

October 26, 2026

ESMO Investor Event

Merck & Co., Inc., Rahway, N.J., USA

Forward-looking statement of Merck & Co., Inc., Rahway, N.J., USA

This presentation of Merck & Co., Inc., Rahway, N.J., USA (the "company") includes "forward-looking statements" within the meaning of the safe harbor provisions of the U.S. Private Securities Litigation Reform Act of 1995. These statements are based upon the current beliefs and expectations of the company's management and are subject to significant risks and uncertainties. There can be no guarantees with respect to pipeline candidates that the candidates will receive the necessary regulatory approvals or that they will prove to be commercially successful. If underlying assumptions prove inaccurate or risks or uncertainties materialize, actual results may differ materially from those set forth in the forward-looking statements.

Risks and uncertainties include but are not limited to, general industry conditions and competition; general economic factors, including interest rate and currency exchange rate fluctuations; the impact of pharmaceutical industry regulation and health care legislation in the United States and internationally; global trends toward health care cost containment; technological advances, new products and patents attained by competitors; challenges inherent in new product development, including obtaining regulatory approval; the company's ability to accurately predict future market conditions; manufacturing difficulties or delays; financial instability of international economies and sovereign risk; dependence on the effectiveness of the company's patents and other protections for innovative products; and the exposure to litigation, including patent litigation, and/or regulatory actions.

The company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise. Additional factors that could cause results to differ materially from those described in the forward-looking statements can be found in the company's Annual Report on Form 10-K for the year ended December 31, 2025 and the company's other filings with the Securities and Exchange Commission (SEC) available at the SEC's Internet site (www.sec.gov).

Agenda

Opening Remarks | Dr. Eliav Barr

ESMO Data Highlights | Dr. Marjorie Green

Closing Remarks | Dr. Eliav Barr

Q&A | All

Opening Remarks

Dr. Eliav Barr
Senior Vice President, Head of Global Clinical Development & Chief Medical Officer



Sustaining durable leadership in oncology through two strategic objectives outlined in 2021

MAXIMIZE

impact and move into earlier stage disease including with KEYTRUDA QLEX

DIVERSIFY

pipeline with novel mechanisms

LEVERAGE

KEYTRUDA as foundation to advance standard of care

Uniquely positioned to advance cancer care with differentiated portfolio and pipeline

Immuno-oncology

Boost anti-tumor immune responses

KEYTRUDA® (pembrolizumab) Injection 100 mg

KEYTRUDA Qlex™ pembrolizumab + berahyaluronidase alfa-pmph Subcutaneous Injection | 165 mg + 2,000 units/mL

MK-2010
Anti-PD1 x VEGF bispecific

intismeran autogene[1]
(V940)
Individualized Neoantigen Therapy

Precision Molecular Targeting

Impact pathways that can drive cancer growth

Lynparza™ olaparib[2]

WELIREG™ (belzutifan) 40 mg tablets

LENVIMA® (lenvatinib) capsules | 10 mg and 4 mg | RESULTS THAT MATTER[3]

nemtabrutinib
(MK-1026)
BTKi

bomedemstat
(MK-3543)
LSD1i

MK-4208
(formerly TERN-701)
Allosteric TKI

opevesostat
(MK-5684)
CYP11A1i

calderasib
(MK-1084)
KRAS G12Ci

MK-2690
(formerly SPR2015)
KRAS G12Di

MK-4716
KRAS

Tissue Targeting

Increase cancer cell sensitivity with ADCs and immune cell engagers

sacituzumab tirumotecan
(MK-2870)
TROP2 ADC

zilovertamab vedotin
(MK-2140)
ROR1 ADC

MK-1045
CD19xCD3 TCE

MK-2750[4]
ADC – undisclosed target

MK-8294[5]
TCE – undisclosed target

Undisclosed preclinical ADCs

I-DXd[4]
(MK-2400)
B7-H3 ADC

R-DXd[4]
(MK-5909)
CDH6 ADC

HER3-DXd[4]
(MK-1022)
HER3 ADC

gocatamig[4]
(MK-6070)
DLL3 TCE

MK-6255
TCE – undisclosed target

1. Collaboration with Moderna 2. Collaboration with AstraZeneca 3. Collaboration with Eisai 4. Collaboration with Daiichi Sankyo 5. Collaboration with Janux Therapeutics

Strategy to maximize benefit for patients with unambiguous efficacy, informed combinations, biomarker enrichment, curative intent settings



Combinable agents

Best outcomes achieved when non-cross resistant therapies are combined or given sequentially

Develop medicines with single agent activity which can be strategically combined with other agents



Biomarkers

Predictive biomarkers enable stratification that can improve therapeutic index of medications

