Degrading Proteins, Making Medicines

MRT-8102 Phase 1 Clinical Data Update

January 7, 2026

Monte Rosa Therapeutics

Forward-Looking Statements

This communication includes express and implied "forward-looking statements," including forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements include all statements that are not historical facts and, in some cases, can be identified by terms such as "may," "might," "will," "could," "would," "should," "expect," "intend," "plan," "objective," "anticipate," "believe," "estimate," "predict," "potential," "continue," "ongoing," or the negative of these terms, or other comparable terminology intended to identify statements about the future. Forward-looking statements contained herein include, but are not limited to, statements about our ability to grow our product pipeline, our ability to successfully complete research and further development and commercialization of our drug candidates in current or future indications, including the timing and results of our clinical trials and our ability to conduct and complete clinical trials, statements regarding our progress and speed of development of only-in-class and first-in-class molecular glue degrader therapeutics, statements around the Company's QuEEN™ discovery engine, its advancement and the broad potential applications of the platform and the Company's ability to create long-term value through focused pipeline execution and strategic collaborations, as well as to expand the targetable protein space for MGD drug discovery, unlocking new opportunities to address previously undruggable therapeutic targets, statements related to the expected potential clinical benefit of any of our candidates, statements regarding the positive interim Phase 1 data and potential benefits of MRT-8102, our expectations regarding the potential of MRT-8102 to transform the treatment of ASCVD and other cardiovascular and cardiometabolic diseases, our belief that our data supports the potential of MRT-8102 to be an oral best-in-class therapeutic among agents targeting the NRLP3/ IL-1/IL-6 pathway and establish the significant potential of MRT-8102 in multiple chronic inflammatory diseases, including ASCVD, our statements regarding the expansion of our proof-of-concept GFORCE-1 study in subjects with elevated CVD risk and acceleration of the anticipated Phase 2 (GFORCE-2) study of MRT-8102 in ASCVD patients, our expectations regarding the timing for sharing data from the GFORCE-1 study of MRT-8102 and timing of initiation of a Phase 2 GFORCE-2 study of MRT-8102 in ASCVD, our statements and expectations regarding our evaluation of additional Phase 2 proof of concept studies in MASH, gout, and recurrent pericarditis, conditions strongly linked to NLRP3 pathway activation, statements regarding our expectations that our collaborator, Novartis, will initiate multiple Phase 2 studies of VAV1-directed MGD MRT-6160 in immune-mediated diseases in 2026, our expectations regarding the submission of an IND application for a next-generation NEK7-directed MGD and timing thereof, our expectations to initiate a MODeFIRe-1 Phase 2 study of MRT-2359 in combination with a second-generation androgen receptor inhibitor in CRPC in 2026, as well as to present updated data from the ongoing Phase 1/2 study of MRT-2359 at the ASCO Genitourinary Cancers Symposium in February 2026, our expectations regarding the submission of an IND application for a CDK2 and/or cyclin E1-directed MGD and timing thereof, statements regarding the clinical significance of the clinical data read-out at upcoming scientific meetings and timing thereof, statements around our ability to capitalize on and potential benefits resulting from our research and translational insights, , including announcements related to preclinical programs, as well as our the ability to optimize collaborations with industry partners on our development programs, statements regarding regulatory filings for our development programs, including the planned timing of such regulatory filings, such as IND applications, and potential review by regulatory authorities, statements around our expectations of success for our programs, strength of collaboration relationships, among others. By their nature, these statements are subject to numerous risks and uncertainties, including those risks and uncertainties set forth in our most recent Annual Report on Form 10-K for the year ended December 31, 2024 filed with the U.S. Securities and Exchange Commission on March 20, 2025, most recent Quarterly Reports on Form 10-Q and any subsequent filings, that could cause actual results, performance or achievement to differ materially and adversely from those anticipated or implied in the statements, as well as the risk that outcomes of preclinical studies may not be predictive of clinical trial results and the risk that initial or interim results from a clinical trial may not be predictive of the final results of the trial or the results of future trials. You should not rely upon forward-looking statements as predictions of future events. Although our management believes that the expectations reflected in our statements are reasonable, we cannot guarantee that the future results, performance, or events and circumstances described in the forward-looking statements will be achieved or occur. Recipients are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date such statements are made and should not be construed as statements of fact. We undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, any future presentations, or otherwise, except as required by applicable law. Certain information contained in these materials and any statements made orally during any presentation of these materials that relate to the materials or are based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of these materials, we have not independently verified, and make no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in these materials relating to or based on such internal estimates and research.

Summary

- MRT-8102, a NEK7-directed molecular glue degrader (MGD), induced rapid and compelling reduction in hsCRP across all doses tested in both healthy volunteers and high-CVD risk subjects (112 subjects in total)

- SAD (48 healthy volunteer subjects) and MAD (40 healthy volunteer subjects) cohorts completed with no adverse safety signals

    - MRT-8102 was dosed from 5 – 400mg (SAD: 40 – 400mg; MAD: 5 – 200mg) and data suggest maximum activity achieved from lowest dose level (5mg MAD)
    - ~80-90% NEK7 degradation noted in T cells at all dose levels tested
    - 78% reduction in hsCRP achieved in subjects with elevated baseline CRP levels after both single and multiple dose administration
    - Favorable AE profile with no adverse safety signal observed as of data cut off date of 12/23/25

- Part 3 (CRP PoC) of Phase 1 study exploring 40 mg MRT-8102 in high-risk CVD subjects (obesity/elevated CRP) is ongoing and 24 subjects have been evaluated up to end of week 4. Preliminary data for these subjects showed:

    - 85% sustained reduction of hsCRP through end of week 4
    - 94% subjects achieved reduction of hsCRP levels to <2 mg/L* after 4 weeks of dosing (baseline hsCRP level of 6.3 mg/L)
    - 31% reduction of fibrinogen after 4 weeks of dosing
    - No SAEs, no severe AEs as of data cut off date of 12/23/25, evaluation ongoing

- Study (now named GFORCE-1) will be expanded and additional dose levels will be explored to accelerate development in ASCVD; data expected in H2 2026

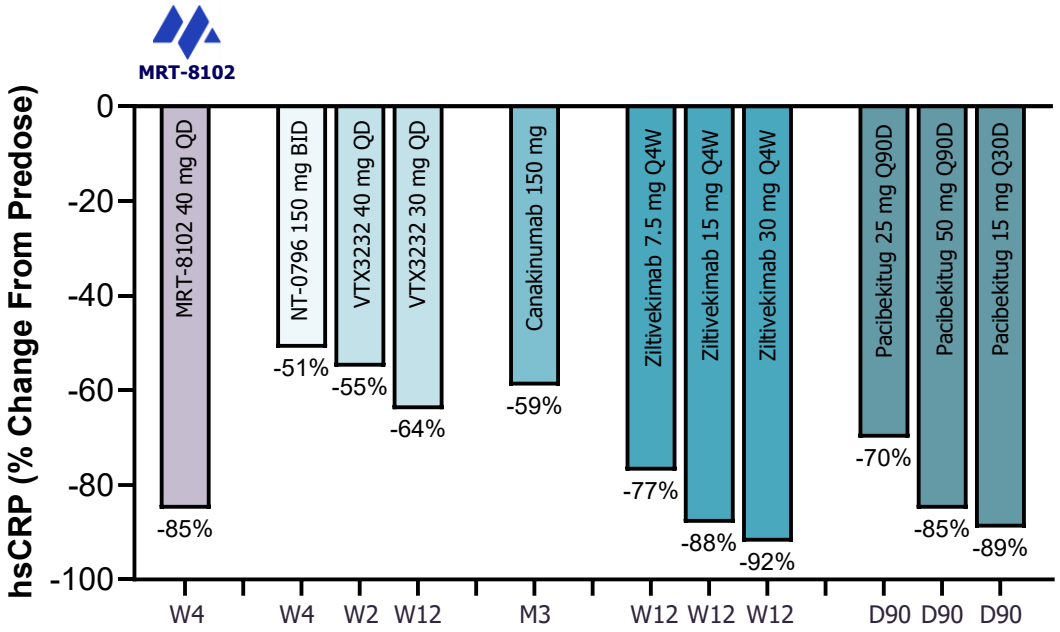
- Early hsCRP results continue to support MRT-8102 development across chronic inflammatory diseases such as ASCVD and MASH

    - hsCRP reduction data compares favorably to previously reported third party data on NLRP3 inhibitors in development and canakinumab (IL-1β antibody) and is on par with IL-6 biologics**

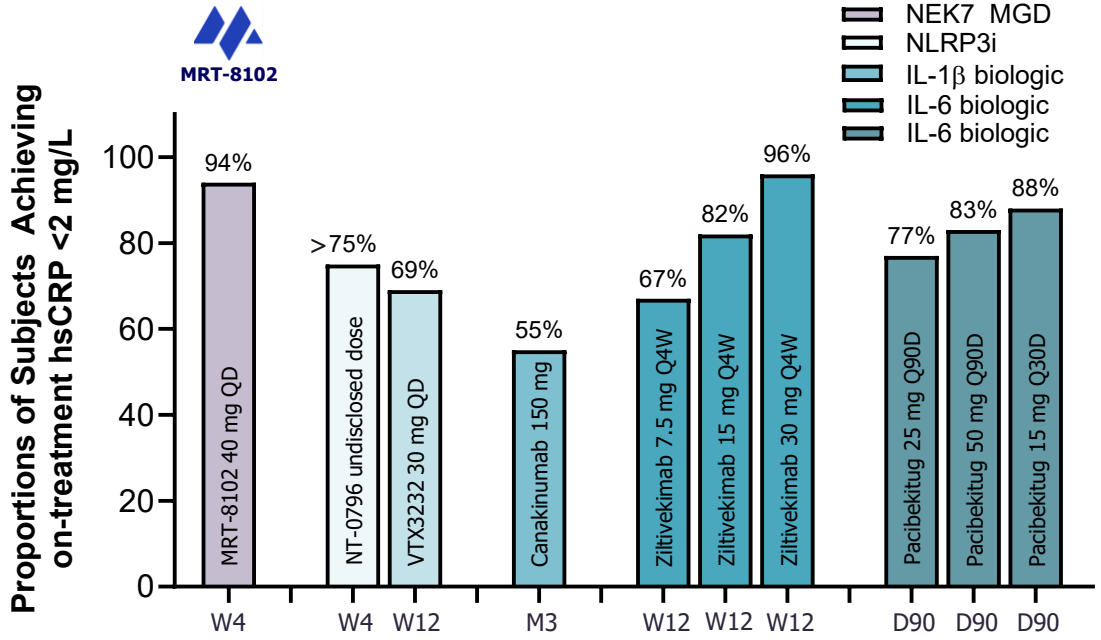
hsCRP, high-sensitivity C-reactive protein
*hsCRP levels of >2 mg/L are associated with elevated CVD risk
**Comparison not based on head-to-head studies

