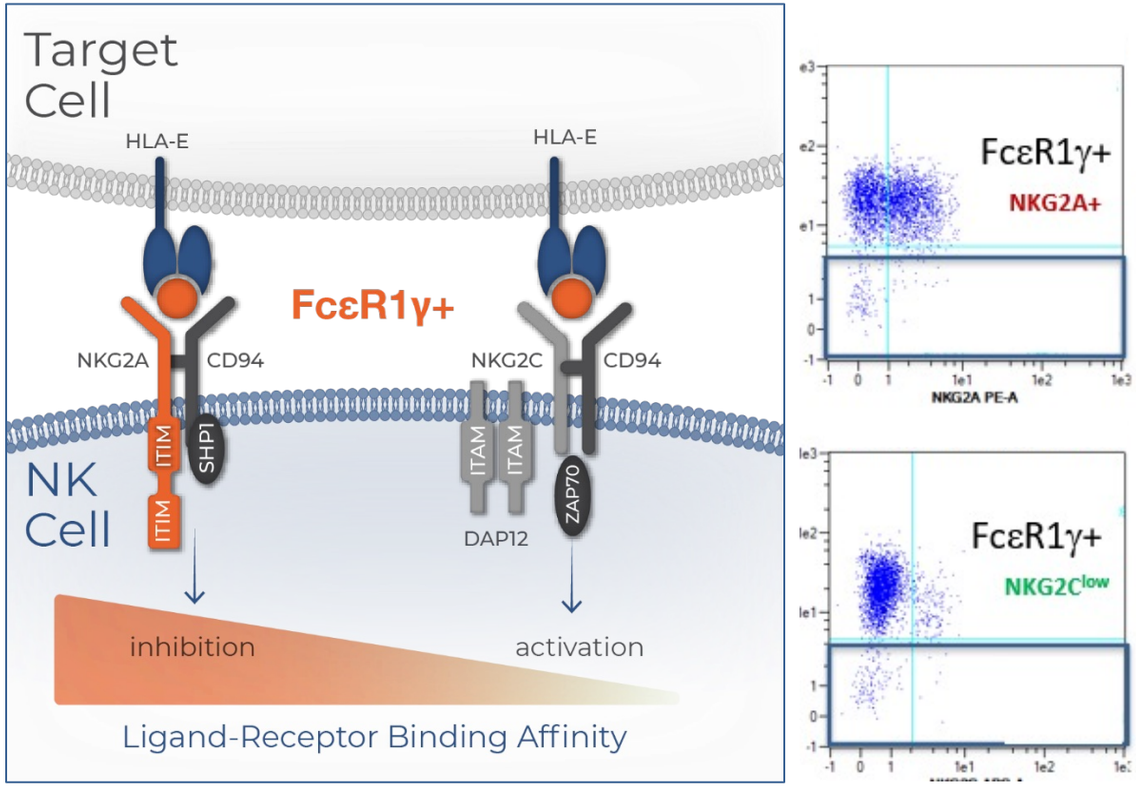
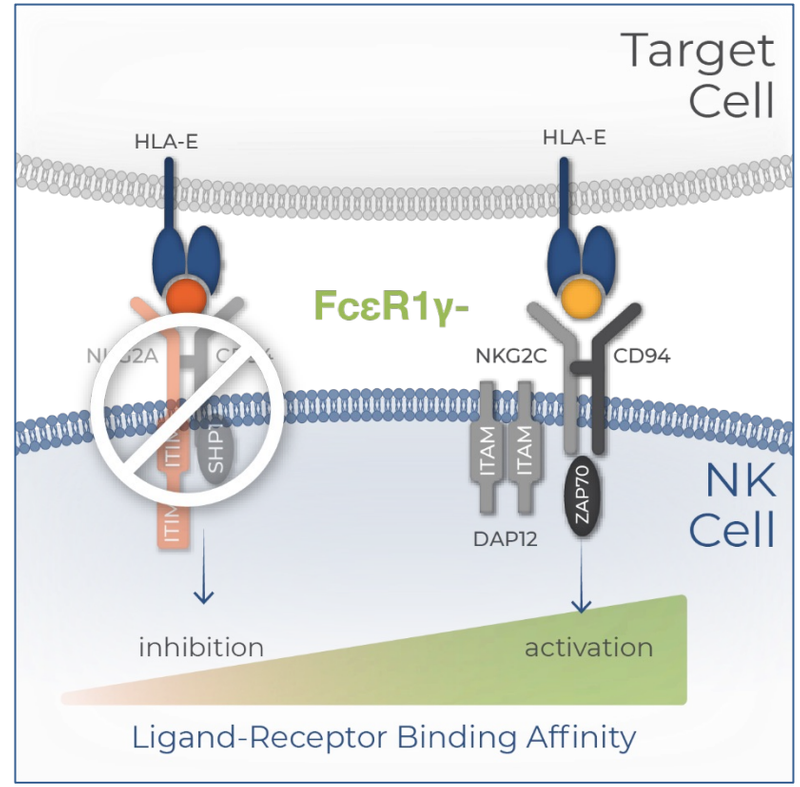
g-NK Cells

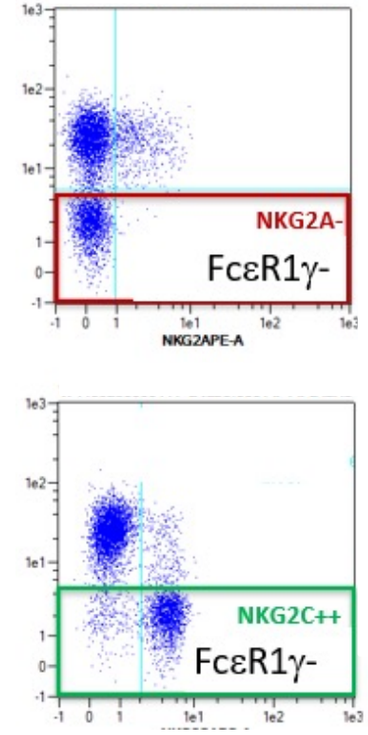
No Inhibition by NKG2A; >Potent Killing of HLA-E+ Targets Via Activating Receptor NKG2C

Conventional NK cells (cNK)
express NK cell checkpoint NKG2A



G-NK cells
are NKG2A negative





Adapted from Siemaszko et al. 2022

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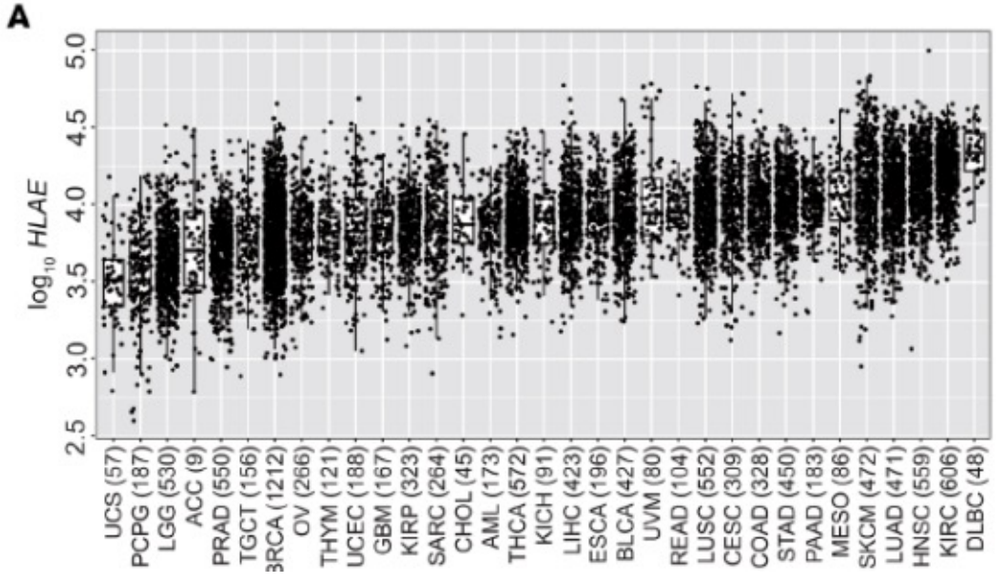
The Inhibitory Effect of The HLA-E /NKG2A Axis is Well Established in Cancer & Autoimmune Disease



HLA-E expression is a common tumor escape mechanism in many cancer types.

