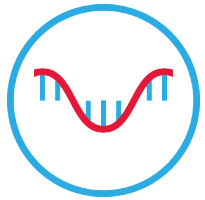
Moderna Science Day 2026

June 25, 2026





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Forward-looking statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including statements regarding: the potential of Moderna's mRNA platform and promise as a multi-modality biotechnology company; Moderna's potential three commercial franchises; Moderna's multi-year revenue growth plan, including up to 10 percent growth in 2026; 2026 value drivers; Moderna's anticipated cash breakeven by 2028; Moderna's ability to use data, AI and machine learning, and robotics to drive innovation; anticipated infectious disease vaccine approvals and launches; anticipated geographic expansion; Moderna's late-stage pipeline opportunities in intismeran and propionic acidemia; Moderna's emerging oncology programs; the potential of mRNA-4194 to address Lynch syndrome and prevent cancer from occurring; the encouraging early clinical signal with mRNA-2808; Moderna's potential to lead in autoimmunity; the potential for mRNA-6007 to unlock a scalable in vivo CAR-T modality; Moderna's regulatory filings for approval; Moderna's ongoing and planned clinical studies; and anticipated progress and milestones for Moderna's programs, including potential near-term data and other catalysts. In some cases, forward-looking statements can be identified by terminology such as "will," "may," "should," "could," "expects," "intends," "plans," "aims," "anticipates," "believes," "estimates," "predicts," "potential," "continue," or the negative of these terms or other comparable terminology, although not all forward-looking statements contain these words. The forward-looking statements in this presentation are neither promises nor guarantees, and you should not place undue reliance on these forward-looking statements because they involve known and unknown risks, uncertainties, and other factors, many of which are beyond Moderna's control and which could cause actual results to differ materially from those expressed or implied by these forward-looking statements. These risks, uncertainties, and other factors include, among others, those risks and uncertainties described under the heading "Risk Factors" in Moderna's Annual Report on Form 10-K for the fiscal year ended December 31, 2025, filed with the U.S. Securities and Exchange Commission (SEC), and in subsequent filings made by Moderna with the SEC, which are available on the SEC's website at www.sec.gov. Except as required by law, Moderna disclaims any intention or responsibility for updating or revising any forward-looking statements contained in this presentation in the event of new information, future developments or otherwise. These forward-looking statements are based on Moderna's current expectations and speak only as of the date of this presentation.

Introduction

Stéphane Bancel
Chief Executive Officer

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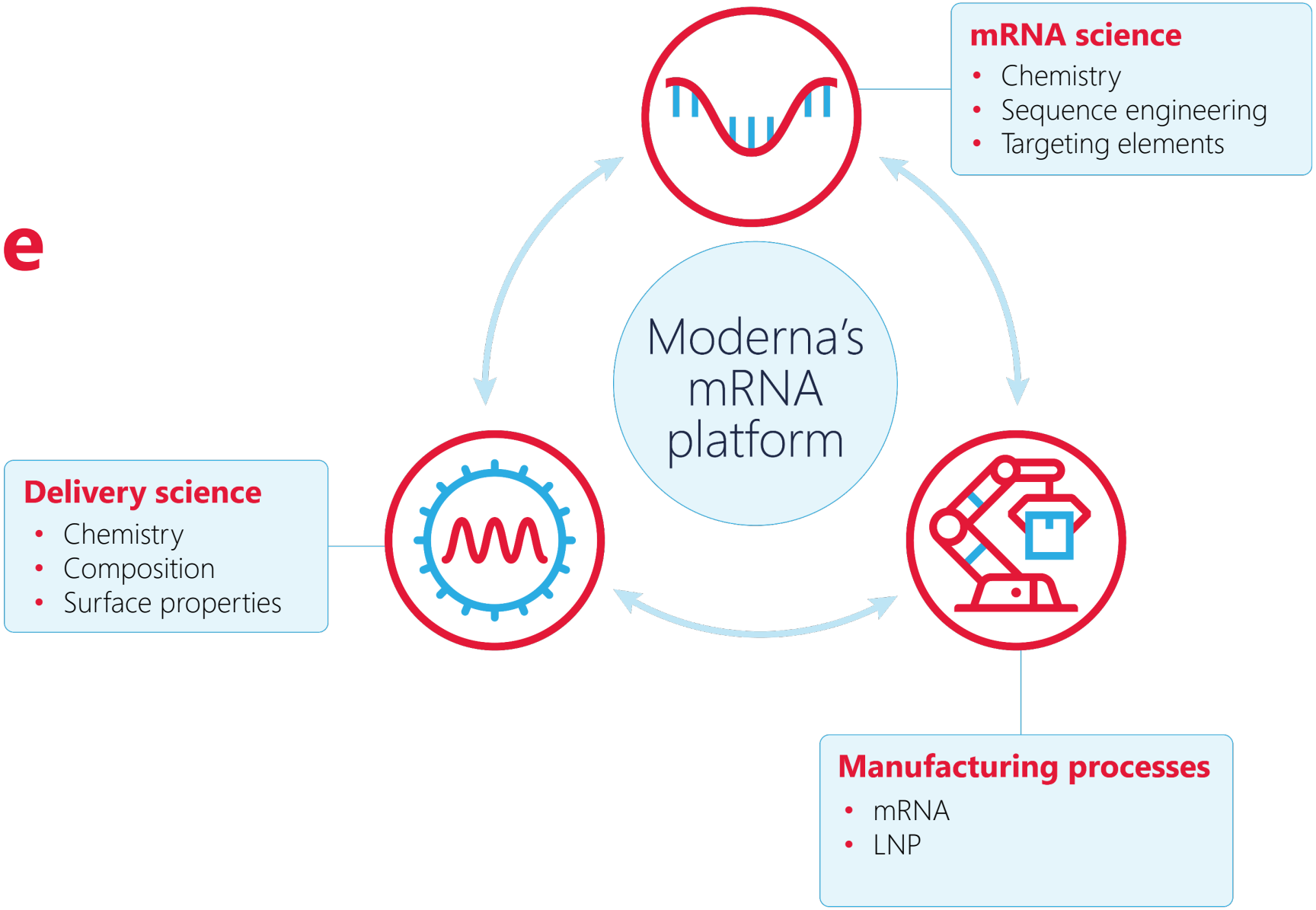
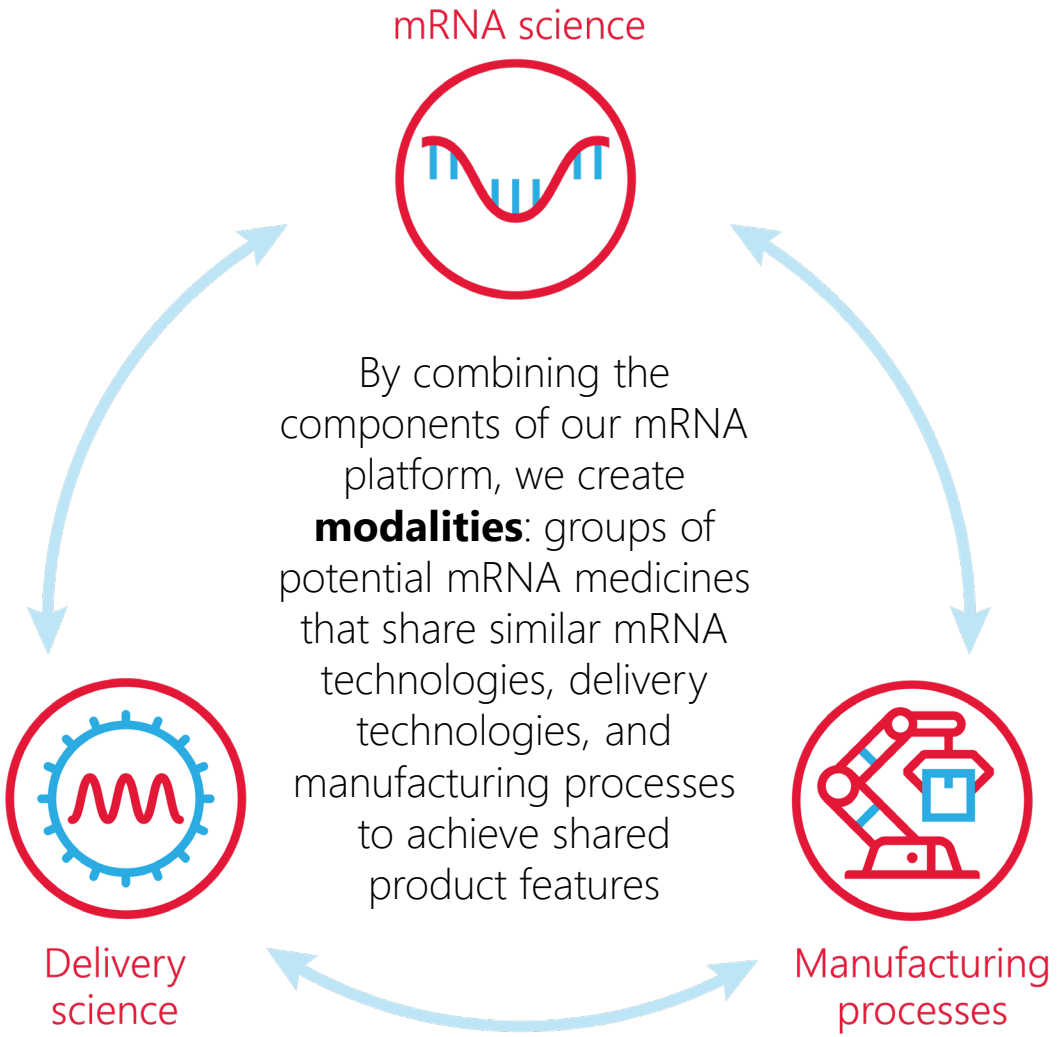
Our mission

Deliver the greatest possible impact to **people** through mRNA **medicines**

Our mRNA platform is built on three integrated pillars



Platform expertise enables multiple modalities



Proof-of-concept data from sentinel programs de-risk modalities and can accelerate development plans

We have three established modalities

MODALITY
Infectious disease vaccines

- **COVID-19 (Spikevax™)**
  approved and commercialized
- **RSV (mRESVIA™)**
  approved and commercialized
- **COVID-19 (mNEXSPIKE™)**
  approved and commercialized
- **Flu + COVID (mCOMBRIAX™)**
  approved
- **Flu**
  filed for approval
- **Norovirus**
  In Phase 3 trial

MODALITY
Intismeran autogene

- **Melanoma**
  Adjuvant: Phase 3 trial
  First-line metastatic: Phase 2 trial
- **Non small cell lung cancer**
  • Adjuvant: Phase 3 trial
  • Adjuvant non-pCR post-neoadjuvant treatment: Phase 3
  • Adjuvant Stage 1: Phase 3
  • First-line metastatic squamous: Phase 2
- **Renal cell cancer**
  • Adjuvant: Phase 2 trial
- **Bladder cancer**
  • Adjuvant muscle-invasive: Phase 2 trial
  • Adjuvant non-muscle-invasive: Phase 2 trial

MODALITY
Rare disease therapeutics

- **PA**
  Registrational trial
- **MMA**
  Phase 2 trial

mRNA platform

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These established modalities represent our first horizon

Horizon 1: Established modalities

Infectious disease vaccines

Potential upcoming catalysts

- U.S. flu vaccine approval
- Norovirus Phase 3 data

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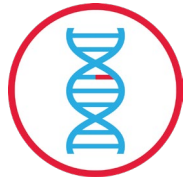
Potential upcoming catalysts

- Phase 3 adjuvant melanoma data; event-driven

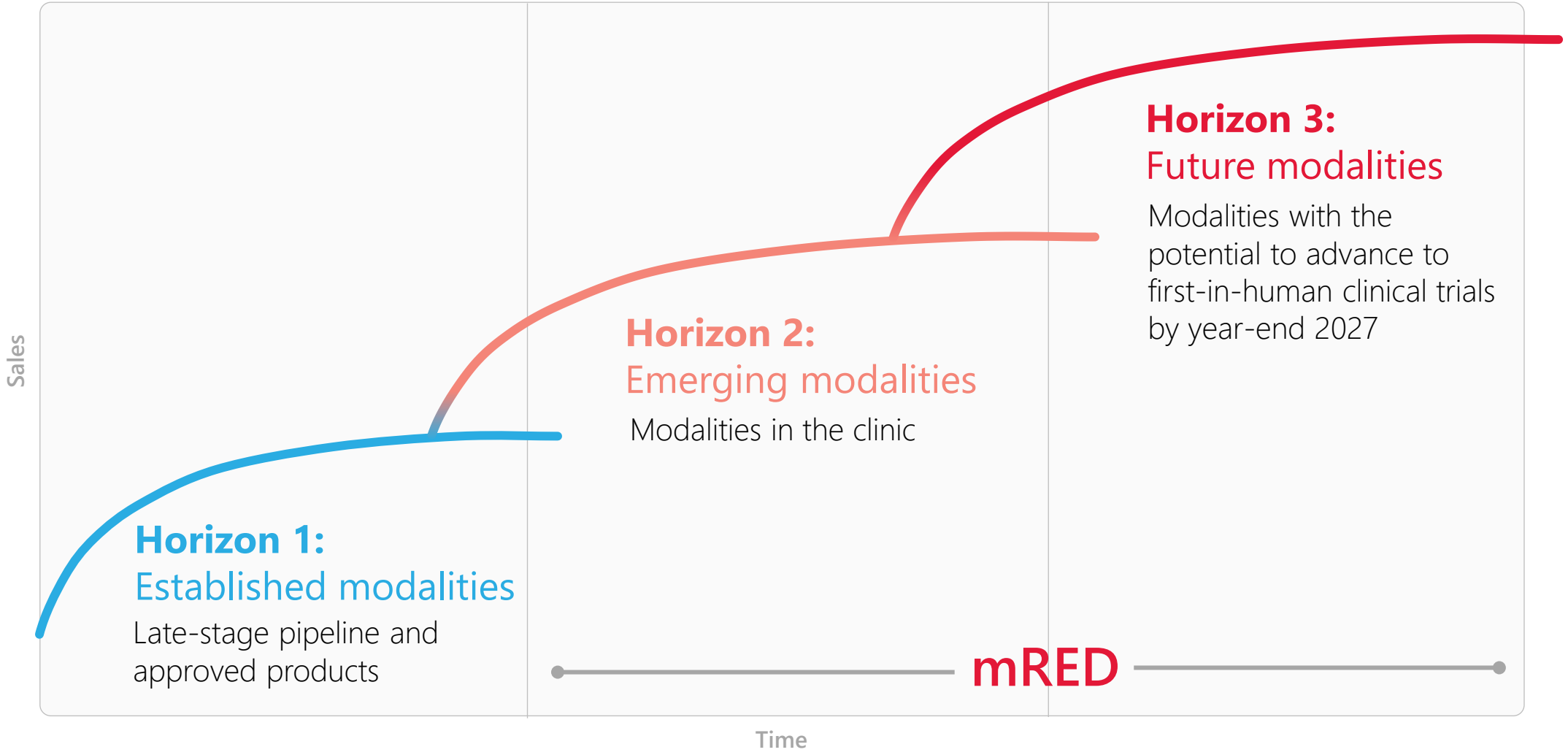


Rare disease therapeutics

Potential upcoming catalysts

- PA registrational study data

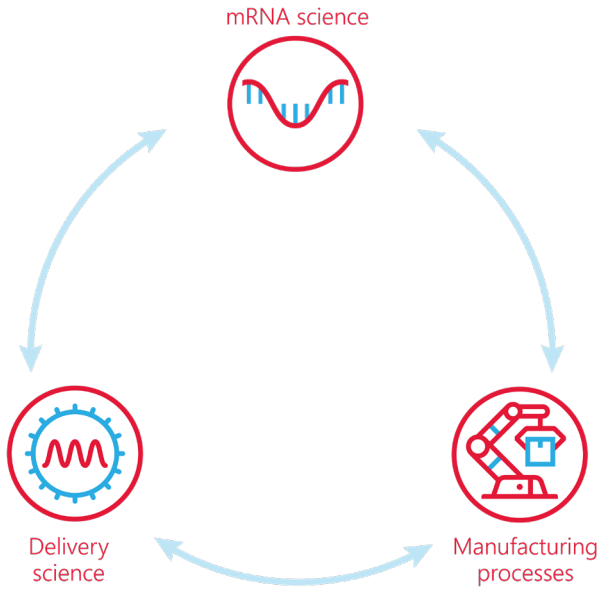
The next horizons are led by Moderna Research & Early Development (mRED)

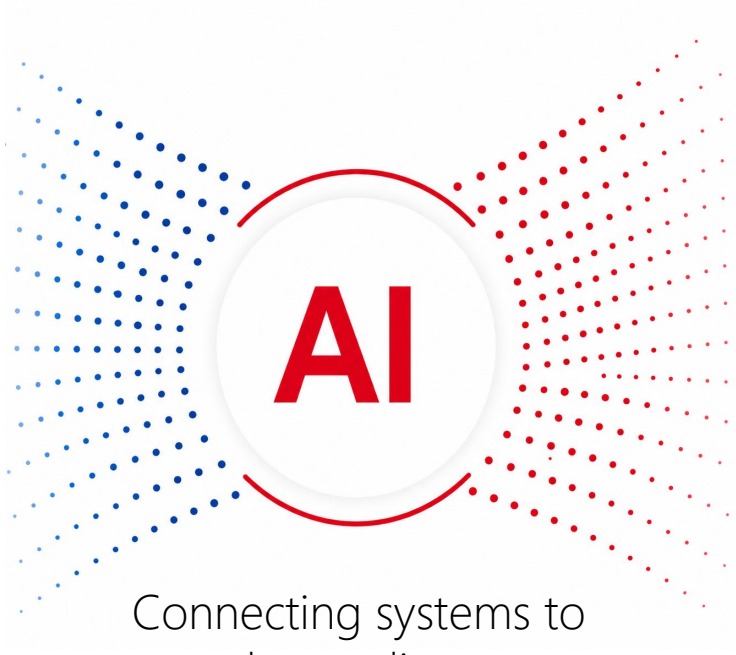


Our AI strategy turns platform data and biological insight into faster innovation

PLATFORM DATA

mRNA technology





Connecting systems to accelerate discovery

INSIGHTS

Biology

- Human biology and disease understanding
- Molecular and clinical data
- Clinical outcomes

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Agenda

Introduction	**Stéphane Bancel** Chief Executive Officer
mRED	**David Berman, MD PhD** Chief Development Officer **Rose Loughlin, PhD** Executive Vice President, Research
Cancer Antigen Therapies	**Kristine McKinney, PhD** Vice President and Head, Cancer Vaccines and Bioinformatics Research **David Church, MBChb, FRCP, DPhil** Professor, Nuffield Department of Medicine, University of Oxford; Cancer Research UK Senior Cancer Research Fellow; Honorary Consultant Medical Oncologist **Sarah Keidel, BM, BCh, MSc** Executive Director, Program Leader, Oncology Development
	Break
T-cell engagers	**Lin Guey, PhD** Chief Scientific Officer, Therapeutics Research
EBV therapeutic (mRNA-1195)	**Sumana Chandramouli, PhD** Executive Director, Program Leader, Infectious Disease
***In vivo* CAR-T**	**Lin Guey, PhD** Chief Scientific Officer, Therapeutics Research
Platform technology	**David Huss, PhD** Chief Technology Officer, Research
Conclusion	**Stéphane Bancel** Chief Executive Officer

mRED

David Berman, MD PhD
Chief Development Officer

Rose Loughlin, PhD
Executive Vice President, Research

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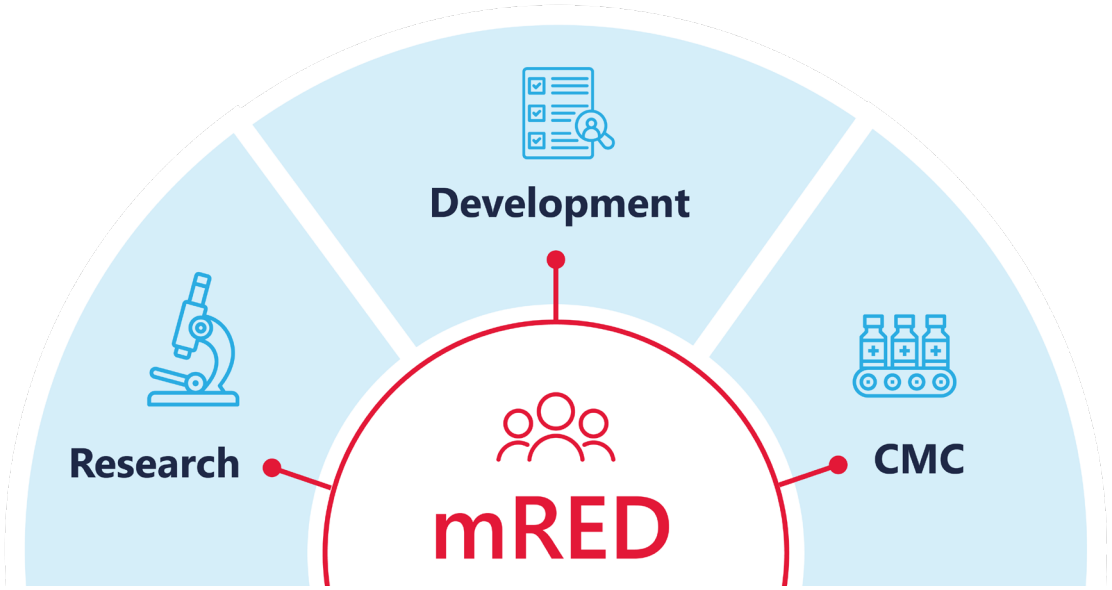
mRED: Moderna's biotech engine for rapid innovation



Decision-making forum focused on moving new modalities from idea to clinical proof-of-concept

Connects strategy, investment, and clinical execution to one senior-leader decision body

Accelerates decision-making with faster, data-triggered decisions

Ensures dedicated focus on the innovation engine fueling the pipeline

Enables science-driven start / stop / continue decisions across mRED

mRED builds Moderna's next growth drivers by advancing new, differentiated, platform-enabled modalities

mRED

Future modalities

Novel modalities and platform technology beyond what Moderna does today

Moves quickly from idea to evidence, generating preclinical data

Emerging modalities

New modalities currently in the clinic

With proof-of-concept, modalities graduate from mRED into late-stage development, either within our pipeline or through partnerships, while platform learnings benefit all of Moderna

Established modalities

Late-stage pipeline and approved products

Innovates on established modalities and model for end-to-end path from discovery through commercialization

A disciplined framework for building the mRED portfolio

Platform advantage
Advance modalities where our platform technology offers distinct competitive advantages and program differentiation

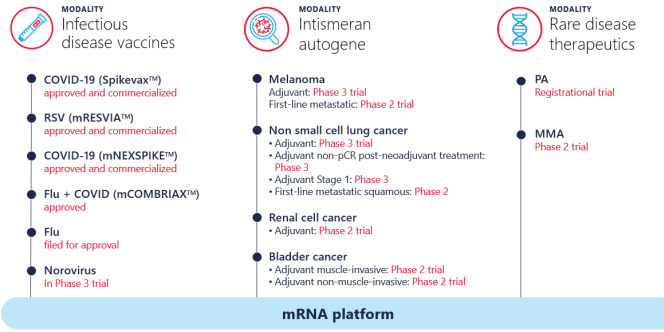
Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction

Portfolio balance
Build a diversified portfolio by balancing breadth across distinct areas of opportunity with disciplined expansion within each modality

Platform competitive advantage

- Multiplexing
- Intracellular proteins
- Transmembrane proteins
- Complex proteins
- T cell response

Proof-of-concept data from sentinel programs de-risk modalities and can accelerate development plans



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mRED portfolio

Horizon 2 — Emerging modalities

Cancer antigen therapies

mRNA-4359

mRNA-4106

mRNA-4200

mRNA-4194

Cell therapy enhancers

mRNA-4203

T-cell engagers (surface antigen)

mRNA-2808

mRNA-2151

Multiple sclerosis therapeutic

mRNA-1195

Horizon 3 — Future modalities

In vivo CAR-T

mRNA-6007

T-cell engagers (intracellular targets)

In vivo CAR-M

Tolerizing therapy

Cancer Antigen Therapies

mRNA-4106: Solid tumors

mRNA-4200: Solid Tumors

mRNA-4194: Lynch Syndrome

Kristine McKinney, PhD
Vice President and Head, Cancer Vaccines and Bioinformatics Research

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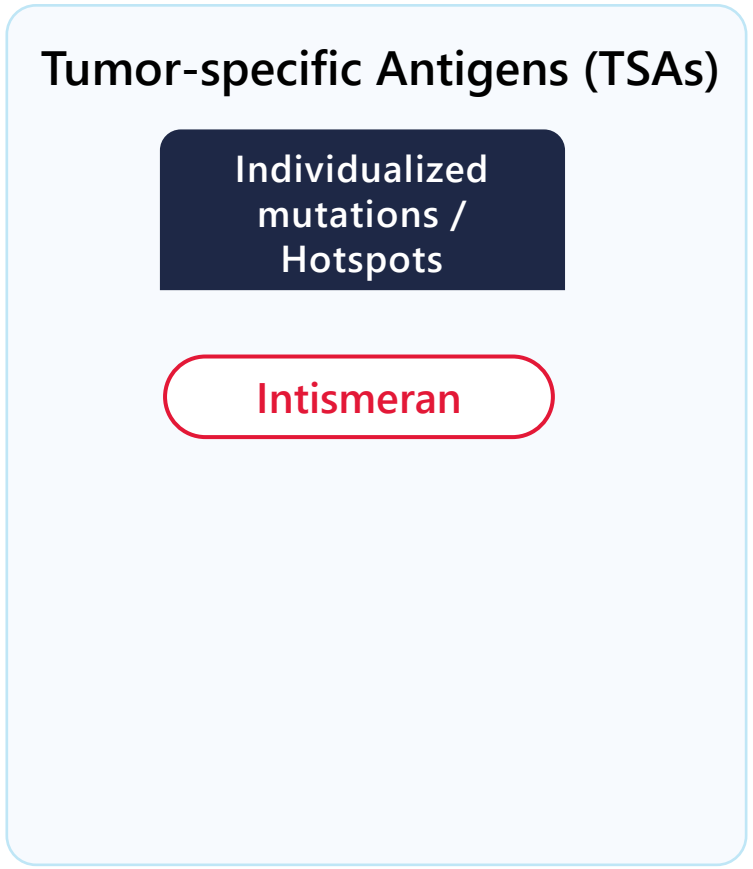
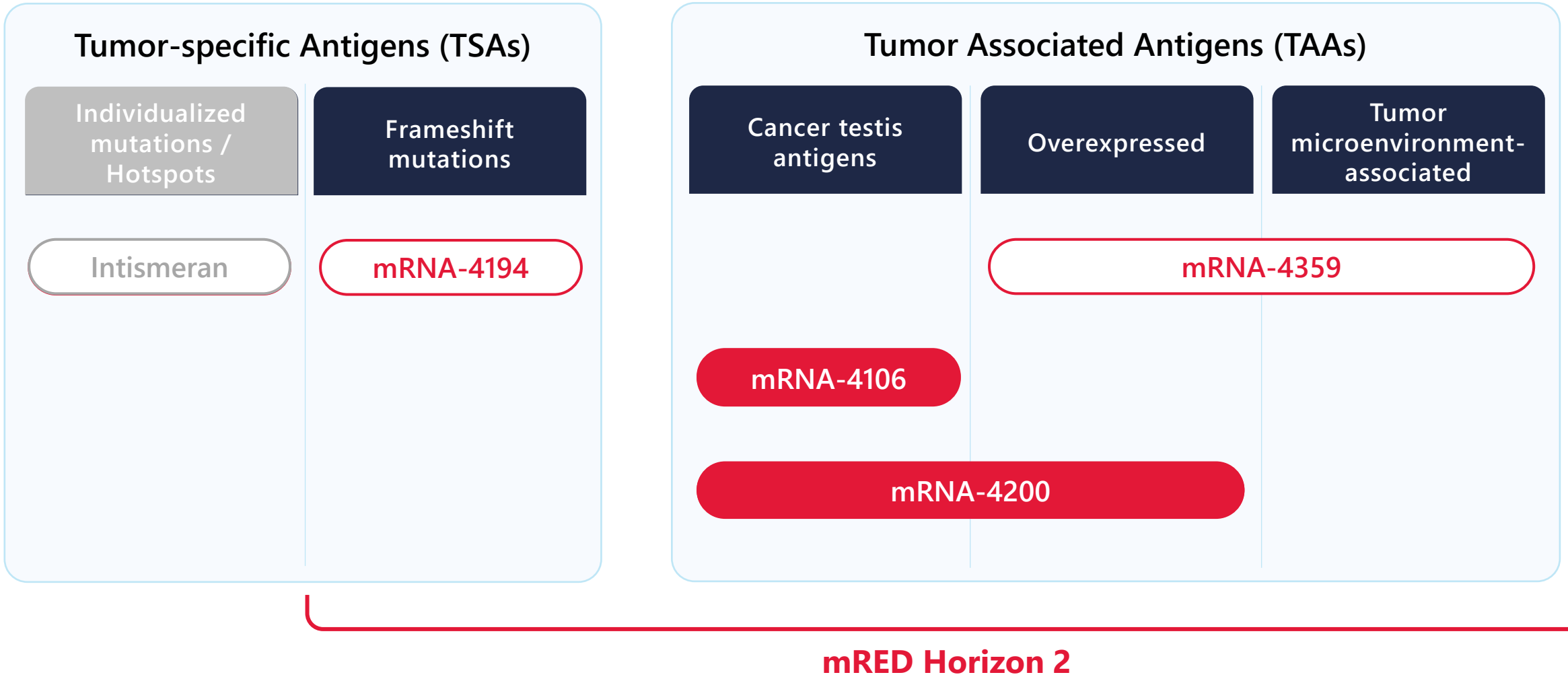
Intismeran's development path de-risks our approach to antigen-targeted cancer therapies



Cancer antigen therapies are designed to target distinct tumor biology



Off-the-shelf cancer antigen therapies: portfolio design principles

1

Optimize for population- and individual-level efficacy
Maximize coverage across patients (more responses) and within patients (deeper responses)

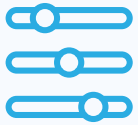
2

Manage biology risk
Prioritize validated antigens and optimize for immunogenicity

3

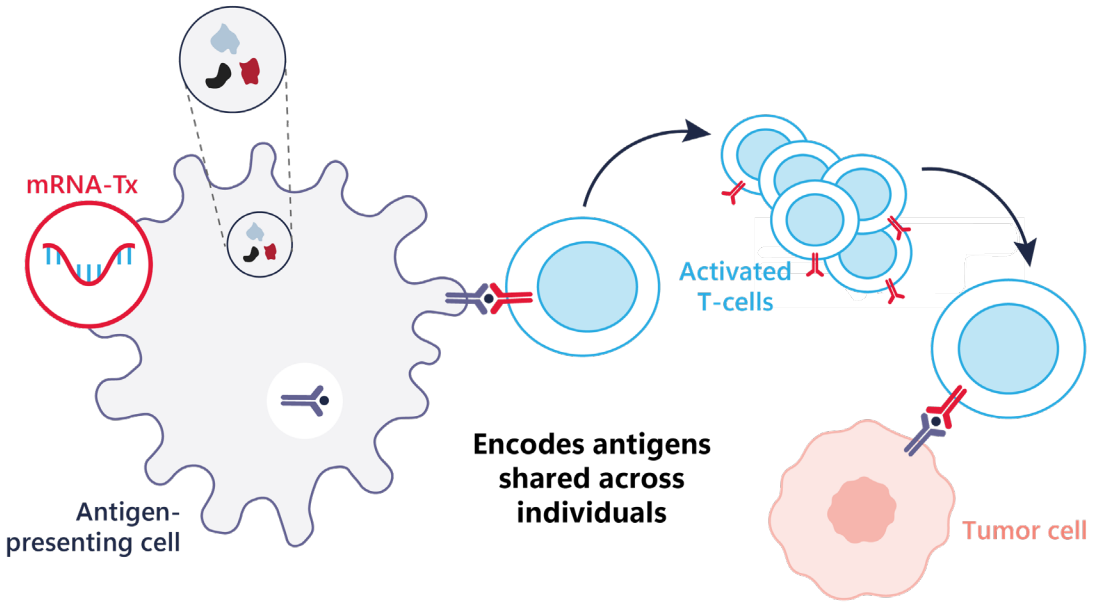
Establish safety
Safely explore design space in data driven manner

4



Enable optionality
Include targets that are also expressed in other tumors, enabling ability to scale and adapt