Improve therapeutic index through precision medicine



Earlier-stage disease

Early treatment improves probability for better outcomes

Develop medicines in earlier stages of disease

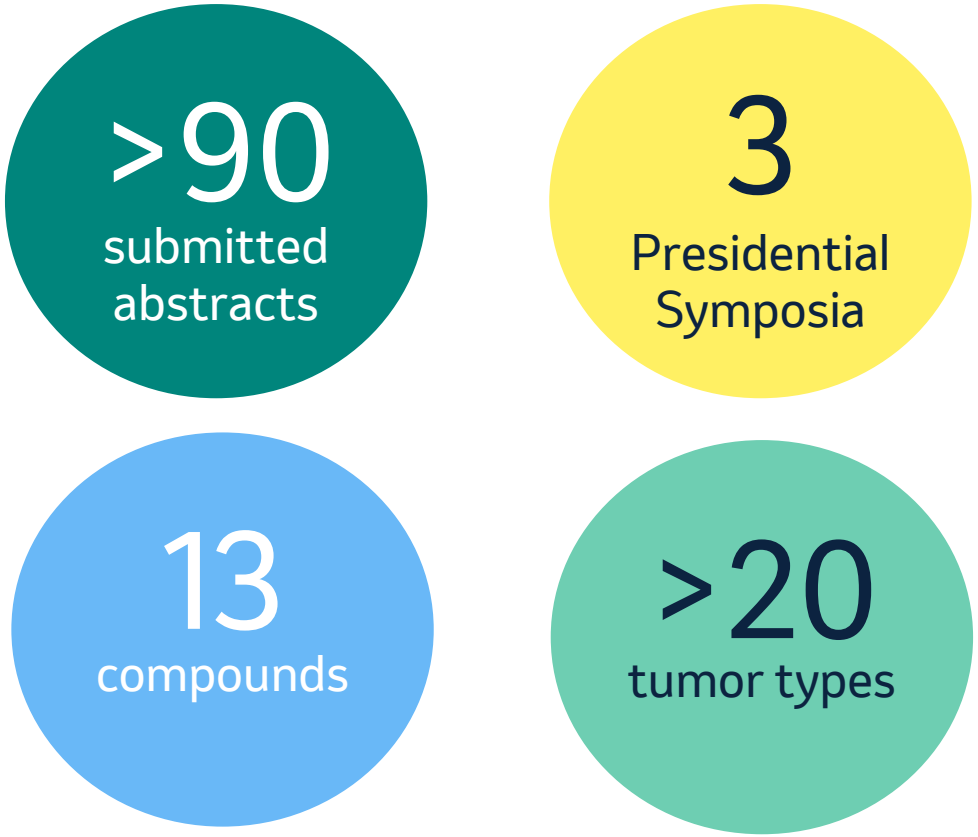
ESMO Data Highlights

Dr. Marjorie Green
Senior Vice President, Head of Oncology
Global Clinical Development



Potentially practice changing data across broad oncology pipeline at ESMO

ESMO at a glance



Presidential Symposia

Intismeran autogene[1]

- **INTerpath-001** (adjuvant stage IIB-IV melanoma)

Sac-TMT

- **TroFuse-005** (post anti PD-1/L1 and platinum chemotherapy endometrial cancer)

KEYTRUDA

- **KEYNOTE-C93** (1L dMMR advanced or recurrent endometrial cancer)

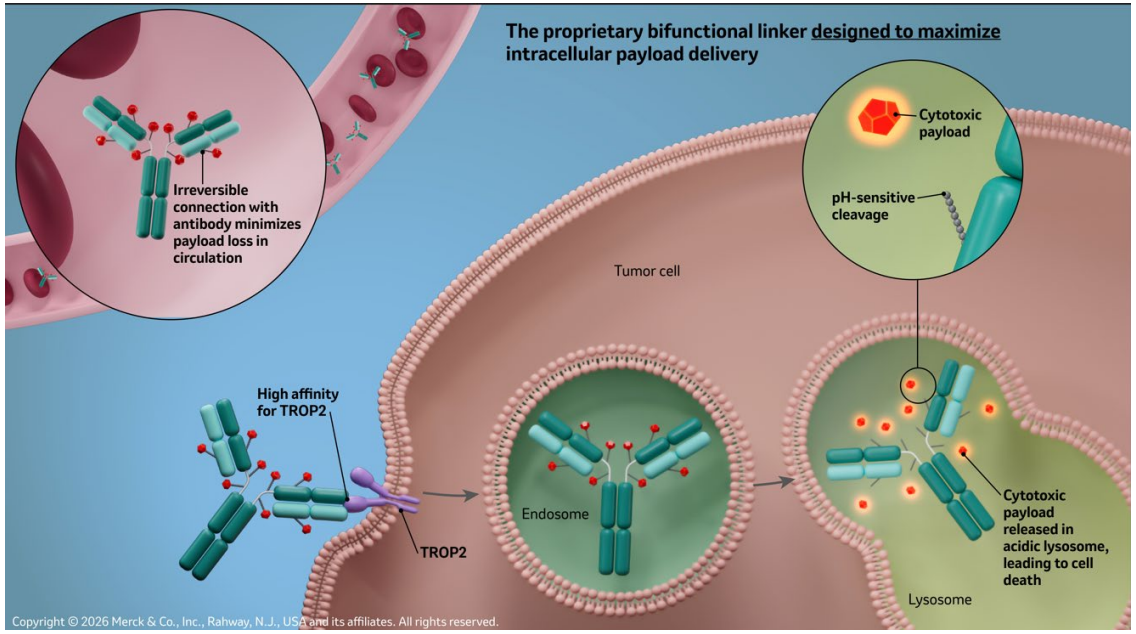
1. Collaboration with Moderna

Sac-TMT designed as cornerstone ADC with potential to deliver best-in-class profile across multiple tumors

Sac-TMT

- **TROP2-directed ADC** with **belotecan-derived topoisomerase I inhibitor payload**
- **Unique bifunctional linker** designed to **maximize payload delivery** to tumor cells
  - **Irreversible connection to antibody** reduces free payload in circulation
  - **pH sensitive connection** ensures intracellular payload release in lysosomes

