Research & Development Day

September 14, 2023

Bristol Myers Squibb™

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

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

Evolving BMS R&D: World-class organization with increased focus on productivity & scientific leadership

Scientific Leadership

- Cardiovascular
- Hematology
- Oncology
- Immunology
- Neuroscience

Established leadership

Re-establishing

Top-tier productivity

- Increasing Probability of Success
- Increasing INDs
- Reducing Cycle Times

Capitalize on differentiated platforms

- Targeted Protein Degradation
- Cell Therapy
- Biotherapeutics
- Small Molecules

Retain & attract the best talent in the industry

- Enabled by a high-performance culture

Leveraging partnerships and AI/Digital Technologies

Build depth across our therapeutic areas

Oncology	Cardiovascular	Hematology	Immunology	Neuroscience
Extend IO leadership • SC nivolumab, Opdualag, & next generation assets	Deepen leadership in cardiomyopathies & heart failure	Extend leadership across the Multiple Myeloma treatment paradigm	Establish new standards of care in pulmonology	Build a diverse pipeline across neurodegenerative & neuroinflammation diseases
Diversification beyond IO	Expand expertise in thrombotic diseases	Broaden portfolio across leukemias, lymphomas and non-malignant hematologic diseases	Strengthen presence in dermatology, rheumatology, & gastrointestinal disorders Rapidly advance Cell Therapy into immunologic diseases	Advance promising clinical assets in Alzheimer's Disease & ALS

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

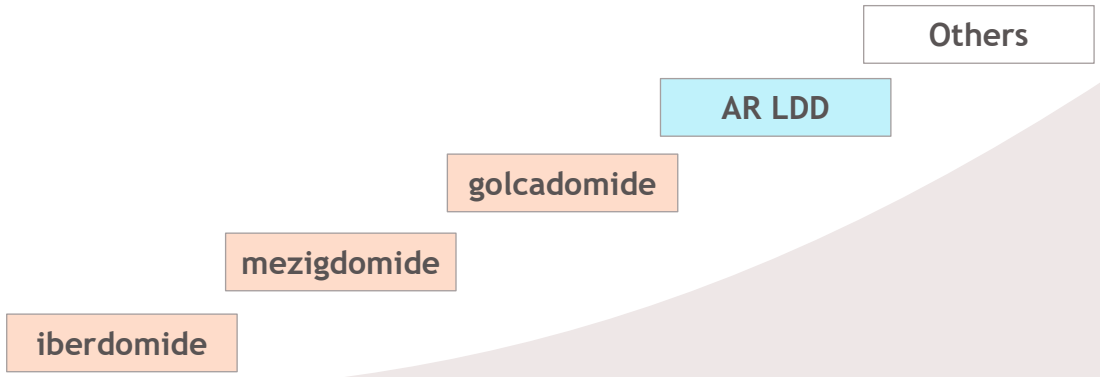
Opportunity for Targeted Protein Degradation

Positioned to deliver ~4 INDs annually

And expand targets to new therapeutic areas:

- ☑ Hematology
- ☑ Oncology
- ☐ Immunology
- ☐ Neuroscience

Waves of agents expected to launch over time:

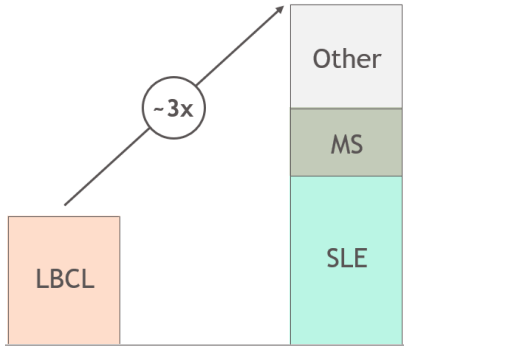


Building on Our Leadership in Cell Therapy

Today		Future	
Abecma (idecabtagene vicleucel)	Breyanzi (lisocabtagene maraleucel)	GPRC5D CAR T	CD19 NEX T
First-in-Class anti-BCMA	*Best-in-Class anti-CD19*	BCMA x GPRC5D dual binding CAR	Innovative Technologies (e.g., allogeneic)

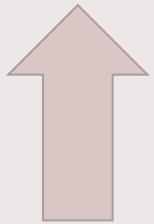
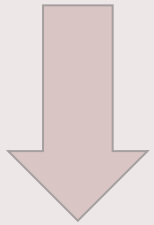
Expansion into Immunology: Starting with CD19 NEX T[1]

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

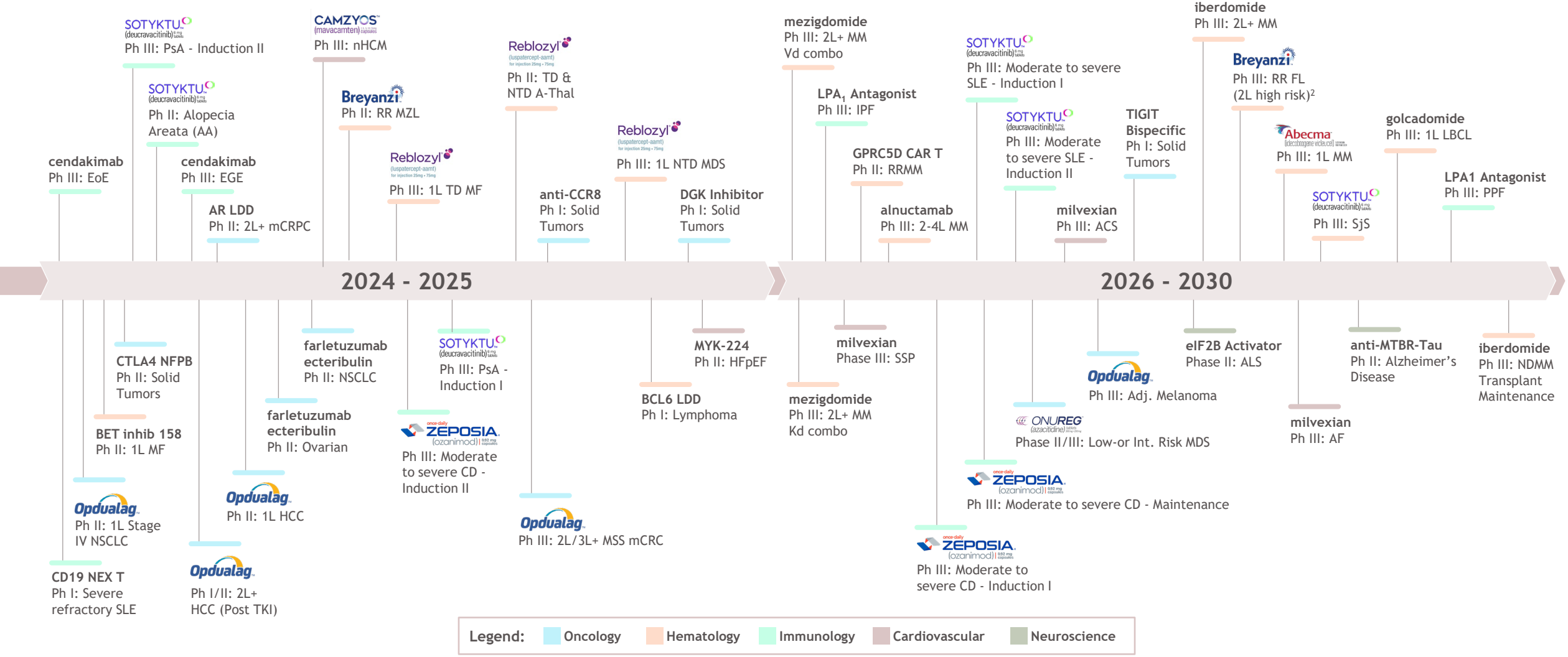


1. 2023 estimates from Decision Resource Group & BMS Internal Analysis; represents U.S. total diagnosed prevalence

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)

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

Oncology | Hematology | Immunology | Cardiovascular | Neuroscience

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

Early and Late Clinical Development

Global Development Operations

Global Regulatory Sciences

Global Biometrics & Data Sciences

Worldwide Patient Safety

Portfolio & Strategic Operations

Strategy & Capabilities

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

TEMPUS · FINNGEN · biobank uk · Open Targets · BROAD INSTITUTE

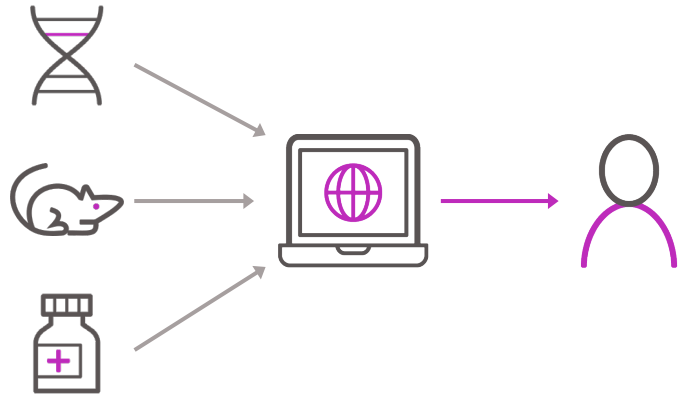
Matching modality to mechanism

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

insitro · Exscientia · SCHRÖDINGER · BigHat BIOSCIENCES

Path to clinical proof-of-concept

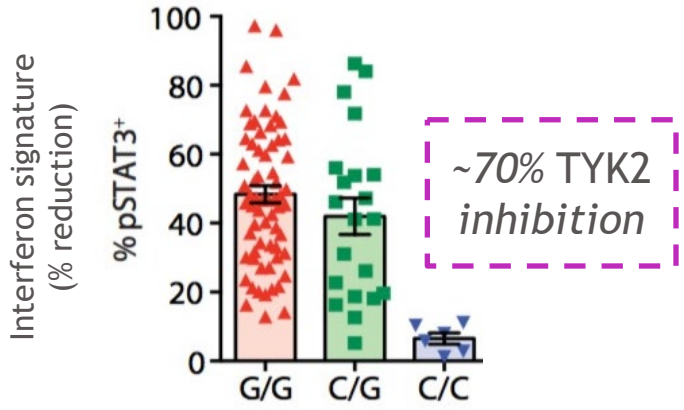
Technologies and diagnostics to enable mechanistic models for dose, schedule, and patient populations



Research framework is effective: *TYK2* genetics and SOTYKTU in immunologic diseases

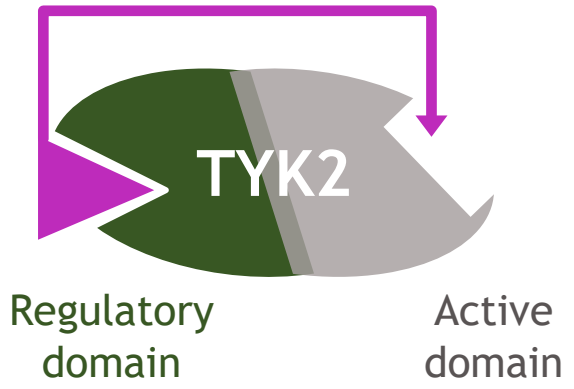
Causal human biology

Human genetics (*P1104A*): implicates TYK2 in multiple immunologic diseases



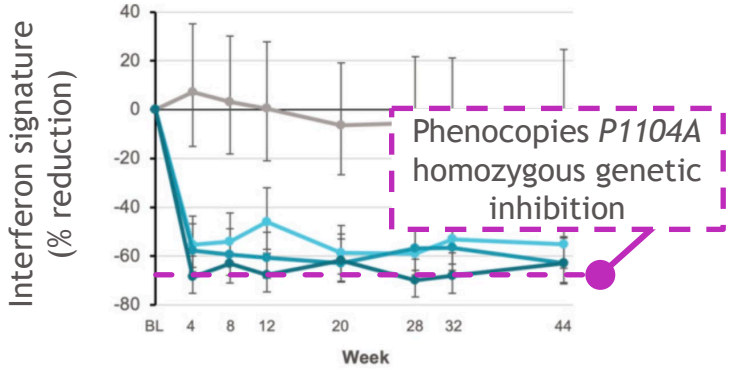
Matching modality to mechanism

Allosteric inhibitor: A highly selective small-molecule drug



Path to clinical proof-of-concept

Initially psoriasis: now systemic lupus erythematosus (SLE)



We now <u>consistently</u> apply this Research framework to all our programs to deliver transformational medicines with an increased probability of success in development

Sci Transl Med 2016 Nov; 8(363): 363ra149
PLoS One 2015 April; 10(4): e0122271
Arthritis Rheumatol. 2023 Feb; 75(2): 242-252.
Clin Transl Sci. 2023 Jan; 16(1): 151-164

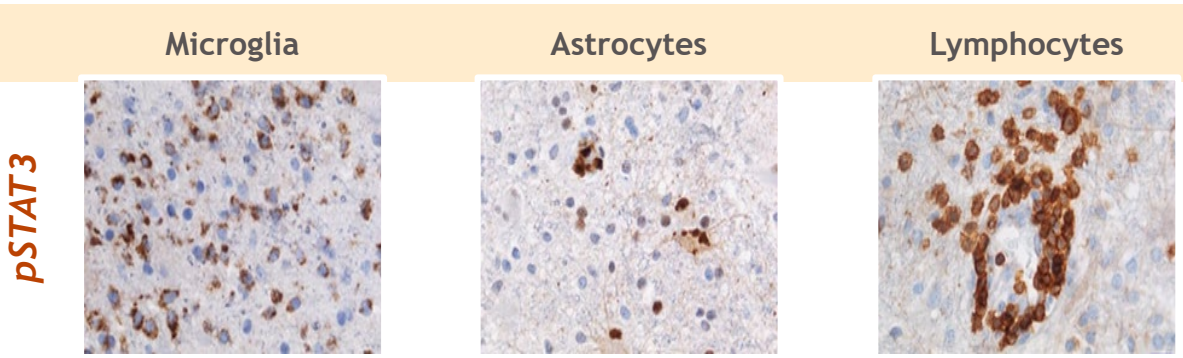
Research framework provides confidence in new programs: novel CNS penetrant TYK2 inhibitor for Multiple Sclerosis (MS)

Transformational potential

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

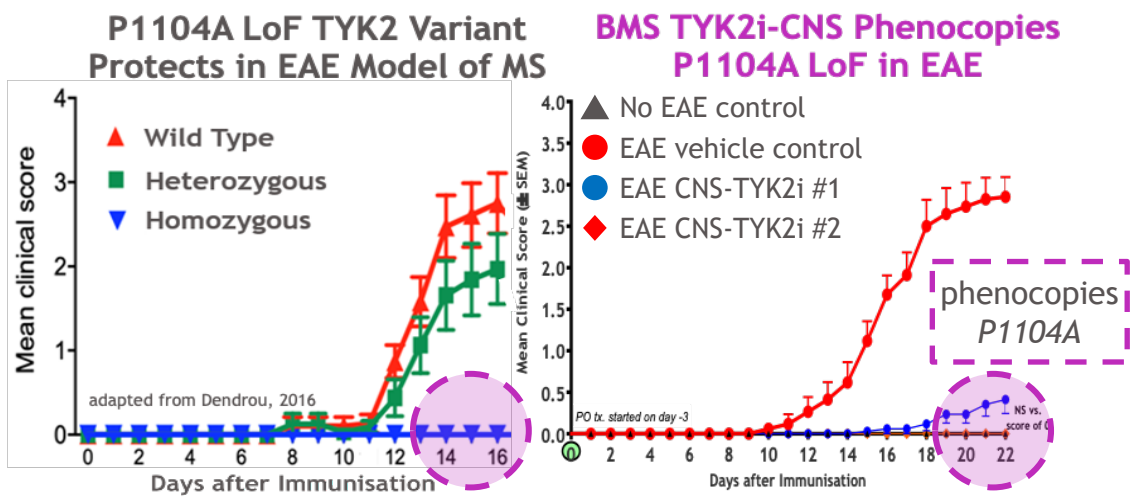
Causal human biology

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



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.

Sci Transl Med 2016 Nov; 8(363): 363ra149

Lu et al, J Neuropathol Exp Neurol 72:1135 (2013);
Dendrou et al:Sci Transl Med 2016 Nov 2;8(363):363ra149. doi: 10.1126/scitranslmed.aag1974

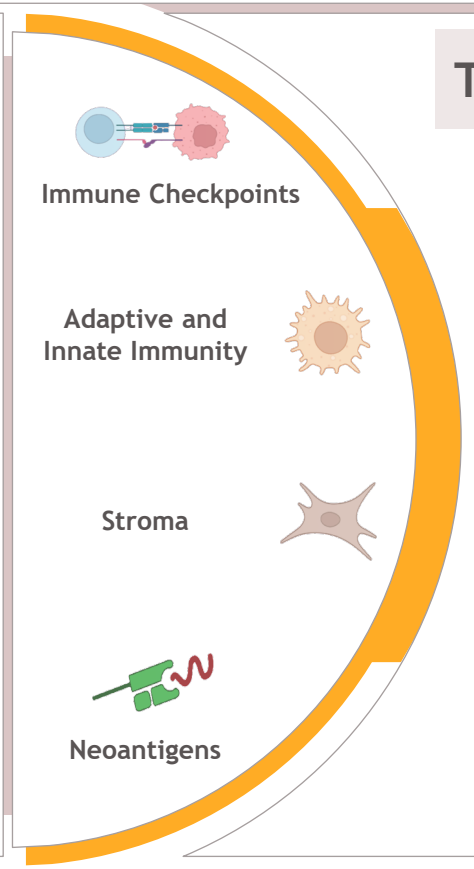
Research framework applied to Oncology builds on our scientific depth in immuno-oncology

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

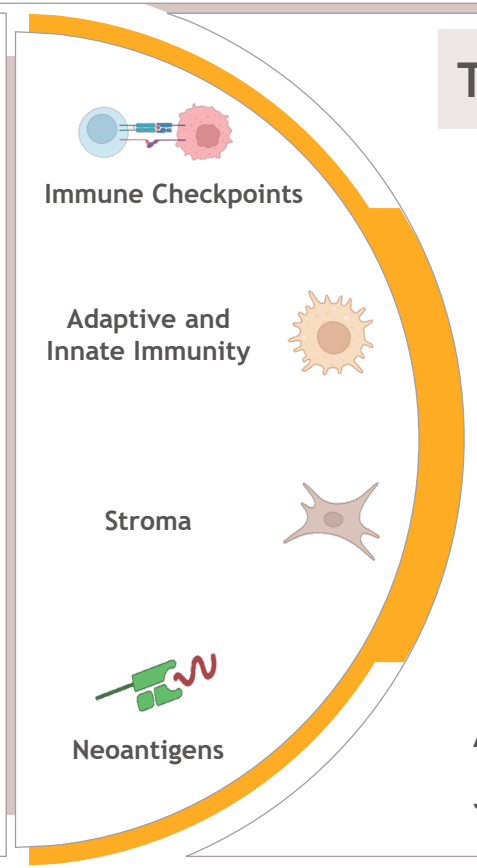
Research framework applied to Oncology builds on our scientific depth in immuno-oncology

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

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

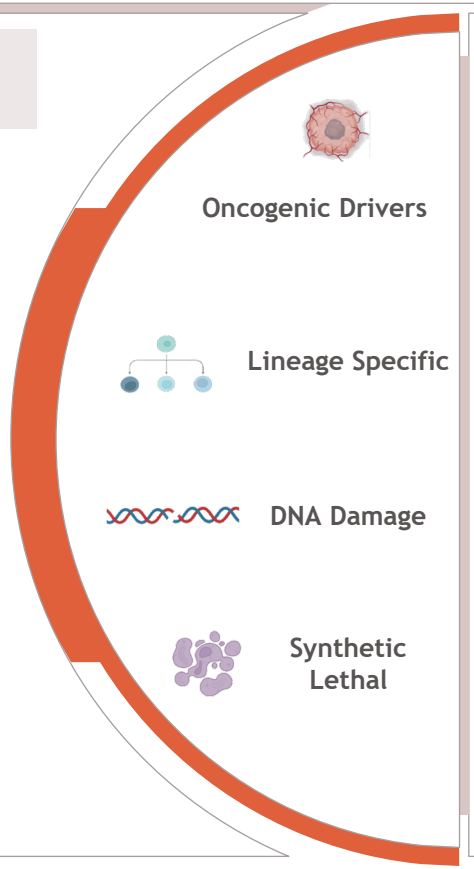
Aberrant Stromal Biology

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



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

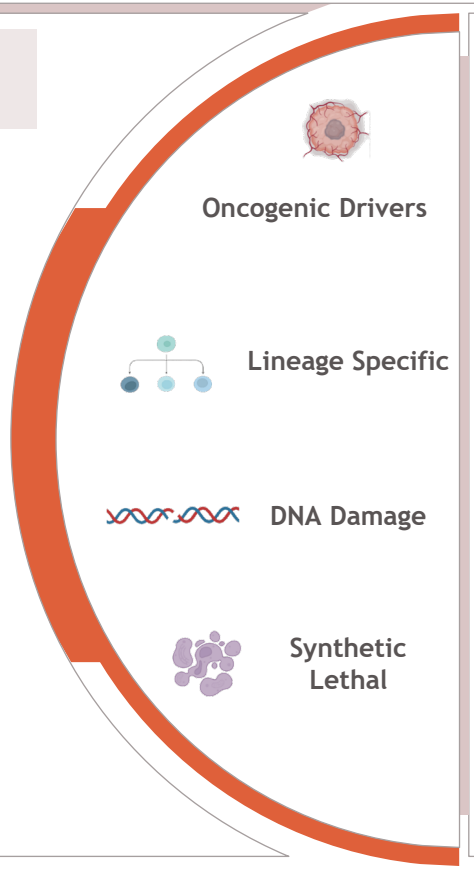
repotrectinib in ROS1+ lung cancer, RAS signaling (SHP2 inhibitor)

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

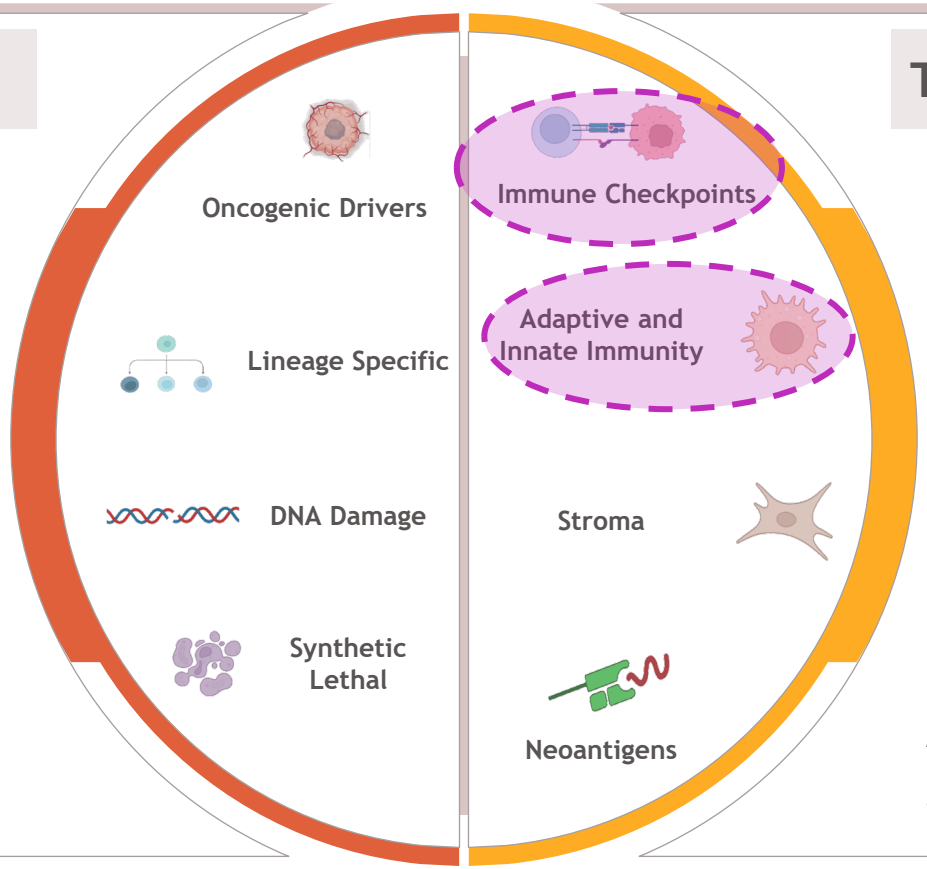
repotrectinib in ROS1+ lung cancer, RAS signaling (SHP2 inhibitor)

Lineage-specific targets

AR LDD in prostate cancer, anti-ganglioside fucosyl-GM1 in SCLC

Cancer cell vulnerabilities

Context specific dependencies (e.g., DNA damage), synthetic lethal interactions



Tumor Extrinsic

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 in action: anti-CCR8 antibody depletes T regulatory cells (Tregs) with combination potential

Transformational potential

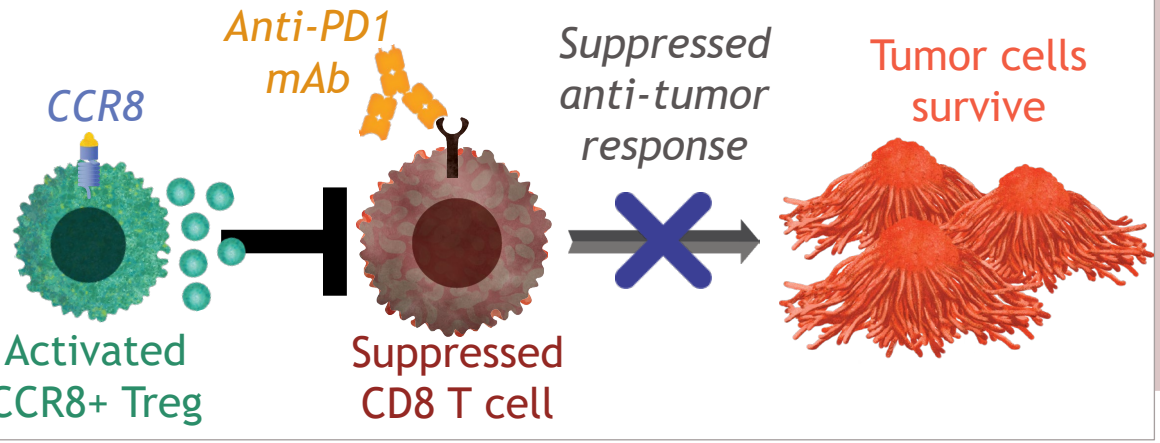
First-in-class, Treg-depleting monoclonal antibody (mAb) with broad combo potential across multiple tumor types.

Matching modality to mechanism

BMS-986340 is an anti-CCR8 IgG1 biologic with enhanced non-fucosylated (NF) Fc that binds CCR8 and potently depletes T regs while sparing effector CD8 T cells.

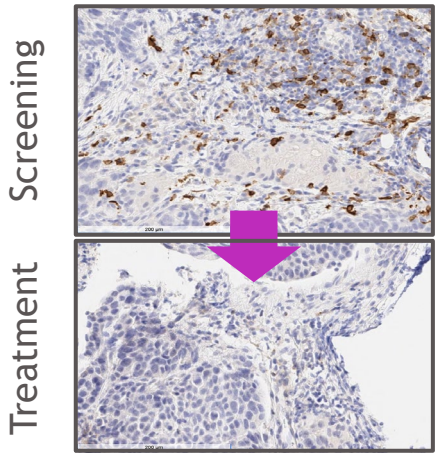
Causal human biology

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

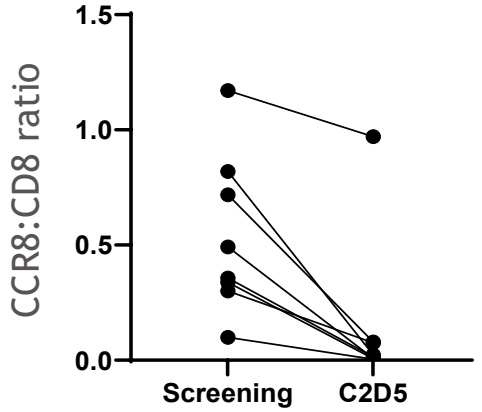


Path to clinical proof-of-concept

Depletion of CCR8+ Tregs in the tumor after 2 cycles



Reduced ratio CCR8 Treg to CD8+ Teff in the tumor



Research framework in action: anti-CCR8 antibody depletes T regulatory cells (Tregs) with combination potential

Transformational potential

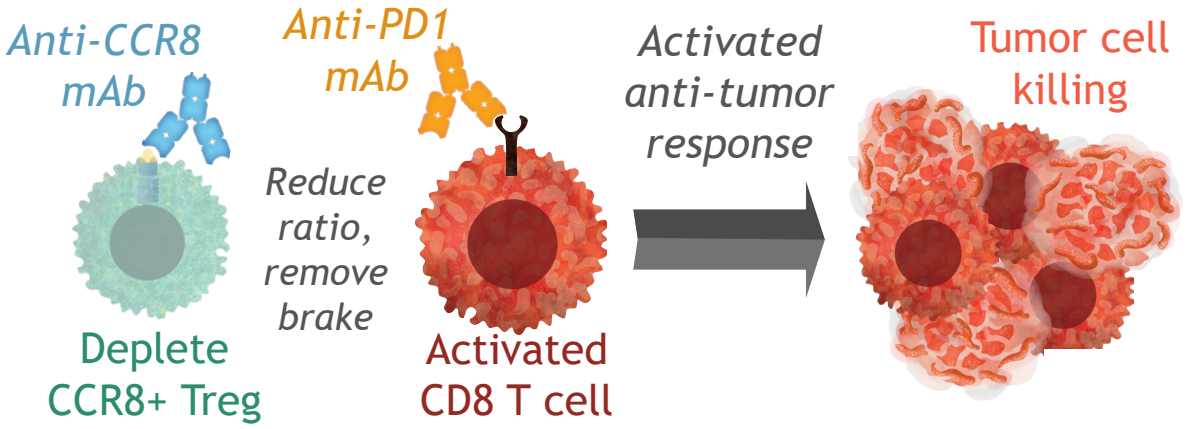
First-in-class, Treg-depleting monoclonal antibody (mAb) with broad combo potential across multiple tumor types.

Matching modality to mechanism

BMS-986340 is an anti-CCR8 IgG1 biologic with enhanced non-fucosylated (NF) Fc that binds CCR8 and potently depletes T regs while sparing effector CD8 T cells.

