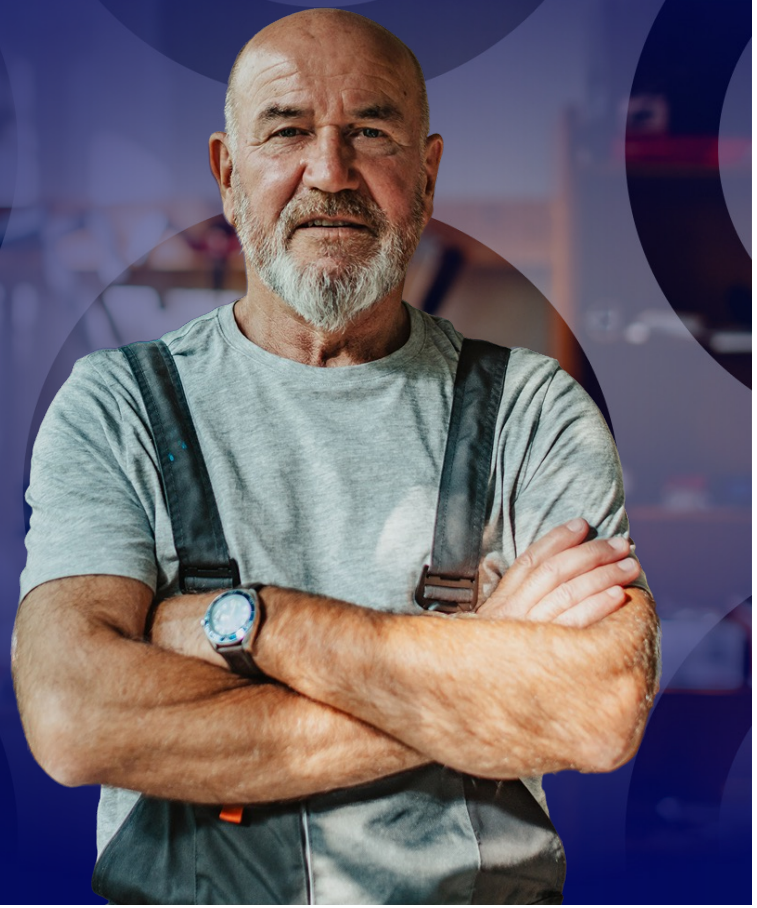
DAMORA THERAPEUTICS

Redefining care for people with blood disorders

Company Overview

August 2026



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Except for statements of historical fact, certain information contained herein constitutes forward-looking statements which include but are not limited to statements regarding: our business strategy, including our ability to develop best-in-class therapeutics to address the full mutant CALR-driven myeloproliferative neoplasm disease spectrum that meaningfully improve both efficacy and convenience compared to INCA033989; the efficacy, safety profile, dosing regime, convenience, and tolerability of DMR-001; Damora's ongoing and future clinical development activities, including the expected timing of Phase 1 clinical proof-of-concept data for DMR-001, and plans for and timing of investigational new drug applications for DMR-002 and DMR-003; estimated market sizes, potential growth opportunities, potential value creation and sample transactions; the length of time that the Company believes its existing cash resources will fund its operations, including expectations of cash runway; and management's assessment of future plans and operations which are based on current internal expectations, estimates, projections, assumptions and beliefs, which may prove to be incorrect. 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Damora Therapeutics: an emerging leader in blood disorders

DAMORA THERAPEUTICS

NASDAQ: DMRA

- **Founded by Fairmount Funds Management** with a mission to fundamentally redefine care for patients with blood disorders
- **Portfolio of mutCALR-targeted therapies with best-in-class potential** for myeloproliferative neoplasms (MPNs), including essential thrombocythemia (ET) and myelofibrosis (MF)
- Corporate and R&D leadership with a **proven track record of clinical and commercial success**
- **Premier investor support, with ~$541 million[1] in cash**, to accelerate lead asset DMR-001 through proof-of-concept and into registrational development

[1] *Cash and cash equivalents as of June 30, 2026.*

Rapidly advancing portfolio uniquely positioned to address the spectrum of patients across mutCALR-driven MPNs

