Creating Transformative Medicines to Improve Patients' Lives

July 24, 2025

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Janux – multiple near-term high value opportunities in cancer and I&I

Clinical Programs

- JANX007: PSMAxCD3-TRACTr undergoing P1b dose expansion trials in mCRPC
  - Position JANX007 to move into early lines of mCRPC therapy
- JANX008: EGFRxCD3-TRACTr undergoing clinical P1a evaluation in multiple solid tumor settings

Development Pipeline

- PSMAxCD28-TRACIr: designed to enhance T cell activation and durability of JANX007
  - IND anticipated in 1H2026
- TROP2xCD3-TRACTr: TRACTr platform provides access to previously intractable TCE target
  - IND-enabling activities planned in 2H2025
- CD19-ARM: novel bispecific with potential in autoimmune disease and heme-onc
  - First patient treated in HNV trial planned in 1H2026

Cash Position

- Robust cash position of $1.01 billion* as of March 2025
  - Runway through JANX007 interim pivotal data, JANX008 P1b data and human proof-of-concept data on TROP2-TRACTr, PSMAxCD28 TRACIr and CD19-ARM

**As of March 31, 2025, includes cash, cash equivalents and short-term investments*

Summary of new programs

PSMAxCD28-TRACIr Program

- Combination with JANX007 to further differentiate depth and durability of patient responses
- CD28-based TRACIr platform designed to enhance T cell activation and durability of CD3-based TRACTr platform
- TRACTr and TRACIr platforms built on same technology with same tumor-activation and PK design features

TROP2-TRACTr Program

- Builds upon learnings from JANX007 and JANX008 clinical programs
- TRACTr technology unlocks access to potential high-value TCE target
- Provides broad solid tumor indication expansion to Janux clinical portfolio

CD19-ARM Program

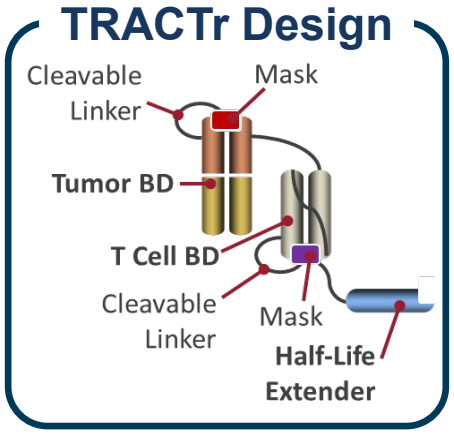
- Builds upon Janux TCE expertise to redesign bispecific T-cell engagers
- Differentiated non-clinical profile provides best-in-class opportunity
- Extends Janux pipeline into autoimmune disease

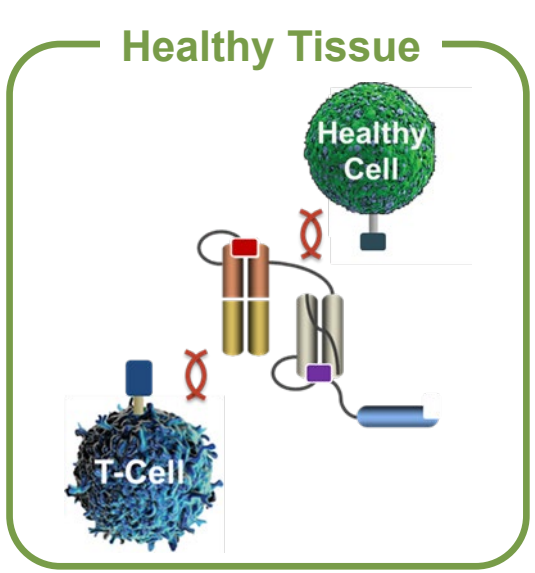
Janux pipeline

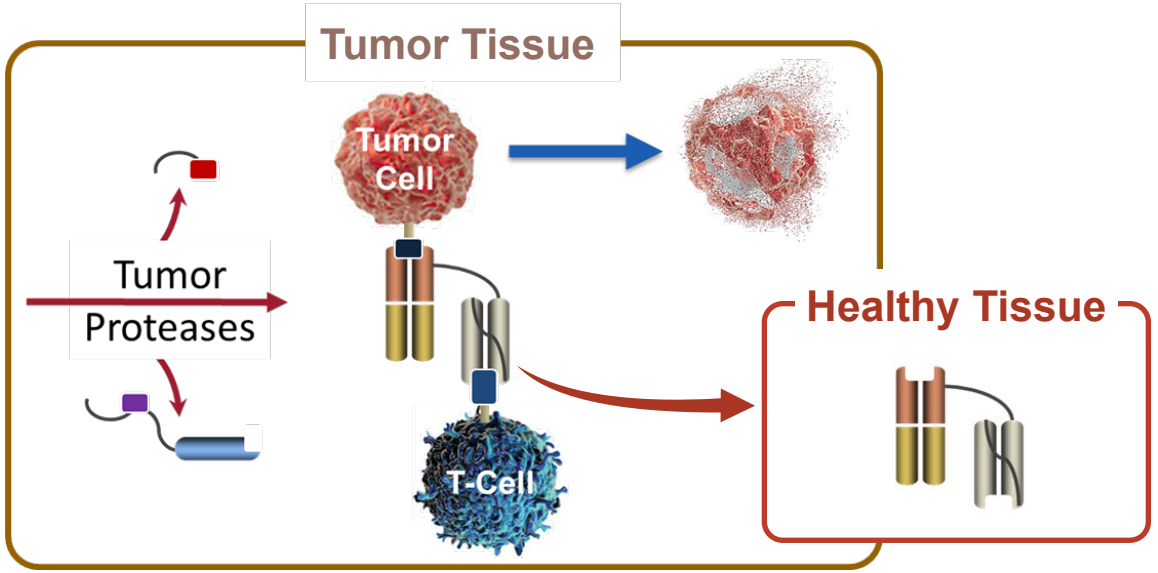
	Target	Indications	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3
Wholly owned TRACTr Programs							
JANX007	PSMA x CD3	mCRPC					
JANX008	EGFR x CD3	EGFR+ solid tumors					
TROP2-TRACTr	TROP2 x CD3	TROP2+ solid tumors					
Wholly owned TRACIr Program							
PSMA-TRACIr	PSMA x CD28	mCRPC					
Wholly owned ARM Program							
CD19-ARM	CD19	AID					
Partnered Programs							
Merck	Two Undisclosed	Undisclosed			Program Progress Not Disclosed		

Janux Tumor Activated T-Cell Engager (TRACTr) platform design principles

Each program is designed as a potent T-cell engager with reduced toxicity







Masks block activity against healthy tissues to reduce CRS and on-target, healthy tissue toxicity

Tumor specific activation to maximize anti-tumor immune response

TCE is rapidly cleared from healthy tissue to limit systemic toxicity

Emerging JANX007 clinical data demonstrates TRACTr platform can potentially improve both safety *and* efficacy compared to contemporary TCEs

Tumor Activated Immunomodulator (TRACIr) Platform

Introduction

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TRACIr platform designed to enhance activity and durability of TRACTr platform

Combination with JANX007 to further differentiate depth and durability of patient responses

Janux *raised the bar* with JANX007†

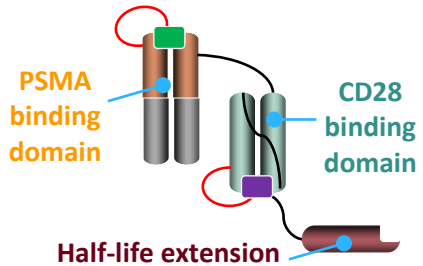
Best-in-treatment potential in prostate cancer

Median Tx line	rPFS (mo) median	% rPFS 6-month	ORR*	Best PSA decline	≥ PSA50 at 12wks	≥ PSA90 at 12wks
5L	7.5-7.9	65-78%	50%	PSA50: 100% PSA90: 63%	75%	50%

Janux aims to *raise the bar again*

Combining PSMA-TRACTr with PSMA-TRACIr

PSMA-TRACIr



- Enhance a potentially best-in-treatment asset, JANX007, with CD28 co-stimulation
- Decrease T-cell exhaustion to increase durability
- Deeper longer clinical responses

Addition of PSMA-TRACIr strengthens Janux's prostate cancer franchise

†Data disclosed from JANX007 Dec 2024 public update and May 2025 public update with respect to rPFS.
**includes confirmed and unconfirmed responses*

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PSMA is an ideal target to evaluate TRACIr platform

Opportunity for rapid proof of concept

What we know in prostate cancer

1. JANX007 clinical results validate* tumor activated platform in mCRPC
2. Predictable dose dependent clinical activity* makes JANX007 ideal combination agent
3. mCRPC is PSMA-TRACTr* and PSMAxCD28-costimulation† responsive

PSMA-TRACTr + PSMA-TRACIr rationale

- PSMA-TRACIr built with the same technology and design principles as PSMA-TRACTr
- Enhanced activity and durability potential
- Janux mCRPC drug development expertise can be applied to generate rapid TRACIr platform proof of concept

PSMA-TRACIr may further differentiate Janux therapies in the prostate cancer treatment landscape

*Note: "Validate" refers to early clinical validation of TRACTr platform from JANX007 clinical data aligning with the platform design principles and expectations *(based on Dec-2024 and May-2025 program public updates).*

†Stein et al 2023 (REGN5678)

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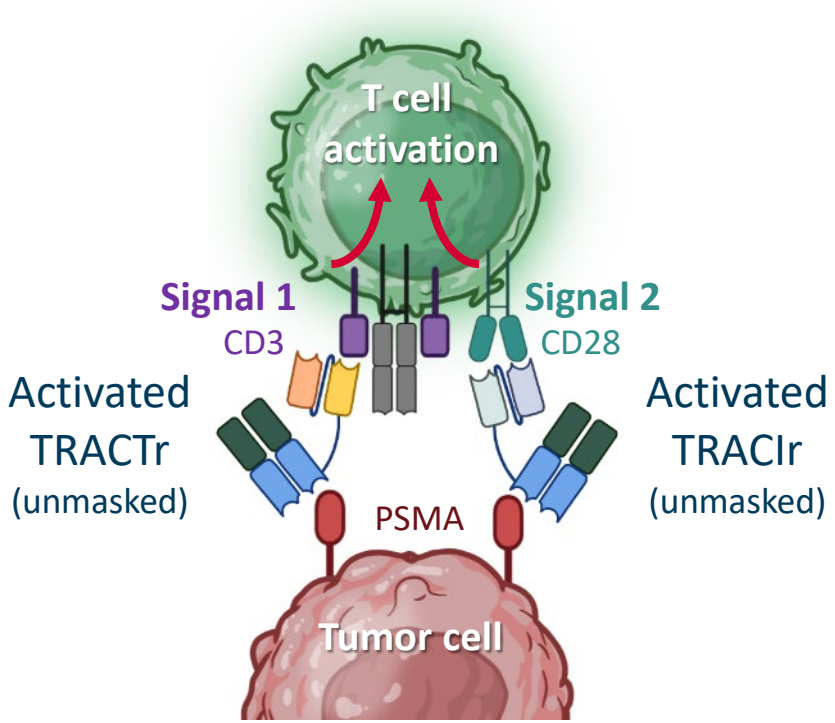
CD28-costimulation to potentially enhance duration of TRACTr mediated anti-tumor activity

Combination designed to further improve T cell immune response against the tumor

Enhanced durability from TRACTr plus CD28-costimulation



Signal-1 + Signal-2
PSMAxCD3 (TRACTr) + PSMAxCD28 (TRACIr)

Activated TRACTr – Signal 1

- Provides tumor recognition element to T cells (signal 1) leading to T cell activation
- Signal 1 drives anti-tumor T cell response and tumor cell killing

Activated TRACIr – Signal 2

- Co-stimulation through PSMAxCD28 (signal 2) enhances T cell activation and expansion
- Signal 2 increases ***durability*** of T cell driven anti-tumor response

Benefit from signal 2 requires presence of signal 1

Complementary tumor activated platforms aim to safely enhance depth and duration of T cell anti-tumor response

TRACTr and TRACIr combination approach

Unique opportunity to differentiate from non-masked approaches

TRACTr + TRACIr – Competitive differentiation

Masked TAA x CD3 + Masked TAA x CD28
designed to safely maximize strength and duration of anti-tumor T cell response

- Masks designed to reduce healthy tissue toxicity and CRS may be required based on clinical toxicity from non-masked assets (AMG160‡, REGN5678*, SAR443216ℍ)
- Potentially access higher doses, increase intra-tumoral drug concentrations, and elicit stronger efficacy
- Flexible dosing and tunable timing for each molecule to optimize patient anti-tumor immune response

Non-masked CD28 approaches limited by toxicity

*PSMA x CD28 (REGN5678)**

- Clinical activity of REGN5678 validates that CD28 co-stimulation can drive efficacy in prostate cancer
- Severe toxicity observed (≥Gr3 53% and 2 Gr5 AEs)