Design and mechanism of action for mRNA-4106 and mRNA-4200



Antigens highly tumor specific and validated

- Targets validated across human tumor tissues with low/no expression in normal tissues
- Preclinical data confirms successful antigen processing, HLA presentation and immunogenicity
- Whole antigen sequence format enhances immunogenicity and supports broad HLA coverage

Multiplexing overcomes limitations of single-targeting

- Reduces risk of tumor clone escape through single antigen loss
- Improves coverage in case of intratumoral and inter-patient antigen heterogeneity

Applicable across multiple tumor types

- Targets are expressed in multiple tumor types

mRNA-4106/mRNA-4200 trial design

Design
Single-arm, open-label, multi-center, dose escalation trial

Number of participants
Up to 42 adults with advanced solid tumors

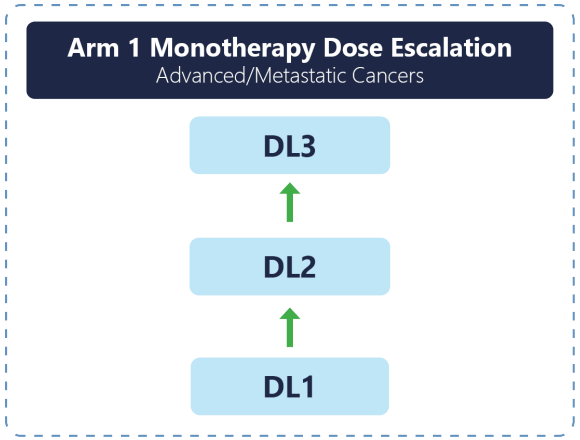
Vaccination schedule
- **mRNA-4106:** mRNA-4106 IM every 3 weeks for up to 9 cycles
- **mRNA-4200:** mRNA-4200 IM for 1 cycle (x 3 weeks), followed by a combination of mRNA-4200 with pembrolizumab for 9 cycles, and continuation of pembrolizumab alone for up to 2 years

Primary Objective:
Safety and tolerability of mRNA-4106 (Arm 1) and mRNA-4200 + pembrolizumab (Arm 2)

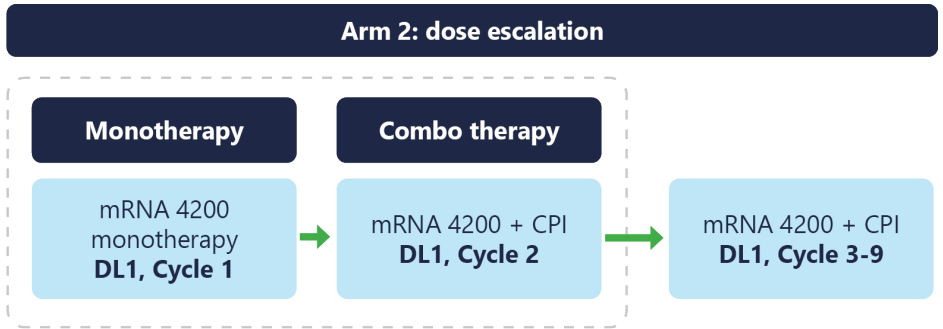
Key Exploratory Objectives:
- Anti-tumor activity of mRNA-4106 (Arm 1) and mRNA-4200 + pembrolizumab (Arm 2)
- T-cell immunogenicity of mRNA-4106 (Arm 1) and mRNA-4200 + pembrolizumab (Arm 2)
- PK, ADA of mRNA-4106 (Arm 1) and mRNA-4200 + pembrolizumab (Arm 2)

Duration
Study participants followed up for 90 days after last study injection

mRNA-4106

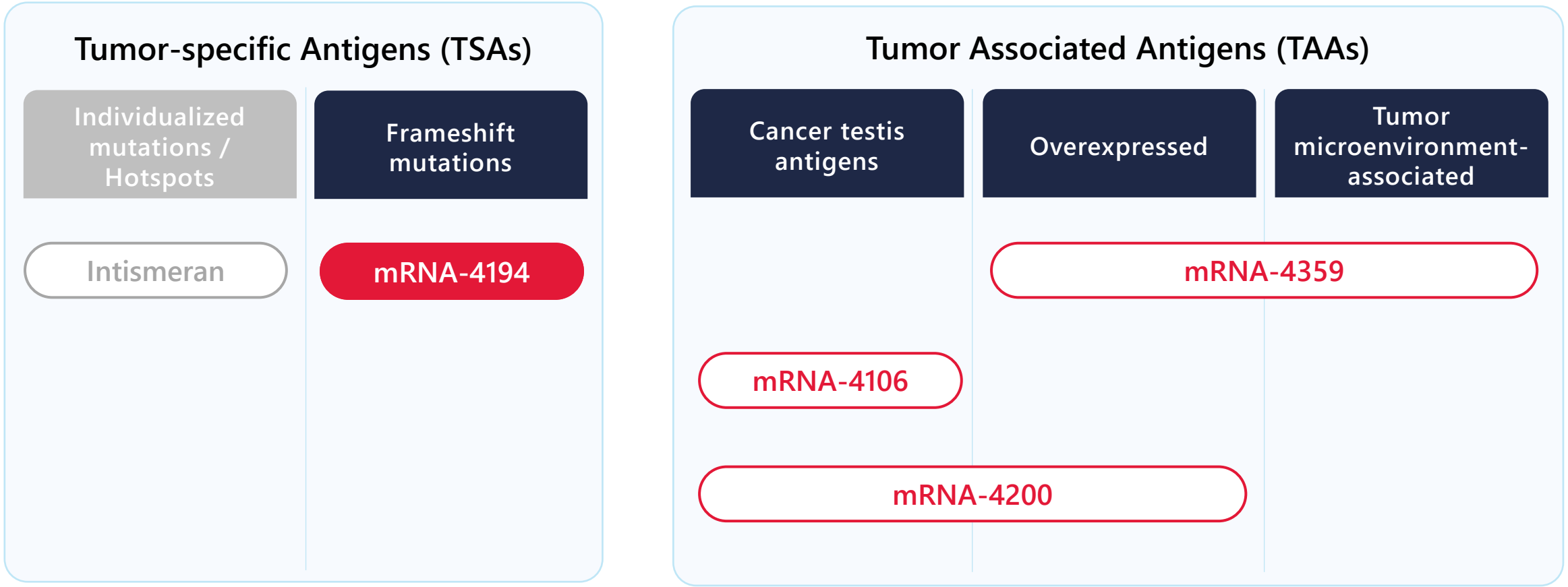


mRNA-4200

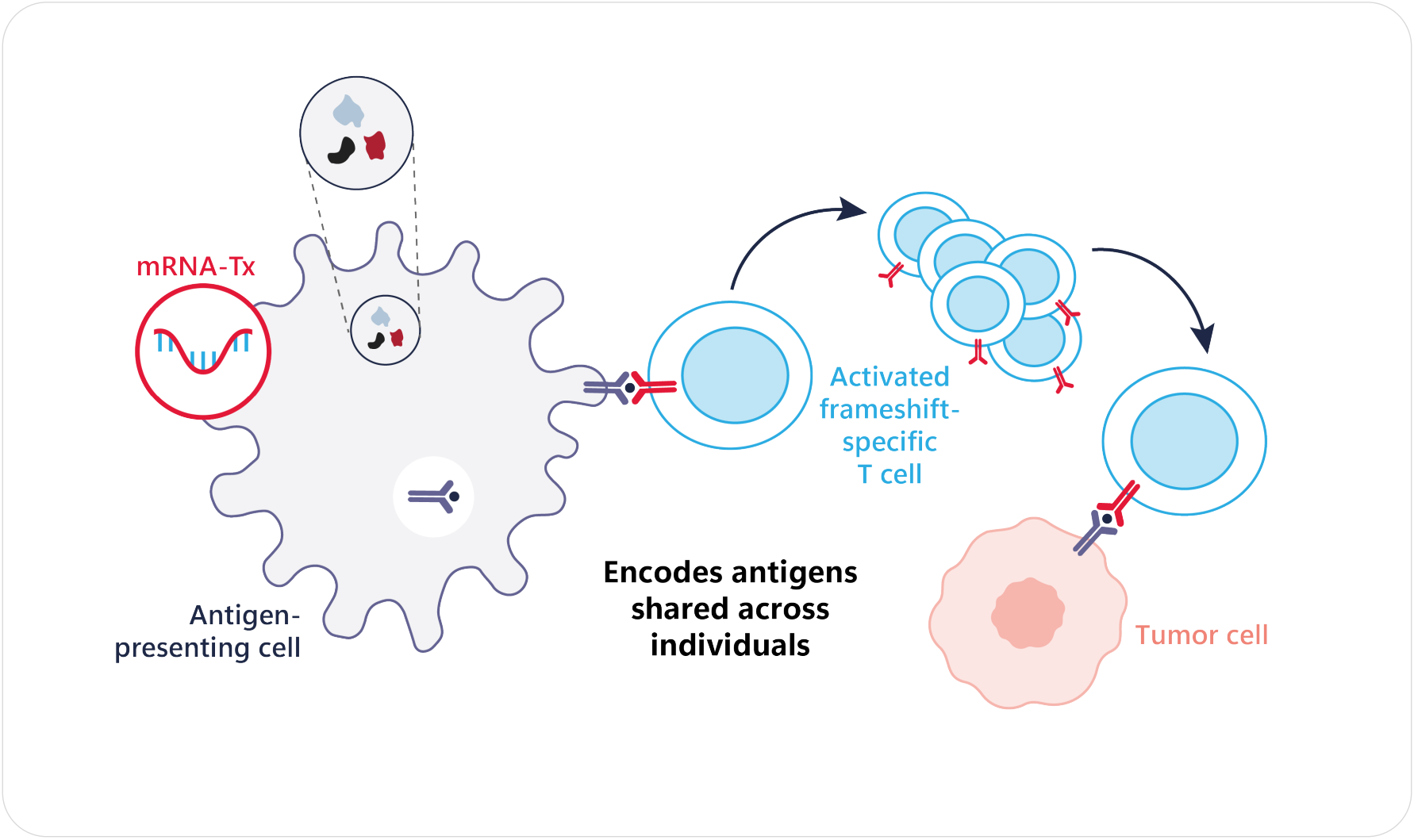


DLT window = 42 days (C1 and C2)

Cancer antigen therapies are designed to target distinct tumor biology



mRNA-4194 extends the potential of cancer antigen therapies into the prevention setting



David Church, MBChb, FRCP, DPhil

David Church is Professor of Precision Oncology and CRUK Senior Cancer Research Fellow at the Centre for Human Genetics, University of Oxford. He is also an Honorary Consultant Medical Oncologist at the Oxford Cancer Centre, and clinical lead for Lynch Syndrome across the Central and South GMSA (population 10.5m).

In addition to leading a research group focused on colorectal and endometrial cancers, he leads the Oxford LynchVax group and the Oxford-Moderna INTERCEPT-Lynch trial, the Genomics England 100KGP endometrial cancer domain, and is a member of several clinical trial translational research groups including TransSCOT and TransPORTEC.

Lynch Syndrome overview

David Church, MBChb, FRCP, DPhil
Professor, Nuffield Department of Medicine, University of Oxford
Cancer Research UK Senior Cancer Fellow
Honorary Consultant Medical Oncologist

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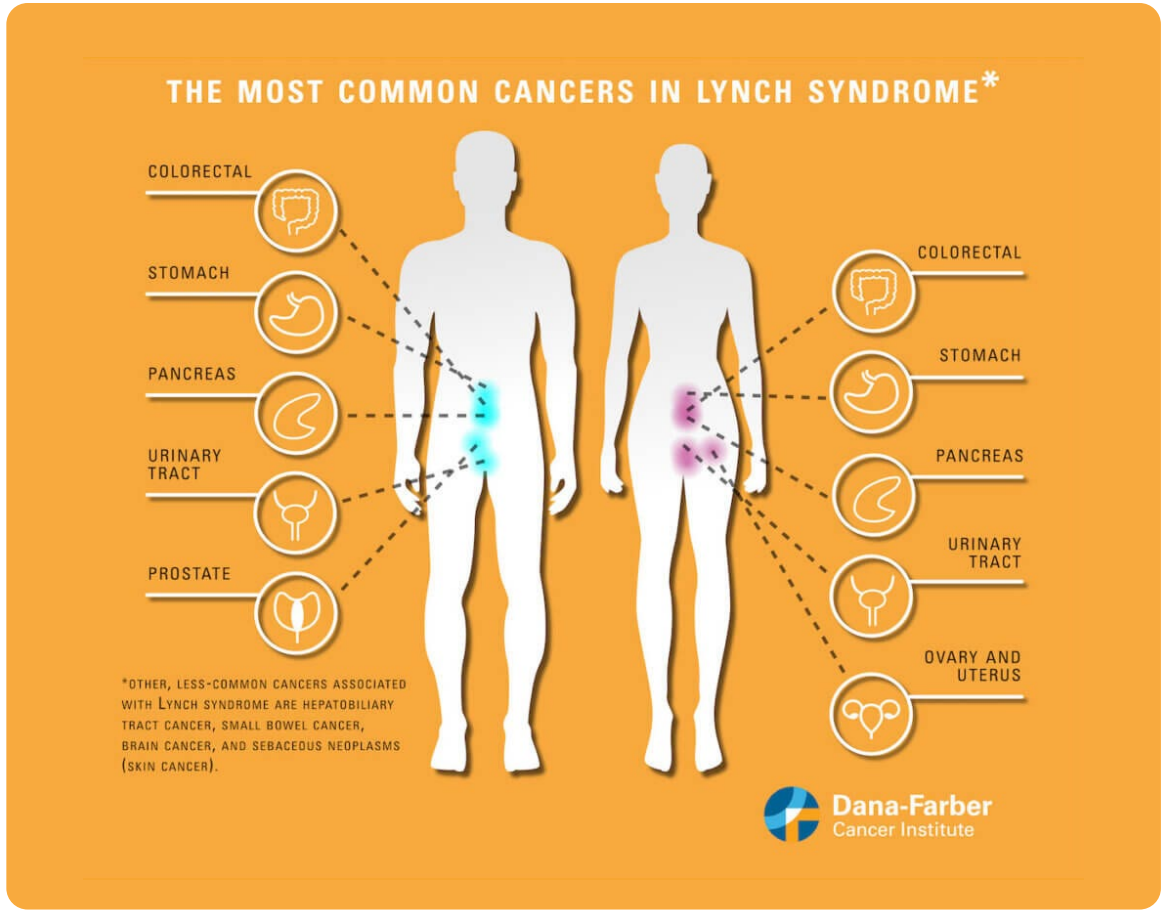
Lynch Syndrome: An inherited cancer predisposition syndrome

Inherited defects in DNA mismatch repair lead to the accumulation of frameshift mutations.

Individuals with Lynch syndrome face elevated lifetime risk of colorectal, endometrial, and other cancers.

Current management relies primarily on surveillance, with aspirin used by some patients as a preventive strategy to reduce colorectal cancer risk.

Frameshift mutations create a potentially targetable source of cancer-specific antigens.



Source: Dana Farber Institute; Monahan et al. 2019;

Lynch Syndrome: Epidemiology and cancer risk

- The true prevalence of LS in the general population is estimated at **1:279**[1] though diagnosed number may be much lower
- Risk of developing LS-associated cancers and age of onset differs based on variant. MLH1 and MSH2/EPCAM carriers confer the highest risk of cancers.

		General Population	MLH1	MSH2	MSH6	PMS2	EPCAM*
Approx. distribution in clinically ascertained LS			**15 – 40%**[1]	**20 – 40%**[1]	**12 – 35%**[1]	**5 – 25%**[1]	**<10%**
Lifetime Risk (%) *Avg. Age of Presentation (years)*	**Any cancer**	**40%**	**71 – 90%**[2]	**52 – 84%**	**58 – 73%**	**34 – 52%**[2]	*Limited data*
	Colorectum	**4.1%**[4] *66 – 69 yo*	**46 – 61%**[4] *44 yo*[1]	**33 – 52%**[4] *44 yo*[1]	**10 – 44%**[4] *42 – 69 yo*[1]	**8.7 – 20%**[4] *61 – 66 yo*[14]	**~75%**[4] -
	Endometrial	**3.1%**[4] *60 yo*	**34 – 54%**[4] *49 yo*[4]	**21 – 57%**[4] *47 – 48 yo*[4]	**16 – 49%**[4] *53 – 55 yo*[1]	**13 – 26%**[4] *49 – 50 yo*[4]	**~12%**[4] -
	Ovarian	**1.1%**[4] *55 – 64 yo*	**4 – 20%**[4] *46 yo*[4]	**8 – 38%**[4] *43 yo*[4]	**1 – 13%**[4] *46 yo*[4]	**1.3 – 3%** *51 – 59 yo*	*n/a*
	Bladder	**2.3%** *73 yo*	**2 – 7%**[4] *59 yo*[4]	**4.4 – 12.8%**[4] *59 yo*[4]	**1 – 8.2%**[4] *71 yo*[4]	**1 – 2.4%** *71 yo*	*n/a*
	Prostate	**4%** *66 – 69 yo*	**7%**[1] *59 – 63 yo*[1]	**16%**[1] *59 – 63 yo*[1]	**5%**[1] *59 – 63 yo*[1]	**5%**[1] *59 – 63 yo*[1]	*n/a*
	Pancreatic	**1.6%**[4] *70 yo*	**3 – 6%**[4] -	**1 – 3.5%**[4] -	**1 – 2%**[4] -	**≤1–2%**[4] -	*n/a*
	Gastric	**0.8%**[4] *68 yo*	**5 – 7%**[4] *52 yo*[4]	**0.2 – 9%**[4] *52 yo*[4]	**1 – 7.9%**[4] -	-	*n/a*
Risk of Subsequent Cancer (any site)		-	**80%**	**81%**	**58%**	-	*n/a*

*While EPCAM is not an MMR gene; rare 3' EPCAM mutations can silence adjacent MSH2, causing functional mismatch-repair deficiency. EPCAM-associated LS is uncommon and less well characterized, limited data exist.

Sources: 1. GeneReviews;; 2. Underkofler et al. Frontiers in Oncology 2023; 3. StatPearls; UpToDate; 4. NCCN

Lynch Syndrome: Testing guidelines in the UK

Since 2017, the National Institute for Health and Care Excellence (NICE) has recommended that all people diagnosed with colorectal cancer are tested for Lynch Syndrome using immunohistochemistry (IHC) or microsatellite instability (MSI) testing.

Since 2020, NICE also recommended testing for Lynch Syndrome in people who are diagnosed with endometrial cancer using IHC.

In 2021, NHS England published guidance to implement Lynch Syndrome testing pathways (right)

NHS England: Implementing Lynch Syndrome Pathways

1	**Stage 1:** Initial Tumour Test	1. Biopsy taken and cancer diagnosed / confirmed 2. Test tumour using immunohistochemistry (IHC) or Microsatellite instability (MSI). Initial tumour testing should be completed in time to inform treatment options
2	**Stage 2:** Germline Testing	3. Test suggests cancer could be caused by Lynch syndrome 4. If not already done, consent to perform germline testing 5. Perform germline testing. This test should take no longer than four weeks to complete.
3	**Stage 3:** Management of Index Case	6. If Lynch syndrome is confirmed, communicate results to patients and refer to genetics service who will upload patients to the Lynch registry portal for national registration and screening 7. Agree on a screening and management plan and refer to relevant services
4	**Stage 4:** Cascade Testing and Surveillance of Family Members	8. Cascade testing of at-risk family members

Sources: NICE HTG430, NICE DG27, NICE DG42, NICE HTG557, NHS England Implementing Lynch syndrome testing and surveillance pathways handbook v1.2, NHS England handbook, NHS England National Genomic Test Directory, NHS England explanation of the National Genomic Test Directory, Example NHS GMS clinical indication page for R210 Inherited MMR deficiency (Lynch syndrome)

Lynch Syndrome: Management

COLORECTAL CANCER

Colonoscopy	Screening colonoscopy is recommended to be given every 2 years, starting from the age of 25 for MLH1/MSH2 carriers, or 35 for MSH6/PMS2 carriers
Aspirin	Daily aspirin is recommended based on the CAPP2 study demonstrating that 600mg daily aspirin reduced risk of CRC in LS patients by approximately half vs. placebo. However, there is currently uncertainty about the optimum dosage of aspirin — the ongoing CAPP3 study aims to identify an optimum dose and may mitigate reluctance to prescribe higher doses of aspirin.
Colectomy (in CRC pts)	For patients with colorectal cancer and MLH1 or MSH2 mutations, decision to perform segmental versus total / near total colectomy should be adaptive and balance risk of metachronous cancer, functional consequence of surgery, and patient age and preference.

SCREENING FOR EXTRACOLONIC CANCERS
has not been shown to be effective, and is not typically recommended. The following can be considered:

Gynecological	Women who have completed their family may consider risk-reducing surgery to remove the uterus, fallopian tubes, and/or ovaries.
Gastric	Screening for and eradication of H. pylori may reduce stomach cancer risk.
Lifestyle changes	i.e., diet, smoking, alcohol intake
Prompt evaluation	Investigation of any symptoms

Source: NHS England Genomics Education Programme

FIH Phase 1 Lynch Syndrome study design

P101 will be divided into 2 parts: Part 1 will test safety and immunogenicity & Part 2 will assess Proof of Biology

Participants with known confirmed LS (carrier of germline pathogenic variant in MLH1, MSH2, MSH6, PMS2, or EPCAM)

Part 1: Dose escalation

N = up to 30

Cohort 4
DL4

↑

Cohort 3
DL3

↑

Cohort 2
DL2

↑

Cohort 1
Starting dose

↓

Cohort 1
Starting dose can be de-escalated if needed

DLT period of 21 days

Select 1-2 dose levels (RDE) for Part 2 based on **safety** and **immunogenicity**

Participants with known confirmed LS and adenomatous colorectal polyps ≥2 & <10mm on colonoscopy (polyps to be left in situ)

Part 2: Dose expansion

N = 80-110

Cohort 1
DL1 (TBD)

Cohort 2
DL2 (TBD)

Wk 28 colonoscopy: polyps measured & resected for translational evaluation

Wk 104 colonoscopy: final colonoscopy for evaluation of new polyps, resection as needed

Endpoints

Primary

- Safety and tolerability of mRNA-4194
- RDE* from Part 1 to Part 2

Secondary

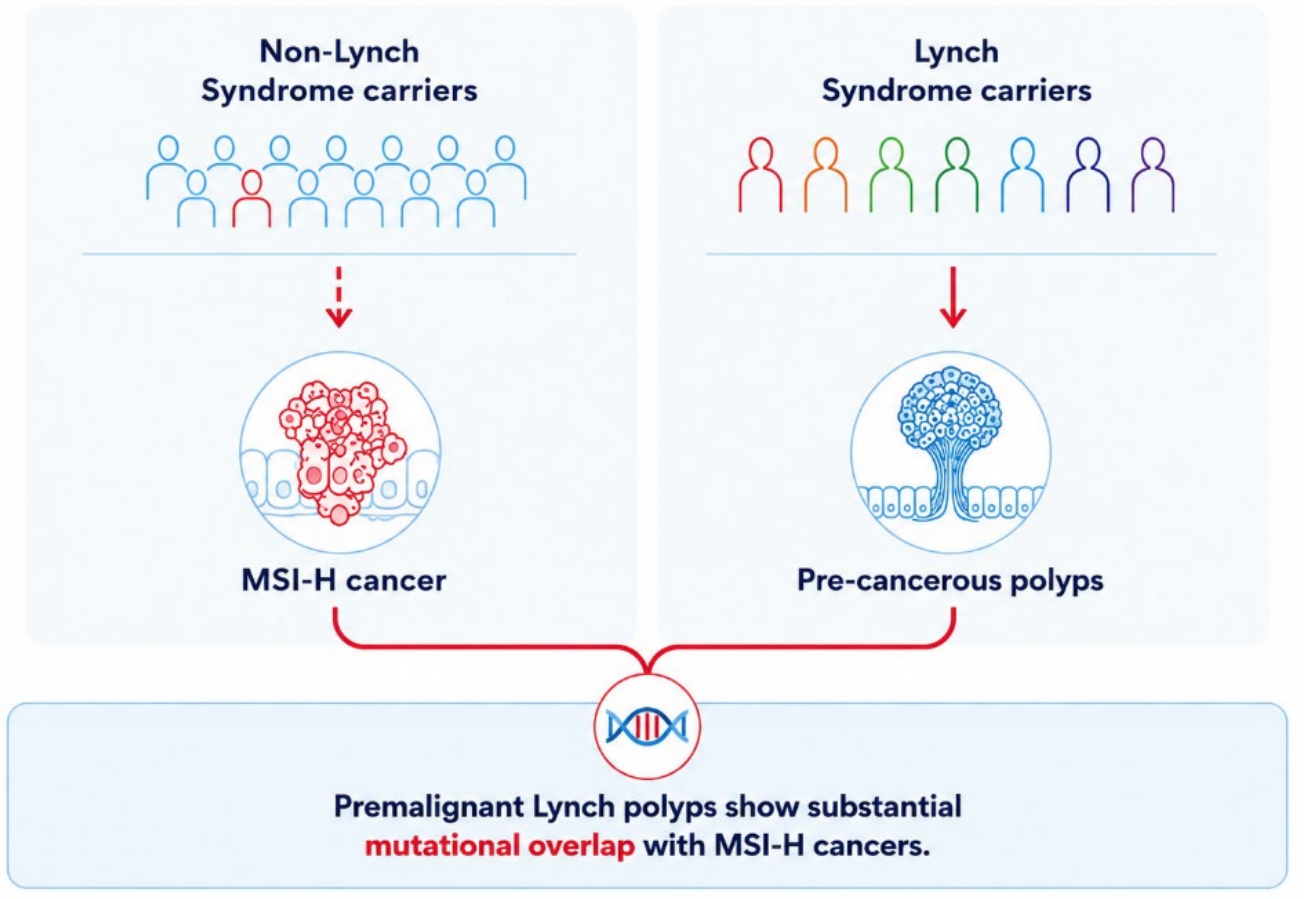
- Percent change in sum of MSI-H adenomatous polyp diameter(s) pre- and post-treatment per colonoscopy central read

Select exploratory

- Assess immunologic activity in blood
- Assess immunologic activity n colorectal tissue (incl. polyps, normal tissue)

RDE = recommended dose for expansion
DLT = dose limiting toxicity

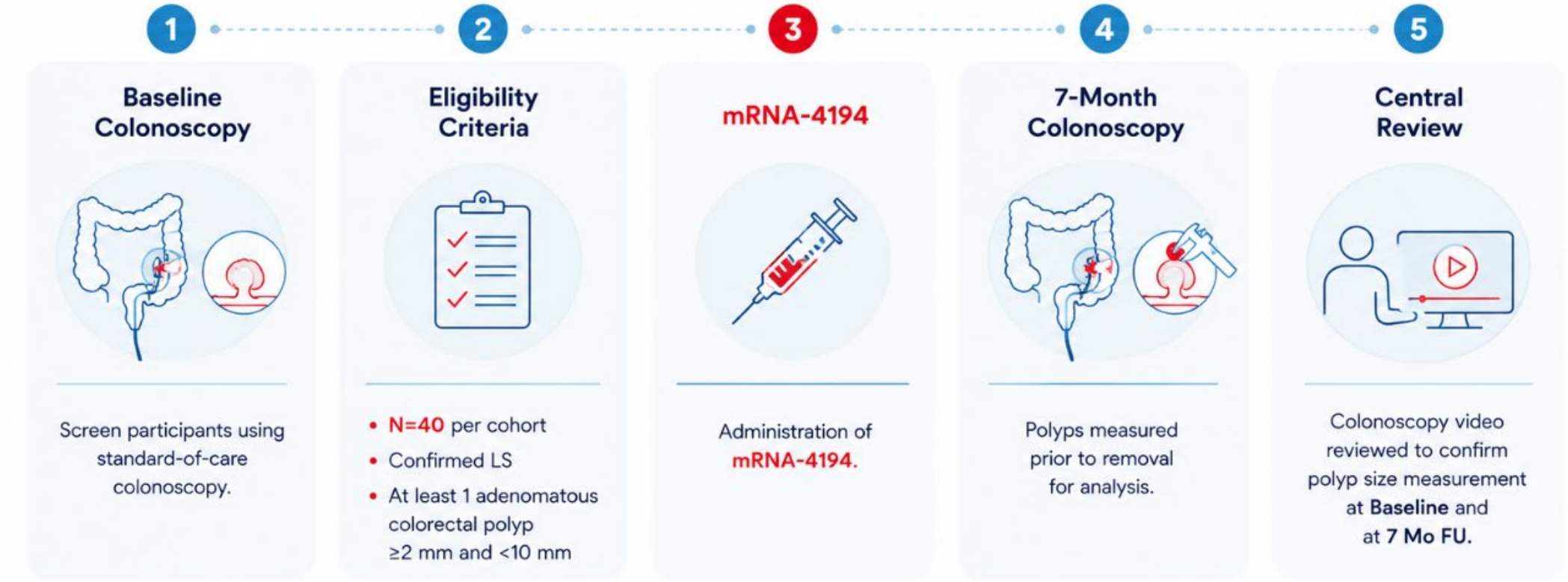
mRNA-4194 targets frameshift antigens in Lynch Syndrome



- **mRNA-4194 antigens are shared across colorectal, endometrial, and gastric MSI-H tumors**, supporting relevance across key Lynch-associated cancer types.
- **The vaccine incorporates 194 frameshift antigens** to create numerous opportunities for immune recognition within and across patients.
- **The selected antigens are designed to elicit immune responses** against targets observed in both premalignant Lynch polyps and MSI-H cancers.

mRNA-4194 is being evaluated for its potential to help prevent cancer in patients with Lynch Syndrome

Part 2 of Phase 1 mRNA-4194 study in Lynch Syndrome



Cancer Antigen Therapies

mRNA-4359: Metastatic first-line melanoma and metastatic first-line non-small cell lung cancer

Sarah Keidel, BM, BCh, MSc
Executive Director, Program Leader, Oncology Development

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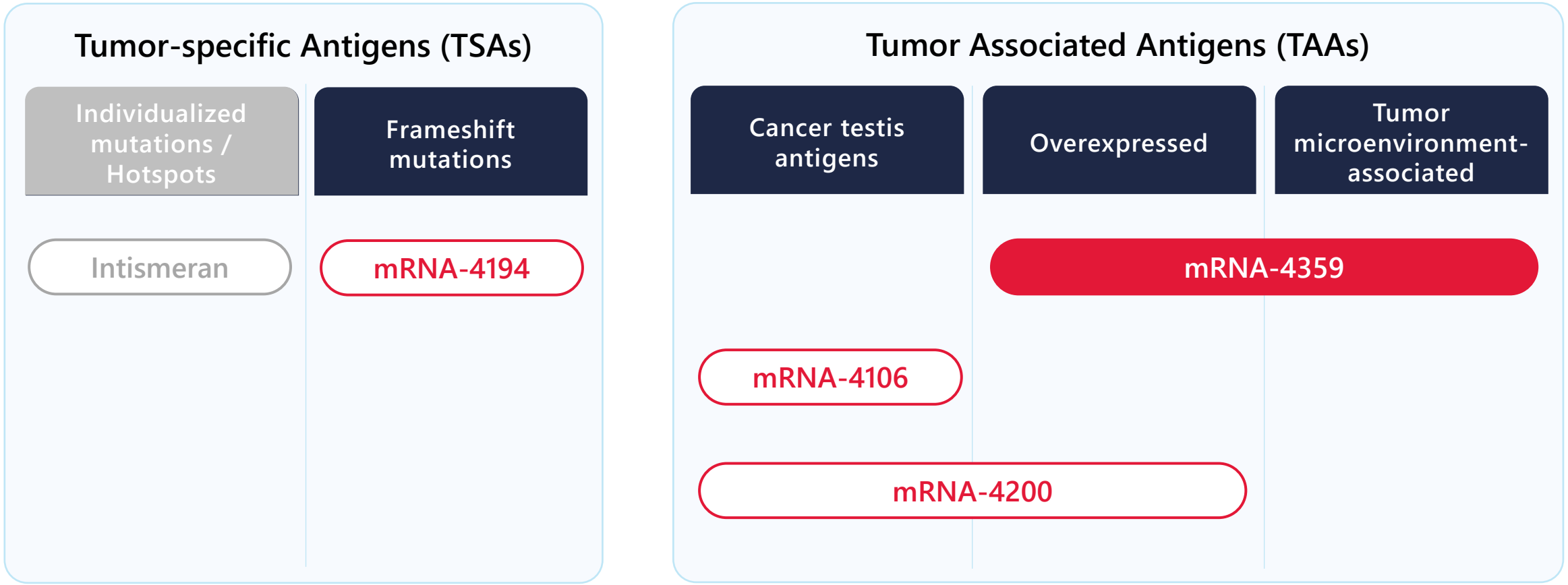
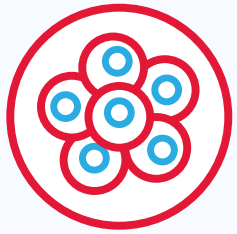
Cancer antigen therapies are designed to target distinct tumor biology



mRNA-4359 product concept

mRNA-4359 is a lipid nanoparticle-encapsulated mRNA-based immune evasion-targeted cancer antigen therapy encoding epitopes of PD-L1 and IDO1 antigens[1]