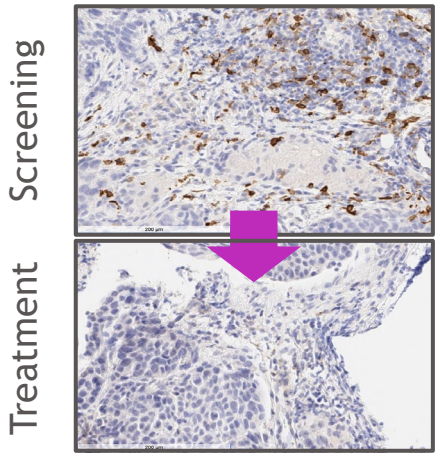
Causal human biology

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

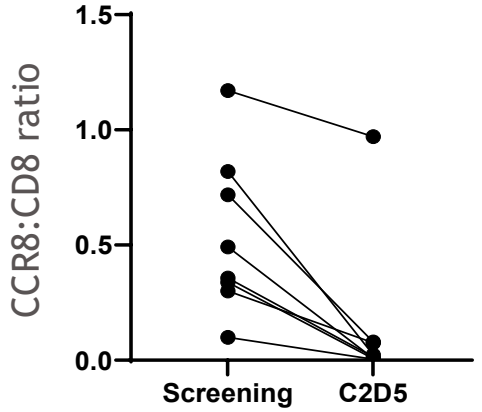


Path to clinical proof-of-concept

Depletion of CCR8+ Tregs in the tumor after 2 cycles



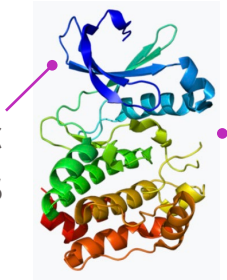
Reduced ratio CCR8 Treg to CD8+ Teff in the tumor



Matching modality to mechanism: Leveraging expertise across multiple modalities

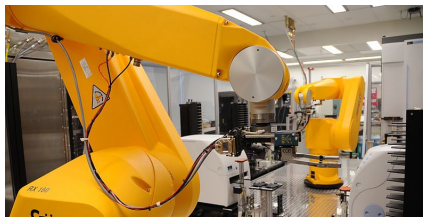
Small molecule chemistry

Allosteric inhibitors



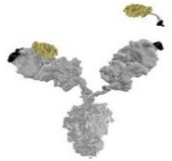
Active site inhibitors

AI-assisted screening



Biotherapeutics

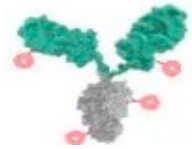
Probody® Therapeutic



Immune cell engagers

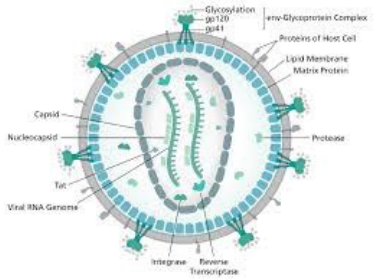
Bi-specifics

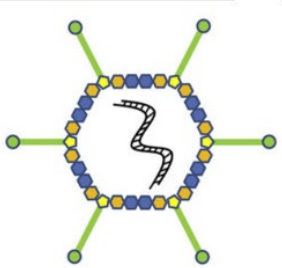
ADCs



Nucleic acid therapies

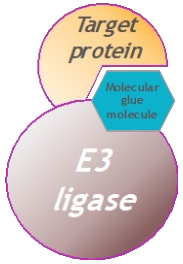
Lentivirus and AAV gene therapy





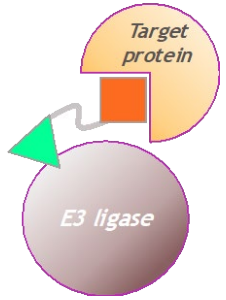
Targeted Protein Degradation

Molecular glue



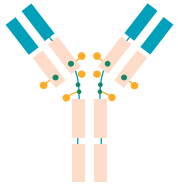
CELMoD

Hetero-bifunctional



LDD

ADC degrader

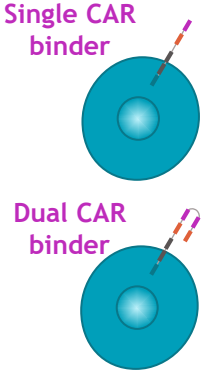


- Cysteine residue
- Drug-Linker

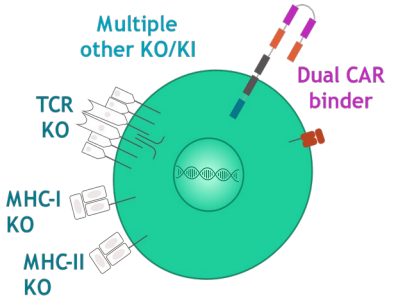
CELMoD ADC

Cell Therapy

Autologous



Next-generation



Allogeneic, iPSC-derived

Established | Emerging | Discuss today

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)

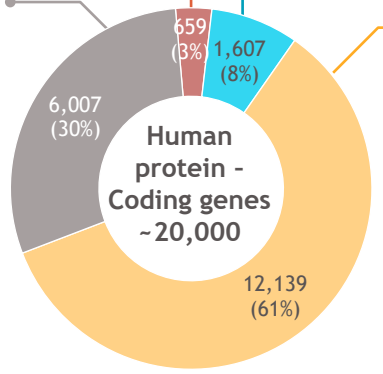
Drugged: Target with approved drugs (3%)

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%)

Small molecule enzyme inhibitor



Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets

Expanded universe of targets

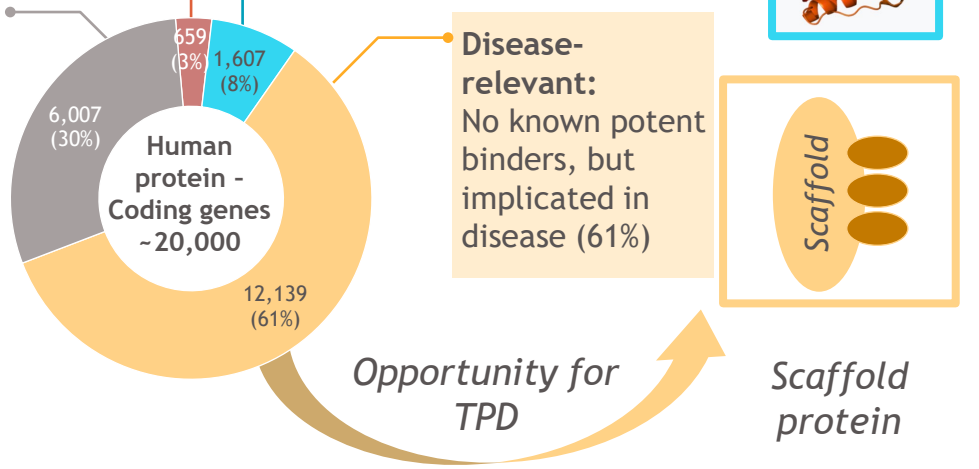
(e.g., scaffolding proteins, transcription factors)

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%)

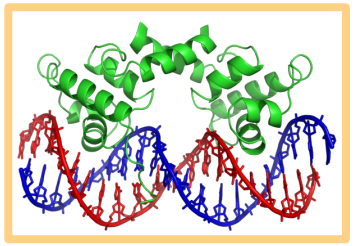
Disease-relevant: No known potent binders, but implicated in disease (61%)



Opportunity for TPD

Small molecule enzyme inhibitor

Scaffold protein



Transcription factor

Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets

Expanded universe of targets

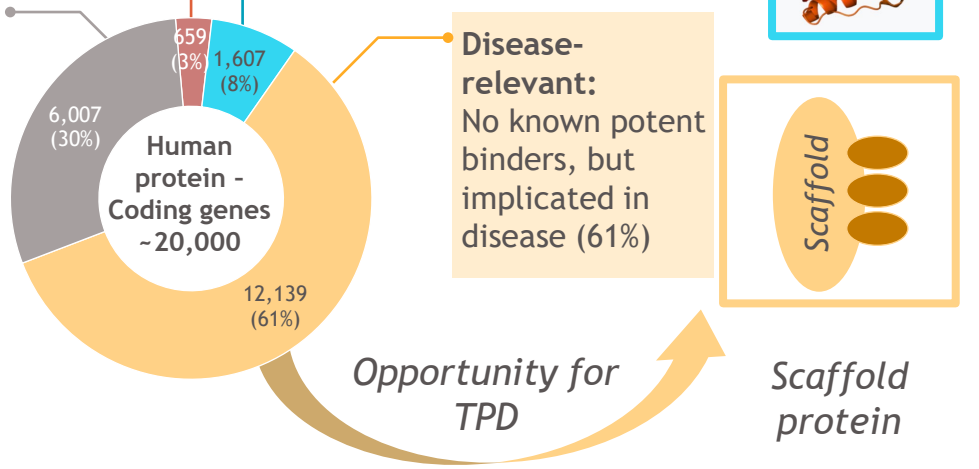
(e.g., scaffolding proteins, transcription factors)

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%)

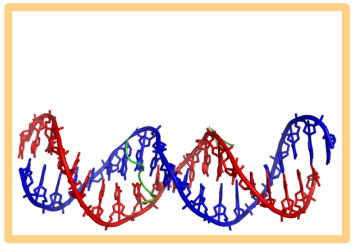
Disease-relevant: No known potent binders, but implicated in disease (61%)



Opportunity for TPD

Small molecule enzyme inhibitor

Scaffold protein

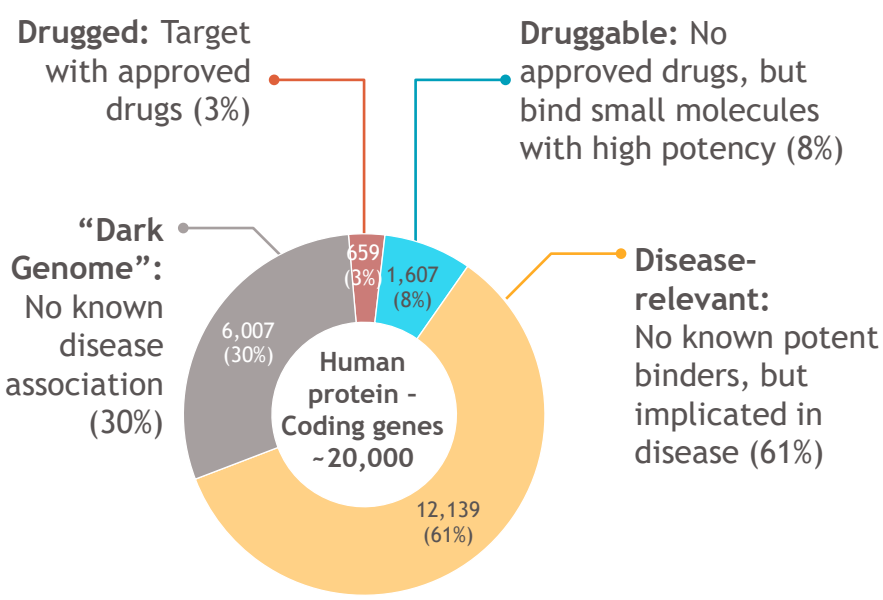


Transcription factor

Targeted Protein Degradation offers the promise of novel targets and clinical differentiation for existing targets

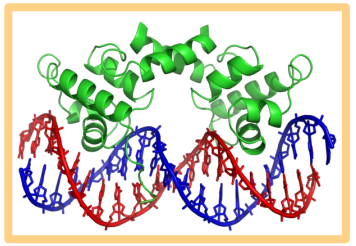
Expanded universe of targets

(e.g., scaffolding proteins, transcription factors)



Small molecule enzyme inhibitor

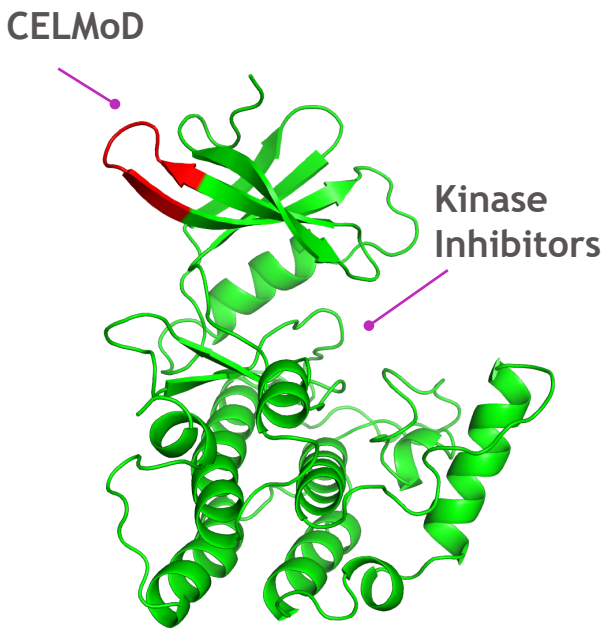
Scaffold protein



Transcription factor

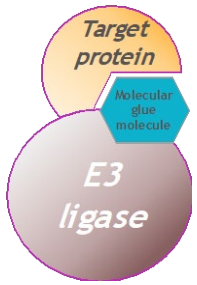
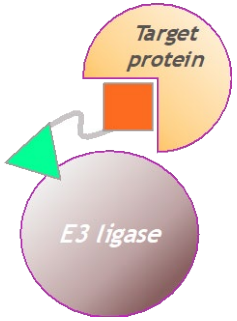
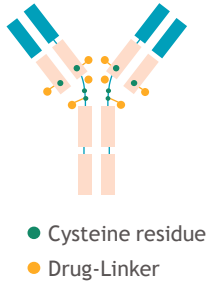
Potentially superior efficacy

(e.g., overcome resistance, higher selectivity)



Our industry leading position in protein degradation is driven by portfolio breadth and depth of expertise

Industry-leading capabilities

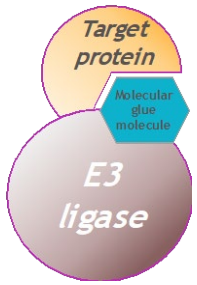
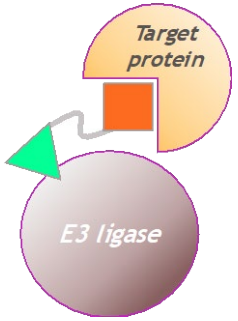
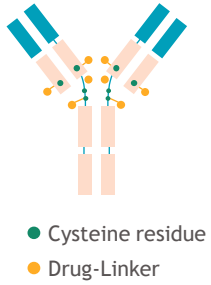
Molecular glue	Hetero-bifunctional	ADC degrader
		
CELMoD	LDD	CELMoD ADC

Berkeley UNIVERSITY OF CALIFORNIA
A-ALPHA BIO
evotec
SGC
SyntheX Targeting the undruggable
AMPHISTA THERAPEUTICS

Our industry leading position in protein degradation is driven by portfolio breadth and depth of expertise

Discuss today

Industry-leading capabilities

Molecular glue	Hetero-bifunctional	ADC degrader
		
CELMoD	LDD	CELMoD ADC

Berkeley UNIVERSITY OF CALIFORNIA · A-ALPHA BIO · evotec · SGC · SyntheX Targeting the undruggable · AMPHISTA THERAPEUTICS

Industry-leading pipeline

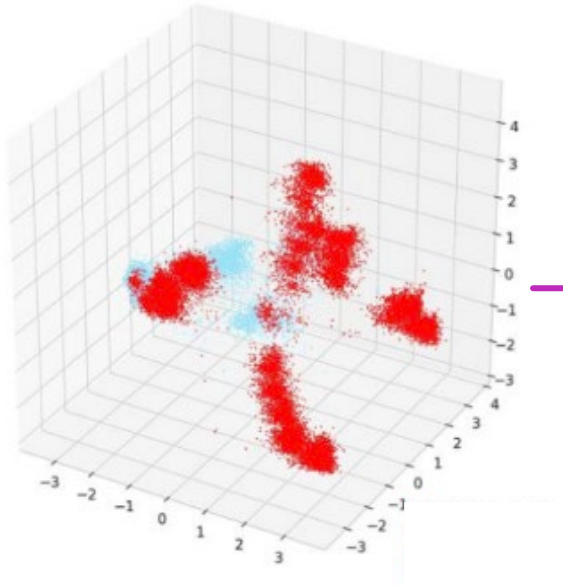
Full Development	**iberdomide:** Multiple Myeloma	**mezigdomide:** Multiple Myeloma
	golcadomide: Lymphoma	
Early Development	**CK1α:** Acute Myeloid Leukemia	**AR LDD:** Prostate
	BCL6 LDD: Lymphoma	**Helios:** Solid Tumors
IND-enabling studies	**HbF CELMoD:** Sickle Cell Disease	**LDD:** Prostate
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

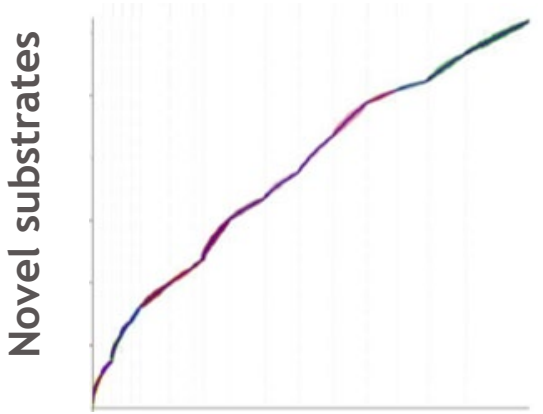
Diversify chemical library

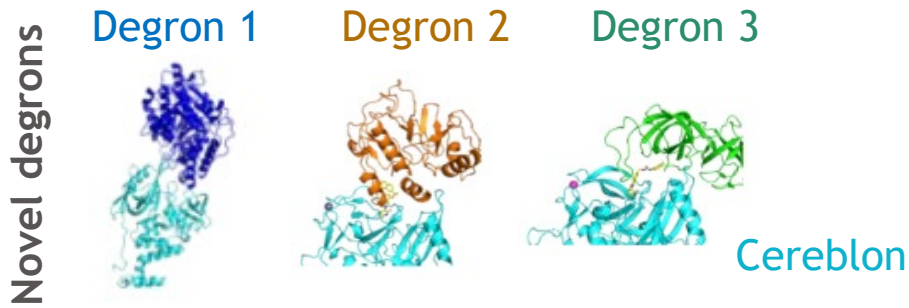


CELMoD library 2019

CELMoD library 2023

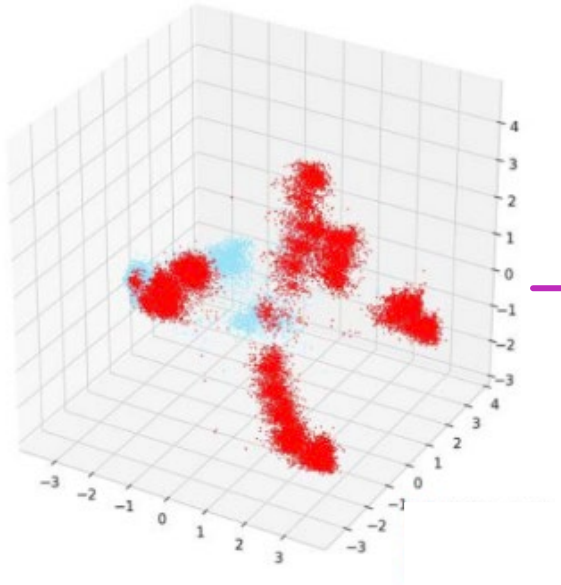
Expanding CELMoD library identifies **novel substrates** and **novel degrons**





The swift expansion of our CELMoD library has enabled key scientific insights and an increased number of IND candidates

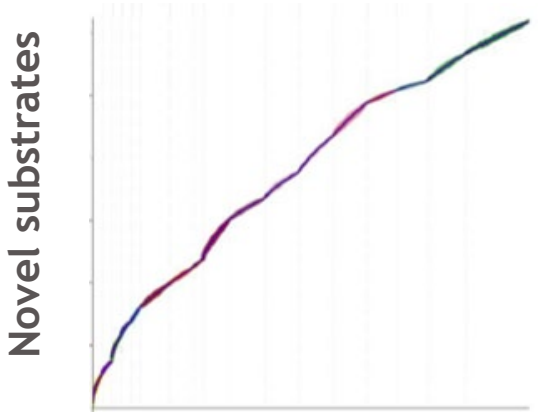
Diversify chemical library



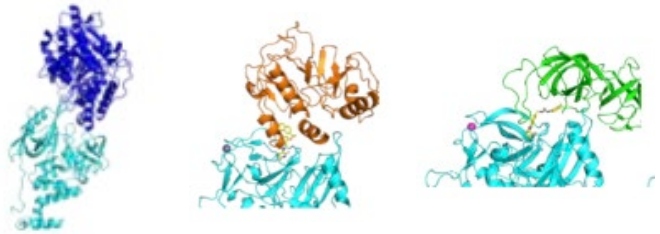
CELMoD library 2019

CELMoD library 2023

Expanding CELMoD library identifies **novel substrates** and **novel degrons**

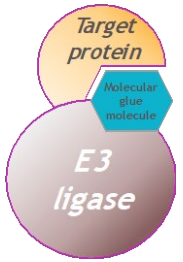


Novel degrons



Causal human biology

10 CELMoDs for Oncology in full discovery & IND enabling studies



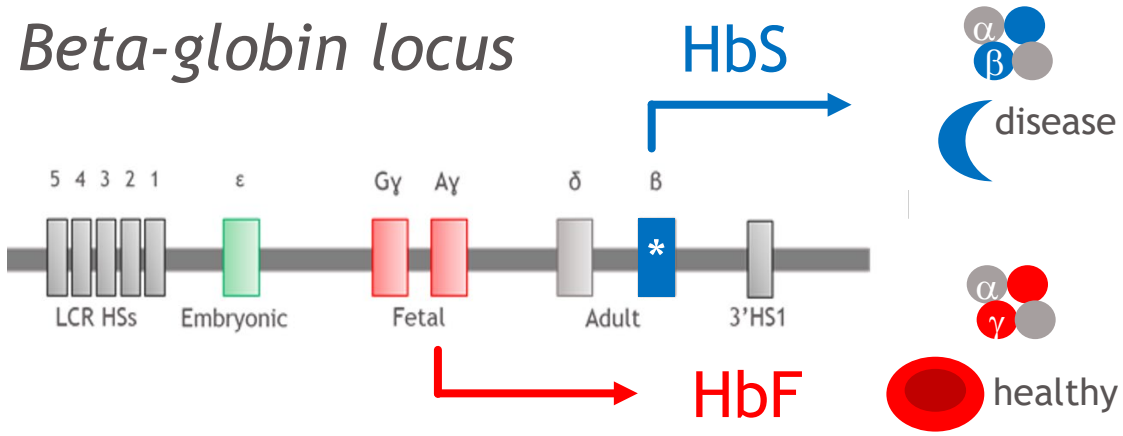
Targeting the previously undruggable: A novel CELMoD for Sickle Cell Disease

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).

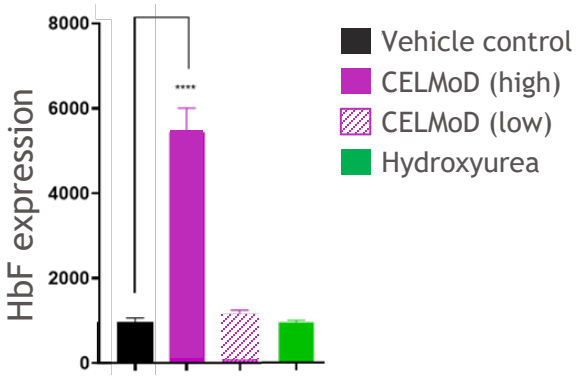
Causal human biology

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

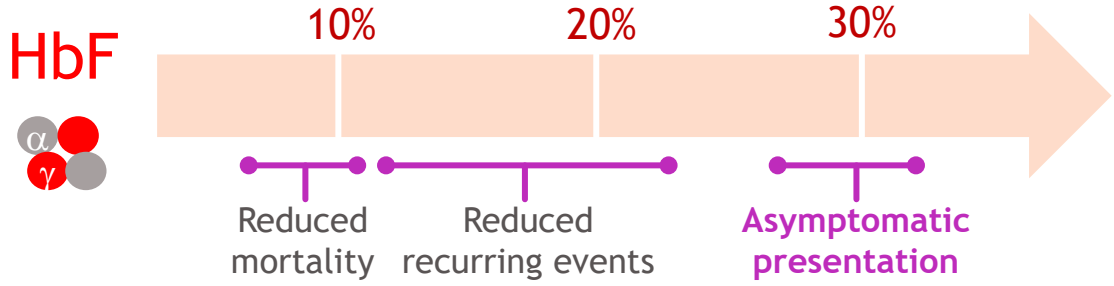


Matching modality to mechanism

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



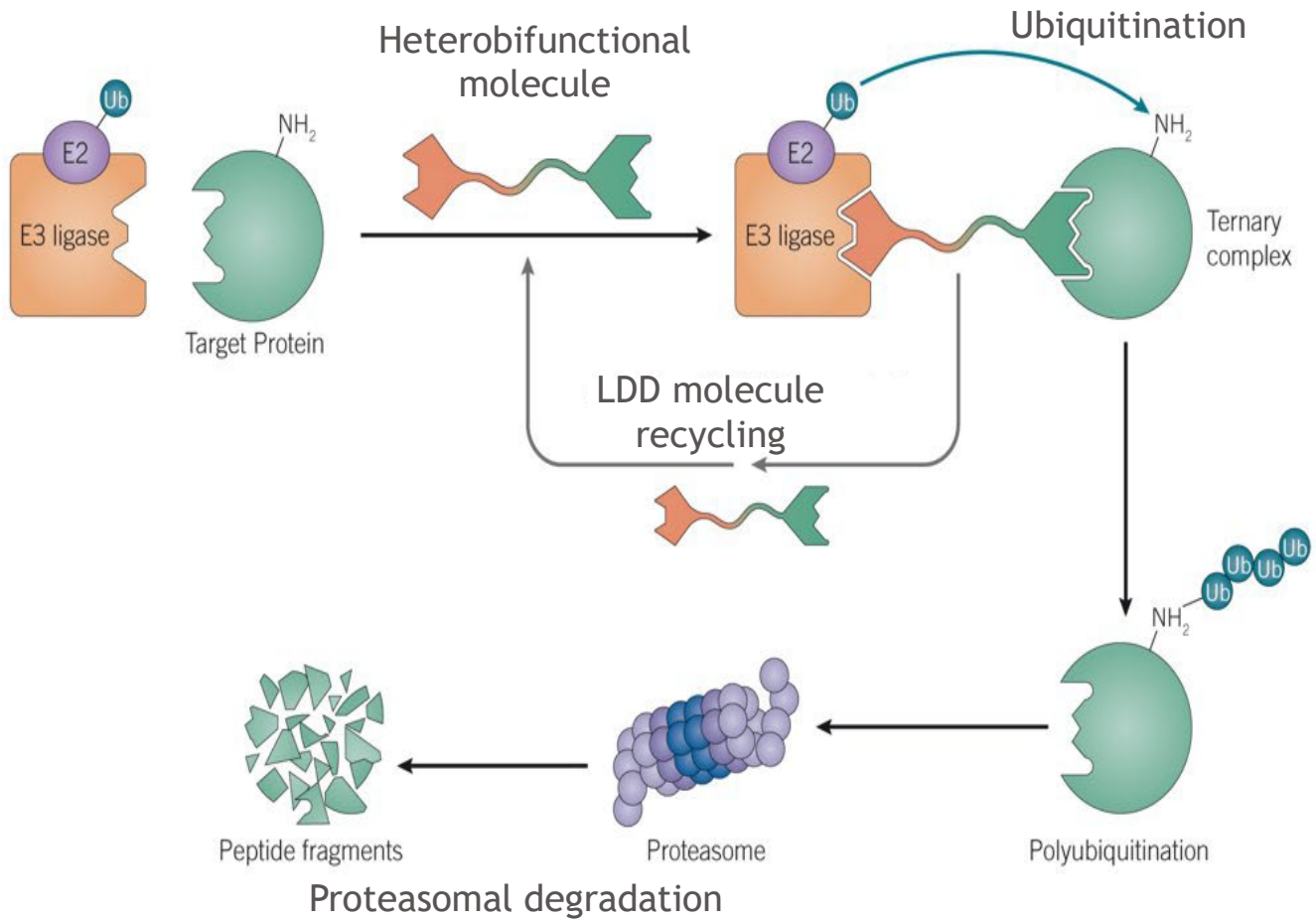
Path to clinical proof-of-concept



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

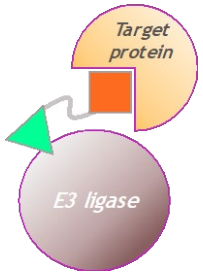
Different from CELMoDs

Same as CELMoDs



Early Development
AR LDD: Prostate
BCL6 LDD: Lymphoma

IND-enabling studies
LDD: Prostate
LDD: Autoimmune



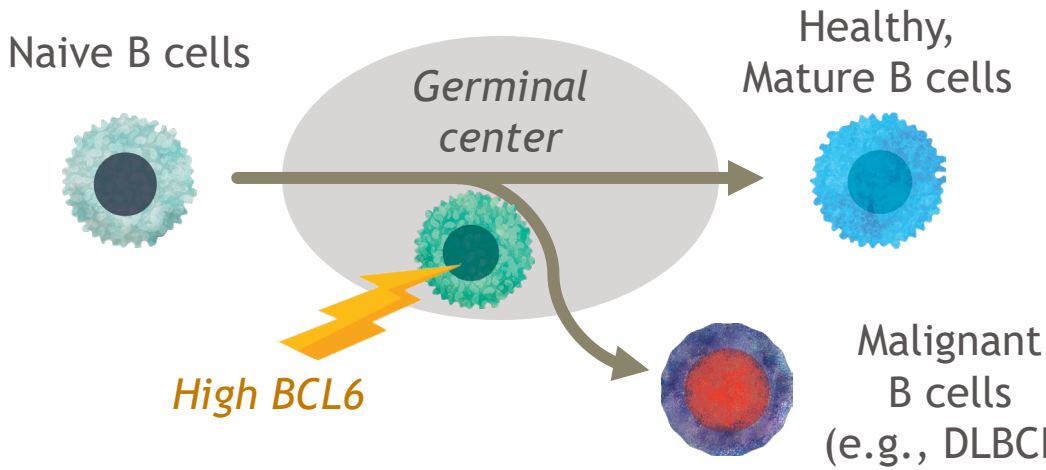
BMS-986458 is a novel ligand directed degrader (LDD) targeting BCL6 in lymphoma

Transformational potential

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

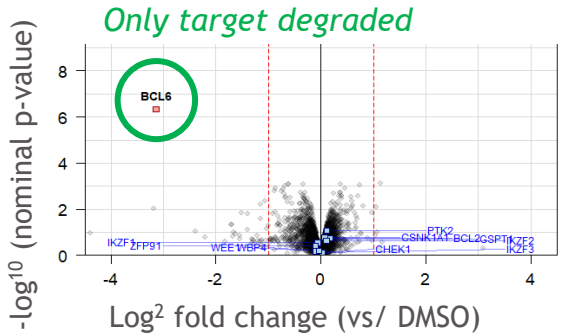
Causal human biology

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



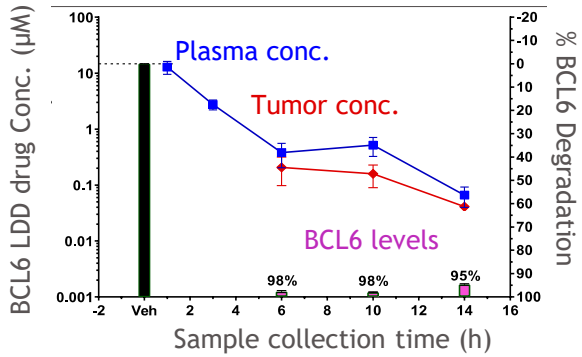
Matching modality to mechanism

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

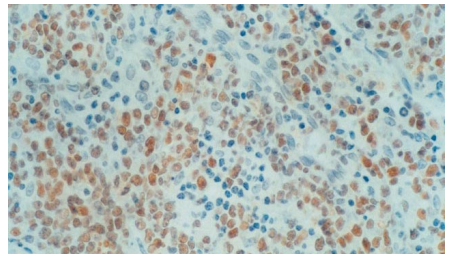


Path to clinical proof-of-concept

Correlate BCL6 degradation with clinical benefit



BCL6 IHC high expression in ~30% DLBCL

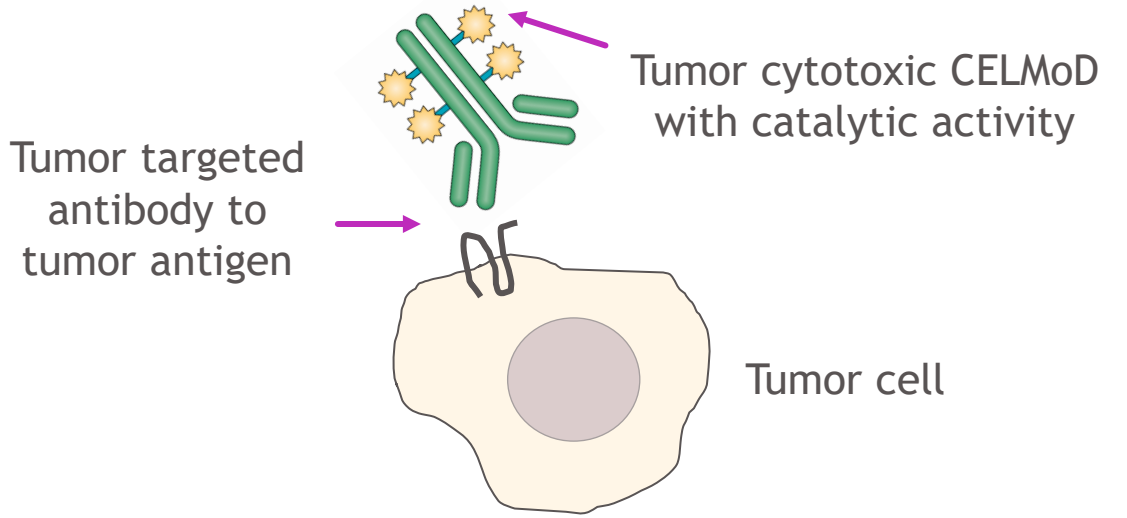


A new frontier: CELMoD ADCs to improve efficacy and safety in hematology/solid tumors