Her2 x CD3 x CD28 (SAR443216)ℍ

- No objective responses with DLTs of cardiac failure where Her2 is expressed‡ and Gr4 transaminases

Trispecifics CD3 x TAA x CD28 limited by design

- Intramolecular stoichiometry constraints may *not* match what is required for optimal T cell activation
- CD3 and CD28 binding in *trans* could lead to T cell fratricide
- Dose and timing of CD3 and CD28 *cannot* be independently varied which may be required for best clinical outcomes

Janux TRACTr + TRACIr complementary platforms with potential best-in-class safety and efficacy

‡Dorff 2024 (AMG160); *Stein 2023 (REGN5678); ‡Eaton 2023
ℍCalvo 2025 (SAR443216; NCT05013554) ESMO https://doi.org/10.1016/j.esmoop.2025.104199);

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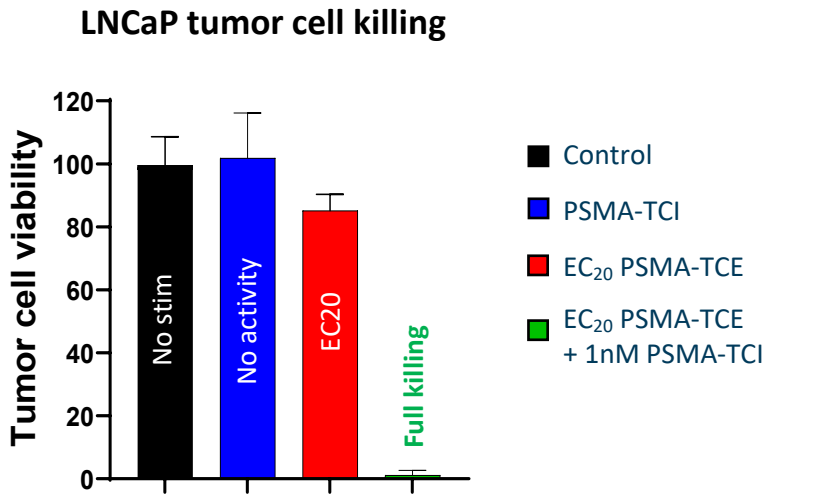
DLT = dose limiting toxicity

PSMA-TRACIr enhancement of T cell mediated tumor cell killing

Masking designed to reduce CRS and healthy tissue toxicity

CD28 co-stimulation enhances TCE activity

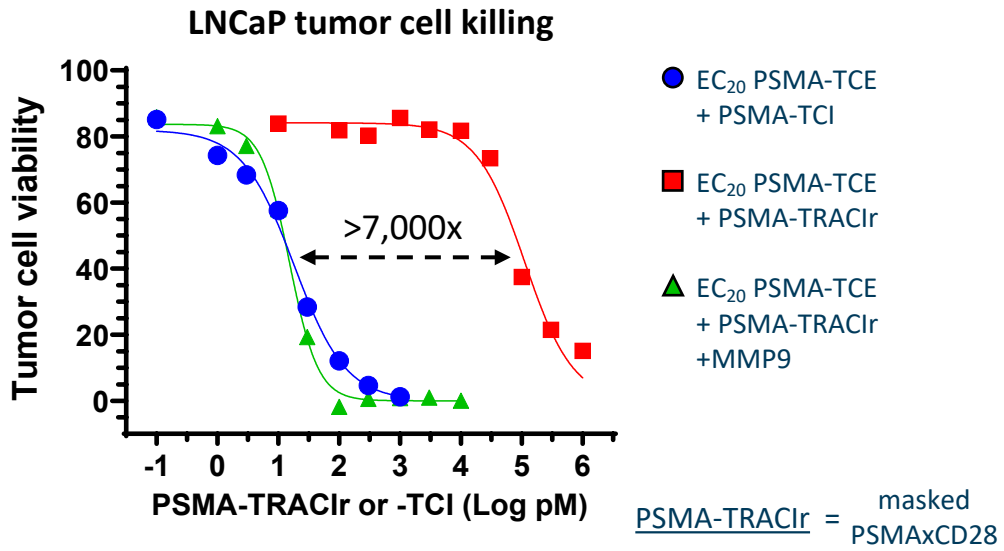


PSMA-TCI = PSMAxCD28

PSMA-TCE = PSMAxCD3

PSMA-TCI lacks activity as a single agent but enhances tumor cell killing of PSMA-TCE in combination

Cleavage dependent functional activity



PSMA-TRACIr = masked PSMAxCD28

PSMA-TRACIr exhibits large functional shift relative to non-masked PSMA-TCI

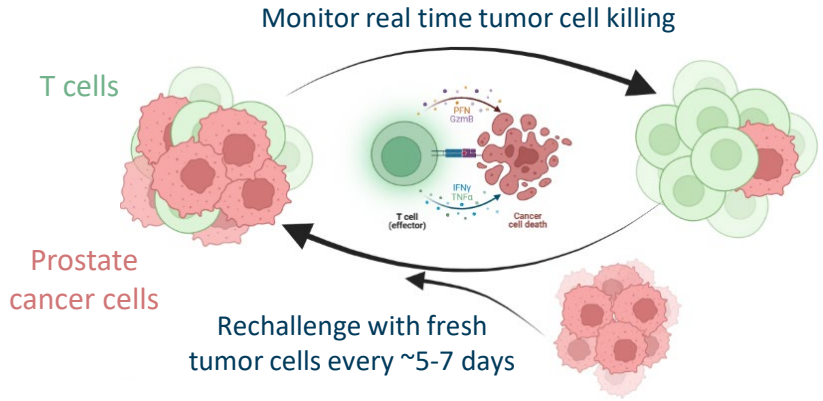
PSMA-TRACIr designed to enhance PSMA-TRACTr in combination

TRACIr extends duration of TRACTr mediated tumor killing

CD28 co-stimulation prolongs T cell cytolytic function

T cell durability assay

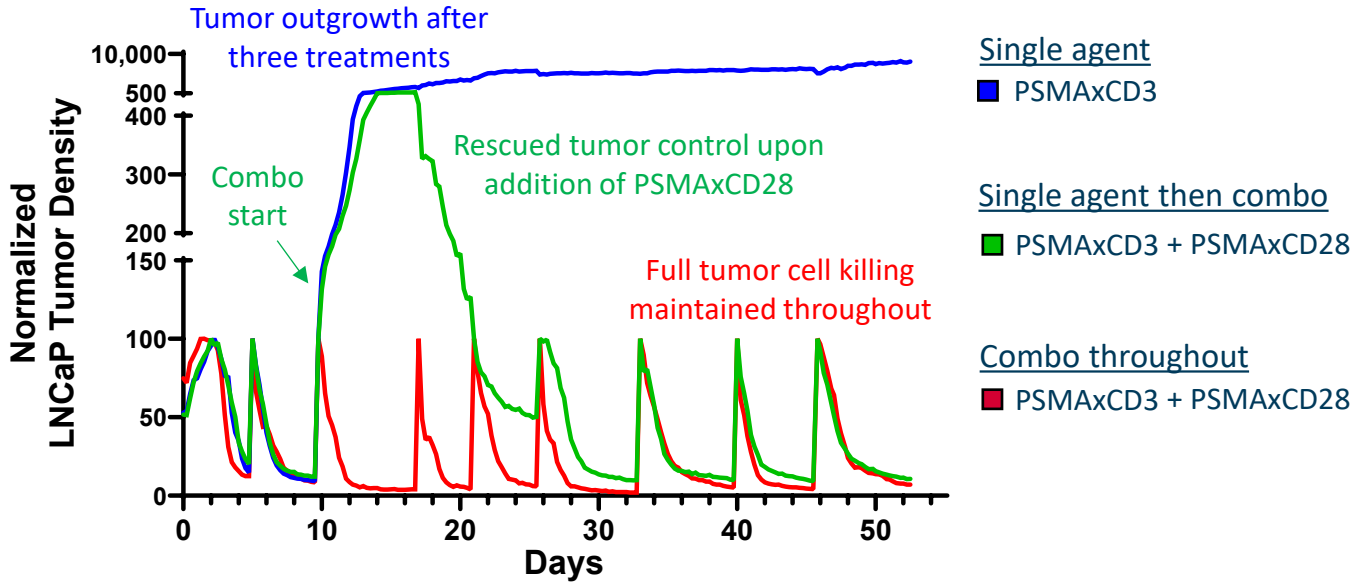
Assay designed to drive rapid T cell exhaustion



Learnings from clinical TCE programs†

Loss in T-cell function during dosing period and upon disease progression has been reported for multiple oncology clinical programs (Blinatumomab, teclistamab, and talquetamab)

CD28 co-stimulation improves duration of T cell mediated cytolytic activity



Non-clinical durability data supports plans to combine PSMA-TRACTr and PSMA-TRACIr in prostate cancer patients

PSMA-TRACIr enhanced duration of the PSMA-TRACTr anti-tumor response highlights the opportunity to further differentiate a potentially best-in-treatment therapy in prostate cancer

†Verkleij et al 2024; † Philipp et al 2022

PSMA-TRACIr profile in cynomolgus monkey

Well tolerated as a single agent or in combination with PSMA-TRACTr

No adverse findings in cynos

Non-masked PSMA-TCE

Transient cytokine induction and elevation in liver enzymes

PSMA-TRACTr

Transient cytokine induction at high doses
No adverse clinical signs or pathology changes

PSMA-TRACTr + PSMA-TRACIr

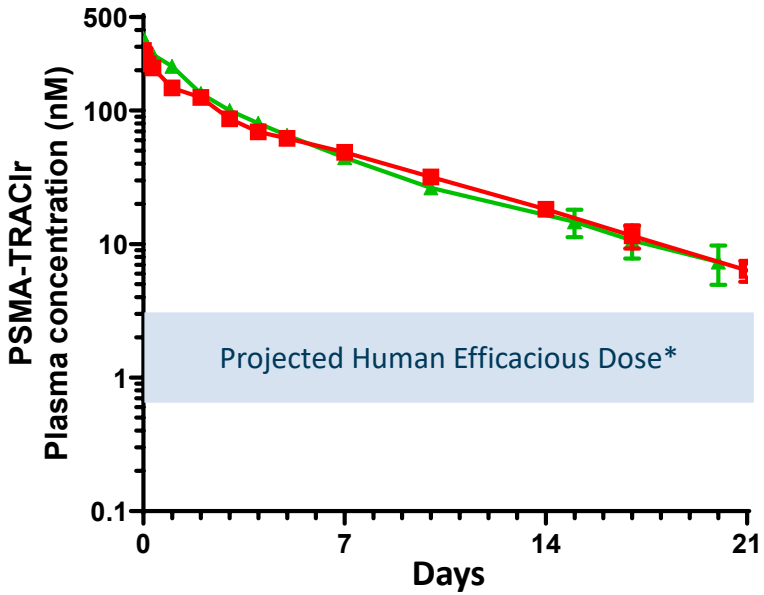
No enhancement of cytokine release, no adverse clinical signs, no observable healthy tissue toxicities

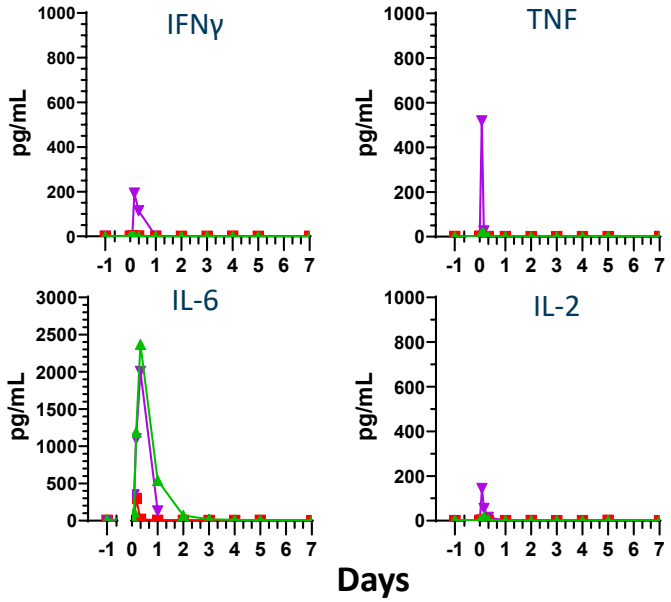
Large safety window in NHPs

Single agent TRACIr: 1mg/kg IV PSMA-TRACIr

Combination TRACTr + TRACIr: 1mg/kg IV PSMA-TRACIr + Active dose PSMA-TRACTr

Single agent TRACTr: Active dose PSMA-TRACTr





Combination PSMA-TRACTr and PSMA-TRACIr exhibits large safety window above anticipated efficacious doses