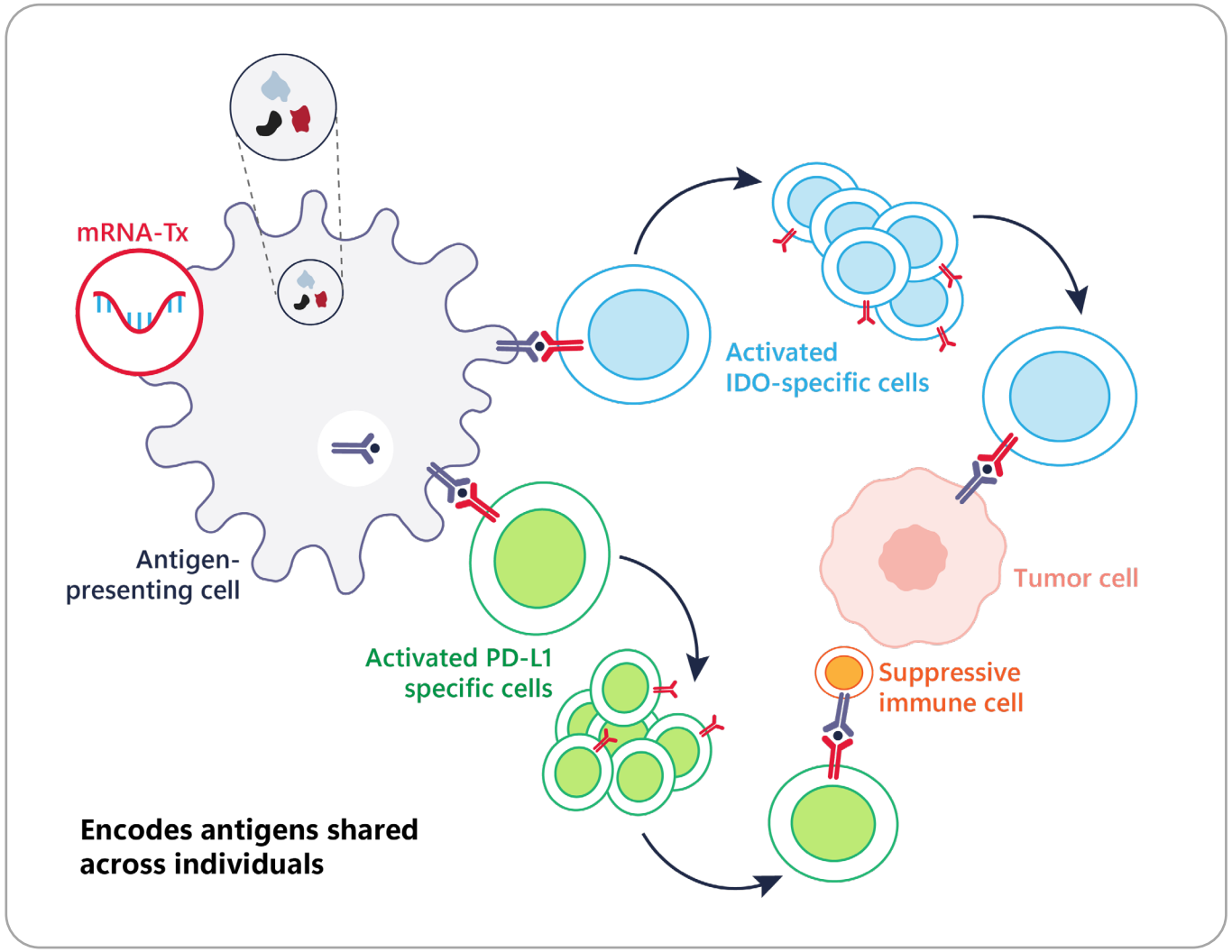


mRNA-4359 targets both immunosuppressive cells and cancer cells and is applicable to many different cancer types

1. Powderly JD, et al. *J Clin Oncol.* 2023;41:TPS2676

mRNA-4359 mechanism of action

Harnessing T-cells with off-the-shelf cancer antigen therapies



MRNA-4359 is designed to elicit T-cell responses against both tumor and immunosuppressive cells, resulting in direct tumor killing and rebalancing of the tumor microenvironment[1]

1. Powderly JD, et al. *J Clin Oncol.* 2023;41:TPS2676

mRNA-4359 Phase 1/2 study design

Phase 1
Monotherapy and combination therapy

Solid tumors

Arm 1a (Dose Escalation)
Advanced or metastatic Solid Tumors
mRNA-4359

Advanced or metastatic melanoma/non small cell lung cancer

Arm 1b (Dose Confirmation)
mRNA-4359+pembrolizumab
Advanced or Metastatic Checkpoint Inhibitor Refractory Melanoma/NSCLC
mRNA-4359+pembrolizumab

KEY OBJECTIVES

- **Safety and tolerability** of mRNA-4359 alone and in combination with pembrolizumab
- **Antitumor activity** of mRNA-4359 alone and in combination with pembrolizumab (ORR, DCR, DOR, PFS)
- **T-cell profile changes** (peripheral and tumor) after treatment of mRNA-4359 alone or in combination with pembrolizumab

Phase 2
Combination therapy

Advanced or metastatic melanoma

Arm 2a
First line melanoma
mRNA-4359+pembrolizumab
n=12

Arm 2c
First line melanoma
mRNA-4359+ipilimumab/nivolumab
n=45

Arm 2d
Second line and beyond melanoma PD-L1 TPS ≥1%
mRNA-4359+pembrolizumab
n=81

Advanced or metastatic non small cell lung cancer

Arm 2b
First line NSCLC PD-L1 TPS ≥50%
mRNA-4359 + pembrolizumab
n=50

KEY OBJECTIVES

Primary endpoints

- **Arms 2a-2c:** Safety and tolerability of mRNA-4359
- **Arm 2d:** Objective response rate based on BICR per on RECIST v1.1

Secondary endpoints

- **Arms 2a-2c:** Objective response rate, disease control rate, duration of response, progression-free survival, Percent change from baseline in T Cell profile in the tumor
- **Arm 2d:** Safety and tolerability; Duration of response, disease control rate, progression-free survival, Overall survival; Quality of Life

Abbreviations: BICR, Blinded Independent Central Review; RECIST v1.1, Response Evaluation Criteria in Solid Tumors Version 1.1

Phase 1/2 arm 1b patient disposition, and baseline characteristics

	mRNA-4359 400 µg Q3W + pembro 400 mg Q6W (n = 14)	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 15)
Follow-up,[a] median (range), wk	22.5 (3.3–84.1)	10.4 (2.0–62.7)
Age, median (range), y	67 (49–83)	65 (29–79)
Male, n (%)	10 (71)	6 (40)
ECOG PS, n (%)		
0	9 (64)	12 (80)
1	5 (36)	3 (20)
PD-L1 TPS,[b] n (%)		
≥1%	6 (43)	4 (27)
<1%	6 (43)	7 (47)
Missing	2 (14)	4 (27)
CPI-R/R disease, n (%)	14 (100)	15 (100)
No. of prior therapy, median (range)	3 (1–8)	3 (1–7)

[a.]Defined as treatment initiation to earliest non-missing date of last known alive, death, or data cutoff. [B.] PD-L1 testing was assessed centrally using PD-L1 IHC 22C3 pharmDx (Agilent, Santa Clara, CA). Data cutoff: February 28, 2025.

mRNA-4359 + pembrolizumab demonstrated a manageable safety profile

- mRNA-4359-related AEs were mostly grade 1/2 injection site reactions and self-limited systemic AEs (eg, fatigue, pyrexia, chills)
- Pembrolizumab AEs were consistent with its known safety profile
  - Pembrolizumab-related AEs occurred in 66% of patients (grade 3, 10%)
  - Pembrolizumab-related AEs with >10% incidence were fatigue (28%), diarrhea, (10%), pruritus (10%), and vomiting (10%)
- 13.8% of patients experienced immune-related AEs (eg, colitis, pancreatitis, gastritis, nephritis, and secondary adrenocortical insufficiency)
- No DLTs occurred for either dose level
- No grade 4 or 5 treatment-related AEs occurred

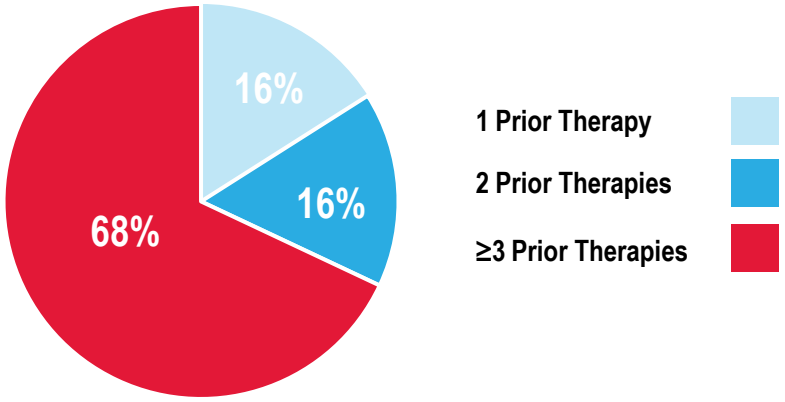
	mRNA-4359 400 µg Q3W + pembro 400 mg Q6W (n = 14)	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 15)
Duration of mRNA-4359 therapy, median (range), wk	12.5 (0.1–81.1)	6.1 (0.1–29.6)
Duration of pembro therapy, median (range), wk	10.1 (0.1–80.6)	5.9 (0.1–60.4)
mRNA-4359–related AEs, n (%)	14 (100)	12 (80)
Grade 3[a]	1 (7)[b]	1 (7)[c]
mRNA-4359–related AEs with incidence ≥20% in either cohort, n (%)		
Injection site pain	10 (71)	8 (53)
Fatigue	7 (50)	7 (47)
Pyrexia	7 (50)	4 (27)
Injection site erythema	4 (29)	1 (7)
Chills	3 (21)	2 (13)
Influenza-like illness	3 (21)	5 (33)
Vomiting	2 (14)	5 (33)
Decreased appetite	2 (14)	3 (20)
Nausea	2 (14)	3 (20)

[a]There were no grade 4 or 5 treatment-related AEs. [b]1 patient experienced grade 3 pulmonary embolism. [c]1 patient experienced grade 3 fatigue and increased blood lactic acid. Data cutoff: February 28, 2025.

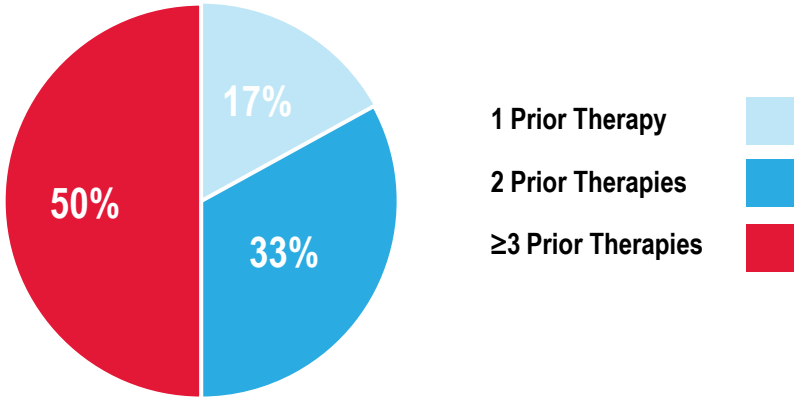
mRNA-4359 + pembro showed antitumor activity in patients with checkpoint inhibitor refractory/resistant melanoma

Evaluable patients	mRNA-4359 400 µg Q3W + pembro 400 mg Q6W (n = 13)	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 12)	All patients (N = 25)
ORR, % (95% CI)[a]	38 (14–68)	8 (0–39)	24 (9–45)
Best overall response, n (%)			
CR	0	1 (8)	1 (4)
PR	5 (38)	0	5 (20)
SD	5 (38)	4 (33)	9 (36)
PD	3 (23)	7 (58)	10 (40)
DCR, % (95% CI)[a]	77 (46–95)	42 (15–72)	60 (39–79)
DOR, median (95% CI),[b,c] wk	NR (NR–NR)	NR (NR–NR)	NR (NR–NR)

Number of Prior Therapies Among 25 Evaluable Patients



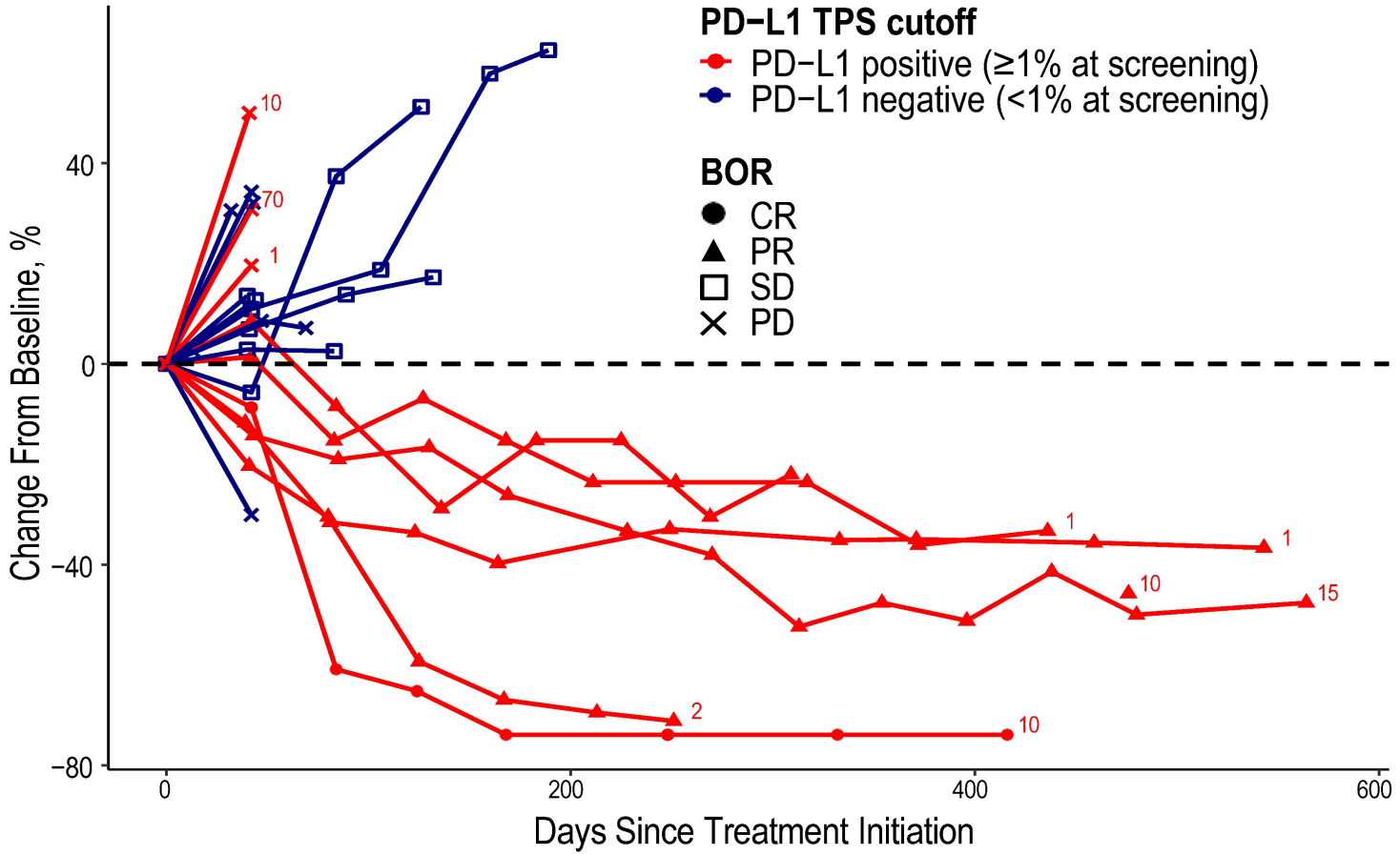
Number of Prior Therapies Among 6 Responders



CR complete response, NR, not reached. PR, partial response. [a]Based on the Clopper-Pearson exact test. [b]Based on Brookmeyer and Crowley methodology, using log-log transformation for calculating CIs. [c]The median follow-up duration of the 6 responders was 71 (range 38–84) wk by the data cutoff date. Data cutoff: February 28, 2025.

Responses were enriched in PD-L1–positive tumors (ORR, 67%), with median duration of response not yet reached, indicating encouraging durability

Tumor Responses Over Time[a]



BOR, best overall response. [a]Baseline PD-L1 TPS scores in patients with PD-L1 positive tumors are displayed at the end of the line.
Data cutoff: February 28, 2025.

Overall response rate:

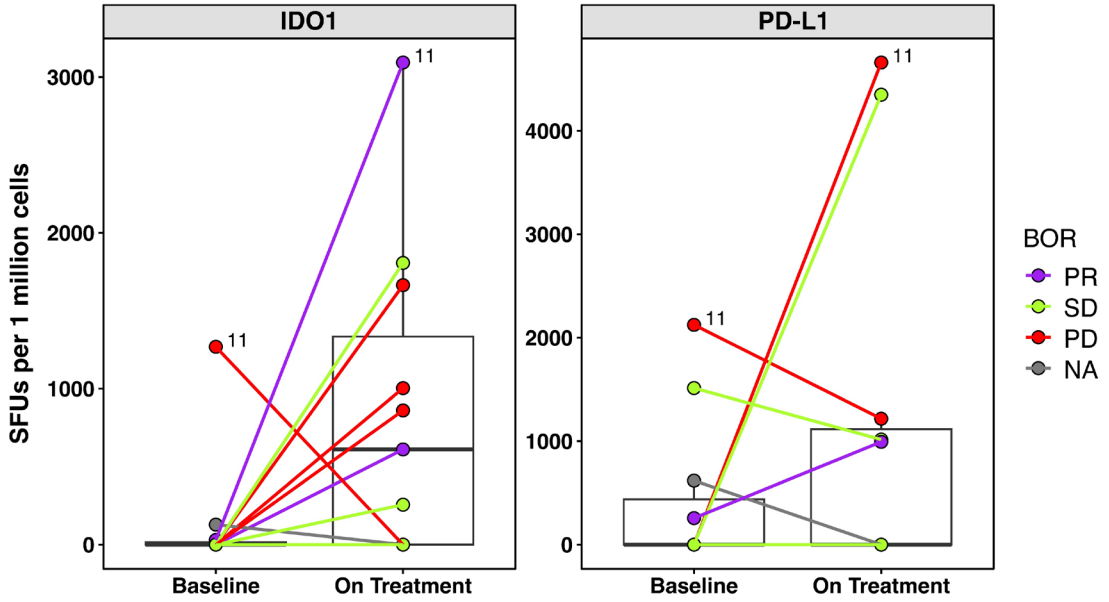
24%

Responses to mRNA-4359 occurred across heavily pretreated patients

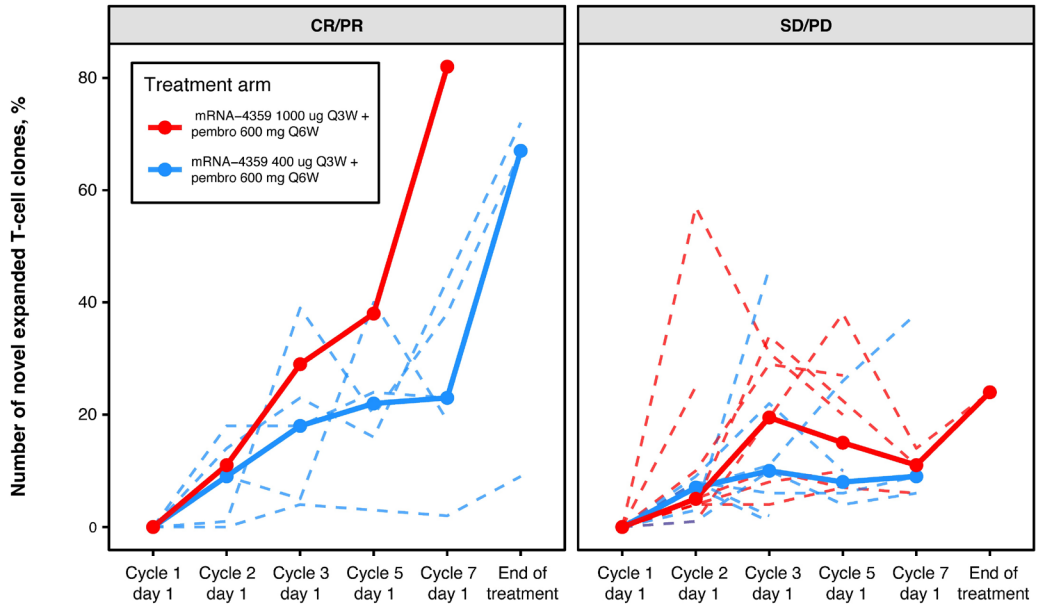
Responders	Prior treatments							Most recent treatment prior to trial enrollment	mRNA-4359 trial enrollment	Progression-free survival duration (as of 05/26/26)
1								1L pembro x 4m **BOR iCPD**		Ongoing in study without progression at 23.7m
2						Neoadj ipi/nivo x 3m **BOR recurrence**	Neoadj CBL-B IO trial x 4m **BOR PD**	1L PRAME-TCR-T trial x 7m **BOR PD**		Progression at 15.6 m
3				Neoadj pembro x 8m **BOR recurrence**	Neoadj nivo/rela trial x 3m **BOR PD**	1L dacarbazine + carboplatin x 2m **BOR PD**	2L IOVANCE TIL trial x 11m **BOR PR**	3L STING agonist + ezabenlimab trial x 2m **BOR PD**		Completed study at 24m without progression
4	1L pembro x 3m **BOR SD**	2L TVEC x 8.5m **BOR SD**	3L Vidutolimod x 1m **BOR PD**	4L pembro x 2.5m **BOR PD**	5L ipi/nivo x 2m **BOR PD**	6L NT-17 + Atezo x 1m **BOR PD**	7L IOVANCE TIL trial x 1m **BOR PD**	8L nivo/rela x 3m **BOR PD**		Completed study at 24m without progression
5						Adj nivo x 10m **BOR recurrence**	1L Imlygic x 5m **BOR PD**	2L Nivo + lerapolturev x 3m **BOR PD**		Progression at 18.4m
6							Adj nivo x 3m **BOR recurrence**	1L ipi/nivo x 1m **BOR PD**		Progression at 16.4m

mRNA-4359 demonstrated biological activity through specific T-Cell responses and novel clonal expansion in the periphery

mRNA-4359 Elicited PD-L1- and IDO1-Specific T cell Responses in the Periphery[a]



Increase in Number of Novel Expanded TCR Clones in the Periphery with mRNA-4359 Treatment



SFU, spot-forming unit; TCR, T cell receptor.
[a]On treatment responses were selected from the 'best' ELISpot response at different time points for each patient.

mRNA-4359 Phase 1/2 study design

Phase 1
Monotherapy and combination therapy

Solid tumors

Arm 1a (Dose Escalation)
Advanced or metastatic Solid Tumors
mRNA-4359

Advanced or metastatic melanoma/non small cell lung cancer

Arm 1b (Dose Confirmation)
mRNA-4359+pembrolizumab
Advanced or Metastatic Checkpoint Inhibitor Refractory Melanoma/NSCLC
mRNA-4359+pembrolizumab

KEY OBJECTIVES

- **Safety and tolerability** of mRNA-4359 alone and in combination with pembrolizumab
- **Antitumor activity** of mRNA-4359 alone and in combination with pembrolizumab (ORR, DCR, DOR, PFS)
- **T-cell profile changes** (peripheral and tumor) after treatment of mRNA-4359 alone or in combination with pembrolizumab

Phase 2
Combination therapy

Advanced or metastatic melanoma

Arm 2a
First line melanoma
mRNA-4359+pembrolizumab
n=12

Arm 2c
First line melanoma
mRNA-4359+ipilimumab/nivolumab
n=45

Arm 2d
Second line and beyond melanoma PD-L1 TPS ≥1%
mRNA-4359+pembrolizumab
n=81

Advanced or metastatic non small cell lung cancer

Arm 2b
First line NSCLC PD-L1 TPS ≥50%
mRNA-4359 + pembrolizumab
n=50

KEY OBJECTIVES

Primary endpoints

- **Arms 2a-2c:** Safety and tolerability of mRNA-4359
- **Arm 2d:** Objective response rate based on BICR per on RECIST v1.1

Secondary endpoints

- **Arms 2a-2c:** Objective response rate, disease control rate, duration of response, progression-free survival, Percent change from baseline in T Cell profile in the tumor
- **Arm 2d:** Safety and tolerability; Duration of response, disease control rate, progression-free survival, Overall survival; Quality of Life

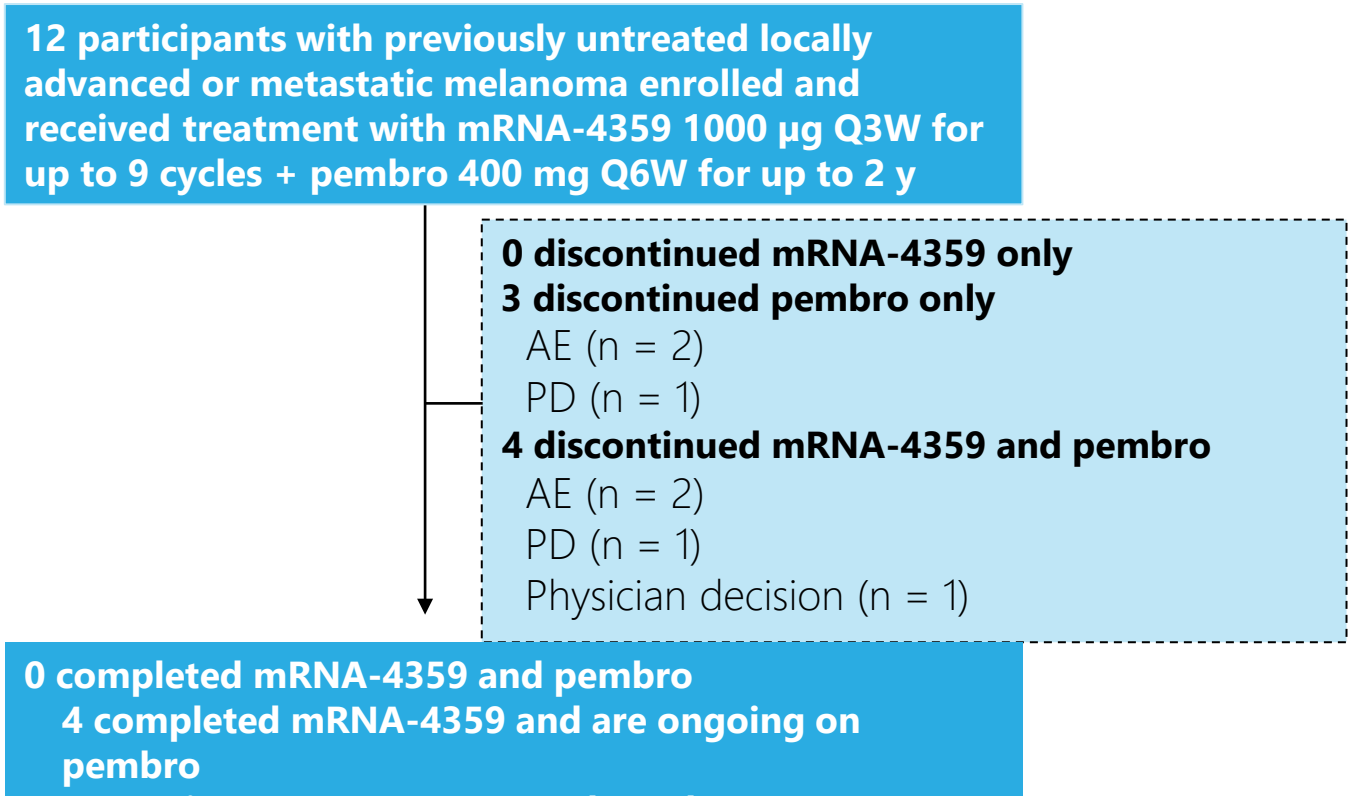
Abbreviations: BICR, Blinded Independent Central Review; RECIST v1.1, Response Evaluation Criteria in Solid Tumors Version 1.1

mRNA-4359 Phase 1/2 arm 2a treatment disposition

Median follow-up at data cutoff:
54.2 (range, 22.3–76.0) wk

Median mRNA-4359 exposure at data cutoff:
9 (range, 3–9) doses

Median pembro exposure at data cutoff:
6.5 (range, 2–12) doses



AE, adverse event; pembro, pembrolizumab; PD, progressive disease.
Data cutoff: December 1, 2025.

mRNA-4359 Phase 1/2 arm 2a baseline characteristics

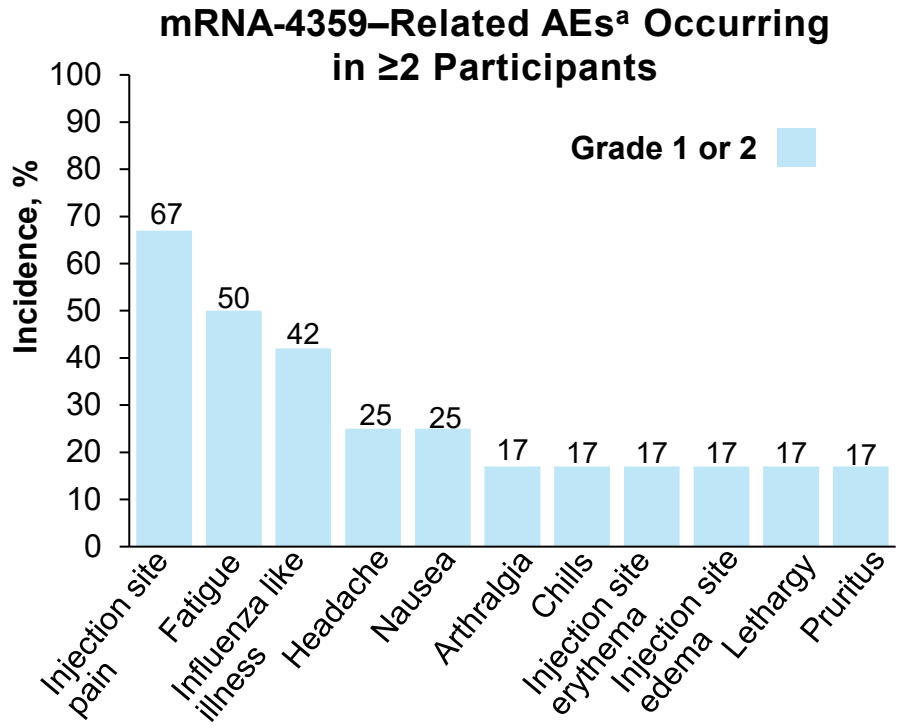
	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 12)[a]
Age, median (range), y	58 (38–80)
<65 y, n (%)	8 (67)
≥65 y, n (%)	4 (33)
Sex, n (%)	
Male	6 (50)
Female	6 (50)
ECOG PS, n (%)	
0	11 (92)
1	1 (8)
Disease stage, n (%)	
III	1 (8)
IV	11 (92)

	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 12)[a]
PD-L1 TPS,[b] n (%)	
≥1%	8 (67)
<1%	3 (25)
Missing	1 (8)
***BRAF* status, n (%)**	
Mutated	6 (50)
Wild-type	6 (50)
Prior (neo)adjuvant therapy,[c] n (%)	4 (33)
Immunotherapy[d]	3 (25)
BRAF/MEK inhibition[e]	2 (17)

TPS, tumor proportion score. [a]1 had mucosal melanoma, 1 acral lentiginous melanoma, and 10 had melanoma of cutaneous (nodular or superficial) or unknown primary origin. [b]PD-L1 testing was assessed centrally using PD-L1 IHC 22C3 pharmDx (Agilent, Santa Clara, CA). [c]3 participants received prior adjuvant therapy and 1 received prior neoadjuvant and adjuvant therapy. [d]All participants received prior nivolumab. [e]Dabrafenib and trametinib or encorafenib and binimetinib. Data cutoff: December 1, 2025.

mRNA-4359–related AEs were all low grade (Grade 1 or 2)

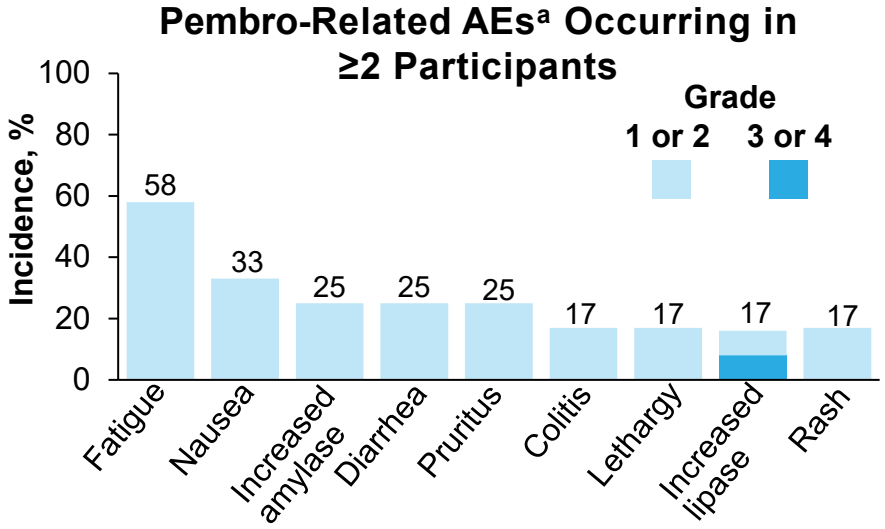
	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 12)
mRNA-4359–related AEs,[a] n (%)	11 (92)
DLTs	0
Grade ≥3	0
Serious	1 (8)
Led to mRNA-4359 delay	2 (17)
Led to mRNA-4359 discontinuation	0
Led to pembro discontinuation	0
Led to death	0



AE, adverse event. [a]Defined as AEs not present before study or worsened on study and deemed related to study drug by investigator..
Data cutoff: December 1, 2025.