MRT-8102 – Beyond an Oral IL-1/IL-6 Modality

MRT-8102 shows favorable reduction of hsCRP compared to other inflammasome and IL-6 targeted agents in development



MRT-8102 achieves favorable rates of hsCRP <2 mg/L compared to other inflammasome and IL-6 targeted agents



MRT-8102 data compares favorably to data reported for NLRP3 inhibitors and appears on par with data reported for IL-6 antibodies

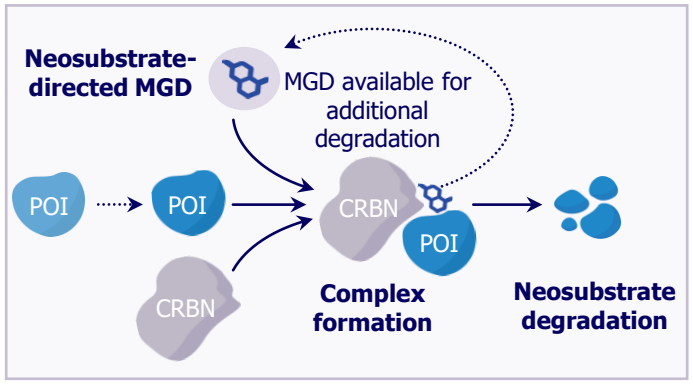
MRT-8102 provides convenience of oral route of administration

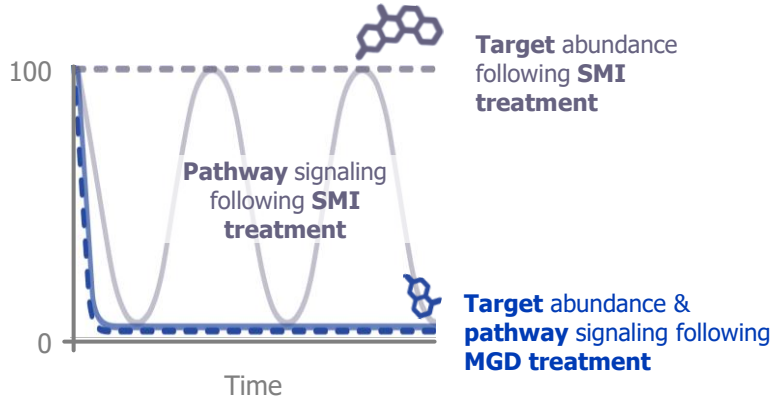
MRT-8102 may also provide potential advantage of inhibiting pyroptotic cell death and hence have a superior effect on local inflammation and plaque stabilization in ASCVD

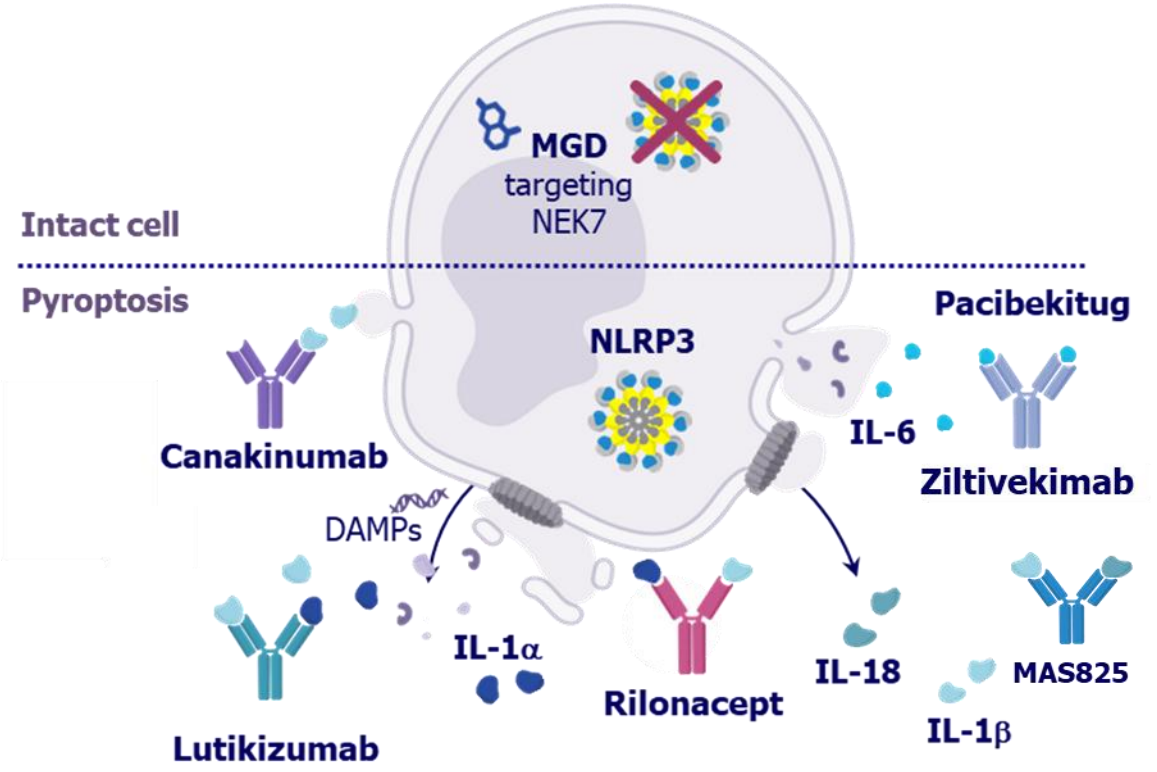
NT-0796 – Clarke N et al. Anti-Neuroinflammatory and Anti-Inflammatory Effects of the NLRP3 Inhibitor NT-0796 in Subjects with Parkinson's Disease. Movement Disorders 2025; Nodthera Press Release June 2024 for Obese Subjects with Cardiovascular risk; V**T3232** – Ventyx Corporate Presentation August 2024 for HV and Ventyx Press Release October 2025 for Subjects with Obesity and Cardiovascular Risk Factor (CRP reduction from FAS, CRP <2 mg/L from MAS); **Canakinumab** - Ridker PM et al. Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease. NEJM 2017; Ridker PM et al. Relationship of C-reactive protein reduction to cardiovascular event reduction following treatment with canakinumab: a secondary analysis from the CANTOS randomized controlled trial. Lancet 2018; **Ziltivekimab** - Ridker PM et al. IL-6 inhibition with ziltivekimab in patients at high atherosclerotic risk (RESCUE): a double-blind, randomized, placebo-controlled, phase 2 trial. Lancet 2021; **Pacibekitug** - Tourmaline Bio Phase2 TranQuility Trial Topline Results May 2025. W –week; M –month; D –day.

Note: Comparisons between MRT-8102 and other therapies represented herein are based on post-hoc analyses comparing MRT-8102 clinical information with publicly available information for other therapies. Any comparisons use information from different clinical trials, conducted by different parties, at different points in time, with differences in trial designs and patient populations. No head-to-head clinical trials have been conducted, cross-trial comparisons should not be made, and this information is provided only for illustrative purposes.

MRT-8102 is Highly Differentiated over Other NLRP3/IL-1/IL-6 Pathway Modalities







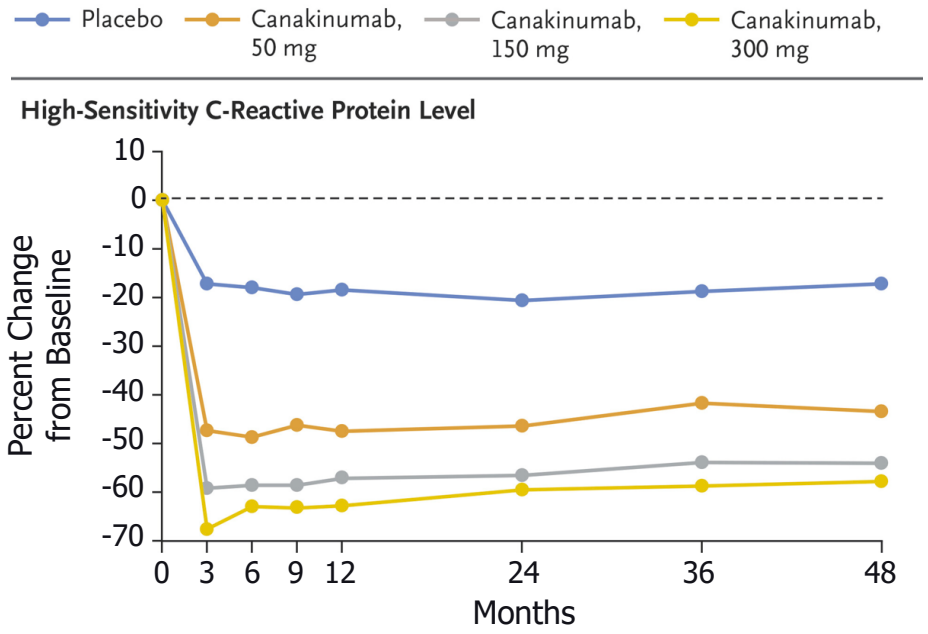
In contrast to small molecule inhibitors (SMIs), MRT-8102 induces catalytic NEK7 degradation, long-lasting inflammasome disassembly, and sustained inhibition of cytokine release

Due to inhibition of NLRP3 assembly, MRT-8102 prevents pyroptotic cell death-mediated release of inflammatory cytokines and DAMPs known to drive disease pathology.