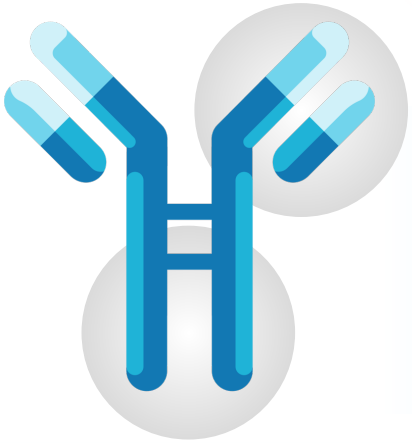
✓ *Phase 1/1b CLARITY-101 trial initiated*

DMR-001

Fc-null, extended half-life

Blocks mutCALR-driven oncogenic signaling, without engaging immune effector functions

Two clinical POC datasets: beginning mid-2027



DMR-003

T-cell engager

mutCALR x CD3 bi-specific recruits and directs T-cell-mediated killing of malignant cells

First regulatory submission: 2027

DMR-002

Fc-enhanced, extended half-life

Afucosylation enhances antibody-dependent cellular cytotoxicity and amplifies natural immune killing of malignant cells

First regulatory submission: 2H 2026

MPNs represent a spectrum of highly debilitating diseases

Essential Thrombocythemia (ET)

- High platelet counts **increase thrombosis and hemorrhage risk**
- Severe headaches, fatigue, mental fogginess, tingling in hands and feet, and other **symptoms negatively impact QoL**
- mutCALR patients are younger with **higher risk of transformation** to myelofibrosis
- **Available therapies do not treat underlying cause of disease** and have significant side effects (e.g., hydroxyurea)

Myelofibrosis (MF)

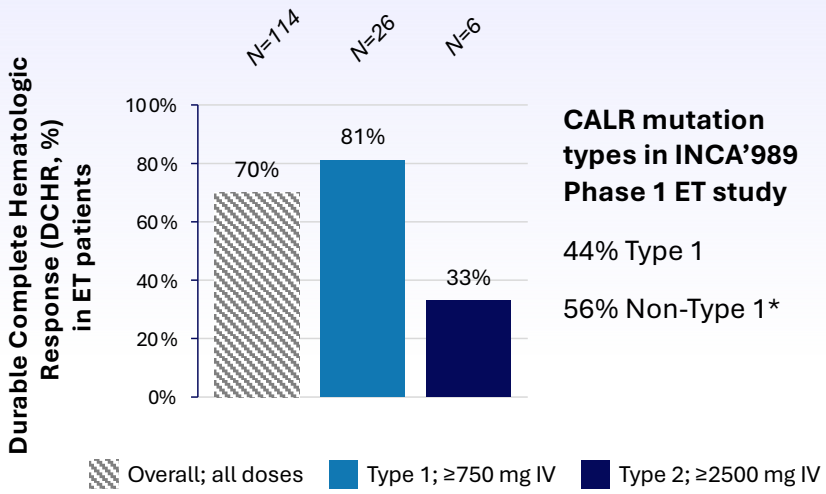
- Bone marrow fibrosis leads to **poor survival and high risk of transformation** to acute myeloid leukemia
- **Debilitating symptoms** include anemia, severe weakness and fatigue, splenomegaly, night sweats and bone pain
- **Available therapies do not treat underlying cause of disease** and largely only address symptoms

Significant need for highly effective, safe and convenient disease-modifying treatments

QoL, quality of life.
Sources: Tefferi 2025 (JAMA); Tefferi 2020 (Am J Hematol).

INCA’989 provided POC, with significant room for improvement

Less potent on non-Type 1 CALR mutations



CALR mutation types in INCA’989 Phase 1 ET study

44% Type 1

56% Non-Type 1*

Frequent, high-volume IV doses

Intravenous administration

Frequent dosing every two weeks

High-volume up to 2,500 mg

Different dose levels needed for Type 1 and Non-Type 1 patients, with dose escalation embedded in design of Phase 3 trial in ET

anti-mutCALR Fc-null antibody therapy was well-tolerated

INCA’989, INCA033989; IV, intravenous.