Causal human biology

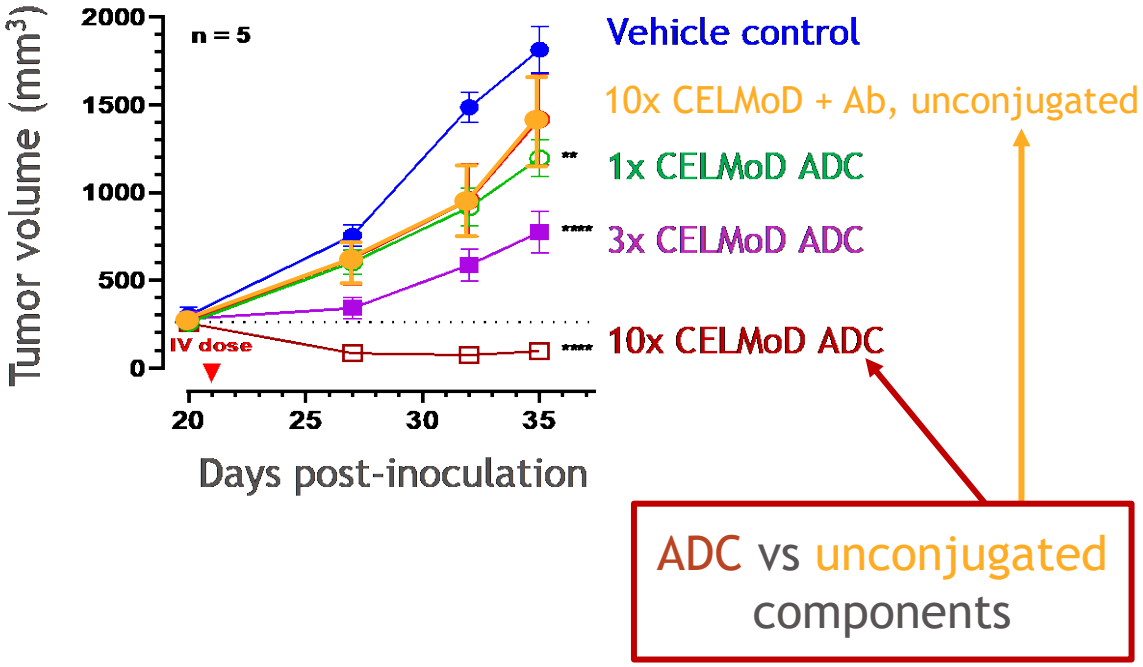
Combine a *clinically validated* tumor targeted antibody with a *clinically validated* tumor cell-biased CELMoD to enhance efficacy and tolerability in hematology/solid tumors

Matching modality to mechanism



Path to clinical proof-of-concept

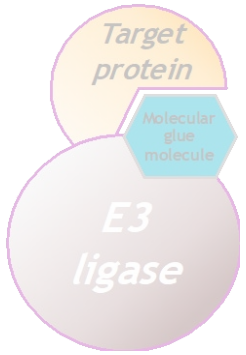
Enhanced efficacy of ADC at lower levels of administered CELMoD



Targeted Protein Degradation and Cell Therapy: Two differentiated platforms for optimizing therapies for patients

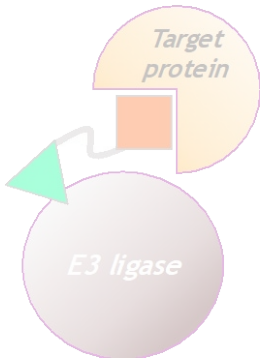
Targeted Protein Degradation

Molecular glue



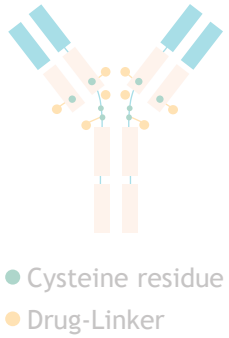
CELMoD

Hetero-bifunctional



LDD

ADC degrader



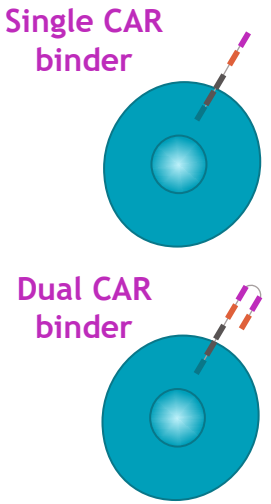
CELMoD ADC

Matching modality to mechanism

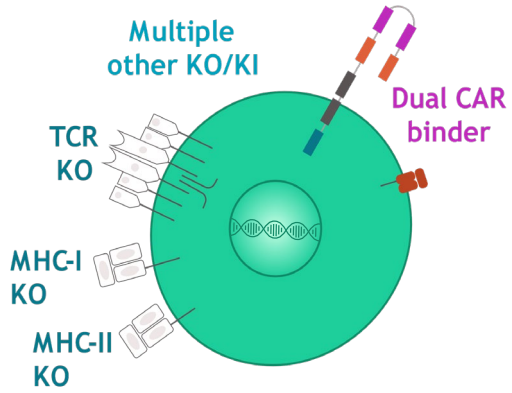
Invention of high-quality therapeutics that match a modality to a molecular mechanism of action

Cell Therapy

Autologous



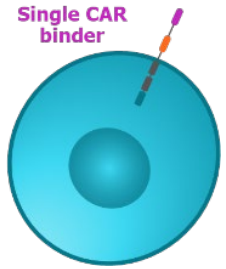
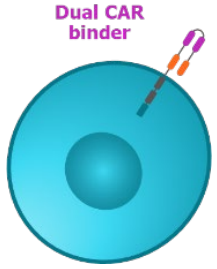
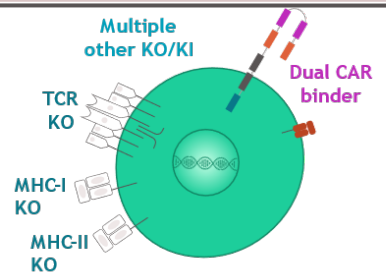
Next-generation



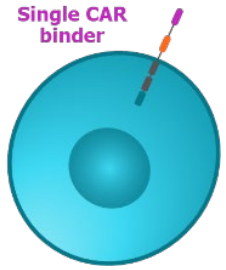
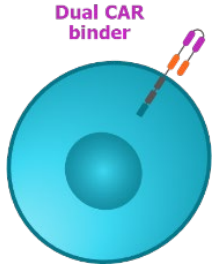
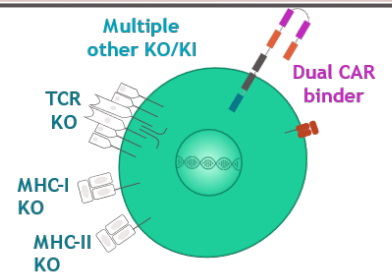
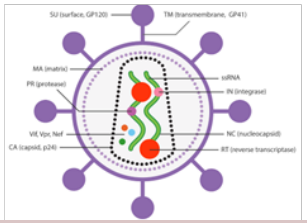
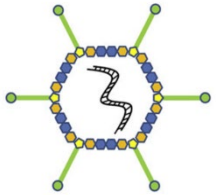
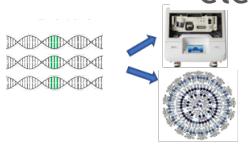
Allogeneic, iPSC-derived

These two platforms unlock novel targets and mechanisms to efficiently deliver INDs with the potential to improve the lives of patients

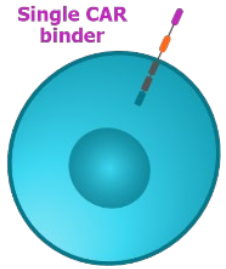
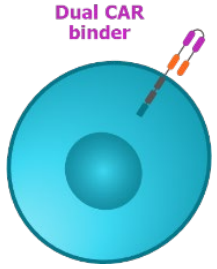
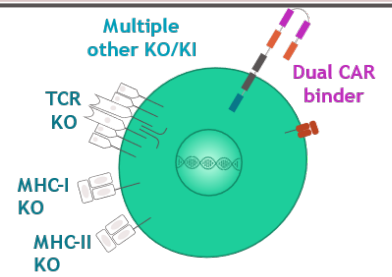
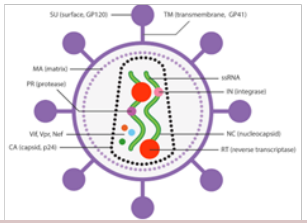
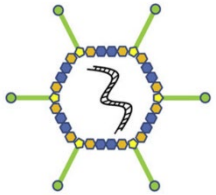
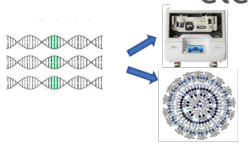
We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency

	Wave 1	Wave 2	Wave 3
Next-gen engineering			
	Monospecific CAR	Dual Targeting CAR	Engineered CAR/TCR
Evolving delivery systems			
Optimizing manufacturing			

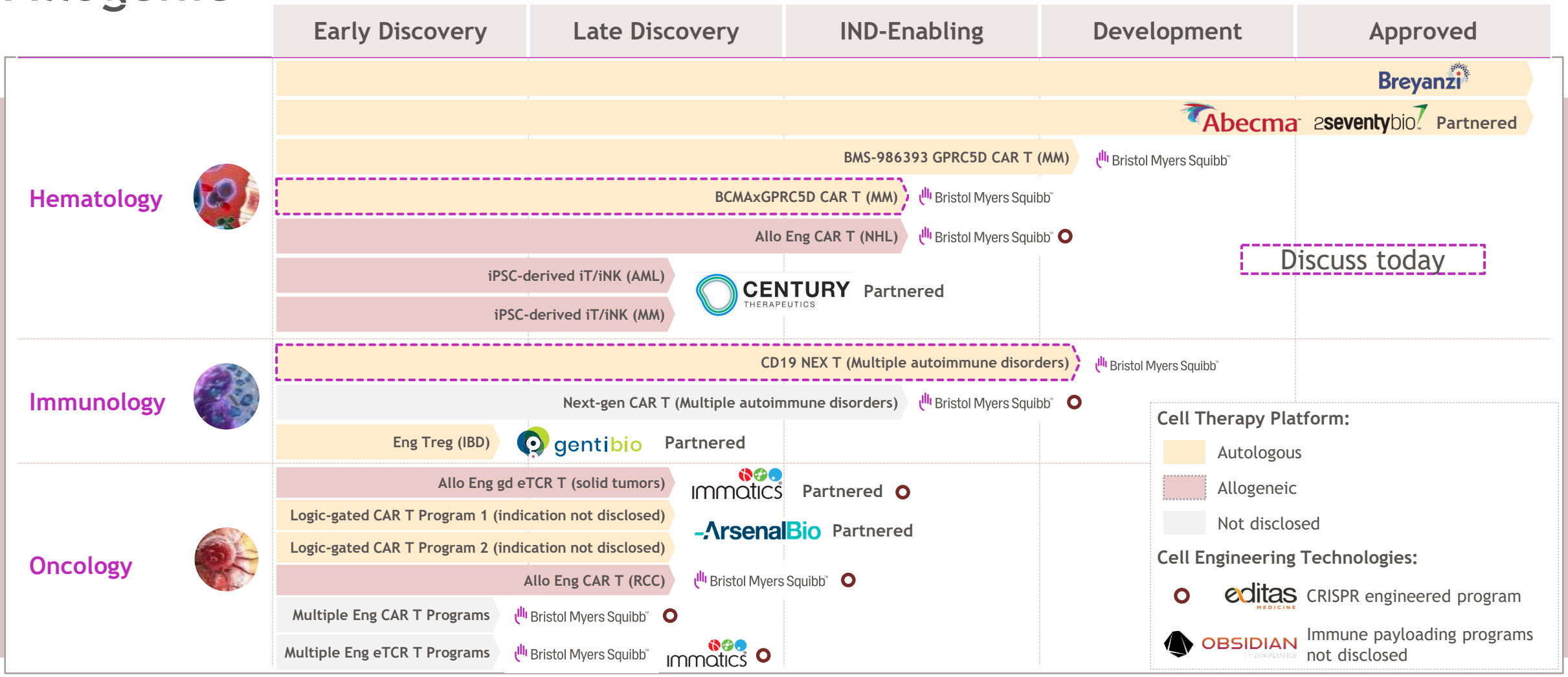
We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency

	Wave 1	Wave 2	Wave 3
Next-gen engineering	 Monospecific CAR	 Dual Targeting CAR	 Engineered CAR/TCR
Evolving delivery systems	 Lentivirus (LVV)	 Adeno-associated virus (AAV)	electroporation DNA  Lipid nanoparticles (LNP) Non-viral delivery (NVD)
Optimizing manufacturing			

We are leveraging expertise to enable expansion beyond Hematology while increasing manufacturing efficiency

	Wave 1	Wave 2	Wave 3
Next-gen engineering	 Monospecific CAR	 Dual Targeting CAR	 Engineered CAR/TCR
Evolving delivery systems	 Lentivirus (LVV)	 Adeno-associated virus (AAV)	electroporation DNA  Lipid nanoparticles (LNP) Non-viral delivery (NVD)
Optimizing manufacturing	Autologous Breyanzi Abecma α/β T cells	Autologous NEX T platform α/β T cells	Allogeneic Healthy donor or iPSC α/β or γ/δ T cells, iNK/iT cells

Next-gen Cell Therapy pipeline: Oncology, Immunology, and Allogenic



Wave 2

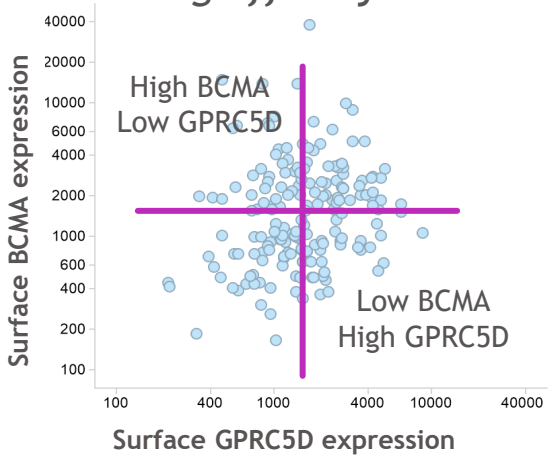
Dual targeting BCMAxGPRC5D CAR T for relapsed/refractory multiple myeloma

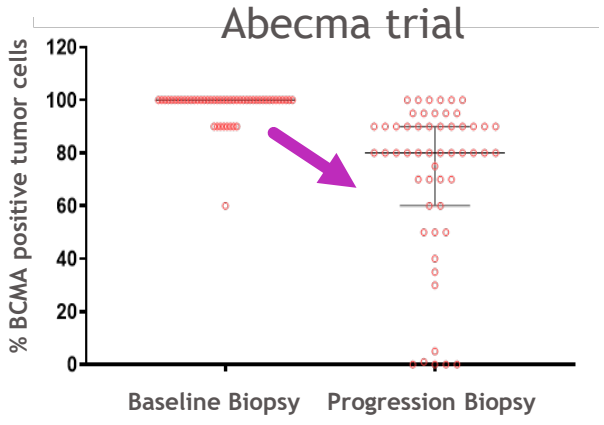
Transformational potential

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

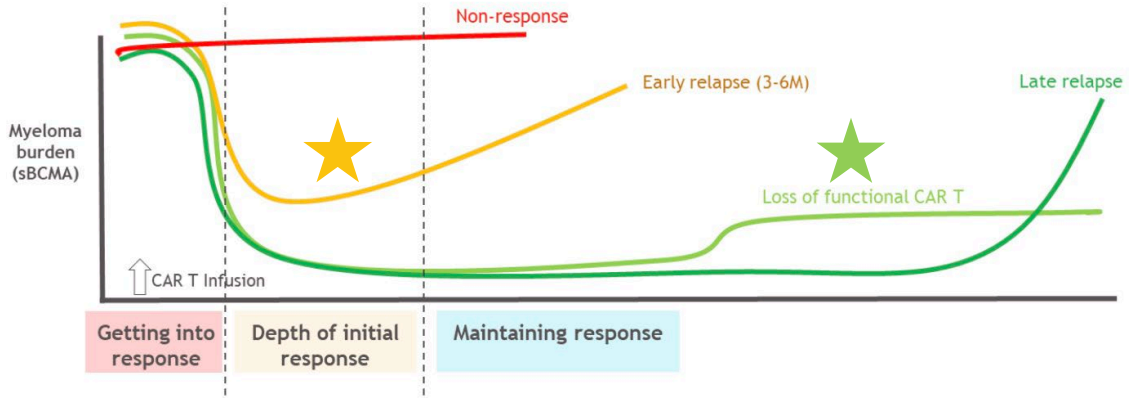




Matching modality to mechanism

- **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



Wave 2

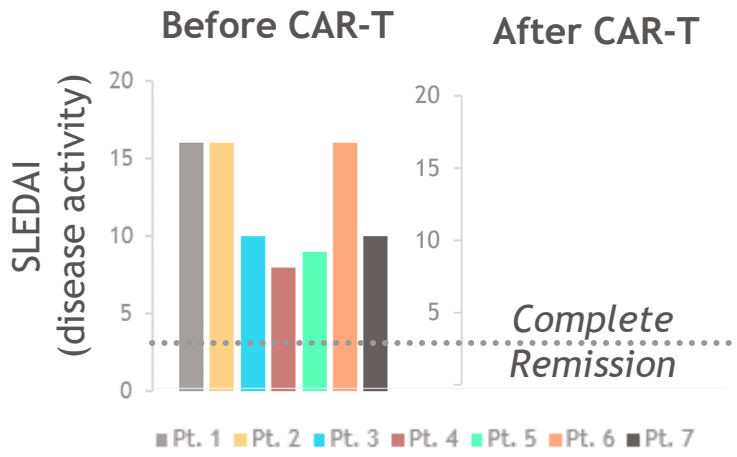
CD19 NEX T to reset the immune system in multiple Immunology indications

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

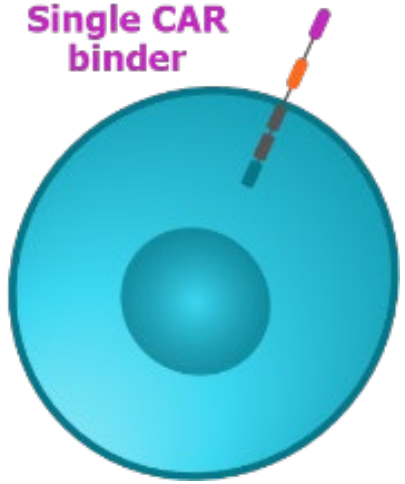
Causal human biology

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



Adapted from Taubmann, J., et al EULAR (2023): 93-94

Matching modality to mechanism



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

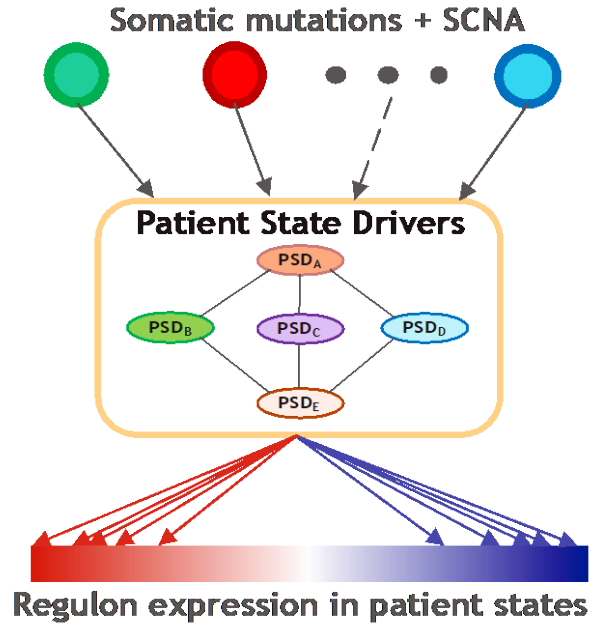
Manufacturing: autologous, single train with shortened turn-around time, lower failure rates

Path to clinical proof-of-concept

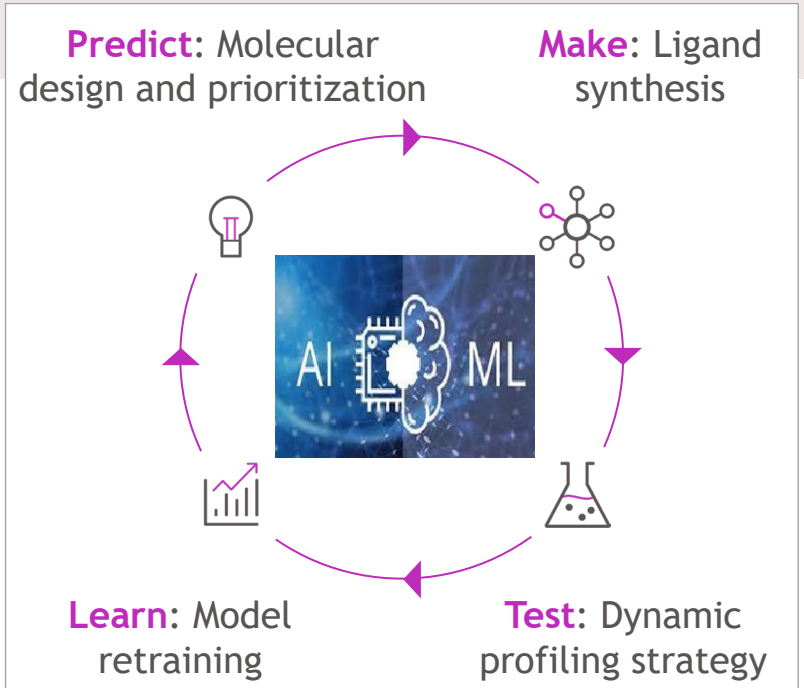
- Expand on findings from academic study in SLE
- Monitor biomarker predictors of cell therapy safety and efficacy
- Demonstrate evidence of resetting immune memory

Computational science, including Artificial Intelligence and Machine Learning, is applied at all stages of Research

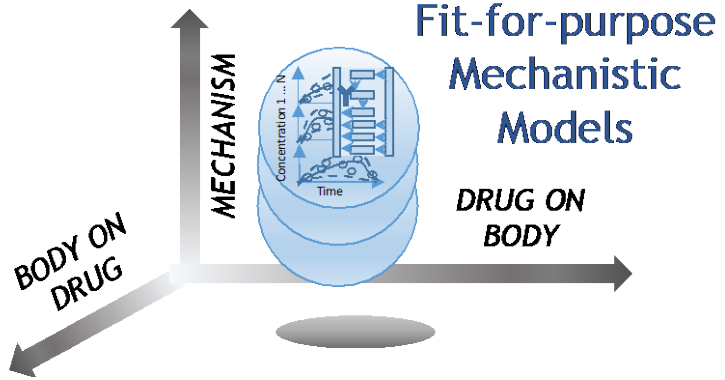
Leverage **patient data** and **predictive analytics** to define causal human biology



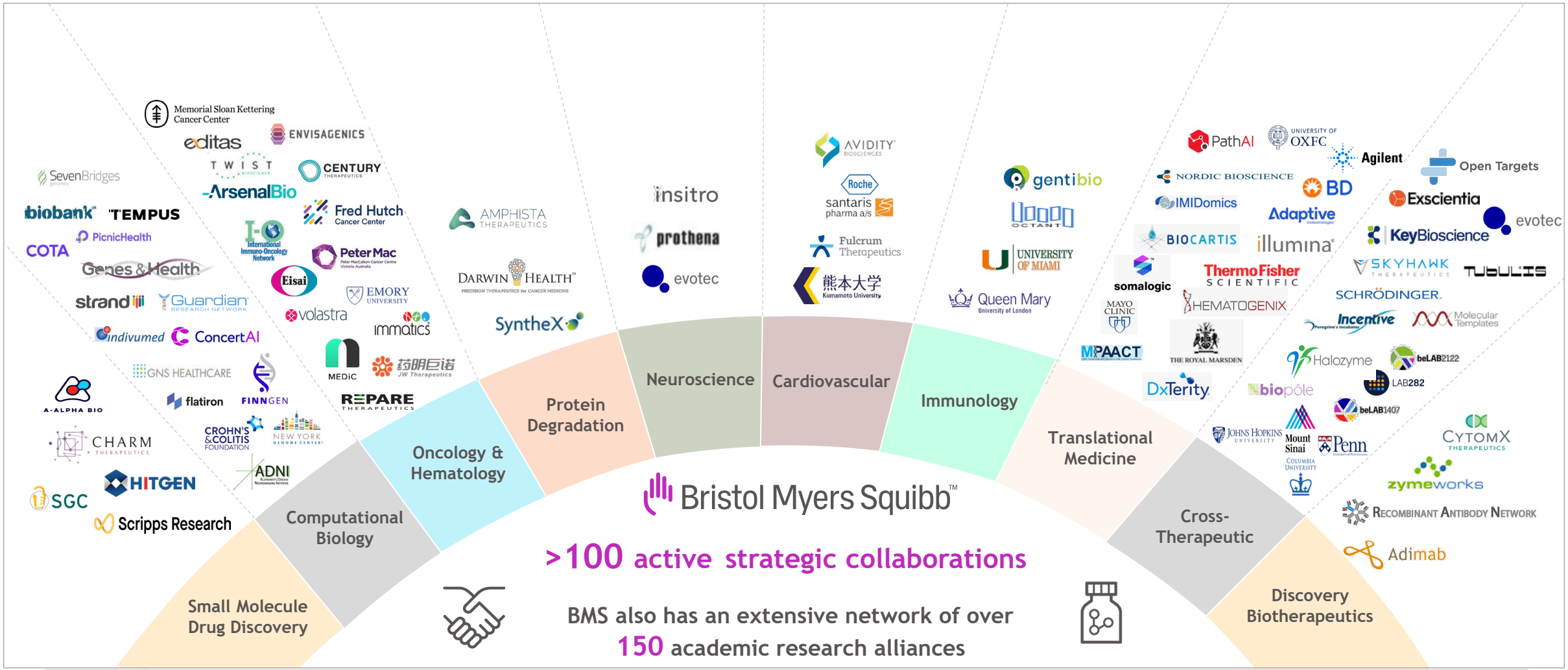
Utilize computational power for **predictive molecule invention** to improve quality and accelerate timelines



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



Internal R&D strengths are amplified through extensive network of external partnerships

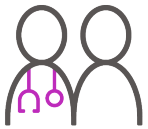


We have the right strategy at the right time to develop transformational medicines & change patients' lives

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

Oncology | Hematology | Immunology | Cardiovascular | Neuroscience

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

Early and Late Clinical Development

Global Development Operations

Global Regulatory Sciences

Global Biometrics & Data Sciences

Worldwide Patient Safety

Portfolio & Strategic Operations

Strategy & Capabilities

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

BMS Pipeline

Data as of September 14th, 2023

	Oncology		Hematology		Immunology		Cardiovascular	Neuroscience
Phase 1	✦AHR Antagonist*^ *Solid Tumors* ✦Anti-CCR8^ *Solid Tumors* ✦Anti-ILT4^ *Solid Tumors* ✦Anti-NKG2A^ *Solid Tumors* ✦AR LDD *1L, 2L mCRPC*	✦Claudin 18.2 ADC *Solid Tumors* ✦DGK Inhibitor *Solid Tumors* ✦JNK Inhibitor *Solid Tumors* ✦Helios CELMoD *Solid Tumors* ✦MAGEA4/8 TCER* *Solid Tumors*	✦SHP2 Inhibitor^ *Solid Tumors* ✦TGFß Inhibitor^ *Solid Tumors* ✦TIGIT Bispecific *Solid Tumors*	✦alnuctamab *RR MM* ✦Anti-SIRPα *Hematologic Malignancies* ✦BCMA NKE *RR MM* ✦BCL6 LDD *Lymphoma* BET Inhibitor (BMS-986158) *Hematologic Malignancies*	✦BET Inhibitor (BMS-986378)^ *RR NHL* ✦CK1α Degrader *Hematologic Malignancies* ✦GPRC5D CAR T *RR MM* golcadomide^ *1L DLBCL* ✦CD33 NKE *RR MM*	✦Anti-CD40 *Autoimmune Disease* afimetoran *CLE* ✦CD19 NEX T *Severe Refractory SLE*	✦IL2-CD25 *Autoimmune Disease* ✦PKCθ Inhibitor *Autoimmune Disease*	✦FXIa Inhibitor *Thrombotic Disorders*	✦Anti-MTBR-Tau *Alzheimer's Disease* ✦eIF2b Activator *Neuroscience* ✦FAAH/MGLL Dual Inhibitor *Neuroscience* ✦BTK Inhibitor *Neuroscience*
Phase 2	✦Anti-CTLA-4 NF Probody® *Solid Tumors* ✦Anti-Fucosyl GM1^ *RR SCLC* ✦Anti-IL-8^ *Solid Tumors* ✦BET Inhibitor (BMS-986378)^ *Solid Tumors*	✦farletuzumab ecteribulin *Solid Tumors* nivolumab+relatlimab *Stage IV 1L NSCLC* nivolumab+relatlimab *1L HCC* nivolumab+relatlimab *2L+ HCC (Post-TKI)*	✦repotrectinib *NTRK Pan-Tumor*	✦golcadomide^ *RR NHL* ✦BET Inhibitor (BMS-986158) *1L MF* ABECMA *1-4L+ MM* BREYANZI *3L+ CLL*	BREYANZI *RR MZL* BREYANZI *RR MCL* REBLOZYL *A-Thalassemia* ONUREG *MDS* BREYANZI *RR FL*	✦afimetoran *SLE* SOTYKTU *DLE*	SOTYKTU *Alopecia Areata* ✦TYK2 Inhibitor (BMS-986322) *Mod-to-Severe Psoriasis*	✦MYK-224 *oHCM* ✦danicamtiv *Dilated Cardiomyopathy* CAMZYOS *HFpEF*	
Phase 3	✦SC nivolumab + rHuPH20 (multi-indications) *2L RCC* OPDIVO *Adjuvant HCC* OPDIVO *Peri-adjuvant MIUC* OPDIVO *Peri-adjuvant NSCLC*	OPDIVO *Stage IB-IIIA Adjuvant NSCLC** OPDIVO + YERVOY *1L HCC* OPDIVO + YERVOY *1L MIUC* OPDIVO + YERVOY *1L+ MSI High CRC*	OPDIVO + YERVOY *St3 Unresectable NSCLC* OPDUALAG *Adjuvant Melanoma* OPDUALAG *2L/3L+ MSS mCRC* ✦SC nivolumab + relatlimab + rHuPH20 *1L Melanoma*	✦iberdomide *2L+ MM* iberdomide *Post-ASCT Maintenance NDMM* ✦mezigdomide *2L+ MM Vd* mezigdomide *2L+ MM Kd*	INREBIC *MF* REBLOZYL *1L TD MF Associated Anemia* REBLOZYL *1L NTD MDS Associated Anemia*	✦cendakimab *Eosinophilic Esophagitis* ✦LPA1 Antagonist *IPF* LPA1 Antagonist *PPF* SOTYKTU Psoriatic Arthritis	SOTYKTU *SLE* SOTYKTU *Sjögren's Syndrome* ZEPOSIA *Crohn's Disease* ✦obexelimab*# *IgG4-Related Disease*	✦milvexian *Secondary Stroke Prevention** milvexian *Acute Coronary Syndrome** milvexian *Atrial Fibrillation** CAMZYOS *nHCM*	

Bristol Myers Squibb™

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

Immunology

Addressing high unmet medical need in Immunology

Asset	Approved	Registrational†	Exploratory/PoC Studies†
SOTYKTU™ (deucravacitinib) 6 mg tablets	Moderate-to-severe Psoriasis	• Psoriatic Arthritis • Sjögren's Syndrome • Systemic Lupus Erythematosus	Alopecia Areata
ZEPOSIA® (ozanimod) 0.92 mg capsules	Moderate-to-severe Ulcerative Colitis	Moderate-to-severe Crohn's Disease	-
CD19 NEX T	-	-	Severe, refractory Systemic Lupus Erythematosus
cendakimab	-	• Eosinophilic Esophagitis • Eosinophilic Gastroenteritis[1]	-
LPA$_1$ Antagonist	-	• Idiopathic Pulmonary Fibrosis • Progressive Pulmonary Fibrosis	-

1. Japan registrational trial; † ongoing or initiating 2023/2024

Significant unmet need in pulmonary fibrosis

Disease

- Fibrotic ILD: Associated with thickening of the lung lining, causing irreversible damage[1]
- IPF: Fatal disease with 3-5 years median survival[2]
- PPF: Heterogenous group of ILDs with a progressive-fibrosing phenotype[1]

Unmet Need

- Ideal novel therapies which can be used alone or in combination with approved anti-fibrotics
- Treatments needed to address underlying fibrosis and reduce decline in lung function
- Tolerable treatment options to increase adherence and QoL improvement

Treatment Opportunity

- Deliver a potential new product with an improved efficacy and tolerability profile over current treatment options
- Approved therapies do not treat underlying fibrosis or halt disease progression

1. Raghu. *Am J Respir Crit Care Med*. 2022 May 1;205(9):e18-e47. 2. Raghu. *Am J Respir Crit Care Med*. 2011 Mar 15;183(6):788-824.