*Based on in vitro MABEL in combination with PSMA-TCE

First patient treated with PSMA TRACIr in combination with JANX007 planned in 2H-2026

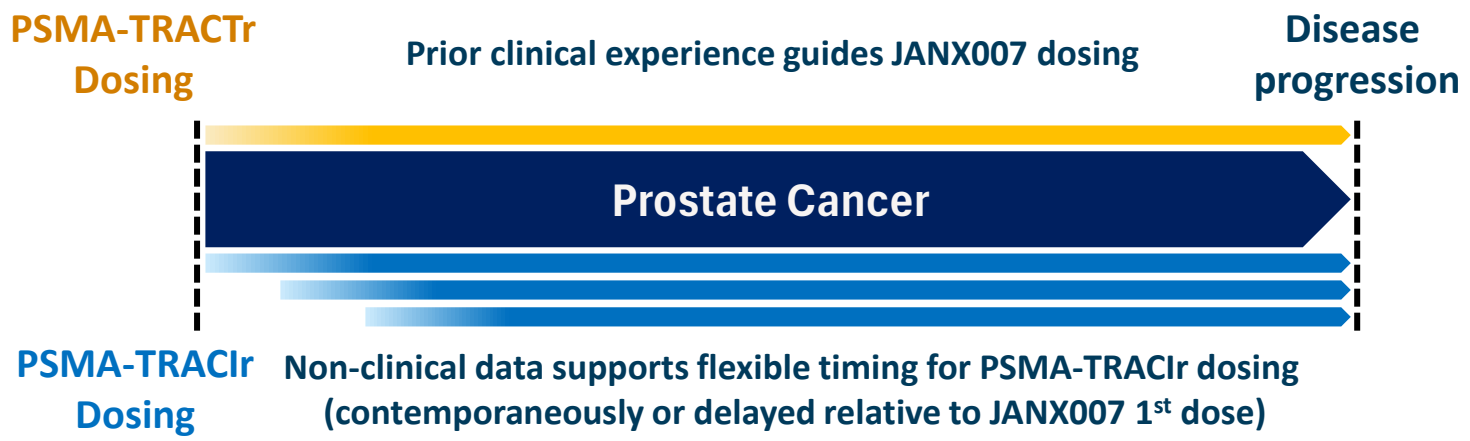
IND enabling studies in progress

IND in 1H-2026

IND enabling studies

- Non-clinical toxicology in NHP
- *In vitro* pharmacology and immunotoxicity safety assessments
- GMP manufacturing
- IND anticipated in 1H-2026

Phase 1 planned in 2H-2026



Key Phase 1 Evaluations

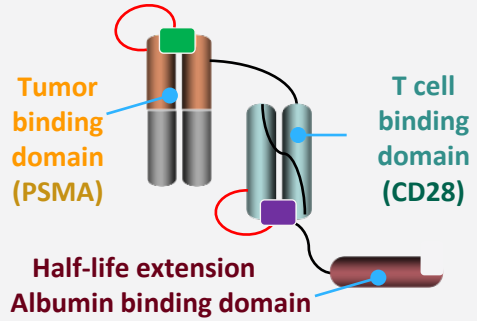
- PK, safety, and tolerability
- PSA, RECIST, durability
- TRACIr platform validation
- Dose regimen finding
- T cell expansion and phenotype

PSMA TRACTr expertise and experience expected to accelerate PSMA TRACIr proof of concept

Janux positioning to strengthen its prostate cancer franchise

Opportunity to raise the bar again in prostate cancer with a TRACTr + TRACIr combination

Clinically validated design principles



PSMA-TRACIr

For treatment of PSMA expressing solid tumors in combination with TRACTrs

Combination with potentially best-in-class JANX007

- JANX007 exhibits predictable dose dependent clinical activity*
- Enhancement in durability from CD28-costimulation demonstrated in non-clinical studies
- Large safety window in NHP at combination exposures well above those anticipated to drive efficacy in humans

Best-in-treatment opportunity

- Prostate cancer is a known PSMA-TRACTr and PSMAxCD28 responsive tumor type
- Potentially stronger responses with prolonged durability
- Reduced risk of CRS and healthy tissue toxicity by design

Rapid TRACIr development path

- JANX007 experience may facilitate accelerated clinical development to enable rapid TRACIr PoC
- FPI planned in 2H-2026

PSMA-TRACIr comprises clinically validated design principles with a rapid path to clinical proof of concept

*Note: "Clinically validated" refers to early clinical validation of TRACTr platform from JANX007 clinical data aligning with the platform design principles and expectations *(based on Dec-2024 and May-2025 program public updates).*

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FPI = First Patient In

TROP2-TRACTr

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TRACTr platform unlocks opportunity for a potential first-in-class TROP2-TCE

Expansion into a high value target opportunity via clinically validated TRACTr platform

Target Selection Rationale – Strategic Focus

1. Leverage learnings from JANX007 and JANX008 clinical programs
2. Accelerate TRACTr development where targeted agents have had clinical success
3. Access new indications across Janux portfolio

Competitive Differentiation

- Designed for improved safety and efficacy vs. ADCs (73% Gr≥3 TEAEs for Trodelvy‡)
- Response potential across full spectrum of TROP2 expression densities (including low expression)
- Designed to enable higher doses, higher intratumoral levels of active drug, and stronger efficacy

High unmet need – Market Potential

TROP2+ tumors - 2L+ incidence USA & EU5ⴖ

NSCLC	209K	OC	25K
HR+ BC	60K	UC	14K
HER2+ BC	25K	CC	14K
TNBC	20K	EC	8K
SCLC	19K	ESCC	4K

>400K treated patients annually
Multi-billion-dollar market potential

TROP2-TRACTr leverages clinically validated platform and target to access new tumor types

‡Bardia 2024; ⴖCompetitive intelligence from company reports and publicly available epidemiology data including SEER.

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TROP2-TCEs likely require tumor-activated approach

Contemporary TCEs unable to access TROP2 target due to broad healthy tissue expression

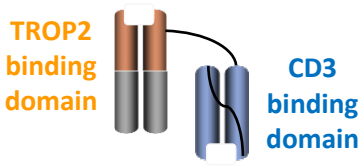
Healthy tissue expression limits contemporary TCEs

- Broad TROP2 expression in skin, kidney, bladder, parotid, thyroid, salivary gland, liver, pancreas
- Poses substantial dose limiting toxicity risk
- TROP2 expressed in overlapping healthy & tumor tissues

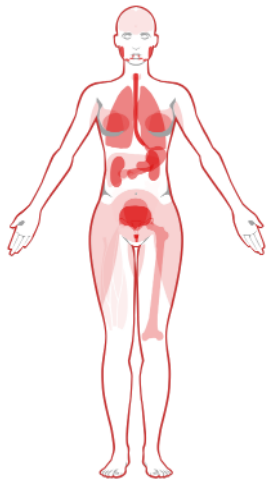
TRACTr designed to reduce healthy tissue toxicity

- Masking to block target engagement and activity in healthy tissue
- Proteolytic cleavage to focus activation to TME
- Switch PK mechanism to enable rapid clearance of active drug once cleared from TME

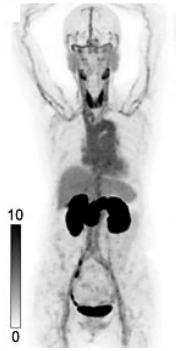
TROP2-TCE



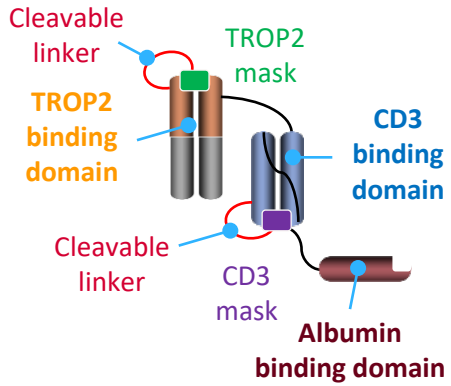
Active wherever TROP2 is expressed

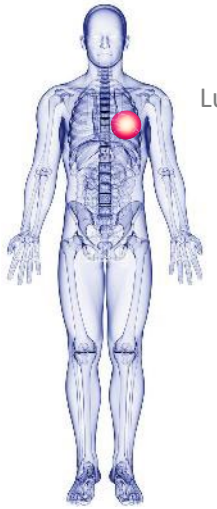


TROP2-PET imaging of healthy volunteers*



TROP2-TRACTr





Lung cancer example

Activity restricted to TROP2+ TME

TROP2-TRACTr designed to reduce CRS and healthy tissue toxicity limitations of conventional TCEs

**Chen et al 2024*

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Optimized TROP2-TRACTr exhibits large activity multiples across tumor cell lines

TROP2-TRACTr exhibits a large functional potency shift relative to non-masked TCE

Skin Cancer: A431

600,000 TROP2/cell

Lung Cancer: H292

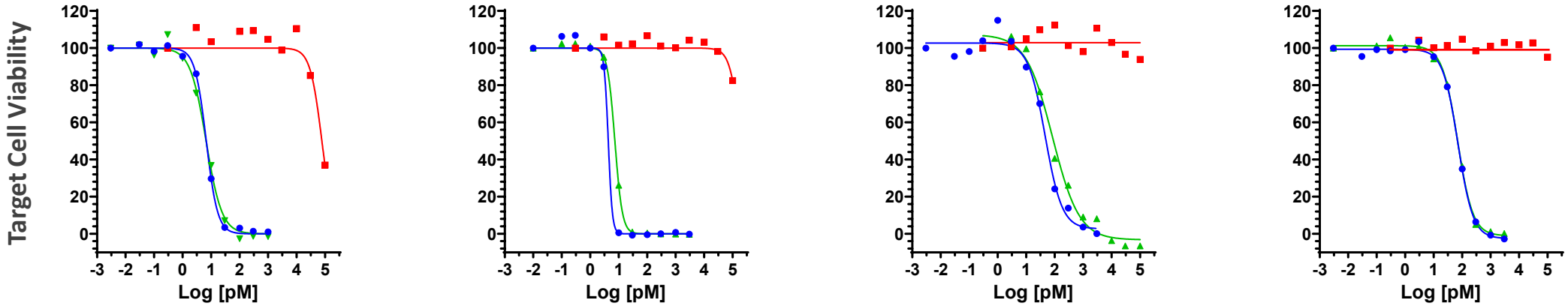
225,000 TROP2/cell

Breast cancer: MDAMB231

35,000 TROP2/cell

Colon cancer: HCT116

15,000 TROP2/cell



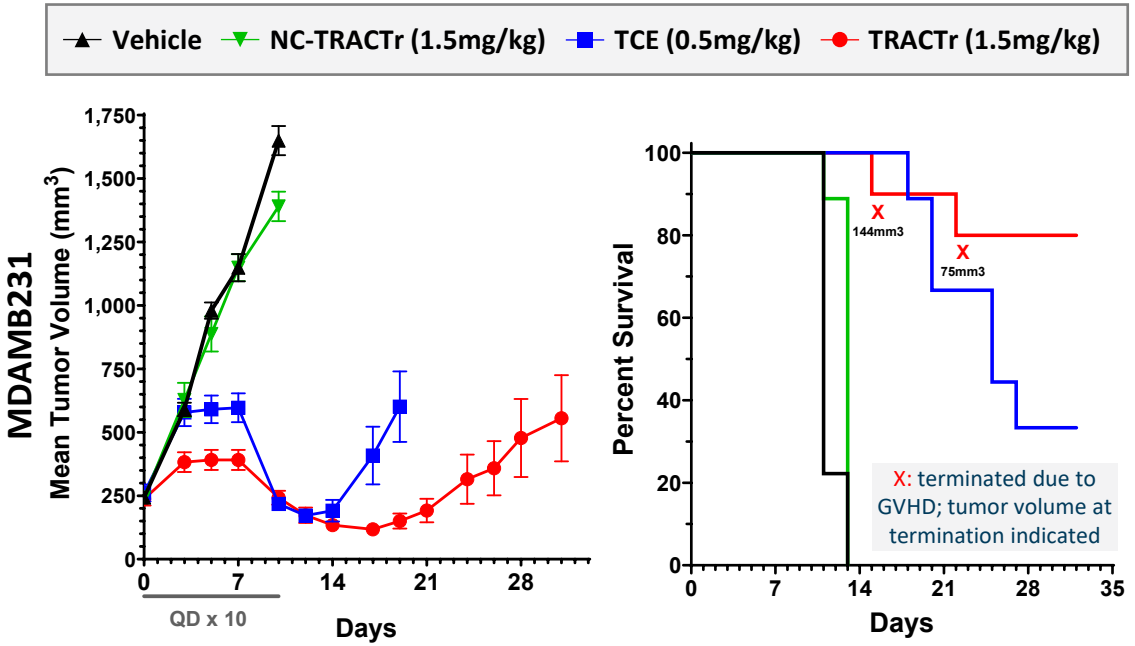
TRACTr provides opportunity to treat tumors with a wide range of receptor densities