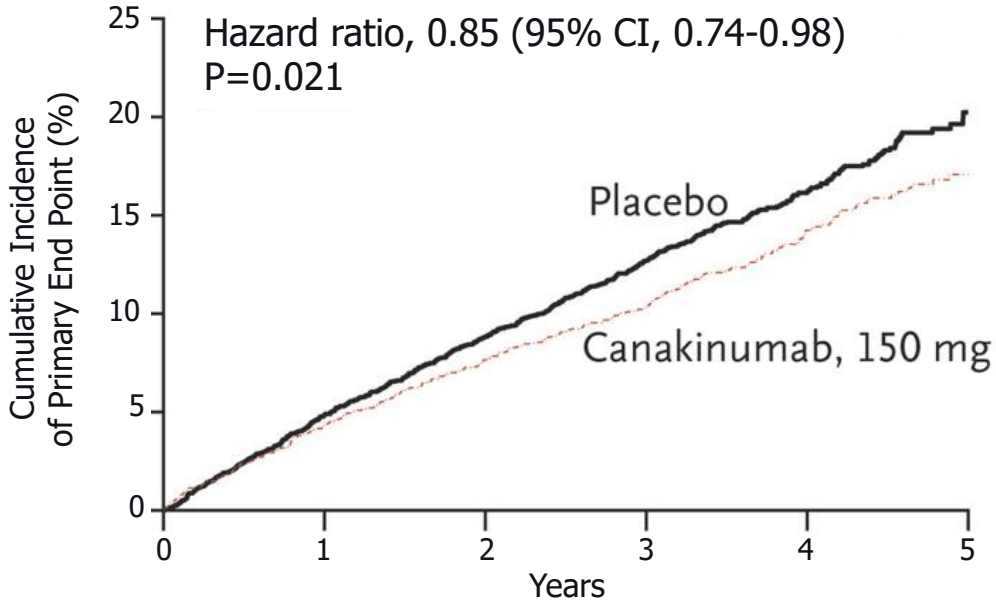
Mono- and bispecific biologics fail to inhibit pyroptosis, leading to incomplete blockage of the pathological drivers of disease

CANTOS Study Established Role of Inflammation in Cardiovascular Disease

50-60% reduction in CRP noted across dose groups following Canakinumab treatment for 48 mo



Significant reduction in recurrent CV events noted at 150 mg dose

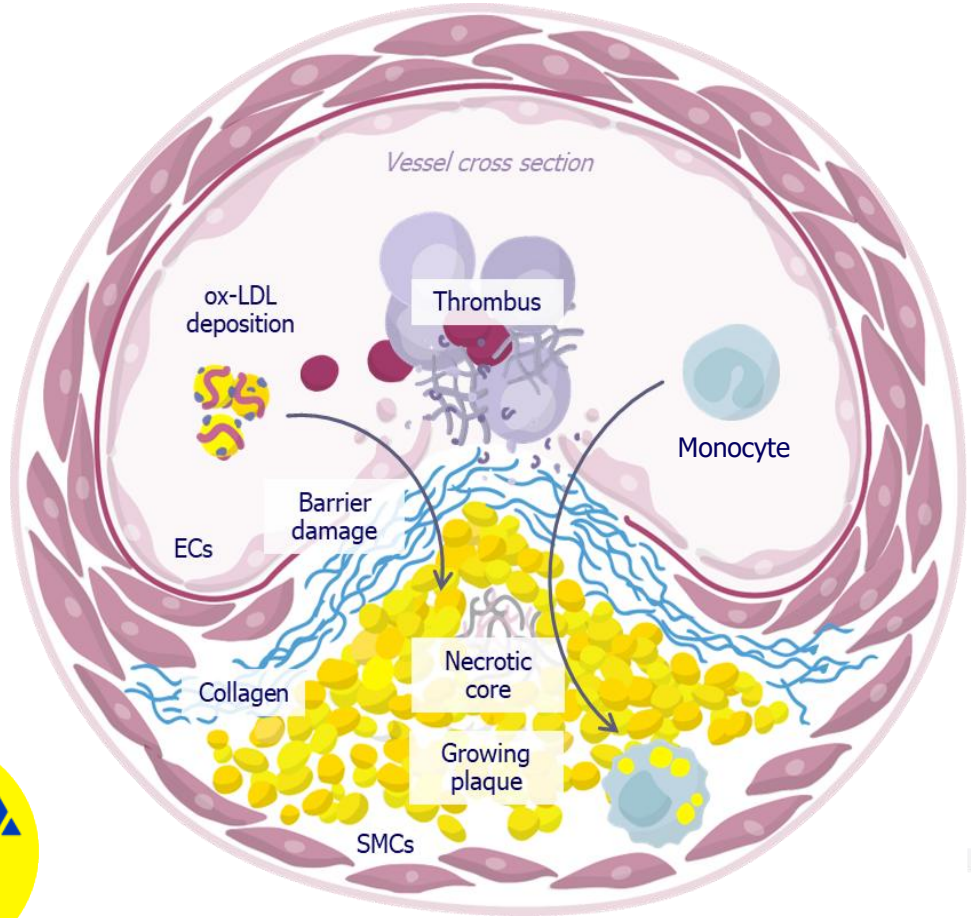


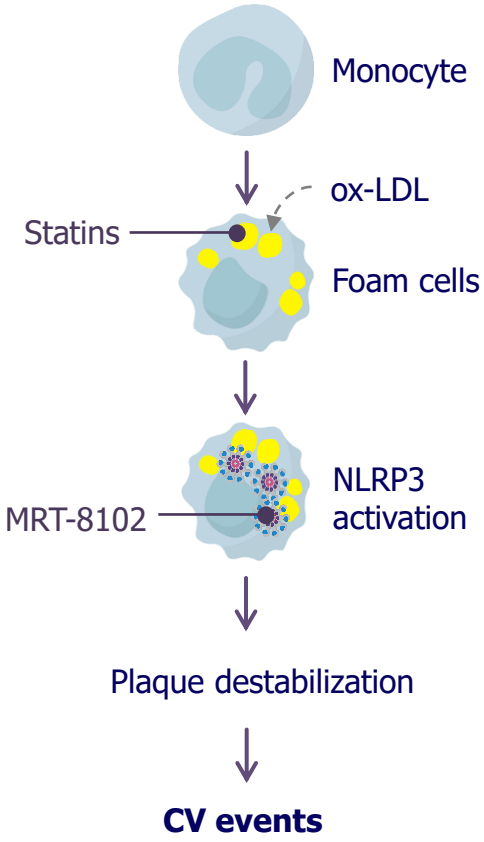
Improving inflammatory status — independent of changes in lipid profile — reduced cumulative incidence of CV events and confirmed the role of inflammation in atherosclerotic disease

Ridker PM et al. NEJM (2017)

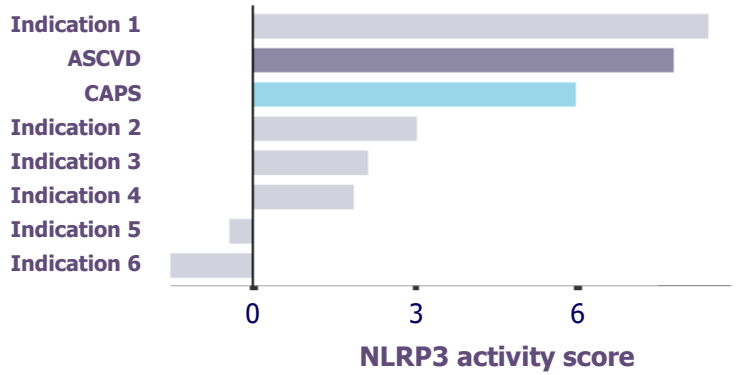
Upstream Targeting of NLRP3/NEK7 Pathway May Have Greater Potential than Downstream IL-6 Biologics in ASCVD

NLRP3 activation promotes plaque growth, destabilization and CV events
MRT-8102 has potential to stabilize plaques preventing thrombosis

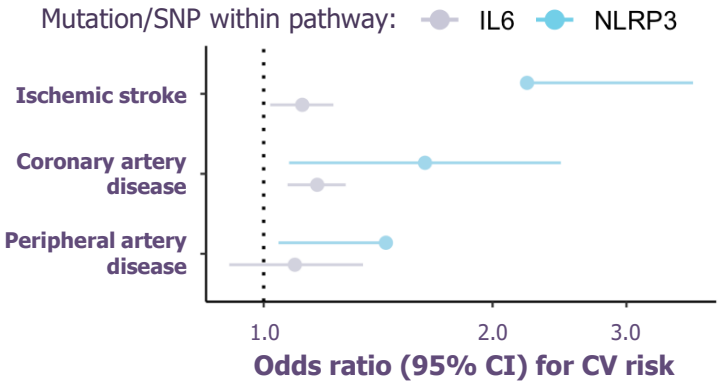




ASCVD ranks amongst top NLRP3 activated indications



Human genetics* supports causal relationship between NLRP3 and ASCVD



* Analysis based on Georgakis et al. Circ Genom Precis Med (2020); Zhu Z et al. Cell Mol Neurobiol (2016); Zhang K et al. Research Square (2021); Zhou D et al., BioMed Research International (2016). Odds ratios were directionally harmonized (OR = 1/OR) to display consistent benefit vs harm.

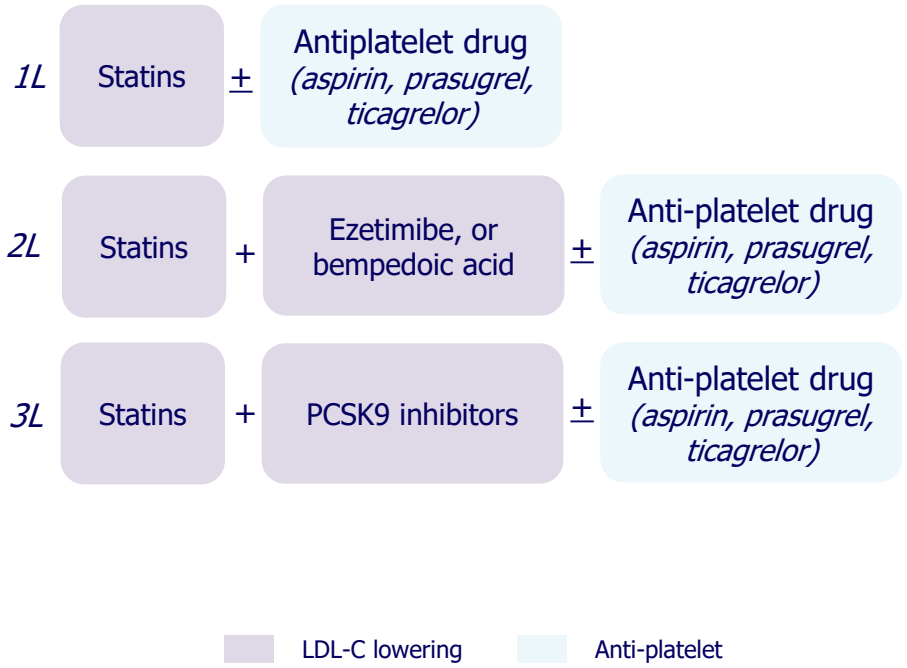
ASCVD Presents a Substantial Market Opportunity for MRT-8102

ASCVD Market Overview

18.7M
Patients in the U.S.

~60%
Patients have a CRP level above 2 mg/L

Treatment Paradigm



Additional interventions: lifestyle change, surgical procedures (angioplasty, endarterectomy, bypass, etc.)