Unbiased analysis of *HLAE* expression in human tumors using data from 10,375 tumor samples, representing 33 tumor types, made available by The Cancer Genome Atlas (TCGA) Research Network.

Finding	Cancer & Autoimmune	Citation
NKG2A associated with bad prognosis	• AML • NHL • PDAC	• *Stringaris 2014* • *Vietzen 2023* • *Liu 2023*
NKG2C associated with good prognosis	• AML & other heme malignancies • NHL • MS • MS	• *Cichocki 2015, 2019* • *Wagner 2020* • *Martinez-Rodrigues 2016* • *Vietzen 2023*
HLA-E negative prognostic factor	• Gastric Cancer • Ovarian & Breast cancer • Gynecological cancer • Colorectal cancer • Laryngeal cancer • MS	• *Morinaga 2022* • *Borst 2020, de Kruif 2010* • *Gooden 2011* • *Levy 2008, Guo 2015* • *Silva 2011* • *Vietzen 2023*

MS = Multiple Sclerosis

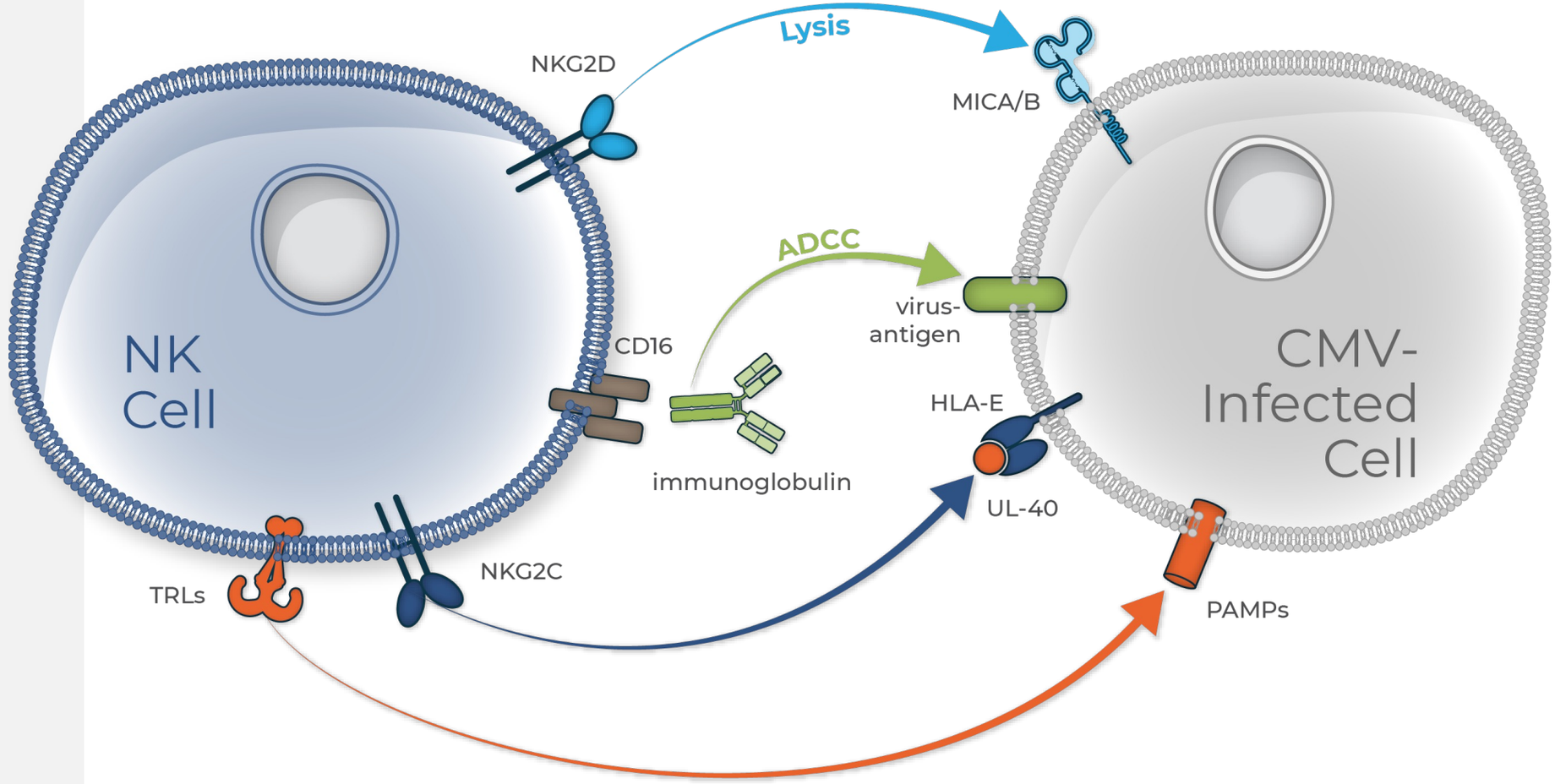
 Kamyia et al 2019

g-NK Cells Eradicate Virally Infected Cells

Viral targets play important roles in cancer (HPV) and autoimmune disease (EBV)

g-NK cells have enhanced anti-viral activity

- Robust ADCC mediated by anti-viral antibodies
- Targeting of viral peptides presented on HLA-E via high levels of NKG2C and low NKG2A expression



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Robust GMP Manufacturing & Cryopreservation

Established manufacturing of PBMC-derived allogenic off-the-shelf product

Leukapheresis

Collect from healthy donors screened for Increased g-NK cells

Manufacturing

Isolate and expand g-NK cells

Storage

Cryopreserve g-NK cells stored until needed

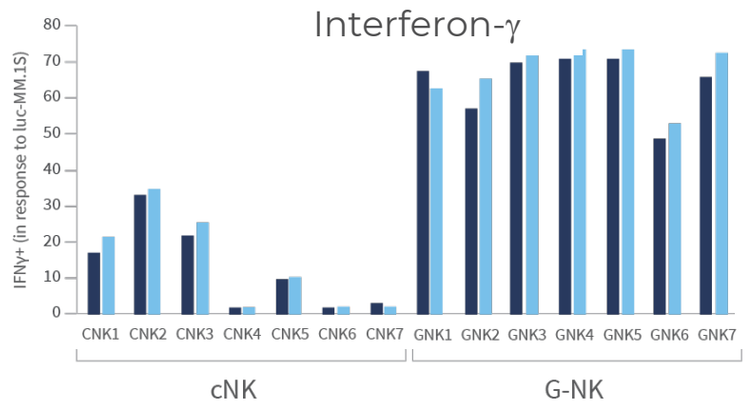
Infusion

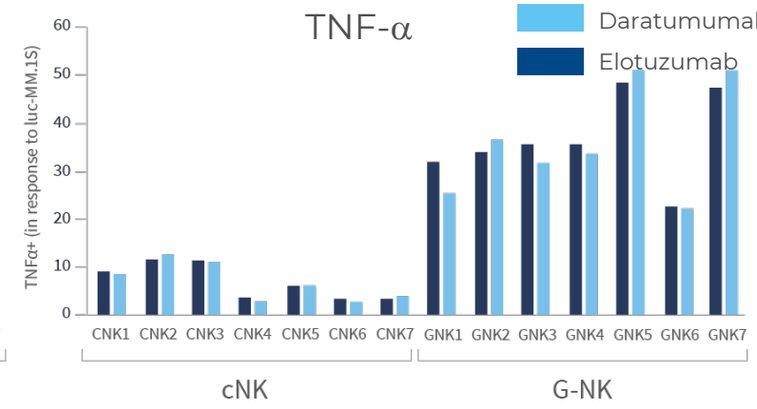
Infuse patient with G-NK cells

- Robust and reproducible process
- Minimal donor-to-donor variability
- Donor selection criteria: high amounts of g-NK cells, geno- and phenotypic profiling, and other proprietary parameters
- Isolation step to enrich for NK cells, then g-NK cells are preferentially expanded for approximately 2 weeks using a proprietary feeder cells and cytokines.
- Solid IP protection