Sources: Mascarenhas 2026 (EHA). Complete response defined as platelet count below 400x10^9/L. Durable defined as maintenance of response for ≥12 weeks . Non-Type 1 mutations include Type 2 and Other CALR mutations.*

DMR-001 has two ways to win: maximize efficacy and meaningfully improve the patient experience

Superior potency and exposure

Next-generation therapy **engineered to maximize efficacy across all CALR mutations** through superior potency and optimized exposure levels

BROAD COVERAGE
Targets both Type 1 & Type 2 mutations

HIGH POTENCY
Potential best-in-class affinity

OPTIMIZED EXPOSURE
Sustained therapeutic levels

Potential **best-in-class** disease modification in ET and MF

Improve patient experience

Differentiation through **a patient-centric design, maximizing the experience on multi-year therapy** with a potential first-to-market, simple, and quick autoinjector

POTENTIAL FIRST TO MARKET
Autoinjector format

INFREQUENT SIMPLE ADMINISTRATION
Quick, low-burden delivery

LONG-TERM ADHERENCE
Designed for chronic use

Potential **first-in-class** convenient solution for chronic therapy

Clinical development strategy is designed to generate rapid POC

Program	MoA	Stage			
		Discovery	IND-enabling	Clinical	Status
DMR-001	**Anti-mutCALR mAb** (Fc-null, half-life extended)				Two clinical POC readouts beginning mid-2027
DMR-002	**Anti-mutCALR mAb** (Fc-enhanced, half-life extended)				First regulatory submission expected 2H26
DMR-003	**Anti-mutCALR x CD3 bsAb** (T-cell engager)				First regulatory submission expected 2027

Large market opportunity, with ~25-35% of essential thrombocythemia & myelofibrosis driven by mutCALR

Assets designed by Paragon Therapeutics, with expertise in developing best-in-class biologics

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Commercial opportunity

Multi- blockbuster potential in mutCALR-driven MPNs

Current MPN treatments leave significant unmet need, with no disease-modifying therapeutics

ET patients are at **increased risk for thrombosis, hemorrhage, and conversion to MF**[1]

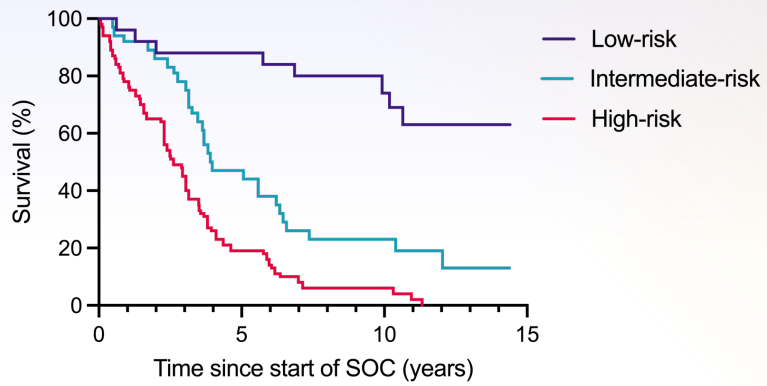
60-70% of ET patients require **cytoreductive therapy** to reduce risk of thrombosis[2]

20-30% of patients receiving SOC cytoreductive therapies are **resistant** or **intolerant**[3]

>17% of mutCALR ET patients **transform to MF**[4]

There is need for highly safe, convenient, and targeted **disease-modifying** therapies

MF leads to **poor survival and significant risk of leukemia**, yet SOC largely addresses symptoms[5]



mutCALR targeted therapy presents an opportunity for much-needed **disease modification** that eradicates neoplasms

Notes: SOC: standard of care.
Sources: Internal KOL calls; [1]Campbell 2005 (Lancet); [2]Loscocco 2024 (Blood); [3]Hernandez-Boluda 2010 (Brit J Hem); [4]Tefferi 2025 (JAMA); [5]Gangat 2023 (Blood Cancer J).