Opportunity

Address residual cardiovascular risk

- Even among patients who achieve their LDL-C targets, **up to 40%** still experience life-threatening cardiovascular events, demonstrating substantial residual risk not fully addressed by LDL-C lowering

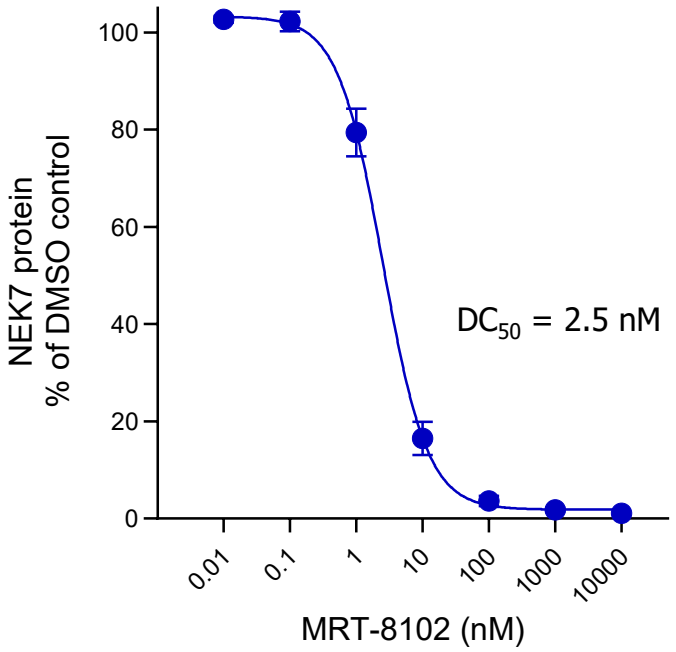
Source: Arnett et al. Circulation (2019); Pesce et al. Front Cardiovasc Med. (2025); Alanaeme et al. Am Heart J Plus (2022); Mazhar et al. Eur Heart J. (2024)

MRT-8102 Profile and Interim Results of Phase I Study

MRT-8102: Preclinical Profile Points to Best-in-Class Potential

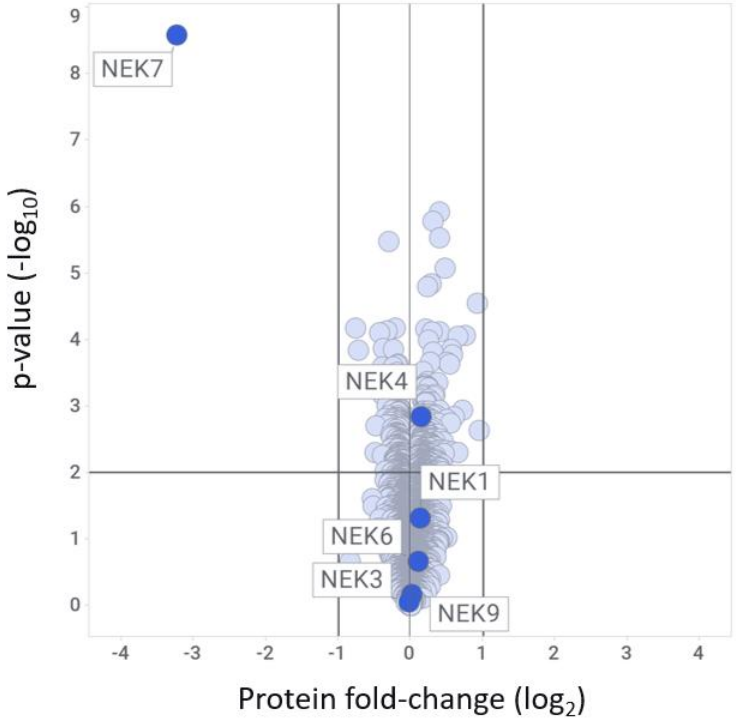
Potency, selectivity, and long-lasting pharmacodynamics differentiate from other IL-1/NLRP3 inflammasome approaches

MRT-8102 potently degraded NEK7



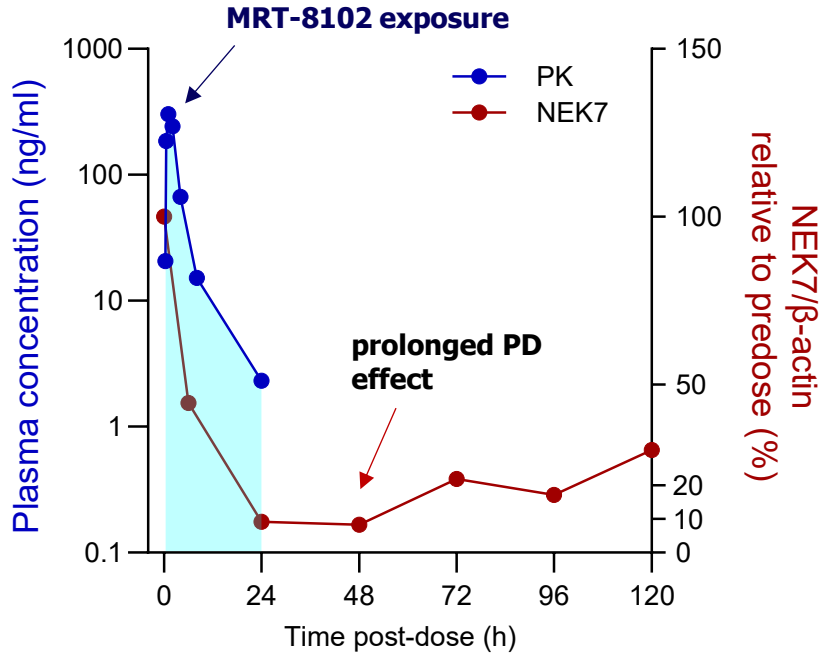
Human PBMC @ 24h treatment

MRT-8102 induced highly selective NEK7 degradation



No degradation of other known CRBN neosubstrates

MRT-8102 exposure resulted in prolonged PD effect



Cyno @ 10 mg/kg single dose

MRT-8102 has potential to avoid on-off pharmacodynamics and off-target toxicities of NLRP3 inhibitors

MRT-8102 Phase I Study – Dose Levels and Endpoints

Primary endpoint

- Safety and tolerability

Key secondary & exploratory endpoints

- PK (blood +/- CSF)
- NEK7 degradation
- Change in CRP level
- IL-6 (blood and CSF)
- Fibrinogen
- Ex-vivo: IL-1β

Completed

SAD cohorts (Part 1)
One oral dose
48 participants

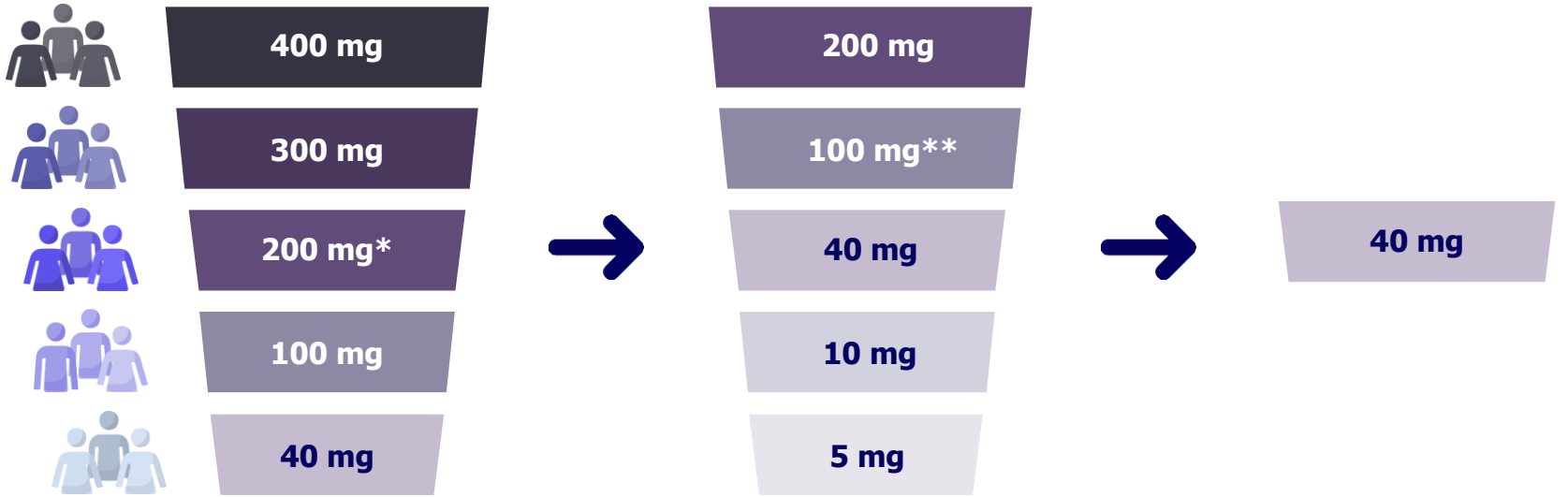
MAD cohorts (Part 2)
7 daily oral doses
40 participants

All cohorts randomized, placebo controlled (6+2)

Ongoing

CRP PoC in elevated CVD risk subjects (Part 3)
28 daily oral doses
~36 participants

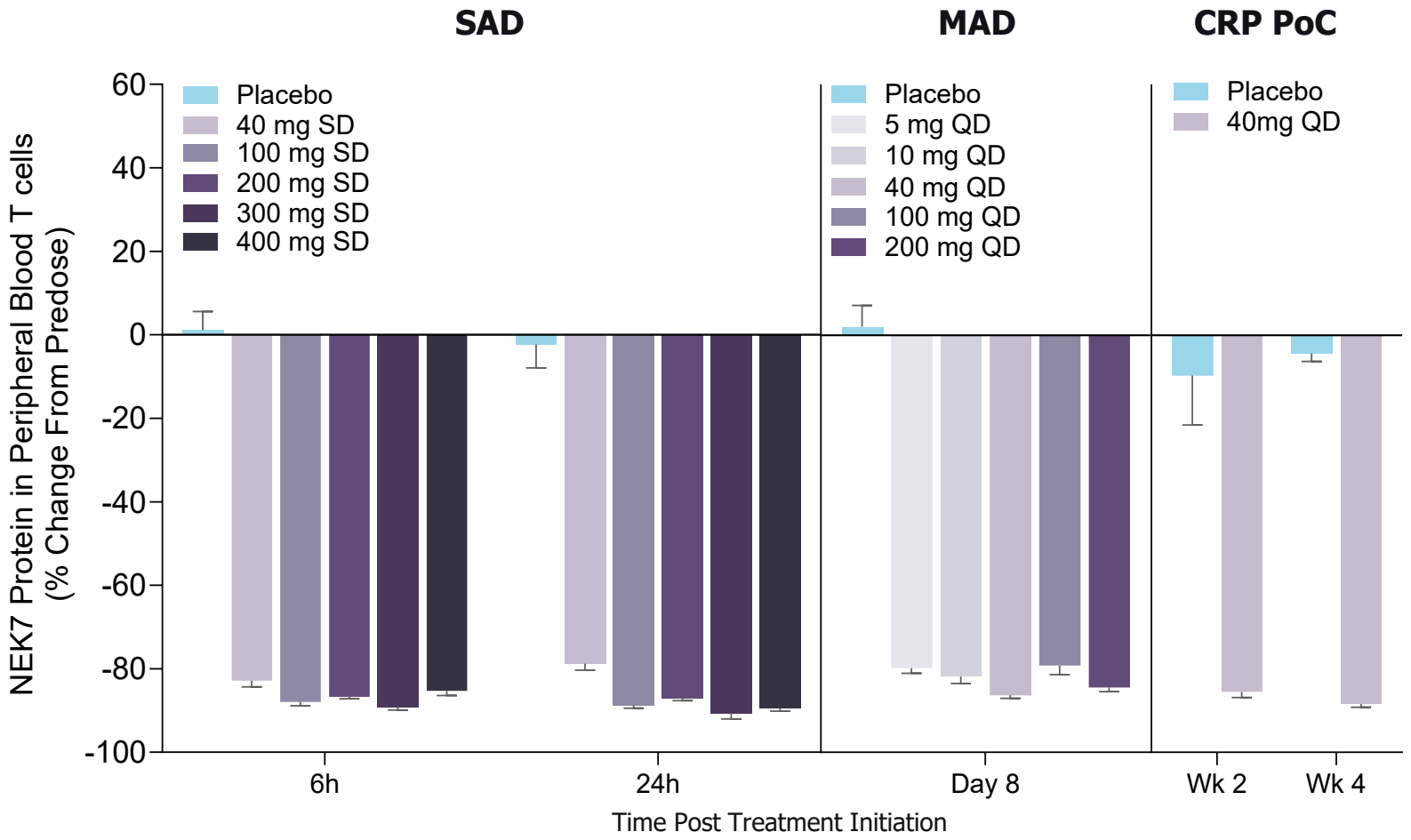
Cohort randomized 3:1 treatment vs placebo



