Forward Looking Statements and Non-GAAP Financial Information

This presentation contains statements about Bristol-Myers Squibb Company’s (the “Company”) future financial results, plans, business development strategy, anticipated clinical trials, results and regulatory approvals that constitute forward-looking statements for purposes of the safe harbor provisions under the Private Securities Litigation Reform Act of 1995. All statements that are not statements of historical facts are, or may be deemed to be, forward-looking statements. Actual results may differ materially from those expressed in, or implied by, these statements as a result of various factors, including, but not limited to, (i) new laws and regulations, (ii) our ability to obtain, protect, and maintain market exclusivity rights and enforce patents and other intellectual property rights, (iii) our ability to achieve expected clinical, regulatory and contractual milestones on expected timelines or at all, (iv) difficulties or delays in the development and commercialization of new products, (v) difficulties or delays in our clinical trials and the manufacturing, distribution and sale of our products, (vi) adverse outcomes in legal or regulatory proceedings, (vii) risks relating to acquisitions, divestitures, alliances, joint ventures and other portfolio actions and (viii) political and financial instability, including changes in general economic conditions. These and other important factors are discussed in the Company’s most recent annual report on Form 10-K and reports on Forms 10-Q and 8-K. These documents are available on the U.S. Securities and Exchange Commission’s website, on the Company’s website or from Bristol-Myers Squibb Investor Relations. No forward-looking statements can be guaranteed.

In addition, any forward-looking statements and clinical data included herein are presented only as of the date hereof. Except as otherwise required by applicable law, the Company undertakes no obligation to publicly update any of the provided information, whether as a result of new information, future events, changed circumstances or otherwise.
Agenda for today

Chris Boerner, PhD, EVP - Chief Operating Officer
Strategic Overview

Robert Plenge, MD, PhD - EVP, Chief Research Officer, Head of Research
Building on our strengths to deliver industry-leading R&D

BREAK (10 min)

Samit Hirawat, MD - EVP, Chief Medical Officer, Drug Development
Accelerating Our Deep Development Pipeline (Immunology, Hematology, & Oncology)

BREAK (10 min)

Samit Hirawat, MD - EVP, Chief Medical Officer, Drug Development
Accelerating Our Deep Development Pipeline (Cardiovascular & Neuroscience)

Chris Boerner, PhD, EVP - Chief Operating Officer
Closing

BMS Leadership
Q&A

Conclusion, lunch reception
Strategic Overview

Chris Boerner, PhD
EVP, Chief Operating Officer
CEO, effective Nov. 1, 2023
Our business has significant opportunities beyond external expectations

### Strong Foundation

- **R&D has delivered:** 9 new medicines, numerous milestones
- **Commercial execution is strong:** Key Inline & New Products continue to grow
- **Business momentum is robust:** Strong base business & expanding New Product Portfolio

### 2023-2030 BMY External vs Internal Revenue Drivers

#### Consensus Drivers
- IRA
- LOE Exposure

#### Drivers of Internal Conviction
- In-line and recently launched products with significant commercial opportunities
- 12 rapidly advancing new medicines in or near registrational development
- R&D productivity and efficiency enhancements
- Strong financial capacity for business development
Numerous levers to drive long-term growth

- Strong Base Business with unrecognized durability
- Increasingly de-risked New Product Portfolio
- Expanding registrational pipeline from 6 to 12 new assets over next 18 months
- Robust early pipeline with 30+ assets & opportunity to deliver ~10 INDs a year
- Increased R&D productivity
- Strategic optionality from Business Development
Our goal is to deliver sustainable growth

Four Key Enablers

- Evolve R&D for scientific leadership
- Strong **commercial execution** to realize value of our marketed portfolio
- Execute **strategic capital allocation** to further strengthen our growth profile
- Foster a **high-performance culture** and attract & retain industry-leading talent

We are driven by our mission: Transforming patients’ lives through science
Focus for today

Four Key Enablers

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We are driven by our mission: Transforming patients’ lives through science
Evolving BMS R&D: World-class organization with increased focus on productivity & scientific leadership

<table>
<thead>
<tr>
<th>Scientific Leadership</th>
<th>Top-tier productivity</th>
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<tr>
<td>Cardiovascular</td>
<td>Increasing Probability of Success</td>
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<tr>
<td>Hematology</td>
<td>Increasing INDs</td>
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<tr>
<td>Oncology</td>
<td>Reducing Cycle Times</td>
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<tr>
<td>Immunology</td>
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<td>Neuroscience</td>
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Capitalize on differentiated platforms

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<tr>
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<th>Retain &amp; attract the best talent in the industry</th>
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<tr>
<td>Targeted Protein Degradation</td>
<td>Enabled by a high-performance culture</td>
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<tr>
<td>Cell Therapy</td>
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<td>Biotherapeutics</td>
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<td>Small Molecules</td>
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Leveraging partnerships and AI/Digital Technologies
Build depth across our therapeutic areas

**Oncology**
- Extend IO leadership
  - SC nivolumab, Opdualag, & next generation assets
- Diversification beyond IO

**Cardiovascular**
- Deepen leadership in cardiomyopathies & heart failure
- Expand expertise in thrombotic diseases

**Hematology**
- Extend leadership across the Multiple Myeloma treatment paradigm
- Broaden portfolio across leukemias, lymphomas and non-malignant hematologic diseases

**Immunology**
- Establish new standards of care in pulmonology
- Strengthen presence in dermatology, rheumatology, & gastrointestinal disorders
- Rapidly advance Cell Therapy into immunologic diseases

**Neuroscience**
- Build a diverse pipeline across neurodegenerative & neuroinflammation diseases
- Advance promising clinical assets in Alzheimer’s Disease & ALS

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Bristol Myers Squibb
Differentiated Platforms: Significantly expand the opportunity in Targeted Protein Degradation & Cell Therapy

Opportunity for Targeted Protein Degradation

Positioned to deliver ~4 INDs annually

- Hematology
- Oncology
- Immunology
- Neuroscience

And expand targets to new therapeutic areas:
- Iberdomide
- Mezigdomide
- Golcadomide
- AR LDD

Waves of agents expected to launch over time:
- Others

Building on Our Leadership in Cell Therapy

Today

- Abecma (GPRC5D CAR T)
- Breyanzi (BCMA x GPRC5D dual binding CAR)

Future

- CD19 NEX T

Expansion into Immunology: Starting with CD19 NEX T

- Severe, refractory lupus
- Multiple Sclerosis
- Other diseases with high unmet need

1. 2023 estimates from Decision Resource Group & BMS Internal Analysis; represents U.S. total diagnosed prevalence
Three R&D productivity objectives to drive long term sustainable growth

1. Increase & Sustain INDs
   - ~10 INDs per year

2. Increase Probability of Success
   - Increase success rate from first-in-human to approval to ~20%

3. Reduce Cycle Times
   - Achieve median of ~6.5 years from first-in-human to approval
Entering a data-rich period supporting potentially first-in-class/best-in-class assets with significant commercial potential

1. Japan or Asia study only 2. Confirmatory trial
Note: excludes assets pending IND approval: HbF Inducer, BCMA x GPRC5D, CD19 NEX T in MS, & TYK2i-CNS
Timeline represents data readouts or key data to inform clinical development (timeline not to scale)

Legend:
- Oncology
- Hematology
- Immunology
- Cardiovascular
- Neuroscience

13 Not for Product Promotional Use
What you will hear today

We are focused on transforming our approach to R&D with an emphasis on:

• Strengthening scientific leadership in our TAs and platforms
• Significantly improving the efficiency and productivity of our R&D engine
• Building a culture of innovation that attracts and retains the best talent

The evolved R&D engine will enhance the data-rich period in the second half of the decade

A number of these assets have the potential to significantly exceed external expectations based on evolving science

Successful execution of our R&D strategy is a core component to enable BMS achieve its strategic goal to achieve sustainable growth
Building on our strengths to deliver industry-leading R&D

Robert Plenge, MD, PhD
EVP, Chief Research Officer, Head of Research
**An integrated approach to research & development**

**Thematic Research Centers (TRCs)**
Biology and translational teams

**Modalities and platforms**
Small molecules, biotherapeutics, cell therapy, targeted protein degradation, nucleic acid therapies

**Research functions**
Computational biology, clinical pharmacology, DMPK, toxicology, translational medicine

Deliver new medicines with transformational potential with an increased probability of success in development

Maximize innovation and productivity to deliver more medicines to patients faster
Three key Research principles to improve R&D productivity

**Causal human biology**
Application of human data (e.g., genetics, longitudinal profiling of patient samples) for rigorous target validation in drug discovery

**Matching modality to mechanism**
Invention of high-quality therapeutics that match a modality to a molecular mechanism of action

**Path to clinical proof-of-concept**
Targeted patient selection (e.g., biomarkers) and clear translational endpoints for improved clinical success

Our ambition is to increase the number of INDs with transformational potential and increased probability of success across all stages of clinical development
Investments in “causal human biology to proof-of-concept” research framework ensure we are industry-leading.

**Causal human biology**
- Human genetics (germline and somatic)
- Translational insights from patients in the real world and BMS clinical trials

**Matching modality to mechanism**
- Diverse modalities, including:
  - Small molecules
  - Biotherapeutics
  - Nucleic acid therapies
  - Targeted Protein Degradation
  - Cell Therapy
- AI-assisted molecule invention

**Path to clinical proof-of-concept**
Technologies and diagnostics to enable mechanistic models for dose, schedule, and patient populations.

**Partnerships**
- TEMPU5
- FINSGEN
- biobank
- insitro
- Exscientia
- Open Targets
- BROAD INSTITUTE
- SCHRÖDINGER
- BigHat BIOSCIENCES

Not for Product Promotional Use 18
We now consistently apply this Research framework to all our programs to deliver transformational medicines with an increased probability of success in development.
Research framework provides confidence in new programs: novel CNS penetrant TYK2 inhibitor for Multiple Sclerosis (MS)

**Transformational potential**
First-in-class, oral, CNS penetrant TYK2 inhibitor with direct anti-inflammatory effects in the CNS to treat neuroinflammatory neurodegenerative disorders.

**Causal human biology**
Mechanism is supported by human genetics (*P1104A* loss-of-function variant), human pathology, clinical fluid biomarkers.

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<tr>
<th>Microglia</th>
<th>Astrocytes</th>
<th>Lymphocytes</th>
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<td>![Microglia Image]</td>
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*pSTAT3*, an indicator of TYK2 activation, is increased in key inflammatory cells of the brain in multiple sclerosis#.

**Matching modality to mechanism**

**Path to clinical proof-of-concept**
Achieve CNS drug exposure to inhibit CNS TYK2 by at least 70% consistent with pre-clinical data in the EAE mouse model (above) and quantitative systems pharmacology modeling of SOTYKTU in psoriasis and SLE.

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# Lu et al, J Neuropathol Exp Neurol 72:1135 (2013);

BMS TYK21-CNS Phenocopies P1104A LoF in EAE

Sci Transl Med 2016 Nov; 8(363): 363ra149
Research framework applied to Oncology builds on our scientific depth in immuno-oncology

**Strategy:**
Build a portfolio of foundational assets to address key tumor intrinsic and tumor extrinsic mechanisms, where combinations will be critical for durable responses with transformational potential.

We have deep expertise in tumor extrinsic biology

Only company with three approved T cell checkpoint inhibitors (CPIs)

Insights from translational datasets to guide the next-generation of transformational medicines

Tumor Extrinsic

- Immune Checkpoints
- Adaptive and Innate Immunity
- Stroma
- Neoantigens
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**Tumor Extrinsic**

**Immune Checkpoints**

**Next-gen T cell CPIs**
- Anti-CTLA4 next-gen, anti-TIGIT bi-specific, dual DGKα/ζ inhibitor

**Other immune cells**
- Tregs - anti-CCR8
- Myeloid - anti-ILT4
- NK cells - anti-NKG2A

**Aberrant Stromal Biology**
- JNK inhibitor, TGFB inhibitor
Research framework applied to Oncology builds on our scientific depth in tumor intrinsic mechanisms

**Strategy:**
Build a portfolio of foundational assets to address key tumor intrinsic and tumor extrinsic mechanisms, where combinations will be critical for durable responses with transformational potential.

**Tumor Intrinsic**

- Oncogenic Drivers
- Lineage Specific
- DNA Damage
- Synthetic Lethal

We have emerging expertise in tumor intrinsic biology

Clinical and pre-clinical programs targeted to specific tumor types and patient subsets

Insights from translational datasets demonstrate the relationship between tumor intrinsic and tumor extrinsic to guide rational combinations
Research framework applied to Oncology builds on our scientific depth in tumor intrinsic mechanisms

**Strategy:** Build a portfolio of foundational assets to address key tumor intrinsic and tumor extrinsic mechanisms, where combinations will be critical for durable responses with transformational potential.