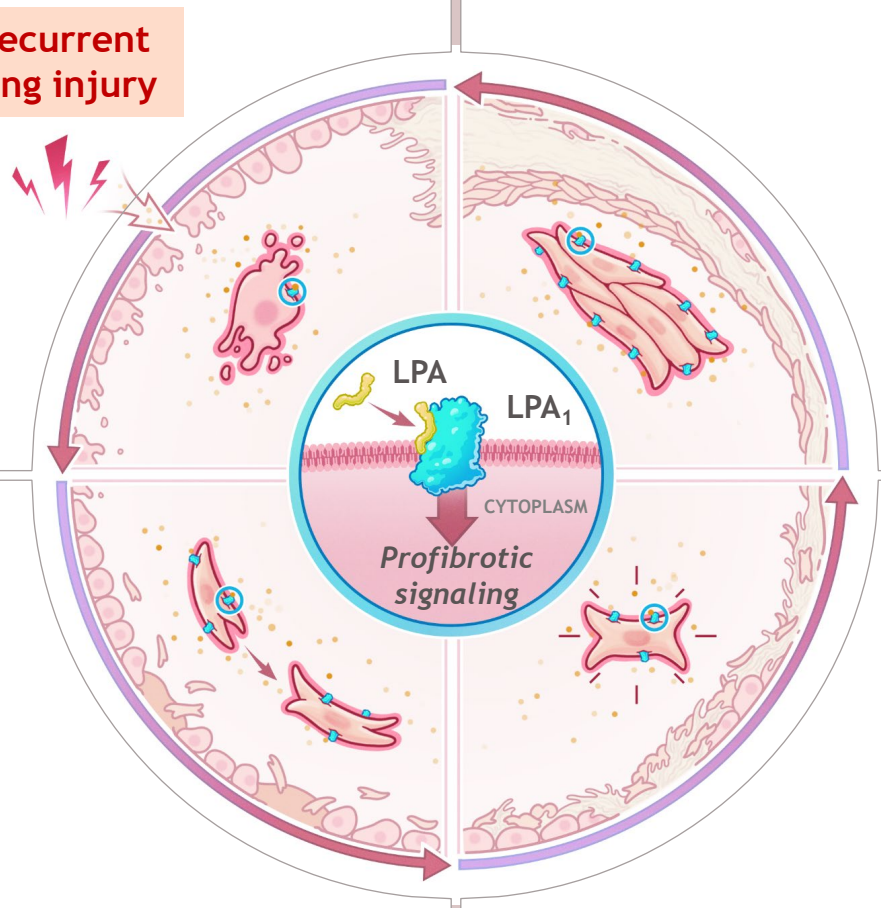
LPA$_1$ signaling is central to the pathogenesis of fibrotic lung diseases

1. Epithelial cell apoptosis[1]

LPA$_1$ signaling promotes apoptosis of alveolar epithelial cells

2. Fibroblast recruitment[2,3]

LPA$_1$ signaling induces fibroblast chemotaxis to the site of injury



3. Fibroblast proliferation & activation[4,5]

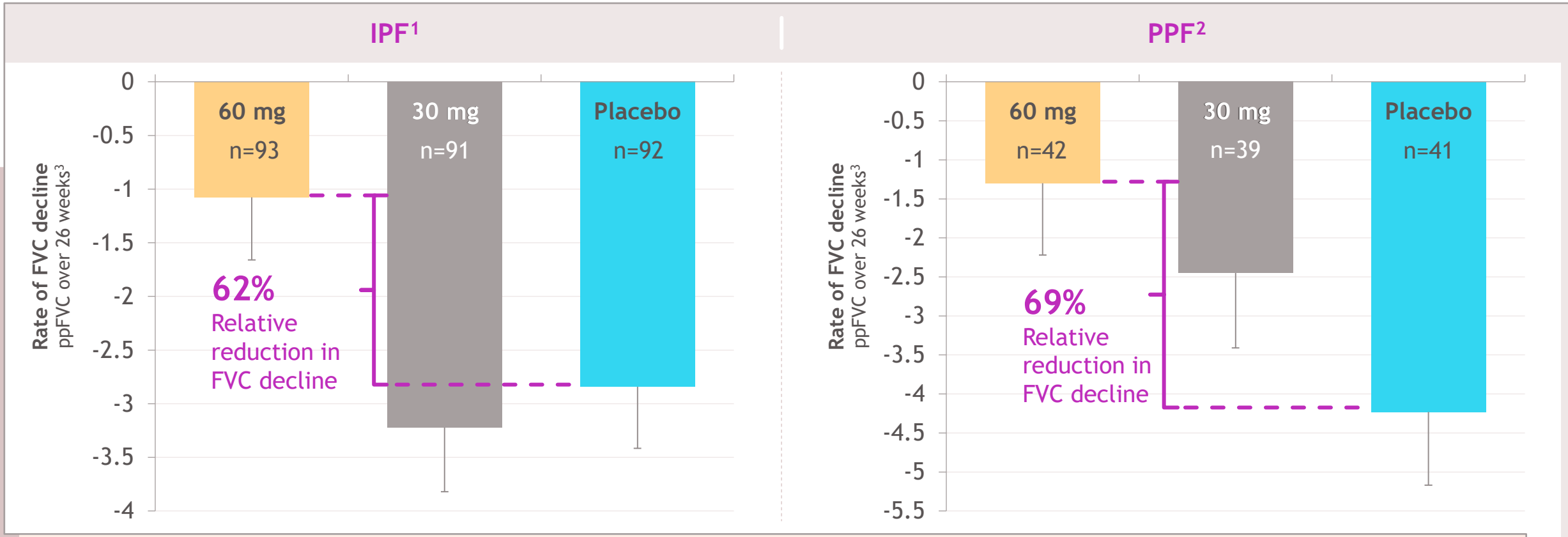
LPA$_1$ signaling stimulates fibroblast proliferation and collagen secretion

4. Fibroblast survival[1]

LPA$_1$ signaling increases fibroblast resistance to apoptosis

1. Funke M, et al. *Am J Respir Cell Mol Biol*. 2012;46(3):355-364. 2. Tager AM, et al. *Nat Med*. 2008;14(1):45-54. 3. Swaney JS, et al. *Br J Pharmacol*. 2010;160(7):1699-1713.
4. Cheng PTW, et al. *J Med Chem*. 2021 Nov 11;64(21):15549-15581. 5. Decato BE, et al. *Respir Res*. 2022;23(1):61.

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

1. Corte TJ, et al. *Am J Respir Crit Care Med*. 2023;207:A2785. 2. Corte TJ, et al. ERS 2023 [Presentation #RCT800]. 3. Mean +SEM, primary endpoint, linear model with supplementary estimand

ALOFT-IPF[1] and ALOFT-PPF[2]: Two parallel Phase 3 registrational studies

Key Inclusion:

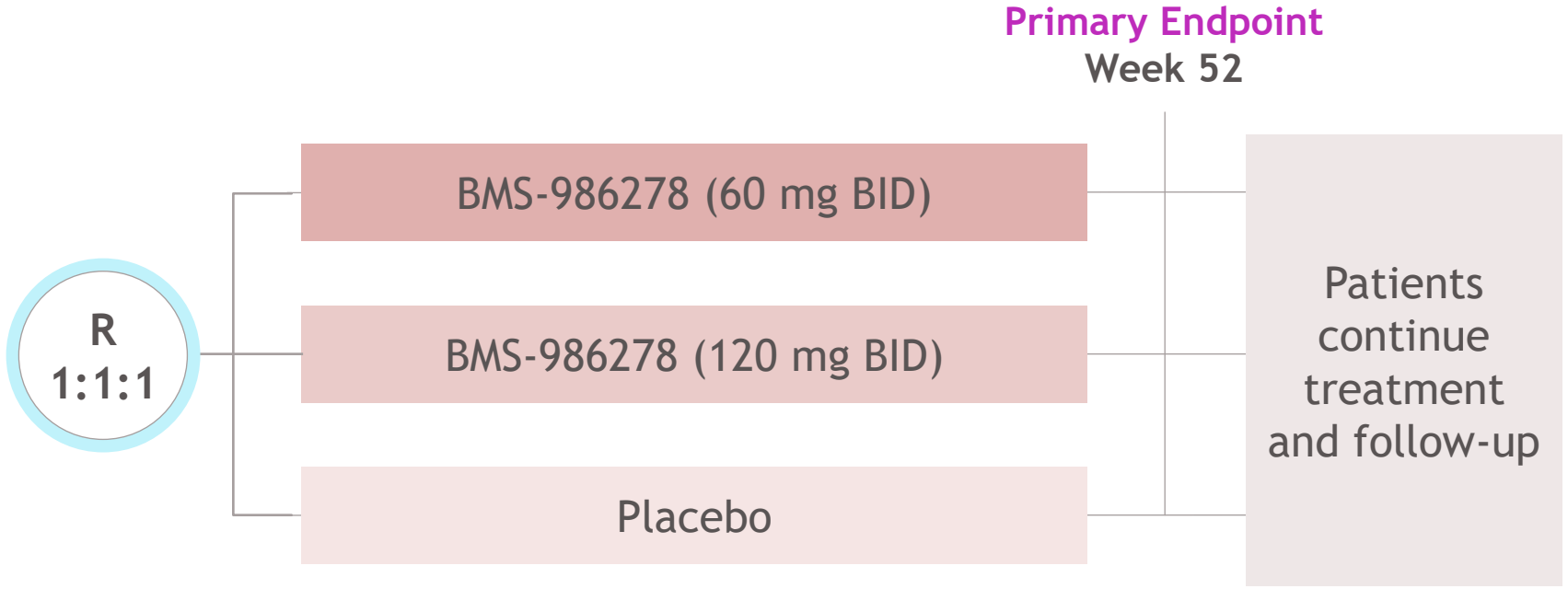
- >40 yo (IPF); >21 yo (PPF)
- FVC ≥40%, DLco >25%
- With or without concomitant background SoC

Primary Endpoint:

- Change in FVC (mL) at week 52

Key Secondary Endpoint:

- Time to disease progression
- Patient-reported outcomes
- Change in 6MWT



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)

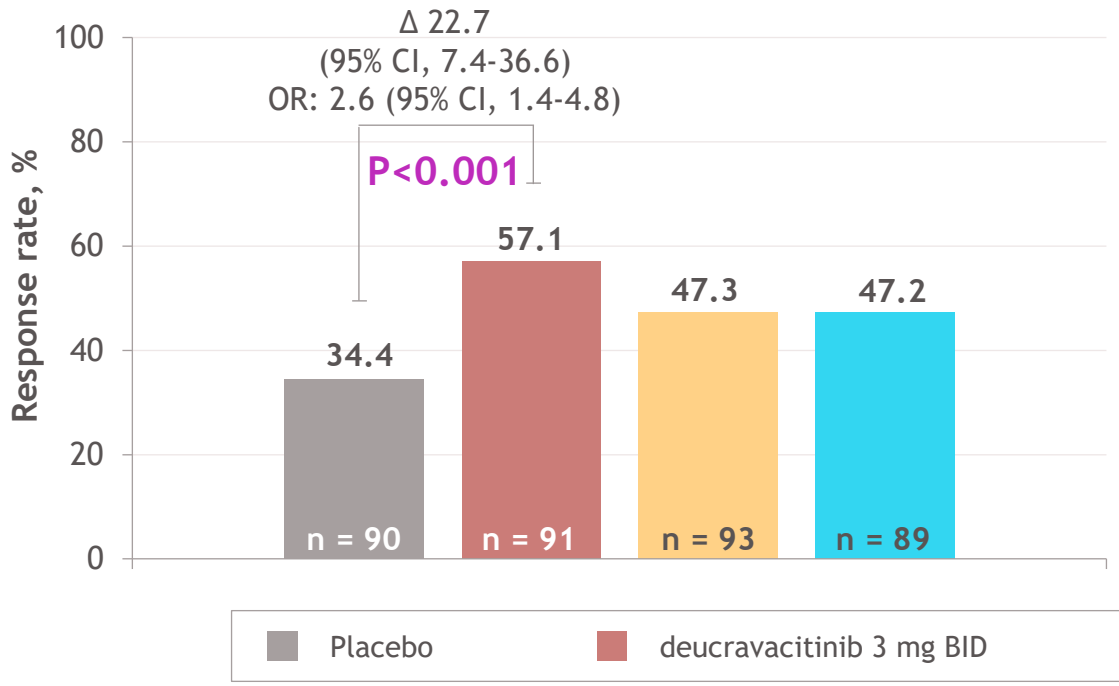
Current Treatment Landscape

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

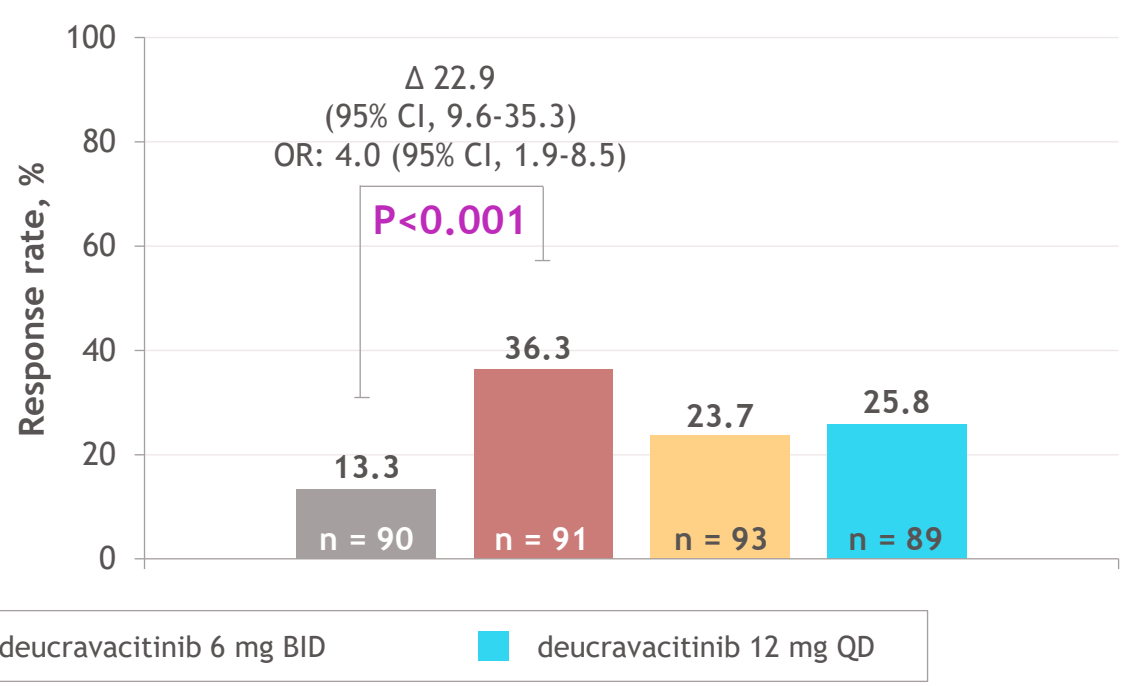
Significant Need: Opportunity for patients to have a novel, oral, effective medicine

SLE Phase 2 results across endpoints provide rationale for Phase 3

SRI(4) response rates at week 48[1]



Achievement of Lupus Low Disease Activity (LLDAS) at week 48[1]



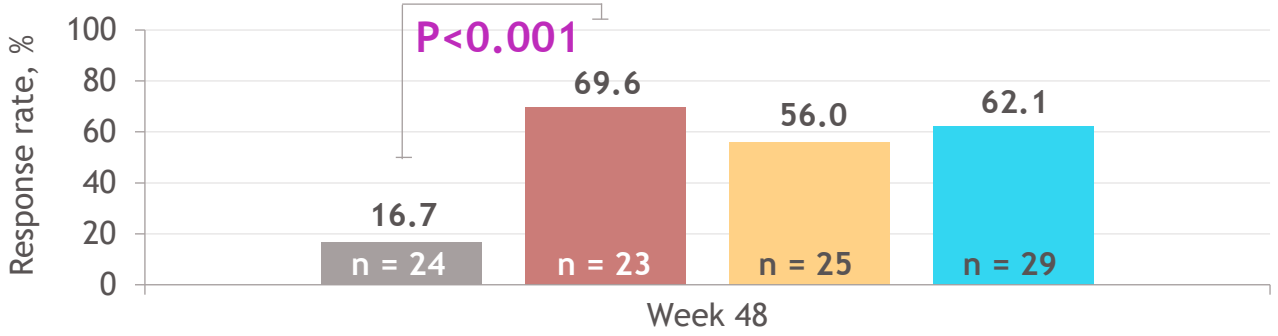
Met the primary endpoint, and all secondary endpoints achieved or meaningfully improved at week 48 with a well tolerated safety profile consistent with earlier trials

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

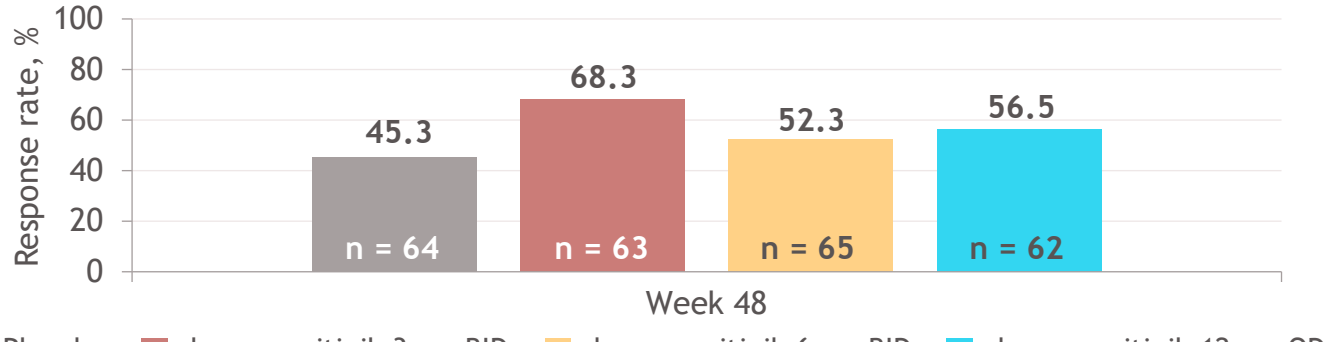
Skin Domain: CLASI-50[1,2]

Baseline CLASI activity score ≥ 10 with ≥ 50% decrease from baseline

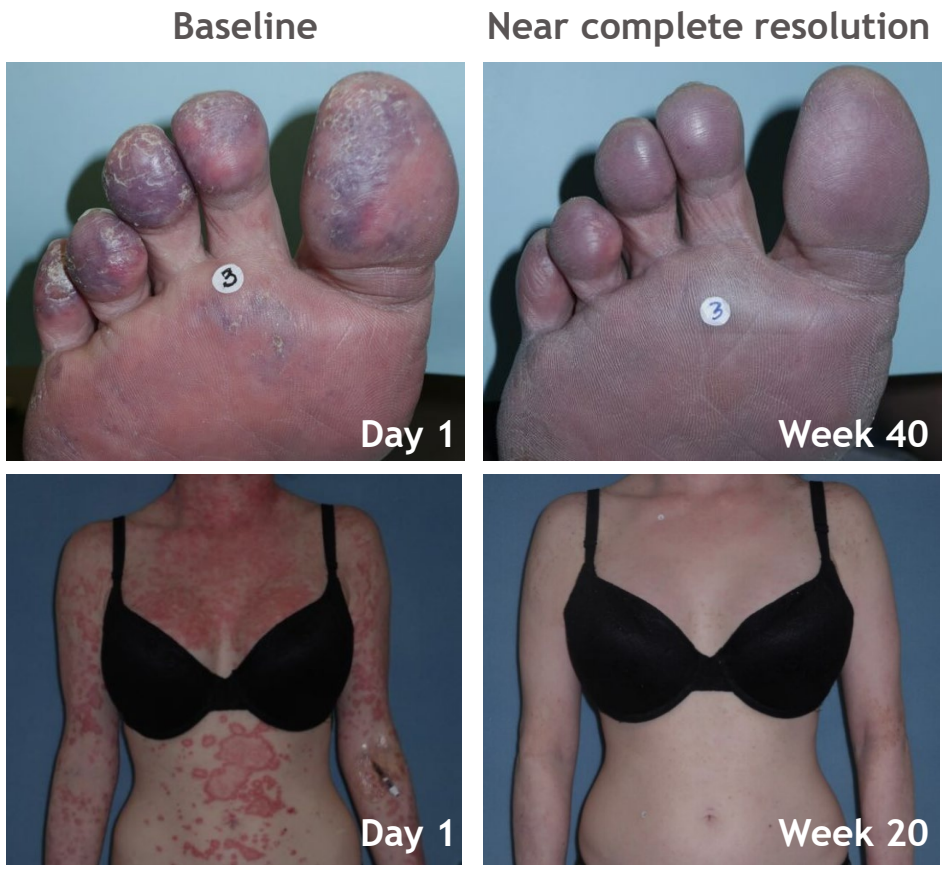


Joint Domain: Joint Count-50[1,3]

≥ 6 active (tender + swollen) joints at baseline, with ≥ 50% decrease from baseline



Patient response treated with deucravacitinib in the PAISLEY Phase 2 study[4]



1. Morand E, et al. *Arthritis & Rheumatology*. 2023 Feb;75(2):242-252. 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

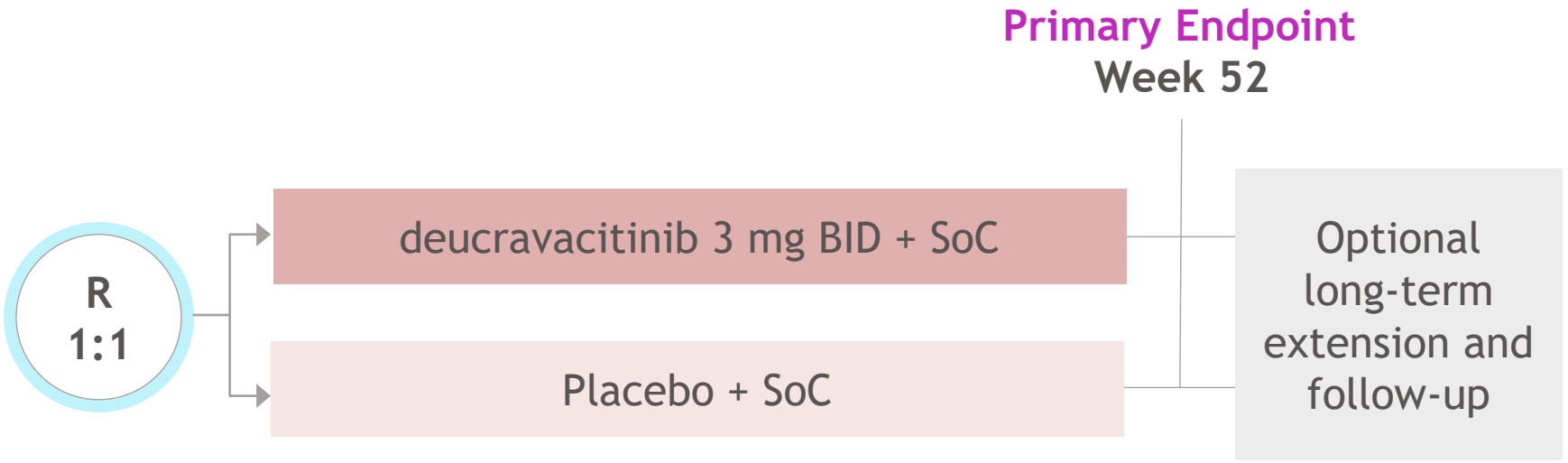
SLE Phase 3 registrational program (POETYK-SLE-1[1] and POETYK-SLE-2[2] parallel studies)

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



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[1]

Disease mechanism and genetic data support reason to believe

- Genetic studies implicate TYK2 pathways in SjS[2]
- Interferon activity is increased systemically and in tissue of patients with SjS[3]
- 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

1. https://sjogrens.org/understanding-sjogrens/treatment; 2. Khatri B, et al. *Nature Communications*. 2022;13(4287):1-17. 3. Brkic Z, et al. *Ann Rheum Dis*. 2013;72:728-735.

Sjögren's Syndrome Phase 3 study (POETYK-SjS-1[1])

Inclusion Criteria:

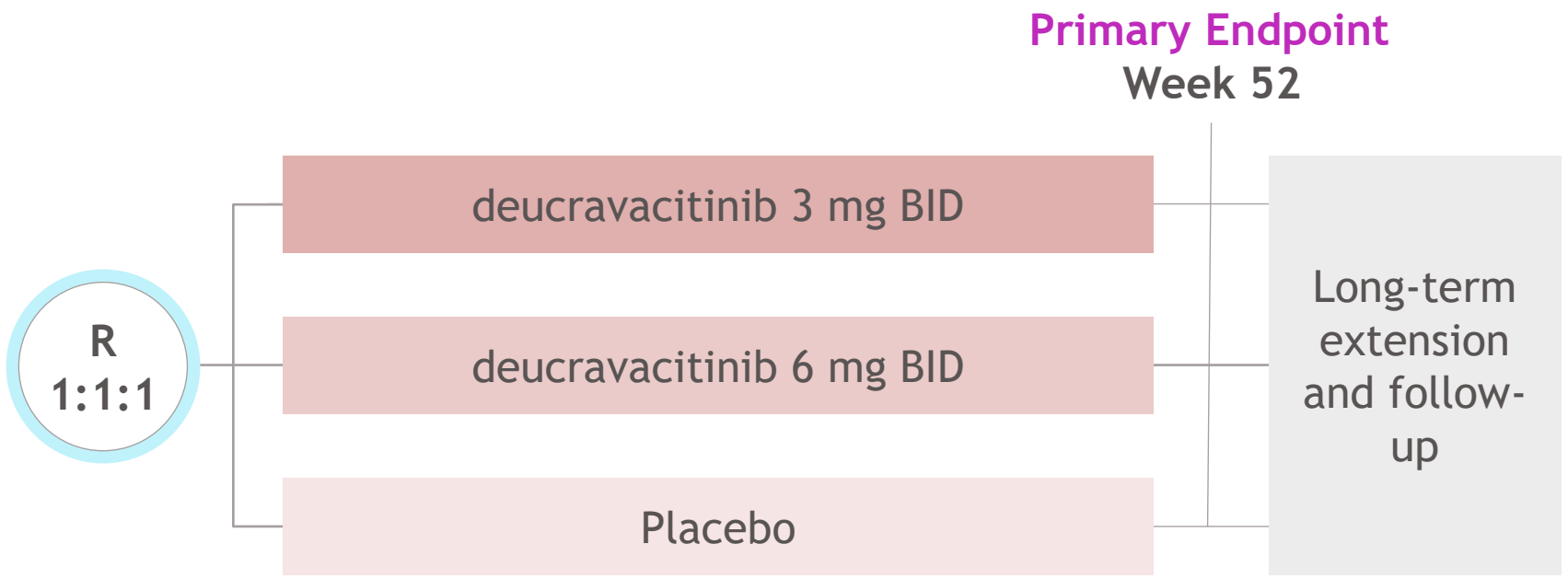
- Meet 2016 ACR/EULAR criteria with disease duration ≤ 7.5 yrs
- Anti-SSA/RO+
- ESSDAI ≥5

Primary Endpoint:

- ESSDAI change from baseline at Week 52

Key Secondary Endpoint:

- ESSPRI



Data anticipated in 2027

1. NCT05946941

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

Today	Near-Term	Future
Psoriasis	**Psoriatic Arthritis**	**Systemic Lupus Erythematosus, Sjögren's Syndrome, & Alopecia Areata**
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[1]	**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[2]

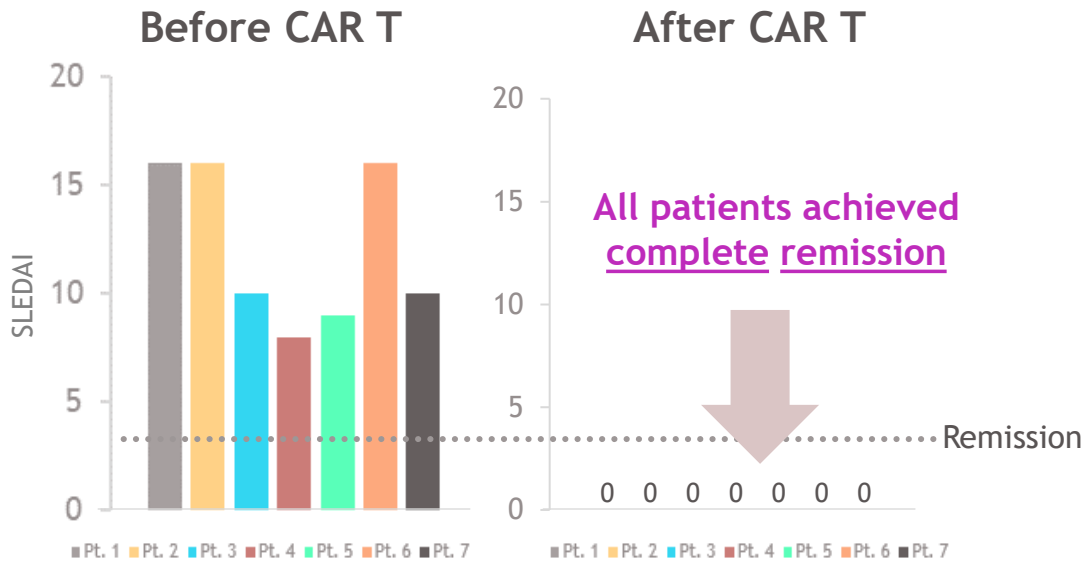
Oral, tolerable, mechanistically differentiated TYK-2 targeting agent provides broad applicability across a range of immune-mediated diseases

1. Mease PJ, Armstrong AW. *Drugs*. 2014;74:423-441. 2. Edelkamp J, et al. ESDR 2022 [poster presentation].

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

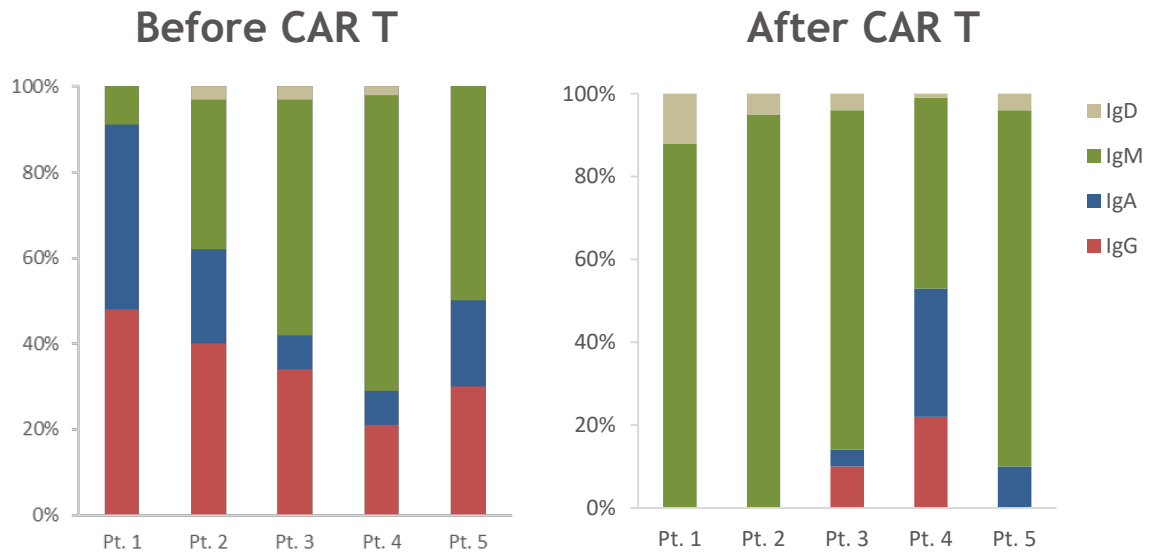
Potential transformational efficacy and favorable safety demonstrated with CD19 CAR T

Disease Remission Post CAR T Treatment[1]



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

Data Suggests Immune System Reset[2]

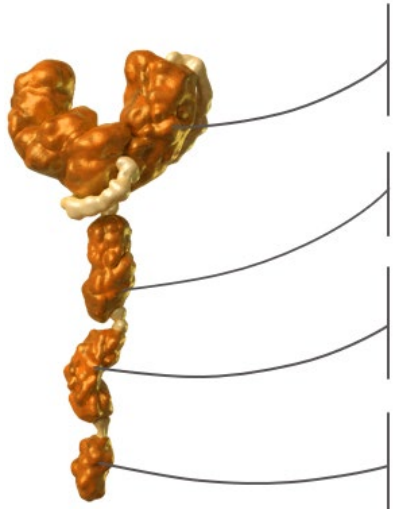


Patient immune system reset after CAR T treatment: Measurement of immunoglobulins shift from mature B-cells expressing IgA & IgG to naïve B-cells expressing IgM & IgD

1. Adapted from Taubmann, J., et al EULAR (2023): 93-94. 2. Adapted from Mackensen, A., Müller, F., Mougiakakos, D. *et al. Nat Med* **28**, 2124-2132 (2022)

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*

CD19 NEX T: Differentiated safety profile of Breyanzi with an enhanced manufacturing process

1. Makita S, et al. *Drugs Context*. 2019;8:212567. 2. Teoh J, et al. *Blood*. 2019;134:593. 3. Jayaraman J, et al. *EBioMedicine*. 2020;58:102931. 4. Weinkove R, et al. *Clin Transl Immunol*. 2019:8;e1049.