Estimate >$5B total addressable market for mutCALR-driven MPNs in the US alone

Essential thrombocythemia

~140,000 prevalent patients in the US

→ **~25%** have CALR driver mutations

Myelofibrosis

~20,000 prevalent patients in the US

→ **~35%** have CALR driver mutations

Estimate ~42,000 patients with mutCALR-driven MPNs in the US,
with majority indicated for cytoreductive / targeted treatment

Sources: Klampfl 2013 (NEJM); prevalence based on range from Mehta 2013 (Leukemia & Lymphoma), Shalis 2021 (Hematol Oncol Clin N Am), and Masarova 2025 (ASCO); Incyte 2025 Q2 report.
Incyte estimates a >$7B US addressable market for anti-mutCALR therapy.

Significant initial opportunity plus high growth potential with expansion into lower-risk populations

Essential Thrombocythemia

~35K patients with mutCALR-driven ET in the US

~21K – 24.5K indicated for cytoreductive Tx

- **20% – 30%** resistant or intolerant to SOC

~10.5K – 14K not indicated for cytoreductive Tx

Myelofibrosis

~7K patients with mutCALR-driven MF in the US

~4.8K – 5.5K with intermediate-to-high-risk MF

~1.4K – 2.1K with low-risk MF

~$1.4B ruxolitinib US revenue attributable to MF alone in 2025

Notes: ET and MF sub-group breakdowns are mutCALR patients based on preliminary internal estimates. Ruxolitinib revenue in MF based on sellside estimates; Incyte does not report revenue by indication.

DMR-001 is competitively positioned to achieve significant market share on convenience alone

INCA'989 formatted for **in-clinic Q2W intravenous dosing**, with on-body injector in early development

Annual doses of anti-mutCALR

INCA'989
IV Q2W

INCA'989
OBI

DMR-001
SC Q4W+

KOLs view **SC dosing as required for wider adoption**, particularly in ET patients

"Any new starts will use [DMR-001], and I may switch patients finding it difficult to come every two weeks for [INCA'989] even if they were doing reasonably well."

-- U.S. Academic HCP

"I think **IV treatment every 2 weeks for [INCA'989] is going to be a deal-breaker** for some ET patients."

-- U.S. Community HCP

"Even at the minimal profile, **I would prefer a biomarker-targeted option versus a JAK inhibitor** [in MF patients], and **[DMR-001] for the SC administration versus IV**."

-- U.S. Academic HCP

DMR-001 is expected to be conveniently administered SC Q4W+ using an autoinjector

Notes: OBI: on-body injector. Annual dose figures reflect dosing frequency projected to match INCA'989 2500mg Q2W IV on Ctrough and assume 2x improvement in Type 2 in vivo potency.

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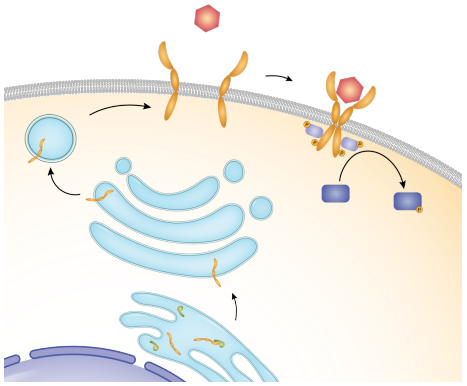
DMR-001

anti-mutCALR Fc-null antibody lead program

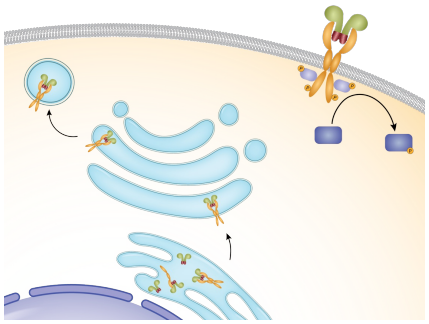
CALR mutations drive constitutive JAK/STAT signaling and uncontrolled proliferation of myeloid progenitors in MPNs

Healthy TPO-dependent activation of TPO receptor



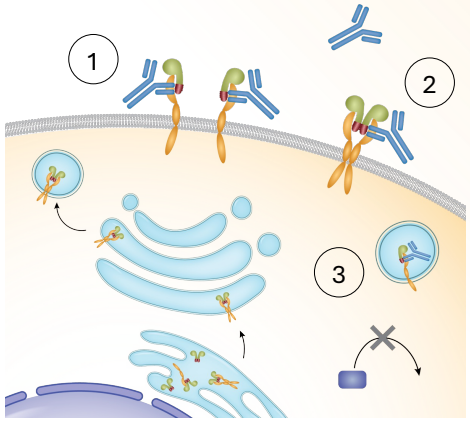
Normal function of wild-type calreticulin is within cell