Mechanism of Action

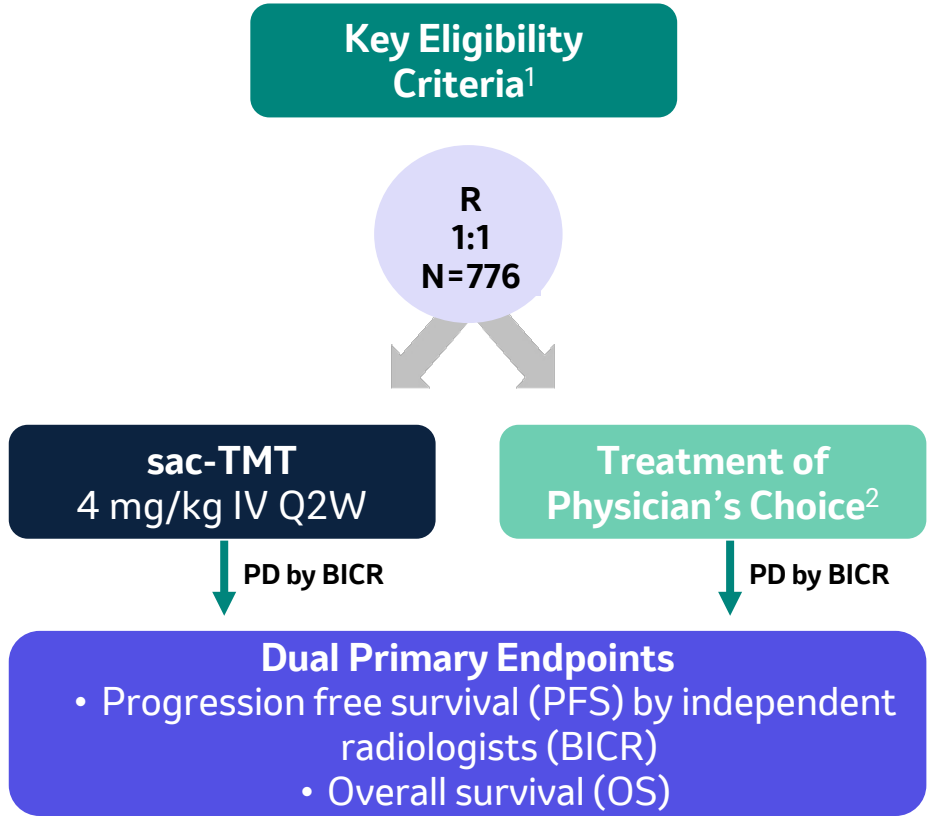


TroFuse-005 aims to address significant remaining unmet need in endometrial cancer

Endometrial cancer

- **6th most common cancer in women with increasing incidence and mortality**
- Persistent **racial disparities** with **>2x mortality rate for black women**
- Following primary treatment, **patients face risk of cancer returning**, often as distant metastasis with poor outcomes
- Different **subtypes of endometrial cancer** exist (e.g. carcinomas, carcinosarcomas, serous adenocarcinoma)
- **Biomarkers** utilized to **predict benefit of therapy**
  - Mismatch repair (dMMR or pMMR); HER2
- Limited treatment options after 1L therapy for advanced disease with **OS usually < 1 year**

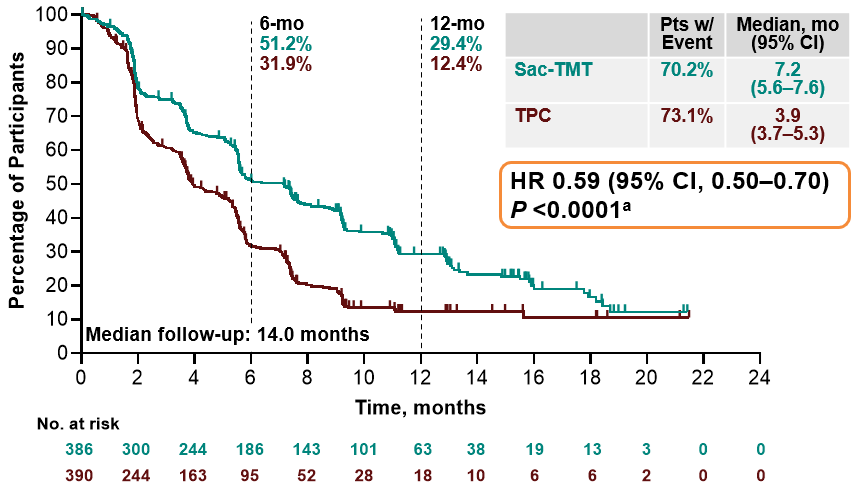
TroFuse-005 Study Design



1. Histologically-confirmed diagnosis of endometrial carcinoma or carcinosarcoma; radiologically apparent measurable or non-measurable disease; prior platinum exposure and prior anti-PD-1/PD-L1 exposure (given separately or in combination), in any setting, including neoadjuvant or adjuvant therapy; recurrence <12 months after completing platinum-based adjuvant therapy, or received platinum in the metastatic setting, or received an IO-based regimen in the recurrent setting after initial adjuvant platinum therapy regardless of platinum-free interval; does not require recurrent drainage of effusions within 6 weeks before randomization 2. Doxorubicin 60 mg/m2 IV Q3W OR Paclitaxel 80 mg/m2 IV on Days 1, 8 and 15 of each 28-day cycle

TroFuse-005 is the first positive global Phase 3 study for sac-TMT from expansive TroFuse clinical development program

PFS by BICR

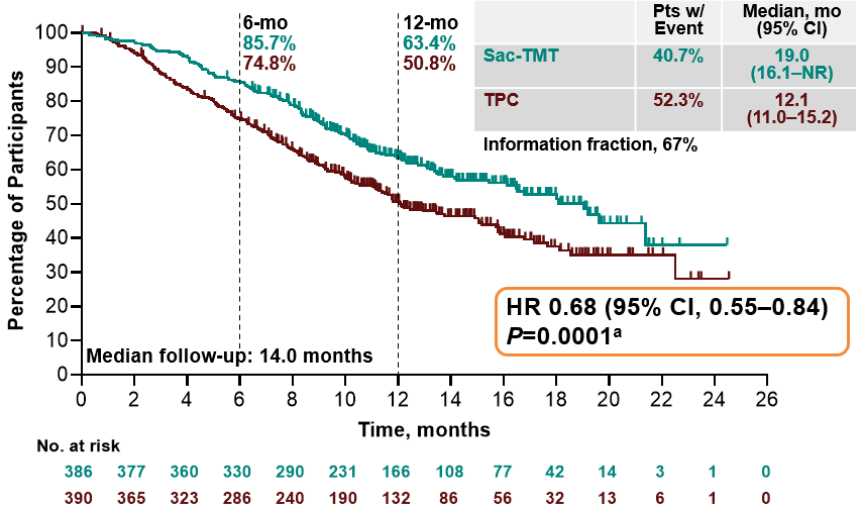


	Pts w/ Event	Median, mo (95% CI)
Sac-TMT	70.2%	7.2 (5.6–7.6)
TPC	73.1%	3.9 (3.7–5.3)

HR 0.59 (95% CI, 0.50–0.70)
P <0.0001[a]

Response assessed per RECIST v1.1 by BICR. aThe observed 1-sided log-rank test p-value met the prespecified boundary for significance (P=0.0025) at this interim analysis. Data cutoff date: March 23, 2026.