Janux TROP2-TRACTr offers an opportunity to treat many solid tumors with high unmet need

TROP2-TRACTr active at low TROP2 expression density levels

Opportunity to treat patients across all levels of TROP2 expression

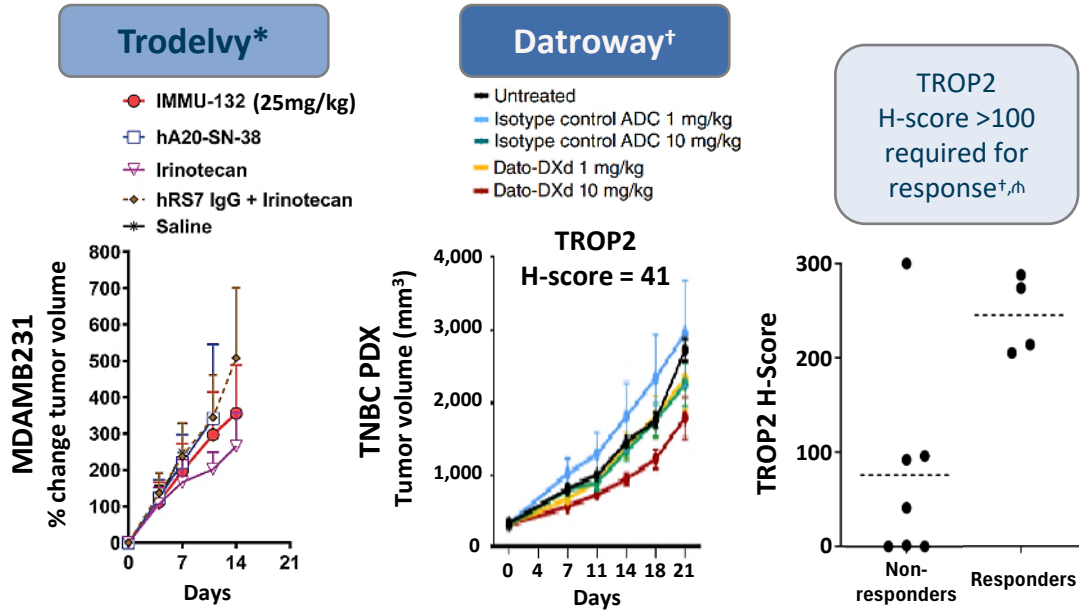
TROP2-TRACTr is active in low TROP2 expression tumor model



Cleavage dependent TROP2-TRACTr activity

TROP2-TRACTr demonstrates prolonged survival benefit

TROP2-ADCs exhibit minimal activity in low TROP2 expression tumor models



Trodelvy and Datroway exhibit minimal activity in tumor models and patients with low TROP2 expression (below ADC activity threshold)

Stronger TROP2-TRACTr anti-tumor activity at low TROP2-expression density differentiates from ADCs

**Goldenberg et al 2015; †Meric-Bernstam et al 2025; ᗰOkajima et al 2021*

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Outcomes in TROP2-ADC treated TNBC patients deteriorate if tumor TROP2 expression is low

TROP2-TRACTr aims to provide stronger benefit to patients with low TROP2 expression

TROP2 expression impacts ADC clinical benefit in TNBC‡

TROP2 Expression (H-Score)	% Patients	Number Patients	Treatment	mPFS (mo)	mOS (mo)	ORR	CR + PR
VERY LOW < 50	13%	21	TPC	Not reported	Not reported	10%	0
		20	SG			15%	3
LOW 0-130	25%	45	TPC	1.5	7.0	4%	2
		35	SG	2.7	8.7	23%	8
MEDIUM 130-220	25%	33	TPC	2.8	8.8	15%	5
		47	SG	4.8	13.4	28%	13
HIGH 220-275	25%	40	TPC	1.6	6.5	0%	0
		39	SG	6.8	15.2	41%	16
VERY HIGH 275-300	25%	32	TPC	2.8	7.1	0%	0
		47	SG	6.9	14.5	45%	21

Treating physician's choice (TPC)

TROP2-ADC: Sacituzumab-govitecan (SG; Trodelvy)

Reduced efficacy in 50% of TNBC patients (H-score ≤ 220) treated with TROP2-ADC‡

- TROP2-TRACTr *in vivo* anti-tumor activity requires less TROP2 expression than ADCs
- Opportunity for TROP2-TRACTr to improve efficacy where ADCs are limited by reduced TROP2 target expression

Opportunity for TROP2-TRACTr in patients with otherwise limited clinical response due to low TROP2 expression

‡Bardia et al 2024

Janux

TROP2-TRACTr is well tolerated in NHP

TRACTr reduced CRS and healthy tissue toxicity compared to non-masked TROP2-TCE

Masking prevents toxicity in NHP

Non-masked TROP2-TCE

Adverse clinical signs consistent with CRS and healthy tissue toxicity:

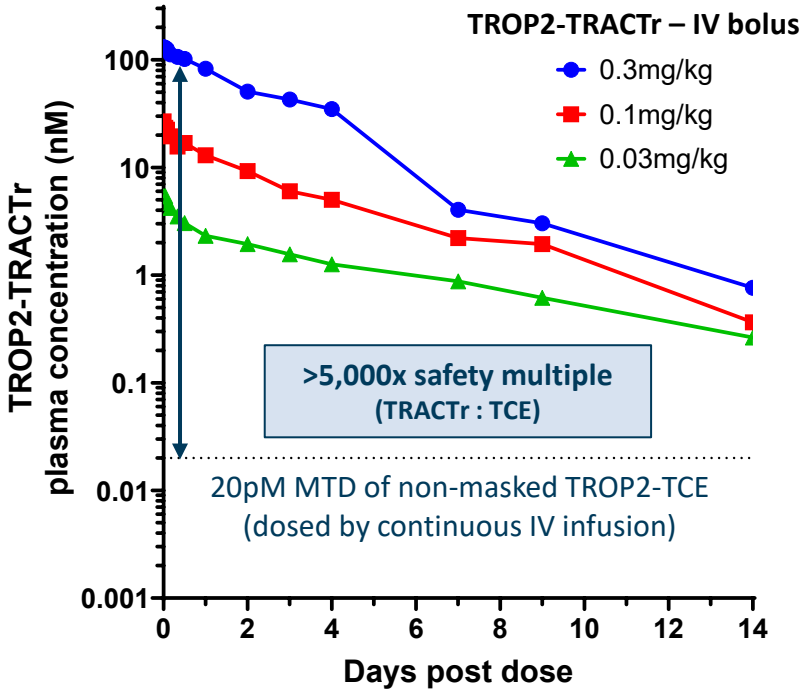
Fever, neurological effects

GI, skin, kidney toxicity

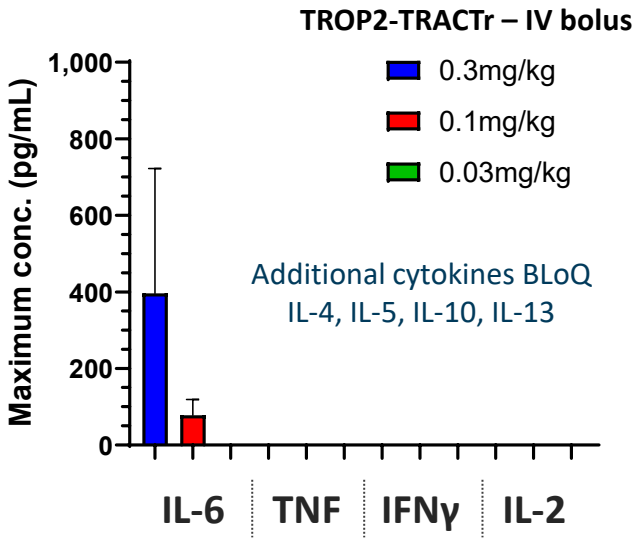
TROP2-TRACTr

No adverse clinical signs or pathology changes consistent with a lack of measurable healthy tissue toxicity

TROP2-TRACTr large safety multiple



Minimal cytokine release



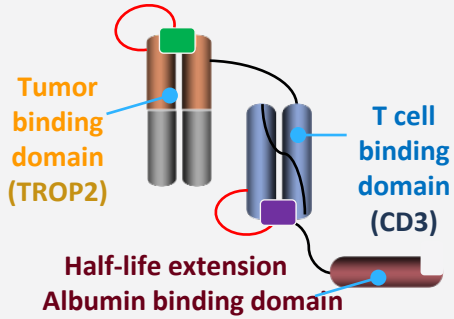
No fever, no clinical signs of CRS

Large safety window may enable increased dose and higher intratumoral drug concentrations for stronger efficacy

Janux well-positioned to access high-value target with clinically validated TRACTr platform

Opportunity for First-In-Class TROP2 targeted TCE

Clinically validated TRACTr platform



TROP2-TRACTr

for the treatment of TROP2 expressing solid tumors

Robust Non-clinical Safety and Activity

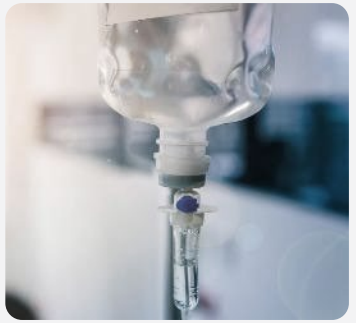
- Activity across full spectrum of TROP2 expression densities and tumor types
- *In vivo* activity demonstrated where TROP2-ADCs have failed
- Large safety multiple in NHP

First-In-Class TROP2-TRACTr Opportunity

- Reduced risk of CRS and healthy tissue toxicity
- Potential stronger efficacy and broader clinical benefit for patients across all levels of TROP2 expression over ADCs
- Add new indications with unmet need into Janux portfolio

Rapid TRACTr Development Path

- Learnings from JANX007 and JANX008 help accelerate development
- Previously deployed strategies have enabled rapid clinical POC
- IND-enabling activities planned in 2H-2025



Janux TROP2-TRACTr first-in-class opportunity combined with potential advantages over ADCs

Adaptive Immune Response Modulator (ARM) Platform

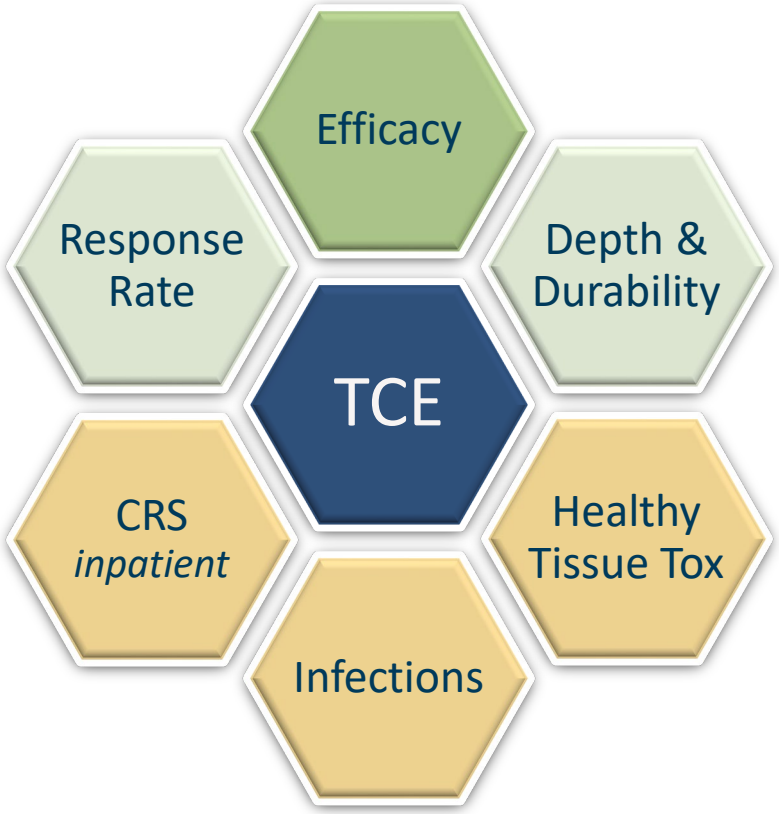
Redesigned bispecific T-cell engagers

Janux

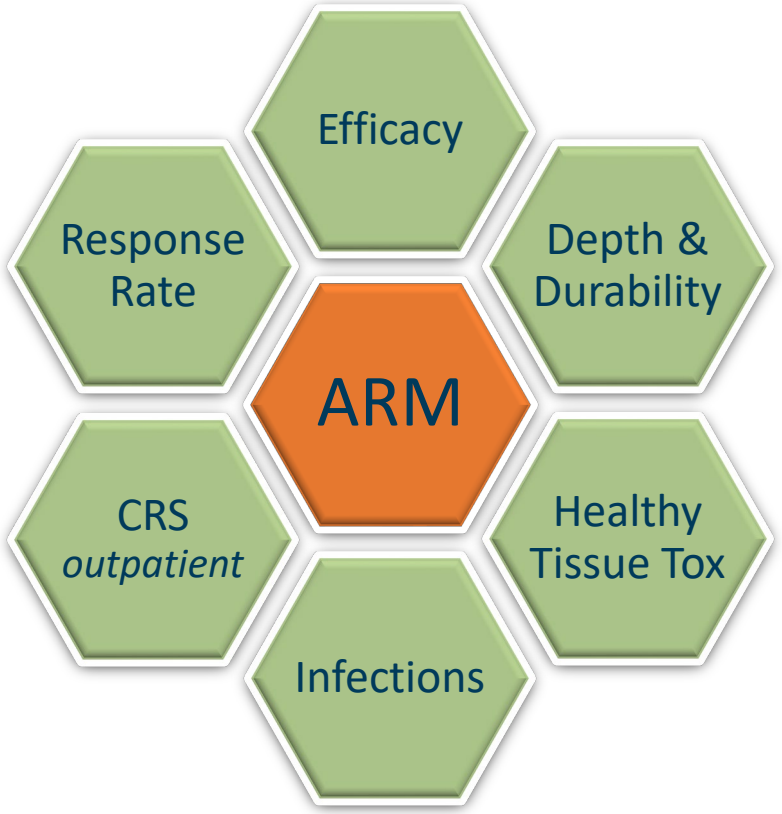
Reimagining T-cell engagers to overcome TCE limitations for improved patient treatments