**Tumor Intrinsic**

- **Oncogenic Mechanisms**
  - repotrectinib in ROS1+ lung cancer, RAS signaling (SHP2 inhibitor)

- **Lineage-specific targets**
  - AR LDD in prostate cancer, anti-ganglioside fucosyl-GM1 in SCLC

- **Cancer cell vulnerabilities**
  - Context specific dependencies (e.g., DNA damage), synthetic lethal interactions

**We have emerging expertise in tumor intrinsic biology**

**Emerging clinical and pre-clinical programs targeted to specific tumor types and patient subsets**

**Insights from translational datasets demonstrate the relationship between tumor intrinsic and tumor extrinsic to guide rational combinations**
Research framework plus tumor intrinsic and extrinsic strategy will deliver productivity in Oncology

Causal human biology

Matching modality to mechanism

Path to clinical proof-of-concept

Tumor Intrinsic

Oncogenic Mechanisms
repotrectinib in ROS1+ lung cancer, RAS signaling (SHP2 inhibitor)

Lineage-specific targets
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Next-gen T cell CPIs
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Other immune cells
Tregs - Anti-CCR8
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Aberrant Stromal Biology
JNK inhibitor, TGFB inhibitor
Research framework in action: anti-CCR8 antibody depletes T regulatory cells (Tregs) with combination potential

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**Screening**
- Anti-PD1 mAb
- Suppressed anti-tumor response
- Activated CCR8+ Treg
- Suppressed CD8 T cell

**Treatment**
- Anti-CCR8 mAb
- Tumor cells survive
- Suppressed anti-tumor response
- Depletion of CCR8+ Tregs in the tumor after 2 cycles

**Graph**
- CCR8:CD8 ratio
- Reduced ratio CCR8 Treg to CD8+ Teff in the tumor

**Image**
- Tumor cells
- Activated CCR8+ Treg
- Suppressed CD8 T cell
Research framework in action: anti-CCR8 antibody depletes T regulatory cells (Tregs) with combination potential

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- **Anti-CCR8 mAb**
  - Deplete CCR8+ Treg
  - Reduce ratio, remove brake

- **Anti-PD1 mAb**
  - Activated anti-tumor response
  - Tumor cell killing

- **Activated CD8 T cell**
  - Activated CD8 T cell

- **Tumor cell killing**

**Hypothesis:**

- **Screening**
  - Activated CD8 T cell
  - Activated anti-tumor response
  - Tumor cell killing

- **Treatment**
  - Depletion of CCR8+ Tregs in the tumor after 2 cycles

**Graph:**

- **CCR8:CD8 ratio**
  - Reduced ratio CCR8 Treg to CD8+ Teff in the tumor

**Clinical trial translational data demonstrate CCR8+ regulatory T cells (Tregs) are a major barrier to effective immune response to anti-PD1 therapy in multiple cancer types.**

**Hypothesis:**

- Activated CD8 T cell
  - Activated anti-tumor response
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**Treatment**

- Depletion of CCR8+ Tregs in the tumor after 2 cycles

**Graph:**

- **CCR8:CD8 ratio**
  - Reduced ratio CCR8 Treg to CD8+ Teff in the tumor
Matching modality to mechanism: Leveraging expertise across multiple modalities

### Small molecule chemistry

| Allosteric inhibitors | Active site inhibitors |

### Biotherapeutics

- **Probody® Therapeutic**
- **Immune cell engagers**
- **Bi-specifics**
- **ADCs**

### Nucleic acid therapies

- **Lentivirus and AAV gene therapy**

### Targeted Protein Degradation

- **Molecular glue**
  - CELMoD
- **Heterobifunctional**
  - LDD
- **ADC degrader**
  - CELMoD ADC

### Cell Therapy

- **Autologous**
- **Next-generation**
  - Allogeneic, iPSC-derived
Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets

**Expanded universe of targets**
(e.g., scaffolding proteins, transcription factors)

- **Drugged**: Target with approved drugs (3%)
- **Druggable**: No approved drugs, but bind small molecules with high potency (8%)
- **“Dark Genome”**: No known disease association (30%)
- **Disease-relevant**: No known potent binders, but implicated in disease (61%)
- **Human protein - Coding genes**: ~20,000
- **Small molecule enzyme inhibitor**
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- **Opportunity for TPD**

- **Small molecule enzyme inhibitor**

**Human protein - Coding genes ~20,000**

- 6,007 (30%)
- 659 (3%)
- 12,139 (61%)
- 1,607 (8%)

- **Transcription factor**
- **Scaffold protein**

**Not for Product Promotional Use**
Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets.

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Opportunity for TPD

Small molecule enzyme inhibitor

- Human protein - Coding genes: ~20,000
  - Drugged: 6,007 (30%)
  - Druggable: 1,607 (8%)
  - Disease-relevant: 12,139 (61%)
  - Dark Genome: 5,59 (3%)

**Druggable**
- Transcription factor
- Scaffold protein

**Scaffolds**

**Not for Product Promotional Use**
Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets

**Expanded universe of targets**
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**Small molecule enzyme inhibitor**

**Potential superior efficacy**
(e.g., overcome resistance, higher selectivity)

- **CELMoD**
- **Kinase Inhibitors**

**Human protein – Coding genes**
- 20,000

**Kinase Inhibitors**
- 6,007 (30%)
- 1,607 (8%)

**Scaffold protein**
- 1,699 (8%)

**Transcription factor**
- 12,139 (61%)
Our industry leading position in protein degradation is driven by portfolio breadth and depth of expertise.
Our industry leading position in protein degradation is driven by portfolio breadth and depth of expertise.

Industry-leading capabilities
- Molecular glue
- Hetero-bifunctional
- ADC degrader

Industry-leading pipeline
- Full Development
  - iberdomide: Multiple Myeloma
  - mezigdomide: Multiple Myeloma
- Early Development
  - golcadomide: Lymphoma
  - CK1α: Acute Myeloid Leukemia
- IND-enabling studies
  - BCL6 LDD: Lymphoma
  - Helios: Solid Tumors
  - HbF CELMoD: Sickle Cell Disease
  - LDD: Prostate
  - CELMoD Solid Tumors
  - LDD: Autoimmune

>15 pre-clinical programs across multiple therapeutic areas

Potential to efficiently deliver ~4 INDs annually and expand beyond Heme/Onc targets (Immunology, CV, Neuroscience)
The swift expansion of our CELMoD library has enabled key scientific insights and an increased number of IND candidates. Expanding CELMoD library identifies novel substrates and novel degrons. Diversify chemical library:

- CELMoD library 2019
- CELMoD library 2023

Molecules profiled in proteomics:
- Novel substrates
- Novel degrons

Degrons:
- Degron 1
- Degron 2
- Degron 3

Cereblon
The swift expansion of our CELMoD library has enabled key scientific insights and an increased number of IND candidates.
Targeting the previously undruggable: A novel CELMoD for Sickle Cell Disease

Transformational potential

Oral small molecule that increases fetal hemoglobin to functionally cure sickle cell anemia (e.g., eliminate pain crisis, prevent long term organ damage).

Causal human biology

Genetically validated targets that lead to persistence of fetal hemoglobin (HbF) are associated with improved clinical outcomes in patients with sickle cell anemia.

Beta-globin locus

Matching modality to mechanism

Through our CELMoD proteomics initiative, we have identified CELMoDs that degrade HbF genetic targets and increase HbF in pre-clinical models.

Path to clinical proof-of-concept

Oral small molecule that increases fetal hemoglobin to functionally cure sickle cell anemia (e.g., eliminate pain crisis, prevent long term organ damage).

Genetically validated targets that lead to persistence of fetal hemoglobin (HbF) are associated with improved clinical outcomes in patients with sickle cell anemia.

Beta-globin locus

- 10% Reduced mortality
- 20% Reduced recurring events
- 30% Asymptomatic presentation

Vehicle control

- CELMoD (high)
- CELMoD (low)
- Hydroxyurea

Vehicle control

- CELMoD (high)
- CELMoD (low)
- Hydroxyurea

LCR Hs
Embryonic
Fetal
Adult
3'HS1

Healthy

Disease

HbF

HbS
Ligand directed degraders (LDD) complement CELMoDs in our approach to Targeted Protein Degradation

Different from CELMoDs

Same as CELMoDs

Early Development
- AR LDD: Prostate
- BCL6 LDD: Lymphoma

IND-enabling studies
- LDD: Prostate
- LDD: Autoimmune
BMS-986458 is a novel ligand directed degrader (LDD) targeting BCL6 in lymphoma

Transformational potential

Oral small molecule medicine to treat B cell lymphomas driven by abnormalities in BCL6 signaling pathway.

Causal human biology

Gain-of-function somatic $BCL6$ mutations lead to B cell lymphomas and deletion of $BCL6$ prevents B cell maturation.

Matching modality to mechanism

We created a BCL6 LDD that has exquisite selectivity relative to the human proteome.

Path to clinical proof-of-concept

Correlate BCL6 degradation with clinical benefit

BCL6 IHC high expression in ~30% DLBCL

Healthy, Mature B cells

High BCL6

Germinal center

Malignant B cells (e.g., DLBCL)

Gain-of-function somatic $BCL6$ mutations lead to B cell lymphomas and deletion of $BCL6$ prevents B cell maturation.
A new frontier: CELMoD ADCs to improve efficacy and safety in hematology/solid tumors

**Causal human biology**

Combine a *clinically validated* tumor targeted antibody with a *clinically validated* tumor cell-biased CELMoD to enhance efficacy and tolerability in hematology/solid tumors.

**Matching modality to mechanism**

Tumor targeted antibody to tumor antigen

Tumor cytotoxic CELMoD with catalytic activity

Tumor cell

**Path to clinical proof-of-concept**

Enhanced efficacy of ADC at lower levels of administered CELMoD

*Vehicle control*

*10x CELMoD + Ab, unconjugated*

*1x CELMoD ADC*

*3x CELMoD ADC*

*10x CELMoD ADC*

ADC vs unconjugated components
Targeted Protein Degradation and Cell Therapy: Two differentiated platforms for optimizing therapies for patients

Matching modality to mechanism

Invention of high-quality therapeutics that match a modality to a molecular mechanism of action

These two platforms unlock novel targets and mechanisms to efficiently deliver INDs with the potential to improve the lives of patients
We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency.

**Wave 1**
- **Next-gen engineering**
- Monospecific CAR

**Wave 2**
- **Evolving delivery systems**
- Dual Targeting CAR

**Wave 3**
- **Optimizing manufacturing**
- Engineered CAR/TCR
We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency.

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<td><strong>Next-gen engineering</strong></td>
<td><strong>Evolving delivery systems</strong></td>
<td><strong>Optimizing manufacturing</strong></td>
</tr>
<tr>
<td>Monospecific CAR</td>
<td>Dual Targeting CAR</td>
<td>Engineered CAR/TCR</td>
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</tr>
<tr>
<td>Lentivirus (LVV)</td>
<td>Adeno-associated virus (AAV)</td>
<td>Non-viral delivery (NVD)</td>
</tr>
</tbody>
</table>

- **Optimizing manufacturing**
- **Evolving delivery systems**
- **Next-gen engineering**
We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency.

<table>
<thead>
<tr>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
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</thead>
<tbody>
<tr>
<td><strong>Next-gen engineering</strong></td>
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</tr>
<tr>
<td><strong>Autologous</strong></td>
<td><strong>Autologous</strong></td>
<td><strong>Allogeneic</strong></td>
</tr>
<tr>
<td>NEX T platform</td>
<td></td>
<td>Healthy donor or iPSC</td>
</tr>
<tr>
<td>α/β T cells</td>
<td>α/β T cells</td>
<td>α/β or γ/δ T cells, iNK/iT cells</td>
</tr>
</tbody>
</table>

- **Electroporation**
- **Lipid nanoparticles (LNP)**
- **DNA**
- **Adeno-associated virus (AAV)**
- **Lentivirus (LVV)**
- **Non-viral delivery (NVD)**
- **Single CAR binder**
- **Dual CAR binder**
- **Multiple other KO/KI**
- **TCR KO**
- **MHCI KO**
- **MHCI–II KO**
- **α/β T cells**
- **γ/δ T cells, iNK/iT cells**
### Next-gen Cell Therapy pipeline: Oncology, Immunology, and Allogenic

<table>
<thead>
<tr>
<th>Early Discovery</th>
<th>Late Discovery</th>
<th>IND-Enabling</th>
<th>Development</th>
<th>Approved</th>
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<tbody>
<tr>
<td><strong>Hematology</strong></td>
<td></td>
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<tr>
<td>BMS-986393 GPRC5D CAR T (MM)</td>
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<tr>
<td>BCMAxGPRC5D CAR T (MM)</td>
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<tr>
<td>Allo Eng CAR T (NHL)</td>
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<tr>
<td>iPSC-derived iT/iNK (AML)</td>
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<td>iPSC-derived iT/iNK (MM)</td>
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<thead>
<tr>
<th><strong>Immunology</strong></th>
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</thead>
<tbody>
<tr>
<td>CD19 NEX T (Multiple autoimmune disorders)</td>
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<tr>
<td>Next-gen CAR T (Multiple autoimmune disorders)</td>
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<tr>
<td>Eng Treg (IBD)</td>
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<tr>
<td>Allo Eng gd eTCR T (solid tumors)</td>
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<tr>
<td>Logic-gated CAR T Program 1 (indication not disclosed)</td>
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<tr>
<td>Logic-gated CAR T Program 2 (indication not disclosed)</td>
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<tr>
<td>Allo Eng CAR T (RCC)</td>
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<table>
<thead>
<tr>
<th><strong>Oncology</strong></th>
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</thead>
<tbody>
<tr>
<td>Multiple Eng CAR T Programs</td>
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</tr>
<tr>
<td>Multiple Eng eTCR T Programs</td>
<td></td>
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</tr>
</tbody>
</table>

**Cell Therapy Platform:**
- Autologous
- Allogeneic
- Not disclosed

**Cell Engineering Technologies:**
- CRISPR engineered program
- Immune payloading programs not disclosed

**Partnered:**
- BMS-986393 GPRC5D CAR T (MM)
- BCMAxGPRC5D CAR T (MM)
- Allo Eng CAR T (NHL)
- iPSC-derived iT/iNK (AML)
- iPSC-derived iT/iNK (MM)
- CD19 NEX T (Multiple autoimmune disorders)
- Next-gen CAR T (Multiple autoimmune disorders)
- Eng Treg (IBD)
- Allo Eng gd eTCR T (solid tumors)
- Logic-gated CAR T Program 1 (indication not disclosed)
- Logic-gated CAR T Program 2 (indication not disclosed)
- Allo Eng CAR T (RCC)
- Multiple Eng CAR T Programs
- Multiple Eng eTCR T Programs

**Bristol Myers Squibb®**

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*Not for Product Promotional Use*
Dual targeting BCMAxGPRC5D CAR T for relapsed/refractory multiple myeloma

**Transformational potential**
Primary and secondary non-response to standard of care therapies remains an unmet medical need in MM.