OS at IA



	Pts w/ Event	Median, mo (95% CI)
Sac-TMT	40.7%	19.0 (16.1–NR)
TPC	52.3%	12.1 (11.0–15.2)

Information fraction, 67%

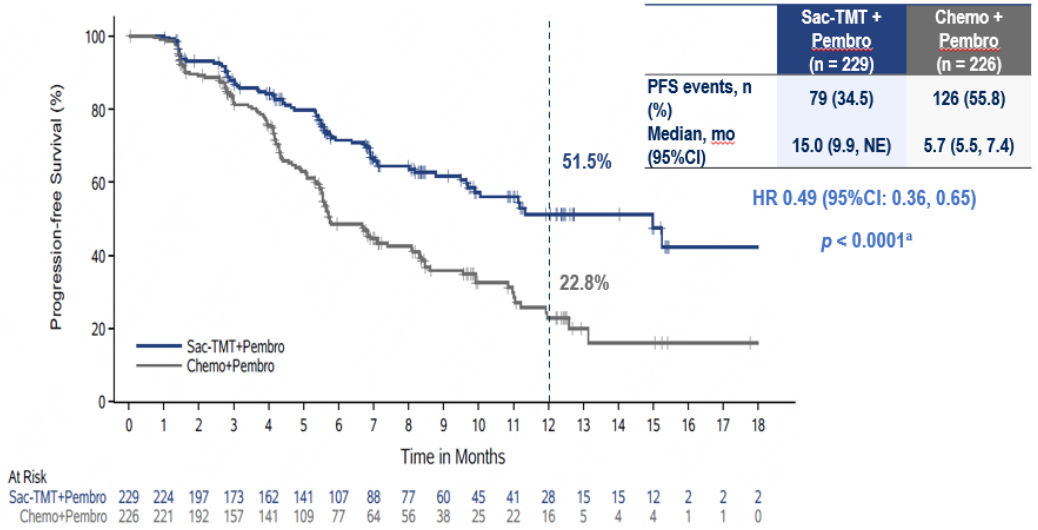
HR 0.68 (95% CI, 0.55–0.84)
P=0.0001[a]

aThe observed 1- sided log-rank test p-value crossed the prespecified significance boundary (P=0.00636) at this interim analysis. At this time, subsequent anticancer therapy was received by 35% of participants in the sac-TMT group and 49% in the TPC group; of those, 4.9% and 12.1%, respectively, had received ADCs. Data cutoff date: March 23, 2026.

- Demonstrated **statistically significant and clinically meaningful improvement** in **OS** and **PFS** in patients with advanced or recurrent endometrial cancer who received platinum-based chemotherapy and anti-PD-1/L1 immunotherapy vs TPC
  - Extended median OS by **6.9 months** to **19 months**
  - **Deep responses** and **prolonged DOR**, indicating a favorable durability and disease control
- Safety profile **consistent** with previously reported studies and **no new signals observed**
- **First and only** global Phase 3 study of a **TROP2 ADC** to improve outcomes for certain patients with **endometrial cancer**

Results from OptiTROP-Lung06[1] strengthens evidence for sac-TMT in 1L NSCLC

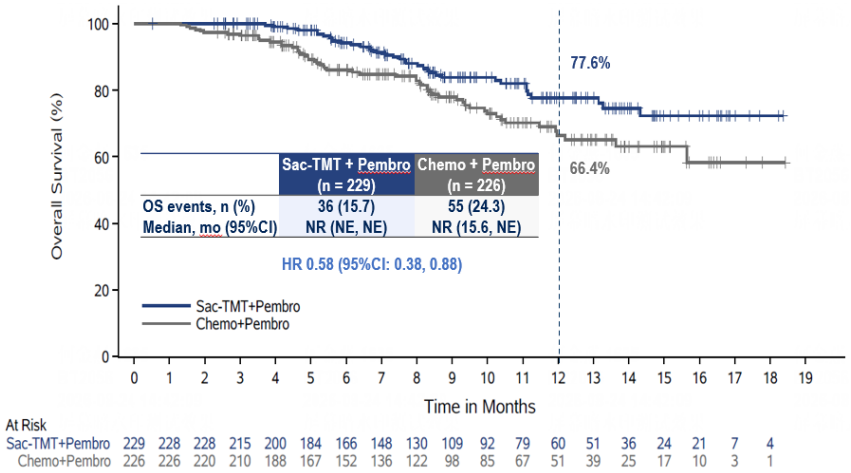
PFS by BICR



	Sac-TMT + Pembro (n = 229)	Chemo + Pembro (n = 226)
PFS events, n (%)	79 (34.5)	126 (55.8)
Median, mo (95%CI)	15.0 (9.9, NE)	5.7 (5.5, 7.4)

a Updated efficacy boundary (corresponding to 205 actual PFS events by BICR): 0.0160 (2-sided). NE, not estimable.