Ligand-independent signaling driven by mutCALR



mutCALR binds to TpoR/MPL complex, and is trafficked to cell surface

Inhibition of mutCALR signaling with DMR-001



Proposed mechanisms: 1) disruption of TpoR/MPL dimer, 2) inhibition of tetramer signaling, 3) internationalization of complex

JAK, janus kinase; MPL, myeloproliferative leukemia protein; mutCALR, mutant calreticulin; STAT, signal transducer and activator of transcription; pJAK, phosphorolated JAK; pSTAT, phosphorolated STAT; TPO, thrombopoietin; TpoR, TPO receptor; wtCALR, wild-type calreticulin;

DMR-001 is a potentially best-in-class anti-mutCALR mAb

Inhibits mutCALR-mediated proliferation with better *in vitro* potency versus reference mutCALR mAb

- **Demonstrated POC** for mechanism of action
- **Superior potency on Type 2 mutCALR**
- **Similar or better potency on Type 1 mutCALR**
- **Favorable safety predicted** – Fc null design, highly selective over wild-type CALR, low volume dosing anticipated

Subcutaneous formulation

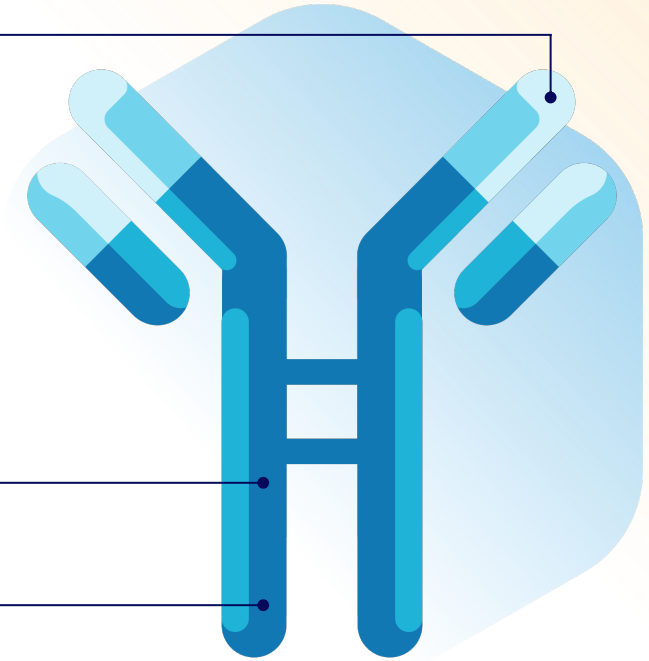
- Targeting lower dose to enable convenient SC autoinjector format

Half-life extension through validated Fc modification

- **Longer exposure** to enhance sustained mutCALR inhibition and **reduce dosing frequency**

Effector-null human IgG1 Fc

Novel IP[(1)] for composition of matter into mid-2040s



DMR-001

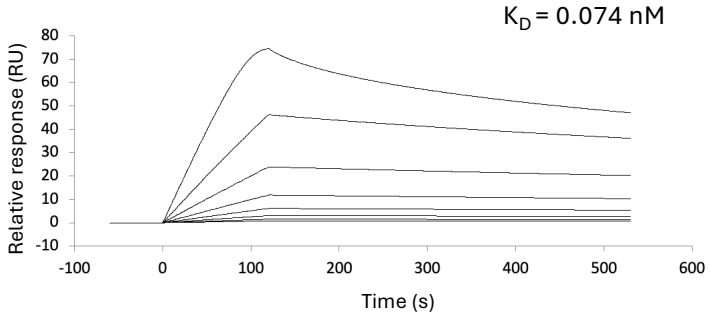
[(1)] *Damora has exercised its option to acquire certain intellectual property license rights pursuant to the antibody discovery and option agreement by and among Damora Therapeutics LLC, Paragon Therapeutics, Inc. and Paramora Holding LLC, dated October 7, 2025.*