**Causal human biology**
- BCMA and GPRC5D are clinically validated targets independently expressed in multiple myeloma
- Antigen heterogeneity and clonal evolution are factors limiting efficacy of BCMA CAR T in multiple myeloma

**Matching modality to mechanism**
- Optimized bispecific construct to overcome intra-and inter-patient antigen variability/heterogeneity and maintain functionality in cases of low BCMA
- Optimized manufacturing to develop at scale process improving product quality and manufacturing failures

**Path to clinical proof-of-concept**

---

**Abecma trial**

- High BCMA Low GPRC5D
- Low BCMA High GPRC5D

- % BCMA positive tumor cells
- Surface BCMA expression vs. Surface GPRC5D expression

- Baseline Biopsy vs. Progression Biopsy
CD19 NEX T to reset the immune system in multiple Immunology indications

Transformational potential

Sequential immunotherapy offers the potential for a functional cure in autoimmunity: 1: Control inflammation; 2: Reset immune memory; 3: Promote homeostasis and repair

Causal human biology

Academic study of CD19 CAR-T demonstrates B cell memory reset and functional cure in SLE.

Matching modality to mechanism

Chimeric antigen receptor (CAR): CD19 and intracellular domains same as Breyanzi

Manufacturing: autologous, single train with shortened turn-around time, lower failure rates

Path to clinical proof-of-concept

• Expand on findings from academic study in SLE
• Monitor biomarker predictors of cell therapy safety and efficacy
• Demonstrate evidence of resetting immune memory
Computational science, including Artificial Intelligence and Machine Learning, is applied at all stages of Research

Leverage **patient data** and **predictive analytics** to define causal human biology

Utilize computational power for **predictive molecule invention** to improve quality and accelerate timelines

Build **mechanistic models** to address specific problems to increase success and accelerate timelines in the clinic

**Predict:** Molecular design and prioritization

**Make:** Ligand synthesis

**Learn:** Model retraining

**Test:** Dynamic profiling strategy

---

[Diagram showing patient state drivers, somatic mutations, SCNA, and regulon expression in patient states.]

[Diagram showing the cycle of predict, make, learn, and test with integration of AI/ML and drug development models.]
Internal R&D strengths are amplified through extensive network of external partnerships

BMS also has an extensive network of over 150 academic research alliances
We have the right strategy at the right time to develop transformational medicines & change patients’ lives

Path to improve R&D productivity

**Causal human biology**
Use of human data for rigorous target validation in drug discovery

**Matching modality to mechanism**
Diverse portfolio of modalities supplemented with AI and ML

**Path to clinical proof-of-concept**
Enable mechanistic models for dose, schedule, and patient selection

- Build on our strength in five core therapeutic areas
- Diverse modalities, including Targeted Protein Degradation and Cell Therapy
- All enabled by translational insights, computational science and BD partnerships

- Increase number of INDs with transformational potential
- Increase success in clinical development
Program will reconvene following a short break

(10 min)
Accelerating our deep development pipeline

Samit Hirawat, MD
EVP, Chief Medical Officer, Drug Development
An integrated approach to research & development

**Thematic Research Centers (TRCs)**
- Biology and translational teams

**Modalities and platforms**
- Small molecules, biotherapeutics, cell therapy, targeted protein degradation, nucleic acid therapies

**Research functions**
- Computational biology, clinical pharmacology, DMPK, toxicology, translational medicine

**Deliver new medicines with transformational potential with an increased probability of success in Development**

**Maximize innovation and productivity to deliver more medicines to patients faster**

- **Research & Development**
  - Early and Late Clinical Development
  - Global Development Operations
  - Global Regulatory Sciences
  - Global Biometrics & Data Sciences
  - Worldwide Patient Safety
  - Portfolio & Strategic Operations
  - Strategy & Capabilities

**Oncology**
**Hematology**
**Immunology**
**Cardiovascular**
**Neuroscience**

---

Bristol Myers Squibb
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### BMS Pipeline

**Data as of September 14th, 2023**

<table>
<thead>
<tr>
<th><strong>Oncology</strong></th>
<th><strong>Hematology</strong></th>
<th><strong>Immunology</strong></th>
<th><strong>Cardiovascular</strong></th>
<th><strong>Neuroscience</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>+ AHR Antagonist*</td>
<td>+ SHP2 Inhibitor*</td>
<td>+ BET Inhibitor (BM-986378)*</td>
<td>+ IL2-CD25</td>
<td>+ Anti-MTBR-Tau Alzheimer’s Disease</td>
</tr>
<tr>
<td>+ Anti-CCR8*</td>
<td>+ alnuctamab</td>
<td>+ BET Inhibitor (BM-986378)*</td>
<td>+ Anti-IL12B</td>
<td>+ Anti-_MTBR-Tau Alzheimer’s Disease</td>
</tr>
<tr>
<td>+ Anti-IL4*</td>
<td>RR MM</td>
<td>RR NHL</td>
<td>+ afimetoran</td>
<td>+ eIF2b Activator Neuroscience</td>
</tr>
<tr>
<td>+ Anti-NKG2A*</td>
<td></td>
<td>+ Anti-SIRPA</td>
<td>+ PKC8 Inhibitor</td>
<td>+ FAAH/MGLL Dual Inhibitor Neurology</td>
</tr>
<tr>
<td>+ AR LDD</td>
<td>+ JNK Inhibitor</td>
<td>+ TGFβ Inhibitor*</td>
<td>+ Beta crt</td>
<td>+ BTK Inhibitor</td>
</tr>
<tr>
<td>1L, 2L mCRPC</td>
<td>Solid Tumors</td>
<td>Solid Tumors</td>
<td>+ Anti-CD40</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>+ Anti-CD40</td>
<td>+ Anti-CD40</td>
</tr>
</tbody>
</table>

- **Phase 1**: + SC nivolumab + rHuPH20 (multi-indications), + AHR Antagonist, + Anti-CCR8, + Anti-IL4, + Anti-NKG2A, + AR LDD
- **Phase 2**: + SC nivolumab + rHuPH20 (multi-indications), + Anti-CTLA-4, + Anti-IL-8, + BET Inhibitor (BM-986378)*
- **Phase 3**: + SC nivolumab + rHuPH20 (multi-indications), + Anti-CTLA-4, + Anti-IL-8, + BET Inhibitor (BM-986378)*

* Partner-run study; ^ Trials exploring various combinations; NME leading indication; # BMS territory

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**Not for Product Promotional Use**
Immunology
<table>
<thead>
<tr>
<th>Asset</th>
<th>Approved</th>
<th>Registrational†</th>
<th>Exploratory/PoC Studies†</th>
</tr>
</thead>
</table>
| **SOTYKTU™**  (deucravacitinib) 6 mg tablets | Moderate-to-severe Psoriasis             | • Psoriatic Arthritis  
• Sjögren’s Syndrome  
• Systemic Lupus  Erythematosus | Alopecia Areata |
| **ZEPOSIA.**  (ozanimod) 3 mg tablets     | Moderate-to-severe Ulcerative Colitis    | Moderate-to-severe Crohn’s Disease | - |
| CD19 NEX T    | -                                      | -               | Severe, refractory Systemic Lupus  Erythematosus |
| cendakimab    | -                                      | • Eosinophilic Esophagitis  
• Eosinophilic Gastroenteritis† | - |
| LPA₁ Antagonist | -                                      | • Idiopathic Pulmonary Fibrosis  
• Progressive Pulmonary Fibrosis | - |

1. Japan registrational trial; † ongoing or initiating 2023/2024
## Significant unmet need in pulmonary fibrosis

<table>
<thead>
<tr>
<th>Disease</th>
<th>Unmet Need</th>
<th>Treatment Opportunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>FibroticILD: Associated with thickening of the lung lining, causing irreversible damage(^1)</td>
<td>• Ideal novel therapies which can be used alone or in combination with approved anti-fibrotics</td>
<td>• Deliver a potential new product with an improved efficacy and tolerability profile over current treatment options</td>
</tr>
<tr>
<td>IPF: Fatal disease with 3-5 years median survival(^2)</td>
<td>• Treatments needed to address underlying fibrosis and reduce decline in lung function</td>
<td>• Approved therapies do not treat underlying fibrosis or halt disease progression</td>
</tr>
<tr>
<td>PPF: Heterogeneous group of ILDs with a progressive-fibrosing phenotype(^1)</td>
<td>• Tolerable treatment options to increase adherence and QoL improvement</td>
<td></td>
</tr>
</tbody>
</table>

LPA₁ signaling is central to the pathogenesis of fibrotic lung diseases

1. **Epithelial cell apoptosis¹**
   LPA₁ signaling promotes apoptosis of alveolar epithelial cells

2. **Fibroblast recruitment²,³**
   LPA₁ signaling induces fibroblast chemotaxis to the site of injury

3. **Fibroblast proliferation & activation⁴,⁵**
   LPA₁ signaling stimulates fibroblast proliferation and collagen secretion

4. **Fibroblast survival¹**
   LPA₁ signaling increases fibroblast resistance to apoptosis

---

Robust Phase 2 IPF and PPF results support development of BMS-986278 across the spectrum of progressive lung fibrosis

Compelling reduction in the decline of lung function at 60 mg in both IPF and PPF cohorts, with a favorable and differentiated tolerability profile

ALOFT-IPF\textsuperscript{1} and ALOFT-PPF\textsuperscript{2}: Two parallel Phase 3 registrational studies

**Key Inclusion:**
- >40 yo (IPF); >21 yo (PPF)
- FVC $\geq 40\%$, DLco $> 25\%$
- With or without concomitant background SoC

**Primary Endpoint:**
- Change in FVC (mL) at week 52

**Key Secondary Endpoint:**
- Time to disease progression
- Patient-reported outcomes
- Change in 6MWT

**Primary Endpoint**
- Week 52

<table>
<thead>
<tr>
<th>Arm</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMS-986278</td>
<td>(60 mg BID)</td>
</tr>
<tr>
<td>BMS-986278</td>
<td>(120 mg BID)</td>
</tr>
<tr>
<td>Placebo</td>
<td></td>
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</tbody>
</table>

**Patients continue treatment and follow-up**

**Phase 3 studies initiating**

Data anticipated in 2026 (IPF) and 2028 (PPF)

1. NCT06003426; 2. NCT06025578
Significant unmet medical need in lupus

Complex Disease

- Chronic auto-immune disorder of widespread inflammation leading to end-organ damage and death
- Impact on QoL due to multiple associated comorbidities (i.e., infections, CV disease)

Current Treatment Landscape

- Few approved branded therapies
- Current options have limited efficacy
- Many therapies require repeated injections

**Significant Need: Opportunity for patients to have a novel, oral, effective medicine**
SLE Phase 2 results across endpoints provide rationale for Phase 3

**SRI(4) response rates at week 48**

- **Δ 22.7** (95% CI, 7.4-36.6)
- OR: 2.6 (95% CI, 1.4-4.8)

**Achievement of Lupus Low Disease Activity (LLDAS) at week 48**

- **Δ 22.9** (95% CI, 9.6-35.3)
- OR: 4.0 (95% CI, 1.9-8.5)

 Met the primary endpoint, and all secondary endpoints achieved or meaningfully improved at week 48 with a well tolerated safety profile consistent with earlier trials

1. Morand E, et al. *Arthritis & Rheumatology*. 2023 Feb;75(2):242-252.; Multiplicity-adjusted secondary end point; Primary endpoint was met: percentage of patients achieving SRI(4) at week 32 (P<0.001)
SLE Phase 2 data demonstrates compelling efficacy across domains

### Skin Domain: CLASI-50

- Baseline CLASI activity score ≥ 10 with ≥ 50% decrease from baseline
- **Response rate, %**
  - Placebo: 16.7% (n = 24)
  - deucravacitinib 3 mg BID: 69.6% (n = 23)
  - deucravacitinib 6 mg BID: 56.0% (n = 25)
  - deucravacitinib 12 mg QD: 62.1% (n = 29)

### Joint Domain: Joint Count-50

- ≥ 6 active (tender + swollen) joints at baseline, with ≥ 50% decrease from baseline
- **Response rate, %**
  - Placebo: 45.3% (n = 64)
  - deucravacitinib 3 mg BID: 68.3% (n = 63)
  - deucravacitinib 6 mg BID: 52.3% (n = 65)
  - deucravacitinib 12 mg QD: 56.5% (n = 62)

**Patient response treated with deucravacitinib in the PAISLEY Phase 2 study**

- **Baseline**
- **Near complete resolution**
- **Day 1**
- **Week 20**
- **Week 40**

---

2. Multiplicity-adjusted secondary end point; Δ 52.9 (95% CI, 21.7-72.7), OR: 10.5 (95% CI, 2.5-43.0).
3. Exploratory non-multiplicity-controlled end point; 4. NCT03252587, Images used with investigator permission.
SLE Phase 3 registrational program (POETYK-SLE-1\(^1\) and POETYK-SLE-2\(^2\) parallel studies)

**Inclusion Criteria:**
- SLEDAI-2K ≥ 6 with skin and/or joint involvement
- BILAG:1A or 2Bs
- Seropositivity
- Stable background therapy
- No severe organ-threatening disease

**Primary Endpoint:**
- SRI(4) at Week 52

---

**Primary Endpoint**
- Week 52

**Optional long-term extension and follow-up**

**Randomization**
- 1:1

**Treatment Groups**
- deucravacitinib 3 mg BID + SoC
- Placebo + SoC

**Data anticipated in 2026**

---

1. NCT05617677; 2. NCT05620407
Development in Sjögren’s Syndrome supported by Phase 2 results in SLE

Unmet Need

- SjS is an autoimmune disease characterized by dry eye and mouth with potential involvement of other organs
- No approved therapies that slow the progression of SjS
- Most patients require supportive care to manage symptoms