OS at IA



	Sac-TMT + Pembro (n = 229)	Chemo + Pembro (n = 226)
OS events, n (%)	36 (15.7)	55 (24.3)
Median, mo (95%CI)	NR (NE, NE)	NR (15.6, NE)

Median follow-up was 9.7 months

1. Study conducted in China 2. Measured by ≥50% SLD reduction

- Demonstrated **statistically significant and clinically meaningful improvement** in **PFS** as first line treatment for certain patients with nonsquamous PD-L1-negative NSCLC
- **Deeper and longer duration of response**
  - Deep response rate[2] of **28.8** vs 17.3
  - Median DOR of **13.6 months** vs 6.8 months
- **Favorable trend in OS**, although data remain immature
- **First positive Phase 3 study** of **sac-TMT plus KEYTRUDA** vs KEYTRUDA plus chemotherapy
- Planning underway for **global Phase 3 studies** evaluating **sac-TMT in combination with either KEYTRUDA** or **MK-2010** in 1L NSCLC

Differentiated Phase 3 TroFuse development program with first mover opportunities to address unmet needs across common tumor types

TroFuse ONCOLOGY CLINICAL TRIALS

	Gynecologic			Breast		NSCLC		GI	GU
	Endometrial	Ovarian	Cervical	HR+	TNBC	EGFRwt	EGFRm	Gastric	Bladder
Earlier Stage					TroFuse-032 TroFuse-012	TroFuse-019			
1L Metastatic	TroFuse-033 ★	TroFuse-021 ★	TroFuse-036 ★	TroFuse-010	TroFuse-011	TroFuse-007 TroFuse-023 ★			
2L+ Metastatic	TroFuse-005 ☑	TroFuse-022 ★	TroFuse-020				TroFuse-004 TroFuse-009	TroFuse-015	TroFuse-031 TroFuse-055

Planning for additional Phase 3 studies of sac-TMT monotherapy and combination studies underway

First mover opportunity | Differentiated profile | Enrollment complete | ★ Maintenance regimen | ☑ Positive topline results

Note: Biomarker strategy in place for multiple studies

Uniquely positioned to explore promising science of PD-1xVEGF with MK-2010

Development considerations

- **Advantaged company** to fully explore potential of PD-1xVEGF axis
  - **Leverage PD-1 and VEGF experience** to inform clinical development program
  - **Portfolio of novel agents** offer combination opportunities
- Initiated **Phase 2 studies in combination** with other agents (e.g. sac-TMT, R-DXd, WELIREG, chemo)
  - Will help inform **signal and dose finding** of combination across solid tumors
- **Productive discussions** with the FDA, anticipate **Phase 3** combination studies with sac-TMT in **1H 2027**

MK-2010 combination studies underway

Study/protocol #	MK-2010-003	MK-2010-002	REJOICE-Ovarian02
Combination partner	Sac-TMT	WELIREG, chemotherapy	R-DXd
Tumor area	Solid tumors	Solid tumors	Ovarian
Development phase	Phase 2	Phase 2	Phase 1/2

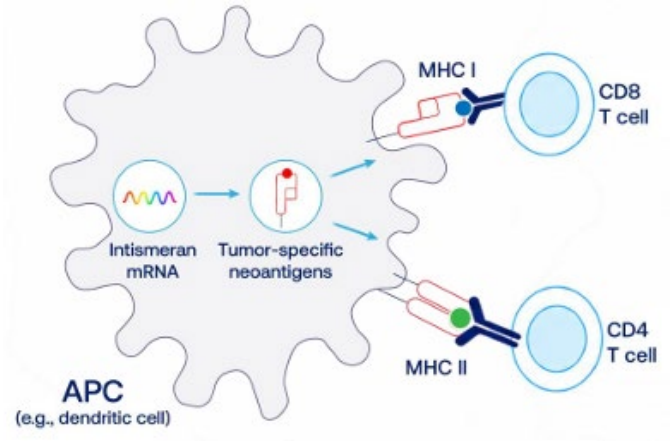
Intismeran autogene is an mRNA-based INT, bringing a new era of personalized cancer research

Intismeran

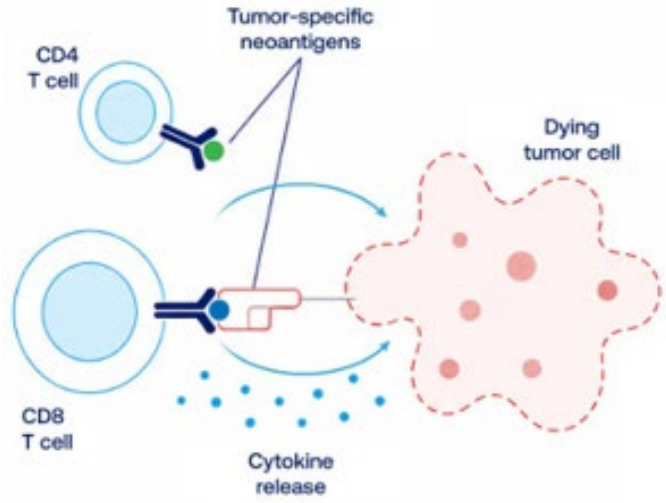
- Investigational **lipid encapsulated mRNA** based individualized neoantigen therapy
- Consists of an mRNA that **encodes neoantigens** designed specifically to each **individual patient's tumor mutanome** and **HLA type**
- Leverages **deterministic machine learning algorithm** to induce de novo and endogenous **neoantigen specific anti-tumor T-cell responses** (both class I (CD8) and class II (CD4))