Interferon-γ TNF-α

G-NK Cell Products Show Low Donor-to-Donor Variability





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Indapta's G-NK Cell Pipeline

Allogeneic product without the need for engineering allows for efficient expansion of pipeline through combinations with mAb

Allogeneic, derived from pre-screened donor PBMC, "natural" NK cell cryopreserved product (IDP-023)

Disease	Indication	Target (mAb)	Preclinical	IND Enabling	Phase 1	Phase 2
Cancer	NHL, MM	CD20 (rituximab), CD38 (daratumumab)				
	AML Low Burden Disease	None	Killing of AML cell lines (Q1 '24)		Amend Protocol Q2 2024	
Autoimmune	Multiple Sclerosis (MS)	CD20 (ocrelizumab)	Killing of autoreactive B & T cells from MS pts (Q2 '24)		IND 2024	
	Autoimmune kidney diseases	CD19 or CD38	B cell depletion from healthy donors & SLE pts (Q1-Q2 '24)		IND 2024	

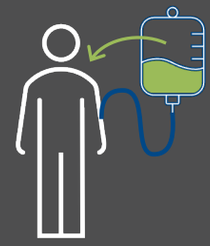
AML=acute myelogenous leukemia;
MRD=measurable residual disease;
NHL=non-Hodgkin's lymphoma

Phase 1 Hematologic Malignancy Trial

Non-Hodgkin's Lymphoma, Multiple Myeloma and Acute Myelogenous Leukemia

Dose escalation in cohorts

Run-in
(initial patients)
- Single Dose
- Multi-Dose
- Addition of IL2
- Two Schedules



MM Cohort
IDP-023 cells x 3 + daratumumab

NHL Cohort
IDP-023 cells x 3 + Rituximab

AML Low Disease Burden Cohort
IDP-023 cells x 3

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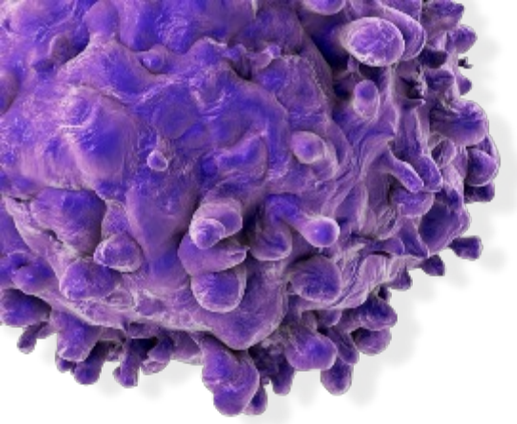

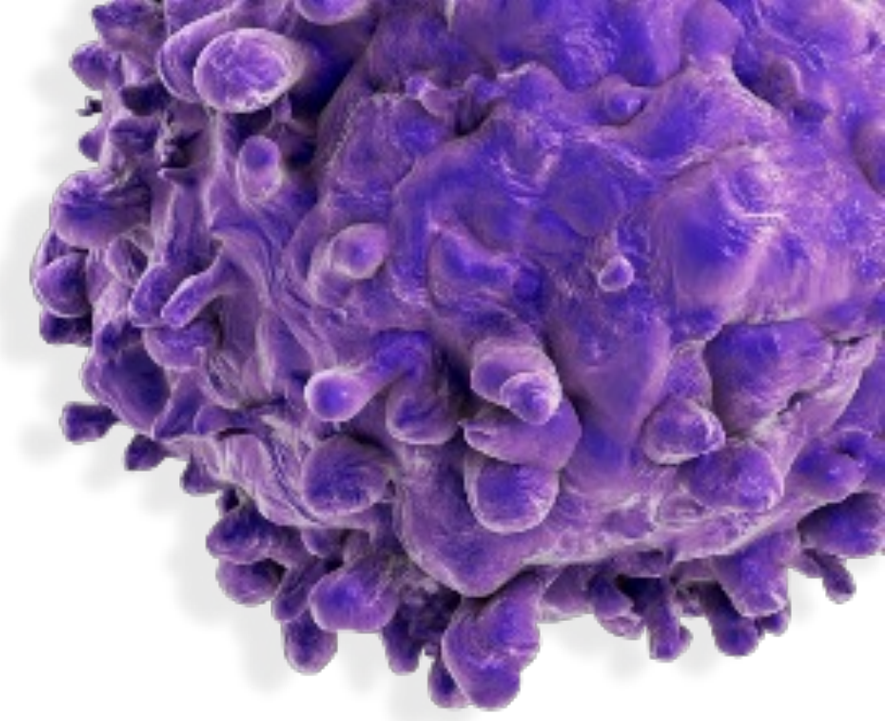

Autoimmune disease: Differentiated Opportunity for IDP-023

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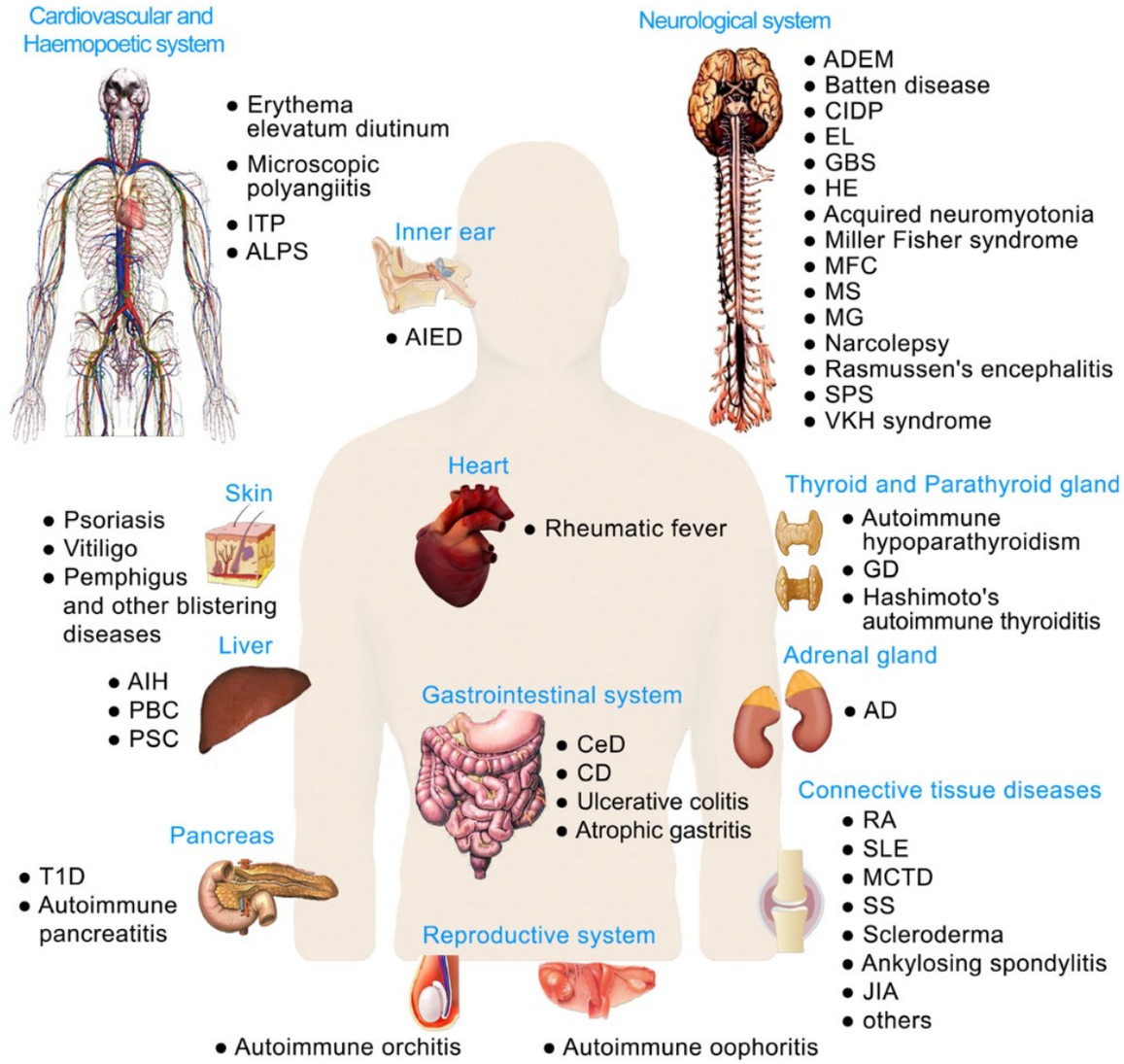
Autoimmune Disease: High Unmet Need