Data cutoff December 23, 2025

* Additional subjects included in 200mg cohort due to sample processing issues; hence a total of 16 subjects have been enrolled in 200mg SAD dose level
**CSF collection

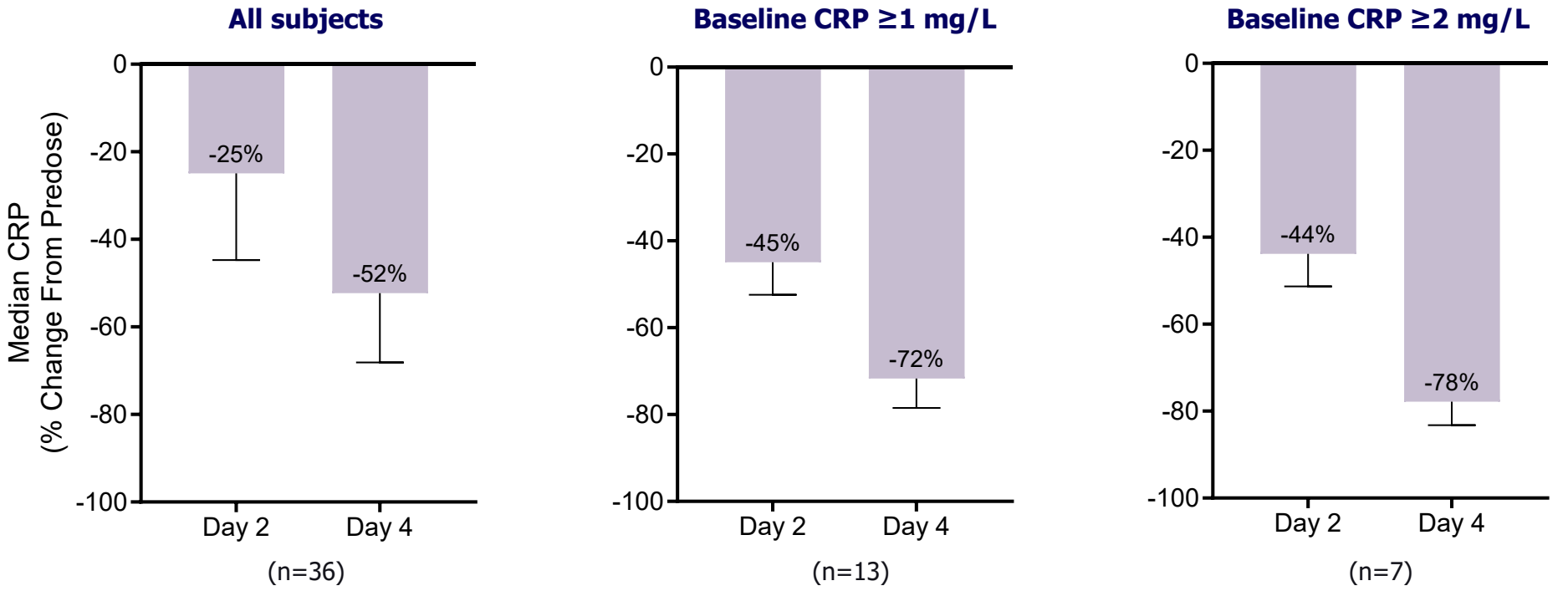
MRT-8102 Achieved 80 – 90% NEK7 Degradation in Peripheral Blood T Cells After Single and Multiple Dose Administration



Rapid and robust degradation of NEK7 noted in peripheral blood T cells **(~80 - 90%)** across all dose levels, consistent with preclinical findings

SAD – 48 subjects (placebo + MRT-8102); MAD - 40 subjects (placebo + MRT-8102); CRP PoC – 16 subjects (placebo + MRT-8102), data delivery pending for remaining 8 subjects.
Data are shown as Mean ± SEM. Day 8, week 2 and week 4 values shown are determined 24h post last dose.

Single Dose of MRT-8102 Led to Significant Reduction in Serum hsCRP



Baseline median CRP – 0.70 mg/L

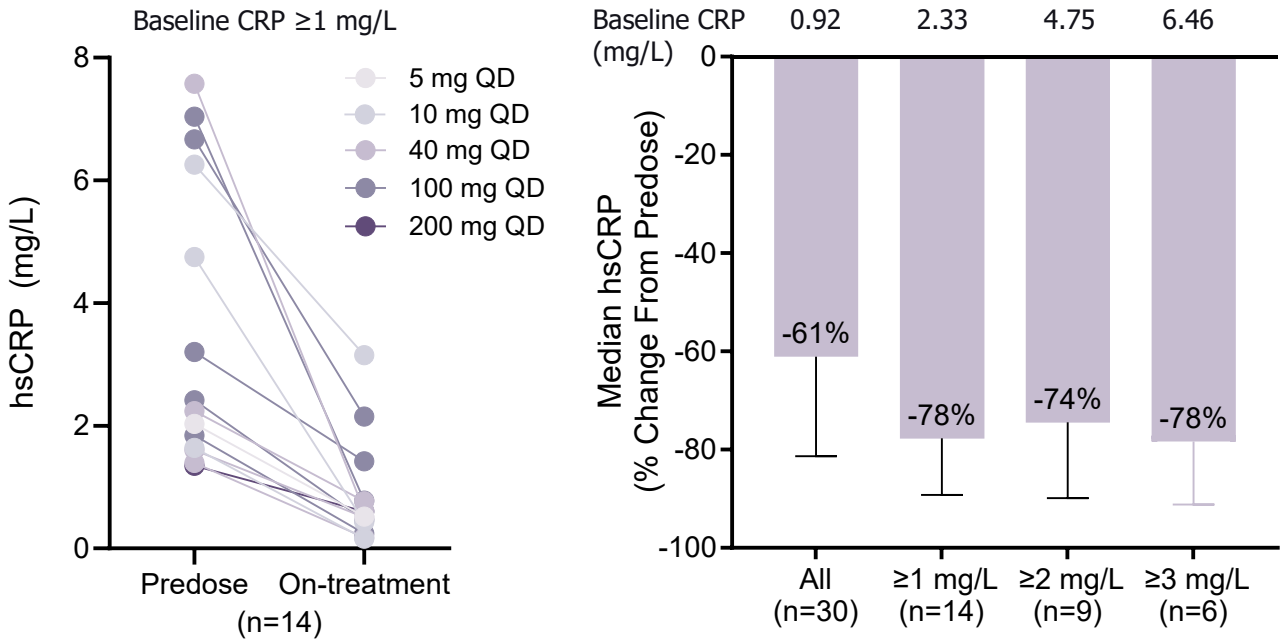
Baseline median CRP – 2.80 mg/L

Baseline median CRP – 3.96 mg/L

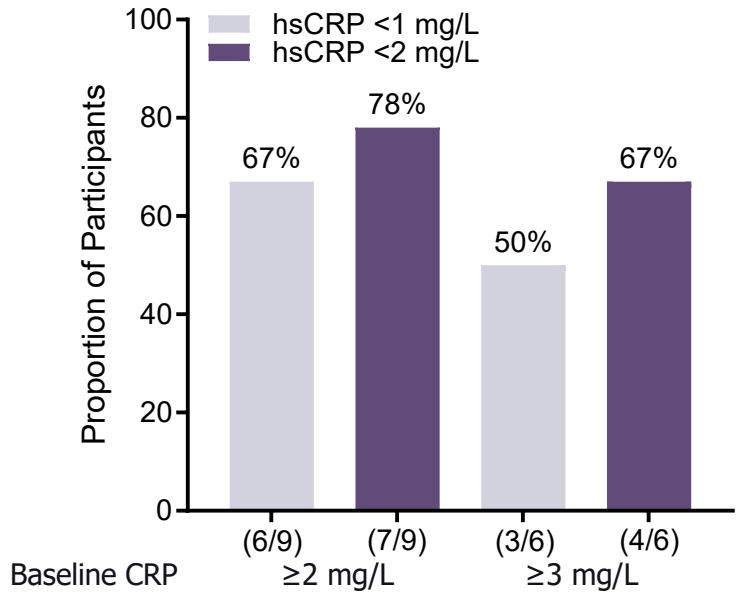
Maximum activity achieved at all SAD dose levels (40 – 400mg)

Multiple Daily Doses of MRT-8102 Led to Significant and Sustained Reduction of Serum hsCRP

MRT-8102 induced significant reduction in serum hsCRP during multiple dose administration*



Significant proportion of subjects achieved hsCRP reduction to <2 mg/L*



61% drop in hsCRP across all subjects regardless of CRP level at baseline; data consistent with maximum MRT-8102 activity achieved at all dose levels

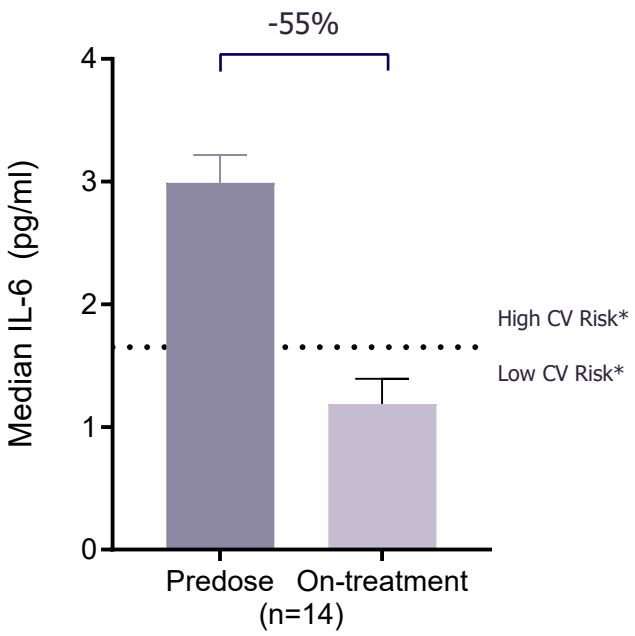
Up to **78%** decrease in hsCRP noted in subjects with elevated CRP levels at baseline