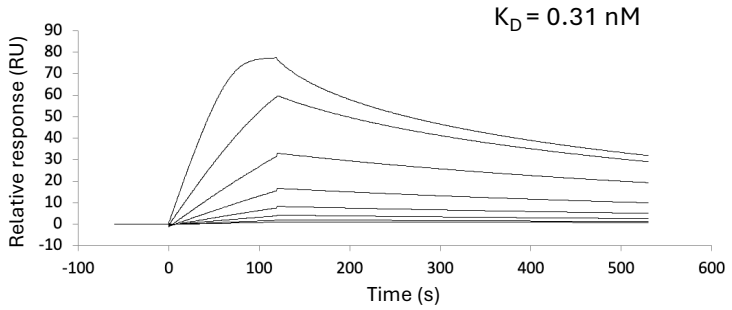
DMR-001 shows higher binding affinity and slower off-rate in both Type 1 and Type 2 mutCALR compared to reference mutCALR mAb

~26-fold improvement in binding affinity in Type 1 mutCALR

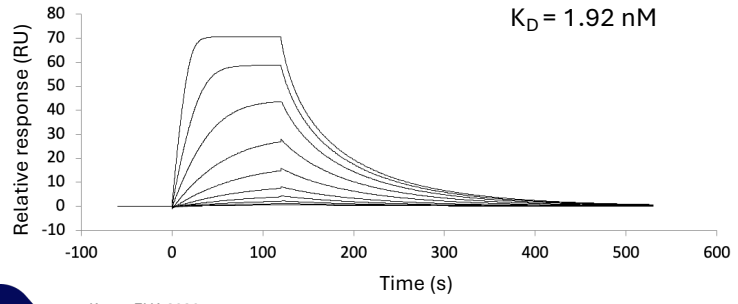
~30-fold improvement in binding affinity in Type 2 mutCALR

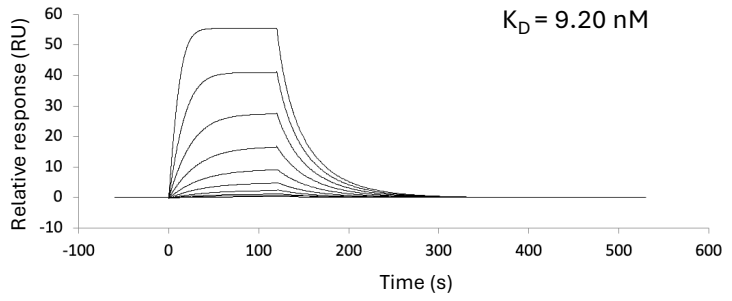
DMR-001





Reference mutCALR mAb

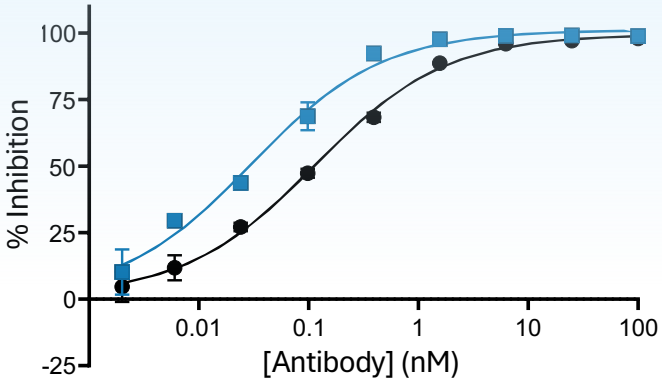




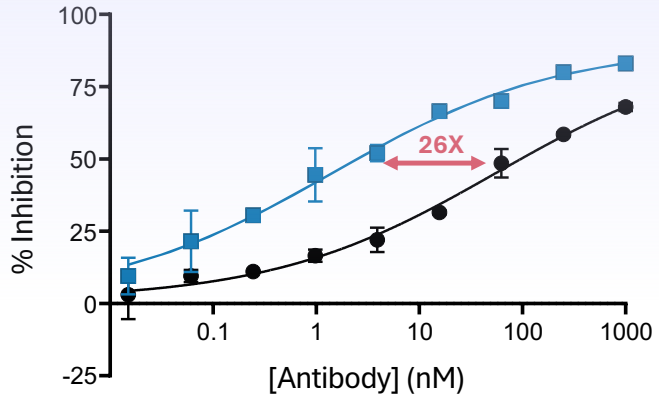
Kwan, EHA 2026.