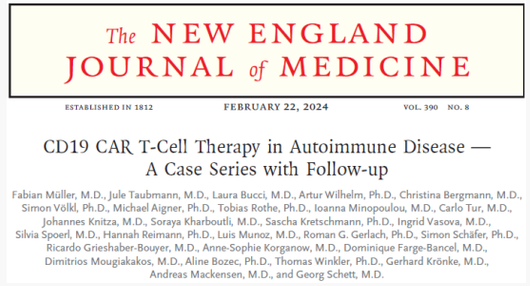
- >10% of population[1]
- 25-31 million people in US[2]
- Current therapies inadequate and often consist of lifelong immune suppression

>100 Diseases Affecting Most Organs & Tissues[3]



1) *Conrad et al., Lancet 2023*
2) *Nat'l. Acad. Sci, Eng, & Med, 2022*
3) *Wang et al., J. Int. Med. 2015*

Impressive Clinical POC from Autologous CD19 CAR T Therapy



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 22, 2024 VOL. 390 NO. 8

CD19 CAR T-Cell Therapy in Autoimmune Disease — A Case Series with Follow-up

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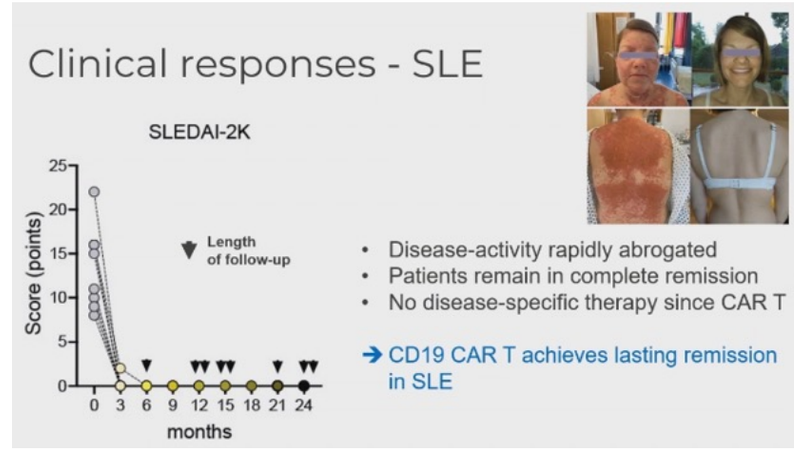
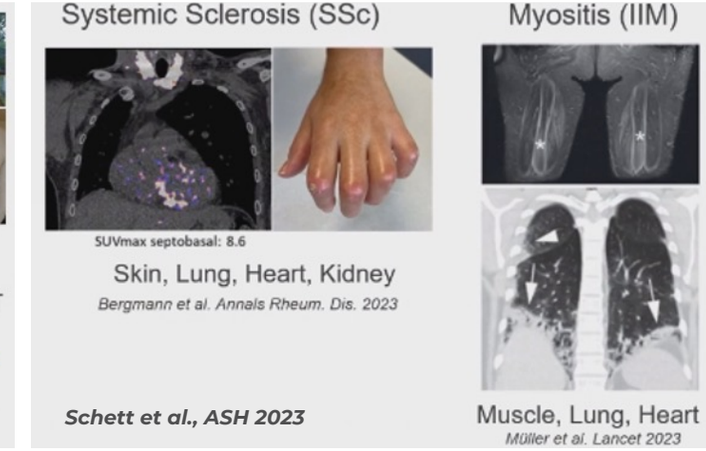
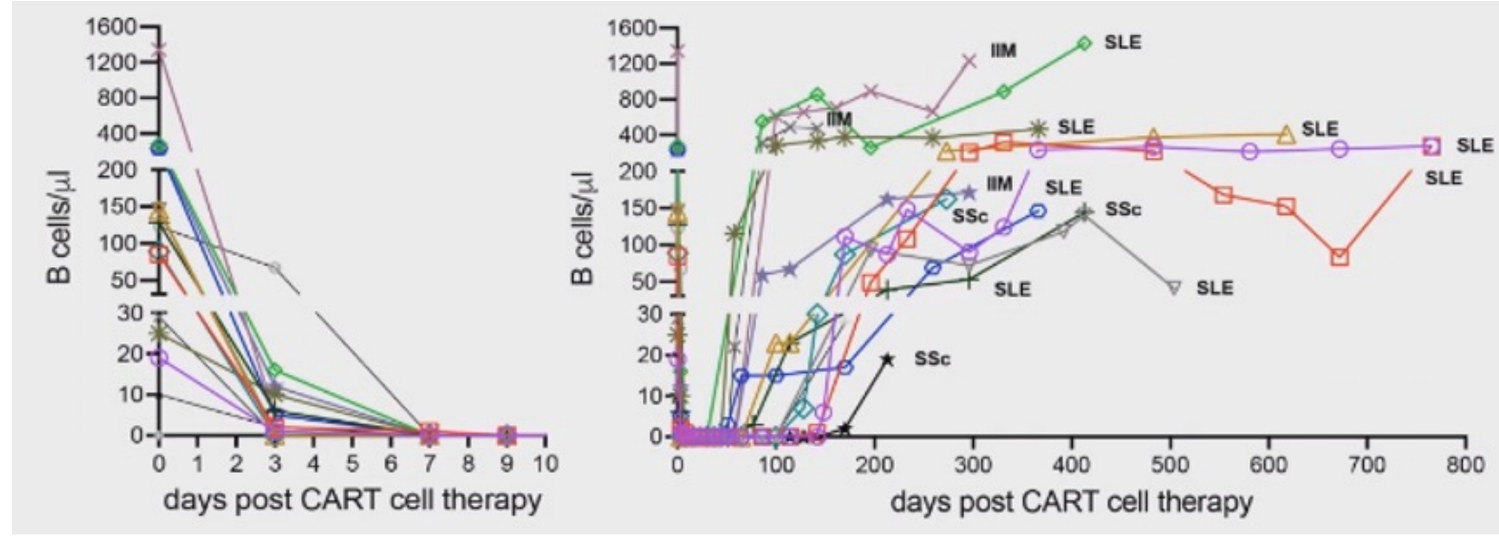
- Rapid resolution of disease activity
- MOA: depletion of B Cells
- Patients remain treatment free despite B cell recovery