78% of subjects with elevated baseline CRP of ≥2 mg/L achieved suppression of hsCRP to <2 mg/L

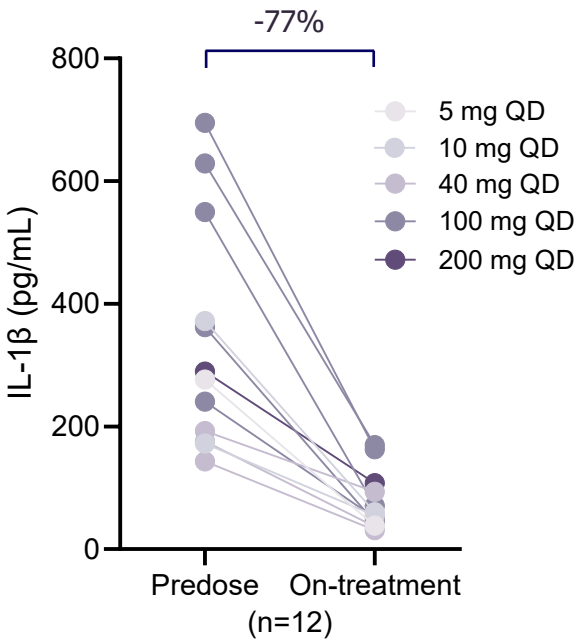
* Values correspond to the best response of hsCRP of day 6, 7, 9 and 14 combined across all MAD dose levels

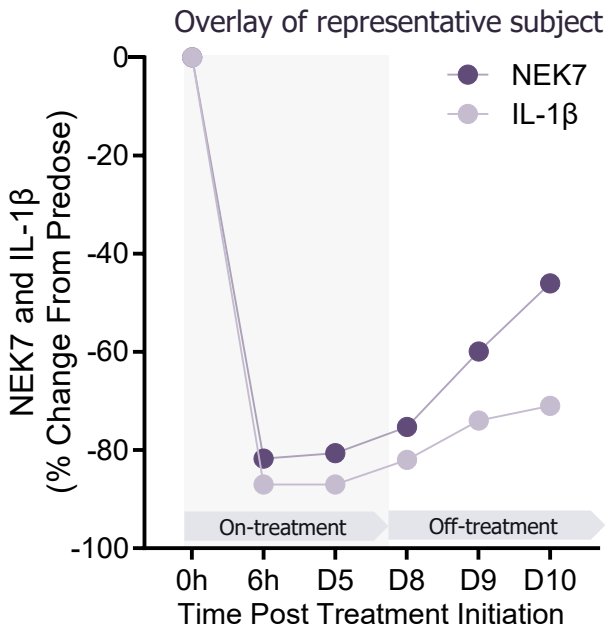
Multiple Daily Doses of MRT-8102 Led to Reductions of IL-6 and IL-1β

55% reduction of endogenous IL-6 plasma levels
Baseline CRP ≥1 mg/L



Rapid and sustained reduction of IL-1β after ex-vivo stimulation
Baseline CRP ≥1 mg/L





Significant reduction in median IL-6 levels to below CV risk threshold noted in subjects with elevated CRP

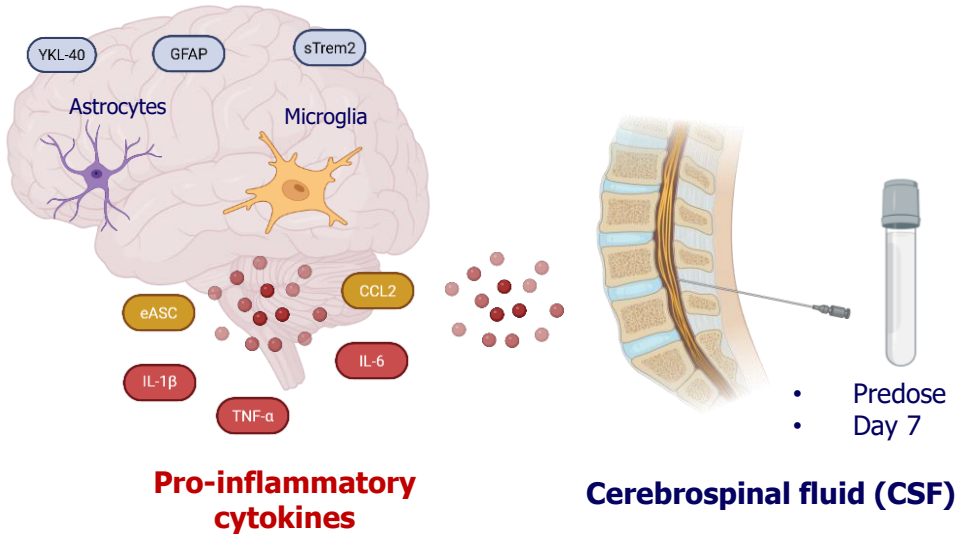
~80% inhibition in IL-1β secretion noted in subjects with elevated CRP at baseline at doses ranging from 5 – 200 mg

Suppression in IL-1β secretion correlates with NEK7 degradation across all time points

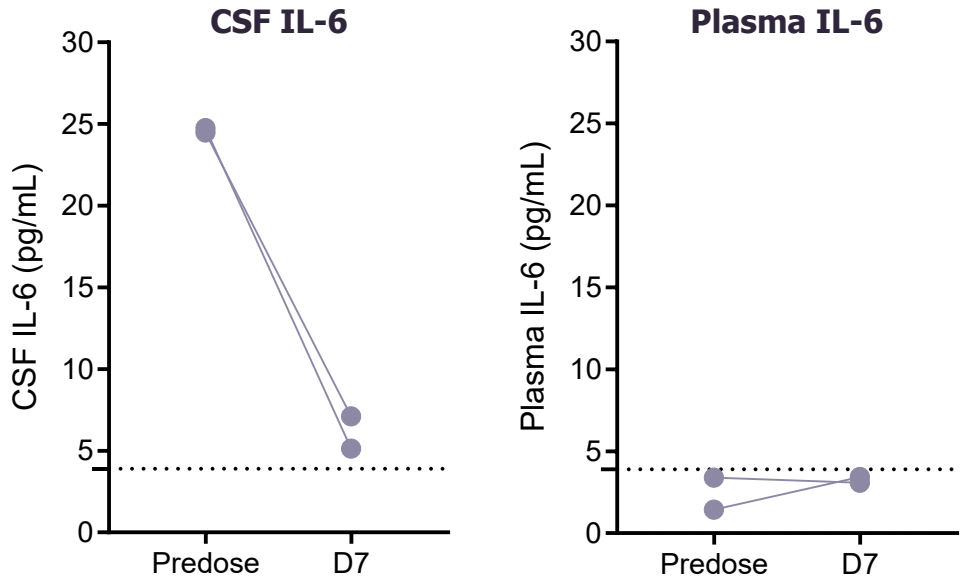
* Ridker PM et al. European Heart Journal (2018)

MRT-8102 Treatment Reduced IL-6 Levels in CSF Consistent with CNS Penetration

Cerebrospinal fluid (CSF) collection
(100 mg QD)



75% reduction of CSF IL-6
in 2 subjects with elevated levels at baseline



100 mg dose achieved levels of MRT-8102 in CSF consistent with pharmacologically active concentrations

Significant decrease in CSF IL-6 noted in two subjects with elevated baseline levels following 7d administration

Plasma IL-6 levels at baseline for these two subjects were low suggesting CNS/CSF-specific effects

Dotted line indicates median established with CSF and plasma samples derived from healthy volunteers (n=20)

CRP PoC (Part 3) Study of MRT-8102 in Subjects with Elevated CVD Risk

Study population

- Obesity (waist ≥ 40” for men or ≥ 35” for women and/or BMI ≥ 30)
- Elevated CRP ≥3 and <15 mg/L

Primary endpoint

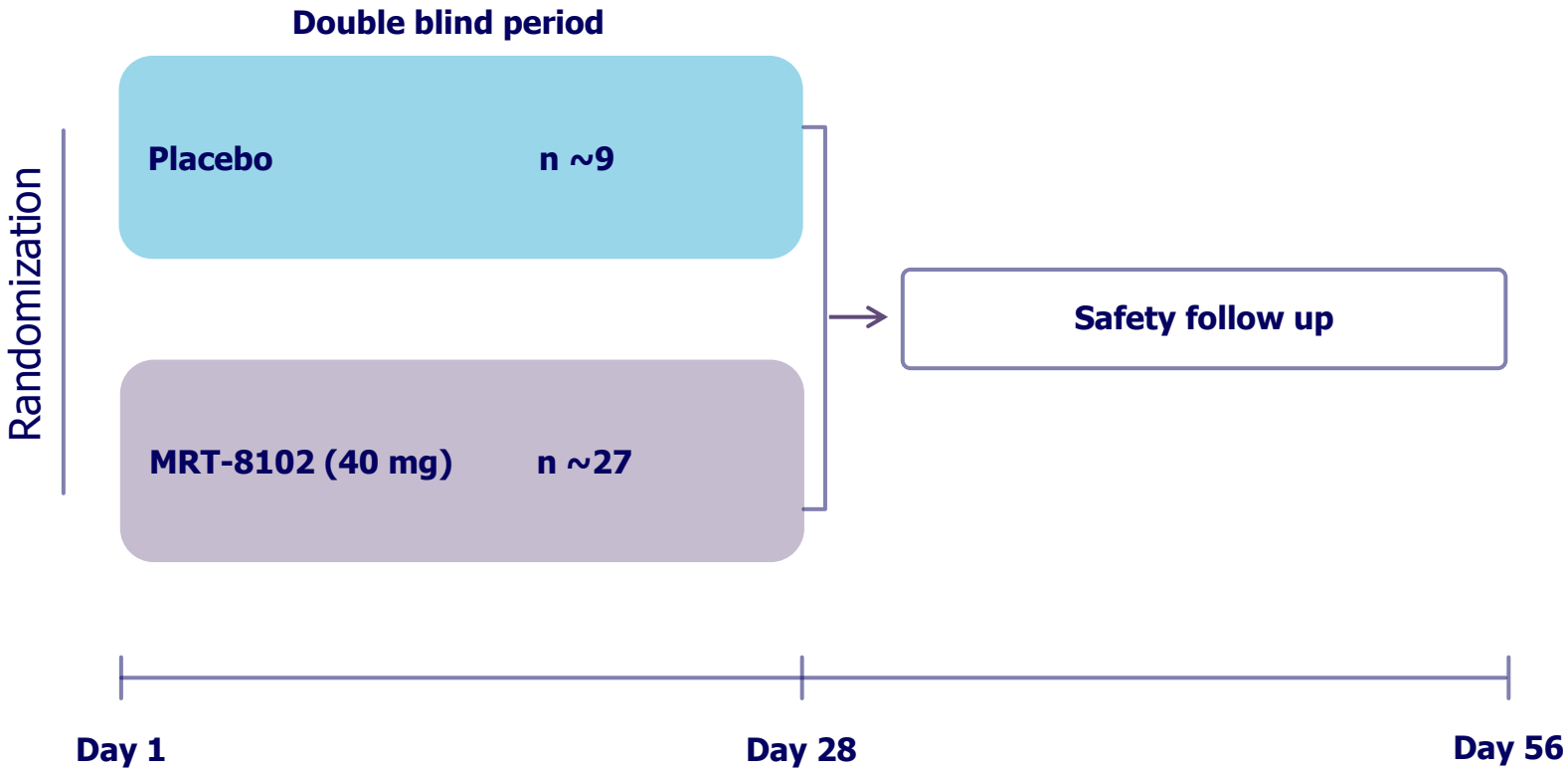
- Safety and tolerability of 28 days dosing