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

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

- **LPA_1 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

1. 2023 estimates from Decision Resource Group & BMS Internal Analysis across indications in the U.S. & EU5

Hematology

Addressing high unmet medical need in Hematology

Asset	Approved	Registrational†	Exploratory/PoC Studies†
Abecma (idecabtagene vicleucel)	5L+ R/R MM[1]	• 3L+ triple-class exposed MM • Sub-optimal response post-SCT	-
Breyanzi (lisocabtagene maraleucel)	• 2L LBCL • 3L+ LBCL	• R/R CLL/SLL • 2L+ FL; 3L+ FL • R/R MCL • R/R MZL	-
Reblozyl (luspatercept-aamt) for injection 25mg • 75mg	• 1L MDS • 2L TD MDS-RS • TD & NTD[2] Beta Thalassemia	• 1L NTD MDS • TD MF	Alpha Thalassemia[3]
alnuctamab	-	2-4L MM	Novel combinations in MM
BET Inhibitor (BMS-986158)			Novel combinations in MF
iberdomide	-	• NDMM post-SCT maintenance • 2-3L MM	-
golcadomide	-	1L LBCL	• 1L DLBCL • R/R PTCL[4]
GPRC5D CAR T	-	Quadruple-class exposed MM	Novel combinations
mezigdomide	-	• 2-4L MM • 2L+ MM	-

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-thalassemia[1]

1. Asia-only study

Phase 3 INDEPENDENCE 1L TD anemia in MF trial design[1]

Key Eligibility Criteria:

- MPN-associated MF
- Stable dose JAK2 inhibitor
- Transfusion dependent

Stratification:

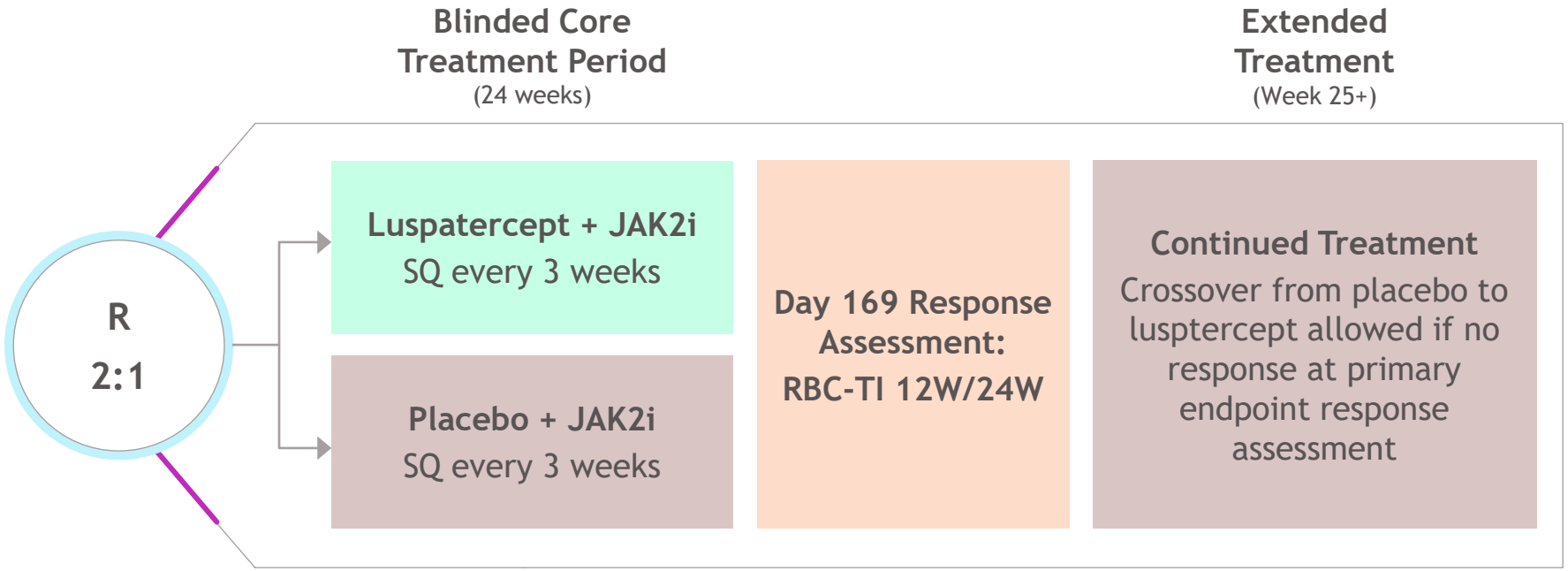
- BL RBC transfusion burden
- DIPSS (intermediate vs. high)

Primary Endpoint:

- RBC transfusion independence for ≥12 weeks

Key Secondary Endpoint:

- RBC transfusion independence for ≥16 weeks



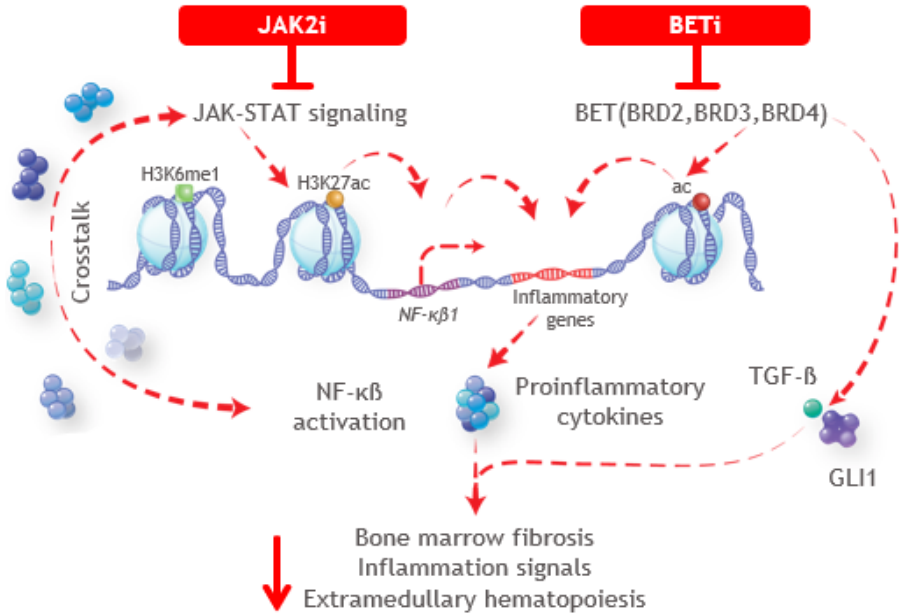
Expected data readout 2025

1. NCT04717414

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**



BETi: Phase 1/2 study ongoing in MF[2]

Dose Escalation Phase	Dose Expansion Phase
1L MF (rux-naïve) BMS-158 + ruxolitinib 15 mg BID	1L MF (rux-naïve) BMS-158 RP2D + ruxolitinib 10 mg BID
	1L MF (add-on to rux) BMS-158 RP2D + ruxolitinib previously tolerated dose
2L MF (rux-exposed) BMS-158 + fedratinib 400 mg QD	2L MF (rux-exposed) BMS-158 RP2D + fedratinib 400 mg QD
	2L MF (rux-exposed) BMS-158 RP2D monotherapy

- **Primary Endpoint:** Safety, tolerability, MTD and/or RP2D
- **Key Secondary Endpoint:** Preliminary efficacy based on SVR

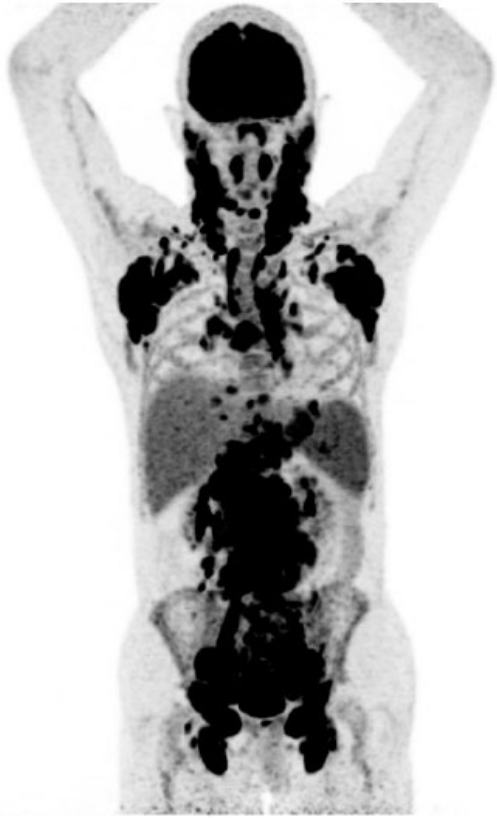
BET Inhibitors alone and in combination with JAK inhibitors have shown clinical benefit in patients with MF[1]

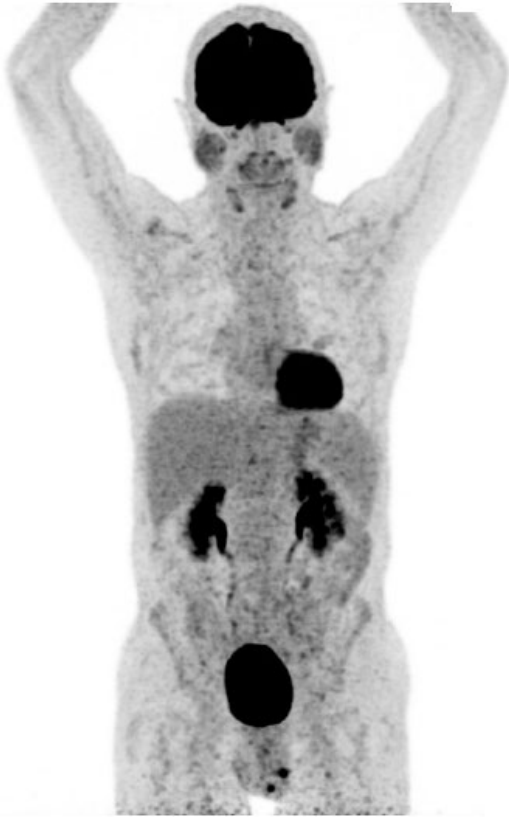
Proof-of-concept data anticipated in 2024

1. Mascarenhas J, et al. Blood 2019; 134(suppl 1):670. 2. NCT04817007

Breyanzi provides transformational benefits to patients

Before Breyanzi infusion	One month after Breyanzi infusion





Follicular Lymphoma Patient from TRANSCEND-FL[1]

1. Images used with investigator permission from TRANSCEND-FL

Best-in-class CAR T across the broadest array of B-cell malignancies

01

- Best-in-class CAR T with the broadest label in 2L+ LBCL
- Differentiated efficacy & safety profile

02

- TRANSCEND-CLL: First & only pivotal trial in high-risk R/R CLL/SLL
- Demonstrated deep and durable responses

03

- Potential best-in-disease in R/R FL
- Unprecedented data in additional lymphoma types including R/R MCL

LBCL Large B-Cell Lymphoma

MCL Mantle Cell Lymphoma

CLL/SLL Chronic Lymphocytic Leukemia/ Small Lymphocytic Lymphoma

FL Follicular Lymphoma

MZL Marginal Zone Lymphoma

Aggressive ← → **Indolent**

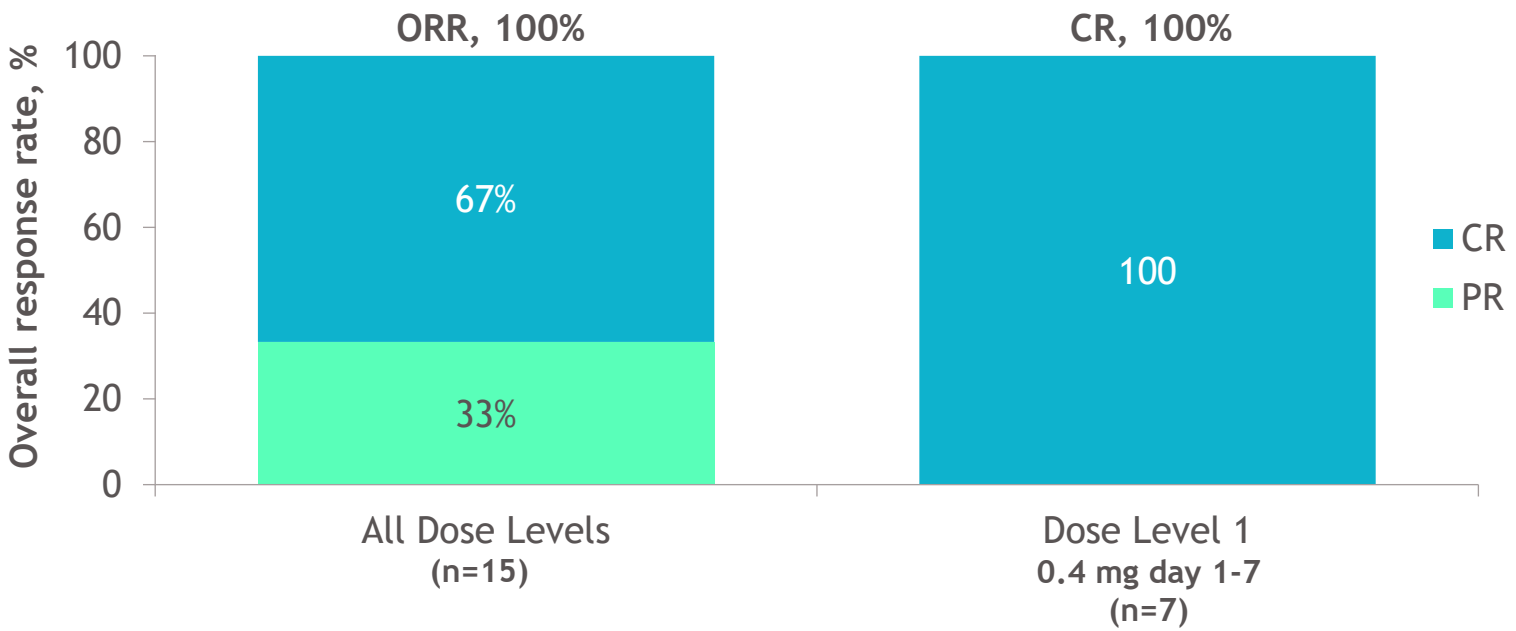
Rapidly progressive but responsive to chemotherapy and often curable

Slowly progressive and responsive to therapy but not typically curable with standard approaches

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[1]



Manageable Safety Profile[1]

- 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

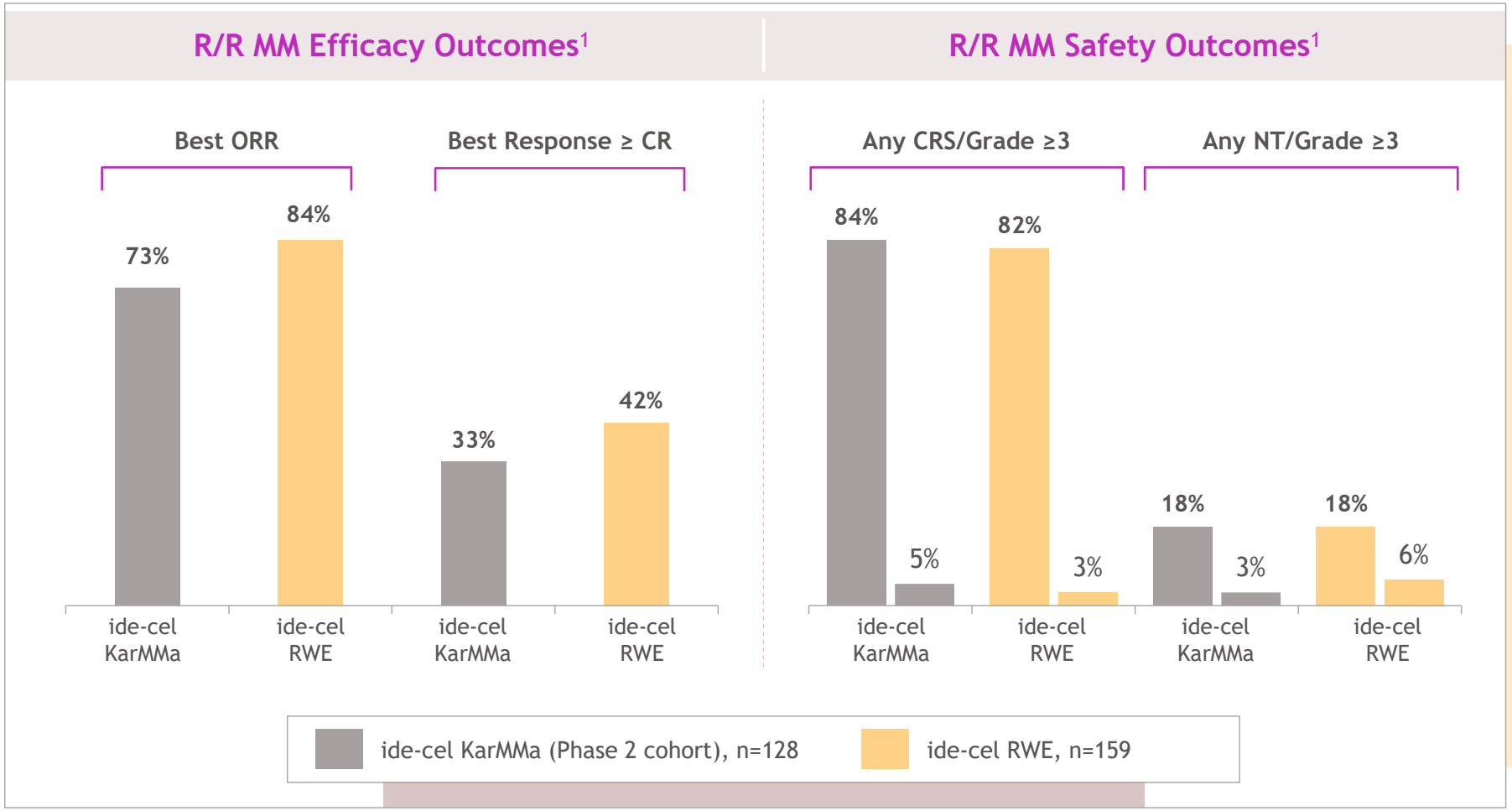
Data anticipated 2027+

1. Munoz J, et al. ICML 2023 (poster #438).

Moving into earlier lines of therapy in multiple myeloma

Today	Near-Term	Future
Abecma™ (idecabtagene vicleucel) SUSPENSION FOR IV INFUSION	**Moving into a triple-class exposed population**	**Phase 3, KarMMa-9 study initiating**
5L+ MM with differentiated real-world evidence	KarMMa-3 is currently under review in U.S., EU, and Japan	Expansion to NDMM with inadequate response to transplant

Confidence in Abecma's competitive profile further reinforced by real world evidence

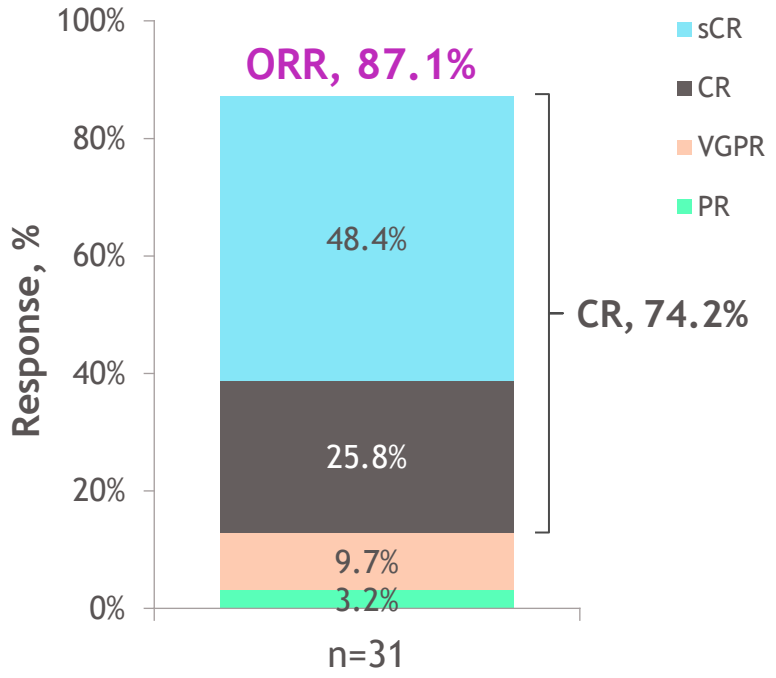


- Predictable safety profile well understood
- Real world data confirms the efficacy profile in a real world population
- Strong manufacturing reliability confirmed: 94% success rate
- Data supports CAR T before other BCMA-targeted therapies in the real world[2]

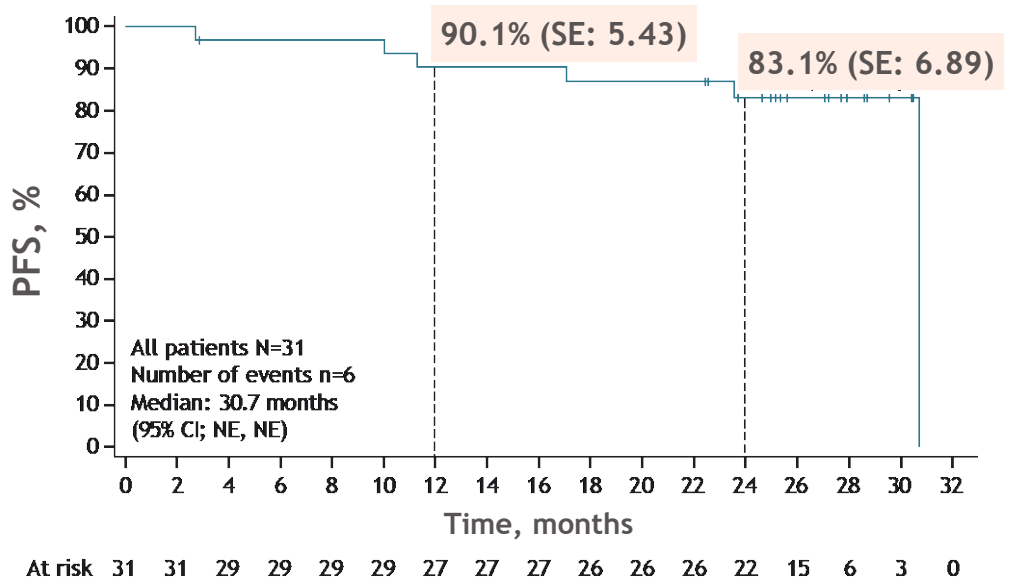
1. Hansen DK, et al. *J Clin Oncol*. 2023;41:2087-97. 2. Ferreri CJ, et al. *Blood Cancer J*. 2023;13:117. Including antibody drug conjugate, bispecific antibody, or BCMA CAR T on clinical trial

KarMMa-2[1]: Strong data supports advancing Abecma into Phase 3 KarMMa-9 study

Overall Response Rate



Progression-Free Survival



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].

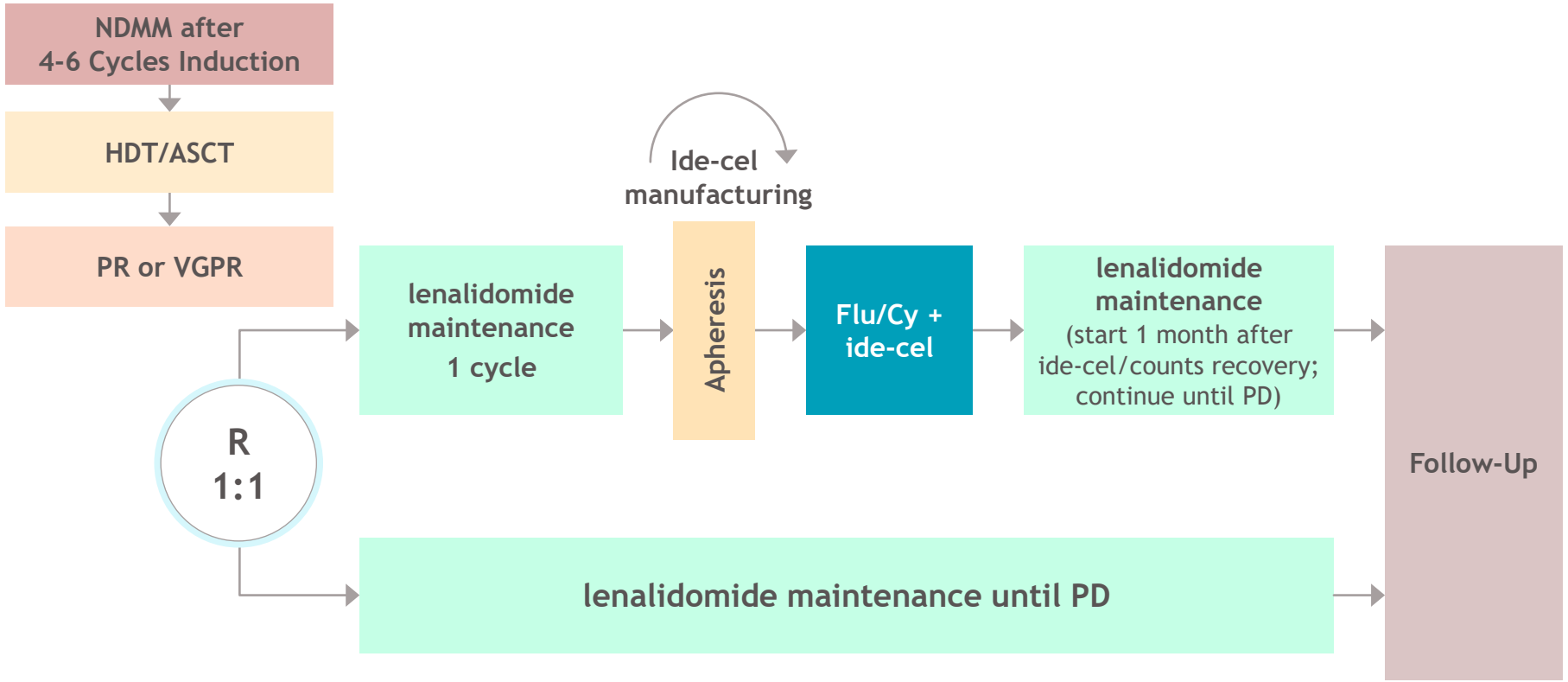
Pivotal KarMMa-9 in patients with sub-optimal response post-ASCT

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

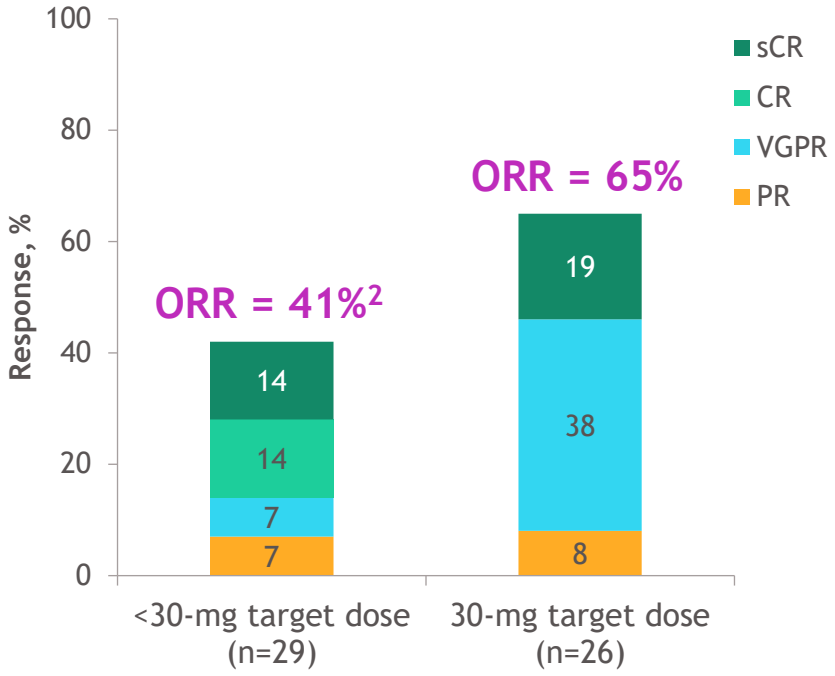


Pivotal KarMMa-9 study initiating

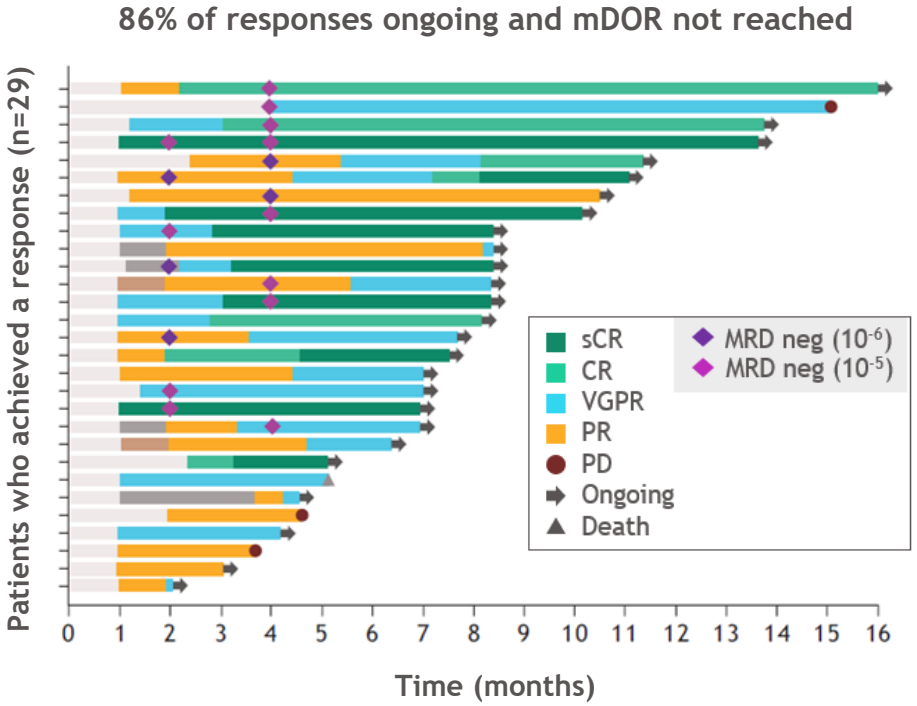
Data anticipated in 2027

Alnuctamab demonstrates deep and durable responses in RRMM

Overall Response Rate: Efficacy supports the optimal Phase 3 dose[1]



Deep and durable responses with clinically important MRD negativity[1]



Safety[1]

Grade ≥3	n=73
CRS	0
ICANS	0
Infections	10%
Hematologic:	
• Neutropenia	42%
• Anemia	25%
• Thrombocytopenia	14%

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

1. Wong SW, et al. EHA 2023 [Poster #P883]. 2. Values do not add to 41% due to rounding.

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

RRMM 1-3 prior lines *alnuctamab monotherapy vs Investigator's Choice SOC*	RRMM ≥3 prior lines (dose escalation) *alnuctamab + GPRC5D CAR T*	RRMM ≥3 prior lines (dose escalation) *alnuctamab + mezigdomide*
• 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

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

GPRC5D-targeted CAR construct

- Anti-GPRC5D domain[1]
- Hinge and transmembrane domain[1]
- 4-1BB[1,2]
- CD3-zeta[1,2]

- Critical need for new targets as the number of post-BCMA treated patients increases[3]
- 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[1]
- Overexpression of GPRC5D is associated with poor disease prognosis[1]

Matching modality to mechanism

TCE	CAR T
Repeated administration	One-time infusion

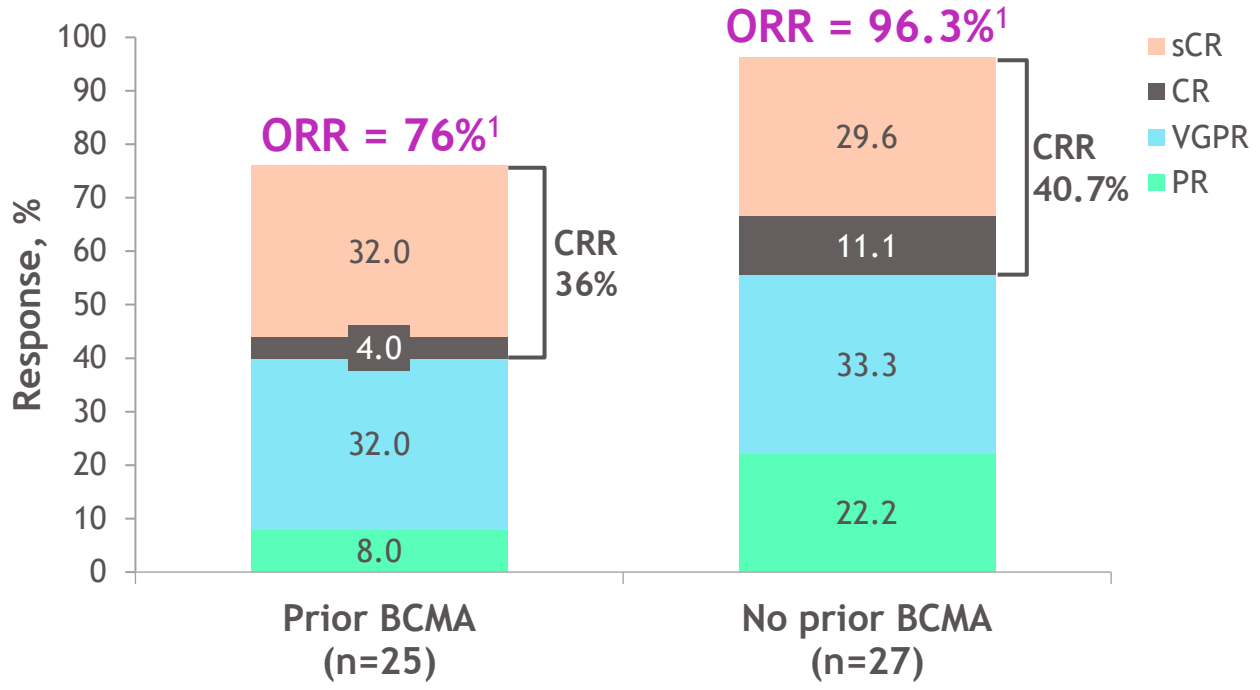
Hypothesis:

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[4]

1. Smith EL, et al. *Science Transl Med*. 2019;11:eaau7746. 2. Song D-G, et al. *Cancer Res*. 2011;71:4617-4627. 3. Mailankody S, et al. *N Engl J Med*. 2022;387:1196-1206. 4. Bal S, et al. EHA 2023 [Presentation #S193].