Mechanism of action

T-Cell Activation



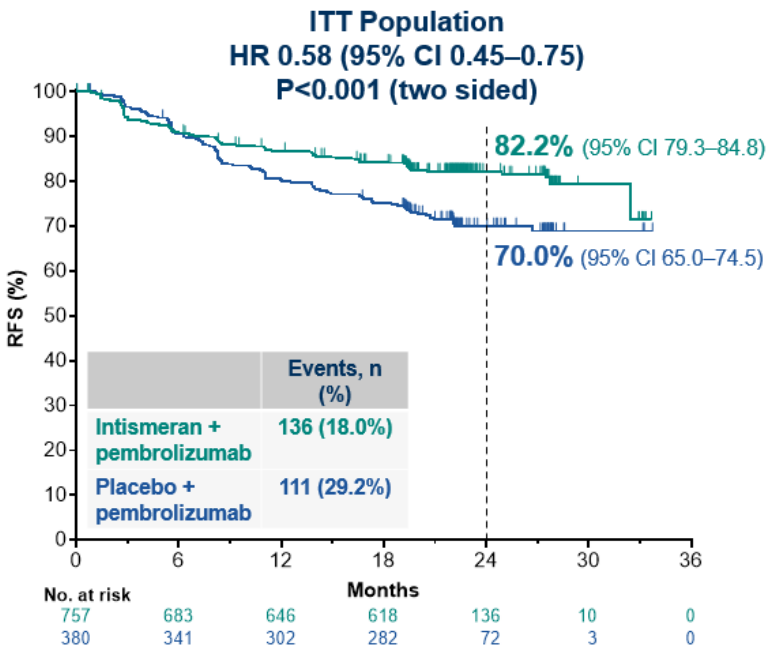
Tumor Destruction



In collaboration

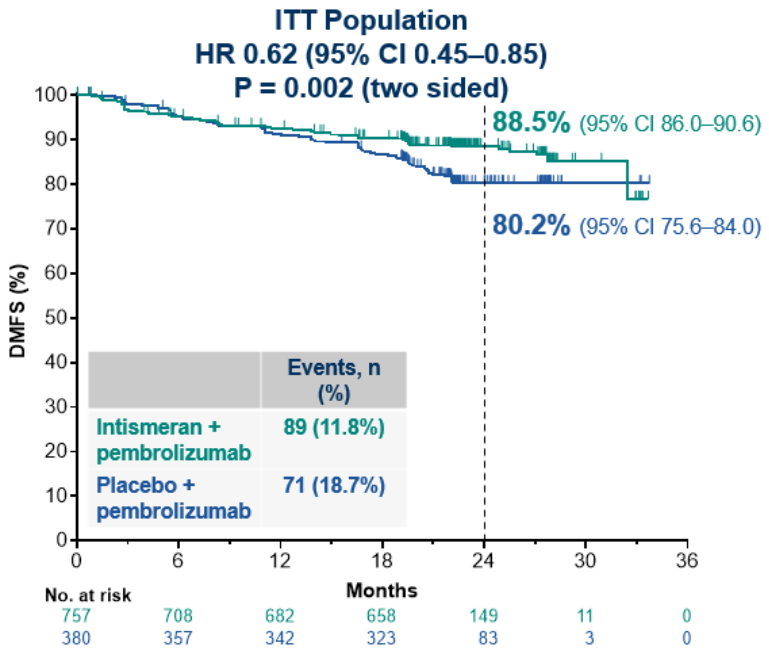
INTerpath001 is the first positive Phase 3 study for an mRNA-based individualized neoantigen therapy

RFS



RFS: time from randomization to the first occurrence of any recurrence or death from any cause. Data cutoff date: June 30, 2026 (median study follow-up, 24.6 mo)

DMFS



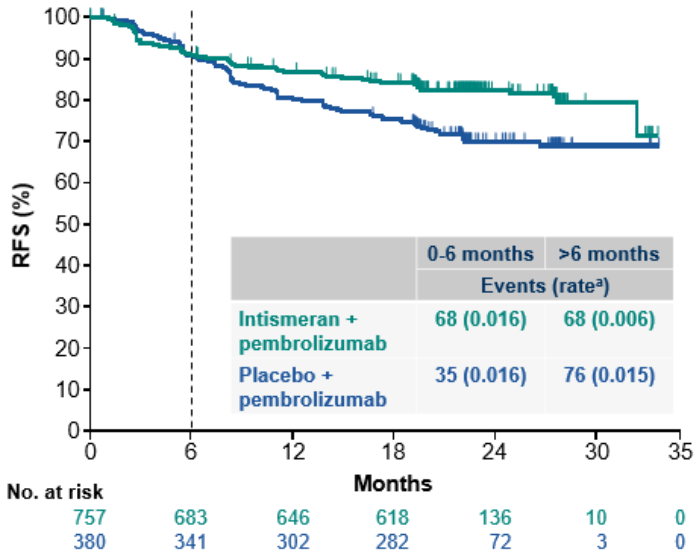
DMFS: time from randomization to first occurrence of distant metastasis or death from any cause. Data cutoff date: June 30, 2026 (median study follow-up, 24.6 mo)

- Demonstrated **statistically significant** and **clinically meaningful** improvement in **RFS** and **DMFS** as adjuvant treatment in certain **patients with completely resected stage IIB-IV melanoma**
- **Favorable trend in OS**, although data remain immature
- Safety was **generally consistent** with previous experience, **no new signals observed**
- **First Phase 3 study** to demonstrate **clinically meaningful improvement** in RFS and DMFS **over KEYTRUDA** in adjuvant treatment of resected melanoma
- Building on evidence from KN-942, results **strengthen confidence in curative potential** of intismeran when given in combination with KEYTRUDA for these patients

Pre-specified piecewise sensitivity analysis shows benefit was greater after six months

RFS

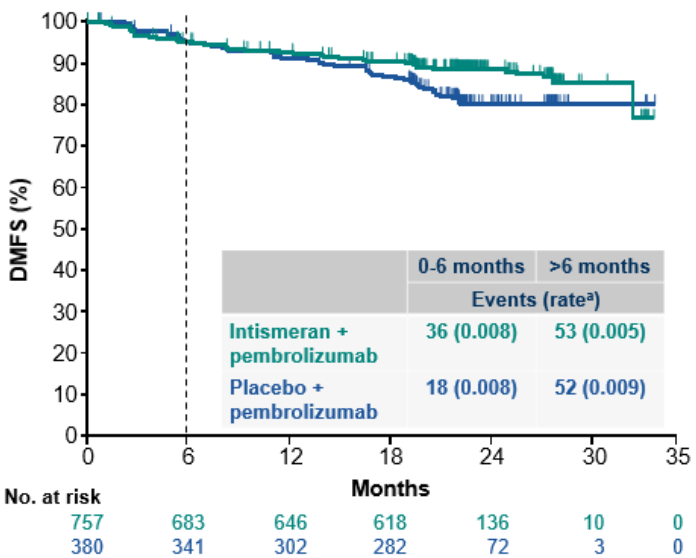
0-6 months: HR 0.99 (95% CI 0.66–1.49)
>6 months: HR 0.42 (95% CI 0.30–0.58)