Disease mechanism and genetic data support reason to believe

- Genetic studies implicate TYK2 pathways in SjS
- Interferon activity is increased systemically and in tissue of patients with SjS
- SjS and SLE have shared pathogenesis with common biomarkers and lab findings

Based on similarity to SLE and high unmet need, the Phase 3 trial in Sjögren’s Syndrome is ongoing

### Immunology

#### Sjögren’s Syndrome Phase 3 study (POETYK-SjS-1^1^)

**Inclusion Criteria:**
- Meet 2016 ACR/EULAR criteria with disease duration ≤ 7.5 yrs
- Anti-SSA/RO+
- ESSDAI ≥5

**Primary Endpoint:**
- ESSDAI change from baseline at Week 52

**Key Secondary Endpoint:**
- ESSPRI

**Primary Endpoint**
- Week 52
  - deucravacitinib 3 mg BID
  - deucravacitinib 6 mg BID
  - Placebo

**Long-term extension and follow-up**

**Data anticipated in 2027**
First-in-class TYK2 inhibitor to treat PsO, with broad potential across PsA, SLE, SjS, and AA

<table>
<thead>
<tr>
<th>Today</th>
<th>Near-Term</th>
<th>Future</th>
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</thead>
<tbody>
<tr>
<td>Psoriasis</td>
<td>Psoriatic Arthritis</td>
<td>Systemic Lupus Erythematosus, Sjögren’s Syndrome, &amp; Alopecia Areata</td>
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</tbody>
</table>

Sotyktu is approved for moderate-to-severe PsO and has reset the bar for oral treatments

Leveraging strong relationship between PsO and PsA, sharing common pathogenic mechanisms

Disruptive potential in SLE and new opportunity in SjS given similar disease pathogenesis

Potential in AA based on inhibition of the IL-12/IFNγ axis

Oral, tolerable, mechanistically differentiated TYK-2 targeting agent provides broad applicability across a range of immune-mediated diseases

Compelling CAR T data in lupus supports expanding new modality to address unmet need

Potential transformational efficacy and favorable safety demonstrated with CD19 CAR T

**Disease Remission Post CAR T Treatment**

- All patients achieved complete remission

**Data Suggests Immune System Reset**

- Patient immune system reset after CAR T treatment: Measurement of immunoglobulins shift from mature B-cells expressing IgA & IgG to naïve B-cells expressing IgM & IgD

7/7 pts achieved durable DORIS complete remission of all therapy; encouraging safety & tolerability with only grade 1 cytokine release syndrome and no neurotoxicity

---

Potential transformative treatment option for patients with certain severe immunologic diseases

BMS-986353 expresses the same CD19 specific CAR construct as best-in-class Breyanzi\(^1,2\)

- **Anti-CD19 Targeting Domain\(^1,2\)**
  - Extracellular single-chain variable fragment to recognize CD19
- **CD28 Hinge/Transmembrane Domain\(^3\)**
- **4-1BB Costimulatory Domain\(^1,2\)**
  - Stimulates CD8\(^+\) central memory T-cell generation and favors CAR T-cell persistence\(^4\)
- **CD3-ζ Activation Domain\(^1,2\)**

Breyanzi achieves rapid and complete B-cell depletion in patients with B-cell malignancies

**NEX T: Next generation technology manufacturing platform balances speed and robustness**

- **Faster turnaround time**
  - Optimized cell expansion time
- **Increased productivity**
  - Leverages a closed and automated manufacturing platform leads to increased yield and lowered cost
- **Innovative technologies**
  - Proprietary harvest technology improves purity

---

Severe, refractory SLE Phase 1 study

Open label\(^1\): Assess the safety, preliminary efficacy, pharmokinetics

Key eligibility criteria:
- 2019 ACR/EULAR classification criteria of SLE
- Presence of anti-dsDNA, anti-histone, anti-chromatin, or anti-Sm antibodies
- ≥ 1 major organ system with a BILAG A score
- Inadequate response to glucocorticoids and to at least 2 treatments

Part A
Dose escalation

Part B
Dose expansion to optimize RP2D

Data anticipated in 2024

---

1. NCT05869955

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Rapidly expanding into other B-cell mediated diseases

Numerous B-cell mediated diseases with select patient populations

- SLE
- Systemic Sclerosis
- Idiopathic inflammatory Myopathies
- Multiple Sclerosis
- Other indications

- Adding cohorts to Phase 1 severe, refractory SLE trial (e.g., myositis and others)
- Phase 1 trial in Multiple Sclerosis to be initiated
Rapidly building our portfolio in Immunology

- **LPA₁ Antagonist**: New potential standard of care in IPF & PPF with registrational Phase 3 programs initiating
- **Sotyktu**: Compelling Phase 2 data supports ongoing registrational Phase 3 programs in SLE & SjS
- **CD19 NEX T**: Phase 1 study in severe, refractory SLE initiated and expanding into other immunologic diseases
- Exciting additional registrational Phase 3 programs:
  - **Cendakimab** in EoE & EGE
  - **Zeposia** in CD
  - **Sotyktu** in PsA
- Exploring 5 additional assets in early development across indications

Addressing immunologic diseases with high unmet need impacting 8M+1 patients
Hematology
Addressing high unmet medical need in Hematology

<table>
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<th>Exploratory/PoC Studies†</th>
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<tbody>
<tr>
<td><strong>Abecma</strong></td>
<td>5L+ R/R MM²</td>
<td>• 3L+ triple-class exposed MM</td>
<td>-</td>
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<tr>
<td></td>
<td></td>
<td>• Sub-optimal response post-SCT</td>
<td></td>
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<td></td>
<td></td>
<td>• R/R CLL/SLL</td>
<td>R/R MCL</td>
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<td></td>
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<td>• 2L+ FL; 3L+ FL</td>
<td>R/R MCL</td>
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<tr>
<td><strong>Breyanzi</strong></td>
<td>2L LBCL; 3L+ LBCL</td>
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<td>1L NTD MDS</td>
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<td>Alpha Thalasemia³</td>
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<td></td>
<td></td>
<td>• TD MF</td>
<td></td>
</tr>
<tr>
<td><strong>Reblozyl</strong></td>
<td>1L MDS; 2L TD MDS-RS; TD</td>
<td>Novo combinátions in MM</td>
<td>Novel combinátions in MM</td>
</tr>
<tr>
<td></td>
<td>&amp; NTD² Beta Thalasemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>alnuctamab</strong></td>
<td>2-4L MM</td>
<td></td>
<td>Novel combinátions in MM</td>
</tr>
<tr>
<td><strong>BET Inhibitor</strong></td>
<td>1L LBCL</td>
<td>NDMM post-SCT maintenance</td>
<td></td>
</tr>
<tr>
<td>(BMS-986158)</td>
<td>2-3L MM</td>
<td>2-3L MM</td>
<td></td>
</tr>
<tr>
<td><strong>iberdomide</strong></td>
<td>-</td>
<td>1L LBCL</td>
<td>1L DLBCL</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>R/R PTCL⁴</td>
</tr>
<tr>
<td><strong>golcadomide</strong></td>
<td>-</td>
<td>Quadruple-class exposed MM</td>
<td>Novel combinátions</td>
</tr>
<tr>
<td><strong>GPRC5D CAR T</strong></td>
<td>-</td>
<td>2-4L MM</td>
<td></td>
</tr>
<tr>
<td><strong>mezigdomide</strong></td>
<td>-</td>
<td>2L+ MM</td>
<td></td>
</tr>
</tbody>
</table>

1. Approved in 4L+ ex-US; 2. NTD approved ex-U.S.; 3. Asia-only study; 4. Japan-only study; † ongoing or initiating 2023/2024
Rapidly expanding use in the treatment of anemia

**FDA approved as first-line treatment** of anemia in adults with lower-risk MDS

- First and only therapy to demonstrate superiority over epoetin alpha in the head-to-head Phase 3 COMMANDS study
- Nearly doubled transfusion independence with concurrent hemoglobin increase vs epoetin alpha with a well-established safety profile
- Demonstrates more durable responses of transfusion independence vs epoetin alpha

Expansion opportunities with ongoing studies in anemia associated with 1L TD MF, 1L NTD MDS, and alpha-thalassemia¹

---

¹. Asia-only study
Phase 3 INDEPENDENCE 1L TD anemia in MF trial design

**Key Eligibility Criteria:**
- MPN-associated MF
- Stable dose JAK2 inhibitor
- Transfusion dependent

**Stratification:**
- BL RBC transfusion burden
- DIPSS (intermediate vs. high)

**Primary Endpoint:**
- RBC transfusion independence for ≥12 weeks

**Key Secondary Endpoint:**
- RBC transfusion independence for ≥16 weeks

<table>
<thead>
<tr>
<th><strong>Blinded Core Treatment Period</strong> (24 weeks)</th>
<th><strong>Extended Treatment</strong> (Week 25+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Luspatercept + JAK2i SQ every 3 weeks</td>
<td>Day 169 Response Assessment: RBC-TI 12W/24W</td>
</tr>
<tr>
<td></td>
<td>Continued Treatment Crossover from placebo to luspatercept allowed if no response at primary endpoint response assessment</td>
</tr>
</tbody>
</table>

- R:2:1
- Day 169 Response Assessment: RBC-TI 12W/24W

**Expected data readout 2025**

1. NCT04717414

---

Not for Product Promotional Use
BMS-986158: Potential-best in-class BET inhibitor with broad applicability

**BETi Mechanism of Action**

Unmet need in MF remains for new treatments which lead to strong & durable spleen volume reduction, symptom improvement, and extended survival

**BET Inhibitors alone and in combination with JAK inhibitors have shown clinical benefit in patients with MF**


**BETi: Phase 1/2 study ongoing in MF**

- **Dose Escalation Phase**
  - 1L MF (rux-naïve) BMS-158 + ruxolitinib 15 mg BID
  - 1L MF (add-on to rux) BMS-158 RP2D + ruxolitinib previously tolerated dose
  - 2L MF (rux-exposed) BMS-158 RP2D + fedratinib 400 mg QD
  - 2L MF (rux-exposed) BMS-158 RP2D monotherapy

- **Dose Expansion Phase**
  - 1L MF (rux-naïve) BMS-158 RP2D + ruxolitinib 10 mg BID

**Primary Endpoint:** Safety, tolerability, MTD and/or RP2D

**Key Secondary Endpoint:** Preliminary efficacy based on SVR

**Proof-of-concept data anticipated in 2024**

Breyanzi provides transformational benefits to patients

Before Breyanzi infusion

One month after Breyanzi infusion

Follicular Lymphoma Patient from TRANSCEND-FL

1. Images used with investigator permission from TRANSCEND-FL
Best-in-class CAR T across the broadest array of B-cell malignancies

<table>
<thead>
<tr>
<th>01</th>
<th>02</th>
<th>03</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Best-in-class CAR T with the broadest label in 2L+ LBCL</td>
<td>- TRANSCEND-CLL: First &amp; only pivotal trial in high-risk R/R CLL/SLL</td>
<td>- Potential best-in-disease in R/R FL</td>
</tr>
<tr>
<td>- Differentiated efficacy &amp; safety profile</td>
<td>- Demonstrated deep and durable responses</td>
<td>- Unprecedented data in additional lymphoma types including R/R MCL</td>
</tr>
</tbody>
</table>

### LBCL
Large B-Cell Lymphoma
- Rapidly progressive but responsive to chemotherapy and often curable

### MCL
Mantle Cell Lymphoma

### CLL/SLL
Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma

### FL
Follicular Lymphoma

### MZL
Marginal Zone Lymphoma
- Slowly progressive and responsive to therapy but not typically curable with standard approaches

---

*Not for Product Promotional Use*
Expanding Targeted Protein Degradation into lymphoma

Unmet Need in 1L LBCL: High-risk disease defined based on the IPI, where R-CHOP leads to cure in less patients

High response rates seen with golcadomide + R-CHOP in 1L DLBCL

Manageable Safety Profile

- No new safety signals were observed with golcadomide monotherapy
- Golcadomide was safely combined with rituximab, with no DLTs observed
- Golcadomide has good combinability with R-CHOP, with manageable safety profile

Plan to initiate 1L LBCL registrational trial in 2024
Data anticipated 2027+

Moving into earlier lines of therapy in multiple myeloma

Today

5L+ MM with differentiated real-world evidence

Near-Term

Moving into a triple-class exposed population

Future

Phase 3, KarMMa-9 study initiating

Expansion to NDMM with inadequate response to transplant
Confidence in Abecma’s competitive profile further reinforced by real world evidence

- Predictable safety profile well understood
- Real world data confirms the efficacy profile in a real world population
- Strong manufacturing reliability confirmed: 94% success rate
- Data supports CAR T before other BCMA-targeted therapies in the real world

<table>
<thead>
<tr>
<th>R/R MM Efficacy Outcomes¹</th>
<th>R/R MM Safety Outcomes¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Best ORR</td>
<td>Any CRS/Grade ≥3</td>
</tr>
<tr>
<td>73% ide-cel KarMMa</td>
<td>84% ide-cel KarMMa</td>
</tr>
<tr>
<td>33% ide-cel KarMMa</td>
<td>5% ide-cel KarMMa</td>
</tr>
<tr>
<td>42% ide-cel RWE</td>
<td>18% ide-cel RWE</td>
</tr>
<tr>
<td>Best Response ≥ CR</td>
<td>Any NT/Grade ≥3</td>
</tr>
<tr>
<td>84% ide-cel KarMMa</td>
<td>82% ide-cel RWE</td>
</tr>
<tr>
<td>18% ide-cel KarMMa</td>
<td>18% ide-cel RWE</td>
</tr>
<tr>
<td>3% ide-cel RWE</td>
<td>6% ide-cel RWE</td>
</tr>
</tbody>
</table>

KarMMa-2\(^1\): Strong data supports advancing Abecma into Phase 3 KarMMa-9 study