Pembrolizumab-related AEs were predominantly low grade

	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W (n = 12)
Pembro-related AEs,[a] n (%)	11 (92)
DLTs	0
Grade 3 or 4	4 (33)
Serious	6 (50)
Led to pembro delay	7 (58)
Led to pembro interruption	1 (8)
Led to pembro discontinuation	3 (25)[b]
Led to mRNA-4359 discontinuation	1 (8)[c]
Led to death	0



- **1 pembro-related grade 4 event occurred (increased lipase)**
- **Pembro-related grade 3 events included immune-mediated pancreatitis in 1 participant, dyspnea in 1 participant, and hepatoxicity, immune-mediated hepatitis, and increased liver function test in 1 participant**

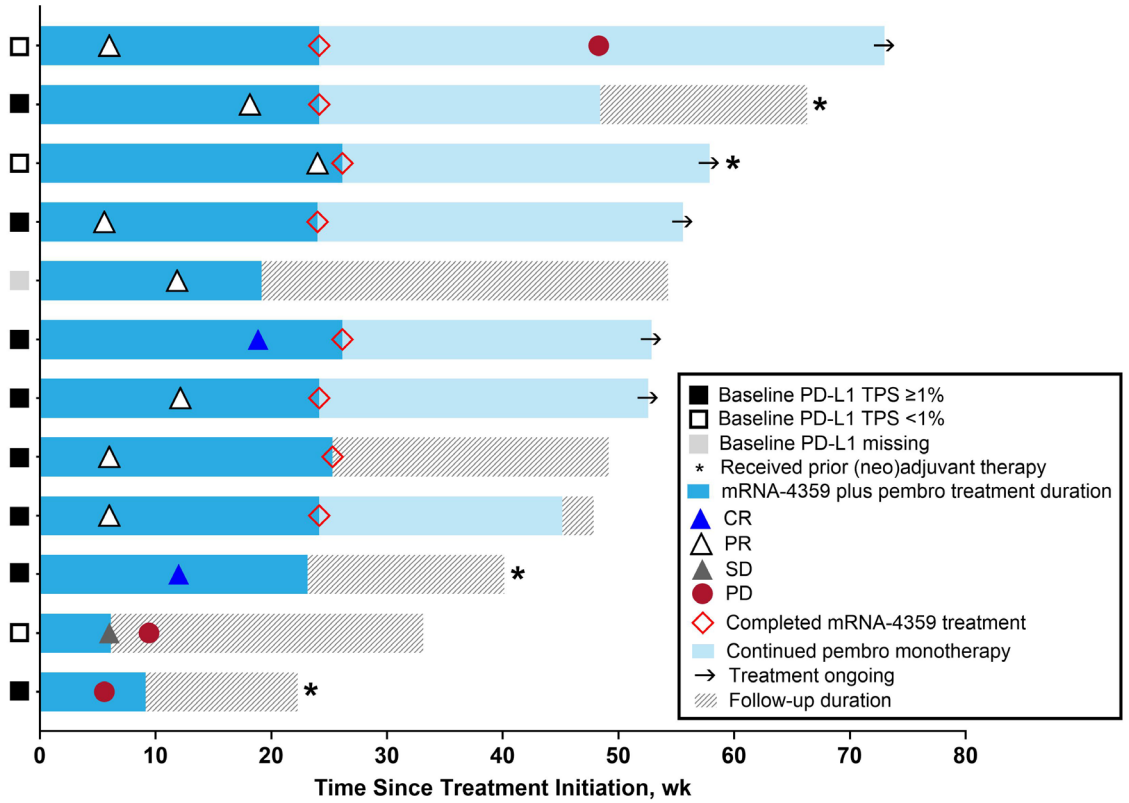
[a]Defined as AEs not present before study or worsened on study and deemed related to study drug by investigator.. [b]Due to grade 1 peripheral sensorimotor neuropathy, grade 2 colitis, and grade 2 immune-mediated pancreatitis (n = 1 each). [c]Due to grade 2 colitis. Data cutoff: December 1, 2025.

Most participants (83%) had tumor response[a] to mRNA-4359 plus pembrolizumab

	mRNA-4359 1000 µg Q3W + pembro 400 mg Q6W		
	PD-L1 TPS ≥1% (n = 8)	PD-L1 TPS <1% (n = 3)	All participants (N = 12)
ORR (95% CI),[b] %	88 (47–100)	67 (9–99)	83 (52–98)
Best overall response,[c] n (%)			
CR	2 (25)	0	2 (17)
PR	5 (63)	2 (67)	8 (67)
SD	0	1 (33)	1 (8)
PD	1 (13)	0	1 (8)
DCR (95% CI),[b] %	88 (47–100)	100 (29–100)	92 (62–100)
DOR, median (95% CI),[d] wk	NR (NR–NR)	NR (NR–NR)	NR (NR–NR)
TTR, median (range),[e] wk	6.0 (5.6–18.1)	15 (6.0–24.0)	6.0 (5.6–24.0)

CR, complete response; NR, not reached; PR, partial response; SD, stable disease; TTR, time to response. [a]Assessed per RECIST version 1.1 by investigator assessment.
[b]Unconfirmed ORR and unconfirmed DCR were based on Clopper-Pearson method. [c]Best overall response was unconfirmed. [d]Estimated using Kaplan-Meier method.
[e]Calculated based on observed values.
Data cutoff: December 1, 2025.

The frequency of progression-free survival (PFS) events was low (2 events)

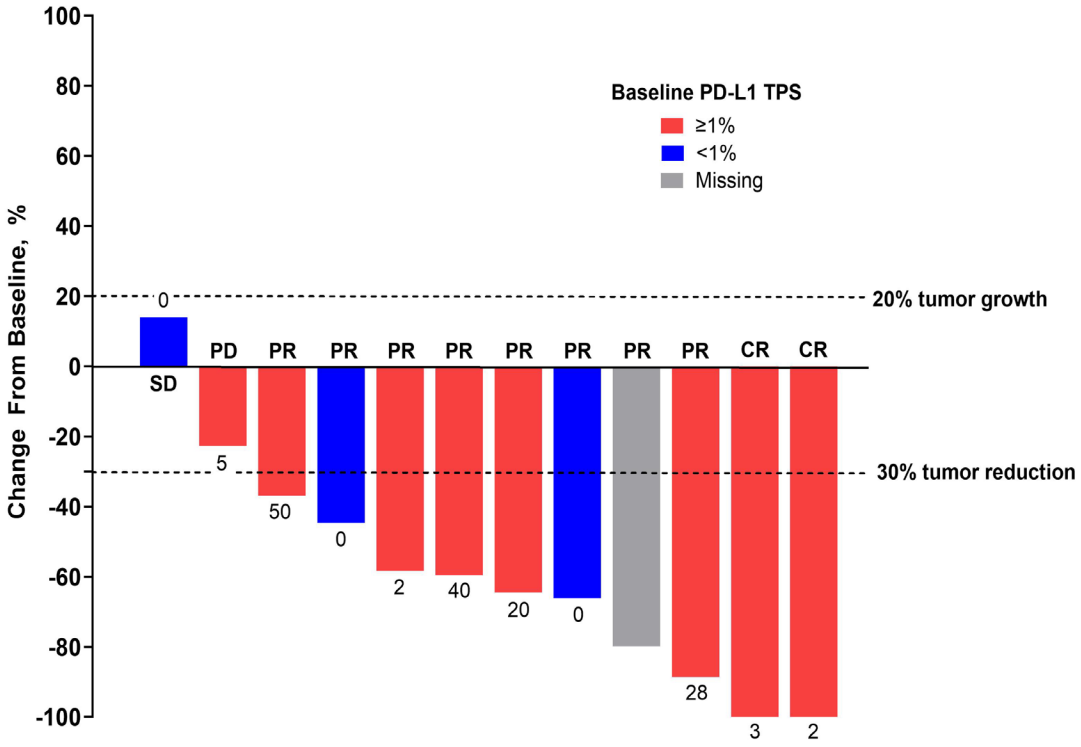


[a]Assessed per investigator assessment. [b]1 participant with PD was censored due to missing tumor assessment. [c]Estimated using Kaplan-Meier method.
Data cutoff: December 1, 2025.

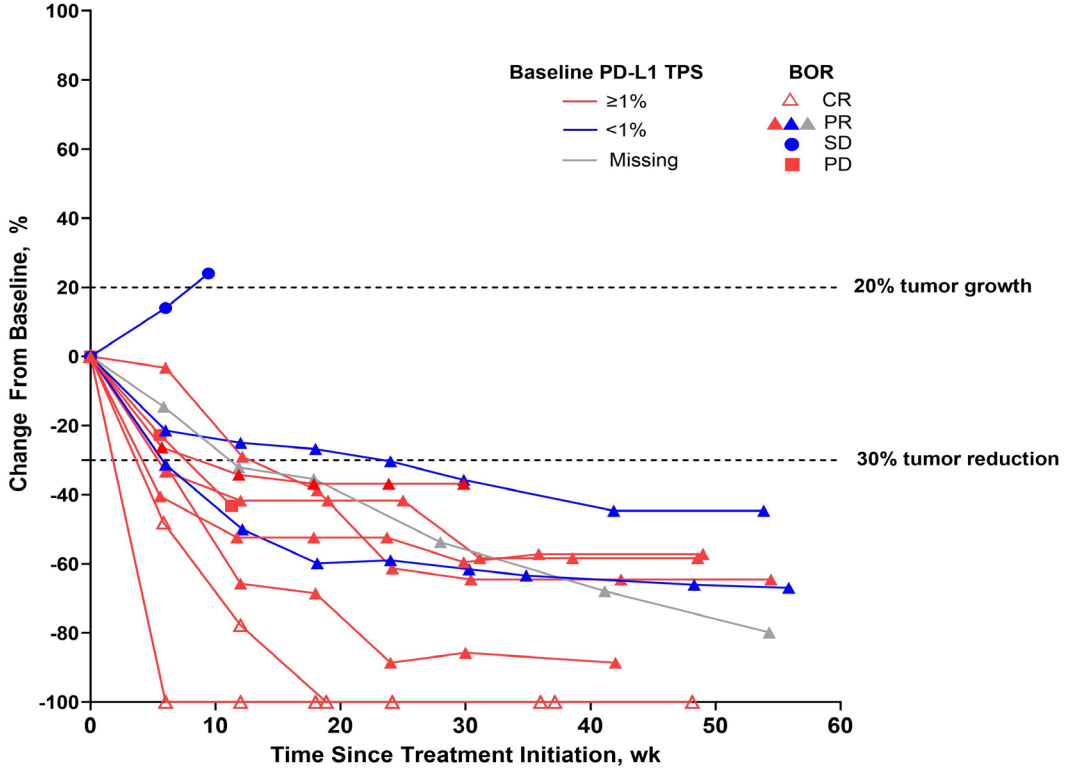
- Three of four participants with prior (neo)adjuvant therapy had an objective response
- As of data cutoff, 2 participants had a PFS event[b]
- Median PFS was NR (95% CI, 9.4 weeks–NR)[c]
- Most participants with a PR are being monitored without any PFS events to date

Most Participants (92%) had a reduction in tumor burden[a]

Maximum Percent Reduction From Baseline in Sum of Diameter of Target Lesions[b]



Percent Change from Baseline in Sum of Diameter of Target Lesions Over Time

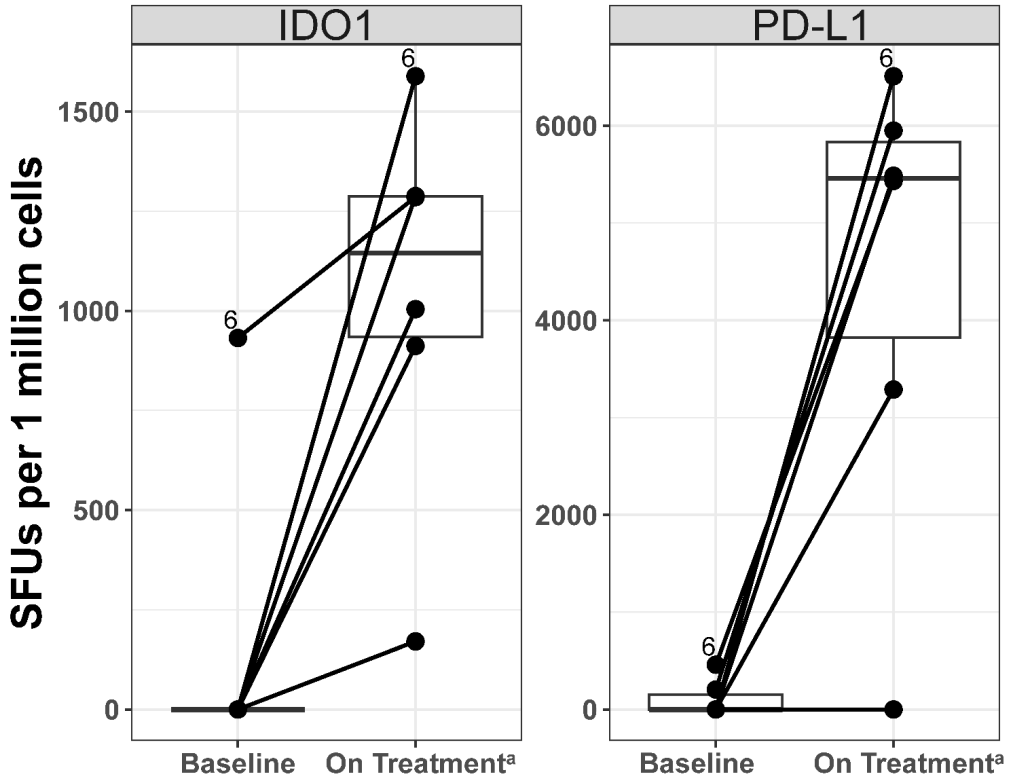


[a]Assessed per investigator assessment. [b]1 participant had a reduction in target lesions but was classified as PD due to the development of a new lesion in the liver.
Data cutoff: December 1, 2025.

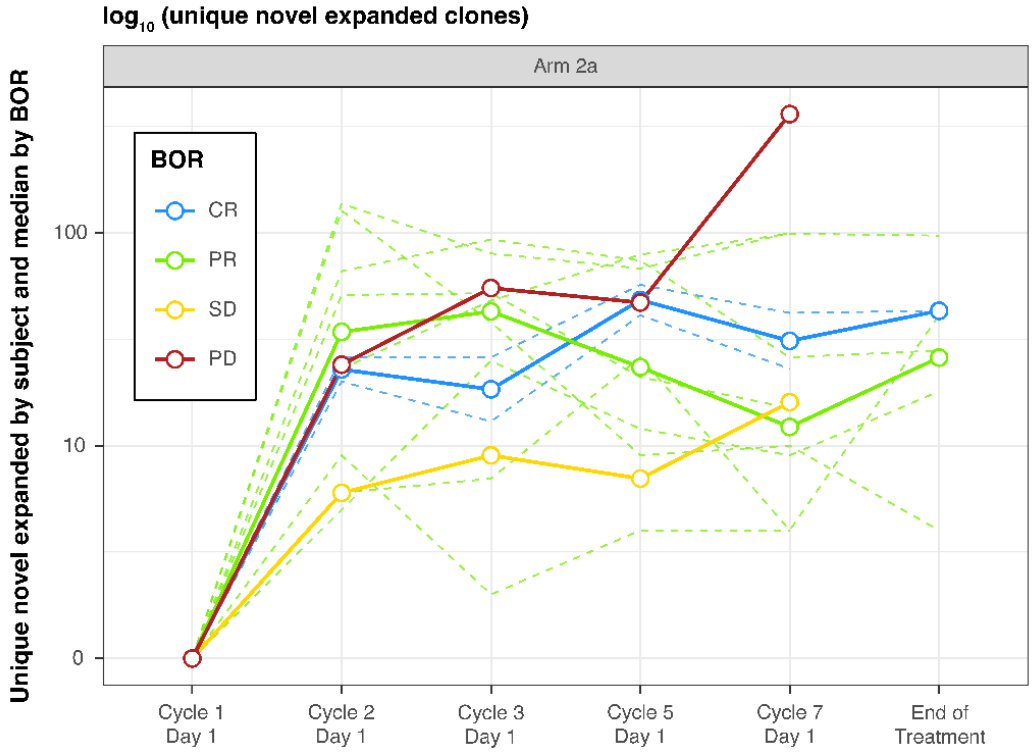
Biological activity demonstrated by antigen-specific T-Cell responses and de novo clonal expansion

ELISpot and TCR sequencing analysis in periphery

mRNA-4359 Plus Pembro Elicited PD-L1- and IDO1-Specific T-cell Responses in Periphery[a]



mRNA-4359 Plus Pembro Increased Number of Novel Expanded TCR Clones in Periphery[b,c]



SFU, spot-forming unit. [a]On-treatment antigen-specific T-cell responses were defined as the best ELISpot response across timepoints for each participant, measured after in vitro expansion of peripheral blood mononuclear cells (PBMCs). [b]Solid lines represent median data for each response group, and dotted lines represent each individual participant. [c]1 participant demonstrated rapid target lesion reduction but was classified as PD due to the development of a new lesion in the liver.

Summary and next steps

Safety	• mRNA-4359 + pembrolizumab showed a manageable safety profile in both checkpoint inhibitor refractory/resistant melanoma and previously untreated locally advanced or metastatic melanoma
Efficacy	• Durable responses seen in CPI-refractory/resistant melanoma, especially in PD-L1 TPS ≥1% patients, where ORR reached 67% and median DOR not reached. • Encouraging antitumor activity and durable responses observed in most participants (83%), irrespective of baseline tumor PD-L1 expression in the first-line setting
Translational data	• mRNA-4359 + pembrolizumab induced antigen-specific T-cell responses and new TCR clones in CPI R/R melanoma patients • Consistent with its mechanistic rationale, mRNA-4359 + pembrolizumab induced antigen-specific T-cell responses and expanded novel TCR clones in all evaluable patients with previously untreated advanced melanoma
Next steps	• Phase 2 data readout

Coffee break

10 minutes

mRED portfolio

Horizon 2 — Emerging modalities

Cancer antigen therapies

mRNA-4359

mRNA-4106

mRNA-4200

mRNA-4194

T-cell engagers
(surface antigen)

mRNA-2808

mRNA-2151

Cell therapy enhancers

mRNA-4203

Multiple sclerosis therapeutic

mRNA-1195

Horizon 3 — Future modalities

In vivo CAR-T

mRNA-6007

In vivo CAR-M

T-cell engagers
(intracellular targets)

Tolerizing therapy

T-cell engagers

mRNA-2808: Multiple myeloma

mRNA-2151: Ovarian cancer

Lin Guey, PhD
Chief Scientific Officer, Therapeutics Research

moderna

T-cell engagers demonstrate how mRED builds modality-based portfolios

Platform advantage
Advance modalities where our platform technology offers distinct competitive advantages and program differentiation

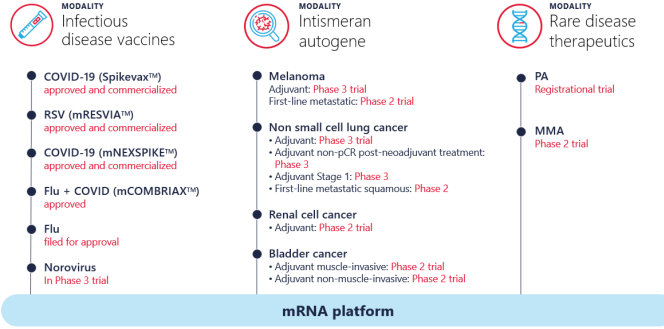
Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction

Portfolio balance
Build a diversified portfolio by balancing breadth across distinct areas of opportunity with disciplined expansion within each modality

Platform competitive advantage

- Multiplexing
- Intracellular proteins
- Transmembrane proteins
- Complex proteins
- T cell response

Proof-of-concept data from sentinel programs de-risk modalities and can accelerate development plans



T-cell engagers demonstrate how mRED builds modality-based portfolios

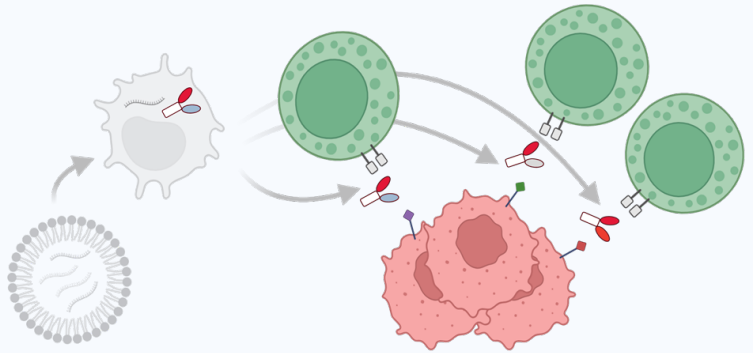
Platform advantage
Advance modalities where our platform technology offers distinct competitive advantages and program differentiation

Platform competitive advantage

- Multiplexing
- Intracellular proteins
- Transmembrane proteins
- Complex proteins
- T cell response

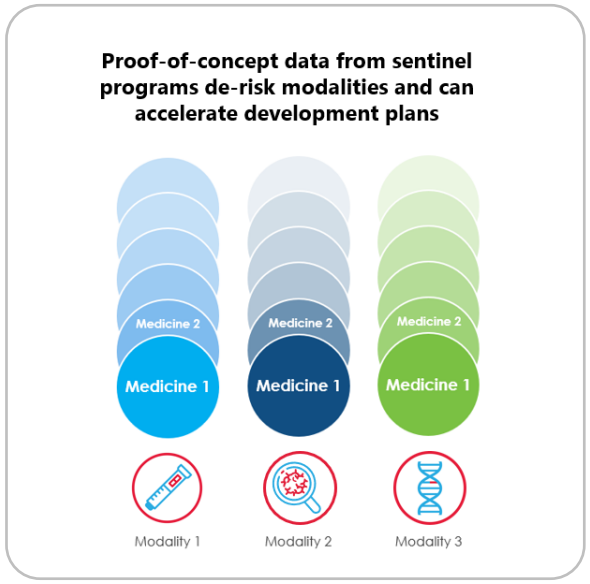
Surface Antigen T-Cell Engagers

- Multiplexes to overcome tumor heterogeneity and antigen escape, well-established resistance mechanism
- Potential to multiplex other T cell targets for co-stimulation



T-cell engagers demonstrate how mRED builds modality-based portfolios

Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction



mRNA-2151

mRNA-2808

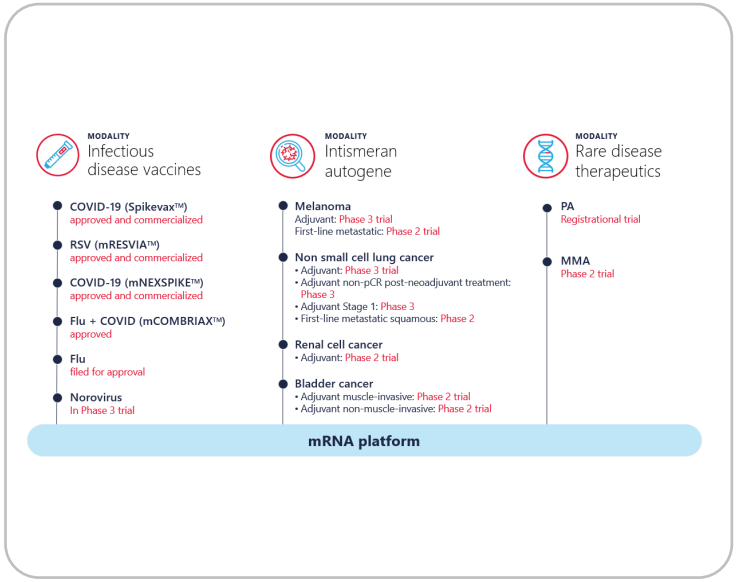
Based on encouraging early clinical signals with mRNA-2808, **investment in mRNA-2151 ungated**

Sentinel program in Phase 1/2 clinical study

Modality:
T-cell engagers

T-cell engagers demonstrate how mRED builds modality-based portfolios

Portfolio balance
Build a diversified portfolio by balancing breadth across distinct areas of opportunity with disciplined expansion within each modality



mRNA-2151

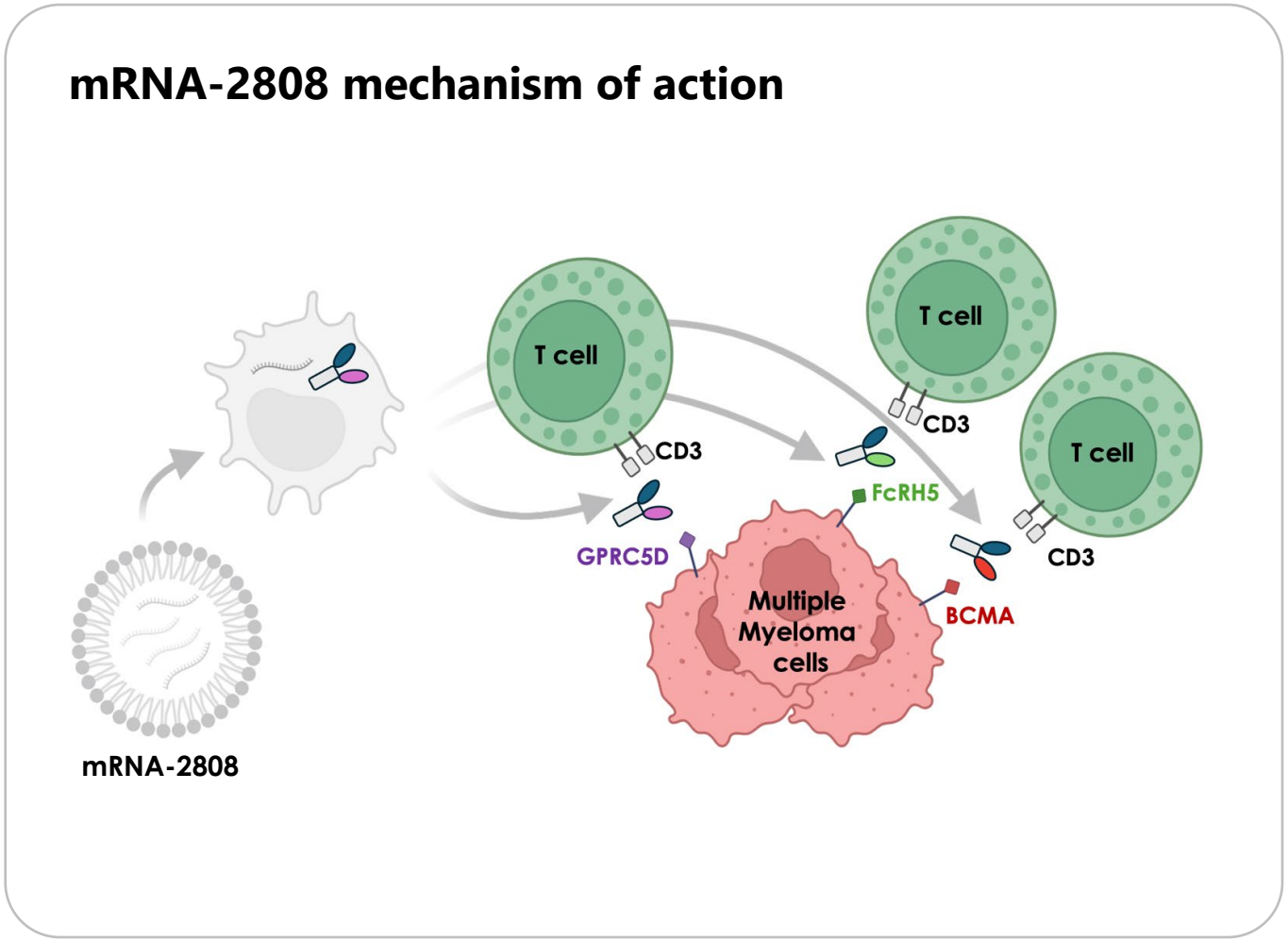
Targeting **ovarian cancer** (solid tumors)

mRNA-2808

Targeting **multiple myeloma**

Modality:
T-cell engagers

mRNA-2808 includes distinct T-cell engagers against clinically-validated multiple myeloma targets; BCMA, GPRC5D, and FcRH5



- Targets T cell CD3 and clinically validated tumor associated antigens (TAAs) that are present on the surface of the tumor
- Multiplexes to overcome antigen escape, well-established resistance mechanism

mRNA-2808 could differentiate in multiple myeloma as the only treatment targeting three disease antigens

T-cell engager landscape in multiple myeloma

First Generation

Single target

Second Generation

Two targets
Bi-specific approach

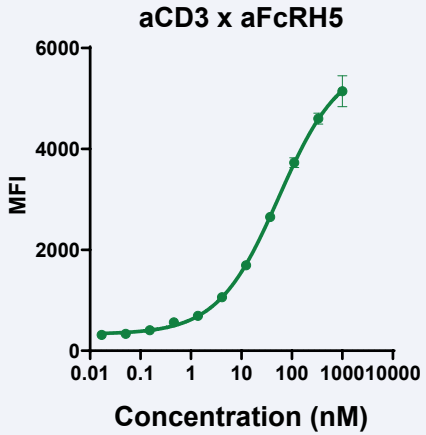
mRNA-2808

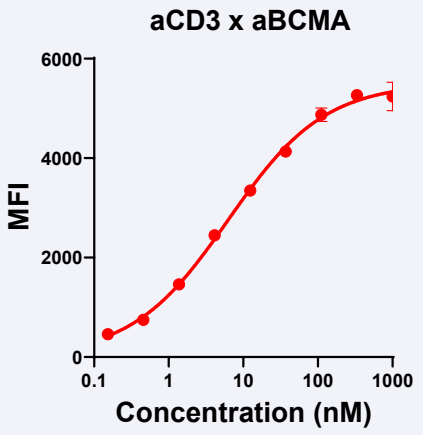
Three targets
Expanded targeting potential

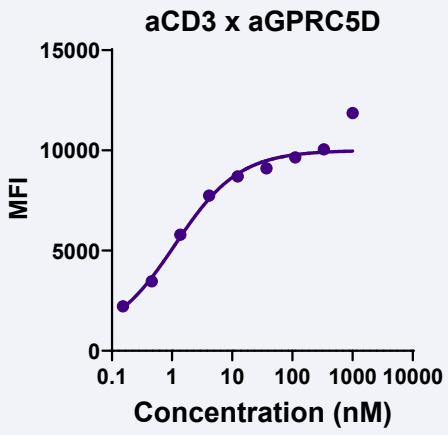
mRNA-2808 encoded TCE proteins exhibit nM binding affinities and pM cytotoxic potencies in vitro