Secondary endpoints

- Change in CRP levels
- PK

Exploratory endpoints

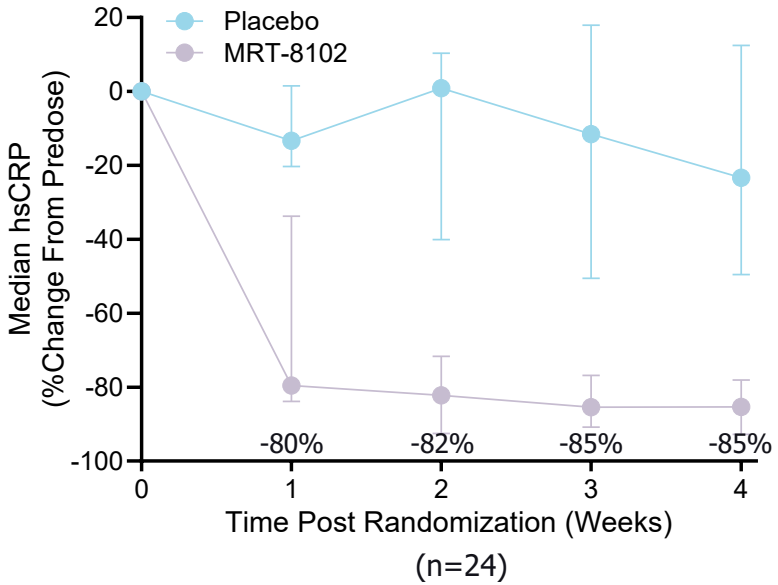
- PD (NEK7, IL-6, IL-18, Fibrinogen, SAA)
- Body weight
- Other markers of CV risk



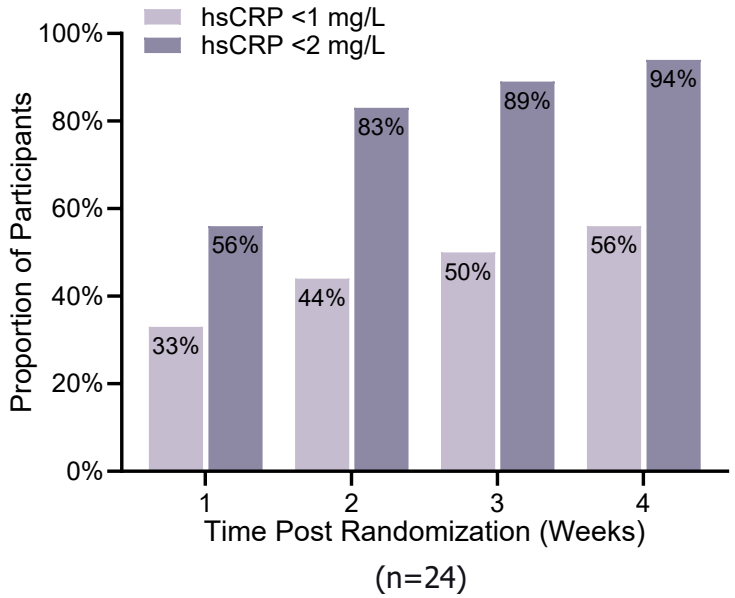
- CRP endpoint: absolute reduction and reduction to <2 mg/L
- CRP measurements: pre-dose, D1, D7, D14, D21, D28, D35

Interim Analysis of CRP PoC Cohort Suggests MRT-8102 Induced Rapid Reductions of hsCRP and Fibrinogen

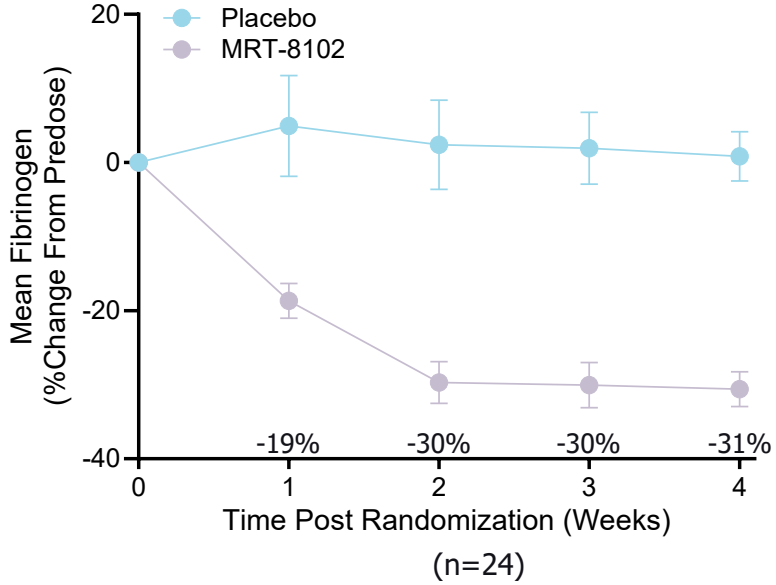
85% decrease of hsCRP after 4 weeks of dosing



94% of subjects show suppression of hsCRP to <2 mg/L*



Up to 31% reduction in fibrinogen after 4 weeks of treatment



85% reduction in CRP noted after 4 weeks of dosing that correlated well with sustained NEK7 degradation during the treatment period

94% of subjects show suppression of hsCRP to <2 mg/L after 4 weeks of dosing

Up to **31%** reduction in fibrinogen, an independent atherosclerotic risk factor**, noted during treatment period

* Baseline median CRP: Placebo - 4.0 mg/L; MRT-8102 - 6.3 mg/L; Baseline mean Fibrinogen: Placebo - 394 mg/dL; MRT-8102 – 431 mg/dL

** Meade TW et al. Lancet (1986); Kannel WB et al. JAMA (1987); Fibrinogen Studies Collaboration, JAMA (2005)

Summary of Blinded Safety Data

As of data cutoff of December 23, 2025, 112 subjects completed dosing:

- SAD/MAD completed, 88 HV participants enrolled
- Part 3 (CRP PoC) ongoing, 24 participants with elevated CVD risk and high CRP levels have concluded hsCRP assessment and are included in the analysis

Safety Profile:

- Well tolerated with favorable safety profile with no SAEs
- Treatment-emergent AEs were mild to moderate
- No evidence of increased infection risk
- No dose dependency
- Evaluation and data collection ongoing for Part 3*

* One participant in Part 3 was diagnosed with asymptomatic, acute infectious hepatitis A while on study (unknown if participant received MRT-8102 or placebo). Participant experienced a transient ALT elevation equivalent to a Gr 3 that improved while on treatment.

GFORCE-1 Study: Dose Exploration of MRT-8102 in Subjects with Elevated CVD Risk

Study population

- Obesity (waist ≥ 40" for men or ≥ 35" for women and/or BMI ≥ 30)
- Elevated CRP ≥3 and <15 mg/L

Primary endpoint

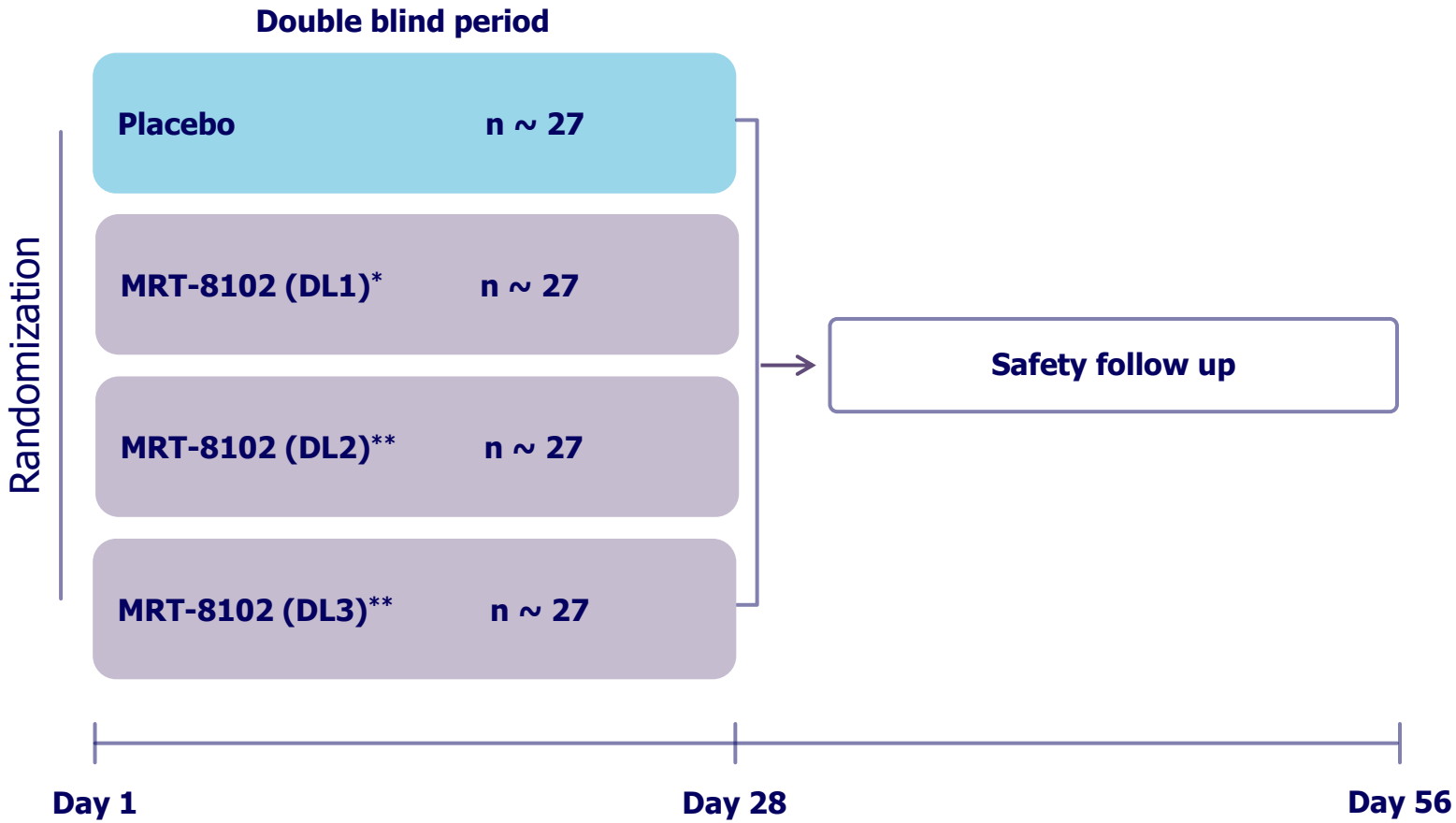
- Safety and tolerability of 28 days dosing