**Overall Response Rate**

- **ORR, 87.1%**
  - sCR: 9.7%
  - CR: 25.8%
  - VGPR: 48.4%
  - PR: 20%
  - CR, 74.2%

**Progression-Free Survival**

- **PFS, %**
  - 90.1% (SE: 5.43)
  - 83.1% (SE: 6.89)

**Safety**

- **Grade ≥3**
  - n=31
  - CRS 0
  - iiNT 3.2%
  - Infections 12.9%

Manageable tolerability profile with low rates of Gr≥3 NT and infections and no Gr≥3 CRS

KarMMa-2 Cohort 2c studied Abecma in patients with clinical high-risk MM due to inadequate response (<VGPR) to frontline ASCT

1. Dhodapkar M, et al. ASH 2022 [Presentation #3314].
**Inclusion Criteria:**
- Adult patients with PR or VGPR to ASCT

**Primary Endpoint:**
- PFS

**Key Secondary Endpoint:**
- OS

**Stratification Factors:**
- R-ISS III at initial diagnosis
- Anti-CD38 induction
- VGPR vs PR

---

**Pivotal KarMMa-9 in patients with sub-optimal response post-ASCT**

- **NDMM after 4-6 Cycles Induction** → **HDT/ASCT** → **PR or VGPR** → **lenalidomide maintenance 1 cycle** → **Apheresis** → **Flu/Cy + ide-cel** → **lenalidomide maintenance (start 1 month after ide-cel/counts recovery; continue until PD)** → **lenalidomide maintenance until PD** → **Follow-Up**

---

**Pivotal KarMMa-9 study initiating**

Data anticipated in 2027
Hematology

Alnuctamab demonstrates deep and durable responses in RRMM

Overall Response Rate: Efficacy supports the optimal Phase 3 dose

Deep and durable responses with clinically important MRD negativity

Safety

<table>
<thead>
<tr>
<th>Grade ≥3</th>
<th>n=73</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRS</td>
<td>0</td>
</tr>
<tr>
<td>ICANS</td>
<td>0</td>
</tr>
<tr>
<td>Infections</td>
<td>10%</td>
</tr>
</tbody>
</table>

Hematologic:
- Neutropenia 42%
- Anemia 25%
- Thrombocytopenia 14%

Responses deepened over time, with CRS limited to low-grade, short-lived events (median duration 2 days)

Aggressive development plan to move into earlier lines and leverage proprietary combinations

<table>
<thead>
<tr>
<th>RRMM 1-3 prior lines</th>
<th>RRMM ≥3 prior lines (dose escalation)</th>
<th>RRMM ≥3 prior lines (dose escalation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>alnuctamab monotherapy vs Investigator’s Choice SOC</td>
<td>alnuctamab + GPRC5D CAR T</td>
<td>alnuctamab + mezigdomide</td>
</tr>
</tbody>
</table>

- Phase 3, placebo-controlled randomized study
- Anti-CD38 mAb & lenalidomide exposed and BCMA-targeting therapy naïve
- Phase 1b, dose escalation and dose optimization study
- Dose escalation: Triple class exposed; prior BCMA or GPRC5D therapies allowed
- Phase 1b, dose escalation and dose optimization study
- Dose escalation: Anti-CD38 mAb exposed or naïve

Initiating Phase 3 trial in 2024
Hematology

GPRC5D CAR T has differentiated MoA/construct, addressing unmet need in post-BCMA treated population

GPRC5D-targeted CAR construct

- Anti-GPRC5D domain
- Hinge and transmembrane domain
- 4-1BB
- CD3-zeta

Critical need for new targets as the number of post-BCMA treated patients increases

GPRC5D is a clinically validated receptor highly expressed on MM cells with limited expression in other tissues and shows great potential for treatment of advanced MM

Overexpression of GPRC5D is associated with poor disease prognosis

Matching modality to mechanism

<table>
<thead>
<tr>
<th>TCE</th>
<th>CAR T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeated administration</td>
<td>One-time infusion</td>
</tr>
</tbody>
</table>

Hypothesis:
Deliver strong anti-tumor efficacy with a better on-target/off-tumor tolerability profile

Though CAR T manufacturing and scalability is bespoke, therapy is a one-time infusion leading to significant efficacy and a manageable tolerability profile

GPRC5D CAR T in post-BCMA patients shows compelling efficacy and differentiated safety

- **ORR** = 96.3%\(^1\) for patients with prior BCMA-targeted therapy (cilta-cel, ide-cel, orva-cel, ALLO 715, others not specified)
- **ORR** = 76%\(^1\) for patients with no prior BCMA-targeted therapy

### Patients with prior BCMA-targeted therapy, n (%) (n = 25)
- CAR T-cell therapy: 19 (76)
- Non-CAR T-cell therapy: 8 (32)

### Patients with no prior BCMA-targeted therapy, n (%) (n = 27)
- CAR T-cell therapy: 6 (32)
- Non-CAR T-cell therapy: 21 (78)

### GPRC5D CAR T on-target/off-tumor safety profile
- Differentiated from bispecifics with lower rates of any grade events, and no Grade ≥ 3 events

---

RegISTRATIONAL TRIAL TO BE INITIATED 1H 2024

- Quadruple class exposed: IMiD, PI, anti-CD38, anti-BCMA
- Explore novel combinations: CELMoDs or anti-BCMA TCE
- Expand in 2L+ vs SOC

- Key segment in RRMM
- Potential to explore head-to-head study vs standard therapies
- Demonstrate high ORR and DOR in a high-need population
- Expand development with combinations in earlier disease setting
- Additional studies planned in 2024+

BRISTOL MYERS SQUIBB
Two multiple myeloma CELMoDs are in registrational trials

**iberdomide**
- Synergistic in vitro activity with anti-CD38 mAb\(^1\)
- Properties enable combinability, enhanced anti-MM activity, and favorable tolerability
- Potential to establish iberdomide in combination with anti-CD38 mAb in earlier lines

**mezigdomide**
- Highly potent, optimized for rapid and maximal degradation of target proteins
- Induces tumor cell death and responses needed to regain control in advanced disease
- Potential to establish mezigdomide-PI triplet combination in later lines, post-lenalidomide and anti-CD38 mAb

<table>
<thead>
<tr>
<th>Asset (indication)</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Projected Data Readout</th>
</tr>
</thead>
<tbody>
<tr>
<td>mezigdomide (RRMM 2-4L)</td>
<td>SUCCESSOR-1(^2)</td>
<td></td>
<td></td>
<td>2026</td>
</tr>
<tr>
<td>mezigdomide (RRMM 2L+)</td>
<td>SUCCESSOR-2(^3)</td>
<td></td>
<td></td>
<td>2026</td>
</tr>
<tr>
<td>iberdomide (RRMM 2-3L)</td>
<td>EXCALIBER-RRMM(^4)</td>
<td></td>
<td></td>
<td>2026</td>
</tr>
<tr>
<td>iberdomide (post-SCT maintenance)</td>
<td>EXCALIBER-MAINTENANCE(^5)</td>
<td></td>
<td></td>
<td>2029</td>
</tr>
</tbody>
</table>

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Extending leadership in multiple myeloma: Opportunity to help patients across their treatment journey

Patient Segments

BMS Assets

Newly Diagnosed Multiple Myeloma (1L)
- Post-SCT consolidation

Early Relapse and Refractory Multiple Myeloma (2L-4L)
- anti-CD38 Sensitive/Naïve
- Post anti-CD38

Late Relapse and Refractory Myeloma (5L+)
- Triple class exposed
- Quadruple class exposed

Approved
- Abecma
- Golcadoamide
- Alnuctamab
- GPRC5D CAR T

Planned
- Iberdomide
- Mezigdomide
- Alnuctamab

Ongoing
- Breyanzi
- GPRC5D CAR T

Hematology
Broadening leadership across malignant and benign Hematology

Reblozyl:
- Recent FDA approval in 1L MDS-associated anemia with a broad label
- 1L TD Myelofibrosis associated anemia Phase 3 ongoing

Numerous assets to extend leadership in Multiple Myeloma:
- Abecma is under regulatory review in the triple class exposed population; Phase 3 initiating in patients with sub-optimal response post-ASCT
- GPRC5D CAR T as a potential first-in-class CAR T with registrational program initiating next year
- iberdomide & mezigdomide registrational data expected in 2026
- alnuctamab initiating Phase 3 next year

Strengthening breadth of leadership across leukemias, lymphomas, and benign hematology:
- Best-in-class Breyanzi expanding across the broadest array of B-cell malignancies
- Golcadomide moving into Phase 3 in 1L LBCL
- BET inhibitor (BMS-986158) as a potential new option for patients with Myelofibrosis

Addressing hematologic diseases impacting 4M+1 patients
Oncology
# Addressing high unmet medical need in Oncology

<table>
<thead>
<tr>
<th>Asset</th>
<th>Approved</th>
<th>Registrational†</th>
<th>Exploratory/PoC Studies†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>OPDIVO (nivolumab)</strong></td>
<td>26 approvals across 11 tumors</td>
<td>9 ongoing trials</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1L melanoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Adj. melanoma</td>
<td></td>
<td>• 1L/2L+ HCC</td>
</tr>
<tr>
<td></td>
<td>• 2L/3L+ MSS CRC</td>
<td></td>
<td>• 1L NSCLC</td>
</tr>
<tr>
<td></td>
<td>• 1L melanoma SC</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Opdualag</strong></td>
<td>1L melanoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• U.S. - All Q2W &amp; Q4W indications (bridging from 2L RCC)</td>
<td>U.S. All Q2W &amp; Q4W indications (bridging from 2L RCC)</td>
<td>NTRK Pan Tumor</td>
</tr>
<tr>
<td>repotrectinib²</td>
<td>-</td>
<td>1L ROS1+ NSCLC</td>
<td></td>
</tr>
<tr>
<td>subcutaneous nivolumab¹</td>
<td>-</td>
<td>U.S. All Q2W &amp; Q4W indications (bridging from 2L RCC)</td>
<td>NTRK Pan Tumor</td>
</tr>
<tr>
<td>AR LDD</td>
<td>-</td>
<td></td>
<td>2L+ mCRPC</td>
</tr>
<tr>
<td>DGK Inhibitor</td>
<td>-</td>
<td></td>
<td>Solid tumors</td>
</tr>
<tr>
<td>farletuzumab ecteribulin</td>
<td>-</td>
<td></td>
<td>NSCLC &amp; ovarian</td>
</tr>
<tr>
<td>TIGIT Bispecific</td>
<td>-</td>
<td></td>
<td>NSCLC &amp; gastric</td>
</tr>
</tbody>
</table>

1. U.S. Regulatory path opens up indications with Q2W and Q4W dose including melanoma, CRC, HCC, 2L NSCLC, UC, ESCC, & Gastric. U.S. PDUFA: November 27, 2023

† ongoing or initiating 2023/2024
## Continuing to grow Opdivo / Dual IO

### Metastatic Setting

<table>
<thead>
<tr>
<th>Tumor/Trial</th>
<th>Status</th>
<th>Tumor/Trial</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subcutaneous nivolumab CM-67T</td>
<td>2023 Readout</td>
<td>MSI-H CRC CM-8HW Opdivo + Yervoy</td>
<td>2025 Readout</td>
</tr>
<tr>
<td>1L MIUC CM-901 Opdivo + Yervoy vs SOC chemo</td>
<td>2024 Readout</td>
<td>1L HCC CM-9DW Opdivo + Yervoy vs sorafenib / lenvima</td>
<td>2025 Readout</td>
</tr>
</tbody>
</table>

### Early-Stage Setting

<table>
<thead>
<tr>
<th>Tumor/Trial</th>
<th>Status</th>
<th>Tumor/Trial</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSCLC (Peri-Adj) CM-77T Neo-adj Opdivo + chemo followed by Adj Opdivo vs chemo</td>
<td>2024 Readout</td>
<td>NSCLC Stage 3 (Unresectable) CM-73L Opdivo mono, O+Y vs Imfinzi</td>
<td>2025 Readout</td>
</tr>
<tr>
<td>NSCLC (Adj) ANVIL Opdivo vs Observation</td>
<td>2024 Readout</td>
<td>HCC (Adj) CM-9DX Opdivo vs Placebo</td>
<td>2025 Readout</td>
</tr>
<tr>
<td>MIBC (Peri-Adj) CA017-078 Opdivo + chemo vs chemo</td>
<td>2024 Readout</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**26 OPDIVO approvals**

**10 YERVOY approvals**

**11 tumors**
SC administration has clear benefits for patients, HCPs, and healthcare systems

**HCPs and Healthcare System**
- **Logistical:** Complex scheduling demands due to higher patient volume
- **Resource utilization:** Overlapping duties for staff, inefficient patient to nurse ratios

**Patients**
- **Time burden:** Inconvenience, opportunity cost/income loss
- **Emotional burden:** Loss of normality, long-term survivorship and ‘chronic care’

---

Subcutaneous nivolumab: Opportunity for a near-term launch potentially benefitting patients into the early 2030s

Checkmate 67T\(^1\): Phase 3 study
- Patients with advanced or metastatic ccRCC who have received prior systemic therapy

**Primary Endpoint:**
- Time-averaged serum concentration over 28 days (Cavgd28)
- Trough serum concentration at steady-state (Cmin)

**Key Secondary Endpoint:**
- ORR

Data expected later this year & launch anticipated in 2024/2025
U.S. Regulatory path opens up indications with Q2W and Q4W dose\(^2\)
Indications encompass majority of Opdivo 2022 net sales in the U.S.