Janux ARMs are designed to improve efficacy and safety of TCEs in oncology and I&I

Contemporary TCEs



Janux ARMs

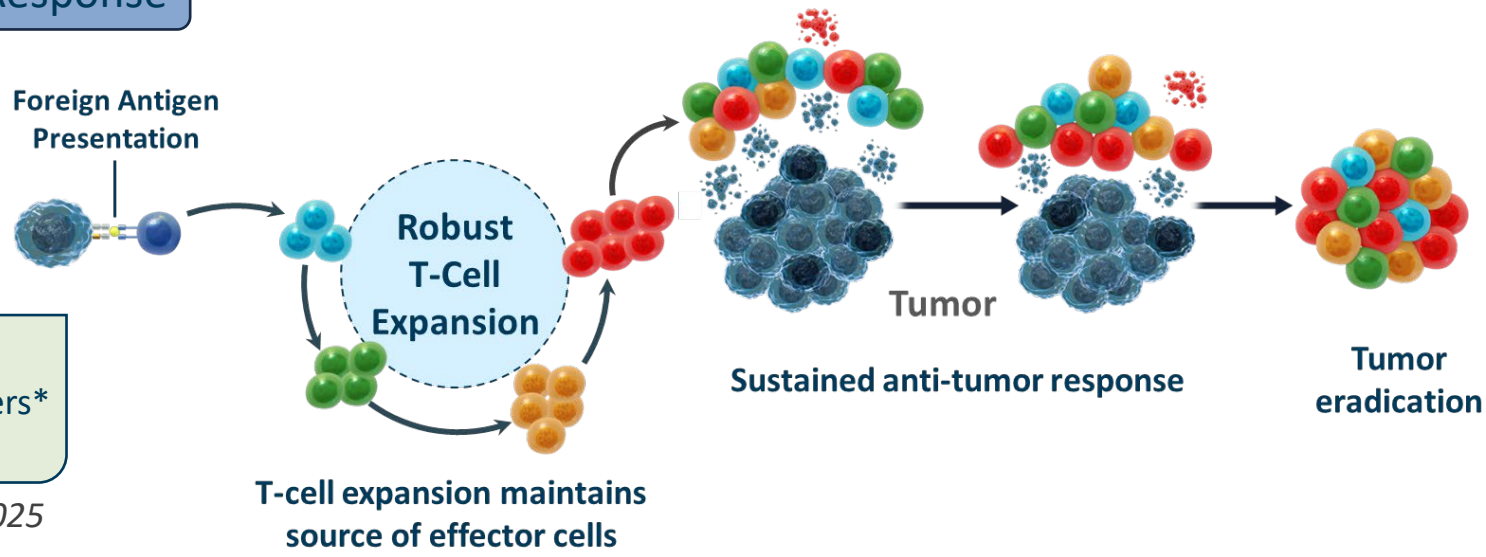


Janux ARM platform is designed to address key deficiencies of contemporary TCEs

Robust T-cell expansion is a key mediator of successful anti-tumor immune responses

Expanded memory reservoir replenishes effector cells to maintain immune response

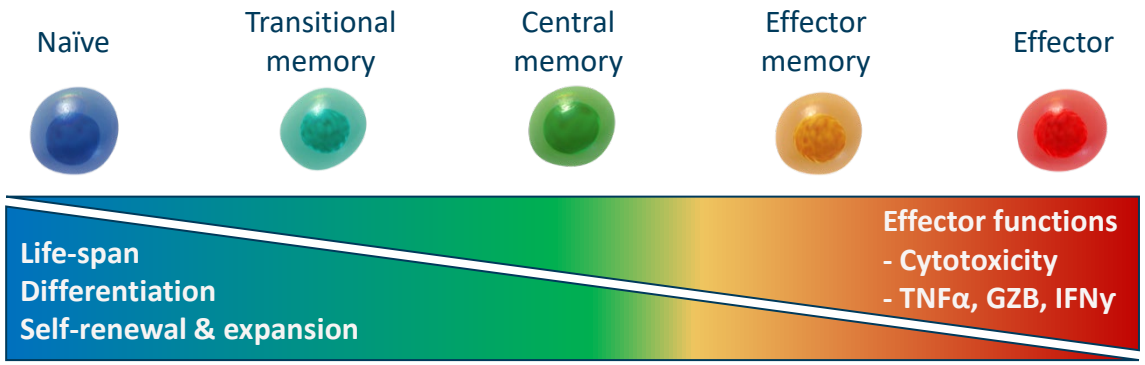
Adaptive T-Cell Immune Response



- Tumor clearance
- Checkpoint antibody responders*
- CAR-T responders**

**Reiner 2025, **Dreyzin 2025*

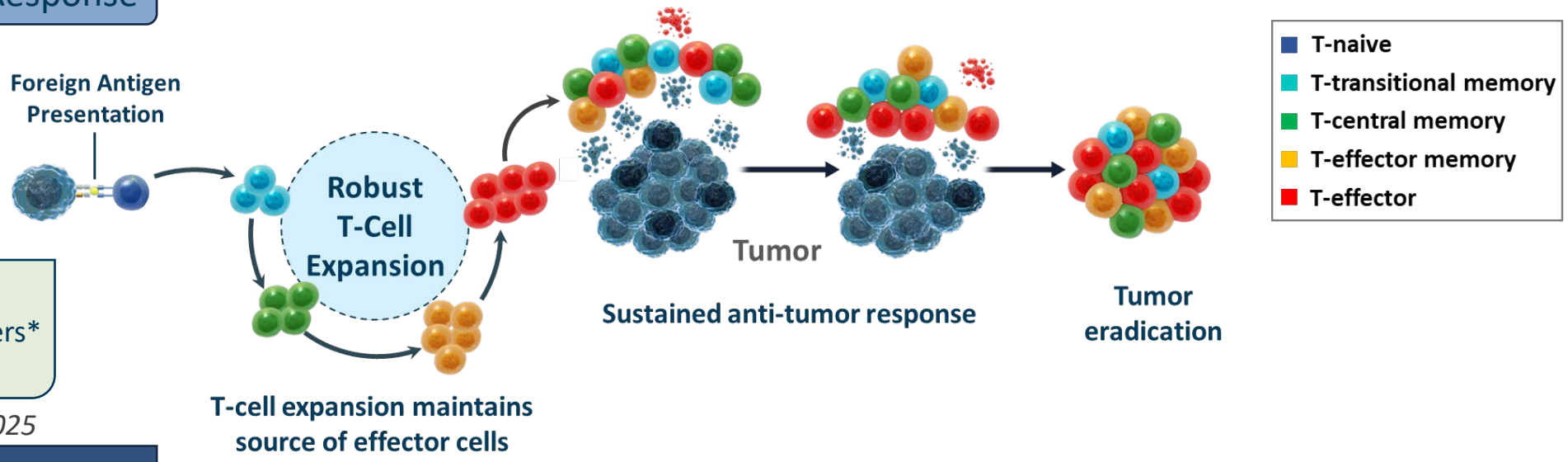
T-Cell Subsets and Functions



Robust T-cell expansion is a key mediator of successful anti-tumor immune responses

Expanded memory reservoir replenishes effector cells to maintain immune response

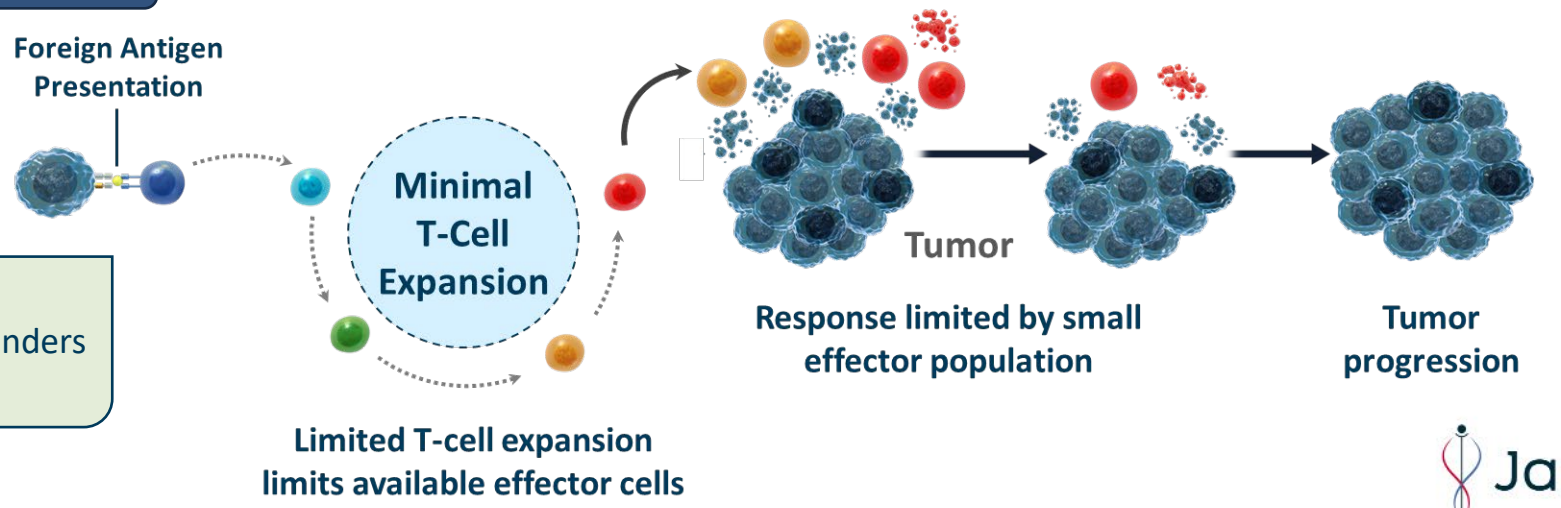
Adaptive T-Cell Immune Response



- Tumor clearance
- Checkpoint antibody responders*
- CAR-T responders**

**Reiner 2025, **Dreyzin 2025*

Ineffective Immune Response

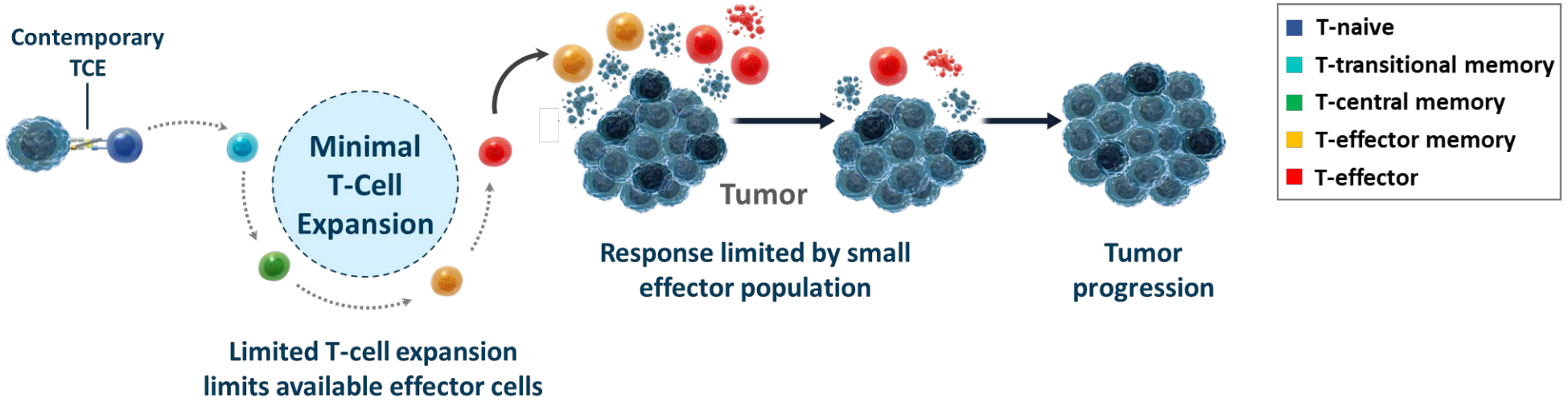


- Progressing tumors
- Checkpoint antibody non-responders
- CAR-T non-responders

Janux

Recent clinical studies have shown that TCEs elicit minimal T-cell expansion in heme-onc and AID patients

TCE T-Cell Immune Response



- Decline in cytolytic function has been reported in ALL patients treated with Blincyto (*Ma 2024*)
  - Naïve, central memory, effector memory, and effector subsets were unchanged following treatment
- Temporary disease improvement has been reported for multiple TCEs in AID patients (*EULAR 2025*)
  - Naïve and central memory subsets were unchanged following Teclistamab treatment (*Bucci, Schett EULAR 2025*)
- Reduced B-cell depletion after treatment with Blincyto, Talquetamab, and Teclistamab has been observed in multiple clinical trials (*Verkleij 2024, Philipp 2022*)

Key question – can a bispecific T-cell engager be redesigned to improve T-cell expansion, depth and durability of response?