Transient B-cell suppression provides durable responses in Multiple autoimmune diseases







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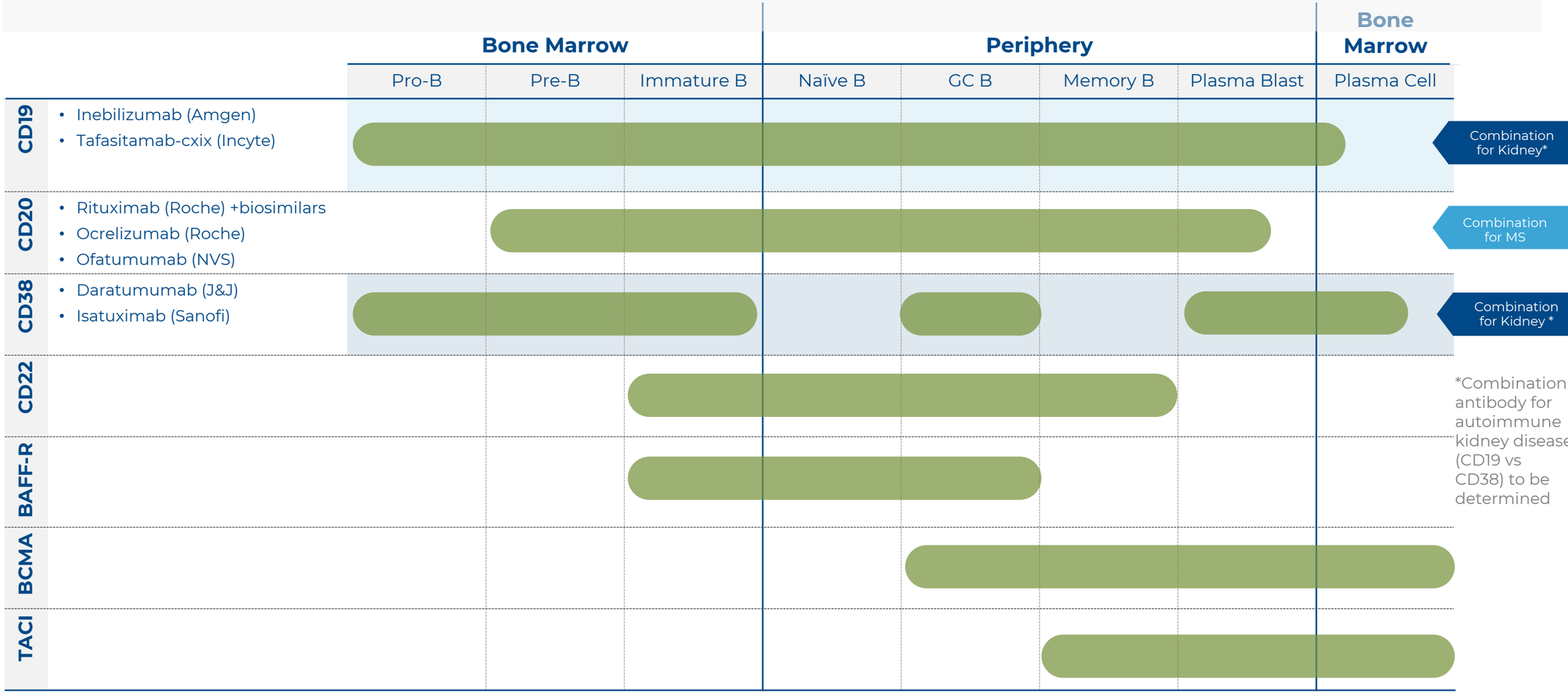
Opportunity for IDP-023 in Autoimmune Disease

- Superior safety to autologous CAR-T would allow for broader population of patients to be treated
  - Low risk of prolonged B-cell aplasia, CRS, ICANs
  - No risk of mutagenicity
- Allogeneic cryopreserved product for on-demand treatment
- NK cells reach peak activity at infusion for rapid target activity
- Multiple MOA beyond B cell depletion: potential to treat much larger spectrum of diseases
  - Superior ADCC mediated B cell depletion in combination with B cell targeting mAbs[1]
  - Low fratricide in combination with CD38 targeting mAb due to low CD38 expression on g-NK cells[1]
  - Direct killing of HLA-E expressing autoreactive T & B cells
  - Elimination of viral reservoir through killing of virally infected cells that may be responsible for disease initiation and maintenance

MOA=Mechanism of Action; [1]*Bigley et al., Blood Adv. 2021*

IDP-023 Can Be Combined with Approved mAbs to Deplete B Cells



Adapted from : Zhang et al., Frontiers in Immunology 2023

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Autoimmune Kidney Disease

IND filing in 2024

Basket trial
- Lupus nephritis (LN)
- Primary membranous nephropathy (PMN)
- IgA nephropathy (IgAN)

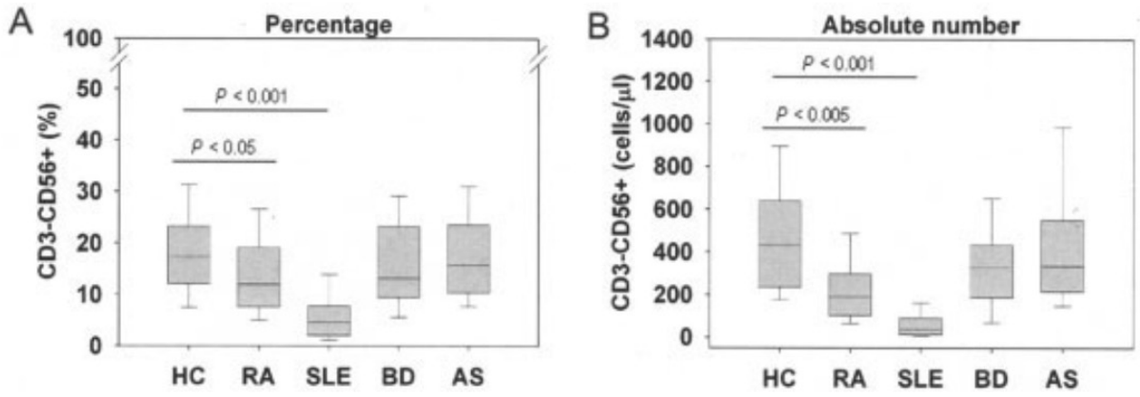
Jan Hillson, MD
Clinical Development Lead for Indapta
(Previously led Phase 3 Global LN trial)

- Rationale for mechanism
  - B cell depletion required for disease resolution in SLE
    - Monotherapy with rituximab only leads to “shallow” depletion of B cells in periphery but does not reach “deeper” into secondary lymphoid organs[1,2]
    - Autologous CD19-CAR T cell therapy resulted in transient but deep B cell depletion and disease resolution[3]
  - B cell depletion with CD38 mAb active in LN & PMN[4,5]
  - SLE & LN patients have decreased NK cell numbers[6]
  - Adoptive transfer of g-NK with superior ADCC[7] allows for combination with mAb or innate immune cell engagers for deeper depletion of B cells
- Short term readouts to quickly reach clinical POC
  - Autoimmune antibody titers
  - Proteinuria
  - Infiltrates on kidney biopsy
- KOL enthusiasm (Georg Schett, Erlangen-Nürnberg)