Binding to TAA+ tumor cell lines

Molm13 cells engineered to express FcRH5 or BCMA+ GPRC5D+ NCI-H929 cells

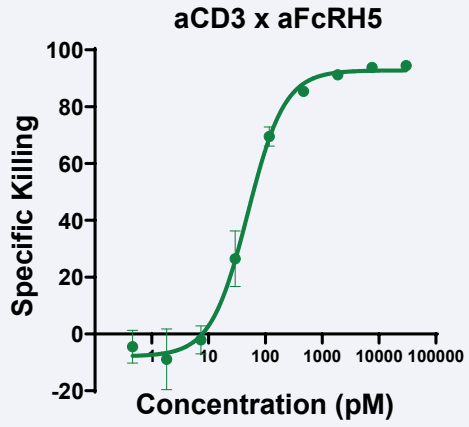


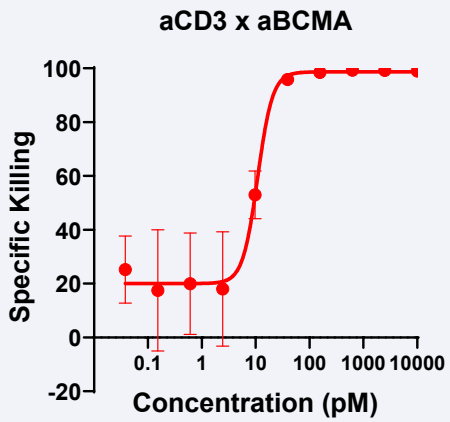


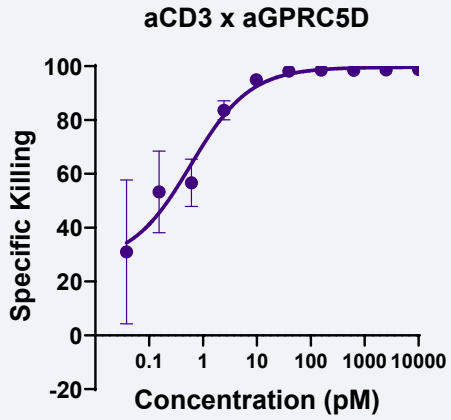


Cytotoxicity against TAA+ tumor cell lines

FcRH5+ SU-DHL-6 cells and GPRC5D+ and BCMA+ NCI-H929 cells



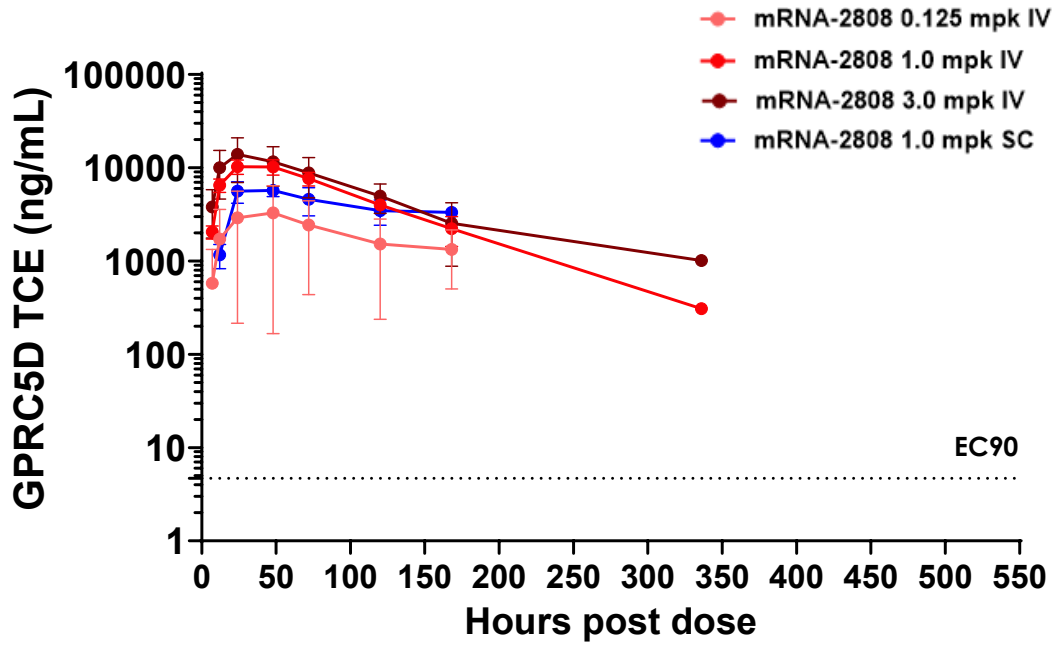




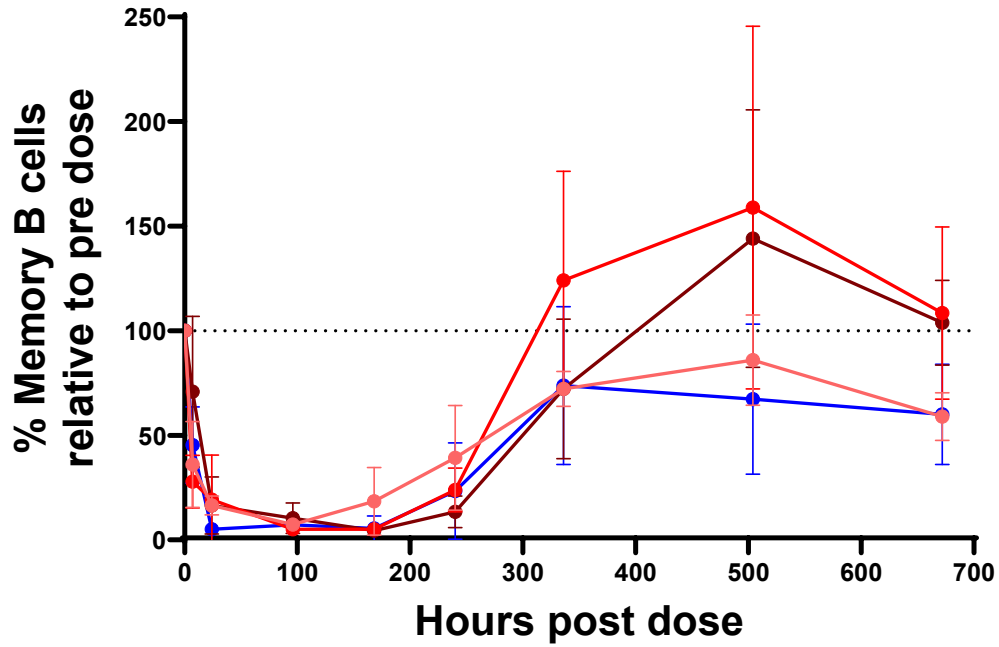
ASH 2024

Repeat dosing in NHPs show therapeutically relevant protein expression resulting in target cell depletion

TCE protein expression



Memory B cell depletion

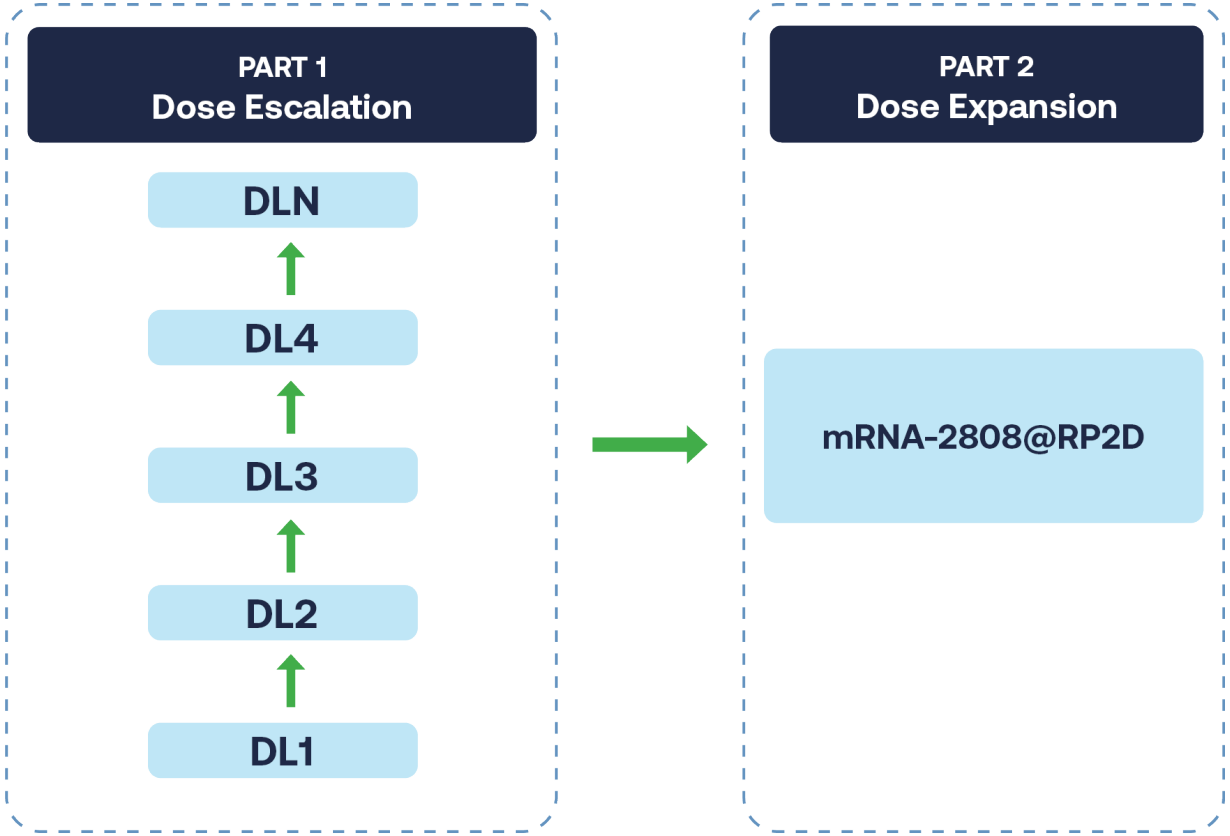


NHPs (n=4/group) received a single IV dose (0.125 mg/kg, 1 mg/kg, or 3 mg/kg) or subcutaneous (SC) injection (1 mg/kg), ASH 2024

In NHPs, mRNA-2808 achieved therapeutically relevant protein expression with potent on-target pharmacology, including TAA-positive B-cell depletion and T-cell activation and proliferation, with an acceptable safety profile

mRNA-2808 phase 1/2 study ongoing in multiple myeloma

Phase 1/2 study design and key objectives



DL: dose level

Key eligibility: Relapsed refractory multiple myeloma with prior exposure to three treatments: a proteasome inhibitor, an immunomodulatory drug (IMiD), and an anti-cluster of differentiation (CD38) monoclonal antibody.

Primary endpoints: Safety, number of participants with dose limiting toxicity, number of participants with treatment-emergent adverse events

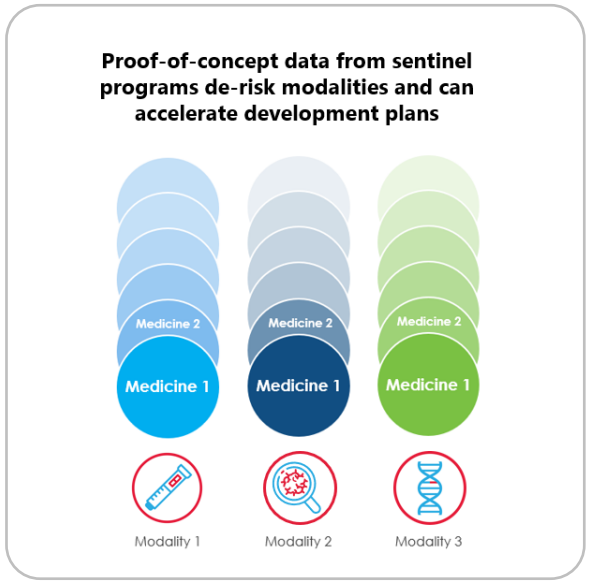
Secondary endpoints: Pharmacokinetics, pharmacodynamics, overall response rate by International Myeloma Working Group (IMWG), duration of response, progression-free survival

T-cell engagers

mRNA-2151: Ovarian cancer

T-cell engagers demonstrate how mRED builds modality-based portfolios; mRNA-2151 is the second TCE in modality

Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction



mRNA-2151

mRNA-2808

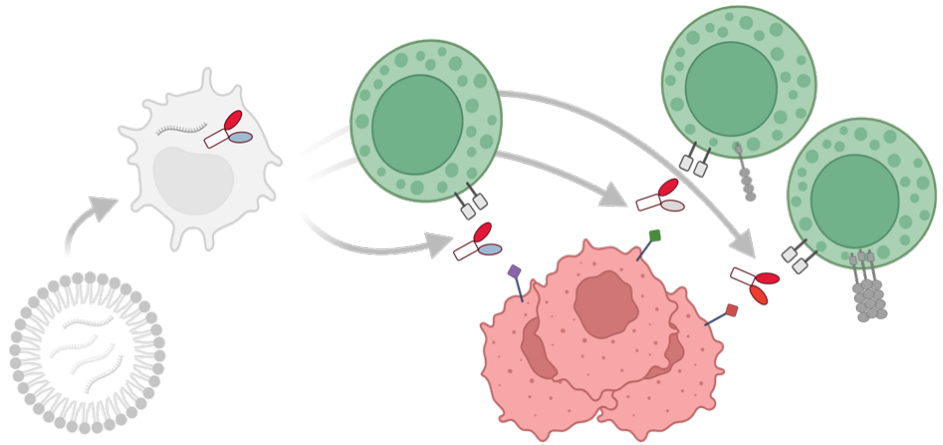
Based on encouraging early clinical signals with mRNA-2808, **investment in mRNA-2151 ungated**

Sentinel program in Phase 1/2 clinical study and has passed stage gates

Modality:
T-cell engagers

mRNA-2151 combines multiple T-cell activating signals to enhance anti-tumor activity in ovarian cancer

mRNA-2151 mechanism of action



Ovarian cancer cells

Signal 1 (TCE1):

- Increase tumor coverage and efficacy
- Broaden addressable patient population expressing at least one TAA

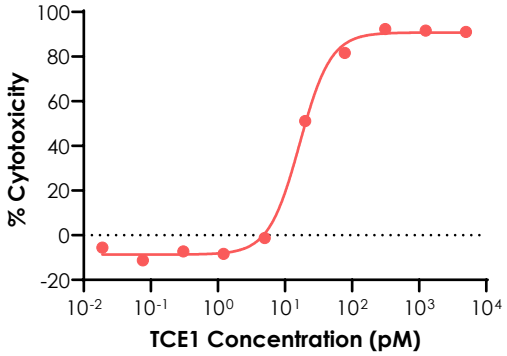
Signal 2 (TCE2):

- Co-stimulation to enhance T cell responses
- Overcome challenges in solid tumors including immunosuppressive TME, poor T cell fitness

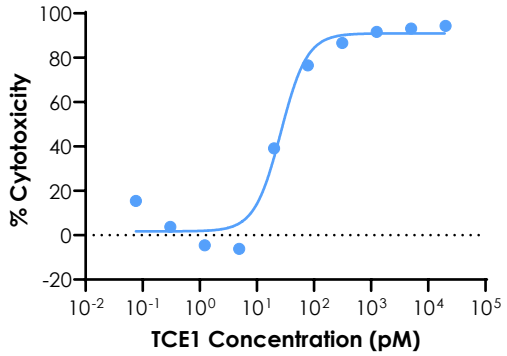
- **Leverages existing Signal 1 antibody** domains; **format derisked** by multiple myeloma TCE mRNA-2808 Phase I
- **Ability to multiplex** other T-cell targets for co-stimulation
- **Plug & play flexibility** for future product concepts

Multiplexed TCE1s exhibit potent anti-tumor activity that is enhanced by TCE2

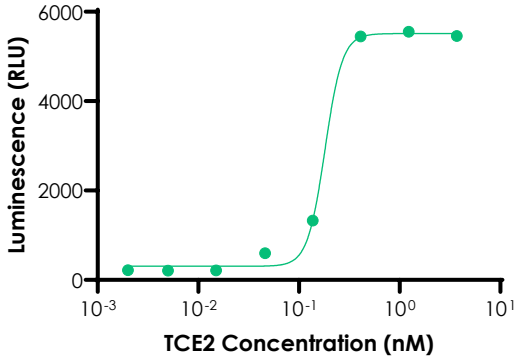
TAA1 TCE1 Cytotoxicity



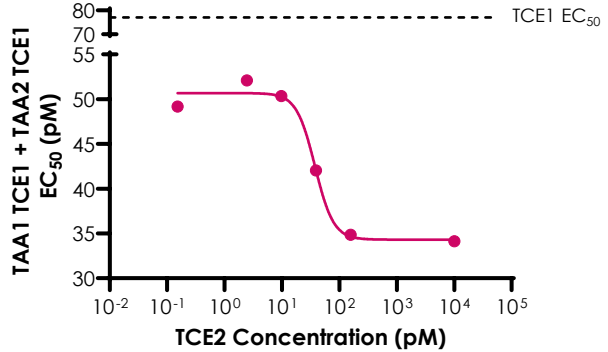
TAA2 TCE1 Cytotoxicity



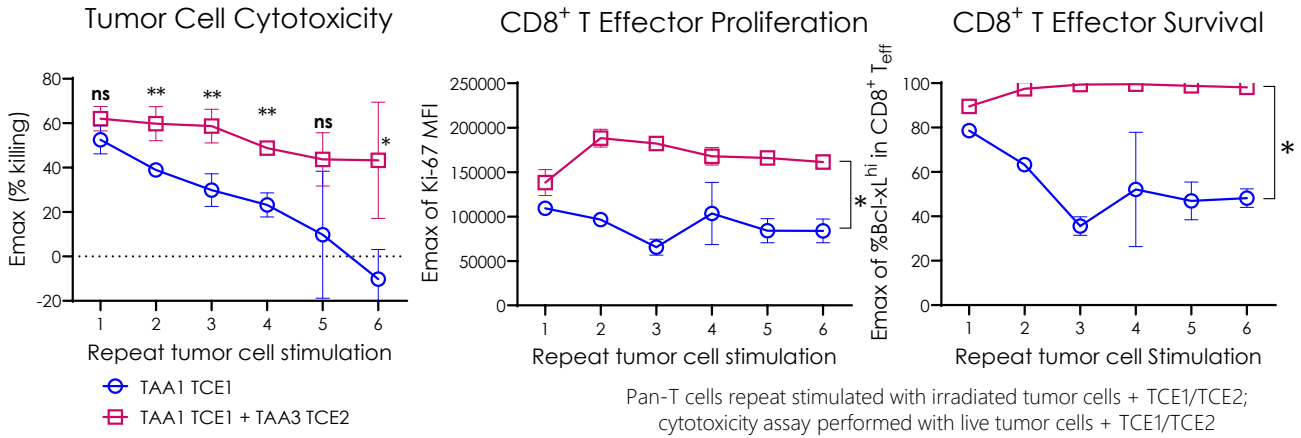
TAA3 TCE2 T Cell Activation



TCE2 enhances potency

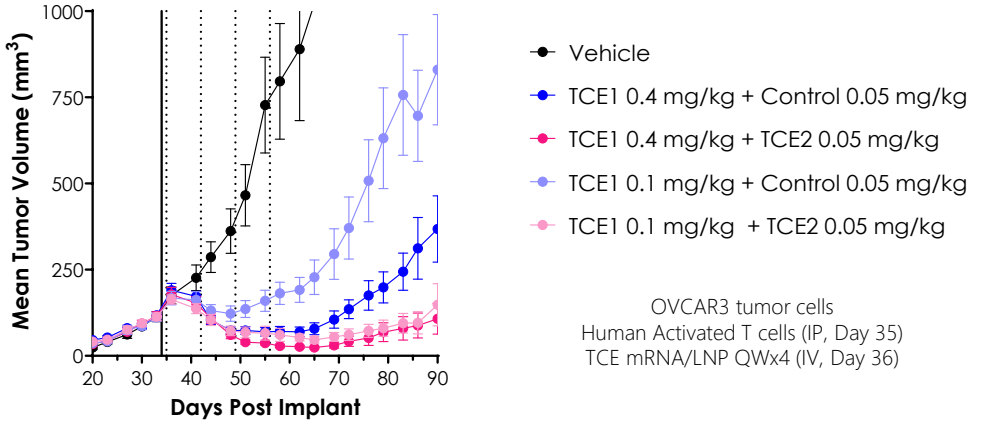


TCE2 improves tumor cell killing and T cell fitness in repeat stimulation assay in vitro



Pan-T cells repeat stimulated with irradiated tumor cells + TCE1/TCE2; cytotoxicity assay performed with live tumor cells + TCE1/TCE2

TCE1 anti-tumor efficacy is enhanced by TCE2 in vivo



OVCAR3 tumor cells
Human Activated T cells (IP, Day 35)
TCE mRNA/LNP QWx4 (IV, Day 36)

Next steps

Completion of IND enabling studies

Initiate Phase 1 study in 2027

mRED portfolio

Horizon 2 — Emerging modalities

Cancer antigen therapies

mRNA-4359

mRNA-4106

mRNA-4200

mRNA-4194

T-cell engagers
(surface antigen)

mRNA-2808

mRNA-2151

Cell therapy enhancers

mRNA-4203

Multiple sclerosis therapeutic

mRNA-1195

Horizon 3 — Future modalities

In vivo CAR-T

mRNA-6007

In vivo CAR-M

T-cell engagers
(intracellular targets)

Tolerizing therapy

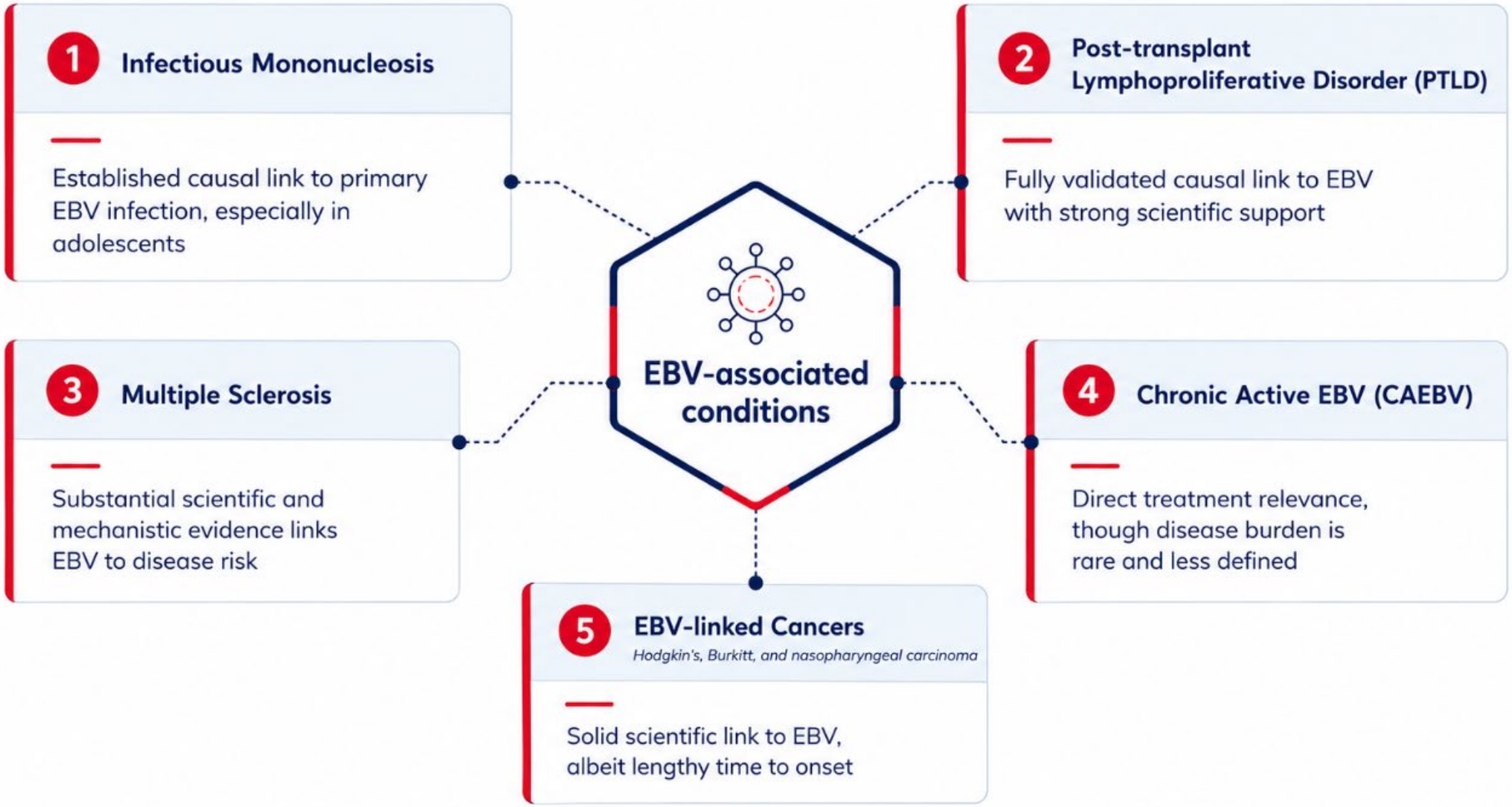
EBV therapeutic

mRNA-1195

Sumana Chandramouli, PhD
Executive Director, Program Leader, Infectious Disease

moderna

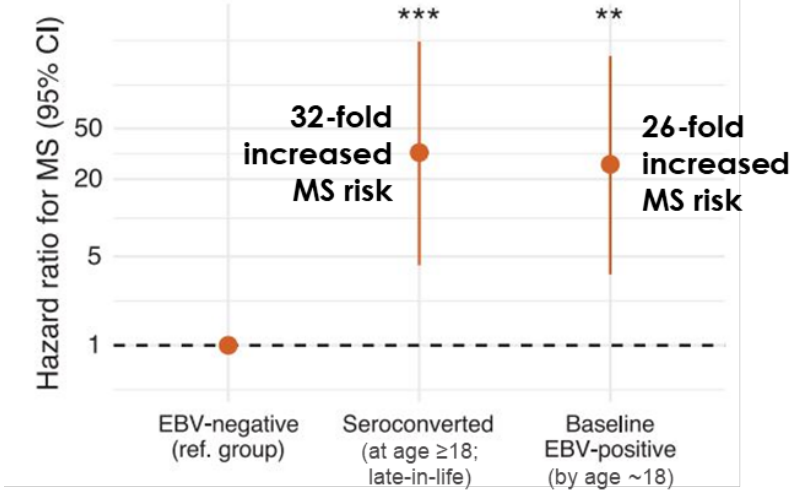
EBV is associated with several serious medical conditions that could be addressed through mRNA



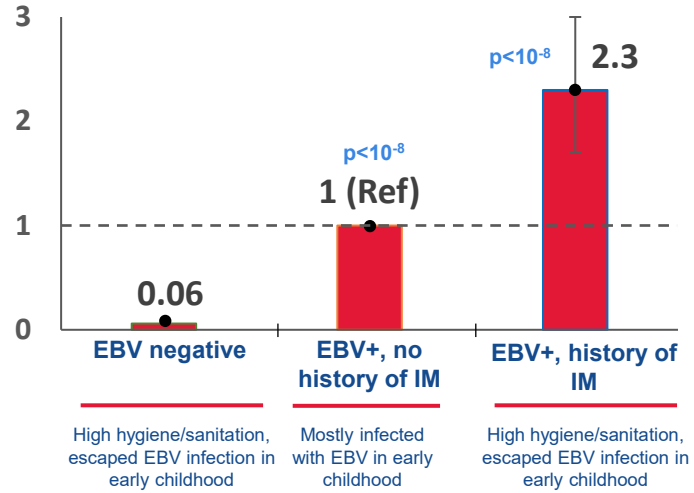
Etiologic link between EBV and multiple sclerosis (MS)

- **Nearly 1M people in the U.S. have MS** [1]
- EBV seropositivity is nearly universal in MS and seronegative individuals have a negligible risk of MS
- Recent landmark study established a **~32 fold increased risk of developing MS following EBV seroconversion**[2]
- It was previously established that **infectious mononucleosis** (IM) is an MS risk factor, beyond the contribution of EBV alone; in addition, the epidemiology of IM and MS are similar

MS Risk by EBV serostatus[2]

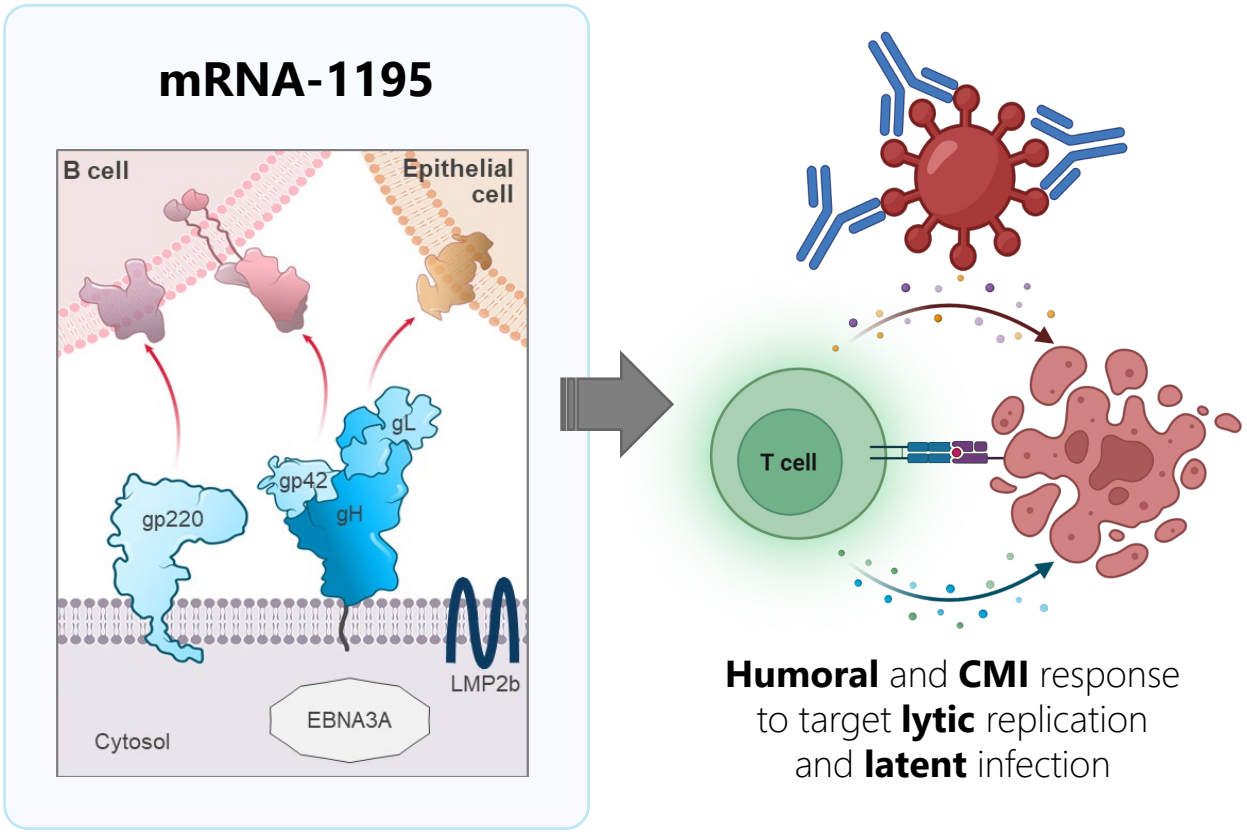


MS Risk by history of IM and EBV serostatus[3]



Sources: 1. https://www.nationalmssociety.org/About-the-Society/MS-Prevalence; 2 Bjornevik et al., Science. 2022 Jan 21;375(6578):296-301. doi: 10.1126/science.abj8222; 3. Ascherio and Munger, Semin Neurol. 2016 Apr;36(2):103-14. doi: 10.1055/s-0036-1579693.

mRNA-1195 is designed to tackle multiple EBV-associated conditions



Humoral and **CMI** response to target **lytic** replication and **latent** infection

Vaccine composed of **lytic** and **latent antigens**

Primary indication(s):

1. **Treatment of Multiple Sclerosis**
   - Immune dysregulation of/by EBV may be one of the underlying mechanisms of action
   - Vaccine MOA: restoring robust immune control of lytic and latent infection

2. **Post-transplant Lymphoproliferative Disorder (PTLD)**

mRNA-1195 Phase 1 Part A trial design (mRNA-1195-P101)

Design
Randomized, observer-blind, placebo-controlled, dose-ranging

Number of participants
350 healthy EBV-seropositive adults (18-55 years old)