---

1. NCT04810078; 2. Supported with data from Checkmate-8KX
Next-generation IO medicine with significant potential to benefit patients into the next decade

<table>
<thead>
<tr>
<th>Today</th>
<th>Near-Term</th>
<th>Future</th>
</tr>
</thead>
</table>
| 2/3L+ MSS Colorectal Cancer & Adjuvant melanoma  
Strong launch supporting rapidly expanding dual IO option for 1L melanoma patients | Phase 3 trials ongoing | Phase 2 trials underway to inform path forward 
Non-Small Cell Lung Cancer & Hepatocellular Carcinoma |
Adjuvant Melanoma: High conviction indication with potential to benefit patients before disease spreads

Clear evidence of neo-adjuvant activity with relatlimab and nivolumab complements proven benefit in metastatic setting

<table>
<thead>
<tr>
<th></th>
<th>pCR, n/N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nivo(^1)</td>
<td>3/12 (25)</td>
</tr>
<tr>
<td>Nivo+Rela(^2)</td>
<td>17/30 (57)</td>
</tr>
</tbody>
</table>

Combination relatlimab and nivolumab yields a high pCR rate and durable clinical benefit in resectable melanoma patients

Favorable safety profile

Opdualag: Potential therapy option for ~21K adjuvant patients vs ~13K 1L metastatic patients in the U.S.\(^3\)

RELATIVITY-098 Phase 3 ongoing: Data expected in 2026

---

MSS CRC: Combination benefit where PD-1 alone has not shown activity

Patient with recurrent, metastatic MSS rectal adenocarcinoma after 3 lines of treatment in the metastatic setting

Baseline 3 months into treatment 9 months into treatment

Partial Response (-38% decrease) in target lesions for 11+ months

RELATIVITY-123 Phase 3 ongoing:
Opdualag vs regorafenib or TAS-102 in later lines of metastatic colorectal cancer
Data expected in 2025

Images courtesy of Dr. Eric Christenson and Dr. Dung Le, Johns Hopkins University
Ongoing Phase 2 studies to inform Phase 3 program

Key takeaways from Phase 2 studies inform and potentially de-risk the Phase 3 program

1. NCT05337137; 2. NCT04967615; 3. NCT04623775
BMS-986442: Differentiated TIGIT & CD96 bispecific antibody in Oncology

Antagonizes TIGIT & CD96 binding to CD155

- Dual TIGIT & CD96 antagonist
- Fc selected to enhance tumor-reactive T cell responses

MoA: Drives T & NK cell anti-tumor immunity

- APC: T-Cell Response
  (Bispecific vs monospecific(s))

Program Overview: Licensed from Agenus

- Phase 1 single-agent trial complete
- Phase 1/2: Evaluating combinations (e.g., PD-1 ± chemotherapy) in dose escalation with data anticipated next year
- Positive data enables clinical development acceleration
- Initial tumors of interest: NSCLC & Gastric cancer

- CD96 & TIGIT are complementary targets in the same pathway & negatively regulate T & NK cell function in the tumor microenvironment\(^1,2,3\)
- BMS-986442 potentially enhances the quality & magnitude of T cell responses (vs TIGIT & CD96 monospecific antibodies) through dual inhibition on APC or tumor cells\(^4\)

---


Not for Product Promotional Use
Dual DGKα/ζ inhibitor builds on our depth in Oncology to potentially deliver a transformational oral CPI

**Transformational potential**
First-in-class, oral therapy as a T cell checkpoint inhibitor (CPI) as monotherapy or in combination with approved CPIs

**Causal human biology**
Translational insights from IO-refractory patients demonstrates mechanisms of resistance related to low antigenicity, lack of co-stimulation, and T cell anergy.

**Matching modality to mechanism**
A dual alpha/zeta inhibitor sensitizes CPI-resistant pre-clinical models through CD8 priming and clonal expansion, leading to tumor cell killing in combination with anti-PD1 and anti-CTLA4 therapies

**Path to clinical proof-of-concept**

<table>
<thead>
<tr>
<th>IO Resistance Mechanisms</th>
<th>DGKi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low TMB</td>
<td>✓</td>
</tr>
<tr>
<td>Low antigenicity</td>
<td>✓</td>
</tr>
<tr>
<td>Low MHCI</td>
<td>✓</td>
</tr>
<tr>
<td>Lack of co-stimulation</td>
<td>✓</td>
</tr>
<tr>
<td>T cell anergy</td>
<td>✓</td>
</tr>
</tbody>
</table>
Effective & tolerable treatment options needed in metastatic castrate resistant prostate cancer (mCRPC)

High unmet need remains in prostate cancer:
- Expected U.S. mortality is ~35K men in 2023
- 5-year OS decreases from >97% to ~32.5% in the localized vs metastatic setting

<table>
<thead>
<tr>
<th>Current SOC - NHT²</th>
<th>AR LDD</th>
</tr>
</thead>
<tbody>
<tr>
<td>• AR is a key driver of prostate cancer and AR-targeted therapies remain current SoC</td>
<td>• AR LDD induces irreversible AR degradation in a catalytic manner leading to deeper, more potent AR inhibition</td>
</tr>
<tr>
<td>• Traditional AR antagonists (e.g., enzalutamide) inhibit AR in a reversible manner</td>
<td>• Potentially paradigm-shifting MoA overcomes resistance mechanisms to NHT including AR WT amplification and mutations</td>
</tr>
<tr>
<td>• This AR inhibition is overcome by upregulation of wildtype (WT) or mutation of AR in cancer cells, leading to resistance:</td>
<td>— Preclinical models demonstrated activity in both settings</td>
</tr>
<tr>
<td></td>
<td>• Potential to improve efficacy, safety, &amp; tolerability in the post-NHT setting</td>
</tr>
<tr>
<td>— AR WT amplification (~50%)³</td>
<td></td>
</tr>
<tr>
<td>— AR mutations (~15-20%)³</td>
<td></td>
</tr>
<tr>
<td>• Post-NHT progression, limited options for patients (e.g., chemo)</td>
<td></td>
</tr>
</tbody>
</table>

AR LDD phase 1 design in 1L & 2L mCRPC

Open label¹: Assess the safety, tolerability and preliminary efficacy

Key eligibility criteria
- Histologically or cytologically confirmed adenocarcinoma of the prostate
- Progressed on ADT and ≥ 1 prior secondary hormonal therapy approved for CRPC
- ECOG performance status (PS) 0 or 1
- Dose escalation/Dose expansion

Primary endpoint
- Safety and tolerability

Key secondary endpoints:
- Confirmed Prostate Specific Antigen (PSA) decline of ≥ 50% from baseline (PSA50)
- Objective soft tissue response (CR or PR), DoR, rPFS, PSA PFS

Part A
Dose escalation

Part B
Dose expansion to optimize RP2D

Data anticipated in 2024

¹. NCT04428788
AR LDD demonstrates on target AR degradation in tumor biopsy

**Patient Example**

**Screening**

AR protein is highly expressed in tumor cells

**On Treatment**

AR protein levels degraded with AR LDD after one cycle

Source: Images from BMS internal database
Confirmation of mechanism of action of AR LDD from first-in-human study

69 yr old male with mCRPC since 2022¹

Prior Tx: ADT, enzalutamide, avelumab + talazoparib

Pt entered study with AR amplification, BRCA2 mutation, rising PSA and progressive soft tissue disease

Treated with AR LDD; responded rapidly with PSA90*

*Observed PSA decreases in this patient only serve to illustrate MoA and are not intended to represent expected outcomes

¹Patient case from BMS internal database
AR LDD: Opportunity to move into pivotal studies in next 18 months

Next Steps

Phase 1 data across mutant/wildtype subgroups to be presented at a medical congress in 2024

Discuss **pivotal study options** with health authorities

Consider expanding into **hormone sensitive** indications

Explore **novel combinations** to potentially enhance efficacy or synergy
Extending IO leadership while diversifying beyond IO

Extending leadership in IO

- **Subcutaneous nivolumab**: Potential to benefit patients into early 2030s with data anticipated this year
- **Opdualag**: 3rd approved IO agent; Approved in 1L melanoma; Phase 3 studies in adjuvant melanoma and mCRC ongoing
  - Ongoing Phase 2 studies in HCC and lung to inform Phase 3 program

Select next-gen IO

- **BMS-986442**: Differentiated TIGIT bispecific antibody targeting both TIGIT and CD96
- **DGK inhibitor**: Potential transformational, oral dual inhibitor in solid tumors

Diversifying beyond IO

- **AR LDD**: Significant opportunity in mCRPC with data expected early next year; initiating pivotal trial in next 18 months
- **repotrectinib**: Potential best-in-class, next generation ROS1/NTRK inhibitor; PDUFA November 27, 2023

Addressing oncologic diseases impacting 1.2M+ patients

---

1. 2023 estimates from Decision Resource Group & BMS Internal Analysis across indications in the U.S. & EU5
Program will reconvene following a short break

(10 min)
Cardiovascular
# Opportunity to develop medicines in important Cardiovascular indications

<table>
<thead>
<tr>
<th>Asset</th>
<th>Approved</th>
<th>Registrational†</th>
<th>Exploratory/PoC Studies†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Camzyos</td>
<td>Obstructive Hypertrophic Cardiomyopathy</td>
<td>Non-obstructive Hypertrophic Cardiomyopathy</td>
<td>-</td>
</tr>
<tr>
<td>milvexian</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MYK-224</td>
<td>-</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>danicamtiv</td>
<td>-</td>
<td></td>
<td>Dilated cardiomyopathy</td>
</tr>
</tbody>
</table>

† ongoing or initiating 2023/2024
Milvexian: Opportunity to expand anticoagulation beyond FXa to benefit millions of patients

Opportunity to address multiple thrombotic conditions of high unmet need

- Anti-platelets
  - SSP
  - ACS
- Factor Xa
  - AF

Target profile: Efficacy comparable or better to FXa with better bleeding profile

- ~7.5M patients\(^2\) in U.S. with thrombotic diseases need treatment

- Robust phase 2 program has demonstrated a differentiated anticoagulant profile

- LIBREXIA program is the largest and most comprehensive phase 3 registrational program ongoing (SSP, ACS & AF)

- U.S. FDA granted Fast Track Designation to all 3 indications ongoing in Phase 3 studies

---

1. Current standard of care for indication(s); 2. Decision Resource Group, BMS Internal Analysis

---

Cardiovascular | milvexian | Camzyos | MYK-224
Our framework reinforces confidence in milvexian as a next-generation anti-thrombotic

### Transformational potential
Oral anti-coagulant with a potential for comparable/better efficacy with reduced bleed risk to a broader range of patients

### Matching modality to mechanism
Milvexian has high affinity and specificity for FXIa, high oral bioavailability and demonstrates a wide therapeutic index in preclinical models of thrombosis

### Causal human biology
Congenital FXI-deficient patients:
- Lower risk for venous thromboembolism & ischemic strokes
- Spontaneous bleeding is uncommon

<table>
<thead>
<tr>
<th>Risk of CV events lower by</th>
<th>Risk of VTE lower by</th>
</tr>
</thead>
<tbody>
<tr>
<td>48%</td>
<td>43%</td>
</tr>
<tr>
<td>In patients with mild deficiency HR 0.52</td>
<td>In patients with moderate-to-severe deficiency HR 0.57</td>
</tr>
<tr>
<td>61%</td>
<td>61%</td>
</tr>
<tr>
<td>In patients with mild deficiency HR 0.39</td>
<td>In patients with moderate-to-severe deficiency</td>
</tr>
<tr>
<td><strong>No VTE events</strong></td>
<td></td>
</tr>
</tbody>
</table>

### Path to clinical proof-of-concept
- Human genetic data
- Epidemiologic observations
- Pre-clinical models
- Phase 2 studies

---

TKR study demonstrates a differentiated monotherapy profile: Supports moving into AF

Monotherapy

AXIOMATIC-TKR study¹

<table>
<thead>
<tr>
<th>Incidence Rate (% / 95% CI)</th>
<th>All VTE + all death</th>
<th>Major bleeding</th>
<th>CRNM</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 mg QD</td>
<td>0.014</td>
<td>0.214</td>
<td>0.25</td>
</tr>
<tr>
<td>25 mg BID</td>
<td>0</td>
<td>0.209</td>
<td>0.236</td>
</tr>
<tr>
<td>50 mg BID</td>
<td>0.014</td>
<td>0.113</td>
<td>0</td>
</tr>
<tr>
<td>100 mg BID</td>
<td>0.007</td>
<td>0.09</td>
<td>0</td>
</tr>
<tr>
<td>200 mg BID</td>
<td>0.007</td>
<td>0.076</td>
<td>0</td>
</tr>
<tr>
<td>25 mg QD</td>
<td>0</td>
<td>0</td>
<td>0.265</td>
</tr>
<tr>
<td>50 mg QD</td>
<td>0.013</td>
<td>0.09</td>
<td>0</td>
</tr>
<tr>
<td>200 mg QD</td>
<td>0.007</td>
<td>0</td>
<td>0.007</td>
</tr>
</tbody>
</table>

Milvexian has potential to offer **comparable/better efficacy with reduced bleed risk to a broader range of patients**

Data from the SSP Phase 2 study gives us confidence to move to Phase 3 in combination with anti-platelet treatments

Combination Therapy

AXIOMATIC-SSP study¹

<table>
<thead>
<tr>
<th>Dose</th>
<th>Incidence rate (%)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>5.5%</td>
<td>4.6%</td>
</tr>
<tr>
<td>25 mg QD</td>
<td>0.3%</td>
<td>0%</td>
</tr>
<tr>
<td>25 mg BID</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>50 mg BID</td>
<td>4.0%</td>
<td>3.5%</td>
</tr>
<tr>
<td>100 mg BID</td>
<td>0.9%</td>
<td>0%</td>
</tr>
<tr>
<td>200 mg BID</td>
<td>0%</td>
<td>0.3%</td>
</tr>
</tbody>
</table>

- 30% RRR versus placebo

Symptomatic ischemic stroke
Fatal bleed & Symptomatic intracranial hemorrhage

Clinically meaningful (~30%) reduction in ischemic stroke (primary endpoint for Phase 3 SSP) across several fold dose range

Efficacy

• No fatal bleeding
• No signal for increase in intracranial bleeds
• No apparent dose response in bleeding

Safety

Dose Response

• Dose response in ischemic stroke with early plateau at 25mg BID
• Phase 3 dose 25mg BID

Phase 2 data supports Phase 3 studies in SSP and ACS

1. Sharma et al, ESC 2022
SSP data provides proof-of-concept in ACS

Acute Coronary Syndrome Unmet Need
- Risk of recurrent CV events remains high despite dual antiplatelet therapy (5-10% annually)\(^1,2\)
- Current treatments (antiplatelets & anticoagulants) decrease CV events, but increase risk of major bleeding
- 900K\(^3\) patients diagnosed in the U.S.