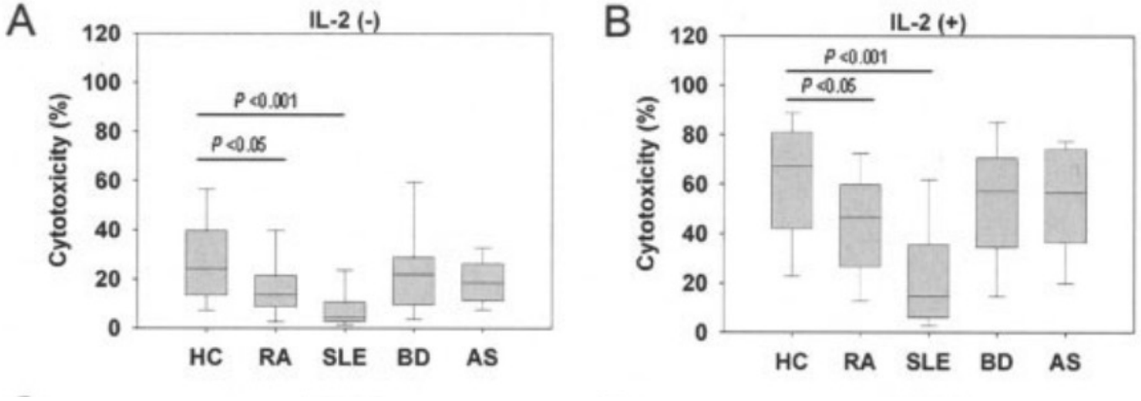
1) *Vos et al., Arthritis and Rheumatism 2007*
2) *Anolik et al., Arthritis and Rheumatism 2007*
3) *Mueller et al., NEJM 2024*
4) *Flesher et al., ASN Kidney Week 2023*
5) *Rocatello et al., Nature Medicine 2023*
6) *Park et al., Arthritis & Rheumatism 2009*
7) *Bigley et al., Blood Advances 2021*

Adaptive Cell Therapy With IDP-023 is Predicted to Deepen B Cell Depletion in Combination with mAb as SLE Patients Have Impaired NK Cell Numbers & Function

Number of NK cells



Cytotoxic Activity



Finding	Citation
NK cells, and their subpopulations of CD56(+) and CD16(+) cells, are decreased in patients with SLE as compared to controls.	• *Thangjam 2023*
NK cytotoxicity of SLE patients was deficient compared to controls and showed an impaired response to IL-15	• *Lin 2017*
Negative correlation of NK cell counts with disease activity i.e., the more severe the disease, the lesser the NK cell count	• *Spada 2015*
Impaired Differentiation and Cytotoxicity of NK cells in SLE	• *Park 2009*

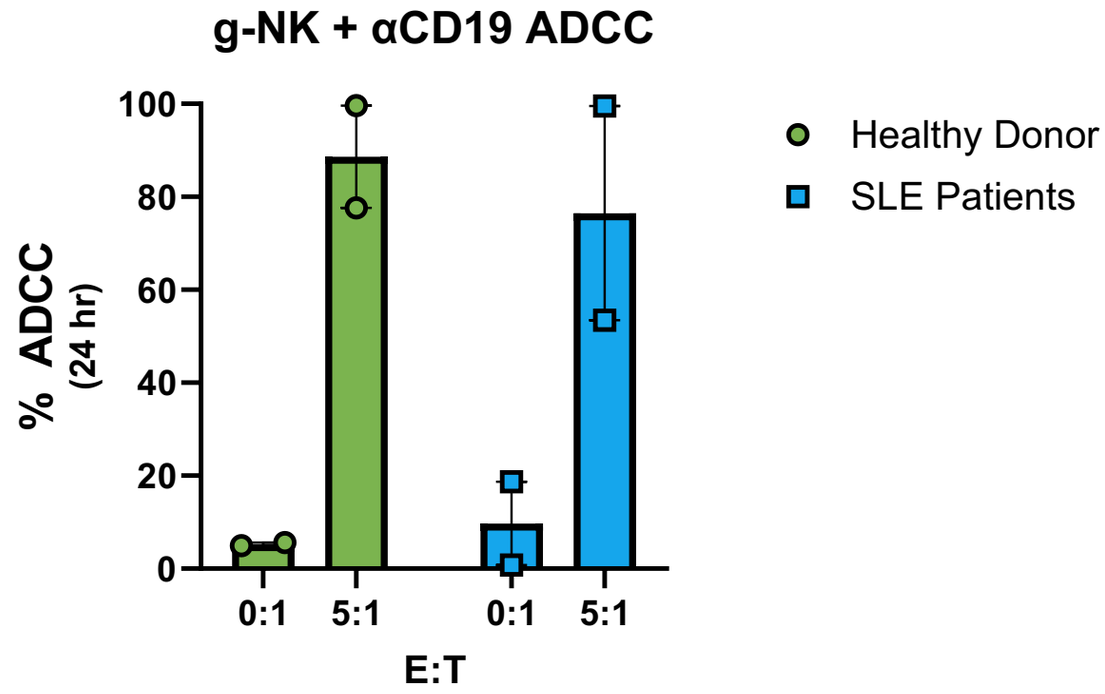
HC- Healthy Control
RA- Rheumatoid Arthritis
SLE- Systemic Lupus Erythematosus
BD- Behcet's Disease
AS- Ankylosing Spondylitis

Park et al., Arthritis & Rheumatism 2009

Potent IDP-023 ADCC activity against B cells from healthy donors or patients with SLE (anti-CD19 mAb)

g-NK cell mediate potent & dose-dependent killing of:

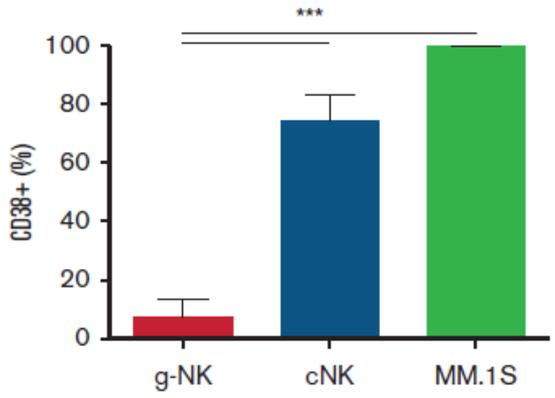
- ✓ Healthy donor B cells
- ✓ B cells from patients with SLE
- • Plasma cells from healthy donors (in progress)
- • Autoimmune T cells (in progress)



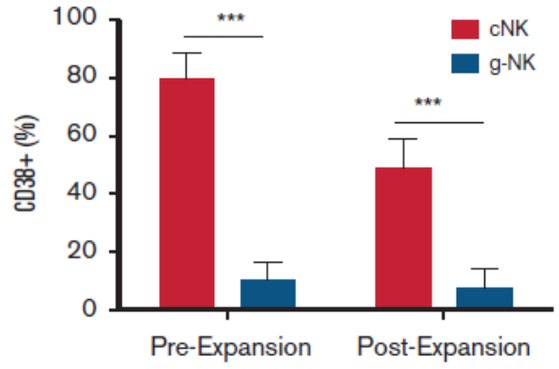
Indapta data; 24hr assay
CD19 mAb (1ug/mL), 1 HD in duplicate, 2 donors SLE
TAB-431CQ-ADCC, Creative Biolabs

Low Levels of CD38 Expression Allow for Effective Combination with Anti-CD38 mAb

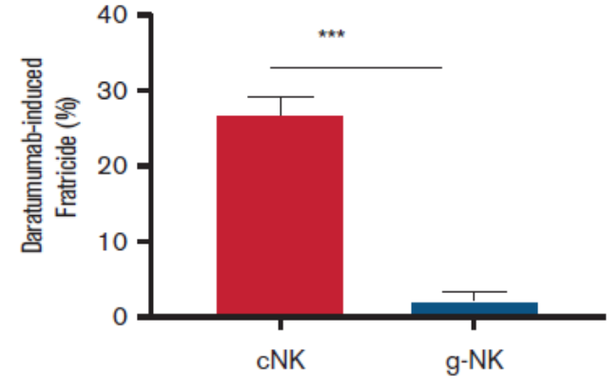
CD38 Expression is significantly lower in g-NK cell than cNK