DMR-001 robustly inhibits cellular proliferation in both Type 1 and Type 2 mutCALR compared to reference mutCALR mAb

Robust anti-proliferative activity
in Type 1 mutCALR



~26-fold greater anti-proliferative activity
in Type 2 mutCALR

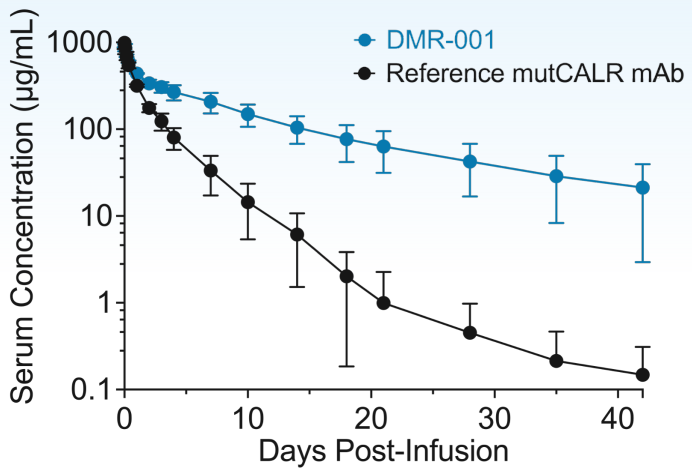


■ DMR-001
◆ Reference mutCALR mAb

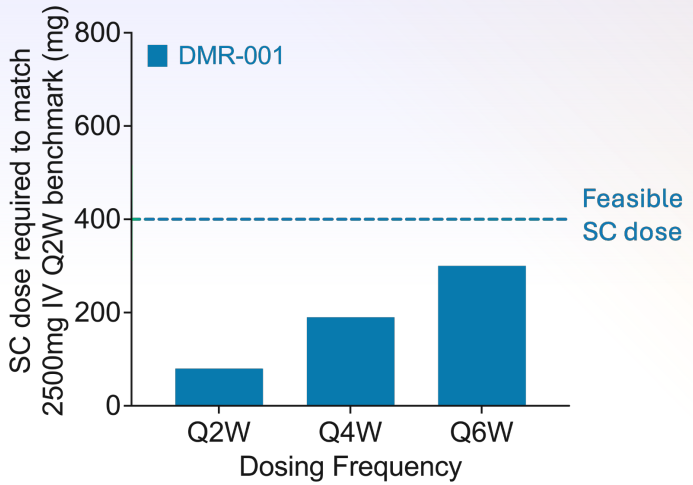
Kwan, EHA 2026.

DMR-001 expected to deliver highly efficacious exposure with convenient, infrequent SC dosing using conservative modeling

~5x longer half-life in NHPs
versus reference mutCALR mAb



Dose required to match INCA'989 2500mg IV Q2W on C_{trough} would enable **Q4W+ SC dosing**



Kwan, EHA 2026. Notes: Reference mutCALR mAb produced recombinantly based on US20230272055A1. Day 42 Interim NHP PK Data after a single 30 mg/kg IV dose (n=4 per group). Bar chart reflects projections to match benchmark mAb 2500mg IV Q2W on C_{trough} and conservatively models 2x improvement in Type 2 potency in vivo. SC, subcutaneous.