ARM demonstrates comparable potency with reduced cytokine release compared to TCE

Healthy human PBMCs – similar data with CD4 T-cells

B-Cell Depletion

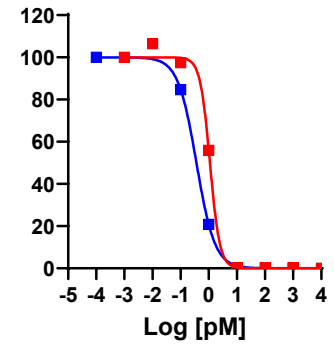
T-Cell Expansion

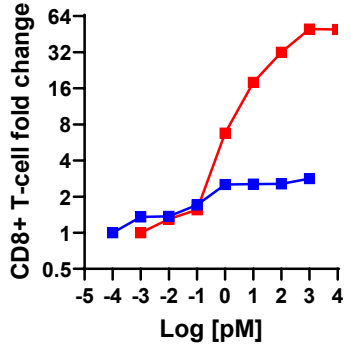
Cytokine Release

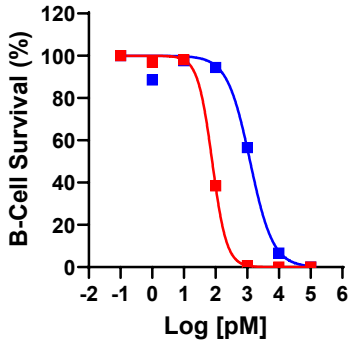
CD19 bispecifics

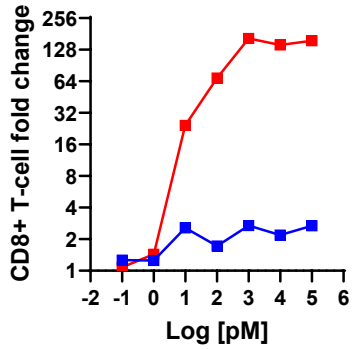
CD20 bispecifics

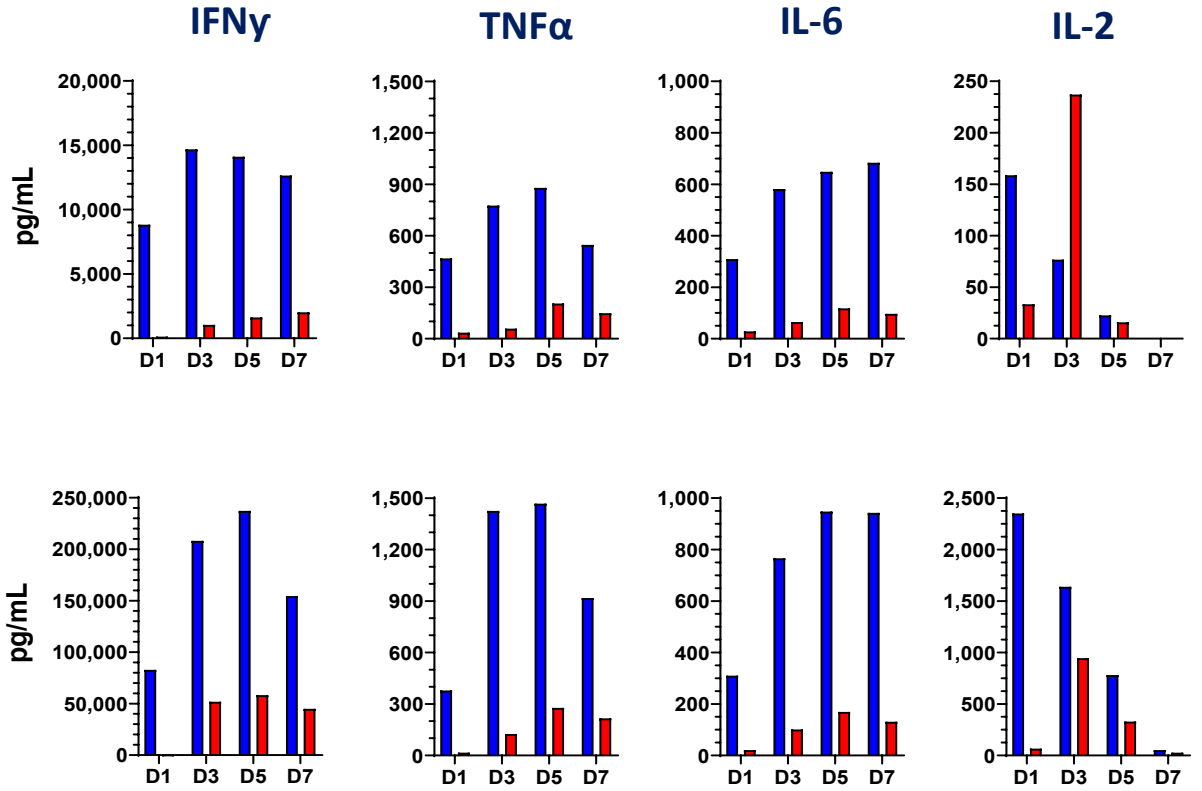
ARM TCE











Complete B cell killing

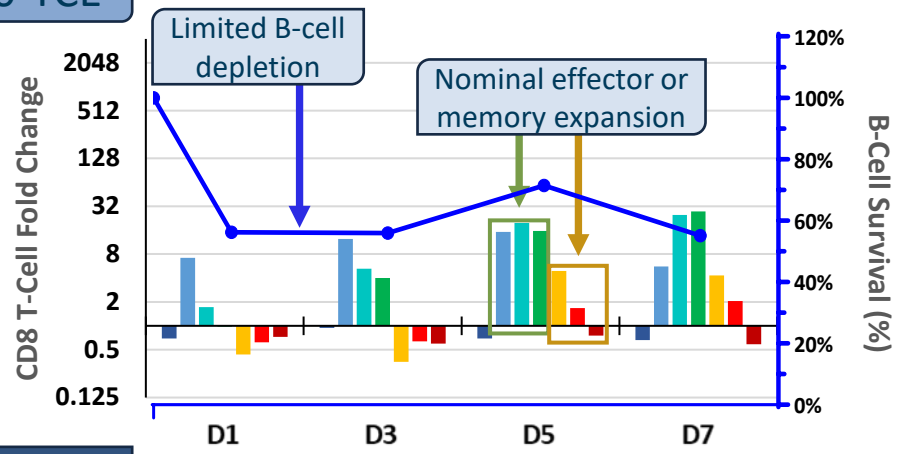
ARM improved expansion

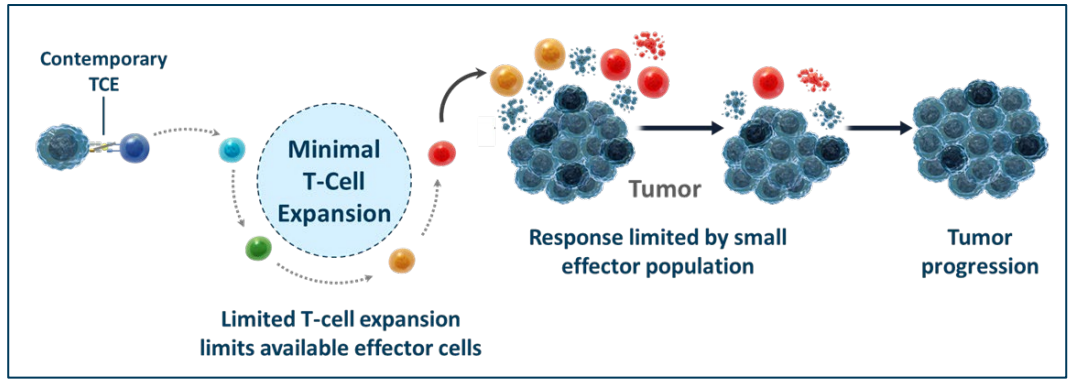
Reduced cytokine release with ARM

Comparison of TCE and ARM CD8 profiles and B-cell depletion

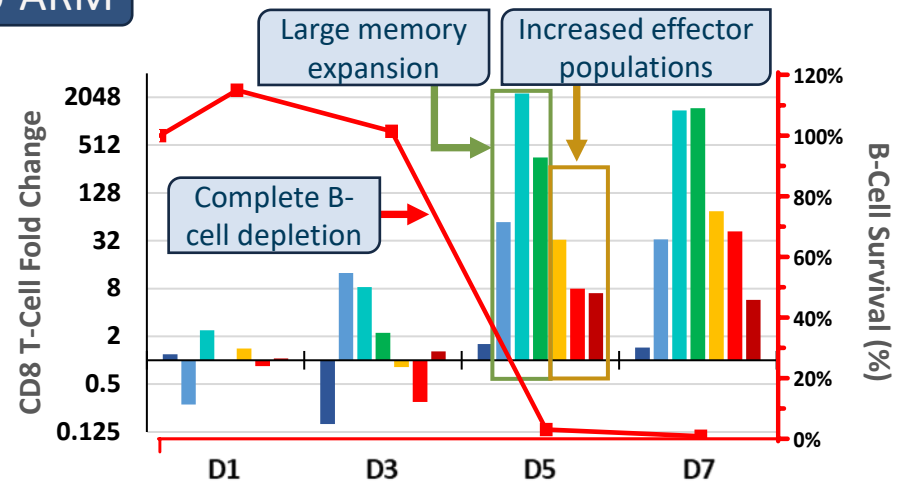
TCE treatment reduces effector populations which limits B-cell depletion

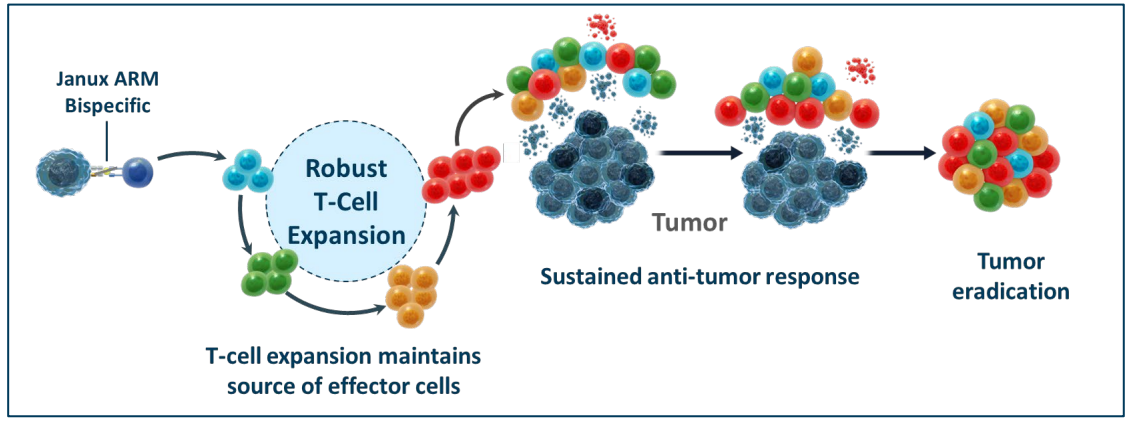
CD20-TCE





CD20-ARM





- Tn
- Tscm
- Ttm
- Tcm
- Tem
- Teff
- Temra

ARM expands memory populations and maintains effector populations for complete B-cell depletion