CD38^{low} phenotype is stable even after expansion



Negligible fratricide in combination with Daratumumab (4hr assay)



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Progressive Multiple Sclerosis

IND filing in 2024

High unmet need

- 100-150K patients in US
- Ocrelizumab only approved therapy and patients still progress[1]

1) *Montalban et al.,NEJM 2017*
2) *Hauser et al., NEJM 2017*
3) *Hauser et al., NEJM 2008*
4) *Greenfield et al., Ann Neurol 2018*
5) *Vietzen et al., Cell 2023*
6) *Martinez-Rodriguez et al., Mult. Scler. 2016*

- Multiple potential mechanisms
  - Deeper B cell depletion via ADCC with anti-CD20 mAb
    - B cell depletion with mAbs effective in MS[1,2,3]
    - Lymph node B cells not fully depleted by anti-CD20 mAb & may provide ongoing source of disease activity[4]
  - Eradication of myelin reactive T & B cells
    - Presence of g-NK cells reduces risk of developing MS in individuals infected with EBV[5]
    - Presence of g-NK cells reduces risk of disease progression in individuals with MS[6]
  - Killing of EBV infected cells
- KOL endorsement
  - “I'm highly enthusiastic about your approach, because I think it has the same potential for success as …Ublituximab when I agreed to ‘chair" the trials’ in 2017” -- *Lawrence Steinman, Professor, Stanford*

Recent Data Highlight Potential for g-NK Cells to Control Multiple Sclerosis

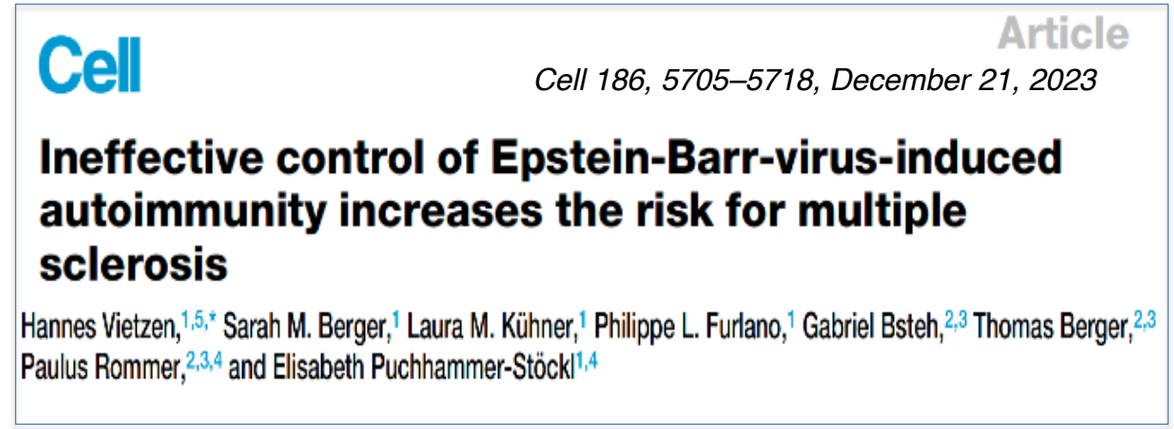
nature reviews immunology Volume 24 | February 2024 | 87–90 | 88

Research highlights

Autoimmunity

Natural killer cells that target autoimmune cells linked with protection against multiple sclerosis

- Authors suggest that **inducing high-levels of NKG2C+ NK cell responses** could be option to limit the progression of MS and offer a new strategy for MS therapy
- **Suggests that g-NK cells are viable adoptive cell therapy for patients with MS**



Cell

Article

Cell 186, 5705–5718, December 21, 2023

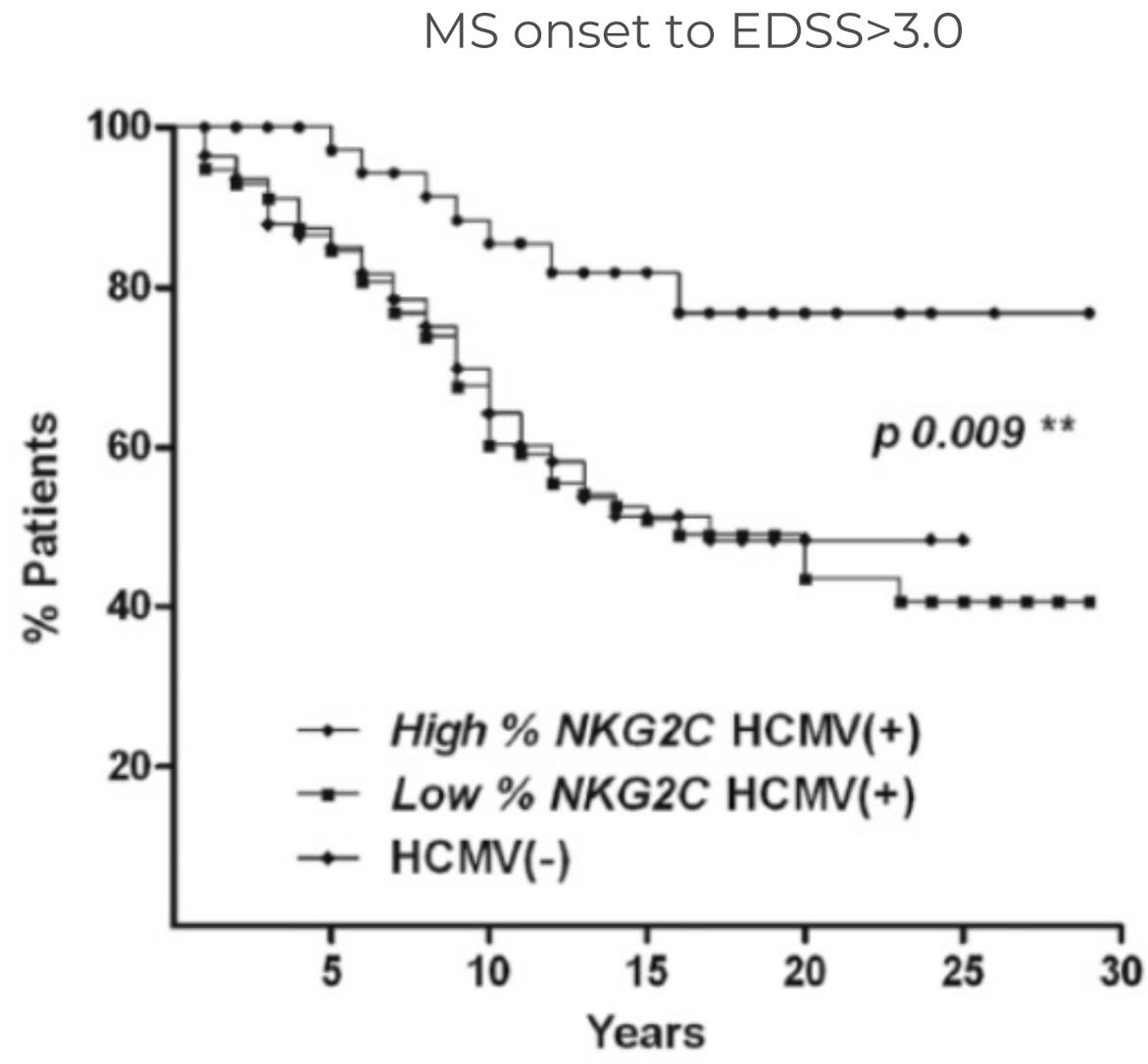
Ineffective control of Epstein-Barr-virus-induced autoimmunity increases the risk for multiple sclerosis