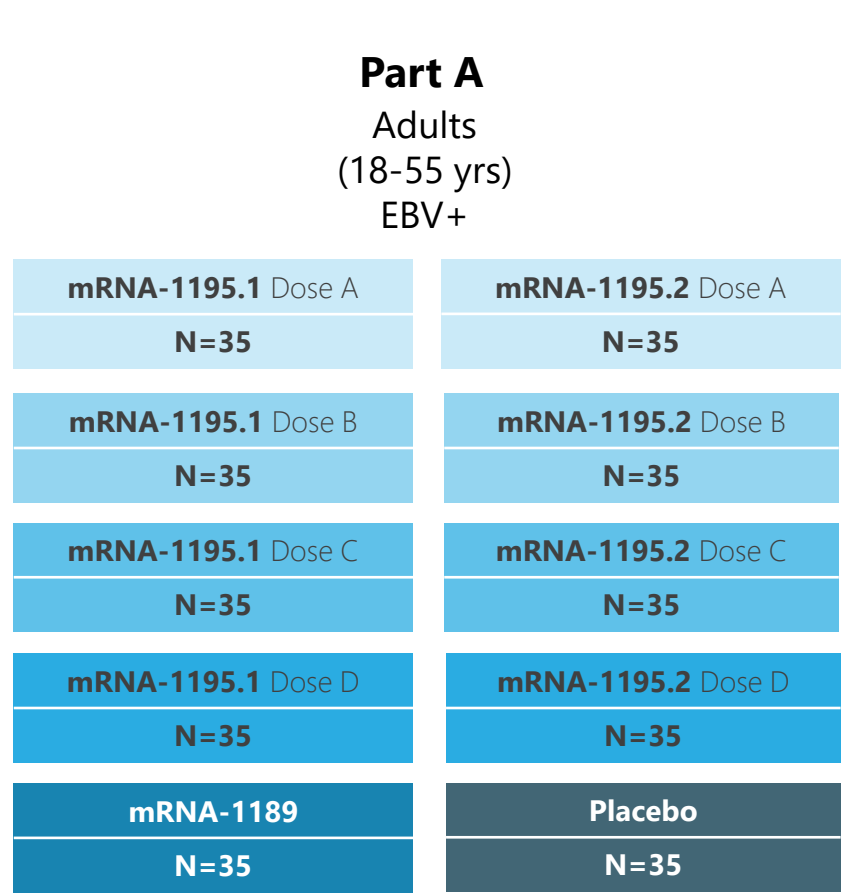
Vaccination schedule
Three injections mRNA-1195 (two different compositions), mRNA-1189 or placebo (0-2-6 month)

Primary Objective:
Safety and reactogenicity of mRNA-1195

Secondary Objective:
- Humoral immunogenicity at Days 1, 85, and 197 – B-cell nAbs, antigen bAbs

Key Exploratory Objectives:
- Humoral immunogenicity (incl. epithelial cell neutralization) at all timepoints
- Impact on EBV viral shedding in saliva
- Cellular immunogenicity (T-cell responses)

Duration
Study participants followed up for 6 months after last study injection



nAbs, neutralizing antibodies; bAbs, binding antibodies

mRNA-1195 was well tolerated with an acceptable safety profile: Local and systemic reactogenicity

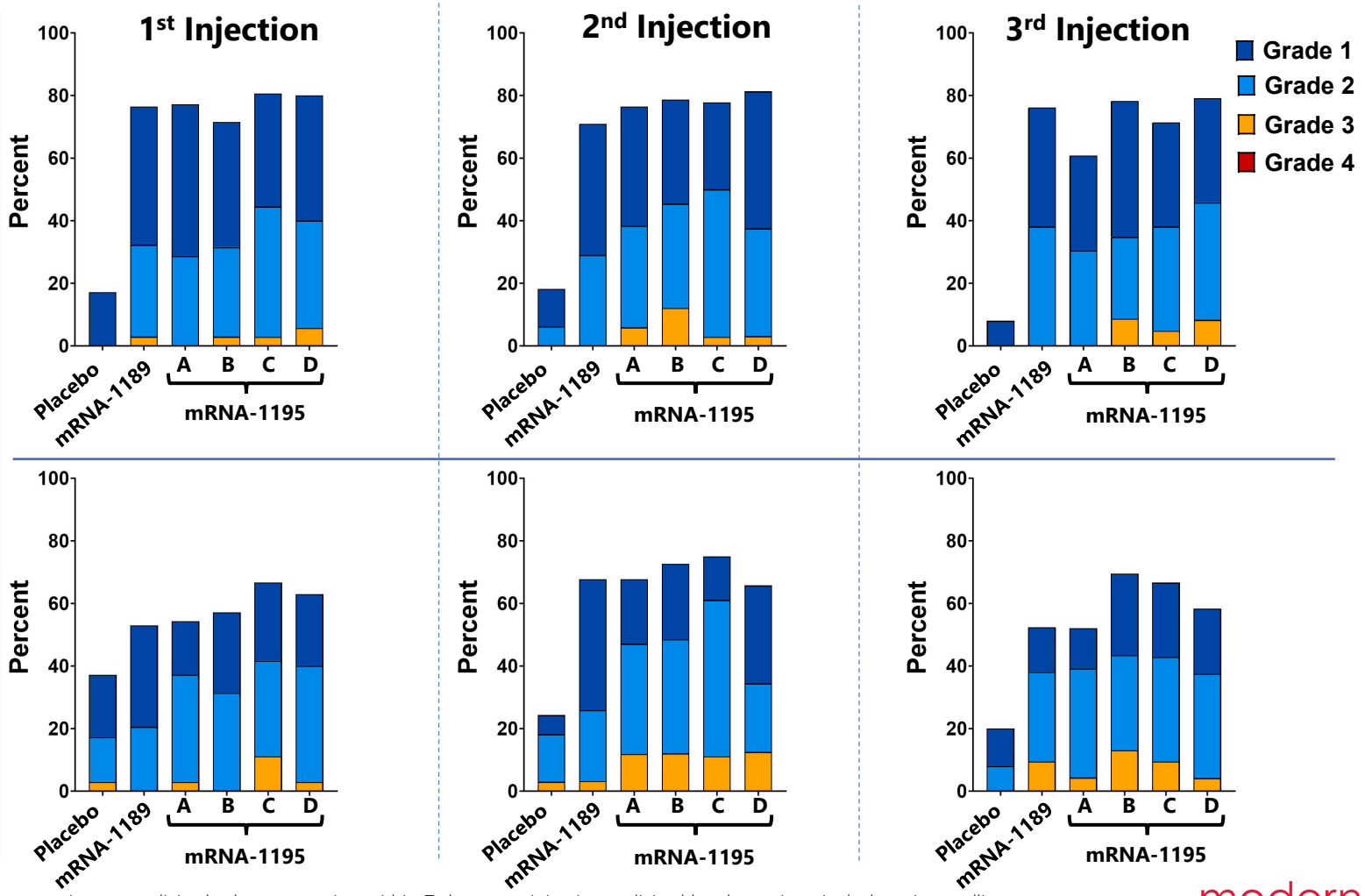
P101 Part A Data | = D1, D57, D169

Local reactogenicity

Pain was the most common local solicited adverse reaction following any injection

Systemic reactogenicity

Headache, fatigue, myalgia, and arthralgia were most common following any injection

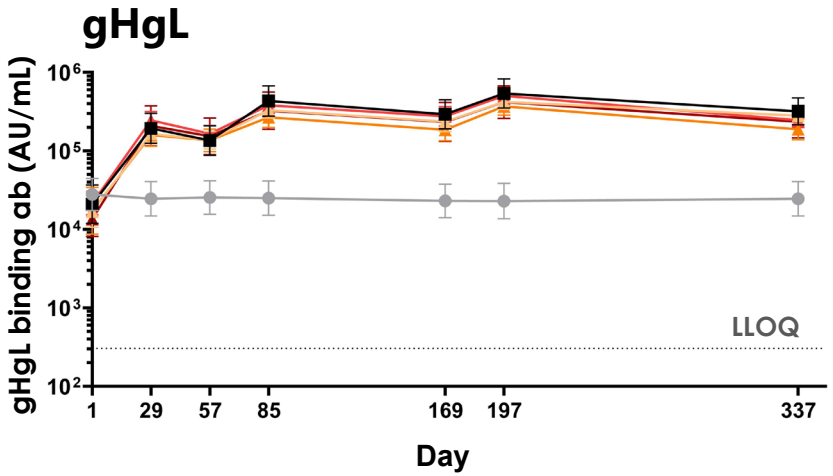


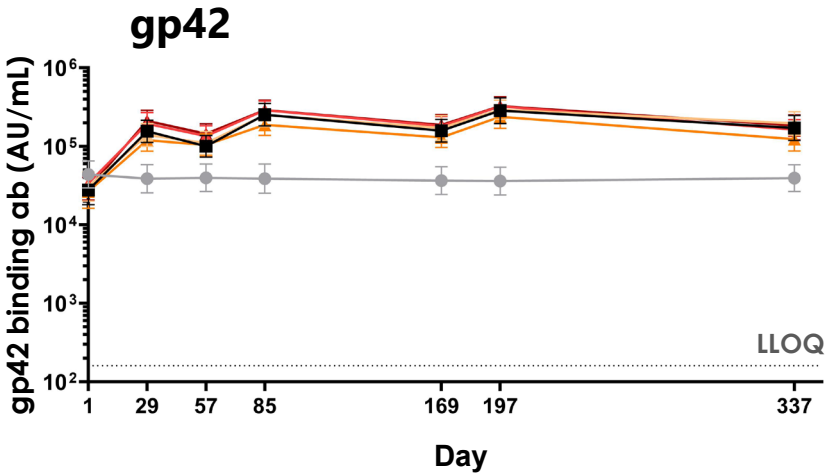
Displayed is percentage of participants reporting any solicited adverse reaction within 7 days post injection; solicited local reactions include pain, swelling, erythema and axillary swelling; solicited systemic reactions include headache, fatigue, myalgia, arthralgia, nausea/vomiting, chills and fever

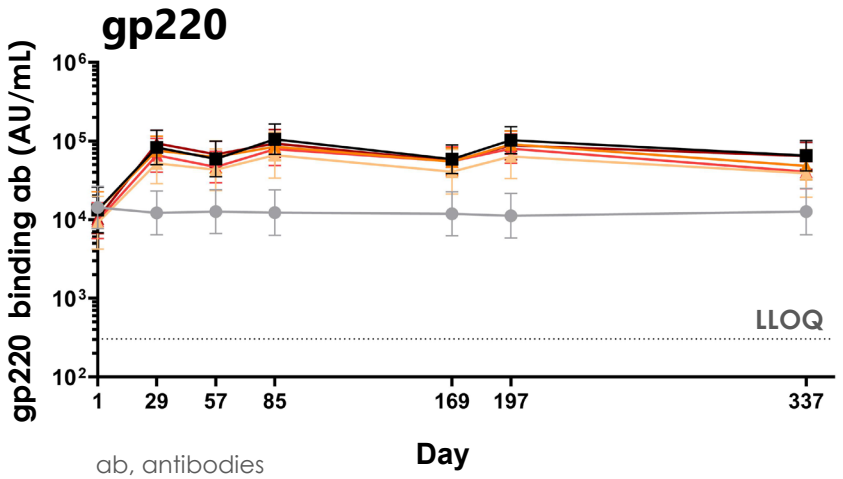
Humoral Immunogenicity: Binding antibodies to glycoproteins are boosted across mRNA-1195 dose levels

P101 Part A Data | = D1, D57, D169

- All mRNA groups had detectable boost in **gHgL, gp42** and **gp220** binding antibodies following mRNA injection
- Responses persisted above baseline through Day 337, 6 months post dose 3
- No pronounced dose response across dose levels tested







ab, antibodies

= Placebo
= mRNA-1189

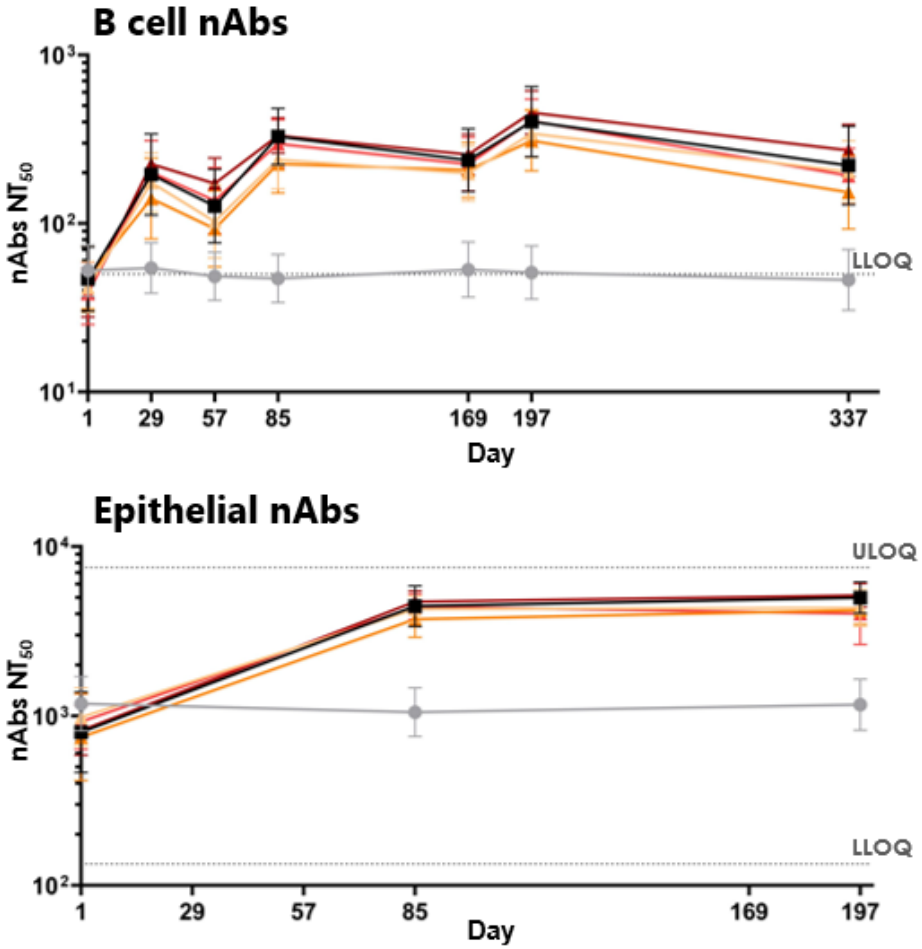
mRNA-1195
= Dose A
= Dose B
= Dose C
= Dose D

Day 1 = Baseline
Day 29 = 1 month post dose 1
Day 85 = 1 month post dose 2
Day 197 = 1 month post dose 3
Day 337 = 6 months post dose 3

Humoral Immunogenicity: Serum B-cell and epithelial nAbs are boosted across mRNA-1195 dose levels

P101 Part A Data | = D1, D57, D169

- All mRNA-1189 and mRNA-1195 groups had detectable boost in **B-cell and Epithelial nAbs** following mRNA injection
- Similar responses across dose levels tested with overlapping confidence intervals
- B cell nAb responses persisted above baseline through Day 337, 6 months post dose 3



= Placebo
= mRNA-1189

mRNA-1195
= Dose A
= Dose B
= Dose C
= Dose D

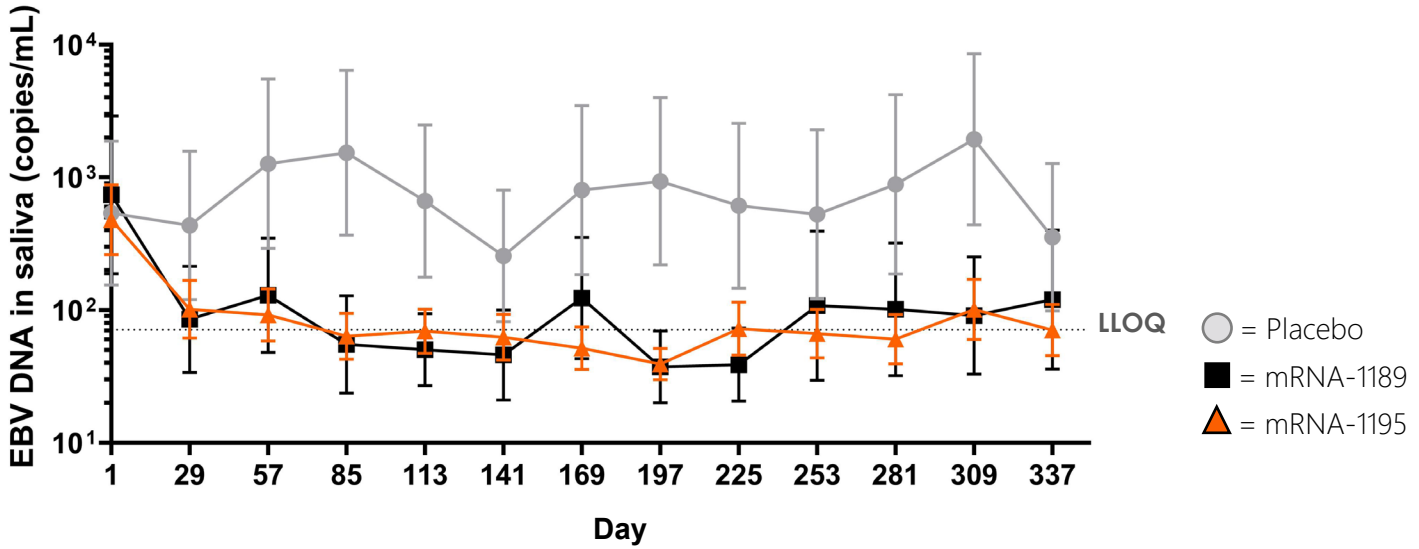
Day 1 = Baseline
Day 29 = 1 month post dose 1
Day 85 = 1 month post dose 2
Day 197 = 1 month post dose 3
Day 337 = 6 months post dose 3

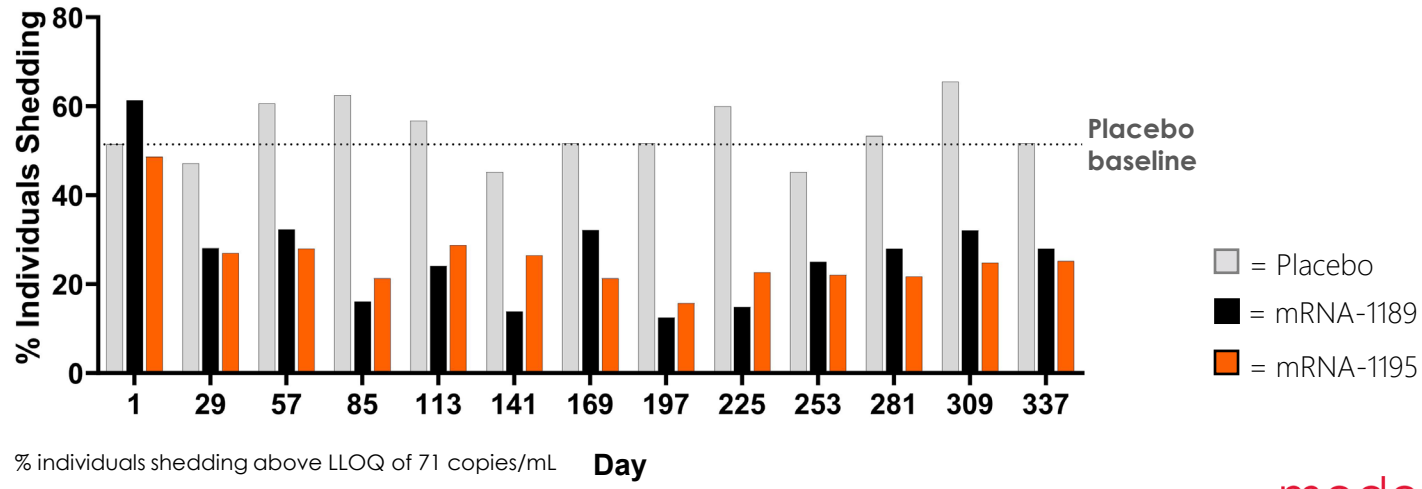
Epithelial nAbs tested only at select timepoints shown

mRNA-1195 impacts EBV shedding in saliva

P101 Part A Data | = D1, D57, D169

- mRNA-1195 dose levels were consolidated and analyzed together to provide better qualitative description of the viral shedding data
- All mRNA groups had detectable **decrease in EBV DNA** shed in saliva compared to placebo group starting 1 month after the 1st injection and persisting through 6 months after the 3rd injection
- Compared to placebo, the **frequency of individuals shedding was reduced** in the mRNA-1195 and mRNA-1189 groups, confirming findings in mRNA-1189 Phase 1

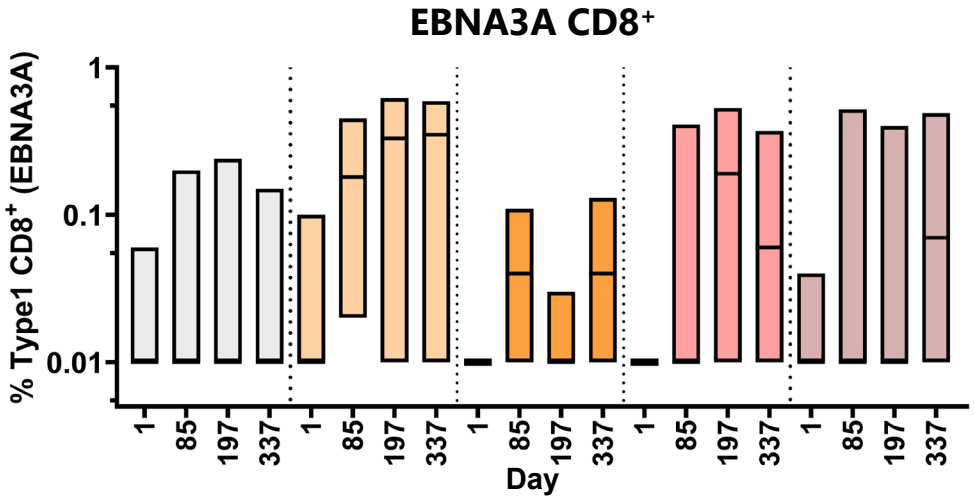


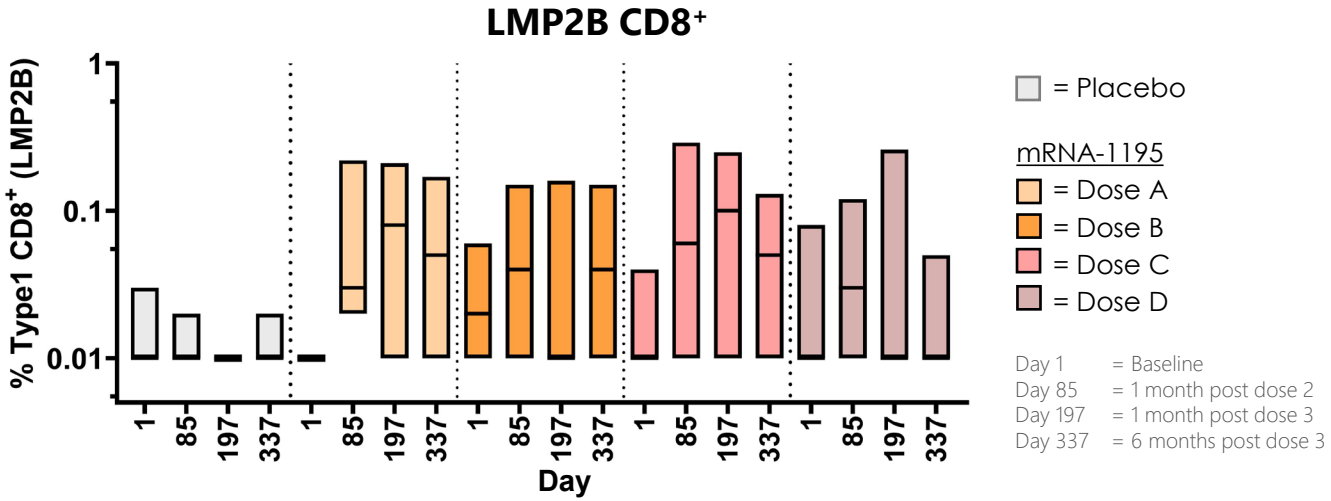


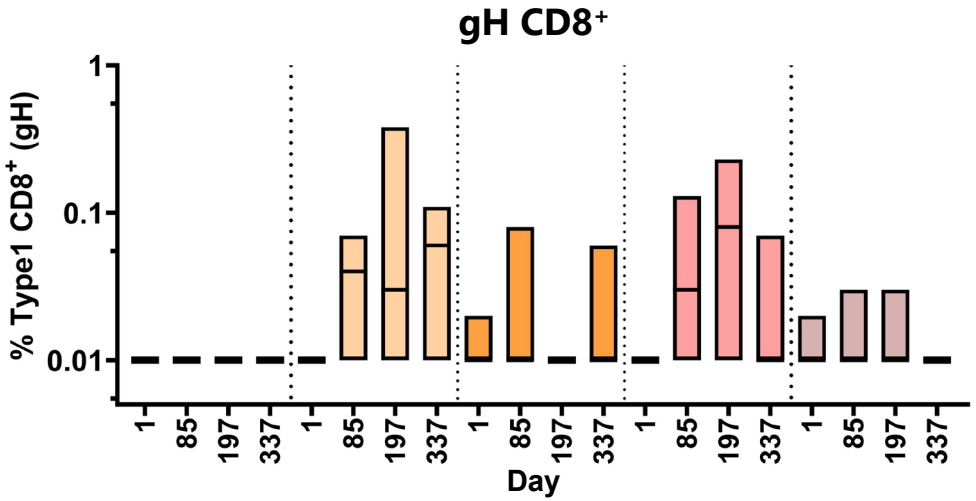
% individuals shedding above LLOQ of 71 copies/mL

Cell-mediated Immunogenicity – CD8⁺ responses to latent and lytic antigens are boosted across mRNA-1195 dose levels

P101 Part A Data | = D1, D57, D169





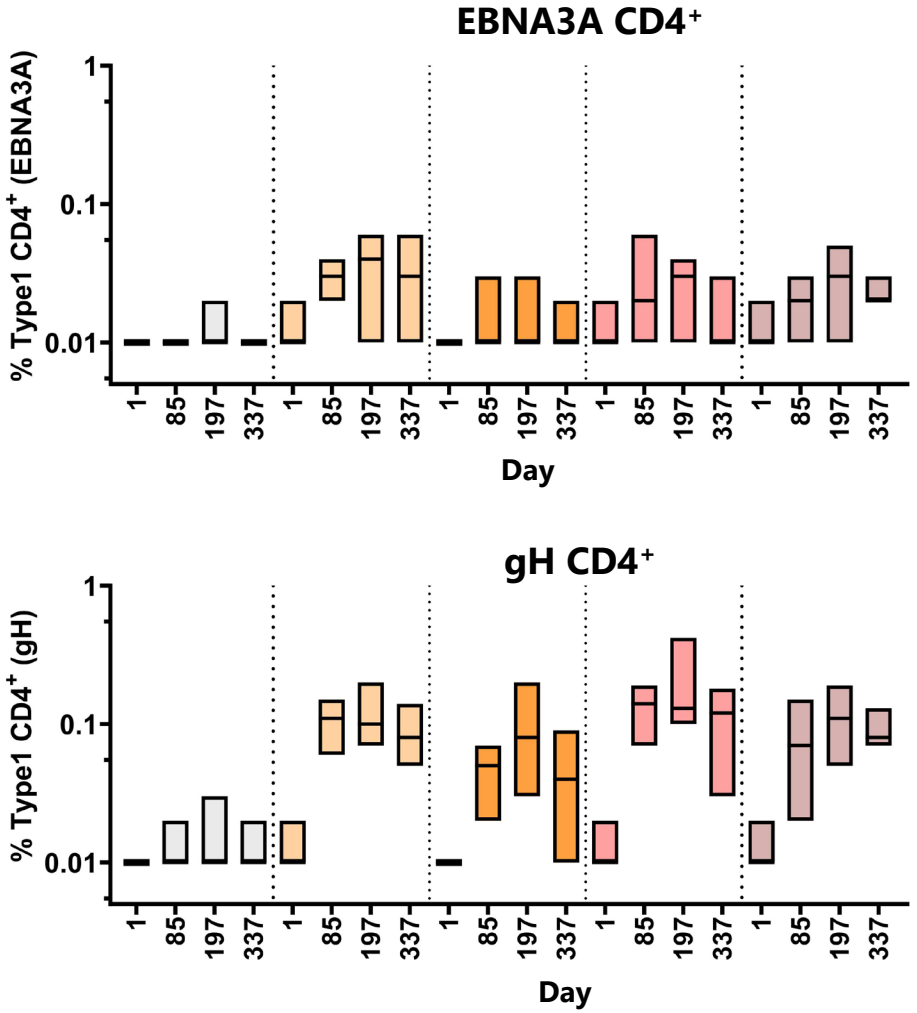


- Range of CD8⁺ responses detected at baseline in EBV+ participants across treatment arms, suggesting heterogeneity in general population
- Overall trend towards increased CD8⁺ responses to EBNA3A, LMP2B and gH following 2 or 3 injections
- Responses persisted through Day 337, 6 months post dose 3, in most mRNA groups

Cell-mediated Immunogenicity – CD4+ responses to latent and lytic antigens are boosted across mRNA-1195 dose levels

P101 Part A Data | = D1, D57, D169

- Baseline CD4+ T cell responses detected at low levels across EBV+ participants in all groups
- Overall trend towards increased CD4+ responses to EBNA3A and gH following 2 or 3 injections
- Responses persisted through Day 337, 6 months post dose 3, in mRNA groups



= Placebo

mRNA-1195
= Dose A
= Dose B
= Dose C
= Dose D

Day 1 = Baseline
Day 85 = 1 month post dose 2
Day 197 = 1 month post dose 3
Day 337 = 6 months post dose 3

Note: Box plots show interquartile range (Q1-Q3) with lines indicating median

EBV (mRNA-1195) Phase 1 Part B trial design

Fully enrolled

Design
Randomized, observer-blind, placebo-controlled, dose-ranging

Number of participants
120 healthy EBV-seronegative and EBV-seropositive adults (18-30 years old)

Vaccination schedule
Three injections of selected mRNA-1195 composition or placebo (0-2-6 month)

Primary Objective:
Safety and reactogenicity of mRNA-1195

Secondary Objective:
- Humoral immunogenicity at Days 1, 85, and 197 – B-cell nAbs, antigen bAbs

Key Exploratory Objectives:
- Humoral immunogenicity (incl. epithelial cell neutralization) at all timepoints
- Impact on EBV viral shedding in saliva (EBV+ only)
- Cellular immunogenicity (T-cell responses)

Duration: 18-months
Study participants followed up for 12 months after last study injection

Part B

Adults
(18-30 yrs)
EBV(-/+)

mRNA-1195 Dose A
N=30 (15/15)
mRNA-1195 Dose B
N=30 (15/15)
mRNA-1195 Dose C
N=30 (15/15)
Placebo
N=30 (15/15)

nAbs, neutralizing antibodies; bAbs, binding antibodies

mRNA-1195-P201 Phase 2 proof-of-concept in MS Design

Ongoing

Design
Randomized 1:1:1, observer-blind, placebo-controlled, dose-ranging

Number of participants
180 EBV+ adults 18 to <55 yrs of age diagnosed with RRMS, CIS, RIS

Vaccination schedule
3 injections at 0, 2, 6 month schedule of selected composition of mRNA-1195 at Dose A or Dose B, or Placebo

Primary Objective:
Safety and reactogenicity

Secondary Objectives:
- Impact on MRI markers of MS disease activity
- Humoral immunogenicity

Key Exploratory Objectives:
- Impact on other markers of MS disease activity and EBV viral activity
- Additional assessments of immunogenicity including cellular immunogenicity

nAbs, neutralizing antibodies; bAbs, binding antibodies; pwMS, people with MS

EBV+ pwMS
(18-55 years of age)
n=180

mRNA-1195 Dose A
N=60
mRNA-1195 Dose B
N=60
Placebo
N=60

Sentinel cohort fully enrolled; DSMB recommendation to proceed with dose escalation

EBV Tx mRNA-1195 summary and next steps

Safety

- Phase 1 interim analysis data demonstrate that mRNA-1195 is generally well tolerated in EBV-seropositive adults 18-55 yrs

Immunogenicity

- EBV-seropositive participants across mRNA-1195 dose groups showed increases in B-cell nAbs and epithelial nAbs, and binding antibodies to glycoproteins from baseline following 3 injections
- mRNA-1195 boosted $CD8^+$ and $CD4^+$ T cell responses to latent and glycoprotein antigens in EBV-seropositive participants across tested dose levels
- Humoral and cell-mediated immunity responses persisted above baseline through 6 months after the last injection
- mRNA-1195 reduced measurable viral shedding in saliva of EBV-seropositive recipients through 6 months after the last injection