ARM demonstrates improved duration of T cell function compared to a TCE

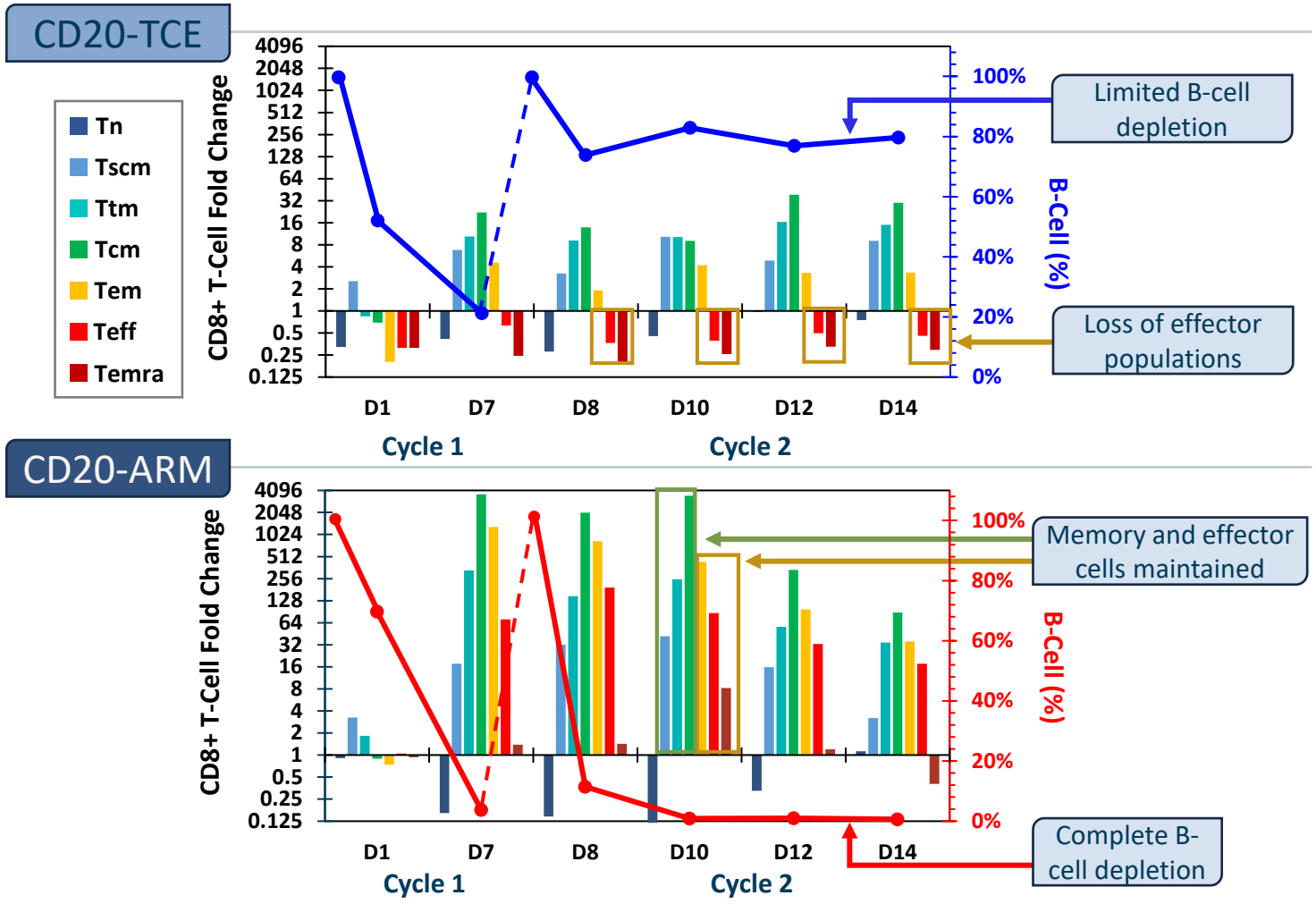
Provides potential to improve clinical durability and reduce infection risk

Learnings from clinical TCE programs*

- Loss in T-cell function during dosing period has been reported for multiple oncology clinical programs
  - Blincyto, teclistamab, and talquetamab
- Limited response durability in AID patients with Teclistamab and Blincyto reported

ARM durability advantage

- ARM maintains T-cell function
  - Improved durability of response compared to a TCE
  - Should decrease exhaustion mediated infection risk
- Progressive loss of T cell function with TCEs is similar to that reported in the clinic



ARM expands memory populations and maintains effector populations for a durable B-cell depletion response

*Philipp 2022, Verkleij 2024; Schett EULAR 2025

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CD19-ARM to Treat AID Patients

Lead Program – Planned FIH Phase 1 for 1H2026

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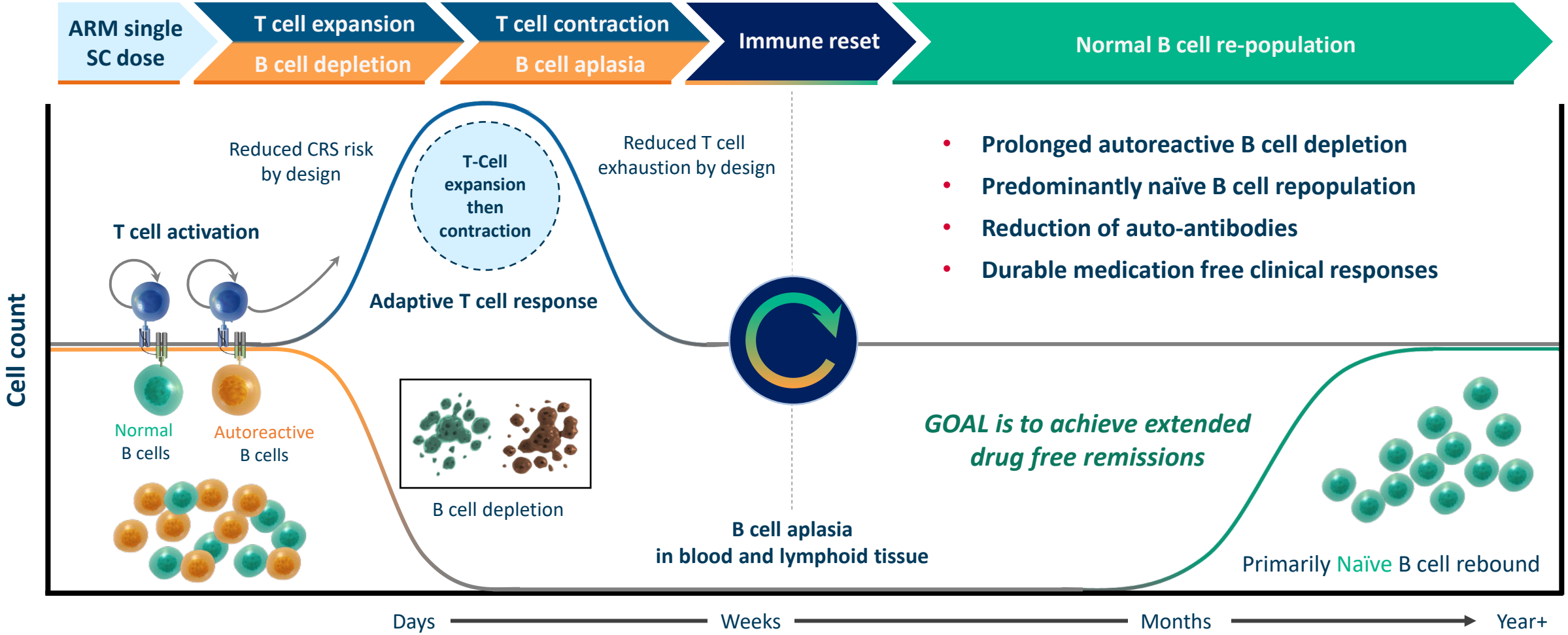
CD19-ARM aims to reset the immune system of autoimmune disease (AID) patients

Deep and rapid B cell depletion from blood and tissue followed by naïve B cell re-population



CD19-ARM designed to drive prolonged drug free remissions as a fully off the shelf re-dosable compound in AID

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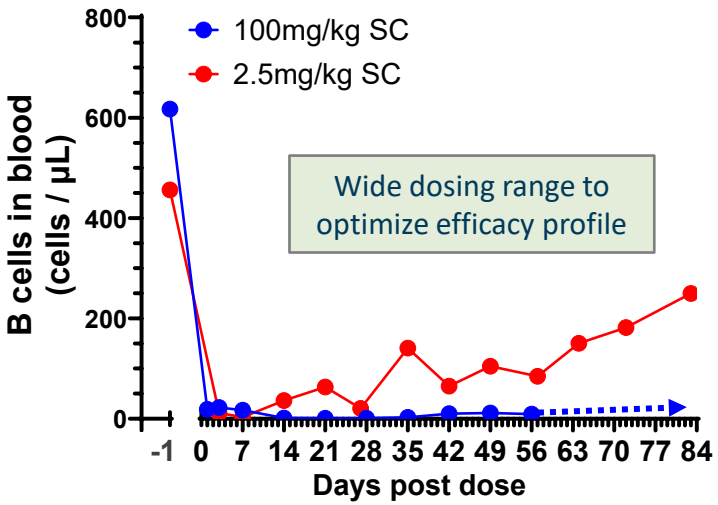
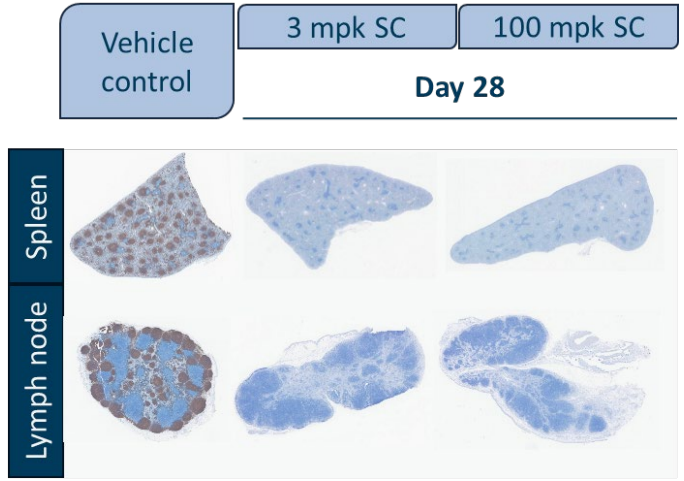
Deep B-cell depletion in periphery and tissues with a prolonged memory cell reset

Single dose NHP results support an extended dosing interval in autoimmune patients

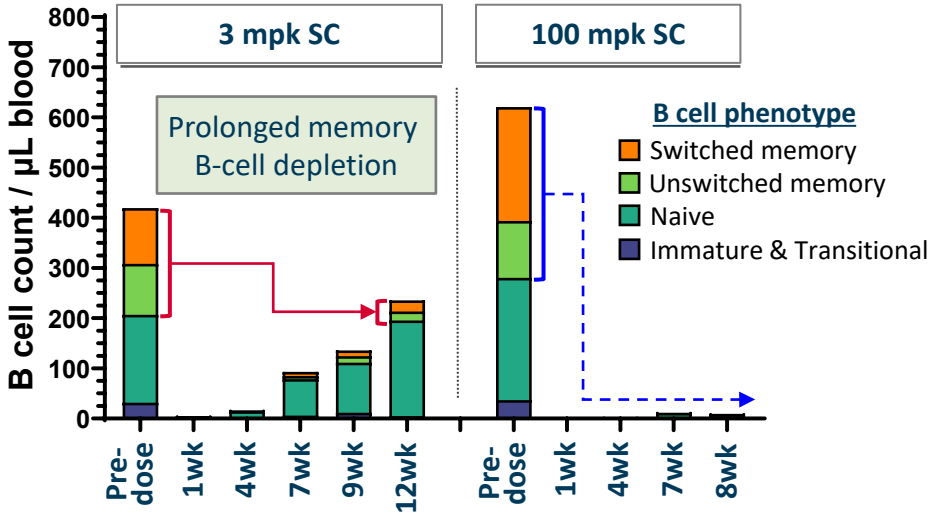
Rapid B-Cell Depletion



Deep Tissue B-cell Depletion



Durable Memory B Cell Reset



Learnings from CD19-CAR T studies in AID patients

- B-cell depletion within 1st month
- Median of 3-months for naïve B-cell repopulation
- Drug free remissions of >2-years have been reported
  - However, AID patient flares have recently been described*

CD19-ARM efficacy profile is similar to CD19-CAR T

- Rapid B-cell depletion occurs within days of dosing
- Primarily naïve B-cell repopulation during first 3-months
- Extended memory population reset
- Ability to re-treat with a single-dose to address disease flares

CD19-ARM provides a wide B-cell depletion dosing range to optimize desired efficacy profile