Scientific rationale for milvexian in ACS

Ischemic stroke and ACS share similar underlying pathophysiology and treatment

FXa on top of dual antiplatelet shows efficacy but with excess bleeding

In AXIOMATIC-SSP, milvexian demonstrated efficacy in reducing recurrent ischemic stroke with no increase in severe bleeding vs. placebo

Phase 3 study in ACS underway

---

Confidence in profile supports three parallel Phase 3 trials in SSP, ACS, and AF

<table>
<thead>
<tr>
<th>Indication</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Projected Data Readout</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secondary Stroke Prevention</td>
<td>LIBREXIA-STROKE¹ (Dose: 25mg BID)</td>
<td></td>
<td></td>
<td>2026</td>
</tr>
<tr>
<td>Acute Coronary Syndrome</td>
<td>LIBREXIA-ACS² (Dose: 25mg BID)</td>
<td></td>
<td></td>
<td>2026</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>LIBREXIA-AF³</td>
<td></td>
<td></td>
<td>2027</td>
</tr>
</tbody>
</table>

The LIBREXIA Phase 3 studies represent the largest, most comprehensive program for a Factor XIa inhibitor

U.S. FDA granted **Fast Track designation** to all 3 indications

---

¹ NCT05702034; ² NCT05754957; ³ NCT05757869
Expanding myosin inhibitor franchise in HCM and HFpEF

**Today**

Strong launch momentum enabling more oHCM patients to benefit from Camzyos

**Medium-Term**

Expansion into nHCM & opportunity to expand diagnosis rates with Artificial Intelligence supported technology

**Long-Term**

Potential opportunity for myosin inhibitors in HFpEF

Continued evolution of data suggests disease modifying ability of Camzyos
**oHCM Patient Case (Electrocardiogram)**

**Before Camzyos Treatment**

**6 Months of Camzyos Treatment**

Normalization trend in the ECG after 6 months of treatment with Camzyos

Source: Courtesy of Dr. Matt Martinez
oHCM Patient Case (Echocardiogram)

Source: Courtesy of Dr. Matt Martinez
Camzyos: Phase 3 trial in nHCM underway

Patients with symptomatic nHCM (NYHA Class II or III)

Key Inclusion:
- Diagnosis of HCM per ACC/AHA and ESC guidelines
- Peak LVOT < 30 mmHg at rest / < 50 mm Hg with provocation
- NYHA Class II or III

Key Exclusion:
- Known infiltrative or storage disorder that mimics nHCM
- H/o unexplained syncope within 6 months prior to screening
- H/o SVT within 6 months prior to screening

Assessment of Primary Endpoints at Week 48:
- Exercise Capacity (pVO2)
- PRO (KCCQ)

Data expected in 2025

1. NCT05582395
Significant unmet need remains in HFpEF

- HF affects ~6.8 million individuals in the US\(^1\)
- HF is classified by clinical signs & symptoms as well as the heart’s ability to eject blood
- 50% of HF patient have HFpEF which is a heterogenous disease contributed to by several comorbidities and/or specific causes, e.g., cardiomyopathy\(^2\)
- Patients with HFpEF typically present with dyspnea and evidence of congestion. There may be evidence of diastolic dysfunction, ventricular stiffening and hypertrophy

HFpEF, heart failure with preserved ejection fraction; SGLT-2i, sodium-glucose cotransporter-2 inhibitors.
1. Heidenreich PA et al. J Am Coll Cardiol. 2022
2. Solomon SD et al. NEJM. 2022
Emerging data suggests a potential role for MYK-224 in HFpEF

**MYK-224 profile as a cardiac myosin inhibitor**

- Pre-clinical animal models show similar exposure-response with mavacamten
  
- MYK-224 oHCM Phase 2 MERCUTIO trial is ongoing to confirm exposure-response similarity in humans

**Role of cardiac myosin inhibitor in HFpEF**

- Encouraging interim observations from mavacamten Phase 2a EMBARK suggests myosin inhibitor benefits in HFpEF

- Leveraging entirety of cardiac myosin inhibitor data and experience to support starting dose for MYK-224 in HFpEF

Initiate MYK-224 PoC in HFpEF in 2023/2024

---

1. Preclinical model only tested in 'prevention', not 'treatment' mode
Opportunity for sustained leadership in Cardiovascular

Successful history of developing leading CV medicines (e.g., Plavix & Eliquis)

Extending our leadership in thrombotic diseases

- **Milvexian**: Robust Phase 2 program supported differentiated clinical profile; Phase 3 studies in SSP, ACS and AF underway

Potential opportunity for myosin inhibitors in cardiomyopathies and heart failure

- **Camzyos**: Expansion into nHCM with Ph3 trial underway
- **MYK-224**: Initiating PoC trial in HfPEF based on supportive data

Addressing cardiovascular diseases impacting 17M+1 patients

---

1. Source: 2023 estimates from Decision Resource Group & BMS Internal Analysis across indications in the U.S. & EU5
Neuroscience
Building an exciting portfolio in neurodegenerative and neuroinflammatory conditions

**Neurodegeneration/Neuroinflammation**

- ALS
- Alzheimer’s Disease
- Huntington’s Disease
- Multiple Sclerosis
- Parkinson’s Disease

**In Development**

- Anti-MTBR-Tau mAb
- eIF2B Activator
- TYK2i-CNS
- FAAH-MGLL

**22 active programs in discovery**

**Powered by internal & external innovation**

1. TYK2i-CNS to transition into clinic soon
BMS-986446 (PRX005): Potential best-in-class antibody to slow or halt the progression of Alzheimer’s Disease

- The propagation of Tau pathology as Alzheimer’s Disease (AD) progresses is thought to be mediated by Tau “seeds” containing the MTBR region of tau\(^1\)
- Tau, not Ab, deposition correlates with age of AD onset, disease duration, and cognitive impairment\(^1\)

A Tau fragment (MTBR-Tau 243) has recently been shown to correlate well with tau accumulation as measured by Tau-PET imaging and cognitive impairment\(^3\)

BMS-986446 targets MTBR-Tau 243 and binds with high affinity to both the 3R and 4R isoforms of tau\(^4,5\)

BMS-986446: Preclinical models showed significant reduction of intraneuronal tau pathology and protection against behavioral deficit in a tau transgenic mouse model in vivo and complete blockade of neuronal tau internalization in vitro\(^5\)

---

Phase 1 data supports rapidly moving BMS-986446 into Phase 2

### Phase 1 Findings

- Safe and well-tolerated in Phase 1 across 3 dose cohorts; no deaths or SAEs observed in healthy participants
- Demonstrated dose-proportional anti-tau concentrations in plasma with CNS penetration in healthy participants
- No persistent anti-tau-induced anti-drug antibodies observed
- Robust exposure in the CSF at 1-month predicts substantial target engagement in CNS

### PoC in Alzheimer’s Disease to initiate in 1H 2024

- Double-blind treatment period
  - Placebo
  - BMS-986446 Dose 1
  - BMS-986446 Dose 2
- Optional long-term extension and follow-up

---

1. Martenyi, et al AAIC 2023 Poster 74181
eIF2B Activator (BMS-986419): Potential across a range of neurodegenerative conditions

Misfolded protein accumulation & evidence of ISR activation is present in multiple neurological conditions

- Stressed cells that develop a chronically activated ISR accumulate misfolded proteins that impair cell functions and can lead to cell death
- The eIF2 complex is an ISR “master regulator” that becomes dysfunctional in chronic disease
- BMS-986419 binds to a subunit in the eIF2 complex (eIF2B) restoring normal ISR function, protein clearance and cellular homeostasis

BMS-986419: Safe and well-tolerated in Phase 1 SAD/MAD study potential opportunity as monotherapy or combinations

Phase 2 study in ALS initiating in 2024

ALS: Rapidly progressing & fatal neurodegenerative disease caused by death of motor neurons:
- Survival is typically only 2-5 years from symptom onset
- ~39k diagnosed prevalent patients in the U.S.
- Limited treatment options

Re-establishing Neuroscience pipeline

Building a diverse pipeline across an array of neurodegenerative & neuroinflammation diseases

Anti-MTBR-Tau moving into POC next year in Alzheimer’s Disease

eIF2B is moving into a Phase 2 trial in ALS

TYK2i-CNS to transition into clinic soon targeting Multiple Sclerosis
We are driving improved operational efficiency to accelerate speed to market

Further enabled through Digital Innovation & AI

- Early Phase Development (I/II)
- Phase III
- Submission Activities

- Optimized study design
- World-class trial operations capabilities, ~40 countries footprint, robust site & patient engagement strategies
- Near-time data review and submissions

Active future timeline
Reduction from previous timeline
Implementing innovative AI/Digital tools to accelerate our R&D productivity

<table>
<thead>
<tr>
<th>What</th>
<th>How</th>
</tr>
</thead>
<tbody>
<tr>
<td>Significantly more powerful hypothesis generation</td>
<td>Building predictive disease models using a vast proprietary data factory</td>
</tr>
<tr>
<td>Digital trial design optimization</td>
<td>Powerful statistical simulation suite that aggregate millions of data points to enable decisions around effect-size, power, patient-selection, timelines &amp; cost</td>
</tr>
<tr>
<td>Enhancing clinical trial operations</td>
<td>Real-time site selection based upon protocol required patient characteristics</td>
</tr>
<tr>
<td>Rapid data interpretation and reporting</td>
<td>Effective automation and visualization technologies to enable timely data insights and clinical trial reporting</td>
</tr>
</tbody>
</table>
Important updates today

### Expanding Currently Launched Products

- **Sotyktu:**
  - Impressive Ph2 SLE data supports Ph3 programs in SLE & SjS
- **Abecma:**
  - KarMMa-3 under regulatory review in triple class exposed population
  - KarMMa-9 registrational trial in post-transplant MM initiating this year
- **Subcutaneous nivolumab:**
  - Potential to benefit patients into early 2030’s with data anticipated this year
- **Camzyos:**
  - Data suggests myosin inhibitors remodel the heart

### New Wave of NME

- **LPA, Antagonist:**
  - Demonstrated compelling Ph2 PPF data and Ph3 studies initiating
- **CD19 NEX T:**
  - Ph1 study initiated in severe, refractory lupus with promise to reset the immune system & expanding into other immunologic diseases
- **BET Inhibitor (BMS-986158):**
  - Proof-of-concept data expected early next year
- **GPRC5D CAR T:**
  - Differentiated profile addressing unmet need post-BCMA targeting treatment; initiating pivotal trial next year
- **alnuctamab:**
  - Initiating Ph3 trial in 2024 in MM
- **iberdomide/mezigdomide:**
  - Ph3 data expected in 2026
- **golcadomide:**
  - Initiating Ph3 trial in 2024 in 1L LBCL
- **AR LDD:**
  - Significant opportunity in mCRPC with data expected early next year
  - Initiating pivotal trial in the next 18 months
- **milvexian:**
  - Compelling rationale for Ph3 programs

### Early Assets to Watch

- **BCMA x GPRC5D:**
  - Entering into POC soon
- **BCL6 LDD:**
  - Novel oral degrader in lymphomas
- **TIGIT Bispecific:**
  - Differentiated IO; targeting TIGIT & CD96
- **DGK inhibitor:**
  - Potential transformational oral, dual inhibitor
- **Anti-CCR8:**
  - Treg depleting mAb therapy with broad combination potential
- **Advancing Neuro PoC trials in 2024:**
  - Anti-MTBR-Tau in Alzheimer’s Disease
  - eIF2B Activator in ALS
- **TYK2i CNS (pre-clinical):**
  - Moving into clinic soon, targeting MS
- **MYK-224:**
  - Progressing into HFpEF

### Productivity

- Increasing registrational assets from 6 to 12 in next 18 months
- Increase INDs to at ~10 per year
- Increase to ~20% PoS from FIH to approval
- Achieve median ~6.5 years from FIH to approval
Conclusion

Chris Boerner, PhD
EVP, Chief Operating Officer
CEO, effective Nov. 1, 2023
Numerous levers to drive long-term growth

- Extended durability of our IO business with subcutaneous nivolumab and Opdualag
- Increasingly de-risked the New Product Portfolio
- Registrational portfolio increasing from 6 to 12 new assets over the next 18 months
- Developing medicines in rapidly growing markets with significant commercial opportunities
- Leading positions with differentiated platforms in Cell Therapy and Targeted Protein Degradation
- Strategic optionality from Business Development

Clearly establish BMS as an R&D leader by the end of the decade
## Extended durability of our I-O business

<table>
<thead>
<tr>
<th>Today</th>
<th>Opportunity</th>
</tr>
</thead>
</table>
| **Subcutaneous nivolumab** | • Potential to benefit patients into early years of next decade  
  • Roughly 65-75% of IV US revenue potentially on-label at launch |
| • Benefits patients into the next decade in 1L melanoma  
  • Potential to benefit beyond melanoma (adj. melanoma, MSS CRC, lung, and HCC) |
New product portfolio significantly de-risked with important catalysts ahead

**CAMZYOS**
- Milestones Achieved: Camzyos oHCM
- Future Catalysts:
  - Camzyos nHCM

**Opdualag**
- Milestones Achieved:
  - Opdualag 1L Mel FDC
- Future Catalysts:
  - Opdualag 1L NSCLC
  - Opdualag Adj. Mel
  - Opdualag 2L+ MSS CRC

**SOTYKTU**
- Milestones Achieved: Sotyktu PsO
- Future Catalysts:
  - Sotyktu PsA
  - Sotyktu SLE

**Reblozyl**
- Milestones Achieved:
  - Reblozyl 2L TD MDS
  - Reblozyl 1L MDS
- Future Catalysts:
  - Reblozyl MF

**Breyanzi**
- Milestones Achieved:
  - Breyanzi 3L+ LBCL
- Future Catalysts:
  - Breyanzi 3L+ CLL
  - Breyanzi 3L+ iNHL

**Zeposia**
- Milestones Achieved:
  - Zeposia MS
  - Zeposia UC
- Future Catalysts:
  - Zeposia CD

**Abecma**
- Milestones Achieved:
  - Abecma 5L+
  - Abecma 3-5L

---

$25B+$
Non-Risk Adjusted*

- CAMZYOS: $4B+
- Opdualag: $4B+
- SOTYKTU: $4B+
- Reblozyl: $4B+
- Zeposia: $4B+
- Abecma: $4B+
- Breyanzi: $4B+
- Zeposia: $3B+
- Abecma: $3B+
- Abecma: $3B+
- Abecma: $1B+

2030

*Non-risk adjusted revenue potential, subject to positive registrational trials and health authority approval
Note: Onureg & Inrebic <$1B NRA
Financial projections may contain non promoted sales, BMS promotes only according to label.