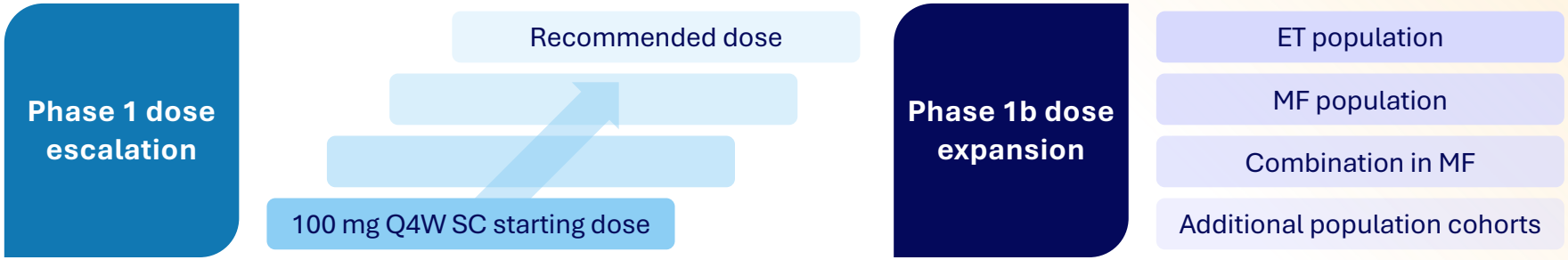
DMR-001 preclinical data supports potential best-in-class profile

	INCA'989 emerging clinical profile	DMR-001 preclinical profile
Dosing	750 mg - 3,500 mg Q2W IV	**~5x increase in half-life** versus reference mutCALR mAb supports **Q4W SC dosing**
Type 1 activity	**ET:**[1] 81% durable CHR (21/26; ≥750 mg) **MF:**[2] 31% SVR35 (8/26; all doses)	**Improved binding affinity** and similarly **robust anti-proliferative activity** versus reference mutCALR mAb
Non-Type 1 activity	**ET:**[1] 33% durable CHR (2/6; 2,500 mg) **MF:**[2] 5% SVR35 (1/19, all doses)	**~30x better binding affinity** and **~26x greater anti-proliferative activity** versus reference mutCALR mAb[3]
Safety	Well tolerated, low discontinuation rate	**Fc null design (similar to INCA'989)**, highly selective, predicted low volume dosing

1 ET resistant or intolerant to ≥1 cytoreductive therapy. 2 MF relapsed, refractory or intolerant to JAK inhibitor. 3 Preclinical data in Type 2 mutCALR.
Harrison, EHA 2026; Mascarenhas, EHA 2026; Kwan, EHA 2026. Note: no head-to-head clinical studies of INCA'989 and DMR-001 have been conducted, and comparisons of clinical and preclinical data should be interpreted with caution. ADCC, antibody-dependent cellular toxicity; CDC, complement-dependent cytotoxicity; CHR, complete hematologic response; SVR35, 35% reduction in spleen volume.

Phase 1/1b CLARITY-101 trial of DMR-001 initiated in Q3 2026

Global, multi-center, open-label, dose escalation and expansion trial in mutCALR-driven ET and MF



- 100 mg Q4W SC starting dose
  - In range of expected therapeutic exposure with high potency, prolonged target coverage
  - Opportunity to reach therapeutic dose with limited number of dose escalation cohorts
- Adaptive Bayesian design enabling cohort backfilling and patient enrichment
- MutCALR antibody experienced patients to be allowed later in dose escalation

- Plan to explore multiple ET and MF populations and settings, including early line treatment and combinations
- Expansion cohorts designed to support the first Phase 3 initiation as early as mid-2028

Key endpoints: Safety, PK, hematologic response, spleen volume response, symptom response, translational biomarkers

Trial design schematic is illustrative, and the number of actual dose escalation and expansion cohorts may differ.

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Financials and future anticipated milestones

Exceptional financial position to accelerate development through proof-of-concept and toward registration

	2026	2027
DMR-001 (Fc-null anti-mutCALR mAb)	✓ Phase 1/1b CLARITY-101 trial initiated	Two clinical POC readouts beginning mid-2027
DMR-002 (Fc-enhanced anti-mutCALR mAb)	2H: first regulatory submission	
DMR-003 (anti-mutCALR x CD3 bsAb)		First regulatory submission
Key external events	2H: INCA'989 update in 1L MF, Design of Phase 3 trial in 2L MF	

Strong cash position with ~$541M of cash and cash equivalents at the end of Q2 2026, with anticipated runway into the second half of 2029

Sources: Incyte public statements.
Notes: ROA: route of administration, MOA: mechanism of action, mAb: monoclonal antibody, bsAb: bispecific antibody, POC: proof-of-concept