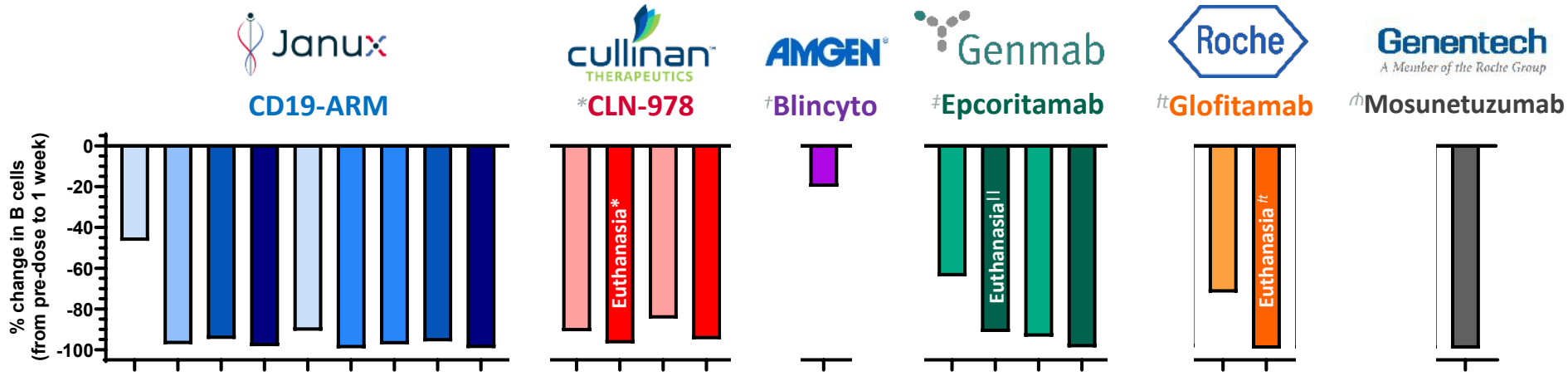
**EULAR 2025*

CD19-ARM has best-in-class safety multiple in NHP relative to contemporary TCEs

ARM overcomes CRS limitations of contemporary TCEs

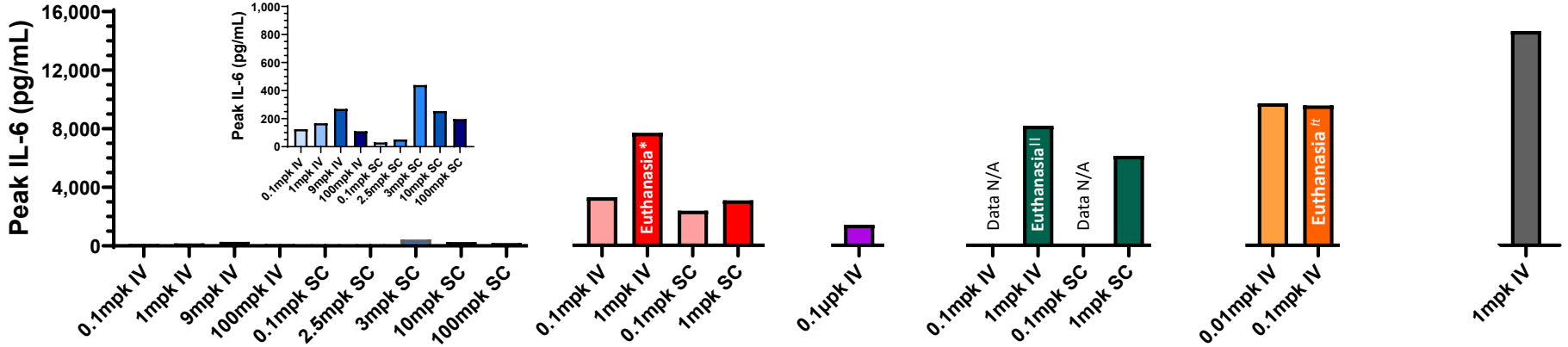
B-Cell Depletion

Competitor's B-cell depletion depth is limited by CRS dosing constraints



Cytokine Release

CD19-ARM provides a ≥100x CRS safety multiple to enable maximum efficacy



CD19-ARM potential best-in-class profile combines efficacy without CRS to enable outpatient treatment opportunity

**Meetze 2023; †BLA-125557; ‡Engelberts 2020; ‖BLA-761324; ᵑSun 2015; ᵑBLA-761263; ǂǂFrances 2022; ǂǂBLA-761309*

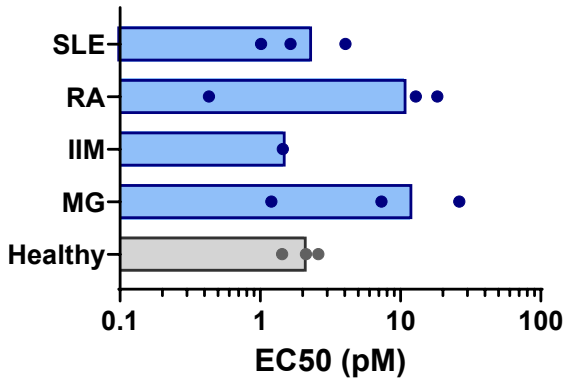
Information provided above is for illustrative purposes only and is not a head-to-head comparison.

Janux

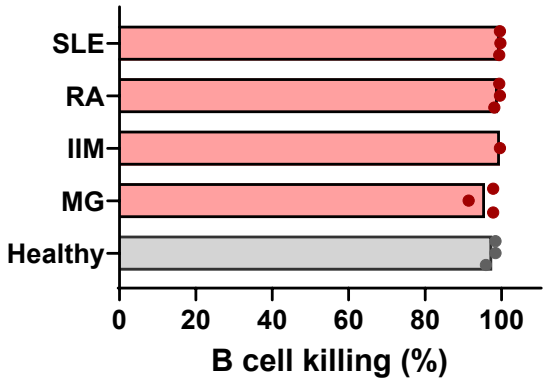
CD19-ARM demonstrates activity in multiple autoimmune patient samples

Consistent B-cell depletion in disease patient PBMCs

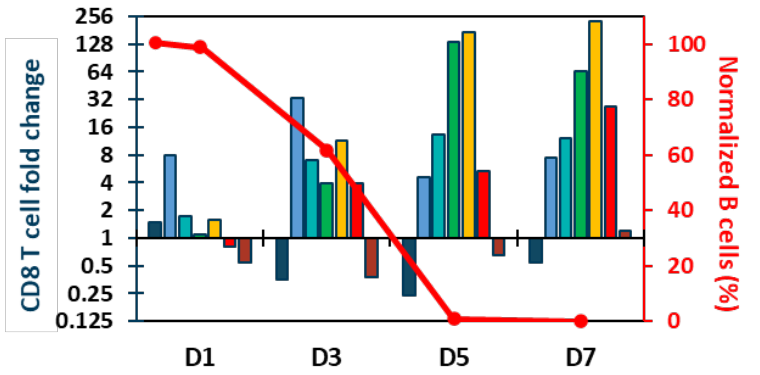
Potent B-Cell Depletion



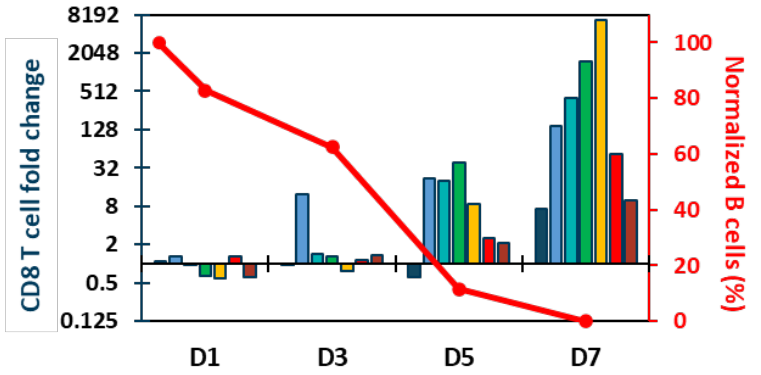
Full B-Cell Depletion



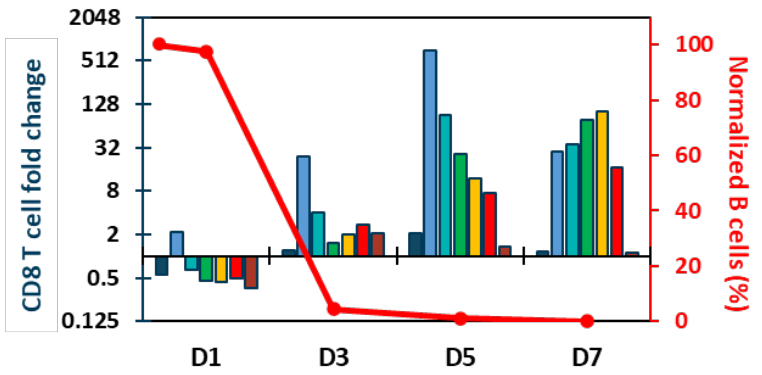
Systemic Lupus Erythematosus (SLE)



Rheumatoid Arthritis (RA)



Myasthenia Gravis (MG)

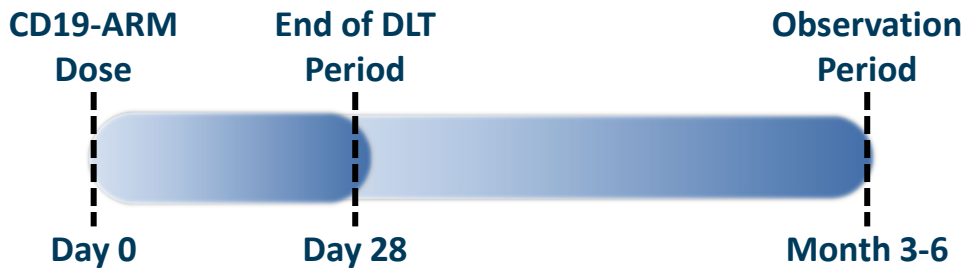


ARM provides opportunity for deep / durable responses in multiple autoimmune indications

Phase 1 study design - first patient treated in HNV trial planned in 1H2026

Healthy Normal Volunteer (HNV) Study

Rapidly understand B-cell depletion and CRS risk



Autoimmune Patient Study

Key Phase 1 Evaluations

- Safety and tolerability
  - CRS/ICANS
- Dose finding
- Pharmacokinetics
- T-cell expansion
- B-cell depletion, immune reset and durability
- Autoantibody serology
- ARM platform validation

HNV study to rapidly understand B-cell depletion and CRS risk, AID study to evaluate multiple indications

Key CD19-ARM developments

Clinical Development

- CD19-ARM planned FIH Phase 1 for 1H2026
- Regulatory filing planned for 4Q2025
- GMP bispecific antibody manufacturing in-progress

Human Dose Projection

- Human efficacious dose projection range is 0.07-0.17 mg/kg SC (5-12 mg total dose)
  - Based upon the fully efficacious dose of 3 mg/kg SC in cyno
  - *CD19-ARM is more potent in human PBMCs compared to cyno PBMCs used to assess MABEL*

Dosing Convenience

- SC dosing coupled with lack of CRS highlights potential for community-based outpatient treatment
- Extended dosing interval without step-doses highlights ease-of-use advantage

ARM Platform

- ARM pipeline is advancing - BCMA, CD20, BAFFr, and trispecifics
- Solid tumor evaluations are in-progress

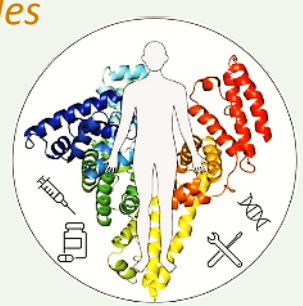
Janux is leading the development of novel TCEs that address efficacy and safety limitations

TRACTr and ARM platforms provide the opportunity to develop best-in-class drugs

Differentiated ARM Platform

Advantageous design principles

- Full cytolytic activity
- Reduced cytokine release
- T cell expansion, longer duration, less T cell exhaustion



Expertise At All Stages of Drug Development

- Demonstrated track record of non-clinical, manufacturing, and clinical TCE development
- Experienced leadership poised to drive best-in-class platform opportunity
- World renowned investigators excited to partner with Janux

CD19-ARM: Best-In-Class Opportunity

B cell depletion has proven clinical efficacy

- Reduced risk of CRS with outpatient/community potential
- Large safety window, high dose flexibility, deep B cell depletion with immune reset potential
- Durable T cell activity may reduce risk of infection

Rapid Path to Clinical PoC

- HNV study (planned 1H2026) accelerates PoC
  - B-cell depletion, safety, immune reset
- HNV PK/PD data informs AID study

CD19-ARM with potential best-in-class profile provides opportunity to match CD19 CAR-T efficacy as an off-the-shelf, outpatient therapy

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