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



Patients with prior BCMA-targeted therapy[1], n (%)	n = 25
CAR T-cell therapy (cilta-cel, ide-cel, orva-cel, ALLO 715, others not specified)	19 (76)
Non-CAR T-cell therapy (ADC, belantamab mafodotin, TCE)	8 (32)

	All treated patients (n = 67)	
On-target/off-tumor TRAEs, n (%)	Any grade	Grade ≥ 3
Skin	14 (20.9)	0 (0)
Dysgeusia/taste disorder	12 (17.9)	0 (0)
Nails	6 (9.0)	0 (0)
Dysphagia	1 (1.5)	0 (0)
Neurotoxicity, n (%)		
ICANS-type neurotoxicity	7 (10.4)	2 (3.0)
Dizziness	7 (10.4)	1 (1.5)
Headache	7 (10.4)	0
Ataxia	2 (3.0)	0
Neurotoxicity	2 (3.0)	0
Gait disturbance	1 (1.5)	0
Dysarthria	1 (1.5)	0
Non-hematologic, n (%)		
CRS	58 (86.6)	3 (4.5)

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

1. Bal S, et al. EHA 2023 [Presentation #S193].

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

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

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

Asset (indication)	Phase 1	Phase 2	Phase 3	Projected Data Readout
mezigdomide (RRMM 2-4L)	SUCCESSOR-1[2]			2026
mezigdomide (RRMM 2L+)	SUCCESSOR-2[3]			2026
iberdomide (RRMM 2-3L)	EXCALIBER-RRMM[4]			2026
iberdomide (post-SCT maintenance)	EXCALIBER-MAINTENANCE[5]			2029

1. Hansen JD, et al. *J Med Chem*. 2020 Jul 9;63(13):6648-6676. 2. NCT05519085; 3. NCT05552976; 4. NCT04975997; 5. NCT05827016

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

Patient Segments	Newly Diagnosed Multiple Myeloma (1L)		Early Relapse and Refractory Multiple Myeloma (2L-4L)		Late Relapse and Refractory Myeloma (5L+)	
	Post-SCT consolidation	Post-SCT maintenance	anti-CD38 Sensitive/Naïve	Post anti-CD38	Triple class exposed	Quadruple class exposed
BMS Assets	Abecma (idecabtagene vicleucel)	iberdomide	iberdomide	Abecma (idecabtagene vicleucel)	Abecma (idecabtagene vicleucel)	GPRC5D CAR T
			mezigdomide	mezigdomide		
			alnuctumab	alnuctumab		
			GPRC5D CAR T	GPRC5D CAR T		

Planned
Ongoing
Approved

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

1. 2023 estimates from Decision Resource Group & BMS Internal Analysis across indications in the U.S. & EU5

Oncology

Addressing high unmet medical need in Oncology

Asset	Approved	Registrational†	Exploratory/PoC Studies†
OPDIVO (nivolumab)	26 approvals across 11 tumors	9 ongoing trials	-
Opdualag	1L melanoma	• Adj. melanoma • 2L/3L+ MSS CRC • 1L melanoma SC	• 1L/2L+ HCC • 1L NSCLC
repotrectinib[2]	-	1L ROS1+ NSCLC	NTRK Pan Tumor
subcutaneous nivolumab[1]	-	U.S. – All Q2W & Q4W indications (bridging from 2L RCC)	-
AR LDD	-	-	2L+ mCRPC
DGK Inhibitor	-	-	Solid tumors
farletuzumab ecteribulin	-	-	NSCLC & ovarian
TIGIT Bispecific	-	-	NSCLC & gastric

1. U.S. Regulatory path opens up indications with Q2W and Q4W dose including melanoma, CRC, HCC, 2L NSCLC, UC, ESCC, & Gastric 2. U.S. PDUFA: November 27, 2023
† ongoing or initiating 2023/2024

Continuing to grow Opdivo / Dual IO

26 OPDIVO approvals

10 YERVOY approvals

11 tumors

Metastatic Setting

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

Early-Stage Setting

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

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

HCPs and Healthcare System

- **Logistical**: Complex scheduling demands due to higher patient volume[1]
- **Resource utilization**: Overlapping duties for staff, inefficient patient to nurse ratios[1,2]

- **Reduces chair time (~5 min)**[3]
- **Allows rapid drug delivery**[3]
- **Reduces staff needed for administration**[3,4]
- **Improves healthcare resource utilization**[3,4]

Patients

- **Time burden**: Inconvenience[5], opportunity cost/income loss[6]
- **Emotional burden**: Loss of normality long-term survivorship and 'chronic care'[7]

- **Reduces time in clinic**[3]
- **Improves scheduling and administration**[8,9,10]
- **Improves patient QOL**[3,4,11]

[1]Lopez-Vivanco G. *Clin Transl Oncol*. 2017;19(12):1454-1461 ; [2]Huang YL, et al. *Journal of Oncology Practice*. 2018; 14(2):e82-e91; [3]O'Shaughnessy J, et al. *Eur J Cancer*. 2021;152:223-232; [4]DuMond B, et al. *J Oncol Pharm Prac*. 2021;27(5):1214-1221. [5]De Cock E, et al. *Cancer Med*. 2016;5(3):389-397. [6]Alzehr A, et al. *Support Care Cancer*. 2022;30(8):6385-6404. [7]Kim Y, Given BA. *Cancer*. 2008;112(11 Suppl):2556-2568. [8]Harvey MJ, et al. *Plos One*. 2022;17(1):e0261336. [9]Jonaitis L, et al. *BMC Proc*. 2021;15(suppl 17):25. [10]Schreiber S, et al. *Adv Ther*. 2022;39:2342-2364. [11]Dent S, et al. *Curr Oncol*. 2019;26(1):e70-e80.

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

Checkmate 67T[1]: Phase 3 study

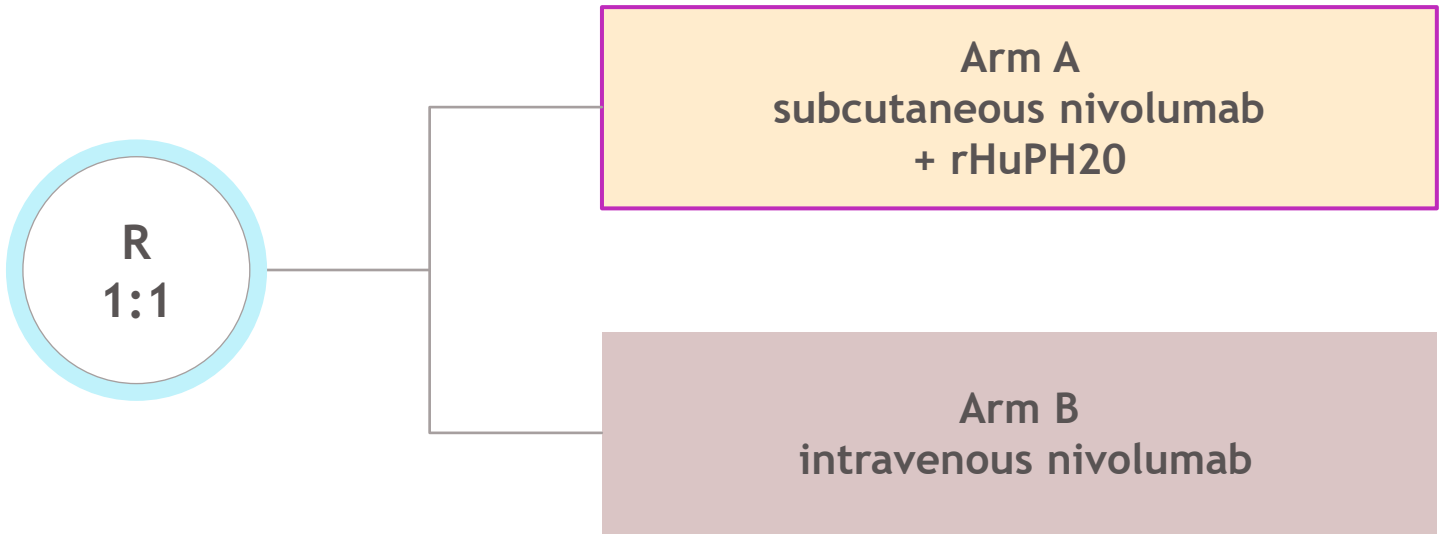
- Patients with advanced or metastatic ccRCC who have received prior systemic therapy

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

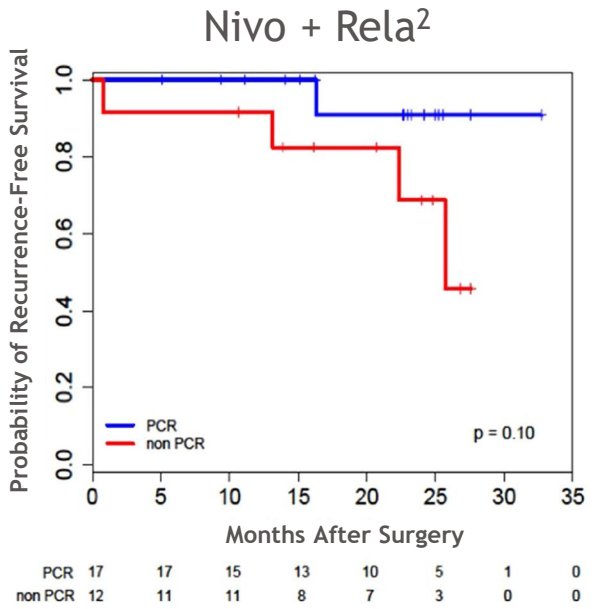
Next-generation IO medicine with significant potential to benefit patients into the next decade

Today	Near-Term	Future
Opdualag™	2/3L+ MSS Colorectal Cancer & Adjuvant melanoma	Non-Small Cell Lung Cancer & Hepatocellular Carcinoma
Strong launch supporting rapidly expanding dual IO option for 1L melanoma patients	Phase 3 trials ongoing	Phase 2 trials underway to inform path forward

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

	pCR, n/N (%)
Nivo[1]	3/12 (25)
Nivo+Rela[2]	17/30 (57)



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

Favorable safety profile

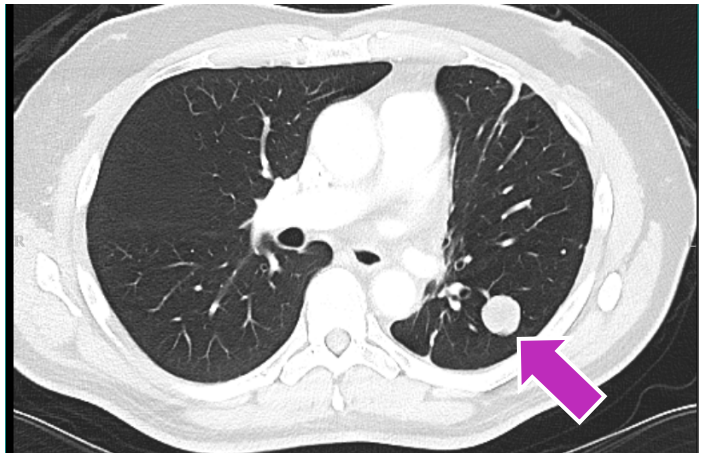
Opdualag: Potential therapy option for ~21K adjuvant patients vs ~13K 1L metastatic patients in the U.S.[3]

RELATIVITY-098 Phase 3 ongoing: **Data expected in 2026**

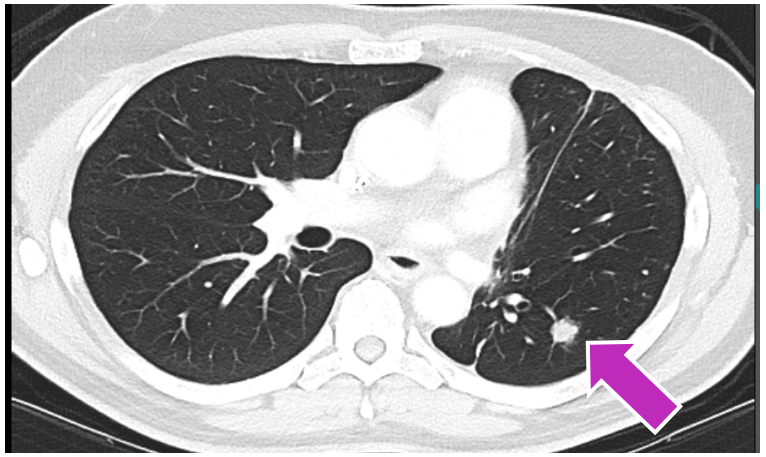
1. Amaria, et al. *Nat Med* 2018; 24: 1649-54; 2. Amaria, et al. *Nat Med* 2022; 611: 155-60; 3. Decision Resources Group, BMS Internal Analysis

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

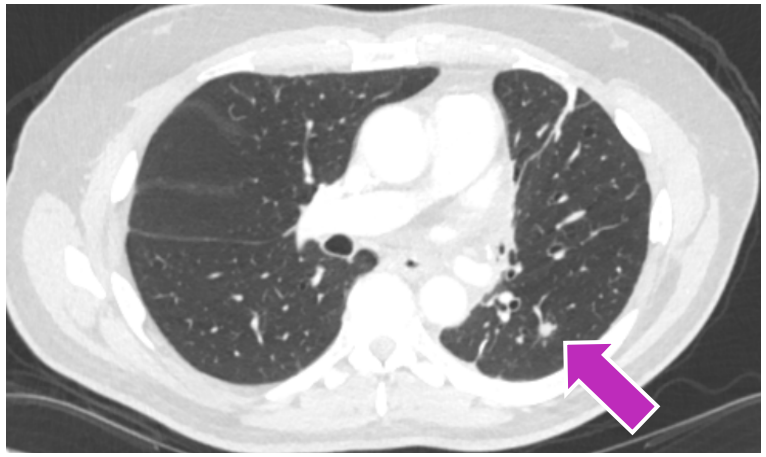
Patient with recurrent, metastatic MSS rectal adenocarcinoma after 3 lines of treatment in the metastatic setting



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

Opdualag™

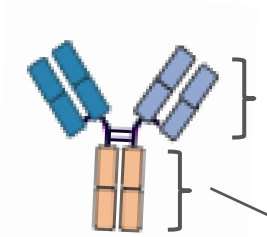
1L HCC[1]	2L + HCC (Post TKI)[2]	1L Stage IV NSCLC[3]
nivolumab + relatlimab + bevacizumab **vs** nivolumab + bevacizumab	nivolumab + relatlimab **vs** nivolumab	nivolumab + relatlimab + chemo **vs** nivolumab + chemo

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

1. NCT05337137; 2. NCT04567615; 3. NCT04623775

BMS-986442: Differentiated TIGIT & CD96 bispecific antibody in Oncology

Antagonizes TIGIT & CD96 binding to CD155



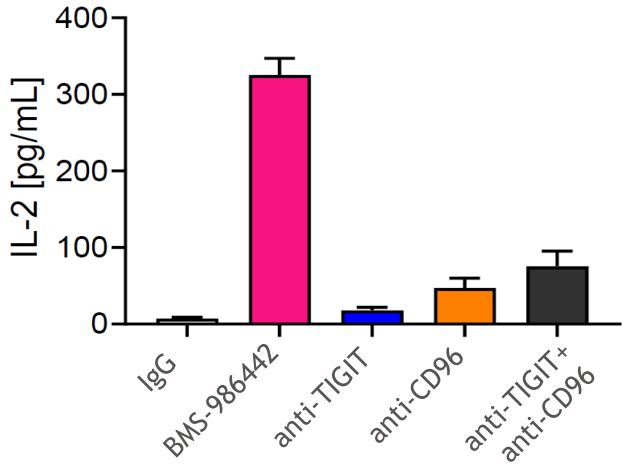
BMS-986442

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

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

MoA: Drives T & NK cell anti-tumor immunity



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

- 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]

1. Waight JD, et al., *Cancer Cell*. 2018. 2. Worboys JD, et al., *Nat. Communi*. 2023. 3. Banta KL, et al., *Immunity* 2023. 4. Dougall WC, et al., Immunol. Rev. 2017

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

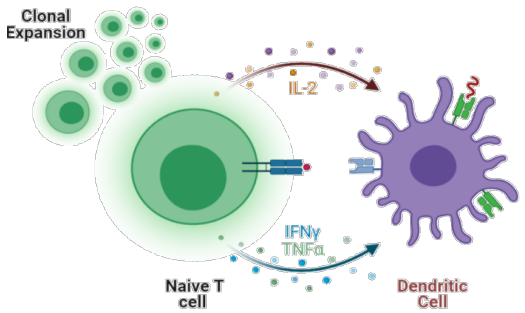
Matching modality to mechanism

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

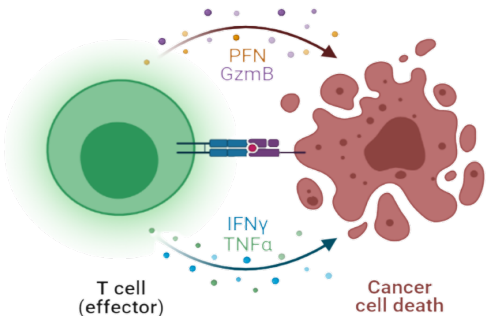
Causal human biology

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

Amplifies CD8 priming & clonal expansion



Amplifies CD8 killing of tumor cells



Path to clinical proof-of-concept

IO Resistance Mechanisms	DGKi
Low TMB	✓
Low antigenicity	✓
Low MHCI	✓
Lack of co-stimulation	✓
T cell anergy	✓

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[1] men in 2023
- 5-year OS[1] decreases from >97% to ~32.5% in the localized vs metastatic setting

Current SOC - NHT[2]

- AR is a key driver of prostate cancer and AR-targeted therapies remain current SoC
- Traditional AR antagonists (e.g., enzalutamide) inhibit AR in a reversible manner
- This AR inhibition is overcome by upregulation of wildtype (WT) or mutation of AR in cancer cells, leading to resistance:
  - AR WT amplification (~50%)[3]
  - AR mutations (~15-20%)[3]
- Post-NHT progression, limited options for patients (e.g., chemo)

AR LDD

- AR LDD induces irreversible AR degradation in a catalytic manner leading to deeper, more potent AR inhibition
- Potentially paradigm-shifting MoA overcomes resistance mechanisms to NHT including AR WT amplification and mutations
  - Preclinical models demonstrated activity in both settings
- Potential to improve efficacy, safety, & tolerability in the post-NHT setting

1. SEER 2023; 2. NHT: e.g., enzalutamide and abiraterone; 3. Robinson, et al., 2015, *Cell* 161:1215; Abida et al., 2019, *PNAS* 116:11428.

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

Open label[1]: 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

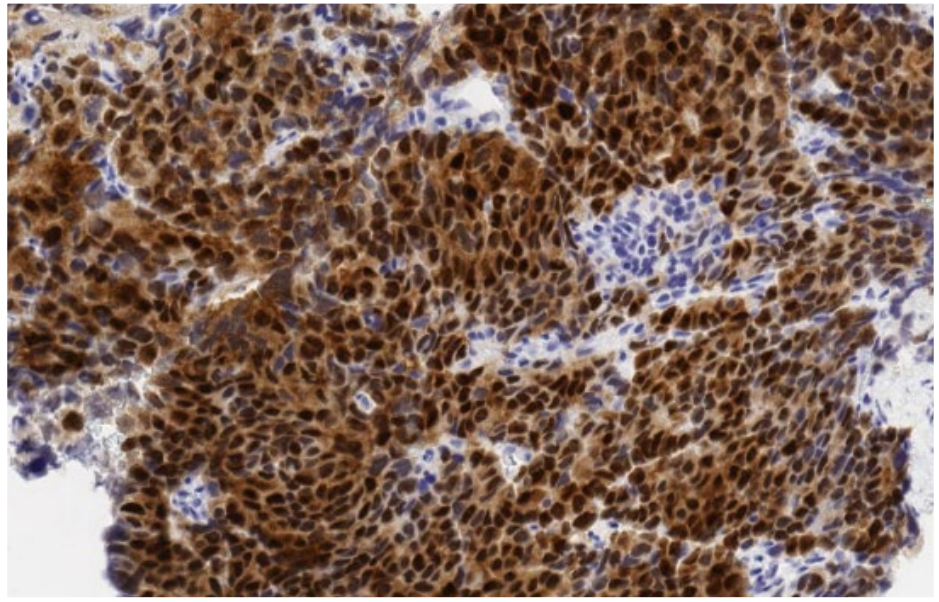
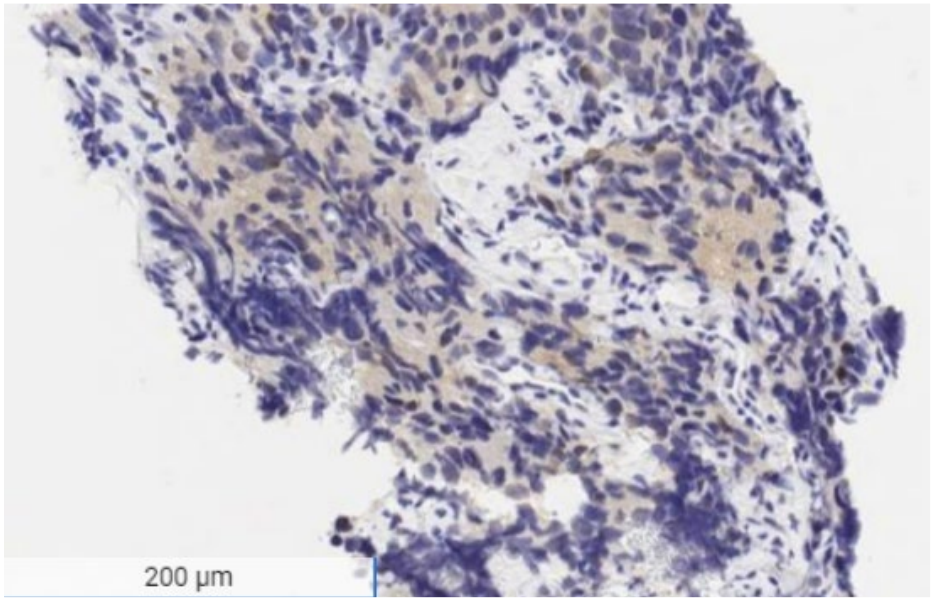
Part A
Dose escalation

Part B
Dose expansion
to optimize RP2D

Data anticipated in 2024

1. NCT04428788

AR LDD demonstrates on target AR degradation in tumor biopsy

	Screening	On Treatment
Patient Example		
	AR protein is highly expressed in tumor cells	AR protein levels degraded with AR LDD after one cycle

Source: Images from BMS internal database

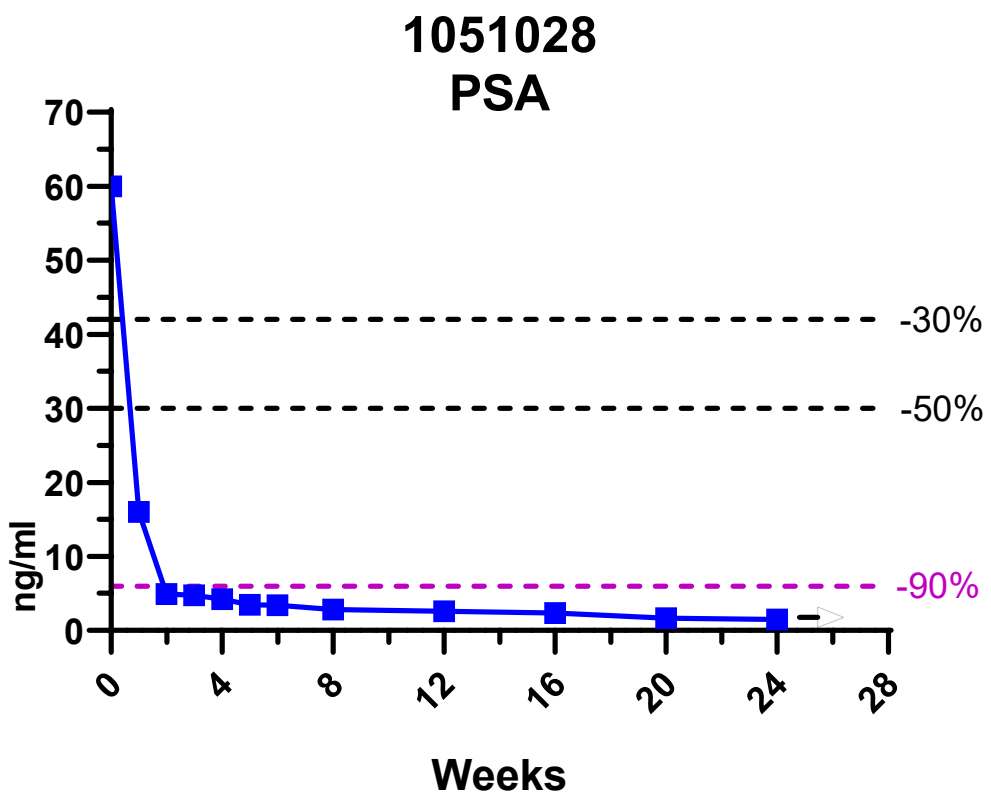
Confirmation of mechanism of action of AR LDD from first-in-human study

69 yr old male with mCRPC since 2022[1]

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

1. Patient 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

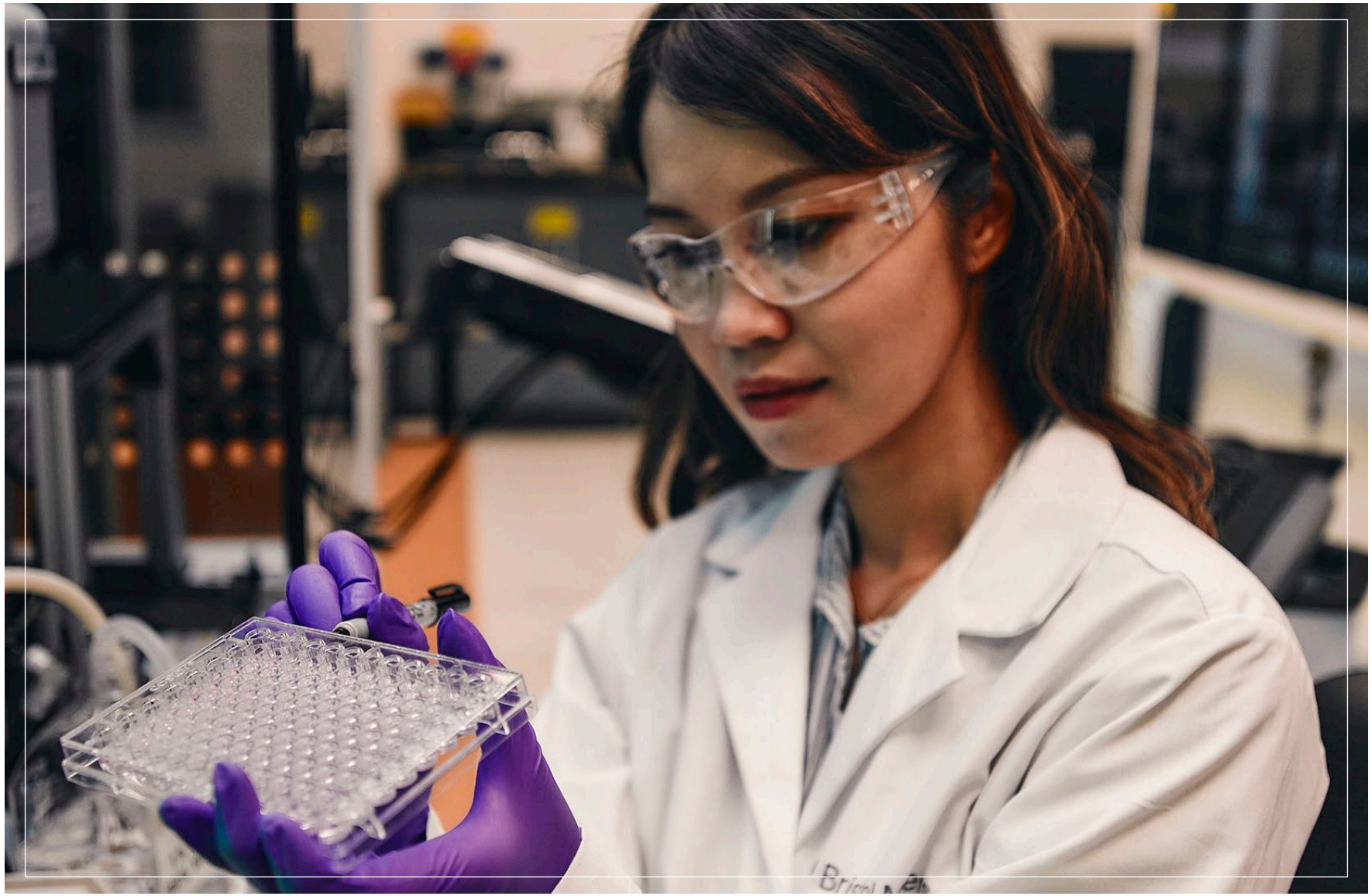
Addressing oncologic diseases impacting 1.2M+[1] patients