	0-6 months	>6 months
	Events (rate[a])	
Intismeran + pembrolizumab	68 (0.016)	68 (0.006)
Placebo + pembrolizumab	35 (0.016)	76 (0.015)

DMFS

0-6 months: HR 1.01 (95% CI 0.58–1.79)
>6 months: HR 0.50 (95% CI 0.34–0.73)



	0-6 months	>6 months
	Events (rate[a])	
Intismeran + pembrolizumab	36 (0.008)	53 (0.005)
Placebo + pembrolizumab	18 (0.008)	52 (0.009)

- **Delayed separation of RFS and DMFS**
- RFS and DMFS benefit **greater after six months**
- **Intismeran** designed to **help overcome tumor-mediated immune suppression** by generating neoantigen-specific T cells, while **KEYTRUDA releases PD-1-mediated inhibition**

aRate calculated as no. of events divided by person-time in each interval. ITT: all randomized patients according to the arm to which they were assigned.
Data cutoff date: June 30, 2026 (median study follow-up, 24.6 mo).

Advancing a robust late-phase development plan for intismeran

Phase 1	Phase 2	Phase 3	Status
INTerpath-001 (adjuvant melanoma)			POSITIVE DATA ★
INTerpath-002 (adjuvant stage II-IIIB NSCLC)			COMPLETED ENROLLMENT
INTerpath-009 (perioperative stage II-IIIB NSCLC)			RECRUITING
INTerpath-014 (adjuvant stage I NSCLC)			RECRUITING
INTerpath-005 (adjuvant MIBC)			COMPLETED ENROLLMENT
INTerpath-004 (adjuvant RCC)			COMPLETED ENROLLMENT
INTerpath-011 (high-risk NMIBC)			RECRUITING
INTerpath-012 (1L melanoma)			RECRUITING
INTerpath-013 (1L squamous NSCLC)			RECRUITING

Earlier-stage indication

Metastatic indication

★ Data presented at ESMO 2026

Closing Remarks

Dr. Eliav Barr
Senior Vice President, Head of Global Clinical Development & Chief Medical Officer



Two potential important approvals next year



Sac-TMT

FDA accepted for priority review PDUFA June 15th

BLA for patients with advanced or recurrent endometrial cancer who have received prior platinum-based chemotherapy and anti-PD-1/L1 immunotherapy

Intismeran autogene[1]

FDA Breakthrough Therapy Designation

With KEYTRUDA for adjuvant treatment of stage IIB-IV high-risk melanoma following complete resection

1. In collaboration

Approaching substantial registration-enabling opportunity set across diversified oncology pipeline

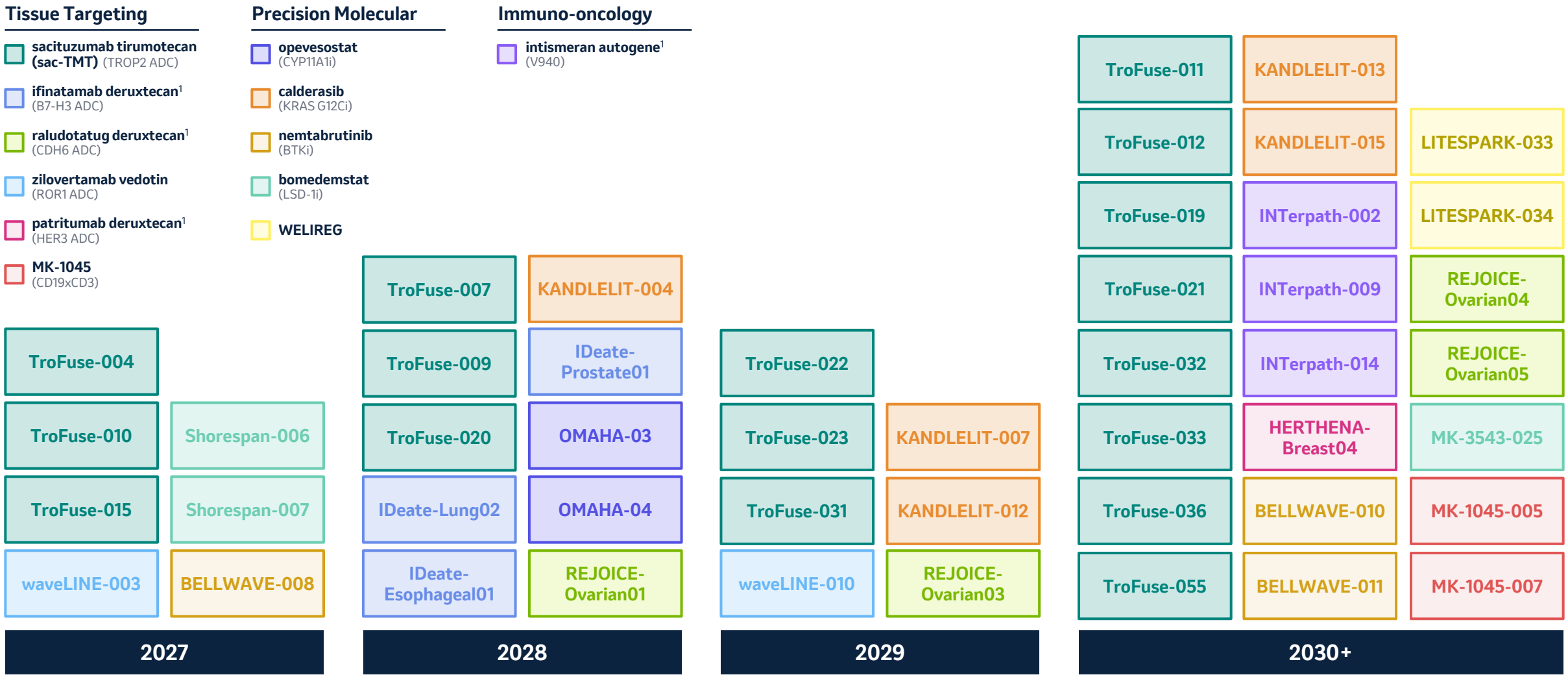
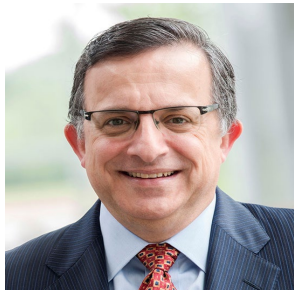