Hannes Vietzen,[1,5,*] Sarah M. Berger,[1] Laura M. Kühner,[1] Philippe L. Furlano,[1] Gabriel Bsteh,[2,3] Thomas Berger,[2,3] Paulus Rommer,[2,3,4] and Elisabeth Puchhammer-Stöckl[1,4]

- Control of autoimmunity by **NKG2C+ NK** cell responses is severely impaired in MS patients
- MS-patient-derived GlialCAM-specific cells evade control via inhibitory **HLA-E/NKG2A axis**
- MS patients are predominantly infected with **EBV** variants that highly upregulate HLA-E
- Specific cytotoxic T cell responses can control EBV-infected GlialCAM-specific B cells

Positive CMV Serostatus and NKG2C+ NK Cell Expansion Delay Disability Progression in MS

Kaplan-Meier analysis of the time from disease onset to the assignment of a sustained Expanded Disability Status Scale (EDSS) score>3.0, classifying MS patients according to NKG2C expression and HCMV serostatus (HCMV(+) high% NKG2C+ NK-cells, n=44; HCMV(+) low% NKG2C+ NK-cells, n=115; HCMV(–) patients; n=87). Log-rank test p-value: **<0.01

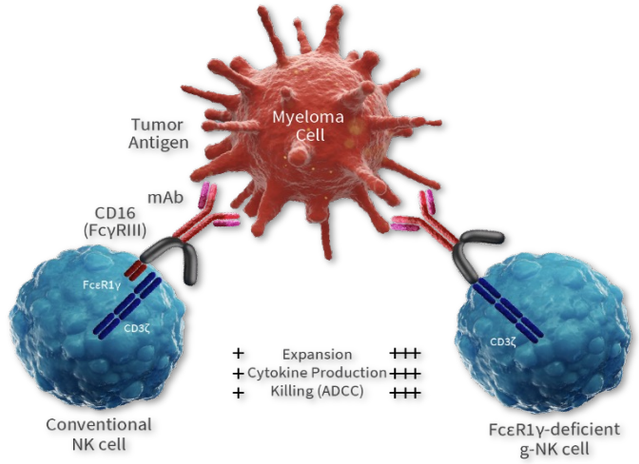
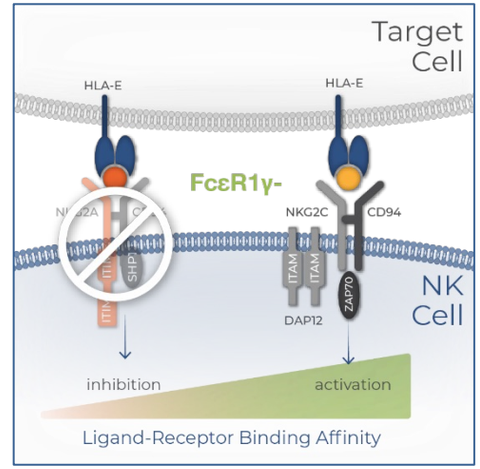
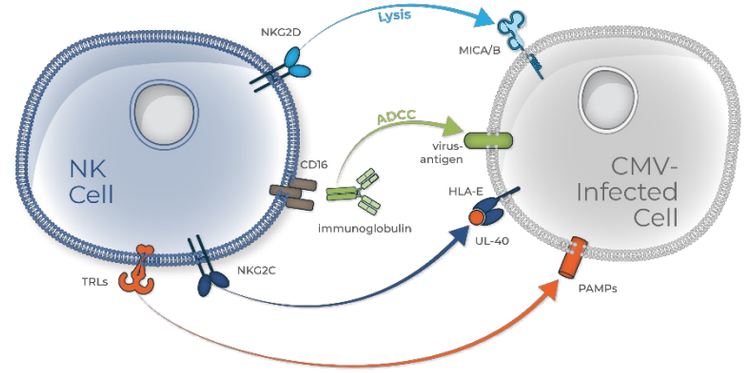


NKG2C^{high} HCMV^{+} individuals are protected

NKG2C^{low} individual (HCMV^{+or-}) have significantly faster EDSS

 Martinez-Rodriguez et al., Mult. Scler. 2016

g-NK Cell Therapy Provides Multifactorial MOA for Curative Treatment of MS

Deep depletion of EBNA-autoantibody producing B cells	Depletion of GlialCAM-specific autoreactive T& B cells*	Elimination of latent viral reservoir*
		

*These mechanisms are unique to g-NK and Indapta's IDP-023

INDAPTA THERAPEUTICS

Two Differentiated Autoimmune INDs in 2024 to Demonstrate POC

Intensive biomarker interrogation to document MOA & demonstrate applicability of IDP-023 to broad spectrum of autoimmune diseases

	Multiple Sclerosis	Autoimmune Kidney Disease
Indications	1° & 2° progressive MS (only approved therapy is ocrelizumab)	Basket trial of lupus nephritis, primary membranous nephropathy, IgA nephropathy & additional orphan kidney diseases
MOA	B cell depletion, killing of autoreactive B & T cells, anti-viral (EBV)	B cell depletion, killing of autoreactive B & T cells, possibly anti-viral (EBV)
Intervention	IDP-023 + ocrelizumab (include control arm of ocrelizumab alone)	IDP-023 + anti-CD19 or anti-CD38 mAb
N	~18	~15
Endpoints	Changes in immune biomarkers along with imaging & clinical endpoints	Changes in proteinuria, levels of pathogenic antibodies, histology, clinical endpoints
IND Filing	2024	2024

Details of trial design, timeline, budget and data readouts in development and will be provided prior to financing

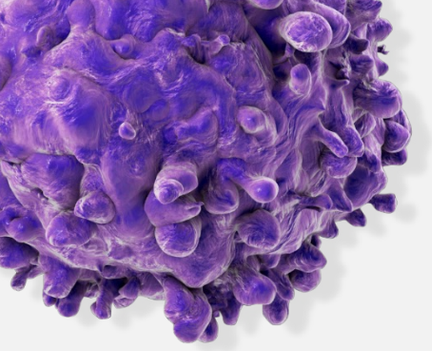
Accomplishments & Execution Milestones

Accomplishments 2023 - 2024

- IND for heme malignancies cleared May 2023
  - 4 patients treated to date
  - IND amended to add AML cohort to heme malignancy trial
- 9 successful GMP runs of IDP-023
- Fast-Track status granted by FDA

Execution Milestones

- Clinical POC heme malignancies (NHL, MM, AML)
  - 6 mo f/u 2H 2025
- POC in MS & autoimmune kidney diseases
  - 2 IND filings 2H 2024



Indapta Value Proposition

Clearly Differentiated Platform Technology

On Demand Product; Favorable Safety

- Allogeneic, cryopreserved product
- No need for genetic engineering or post expansion cell selection

Superior MOA

- Superior ADCC & antibody independent killing
- Greater
  - Cytokine secretion
  - Cytolytic activity
  - Persistence
- HLA-E targeting (NKG2C+/NKG2A-)
- Anti-viral targeting

Robust GMP Manufacturing

- Optimized donor screening
- Low batch-to-batch variability
- 9 successful GMP runs completed
- Multiple doses per donor

Phase 1 in Hematologic Malignancies

- IND cleared April 2023
- Phase 1 initiated Q4 2023
  - NHL
  - Multiple myeloma
  - AML amendment
- FastTrack designation

Autoimmune: Rapid Timeline to FIH

- Efficient Pipeline development
- 2 INDs 2024

Strong IP

- Broad granted patent protection
- Portfolio of pending applications