Secondary endpoints

- Change in CRP levels
- PK

Exploratory endpoints

- PD (NEK7, IL-6, IL-18, Fibrinogen, SAA)
- Body weight
- Other markers of CV risk



CRP endpoint: absolute reduction and reduction to <2 mg/L
CRP measurements: pre-dose, D1, D7, D14, D21, D28, D35

Expanded dose exploration to accelerate Phase 2 ASCVD study: Ph 2 initiation planned in 2026

GFORCE, Glue for CRP Elimination
* DL of 40 mg (n=27) with corresponding placebo (n=9) completed enrollment
** Two additional DLs with corresponding placebo to be included in the next amendment

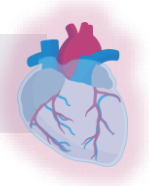
Additional Attractive Opportunities Exist for MRT-8102

Cardio-Immuno-Metabolic

Metabolic dysfunction-associated steatohepatitis

- **~7-9M** U.S. addressable population
- **Large** and **rapidly-growing** market; development effort mainly focuses on reducing steatosis
- Growing scientific evidence that **targeting inflammation** could provide **additional clinical benefit**

Recurrent pericarditis



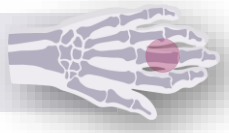
- **~40K** U.S. addressable population
- Significant unmet need for therapies with **improved safety and tolerability** profile given typically long duration of treatment
- Strong preference for **oral** treatments among patients

Rheumatology

Gout

- **~3M** U.S. addressable population, representing the approximately one-third of gout patients with stage 3+ CKD
- Gout SOC therapies are **contradicted** or **lack safety data** in CKD patients, imposing a major treatment challenge
- High unmet need among **chronic refractory** patients, who often suffer from **breakthrough flares**

Osteoarthritis



- **~3-4.5M** U.S. addressable population
- Canakinumab decreased rates of knee and hip replacement
- A **safe, novel** anti-inflammatory option for long-term management
- **4-5L** treatment option for patients refractory to SYSADOA* and NSAIDs

Allergic Diseases

Asthma

- **~1-3M** U.S. addressable population, with non-Type 2 disease representing 30-50% of severe asthma
- High NLRP3 inflammasome activity in non-type 2 inflammatory subtype
- Non-type 2 presents lack of response to corticosteroid therapy, responsive to anti-TSLP
- Potential to expand to Type 2 inflammatory subtype

*SYSADOA: symptomatic slow-acting drugs for osteoarthritis, such as chondroitin sulfate, glucosamine, hydrochloride, diacerin

Source: Clarivate; Alanaeme et al. (2022); Mazhar et al. (2024); Hagstrom et al. (2024); Zhu et al. (2012); Mohammed et al. (2019); Settipane et al. (2019); Pollack et al. (2022); Hinks et al. (2021); Esteban-Gorgojo et al. (2018); MRTx interviews, survey, and analysis

Summary and Pipeline Update

Summary and Next Steps

- SAD (48 healthy volunteer subjects) and MAD (40 healthy volunteer subjects) cohorts completed with no adverse safety signals, ~80-90% NEK7 degradation noted in T cells at all dose levels tested, and compelling reduction in hsCRP in subjects with elevated baseline CRP levels

- Part 3 (CRP PoC) of Phase 1 study exploring 40 mg MRT-8102 in high-risk CVD subjects (obesity/elevated CRP) is ongoing and 24 subjects have been evaluated up to end of week 4. Preliminary data for these subjects showed:
    - 85% sustained reduction of hsCRP through end of week 4
    - 94% subjects achieved reduction of hsCRP levels to <2 mg/L* after 4 weeks of dosing (baseline hsCRP level of 6.3 mg/L)
    - 31% reduction of fibrinogen after 4 weeks of dosing
    - No SAEs, no severe AEs as of data cut off date of 12/23/25, evaluation ongoing

- Study (now named GFORCE-1) will be expanded and additional dose levels will be explored to accelerate development in ASCVD; data expected in H2 2026, with plans to initiate Phase 2 GFORCE-2 study of MRT-8102 in ASCVD in 2026

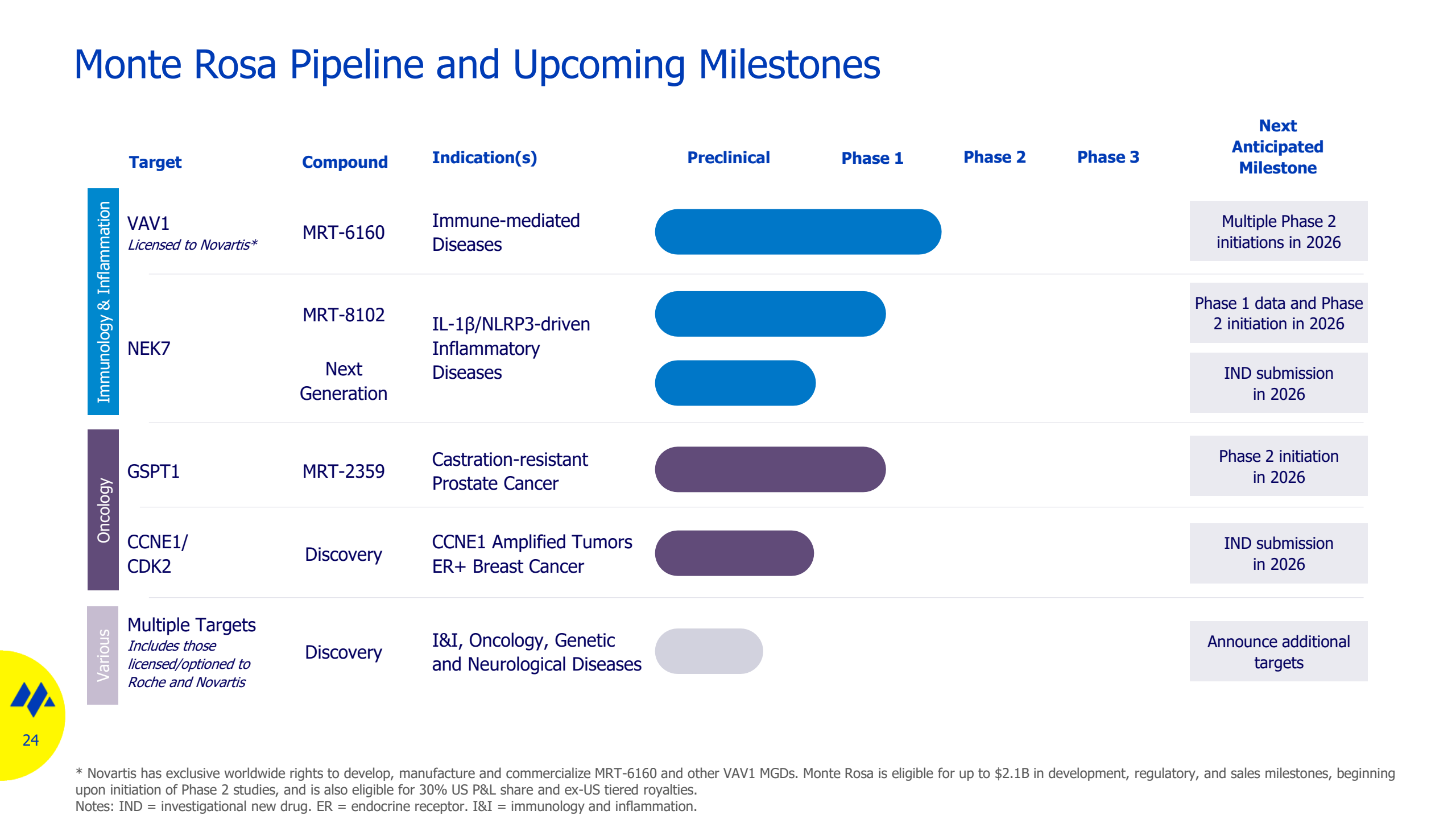
- Monte Rosa will expand its NEK7 program to next-generation MGDs with attractive properties supporting a range of indications

- Monte Rosa considering to advance MRT-8102 or next-generation MGD into multiple other indications

*hsCRP levels of >2 mg/L are associated with elevated CVD risk

Monte Rosa Pipeline and Upcoming Milestones

	Target	Compound	Indication(s)	Next Anticipated Milestone
Immunology & Inflammation	VAV1 *Licensed to Novartis**	MRT-6160	Immune-mediated Diseases	Multiple Phase 2 initiations in 2026
	NEK7	MRT-8102	IL-1β/NLRP3-driven Inflammatory Diseases	Phase 1 data and Phase 2 initiation in 2026
		Next Generation		IND submission in 2026
Oncology	GSPT1	MRT-2359	Castration-resistant Prostate Cancer	Phase 2 initiation in 2026
	CCNE1/ CDK2	Discovery	CCNE1 Amplified Tumors ER+ Breast Cancer	IND submission in 2026
Various	Multiple Targets *Includes those licensed/optioned to Roche and Novartis*	Discovery	I&I, Oncology, Genetic and Neurological Diseases	Announce additional targets



* Novartis has exclusive worldwide rights to develop, manufacture and commercialize MRT-6160 and other VAV1 MGDs. Monte Rosa is eligible for up to $2.1B in development, regulatory, and sales milestones, beginning upon initiation of Phase 2 studies, and is also eligible for 30% US P&L share and ex-US tiered royalties.
Notes: IND = investigational new drug. ER = endocrine receptor. I&I = immunology and inflammation.

Q&A

Thank you

Monte Rosa Therapeutics