Chart reflects Phase 2/3 or Phase 3 primary completion dates on clinicaltrials.gov as of October 15, 2026 1. In collaboration

Q&A



Dr. Eliav Barr
SVP, Head of Global Clinical Development & Chief Medical Officer



Dr. Marjorie Green
SVP, Head of Oncology Global Clinical Development



Peter Dannenbaum
SVP, Investor Relations



Appendix

Acronyms

1L = First-Line
ADC = Antibody-drug conjugate
B7-H3 = B7 homolog 3
BICR = Blinded independent central review
BLA = Biologics license application
BTKi = Bruton Tyrosine Kinase inhibitor
CD = Cluster of Differentiation
CDH6 = Cadherin-6
CI = Confidence interval
CYP11A1i = Cytochrome P450 Family 11 Subfamily A Member 1 inhibitor
DLL3 = Delta-like ligand 3
DOR =Duration of Response
DMFS = Distant Metastasis-Free Survival
dMMR = Mismatch Repair Deficient
EC = Endometrial cancer
EGFR = Epidermal growth factor receptor
EGFRm = Epidermal growth factor receptor mutant
EGFRwt = Epidermal growth factor receptor wild type
GI = Gastrointestinal
GU = Genitourinary
HER2 = Human epidermal growth factor receptor 2
HER3 = Human epidermal growth factor receptor 3
HLA = Human leukocyte antigen
HR = Hazard ratio
HR+ = Hormone receptor-positive
IA = Interim analysis
INT = Individualized neoantigen therapy
IO = Immuno-oncology
ITT = Intent-to-treat
IV = Intravenous
KRAS = Kirsten rat sarcoma viral oncogene homolog
LA = Locally Advanced
LSD1i = Lysine-Specific Demethylase 1 inhibitor
MHC = Major Histocompatibility Complex
MIBC = Muscle-invasive bladder cancer
MOA = Mechanism of action
mOS = Median Overall Survival
mRNA = Messenger RNA
NMIBC = Non-Muscle Invasive Bladder Cancer
NR = Not reached
NSCLC = Non-small cell lung cancer
OS = Overall survival
PD-1 = Programmed cell death protein 1
PD-L1 = Programmed cell death ligand 1
PFS = Progression free survival
pMMR = Proficient Mismatch Repair
Q2W = Every two weeks
RCC = Renal cell carcinoma
R-DXd = Raludotatug Deruxtecan
RFS = Recurrence free survival
ROR1 = Receptor tyrosine kinase-like orphan receptor 1
sac-TMT = Sacituzumab Tirumotecan
SLD = Sum of longest diameters
TCE = T cell engager
TKI = Tyrosine kinase inhibitor
TNBC = Triple negative breast cancer
TPC = Treatment of physician's choice
TROP2 = Trophoblast cell surface antigen-2
VEGF = Vascular endothelial growth factor

Sensitive

Eliav Barr, MD



Senior Vice President, Head of Global Clinical Development, Chief Medical Officer

Dr. Eliav Barr is senior vice president and head of Global Clinical Development and Chief Medical Officer at Merck Research Laboratories. He leads all late-stage clinical development for Merck's human health portfolio and pipeline.

Prior to his current role, Eliav led MRL's Global Medical Affairs organization expanding Merck's scientific engagement and implementation efforts in oncology, vaccines and infectious diseases. Since joining Merck in 1995, Eliav has held positions of increasing responsibility including leadership roles in oncology and infectious diseases clinical development. He was also previously Therapeutic Area Head for Infectious Diseases and managed product development teams in Oncology and Infectious Disease.

Eliav is a cardiologist by training. He received his undergraduate degree from Penn State University and his medical degree from Thomas Jefferson University. He completed his Internal Medicine residency and Cardiology Fellowship at Johns Hopkins University, and subsequently pursued post-doctoral training at the University of Michigan. Prior to joining Merck, he held a faculty position at the University of Chicago.

Sensitive

Marjorie Green, MD



Senior Vice President,
Head of Oncology Global Clinical Development

Dr. Marjorie Green is senior vice president and head of oncology global clinical development at Merck Research Laboratories.

Marjorie joined Merck from Seagen, where she was senior vice president and head of late-stage oncology, leading clinical development of a diverse portfolio of oncology candidates including multiple antibody drug conjugates. She previously held positions of increasing responsibility at Genentech culminating in her tenure as vice president, Global Product Development, head of breast and gynecologic tumor franchise. Previously, she was assistant professor and medical director of the Nellie B. Connally Breast Center and vice-chair of the Institutional Review Board at the MD Anderson Cancer Center, Houston, Texas. During her tenure a MD Anderson, Marjorie established herself as a nationally recognized clinical expert in the management of breast cancer and the treatment and prevention of associated bone metastases and has authored multiple manuscripts and book chapters on preoperative chemotherapy.

Marjorie received her Bachelor of Arts from the University of Notre Dame and her medical degree from the University of Texas Medical Branch. She conducted an internal medicine residency at University of Virginia School of Medicine and completed fellowships in medical oncology and hematology at the MD Anderson Cancer Center.