Not for Product Promotional Use
We are increasing registrational portfolio from 6 to 12 potentially first-in-class/best-in-class assets over the next 18 months.

<table>
<thead>
<tr>
<th>In registrational studies</th>
<th>Registrational studies pending</th>
</tr>
</thead>
<tbody>
<tr>
<td>mezigdomide</td>
<td>CD19 NEX T</td>
</tr>
<tr>
<td>repotrectinib*</td>
<td>BET Inhibitor (BMS-986158)</td>
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<td>iberdomide</td>
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<tr>
<td>cendakimab</td>
<td>alnuctamab</td>
</tr>
<tr>
<td>milvexian</td>
<td>golcudomide</td>
</tr>
<tr>
<td>LPA\textsubscript{1} Antagonist</td>
<td>AR LDD</td>
</tr>
</tbody>
</table>

*Filed, U.S. PDUFA 11/27/2023
Developing medicines in rapidly growing markets with significant commercial opportunities

<table>
<thead>
<tr>
<th>Category</th>
<th>2023</th>
<th>2030</th>
</tr>
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<td>Neuroscience</td>
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*CV markets impacted by generic entry in Atrial Fibrillation
Source: EvaluatePharma estimates
Building a competitive advantage in Cell Therapy

Manufacturing capacity is expanding

*Plan to deliver top-tier supply chain, manufacturing capacity & reliability:*

- Expanding drug product capacity
- Strengthening vector supply
- Increasing efficiency

Innovative pipeline is advancing

- Expanding to immunologic diseases
- Developing new targets
- Exploring innovative technologies e.g., dual binding CAR & allogeneic

Well-positioned at the center of the innovation ecosystem
Targeted Protein Degradation platform is poised for a step-change in productivity

Growing asset library
- Extensive number of potential INDs identified
- Opportunities across therapeutic areas

Industry-leading capabilities
- Significant experience applying preclinical, manufacturing, translational, AI/digital and clinical tools to optimize candidates

Engine expected to deliver approximately 4 INDs annually
Enhancing BMS leadership by the end of the decade

**Strong track record**
- 9 new products delivered since 2019
- 3 first-in-class medicines in 2022
- 20+ new indications & 45+ approvals across U.S., EU & Japan since 2020

**High quality pipeline**
- Registrational pipeline increasing to 12 new assets over the next 18 months
- Targeting high unmet need with growing commercial potential

**Improved productivity**
- Deliver ~10 INDs per year
- Increase to ~20% PoS from FIH to approval
- Achieve median of ~6.5 years from FIH to approval

Continual generation of new first-in-class or best-in-class medicines
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Term</th>
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<td>Six Minute Walk Test</td>
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<td>American College of Rheumatology</td>
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<td>ADC</td>
<td>Antibody-Drug Conjugate</td>
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<td>APC</td>
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<td>AR LDD</td>
<td>Androgen Receptor Ligand-Directed Degrader</td>
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<td>DPSS</td>
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<td>DLT</td>
<td>Dose-Limiting Toxicity</td>
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<td>DMPK</td>
<td>Drug Metabolism Pharmacokinetics</td>
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<td>EGE</td>
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<td>Flu/Cy</td>
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<td>UC</td>
<td>Ulcerative Colitis</td>
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<tr>
<td>VGPR</td>
<td>Very Good Partial Response</td>
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</table>
R&D efforts align with ESG values

Addressing areas of high unmet need

- 40+ disease areas studied, including several rare diseases
- Novel drugs with three first-in-class medicines launched in 2022
- Diverse modalities with 10 drug platforms (e.g., cell therapy)

Enhancing health equity and clinical trial diversity

Numerous initiatives related to ensuring clinical trial diversity:

- 58% of US clinical trial sites located in highly diverse communities
- Racial diverse participants at 22% (goal 20%) in 2022

Responsibly driving innovation to maximize impact

- “Green by design” principles and a green chemistry approach, reducing total waste generated
- “Green chemistry reviews” to identify opportunities for reducing safety and environmental impact

Representative examples, not exhaustive
# Changes to the Development Pipeline

<table>
<thead>
<tr>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
<th>Registrational Submissions</th>
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<td>New or Phase Transition</td>
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<tr>
<td>✫ BCL6 LDD in Lymphoma</td>
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<td>✫ LPA1 Antagonist in IPF</td>
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<td>✫ LPA1 Antagonist in PPF</td>
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<td>✫ obexelimab*# in IgG4-Related Disease</td>
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<td>Approvals (n=2)</td>
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<td>✫ NME 2</td>
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<td>• OPDIVO in Adj Melanoma (EU)</td>
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<td>✫ CD47xCD20</td>
<td>✫ HSP47</td>
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<td>• REBLOZYL in 1L MDS associated anemia (US)</td>
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<td>✫ GSPT1 CELMoD (CC-90009)</td>
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<tr>
<td>✫ RIPK1 Inhibitor</td>
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</tbody>
</table>

* Partner-run study; ✫ NME leading indication; # BMS territory

Data as of September 14th, 2023
Addressing high unmet medical need in Oncology & Hematology

**Oncology**

- **2023**
  - Lung $33B+
  - Bladder $5B+
  - H&N $3B+
  - Renal $9B+
  - Liver $3B+
  - Melanoma $9B+
  - Ovarian $3B+
  - CRC $6B+
  - GI $1B+
  - Prostate $14B+
  - **Total**: $85B+

- **2030**
  - Lung $61B+
  - Bladder $10B+
  - H&N $5B+
  - Renal $8B+
  - Liver $4B+
  - Melanoma $14B+
  - Ovarian $9B+
  - CRC $7B+
  - GI $4B+
  - Prostate $20B+
  - **Total**: $140B+

**Hematology**

- **2023**
  - CLL $7B+
  - AML $1B+
  - MF $2B+
  - MDS $1B+
  - cHL $2B+
  - MM $22B+
  - NHL $14B+
  - **Total**: $49B+

- **2030**
  - CLL $11B+
  - AML $10B+
  - MF $3B+
  - cHL $3B+
  - MDS $6B+
  - MM $31B+
  - NHL $27B+
  - **Total**: $90B+

Source: EvaluatePharma estimates; totals may not add due to rounding
Addressing high unmet medical need in Immunology, Cardiovascular & Neuroscience

**Immunology**
- Psoriasis $26B+
- Pulmonary Fibrosis $5B+
- Lupus $2B+
- Alopecia $0.6B+
- EoE $40M+
- SjS $19M+

**Cardiovascular**
- ACS $0.5B+
- Stroke $1.8B+
- HFpEF $8B+
- AF $19B+

**Neuroscience**
- MS $21B+
- ALS $1B+
- Alzheimer's $1B+

**2023**
- $60B+
- UC $7B+
- PsA $5B+
- CD $16B+

**2030**
- $75B+
- UC $10B+
- Pulmonary Fibrosis $2B+

*CV markets impacted by generic entry in Atrial Fibrillation

Source: EvaluatePharma estimates; totals may not add due to rounding.
Farletuzumab ecteribulin (FZEC): Novel folate receptor alpha (FRα) ADC

Overview

- FRα is a folate-binding protein that has limited expression on normal tissues and is overexpressed in malignant cells²
- FZEC binds to FRα on the surface of tumor cells, is internalized and cleaved to release the payload, eribulin
  - Eribulin inhibits microtubule growth resulting in cell death
- FZEC may potentially target tumors with heterogenous FRα expression through bystander effect of eribulin on nearby FRα-negative cells

Development plan

- PoC trials ongoing in NSQ NSCLC, ovarian & endometrial cancers
- In dose expansion to optimize the therapeutic index with data anticipated in 2024

MOA: Target delivery of differentiated payload, eribulin

High addressable population based on range of FR expression

1. In partnership with Eisai; 2. Level of overexpression may vary depending on tumor type

---

150
Repotrectinib: Potential Best-in-Class ROS1 Inhibitor in NSCLC

Highly Potent & Differentiated Small Molecule

<table>
<thead>
<tr>
<th>ROS1+ TKI-Naïve NSCLC; ORR (95% CI)</th>
<th>79% (67.6, 87.7)</th>
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</thead>
<tbody>
<tr>
<td>TKI-Pretreated Activity</td>
<td>✓ ORRs of 28-42% (n=100)</td>
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<tr>
<td>CNS Activity (ROS1+ NSCLC)</td>
<td>✓</td>
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<tr>
<td>ROS1+ TKI-Naïve NSCLC Durability</td>
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<tr>
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<td>• ≥12-month DOR: 83.1% (73.1, 93.2)</td>
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<td>• mDOR: 34.1 (25.6-NE)</td>
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<tr>
<td>PFS</td>
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<tr>
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<td>• ≥12-month PFS: 76.6% (66.2, 87.0)</td>
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<tr>
<td></td>
<td>• mPFS: 35.71 (27.40, NE)</td>
</tr>
</tbody>
</table>

Generally Well Tolerated Safety Profile

Source: Cho BC, et al. IASLC WCLC 2023

Clinically differentiated profile in NSCLC

Market Potential

ROS1 Prevalence: ~1.5% of NSCLC patients²

Existing ROS1 market: ~$500-$600M³

Opportunity to roughly **double** the ROS1 market & achieve best-in-class share based on:
- Longer duration of response
- Higher response rate
- Better safety / tolerability profile

U.S. PDUFA November 27, 2023
BMS-986288: A next generation CTLA-4 antibody

Overview

• CTLA-4: established MOA, with Yervoy approved across solid tumors
• Challenges (toxicity and patient selection) associated with targeting CTLA-4 have limited development
• BMS-986288 is a next-generation CTLA-4 designed to improve the benefit/risk:
  – NF (enhanced CD16 binding) biology increases immune priming via Fc engagement enhancing anti-tumor response
  – Improves safety profile with Probody® added to NF allowing for combinations and moving to earlier lines of therapy

Development plan

PoC trials in NSCLC & MSS CRC ongoing; data anticipated in 2024

MOA: A masked non-fucosylated anti-CTLA-4 antibody which improves immune priming and the safety profile
PsA: Sotyktu Phase 2 responses provide confidence for Phase 3

Unmet need: Effective, tolerable, oral options with a novel mechanism to address both skin and joint involvement

Phase 2 Primary Endpoint: ACR20 over time

week 16 primary endpoint: ACR20

All primary and key secondary endpoints were achieved in patients with active PsA

Treatment was well-tolerant with a safety profile consistent with prior studies

Phase 3 program ongoing

data anticipated 2024/2025

POETYK-PSA-1

• Active disease; biologic DMARD-naive
• ≥ 1 PsA-related hand and/or foot joint erosion on X-ray

Week 16 Primary endpoint: ACR20

Placebo  deucravacitinib 6 mg QD  LTE

R 1:1

deucravacitinib 6 mg QD

POETYK-PSA-2

• Active disease; biologic DMARD-naive OR TNF inhibitor experienced

Week 16 Primary endpoint: ACR20

Placebo  deucravacitinib 6 mg QD  LTE

R 3:3:1

deucravacitinib 6 mg QD  apremilast 30 mg BID


153
Established IBD presence with Zeposia in UC, with potential expansion to Crohn’s Disease

**Zeposia in IBD**

**Ulcerative Colitis**

Approved in the U.S. & EU

Zeposia provides UC patients with efficacy comparable to biologics, and a favorable safety profile in an oral medicine.

**Primary endpoints:**
- Induction studies: Week 12 clinical remission
- Maintenance study: Co primary @ Week 52 clinical remission and endoscopic response

**Crohn’s Disease**

Phase 3 YELLOWSTONE program ongoing

Maintenance study data anticipated 2026

### Study 3201

- **N = 400**
  - Zeposia
  - Placebo

### Study 3202

- **N = 400**
  - Zeposia
  - Placebo

Zeposia responders or remitters are re-randomized 1:1 to Zeposia or Placebo

- **12 wk induction study**
- **52 wk maintenance study**

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1. NCT03440372; 2. NCT03440385; 3. NCT03464097
Eosinophilic Esophagitis + Cendakimab

- Binds to IL-13 ligand
- Blocks IL-13 binding to both IL-13Ra1 & IL-13Ra2 subunits

EoE is a life altering disease affecting ~700k patients (combined U.S./EU5)

Potentially differentiated MoA addressing a significant unmet need for a highly efficacious treatment that improves both inflammation & fibrosis/remodeling

Co-primary (week 24):
- Change in dysphagia days
- Histologic response: eos ≤6/hpf

Key secondary (weeks 24 & 48):
- Histologic response: eos <15/hpf
- EREFS
- EoE-HSS
- mDSD composite score

Data anticipated in 2024

1. Source: Decision Resources Group; BMS Internal Analysis; 2. NCT04753697