Next steps

- Phase 1 part B data expected in 2H2026
- Phase 2 proof-of-concept MS study data

mRED portfolio

Horizon 2 — Emerging modalities

Cancer antigen therapies

mRNA-4359

mRNA-4106

mRNA-4200

mRNA-4194

Cell therapy enhancers

mRNA-4203

T-cell engagers (surface antigen)

mRNA-2808

mRNA-2151

Multiple sclerosis therapeutic

mRNA-1195

Horizon 3 — Future modalities

In vivo CAR-T

mRNA-6007

T-cell engagers (intracellular targets)

In vivo CAR-M

Tolerizing therapy

In vivo CAR-T

mRNA-6007: Autoimmune disease

Lin Guey, PhD
Chief Scientific Officer, Therapeutics Research

moderna

Background of B-cell mediated autoimmune disease and current and developmental stage therapies

B cells play a central role in autoimmune diseases, via both antibody-dependent and independent mechanisms

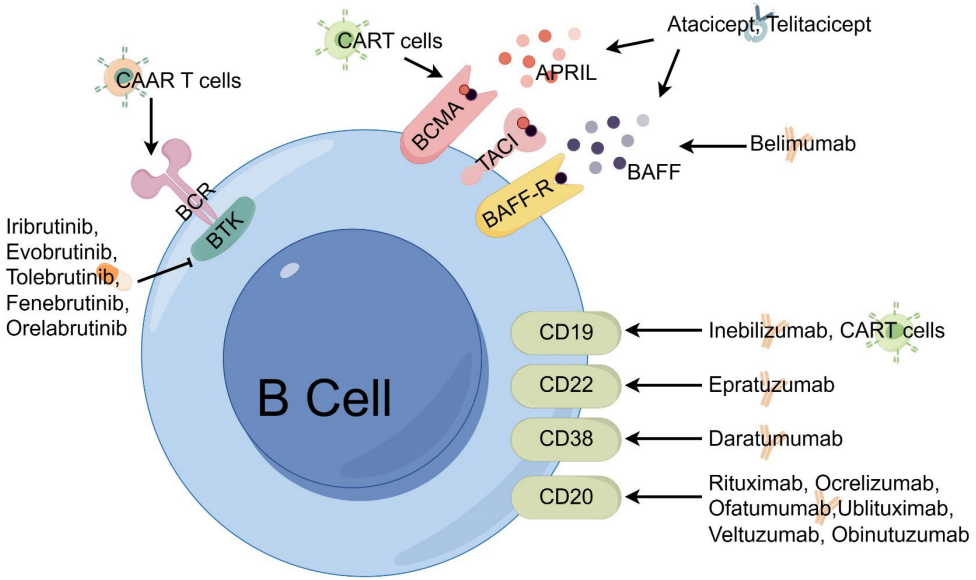
Current standard of care (SOC) manages disease, but does not reset the immune system

- Corticosteroids and immunosuppressants
- Targeted biologics (e.g., anti-CD20 mAbs)

mAbs can deplete circulating B cells, but may have limited tissue penetration, leading to incomplete depletion in key diseases sites (e.g., lymph nodes, spleen)

CAR-T enables deeper and broader B cell depletion, including in tissues

- May overcome antigen variability and low-expression targets
- Early data show more complete depletion vs mAbs (e.g., lymph node clearance)



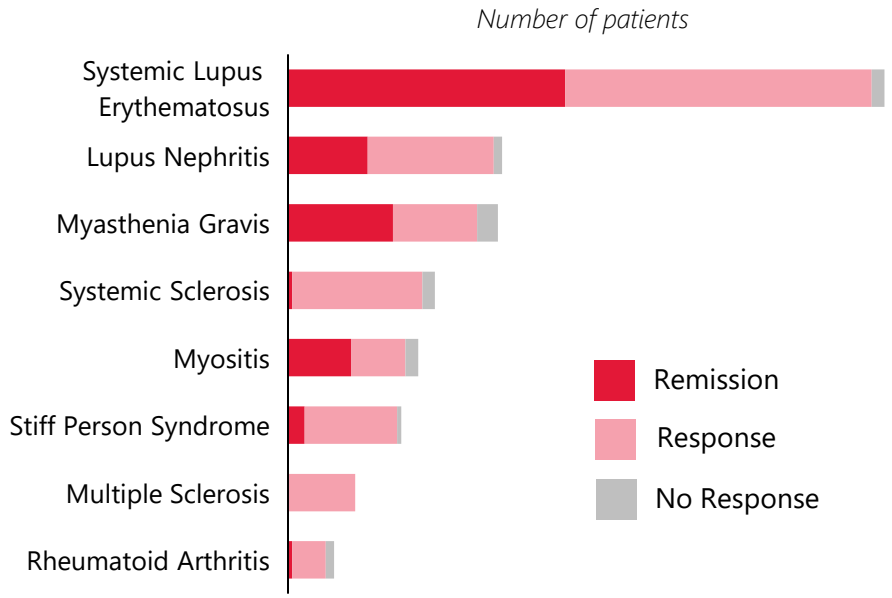
B cell targets and corresponding drugs and MOAs (Li et. al., Front Immunol. 2023)

Emerging clinical data derisks *ex vivo* CAR-T for B cell mediated autoimmune diseases

400+
Autoimmune patients treated to date
Across ~50 publications

270+
Total years of follow-up

Ex vivo CAR-T responses in autoimmunity



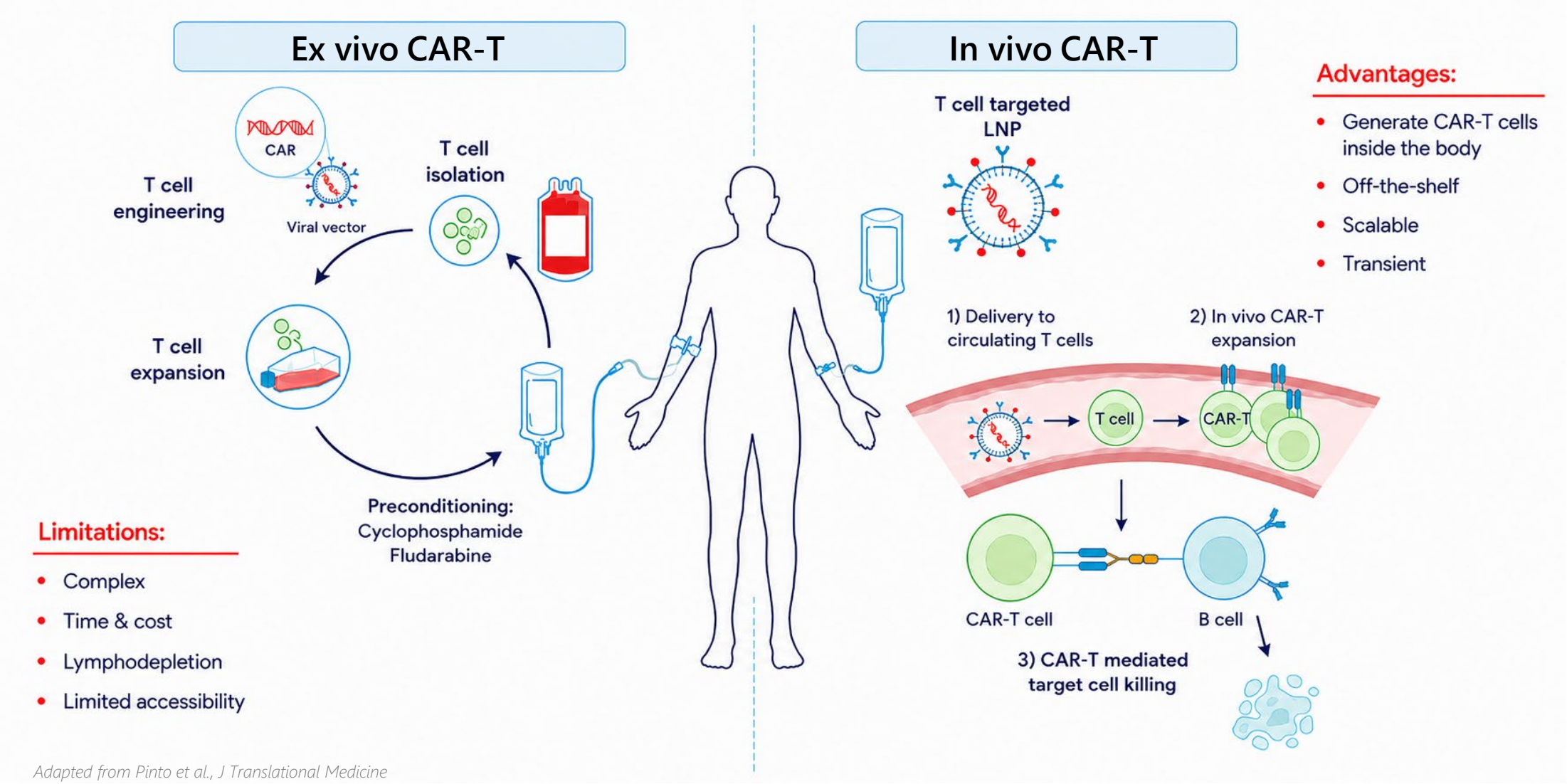
Growing body of data; many case studies in additional autoimmune indications

Efficacy
- Strong remission rates in highly refractory patients
- Clinical data supports 1+ yr durability, although long-term data are still emerging

Dose and Persistence
- Similar or slightly lower dose levels and expansion to oncology, but significantly shorter persistence
- Lymphodepletion largely used in *ex vivo* CAR-T studies

Tolerability
- Acceptable safety profile, with only mild CRS (generally Grade 1-2) and minimal low-grade ICANs
- Improved safety compared to oncology
- Some severe infections and IVIG supplementation, but usually managed through standard treatment

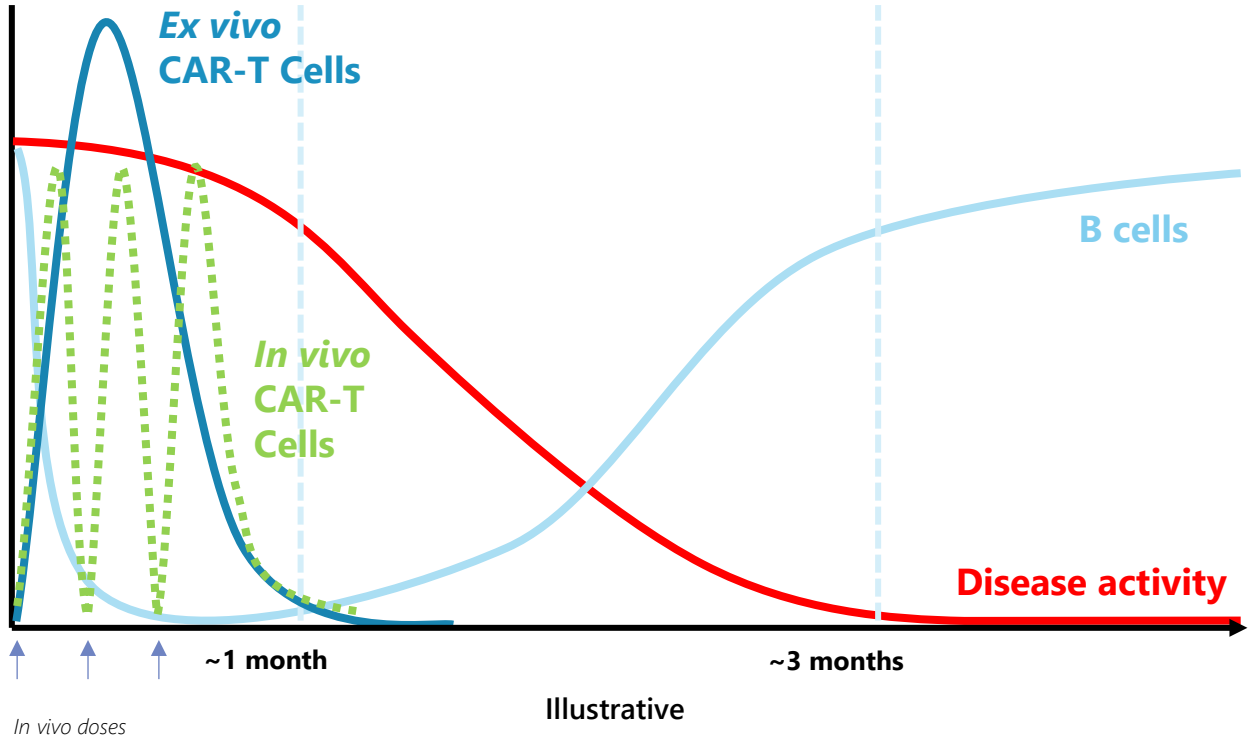
A different approach: *in vivo* CAR-T



Adapted from Pinto et al., J Translational Medicine

In vivo CAR-T offers the potential to induce deep immune reset with the convenience of an 'off-the-shelf' therapy

Transient CAR expression has the potential to drive deep responses in autoimmune diseases with limited dosing



- Infused *ex vivo* CAR-T cells only last ~15 – 60 days
- B cell depletion is rapid with reconstitution after a few months
- Disease control can last months to years
- *In vivo* CAR-T may require several doses but provide similar impact on B cell depletion and disease control

In vivo CAR-T exemplifies the principles mRED uses to build a new modality

Platform advantage
Advance modalities where our platform technology offers distinct competitive advantages and program differentiation

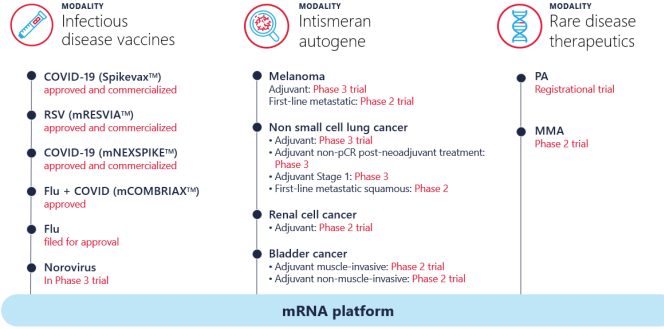
Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction

Portfolio balance
Build a diversified portfolio by balancing breadth across distinct areas of opportunity with disciplined expansion within each modality

Platform competitive advantage

- Multiplexing
- Intracellular proteins
- Transmembrane proteins
- Complex proteins
- T cell response

Proof-of-concept data from sentinel programs de-risk modalities and can accelerate development plans



In vivo CAR T, mRNA-6007, was selected as lead candidate based on comprehensive screening, optimization, and preclinical evaluation

Platform advantage
Advance modalities where our platform technology offers distinct competitive advantages and program differentiation

Platform competitive advantage

- Multiplexing
- Intracellular proteins
- Transmembrane proteins
- Complex proteins
- T cell response

In vivo, mRNA-LNP CAR-T is a scalable, controllable way to reset pathogenic B-cell immunity; Moderna's mRNA platform makes us uniquely positioned to lead in autoimmunity

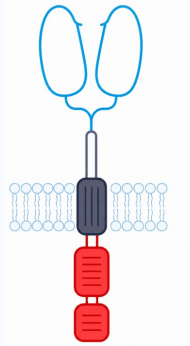
LNP

Clinically validated base particle

Targeting Moiety

- Internally discovered
- Humanized & characterized
- Validated linker chemistry

CAR Strategy



- Humanized binders & clinically validated CAR formats
- mRNA platform allows for multiplexing

mRNA-6007 is the sentinel program designed to unlock a scalable *in vivo* CAR-T modality

Scalability
Prioritize modalities that can generate multiple follow-on programs after sentinel risk reduction

Portfolio balance
Build a diversified portfolio by balancing breadth across distinct areas of opportunity with disciplined expansion within each modality

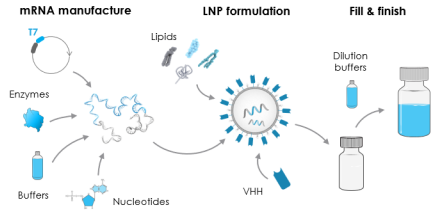
Follow-on product concepts leveraging the same LNP include *in vivo* TCR/CAR-T for oncology (mRNA and gene insertion cargos) and T cell reprogramming

CAR-T with applicability across multiple autoimmune diseases involving B and plasma cells. Current Phase 1 focus on systemic lupus erythematosus (SLE) and other B cell mediated autoimmune diseases

Modality:
***In vivo* CAR-T**

mRNA-6007 builds on validated technology, regulatory experience, CMC expertise, and end-to-end manufacturing

ID vaccines	*Billions of doses*	**Commercial**
Intismeran	*Individualized*	**Late-stage clinical**
Rare disease	*Mid-scale Chronic*	**Late-stage clinical**



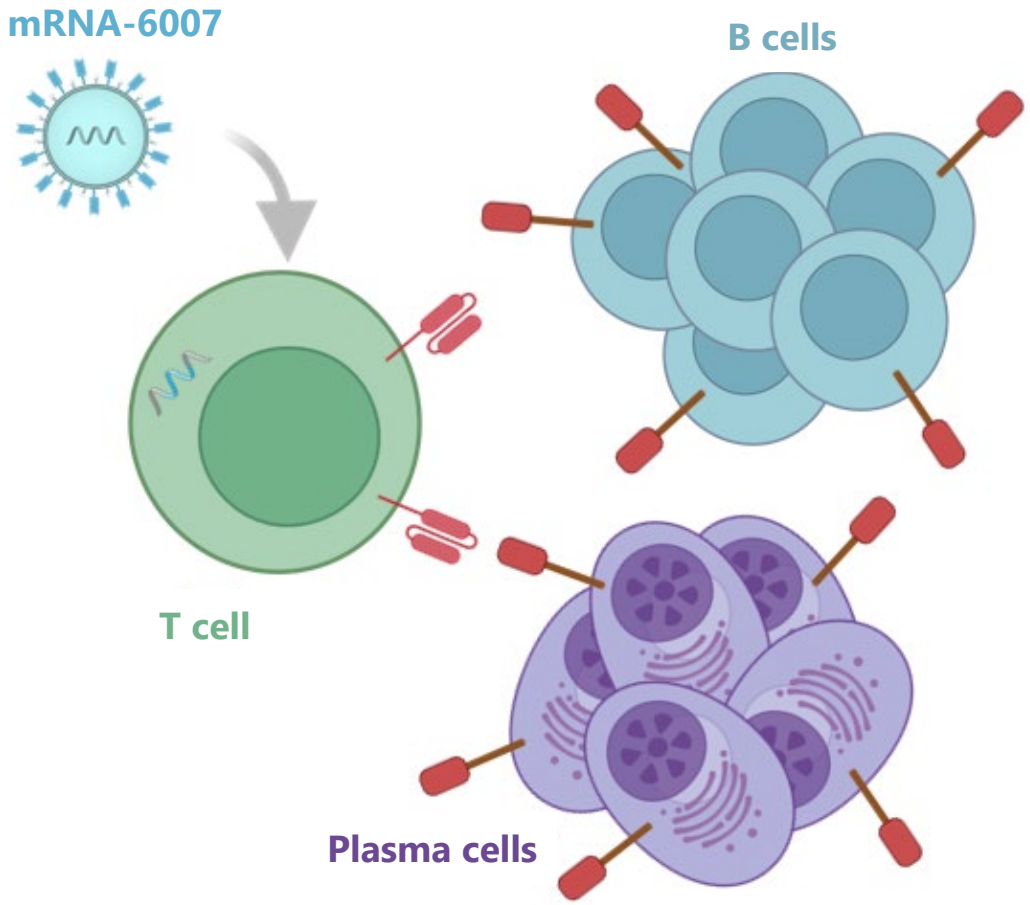
Validated technology
Proven mRNA/LNP platform with extensive clinical experience

Proven regulatory strategies
Established health authority approach and platform toxicology and non-clinical data to support filings

De-risked CMC path
Proprietary processes for mRNA and LNP and scaled processes and analytics to support pivotal studies

In-House end-to-end manufacturing
Integrated plasmid to drug product and quality control with global network supporting large capacity

mRNA-6007 mechanism of action



Targeted LNPs deliver mRNA into CD4 and CD8 T cells and NK cells, where mRNA is translated into **CARs targeting B cell antigens**

CAR-T cells circulate in the body and recognize B and plasma cells with the antigens (including pathogenic cells), leading to **cytotoxicity**

Pathogenic B cells are depleted, leading to eventual repopulation with naïve B cells and an **immune "reset"**

CD19 CAR mRNA/LNP-A shows dose-dependent CAR-T expression and complete B cell depletion in spleens of humanized mice

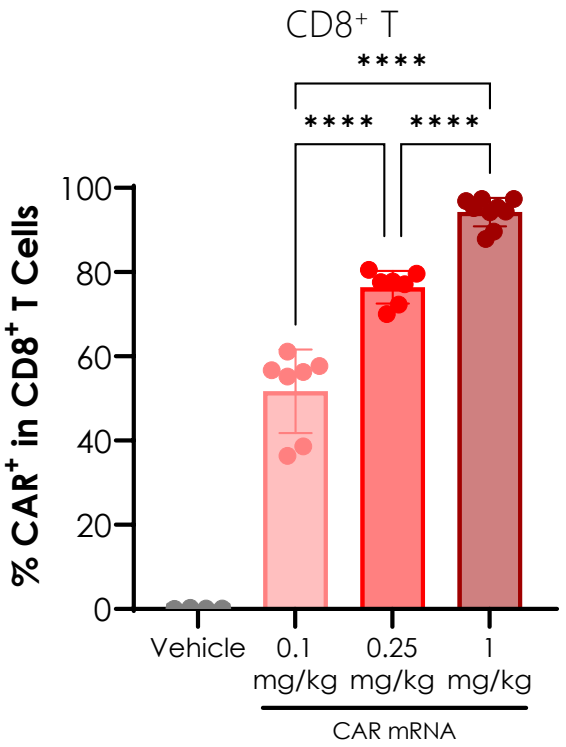
hCD34⁺ NSG

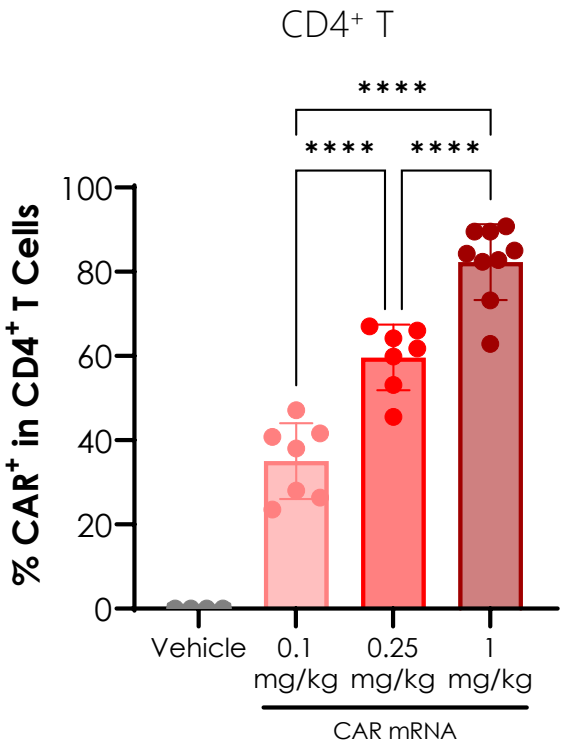
CD19 CAR mRNA
LNP-A

IV
Q3/4D×3
(D1, D4, D8)

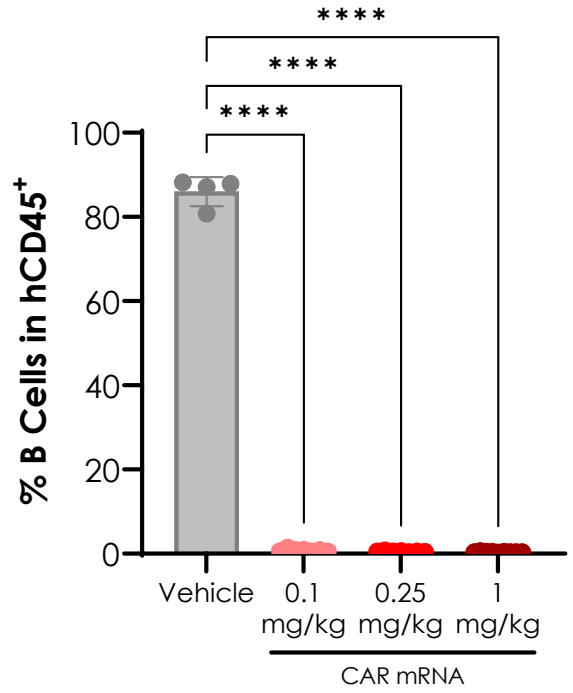
Termination 24h
post-last dose

CAR⁺ T cells (spleen)





B cells (spleen)



24 hours post 2nd dose (results similar post 3rd dose)

In NHP, no significant change in liver enzymes from mRNA/LNP treatment or CD20 CAR

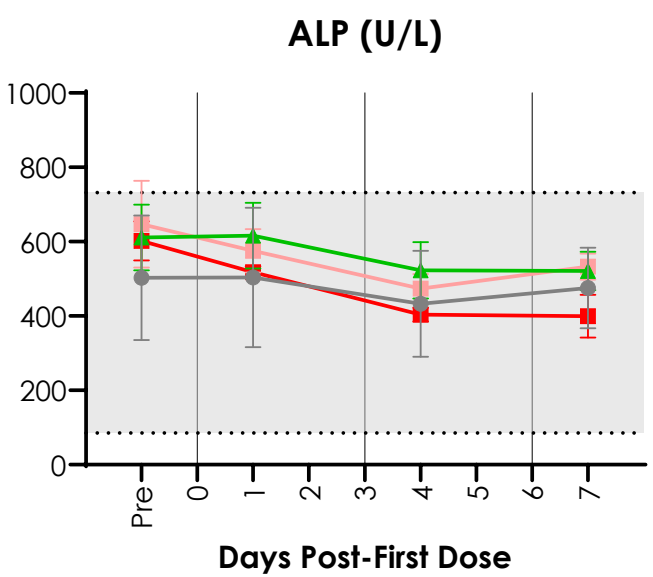
Cynomolgus macaques

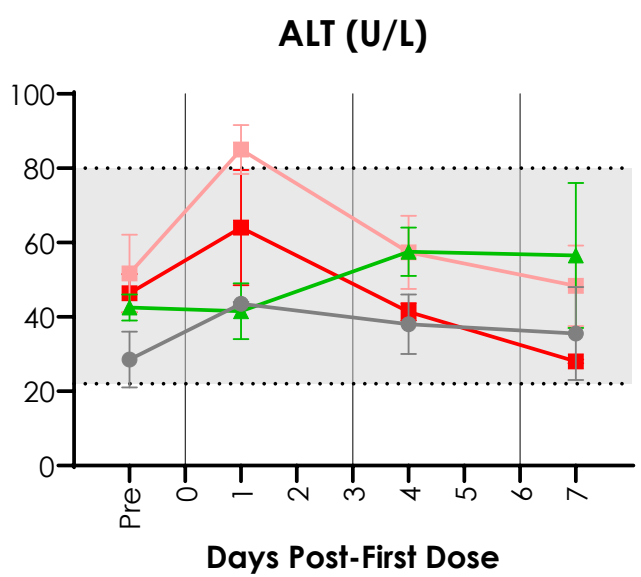
CD20 CAR mRNA
LNP-A

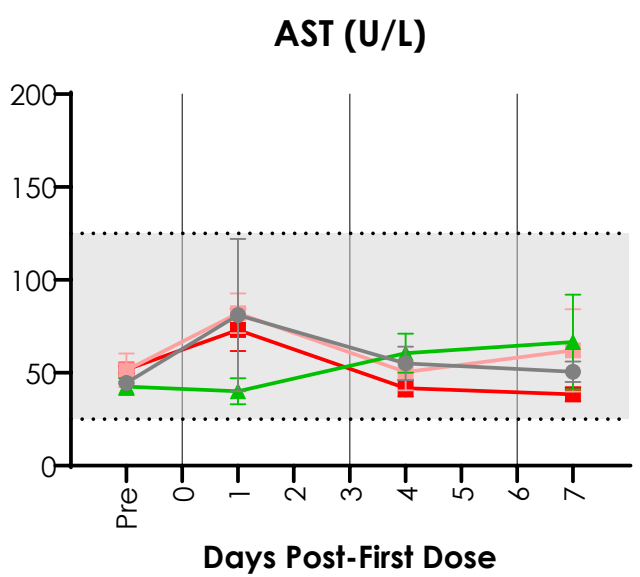
0.5 or 1 mpk IV
Q3D×3
(D0, D3, D6)

No pre-treatment before dosing

Liver enzymes







No significant changes in body temperature or body weight

Shaded areas = range in healthy NHPs

- Vehicle
- mGL/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (1 mg/kg)

~70-90% of CD8⁺ T cells across periphery & tissues express fluorescent reporter after three doses of CD7-targeted LNP-A delivering mGL mRNA

Cynomolgus macaques

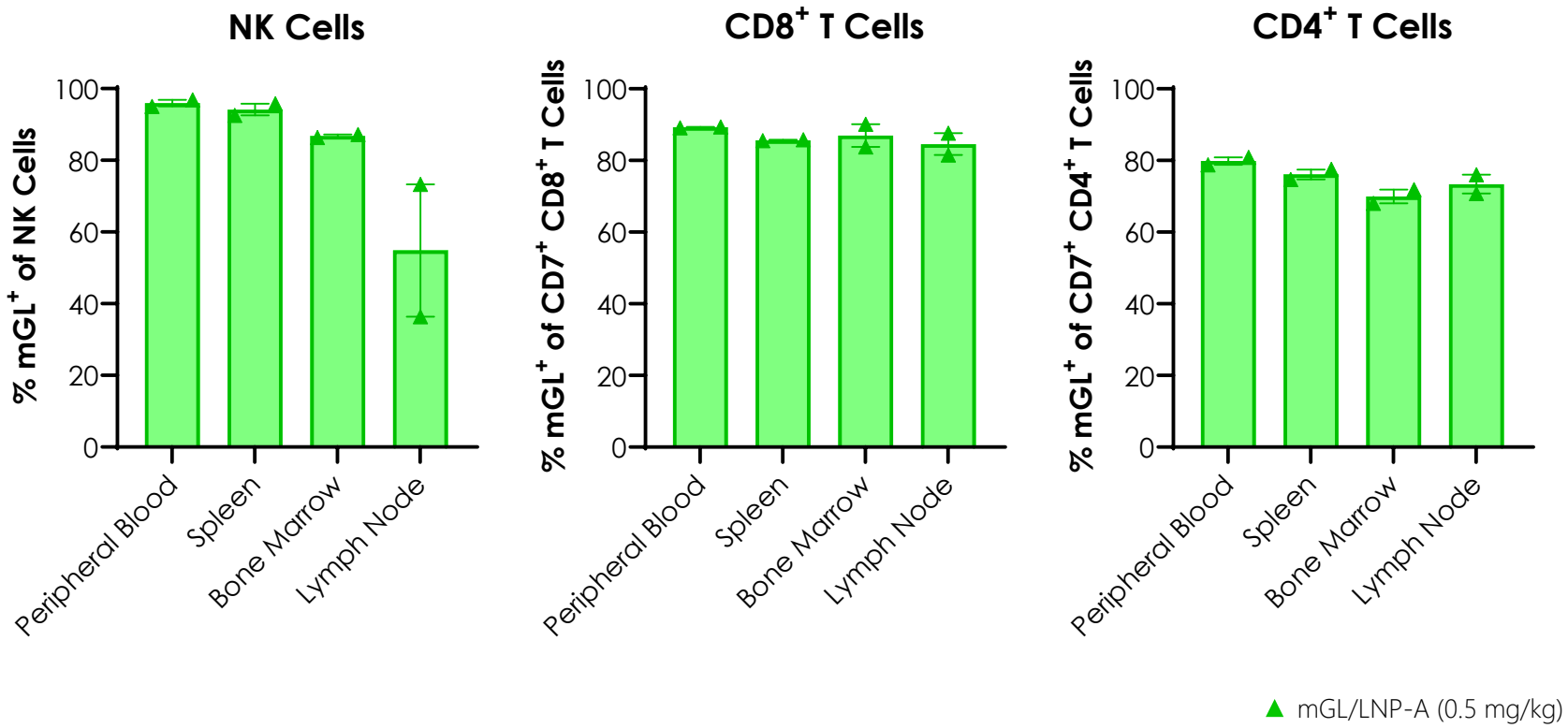
mGL mRNA
LNP-A

0.5 mpk IV
Q3D×3
(D0, D3, D6)

No pre-treatment before dosing

Flow analysis 48h post 3rd dose (D8)

mGreenLantern reporter expression in CD7⁺ immune cells after three doses



Rapid and complete peripheral B cell depletion observed in NHP

Cynomolgus macaques

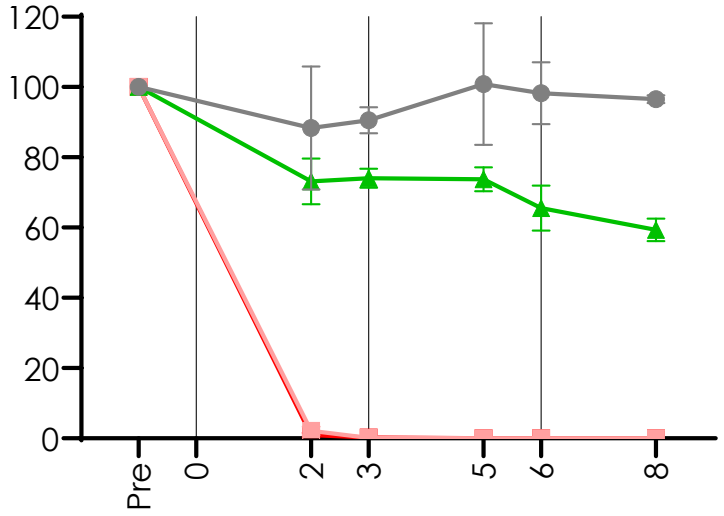
CD20 CAR mRNA LNP-A

0.5 or 1 mpk IV Q3D×3 (D0, D3, D6)

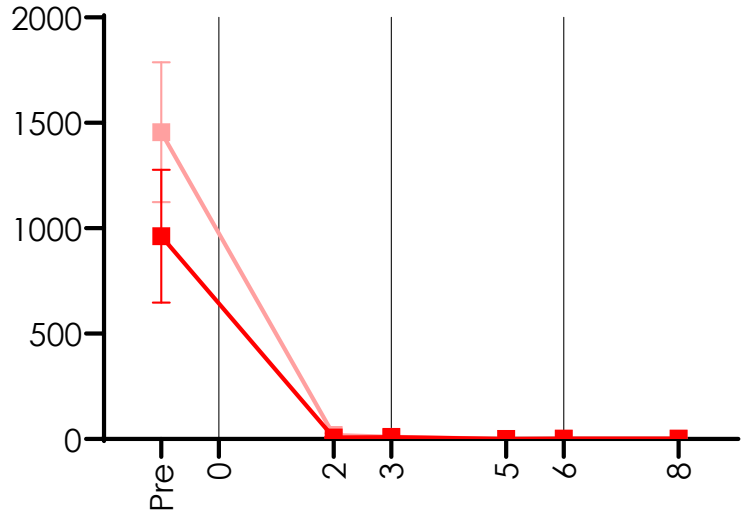
No pre-treatment before dosing

B cell depletion in blood

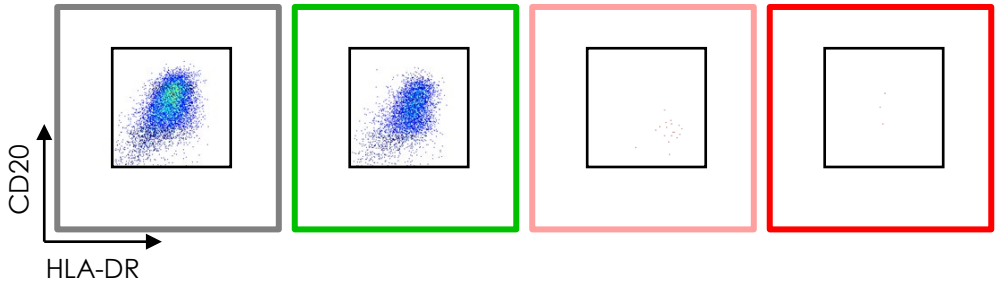
CD20$^+$ B cell fraction (% of pre-dose)



Total CD20$^+$ B Cells / µL Blood



48h post-1st dose



- Vehicle
- mGL/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (1 mg/kg)

B cell depletion observed in NHP tissues

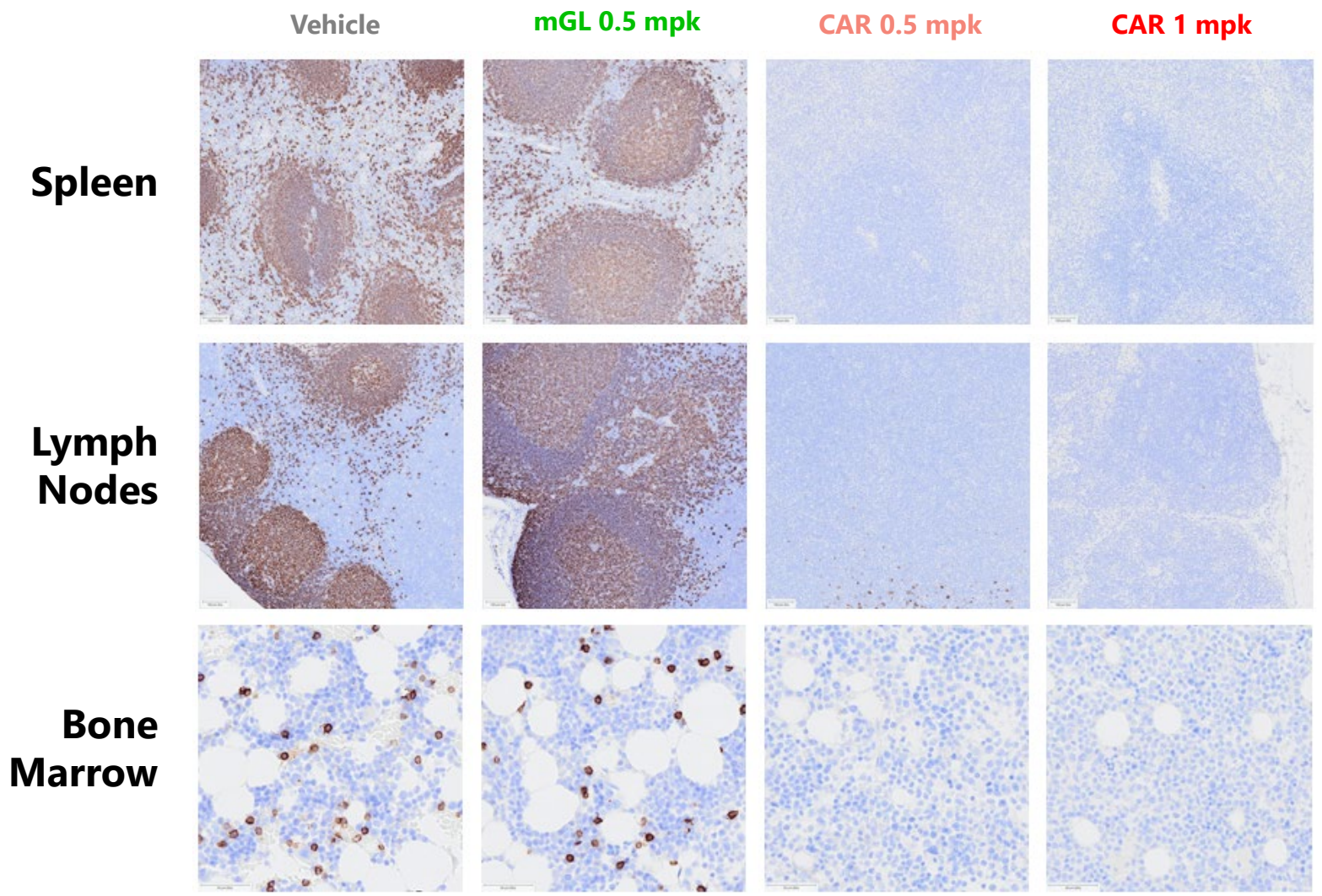
Cynomolgus macaques

CD20 CAR mRNA
LNP-A

0.5 or 1 mpk IV
Q3D×3
(D0, D3, D6)

No pre-treatment before dosing

Tissue analysis 48h post 3rd dose (D8)



CD20 CAR mRNA/LNP mediates acute CAR-related cytokine induction after first dose

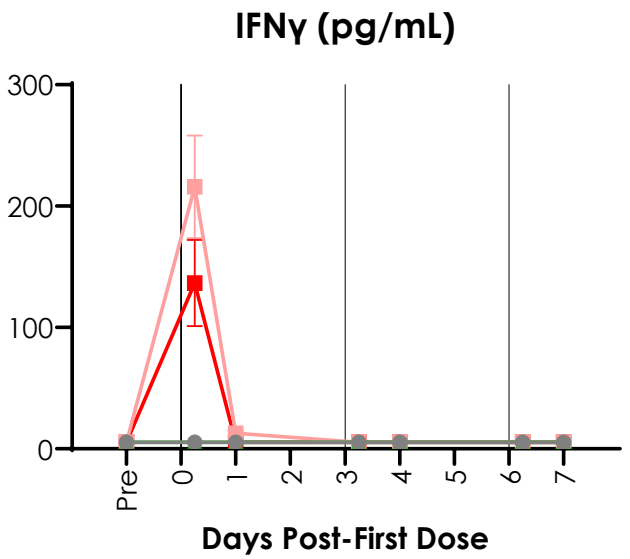
Cynomolgus macaques

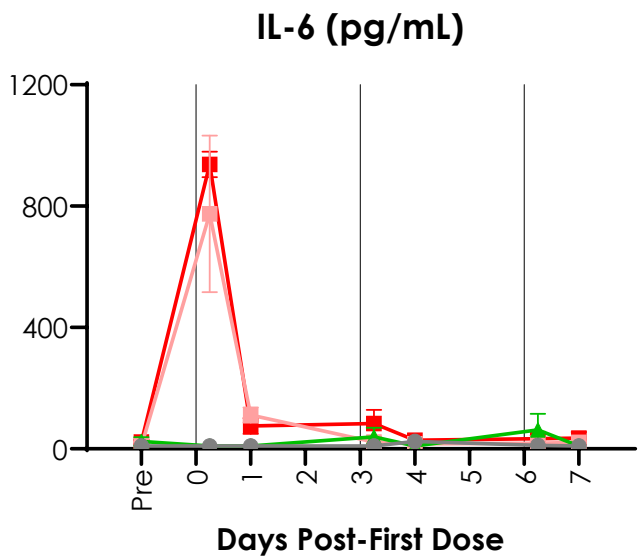
CD20 CAR mRNA LNP-A

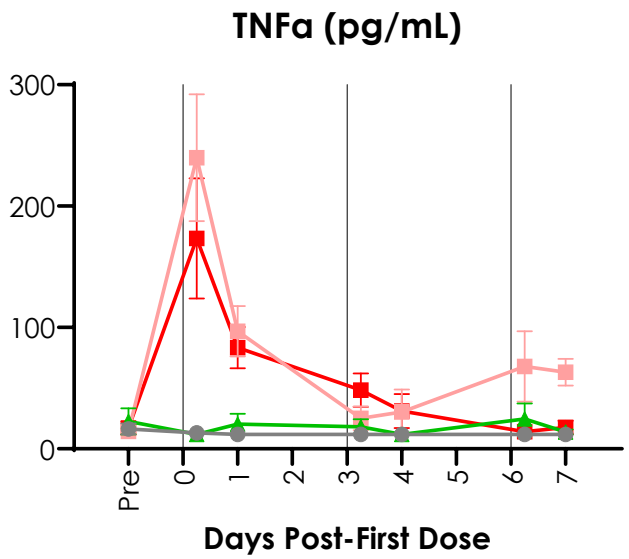
0.5 or 1 mpk IV Q3D×3 (D0, D3, D6)

No pre-treatment before dosing

Pro-inflammatory cytokines



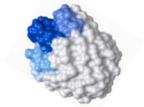
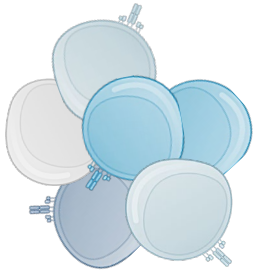




Response after first dose driven mainly by inflammatory and chemotactic cytokines
Response is predominantly acute and does not appear to accumulate across subsequent doses

- Vehicle
- mGL/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (0.5 mg/kg)
- CD20 CAR/LNP-A (1 mg/kg)

NHP data underestimates expected human pharmacology due to species-specific CD7 expression levels and VHH engagement

	VHH binding to CD7	Weak[1]	**Sub nM**[2]	*Strong human VHH CD7 binder not cross-reactive with cyno*
	T cell transfection by VHH-LNP	→	**18-fold**[3]	*Order of magnitude higher activity in human PBMCs compared to cyno*
	CD7⁺ of CD8⁺ T cells	~65%	**~100%**[4]	*Full addressability of CD8⁺ T cells via CD7 in human highly reduced in cyno*
	CD7⁺ of CD4⁺ T cells	<80%	**>90%**[4]	*Close to full addressability of CD4⁺ T cells via CD7 in human diminished in cyno*
	CD4⁺ / CD8⁺ of CD3⁺ T cells	CD8⁺ skewed	**CD4⁺ skewed**	*CD8⁺ smaller share of CD3⁺ in human compared to cyno*

1. Bivalent VHH-Fc 2. Monomeric VHH-His;; 3. CAR⁺ EC50 ; 4. In both healthy and SLE patients;

mRNA-6007 next steps

Completion of IND enabling studies

Health authority interactions

Platform technology

David Huss, PhD
Chief Technology Officer, Research

moderna

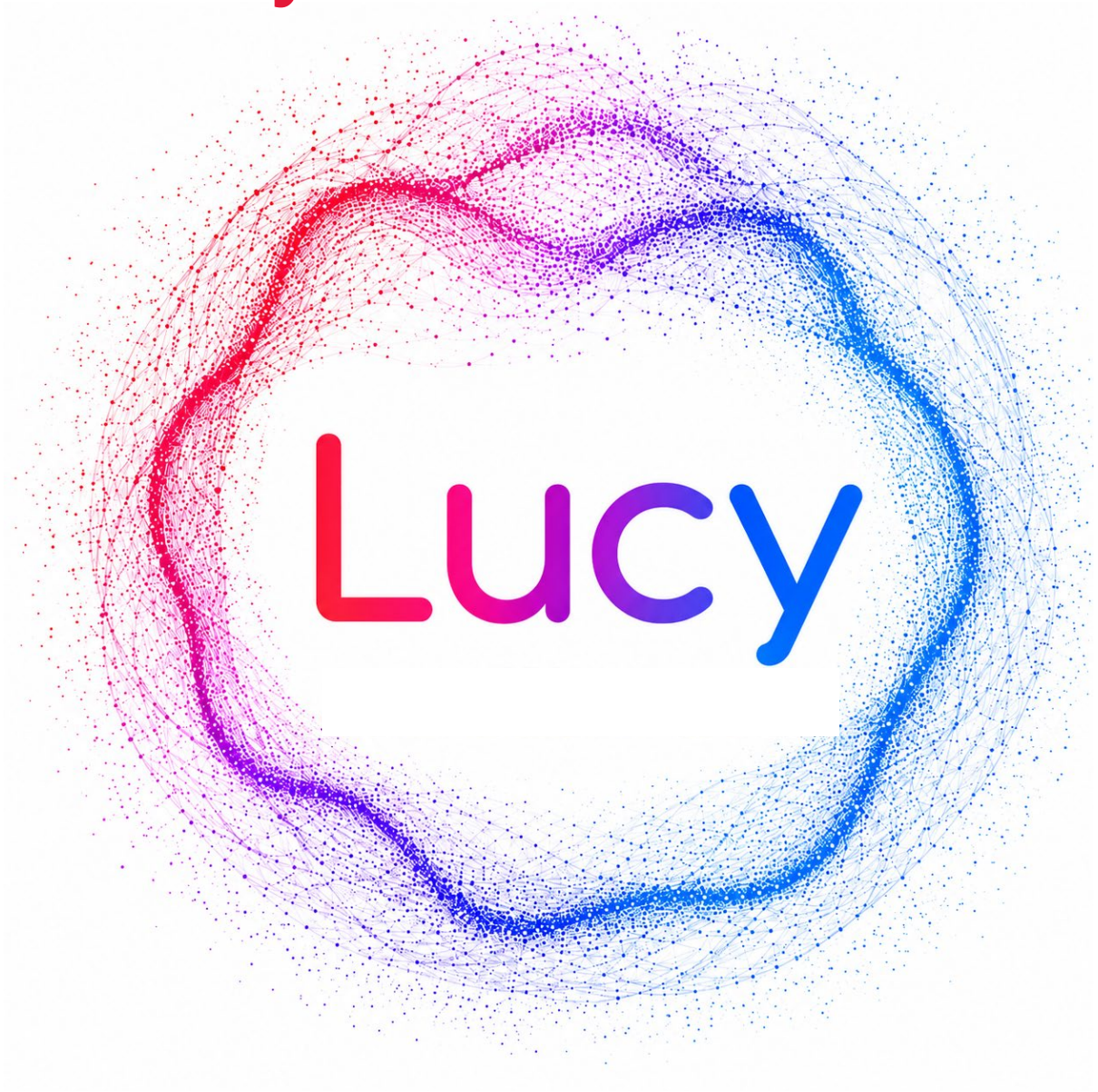
We are entering a new era of scientific exploration

AI and automation are eliminating the traditional limits on hypothesis generation and experimentation

Scientific advantage now belongs to those that **learn the fastest**

Learning at scale requires a fundamentally different experimentation and data infrastructure

Meet Lucy

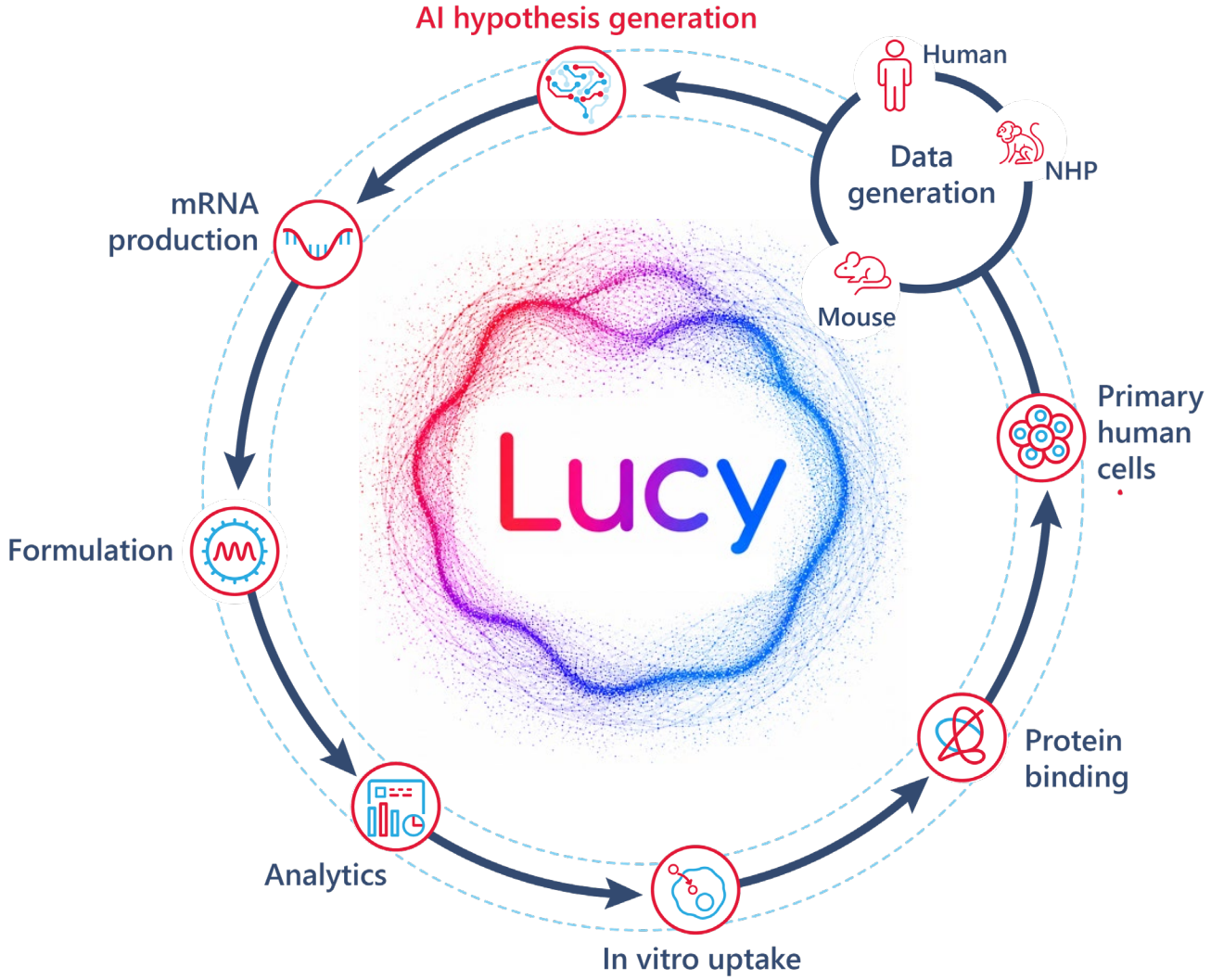


Lucy connects AI, automation, experimentation, and data into a **continuously improving learning system**

Every cycle makes the next discovery **faster**, **smarter**, and **more predictable**

Over time, **Lucy becomes a strategic advantage** that compounds across Moderna

Lucy and our digital-first automation platform create the ultimate scientific flywheel, transforming data into intelligence



Moderna's scientific intelligence engine incorporates Lucy and internal and external biomedical data, supercharged with AI tools

Moderna's **Scientific Intelligence Engine**

Vibe coding

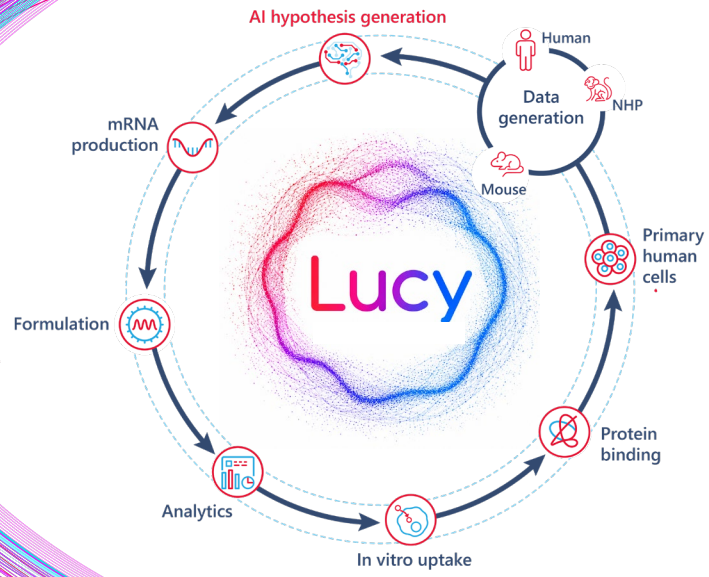
Open AI

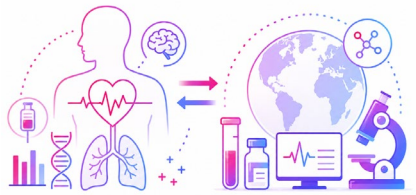
Emerging AI tools

Other LLMs

Machine learning

Quantum computing





Internal, proprietary, and publicly available biomedical data

moderna

Conclusion

Stéphane Bancel
Chief Executive Officer

moderna

Executing for near-term growth while fueling the next generation of mRNA medicines

Broaden our portfolio and global reach

Execute infectious disease vaccine launches, expand geographically, and establish new growth pillars in intismeran and PA

Focus mRED-led science in emerging and future modalities

Advance high-potential programs toward clinical proof-of-concept and first-in-human milestones, derisking novel modalities

Power innovation through our Scientific Intelligence Engine

Harness data, AI/ML, automation, and robotics to accelerate discovery and continuously improve how we work

Horizon 1
Established and de-risked modalities

Horizons 2 and 3 (mRED)
Emerging and future modalities

Scientific Intelligence Engine

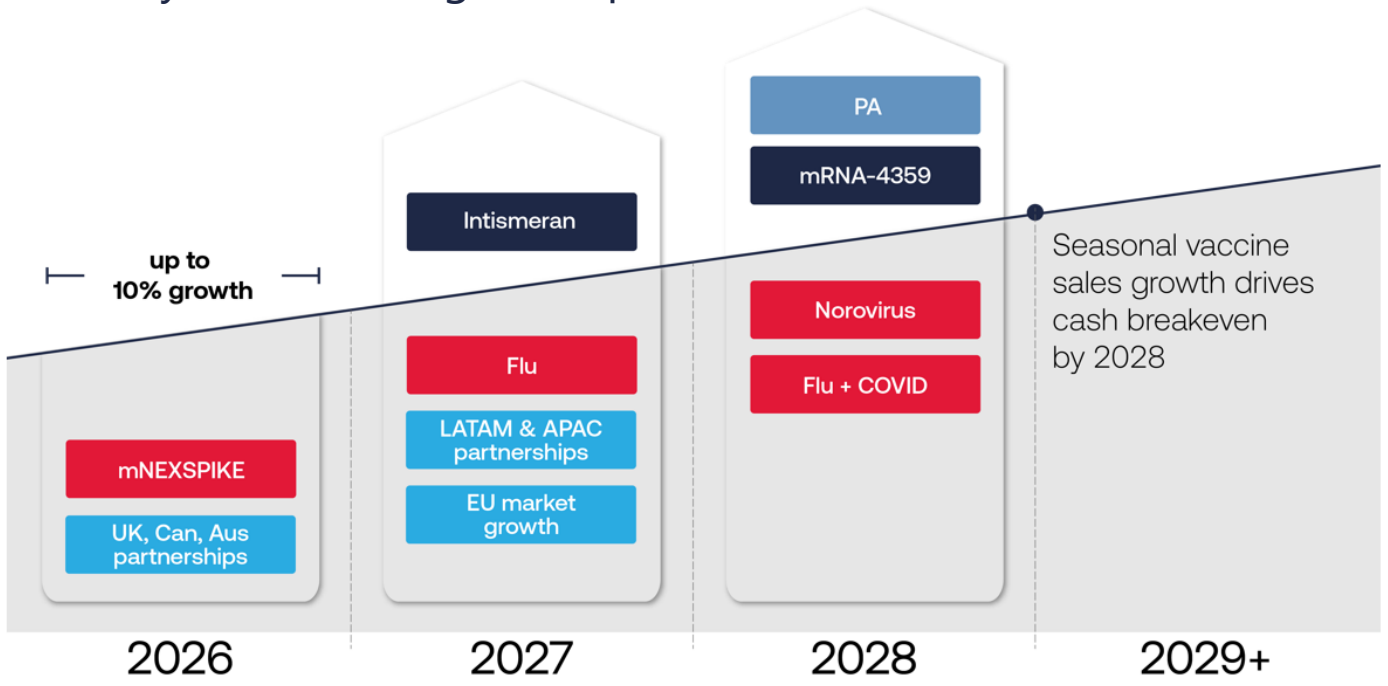
Executing for near-term growth while fueling the next generation of mRNA medicines

Broaden our portfolio and global reach

Execute infectious disease vaccine launches, expand geographically, and establish new growth pillars in intismeran and PA

Horizon 1
Established and de-risked modalities

Multi-year revenue growth plan



2026 value drivers

Commercial + financial performance

- **Up to 10% revenue growth** in 2026
- **2026 adjusted cash cost[1] target: $4.2B,** inclusive of mRED investments

Potential approvals

- **mNEXSPIKE** in ✅ EU, ✅ Japan, Switzerland, Taiwan
- **mCOMBRIAX** in ✅ EU, Canada, Australia
- **Flu** (mRNA-1010) in the U.S., Canada, Australia

Potential clinical milestones

- **Intismeran**
  - ✅ Five-year Phase 2 adjuvant melanoma data
  - Phase 3 adjuvant melanoma data; event-driven
  - Phase 2 adjuvant renal cell carcinoma data; event-driven
  - Phase 1 adjuvant pancreatic and peri-operative gastric data
- **mRNA-4359** Phase 2 data
- **Norovirus** Phase 3 data subject to case accruals
- **PA** registrational study data

Executing for near-term growth while fueling the next generation of mRNA medicines

Focus mRED-led science in emerging and future modalities

Advance high-potential programs toward clinical proof-of-concept and first-in-human milestones, derisking novel modalities

Horizons 2 and 3 (mRED)
Emerging and future modalities

mRNA-2151

Based on encouraging early clinical signals with mRNA-2808, **investment in mRNA-2151 ungated**

mRNA-2808

Sentinel program in Phase 1/2 clinical study and has passed stage gates

Modality:
T-cell engagers

Follow-on programs

Follow-on product concepts leveraging the same LNP include *in vivo* TCR/CAR-T for oncology (mRNA and gene insertion cargos) and T cell reprogramming

mRNA-6007

CAR-T with applicability across multiple autoimmune diseases involving B and plasma cells. Current Phase 1 focus on systemic lupus erythematosus (SLE) and other B cell mediated autoimmune diseases

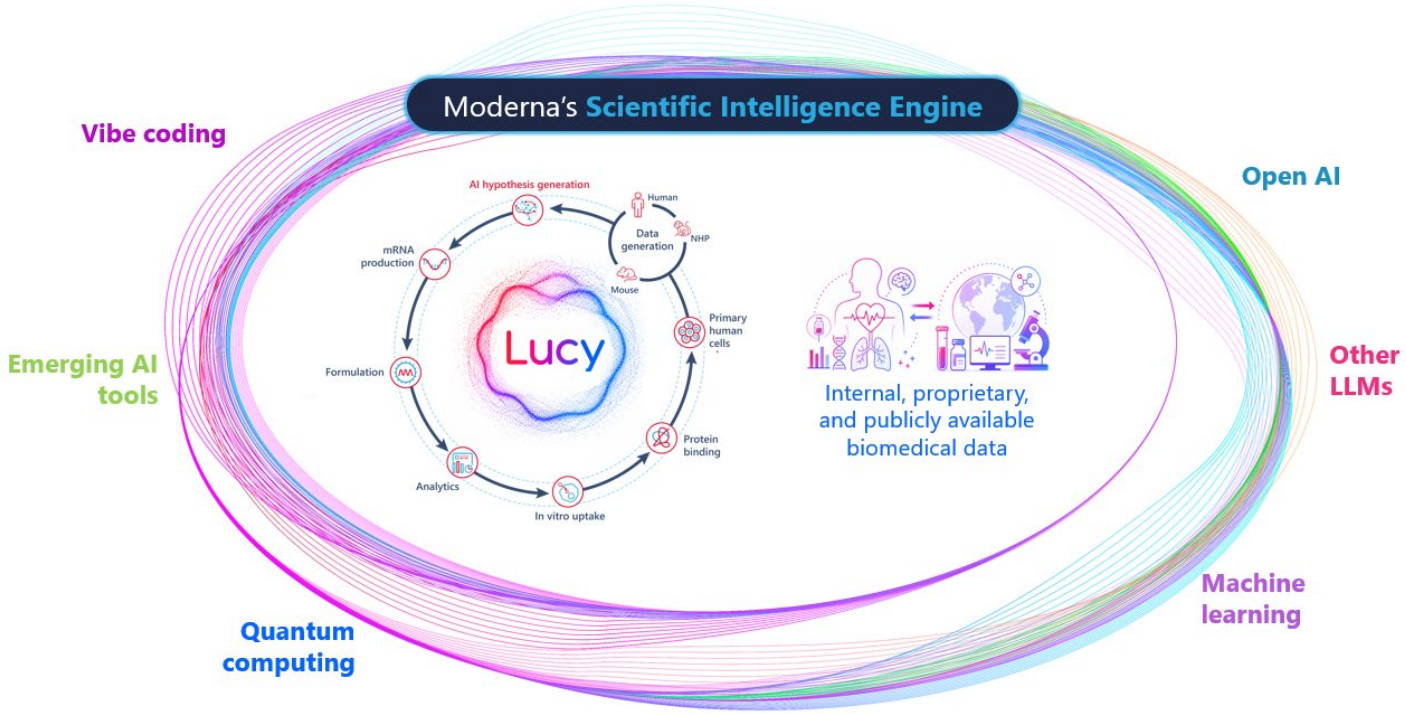
Modality:
***In vivo* CAR-T**

Executing for near-term growth while fueling the next generation of mRNA medicines

Power innovation through our Scientific Intelligence Engine

Harness data, AI/ML, automation, and robotics to accelerate discovery and continuously improve how we work

Scientific Intelligence Engine



Our mission

Deliver the greatest possible impact to **people** through mRNA **medicines**

moderna

Q & A