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



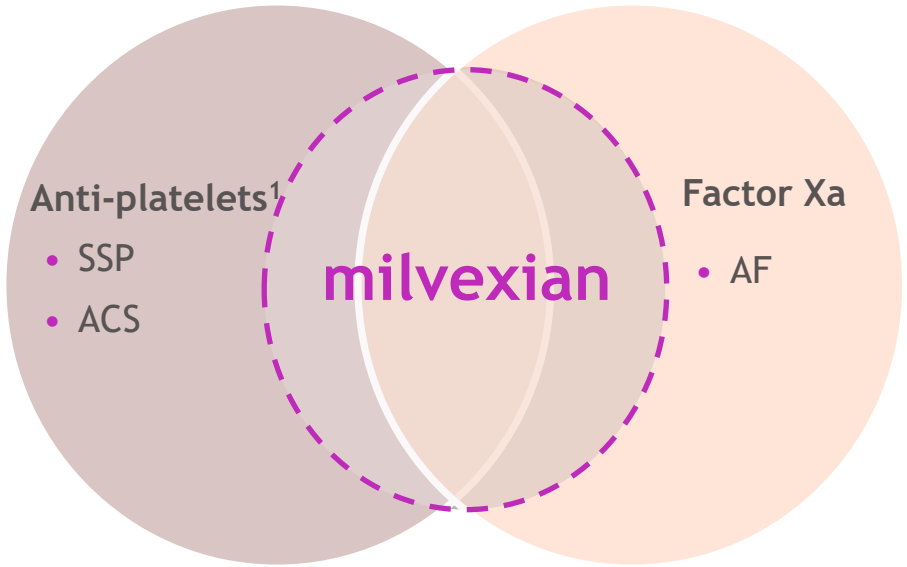
Opportunity to develop medicines in important Cardiovascular indications

Asset	Approved	Registrational†	Exploratory/PoC Studies†
CAMZYOS™ (mavacamten) 2.5, 5, 10, 15mg capsules	Obstructive Hypertrophic Cardiomyopathy	Non-obstructive Hypertrophic Cardiomyopathy	-
milvexian	-	• Secondary Stroke Prevention • Acute Coronary Syndrome • Atrial Fibrillation	-
MYK-224	-	-	• Obstructive Hypertrophic Cardiomyopathy • Heart Failure with preserved Ejection Fraction
danicamtiv	-	-	Dilated cardiomyopathy

† 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



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

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

Risk of CV events lower by[1]		Risk of VTE lower by	
▼ 48%	▼ 43%	▼ 61%	No VTE events
In patients with mild deficiency **HR 0.52**	In patients with moderate-to-severe deficiency **HR 0.57[1]**	In patients with mild deficiency **HR 0.39**	In patients with moderate-to-severe deficiency[1]

Path to clinical proof-of-concept

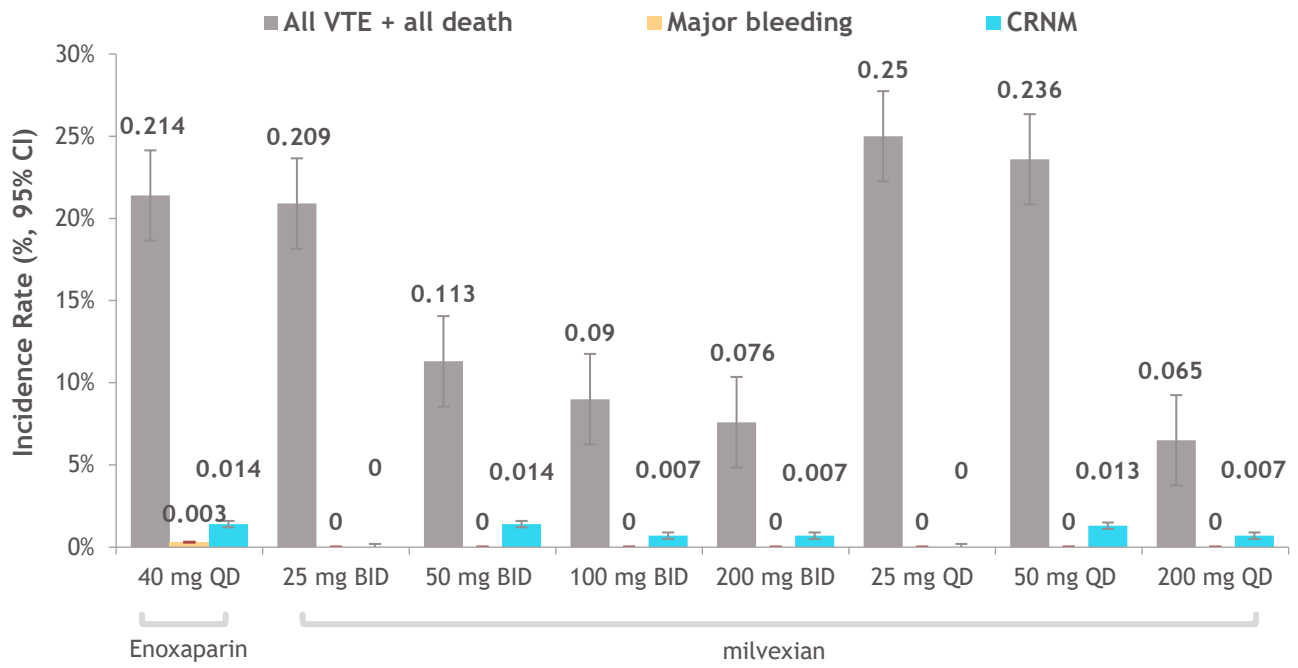
- Human genetic data
- Epidemiologic observations
- Pre-clinical models
- Phase 2 studies

1. Preis M, et al. *Blood*. 2017; Mar;129(9):1210-1215.

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

Monotherapy

AXIOMATIC-TKR study[1]



AXIOMATIC-TKR[1] Phase 2 data

Efficacy — Robust efficacy with superiority vs enoxaparin

Safety — No major bleeds observed in milvexian arms

Dose Response — Clear efficacy & no dose response in bleeding observed → distinct from existing anticoagulants

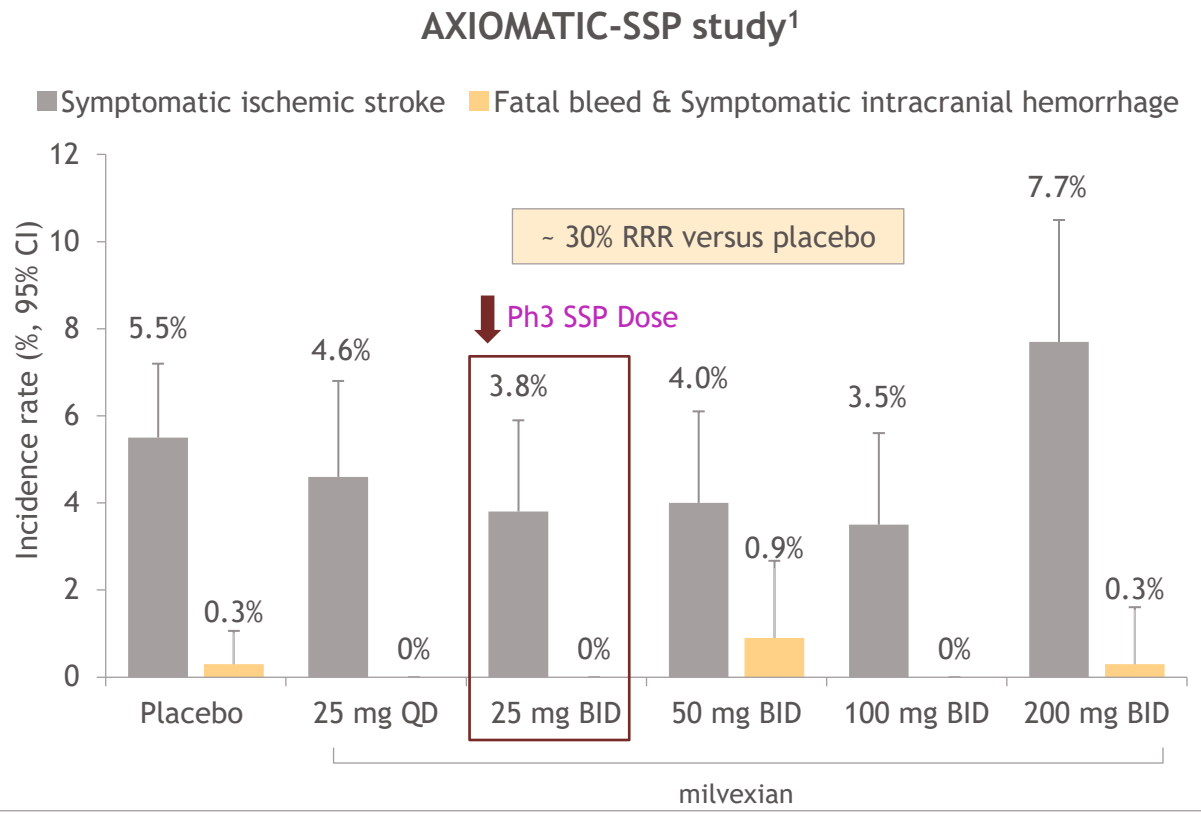
Supports Phase 3 AF trial

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

1. Weitz J, et al. *N Engl J Med.* 2021; 385:2161-2172.

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

Combination Therapy



Efficacy

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

Safety

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

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

1. Steg G, et al. *JAMA*. 2007. 2. Sorbets E, et al. *J Eur Heart J*. 2020. 3. Decision Resources Group, BMS Internal Analysis

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

Indication	Phase 1	Phase 2	Phase 3	Projected Data Readout
Secondary Stroke Prevention	LIBREXIA-STROKE[1] (Dose: 25mg BID)			2026
Acute Coronary Syndrome	LIBREXIA-ACS[2] (Dose: 25mg BID)			2026
Atrial Fibrillation	LIBREXIA -AF[3]			2027

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

CAMZYOS™ (mavacamten) 2.5, 5, 10, 15mg capsules

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

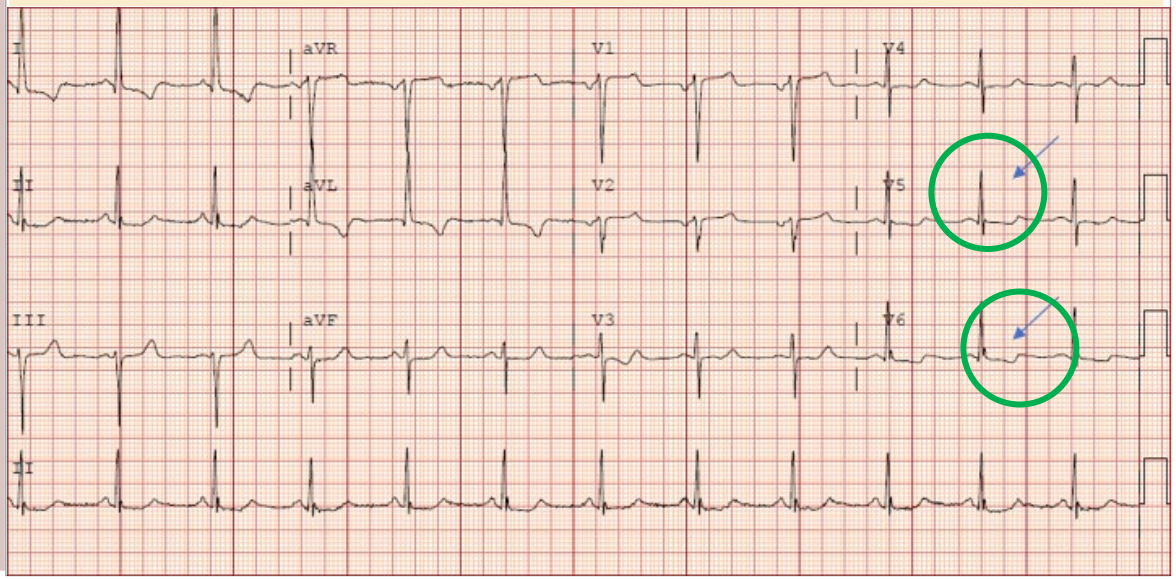
Long-Term

Potential opportunity for myosin inhibitors in HFpEF

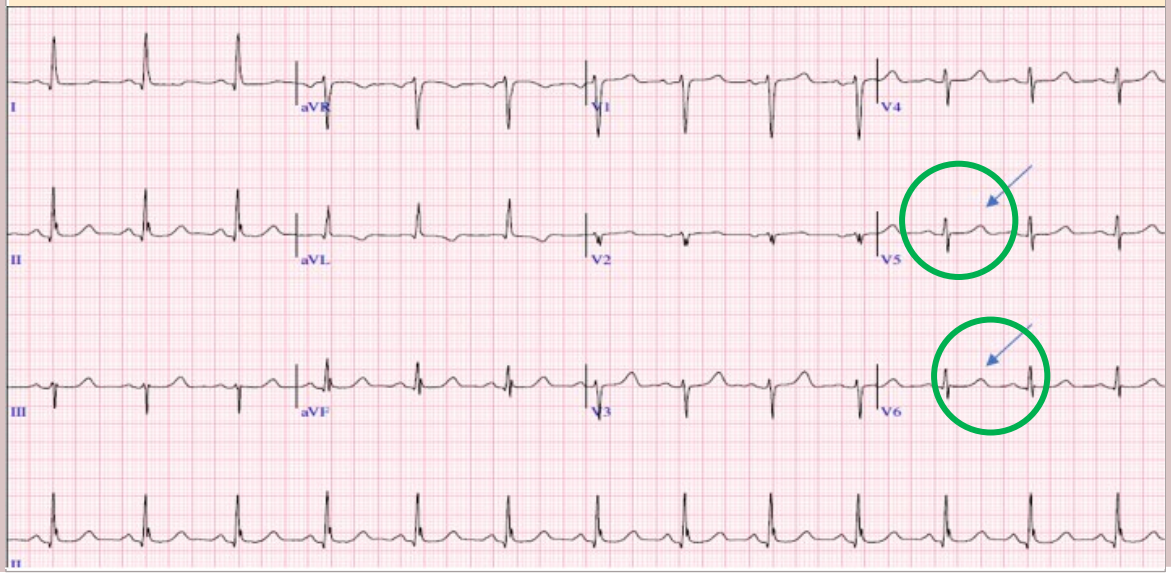
Continued evolution of data suggests disease modifying ability of Camzyos

oHCM Patient Case (Electrocardiogram)

Before Camzyos Treatment



6 Months of Camzyos Treatment

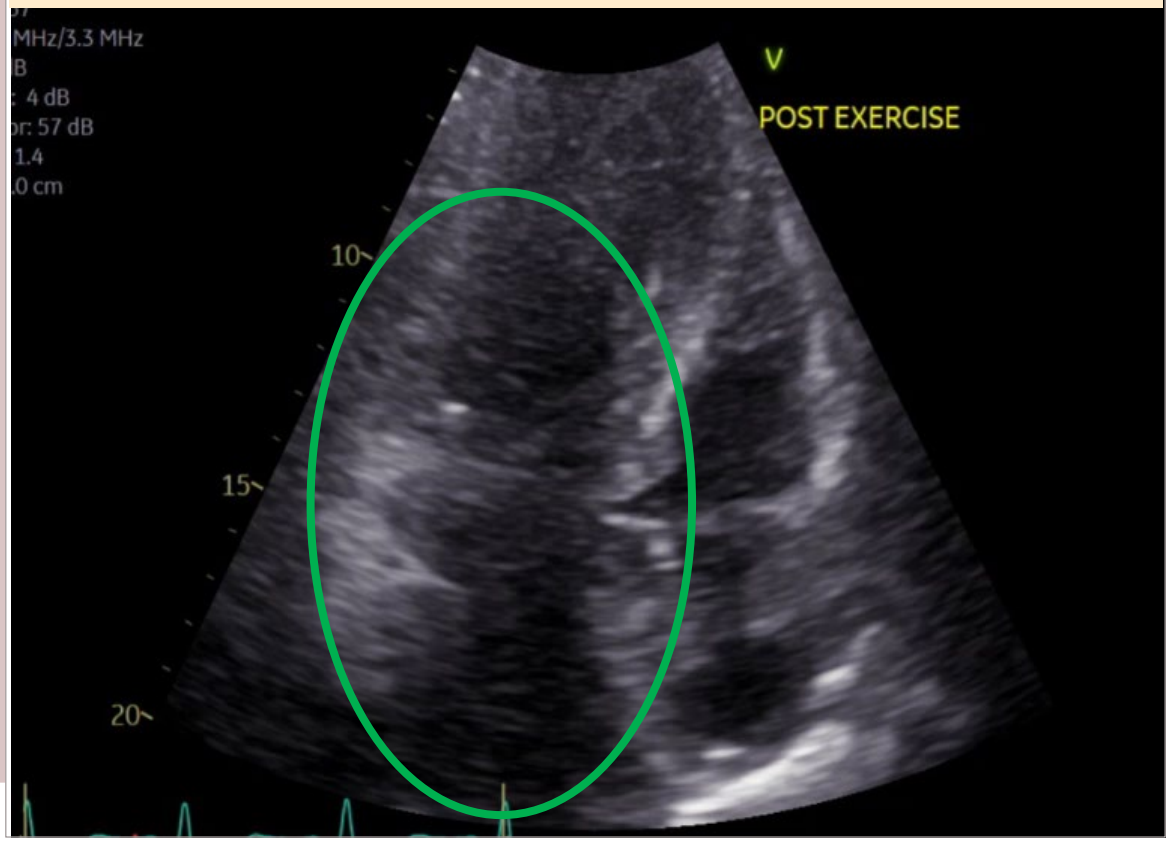


Normalizing trend in the ECG after 6 months of treatment with Camzyos

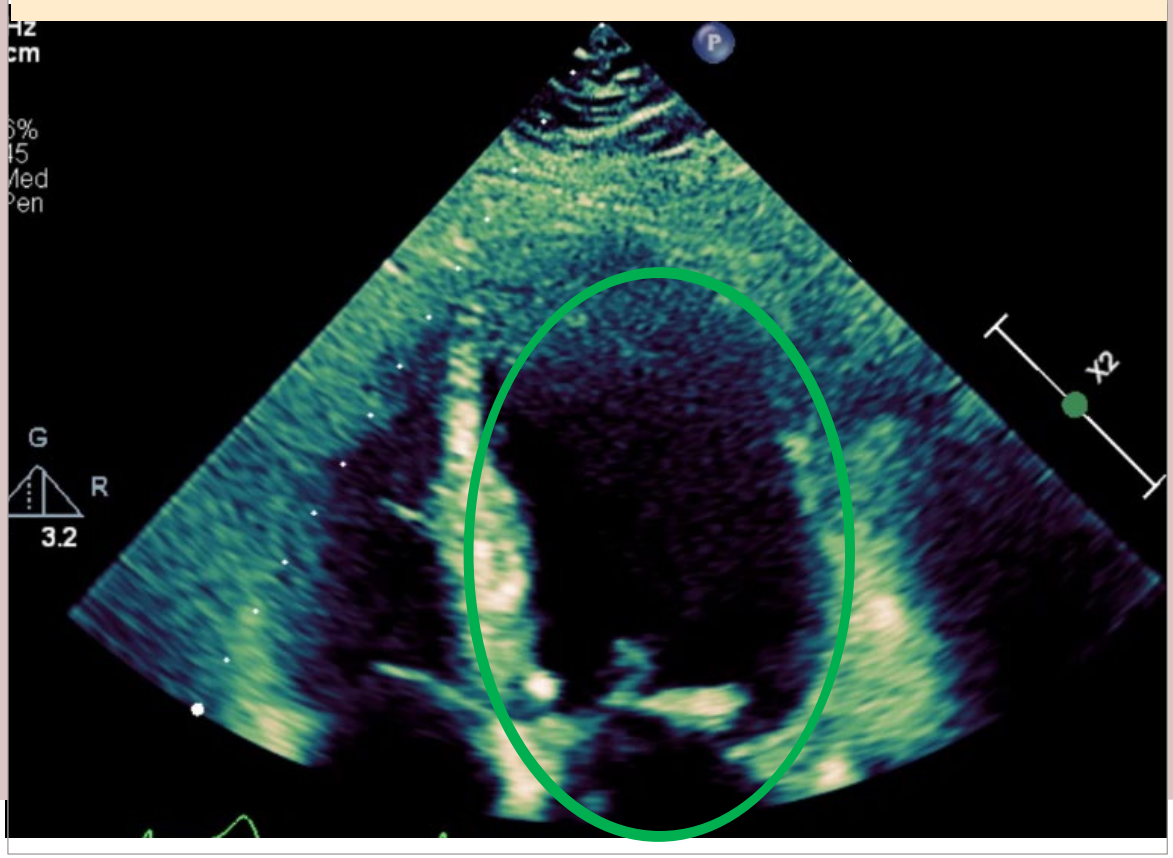
Source: Courtesy of Dr. Matt Martinez

oHCM Patient Case (Echocardiogram)

Before Camzyos Treatment



6 Months of Camzyos Treatment



Source: Courtesy of Dr. Matt Martinez

Camzyos: Phase 3 trial in nHCM underway

ODYSSEY HCM

Patients with symptomatic nHCM (NYHA Class II or III)

Key Inclusion:

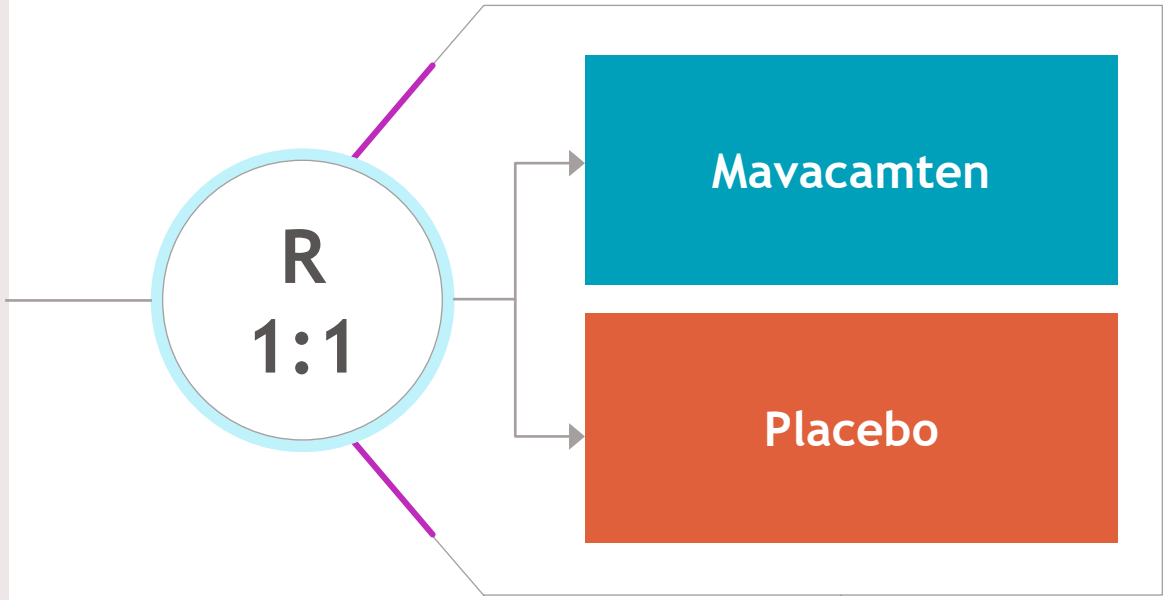
- Diagnosis of HCM per ACC/AHA and ESC guidelines
- Peak LVOT < 30 mmHg at rest / < 50 mm Hg with provocation
- NYHA Class II or III

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)

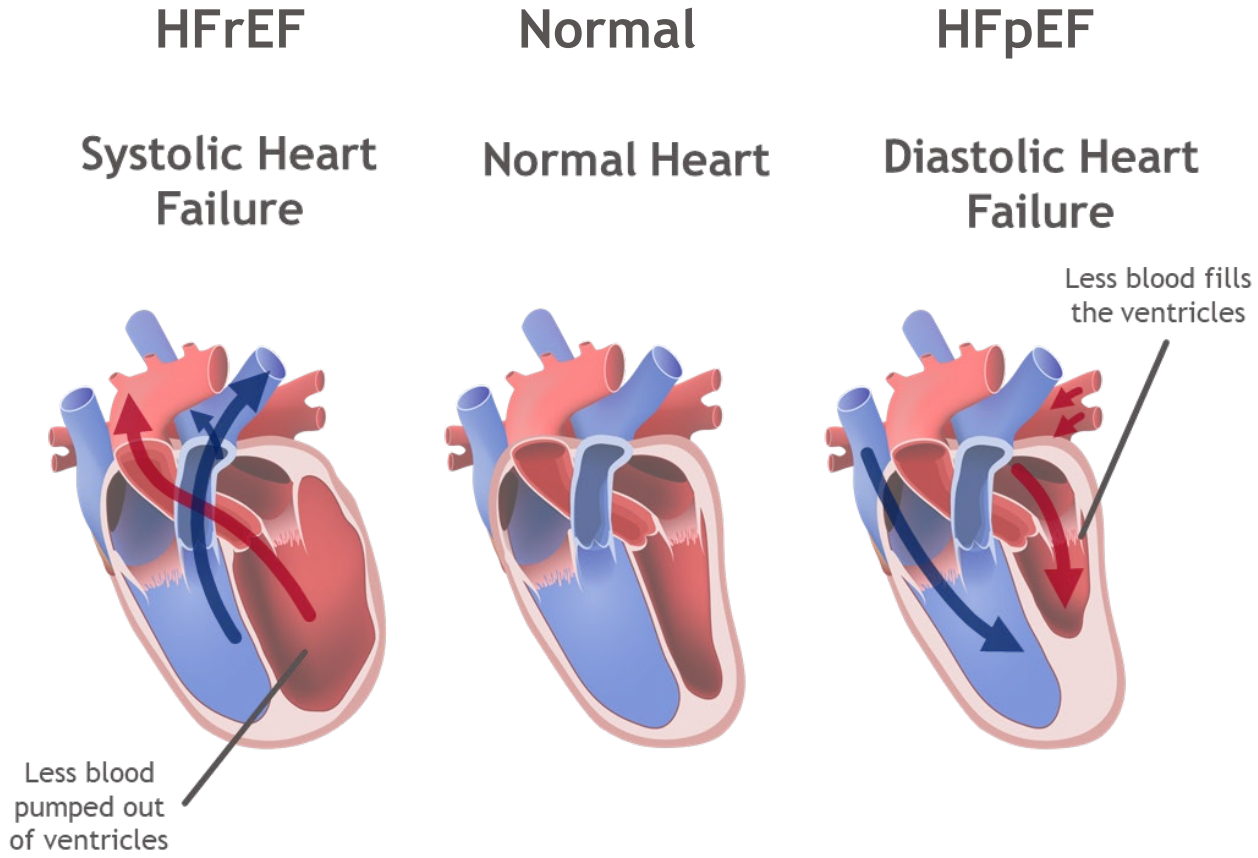


Data expected in 2025

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[1]
- 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[1]**
- **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]

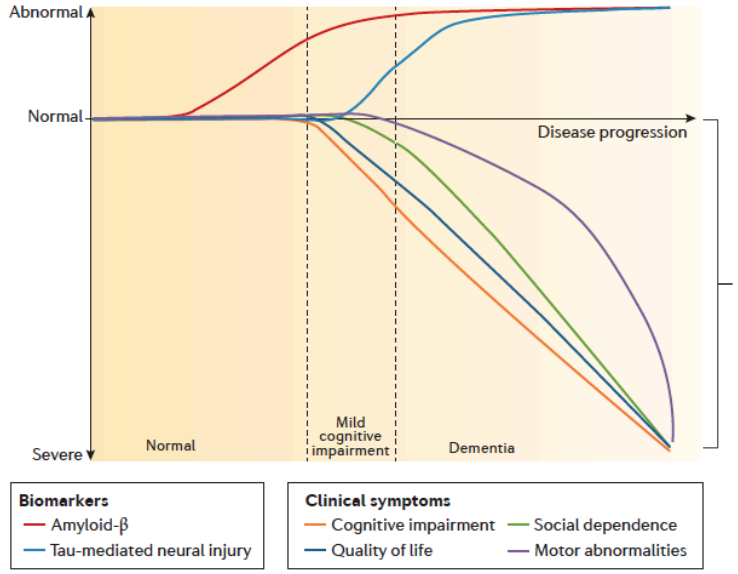


Figure adapted from *Masters et al, Nat Rev Dis Prim 2015*

- 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]

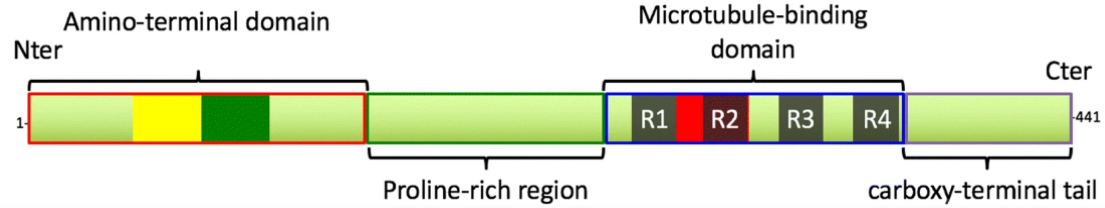


Figure adapted from *Colin et al, Acta Neuropathologica 2020*

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]

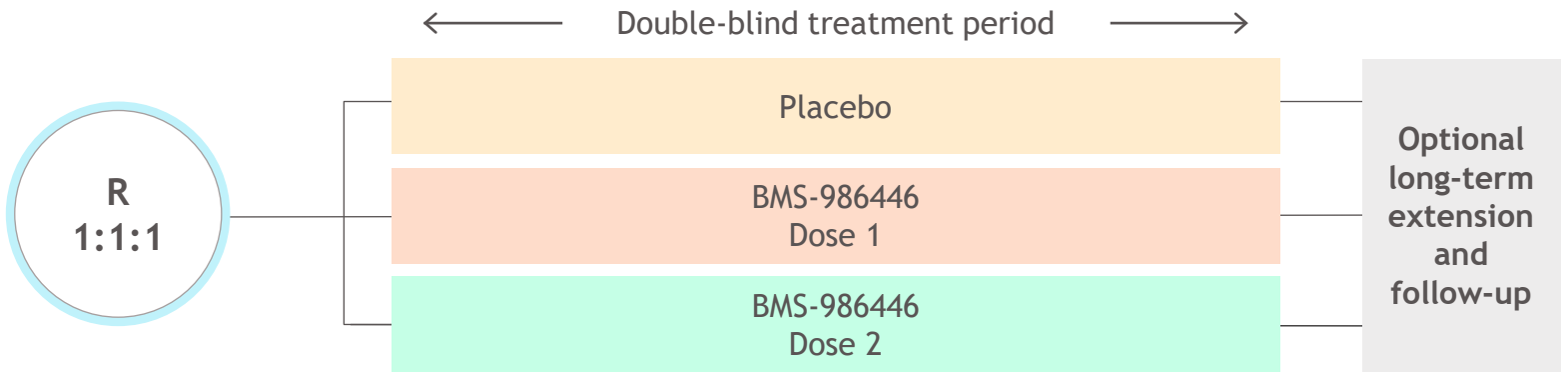
1. Horie, et al. *Brain* 2021. 2. Murray, et al. *Brain*. 2015. 3. Horie, et al. *Nat Med*. 2023. 4. Data on file: Prothena Press release 7/10/2023. 5. Dolan ADPD 2021

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

Phase 1 Findings[1]

- 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



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[1]

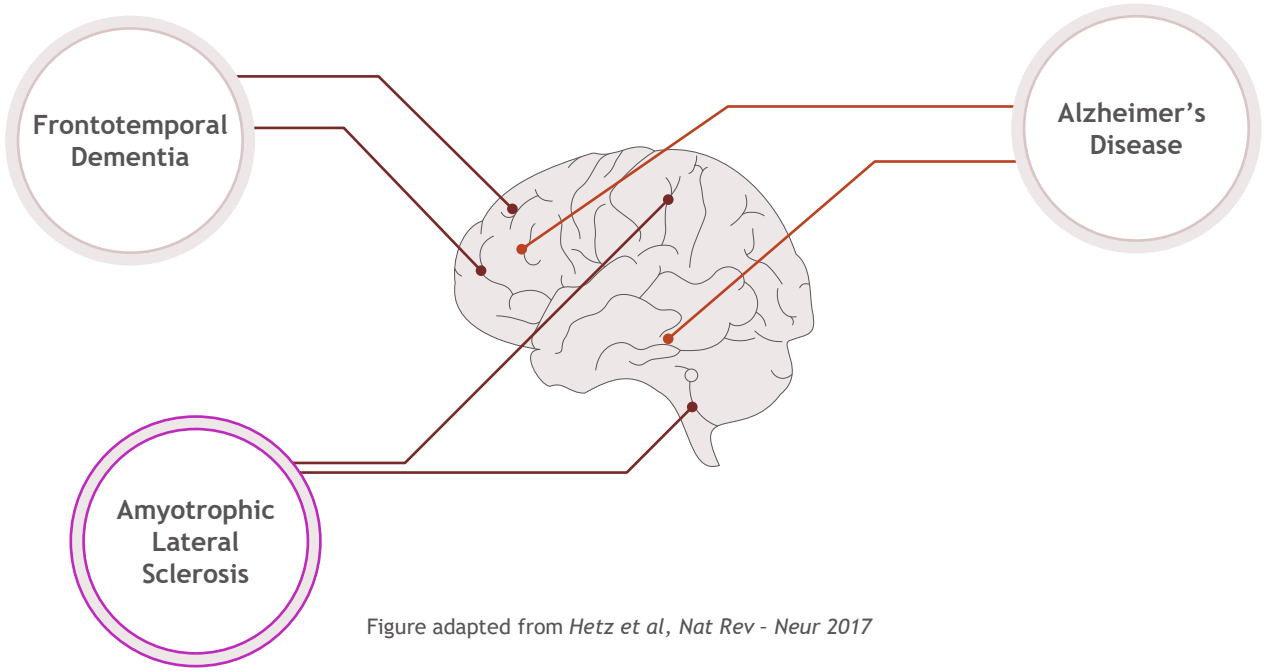


Figure adapted from *Hetz et al, Nat Rev - Neur 2017*

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

- 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

Phase 2 study in ALS initiating in 2024

ALS: Rapidly progressing & fatal neurodegenerative disease caused by death of motor neurons:

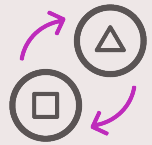
- Survival is typically only 2-5 years from symptom onset[3]
- ~39k[4] diagnosed prevalent patients in the U.S.
- Limited treatment options

1. Hetz et al, *Nat Rev - Neur*. 2017. 2. Smith et al, *Brain* 2016. 3. Masron et al, *Eur J Neur* 2020. 4. Decision Resources Group, BMS Internal Analysis;
ISR: Integrated Stress Response

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

Implementing innovative AI/Digital tools to accelerate our R&D productivity

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

Cytel · NVIDIA · nurocor · OWKIN · Columbia University Medical Center · TEMPUS · TriNetX · COTA · EDETEK · flatiron · ConcertAI

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_1 Antagonist:**
  - *Demonstrated compelling Ph2 PPF data and Ph3 studies initiating*
- **CD19 NEX T:**
  - *Ph 1 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

Today	Opportunity
OPDIVO® (nivolumab) INJECTION FOR INTRAVENOUS USE 10 mg/mL	**Subcutaneous nivolumab** • Potential to benefit patients into early years of next decade • Roughly 65-75% of IV US revenue potentially on-label at launch

Today	Opportunity
Opdualag™ (nivolumab and relatlimab-rmbw) Injection for intravenous use | 480 mg/160 mg	• 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
- ✓ Breyanzi 2L LBCL

Future Catalysts
- • Breyanzi 3L+ CLL
- • Breyanzi 3L+ iNHL

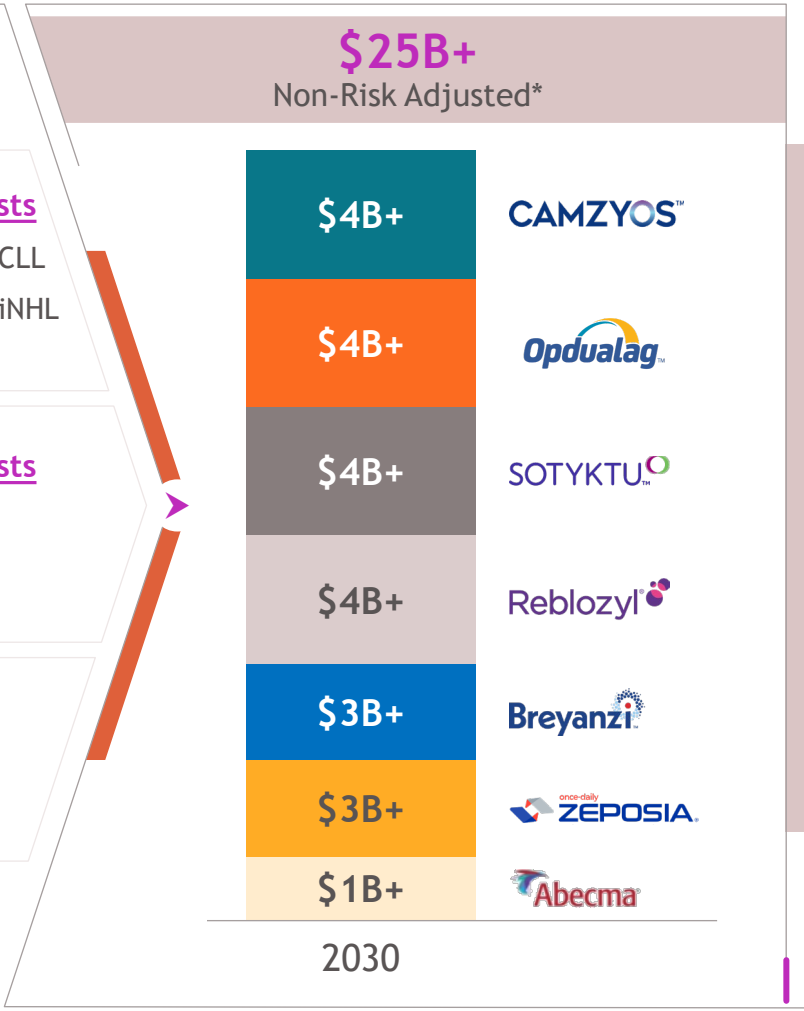
ZEPOSIA

Milestones Achieved
- ✓ Zeposia MS
- ✓ Zeposia UC

Future Catalysts
- • Zeposia CD

Abecma

Milestones Achieved
- ✓ Abecma 5L+
- ✓ Abecma 3-5L



*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

We are increasing registrational portfolio from 6 to 12 potentially first-in-class/best-in-class assets over the next 18 months

In registrational studies

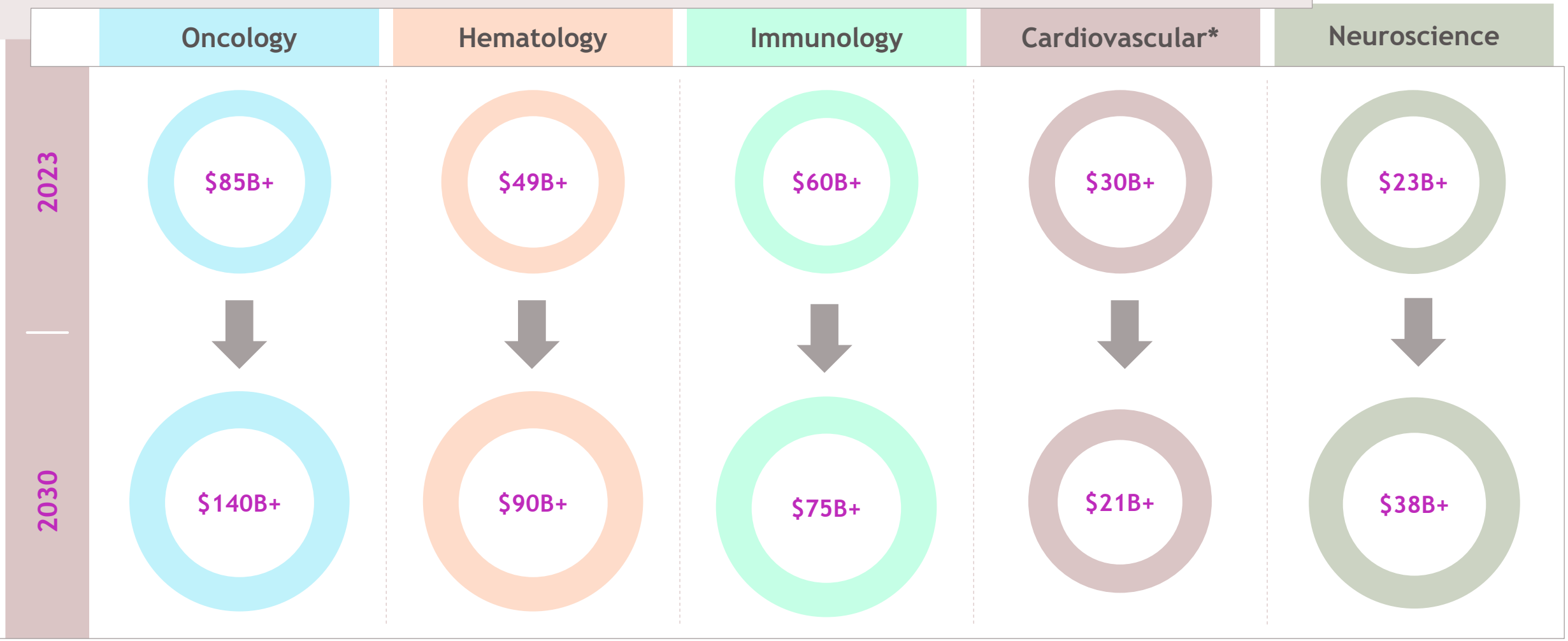
- mezigdomide
- repotrectinib*
- iberdomide
- cendakimab
- milvexian
- LPA_1 Antagonist

Registrational studies pending

- CD19 NEX T
- BET Inhibitor (BMS-986158)
- GPRC5D CAR T
- alnuctamab
- golcadomide
- AR LDD

*Filed, U.S. PDUFA 11/27/2023

Developing medicines in rapidly growing markets with significant commercial opportunities



*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

Q&A

Bristol Myers Squibb™

Acronyms

6MWT	Six Minute Walk Test	**HDT**	High Dose Therapy	**NTRK**	Neurotrophic Tyrosine Receptor Kinase
AA	Alopecia Areata	**HFpEF**	Heart Failure with Preserved Ejection Fraction	**NYHA**	New York Heart Association
ACR	American College of Rheumatology	**HFrEF**	Heart Failure with Reduced Ejection Fraction	**oHCM**	Obstructive Hypertrophic Cardiomyopathy
ACS	Acute Coronary Syndrome	**IBD**	Inflammatory Bowel Disease	**ORR**	Overall Response Rate
ADC	Antibody-Drug Conjugate	**ICANS**	Immune Effector Cell-associated Neurotoxicity Syndrome	**OS**	Overall Survival
ADT	Androgen Deprivation Therapy	**iiNT**	Investigator-identified Neurotoxicity	**PFS**	Progression-free Survival
AF	Atrial Fibrillation	**ILD**	Interstitial Lung Disease	**PI**	Proteosome Inhibitor
AI	Artificial Intelligence	**IMiD**	Immunomodulary Drug	**PoC**	Proof of Concept
ALS	Amyotrophic Lateral Sclerosis	**IND**	Investigational New Drug	**PoS**	Probability of Success
AML	Acute Myeloid Leukemia	**IO**	Immuno-Oncology	**PPF**	Progressive Pulmonary Fibrosis
APC	Antigen-Presenting Cell	**IPF**	Idiopathic Pulmonary Fibrosis	**ppFVC**	Percent of Predicted Forced Vital Capacity
AR LDD	Androgen Receptor Ligand-Directed Degrader	**IPI**	International Prognostic Index	**PR**	Partial Response
ASCT	Autologous Stem Cell Transplant	**IRA**	Inflation Reduction Act	**PsA**	Psoriatic Arthritis
BCMA	B-cell Maturation Antigen	**ISR**	Integrated Stress Response	**PSA**	Prostate Specific Antigen
BID	Twice Daily	**JAK2i**	Janus Kinase 2 Inhibitor	**PsO**	Psoriasis
BILAG	British Isles lupus Assessment Group index	**LBCL**	Large B-cell Lymphoma	**PTCL**	Peripheral T-cell Lymphoma
BL	Baseline	**LDD**	Ligand-directed Degrader	**QD**	Once Daily
CAR T	Chimeric Antigen Receptor T-cell	**LOE**	Loss of Exclusivity	**QoL**	Quality of Life
CD	Crohn's Disease	**LPA1**	Lysophosphatidic Acid Receptor 1	**R**	Randomized
CELMoD	Cereblon E3 Ligase Modulator	**mAB**	Monoclonal Antibody	**RA**	Rheumatoid Arthritis
CLASI	Cutaneous Lupus Activity Index	**MCL**	Mantle Cell Lymphoma	**RBC**	Red Blood Cell
CLL	Chronic Lymphocytic Leukemia	**mCRPC**	Metastatic Castration-resistant Prostate Cancer	**RCC**	Renal Cell Carcinoma
CPI	Checkpoint Inhibitor	**mDOR**	Median Duration of Response	**ROS**	C-ros Oncogene
CRPC	Castration-Resistant Prostate Cancer	**MDS**	Myelodysplastic Syndrome	**RWE**	Real-World Evidence
CRS	Cytokine Release Syndrome	**MF**	Myelofibrosis	**SC**	Subcutaneous
CV	Cardiovascular	**MIUC**	Muscle-invasive Urothelial Carcinoma	**sCR**	Stringent Complete Response
DIPSS	Dynamic International Prognostic Scoring System	**ML**	Machine Learning	**SjS**	Sjogren's Syndrome
DLT	Dose-Limiting Toxicity	**MM**	Multiple Myeloma	**SLE**	Systemic Lupus Erythematosus
DMPK	Drug Metabolism Pharmacokinetics	**MoA**	Mechanism of Action	**SLEDAI-2K**	SLE Disease Activity Index 2000
EGE	Eosinophilic Gastroenteritis	**mPC**	Metastatic Prostate Cancer	**SLL**	Small Lymphocytic Lymphoma
EoE	Eosinophilic Esophagitis	**MPN**	Myeloproliferative Neoplasm	**SoC**	Standard of Care
ESSDAI	EULAR Sjögren's Syndrome Disease Activity Index	**MRD**	Minimal Residual Disease	**SRI(4)**	Systemic Lupus Erythematosus Responder Index 4
ESSPRI	EULAR Sjögren's Syndrome Patient Reported Index	**MS**	Multiple Sclerosis	**SSP**	Secondary Stroke Prevention
EULAR	European League Against Rheumatism	**MSS mCRC**	Microsatellite Stable Metastatic Colorectal Cancer	**TA**	Therapeutic Area
FL	Follicular Lymphoma	**MZL**	Marginal Zone Lymphoma	**TCE**	T-cell Engager
Flu/Cy	Fludarabine and Cyclophosphamide	**NDMM**	Newly Diagnosed Multiple Myeloma	**TD**	Transfusion Dependent
FVC	Forced Vital Capacity	**NFPB**	Non-fucosylated Probody	**TIGIT**	T-cell Immunoglobulin and ITIM Domain
FXa	Factor 10a	**nHCM**	Non-obstructive Hypertrophic Cardiomyopathy	**TKI**	Tyrosine Kinase Inhibitor
FXIa	Factor 11a	**NHL**	Non-hodgkin's Lymphoma	**TKR**	Total Knee Replacement
GI	Gastrointestinal	**NHT**	Novel Hormone Therapy	**TRAE**	Treatment-related Adverse Event
GPRC5D	G Protein Coupled Receptor, Class C, Group 5, Member D	**NSCLC**	Non-small Cell Lung Cancer	**TYK2**	Tyrosine Kinase 2
HbF	Fetal Hemoglobin	**NT**	Neurotoxicity	**UC**	Ulcerative Colitis
HCC	Hepatocellular Carcinoma	**NTD**	Non-transfusion Dependent	**VGPR**	Very Good Partial Response

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

Changes to the Development Pipeline

	Phase I	Phase II	Phase III
New or Phase Transition	✦BCL6 LDD in Lymphoma		✦LPA1 Antagonist in IPF LPA1 Antagonist in PPF ✦obexelimab*# in IgG4-Related Disease
Removed	✦NME 2 ✦CD47xCD20 ✦GSPT1 CELMoD (CC-90009) ✦RIPK1 Inhibitor	✦Anti-TIGIT ✦HSP47	

Registrational Submissions

Approvals (n=2)

- OPDIVO in Adj Melanoma (EU)
- REBLOZYL in 1L MDS associated anemia (US)

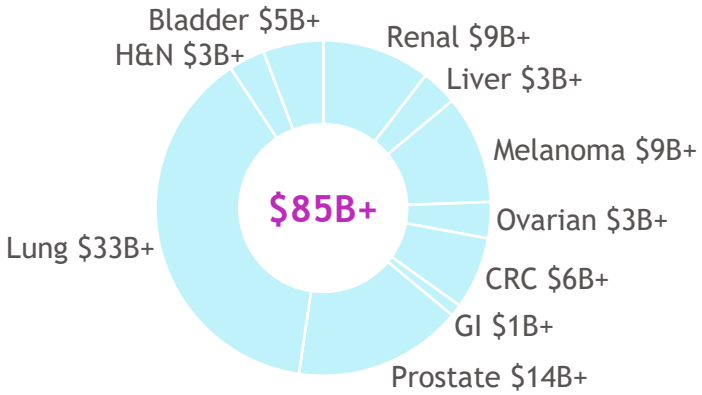
* Partner-run study; ✦NME leading indication; # BMS territory

Oncology | Hematology | CV | Immunology | Neuroscience

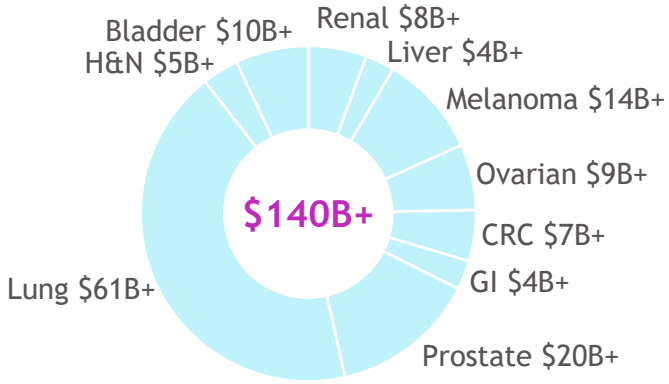
Addressing high unmet medical need in Oncology & Hematology

Oncology

2023

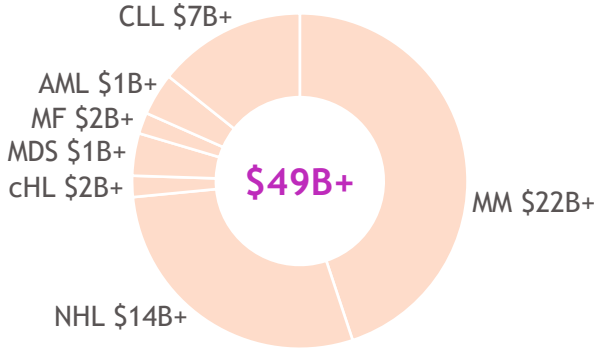


2030

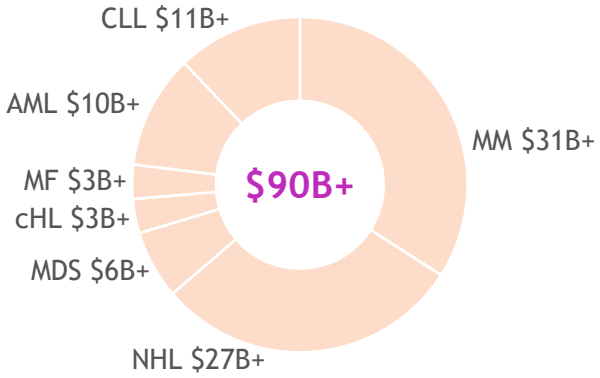


Hematology

2023

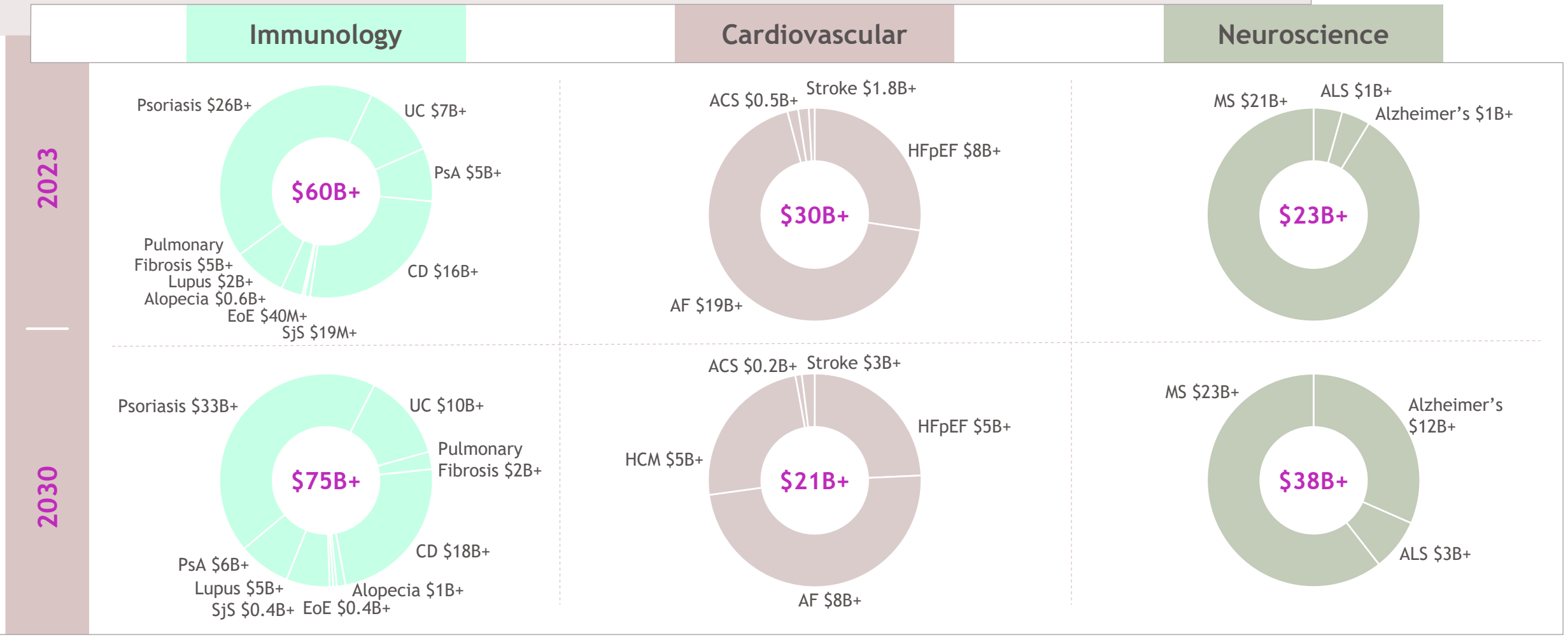


2030



Source: EvaluatePharma estimates; totals may not add due to rounding

Addressing high unmet medical need in Immunology, Cardiovascular & Neuroscience



*CV markets impacted by generic entry in Atrial Fibrillation
Source: EvaluatePharma estimates; totals may not add due to rounding

Farletuzumab ecteribulin (FZEC)[1]: Novel folate receptor alpha (FRα) ADC

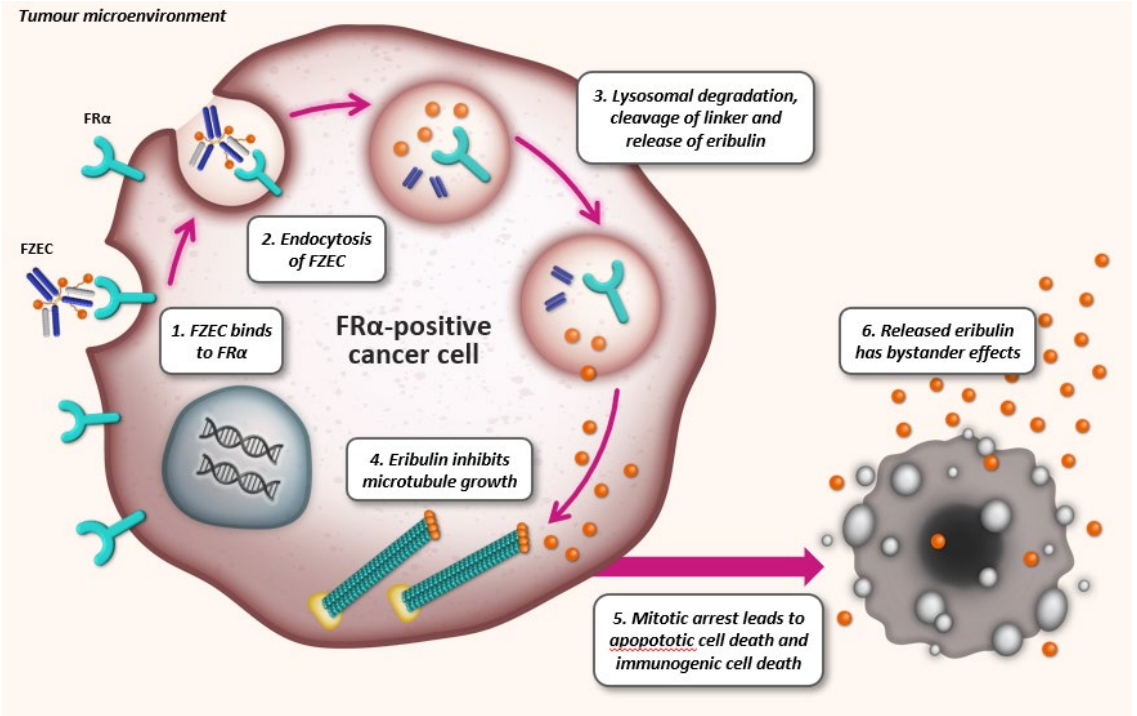
Overview

- FRα is a folate-binding protein that has limited expression on normal tissues and is overexpressed in malignant cells[2]
- 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

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

Highly Potent & Differentiated Small Molecule

ROS1+ TKI-Naïve NSCLC; **ORR** (95% CI)		**79%** (67.6, 87.7)
TKI-Pretreated Activity		✓ ORRs of 28-42% (n=100)
CNS Activity (*ROS1*+ NSCLC)		✓
***ROS1*+ TKI-Naïve NSCLC Durability**	**DOR**	• ≥12-month DOR: 83.1% (73.1, 93.2) • mDOR: 34.1 (25.6-NE)
	PFS	• ≥12-month PFS: 76.6% (66.2, 87.0) • mPFS: 35.71 (27.40, NE)
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 patients[2]

Existing ROS1 market:
~$500-$600M[3]

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

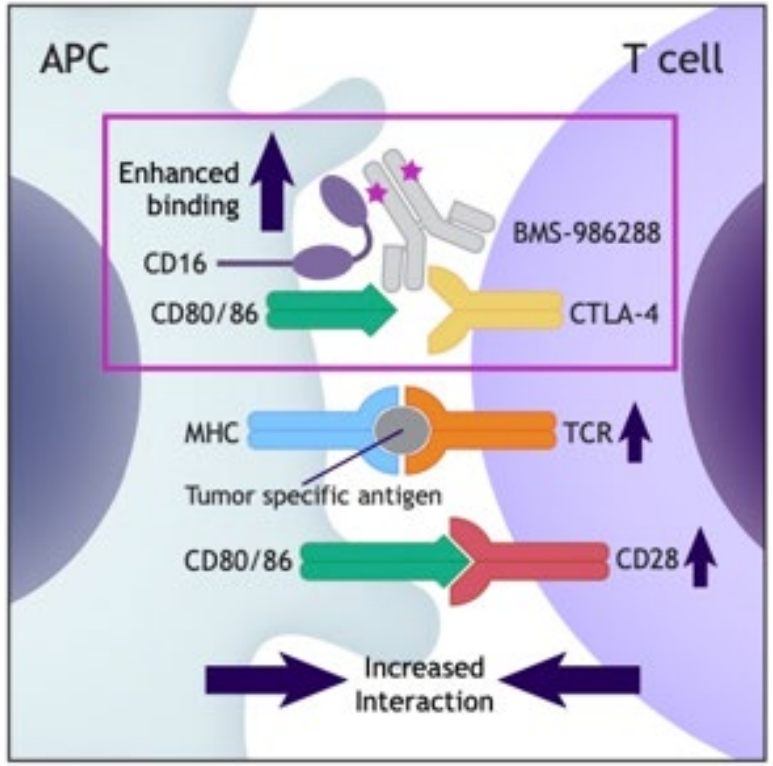
Overview

- 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

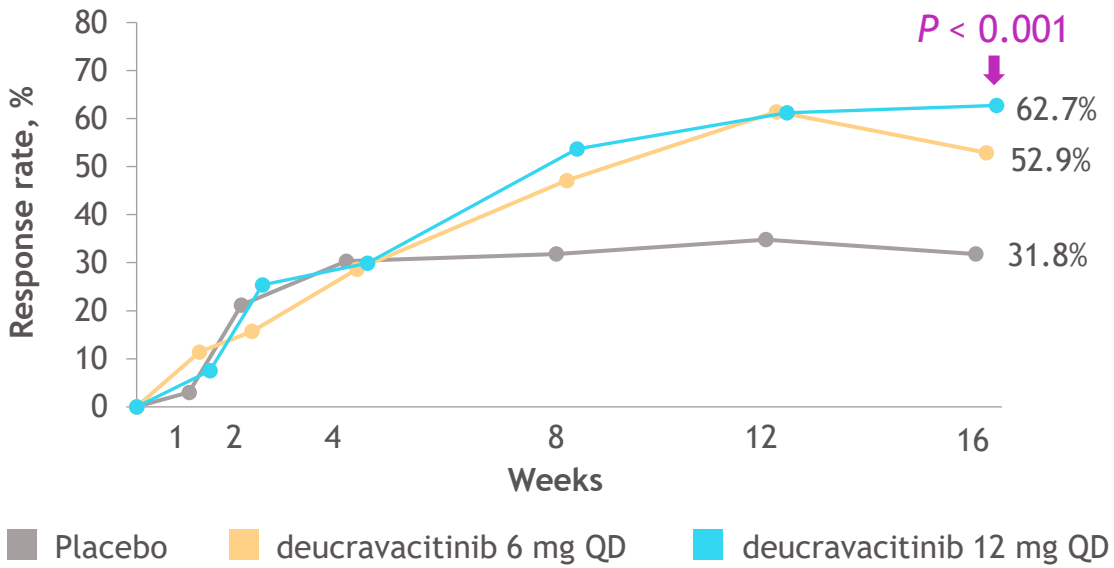


Broad range of development opportunities

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[1]



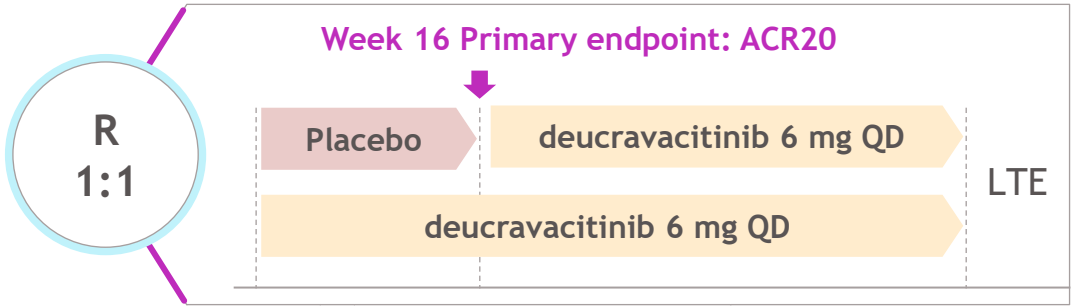
All primary and key secondary endpoints were achieved in patients with active PsA

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

Phase 3 program ongoing
data anticipated 2024/2025

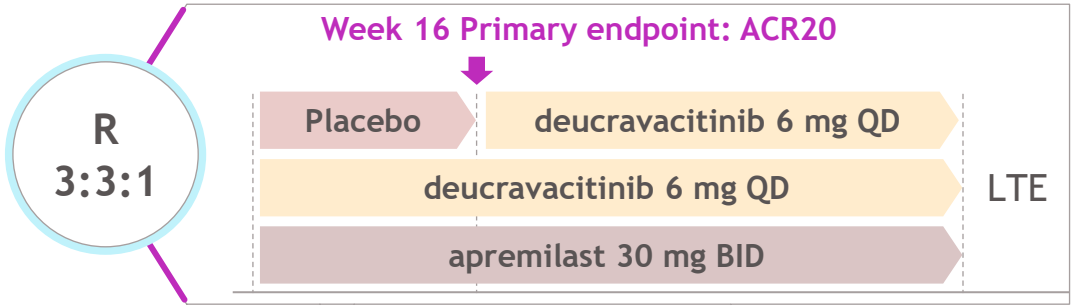
POETYK-PSA-1[2]

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



POETYK-PSA-2[3]

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



1. Mease PJ et al. *Ann Rheum Dis*. 2022;81:815-822. 2. NCT04908202; 3. NCT04908189

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

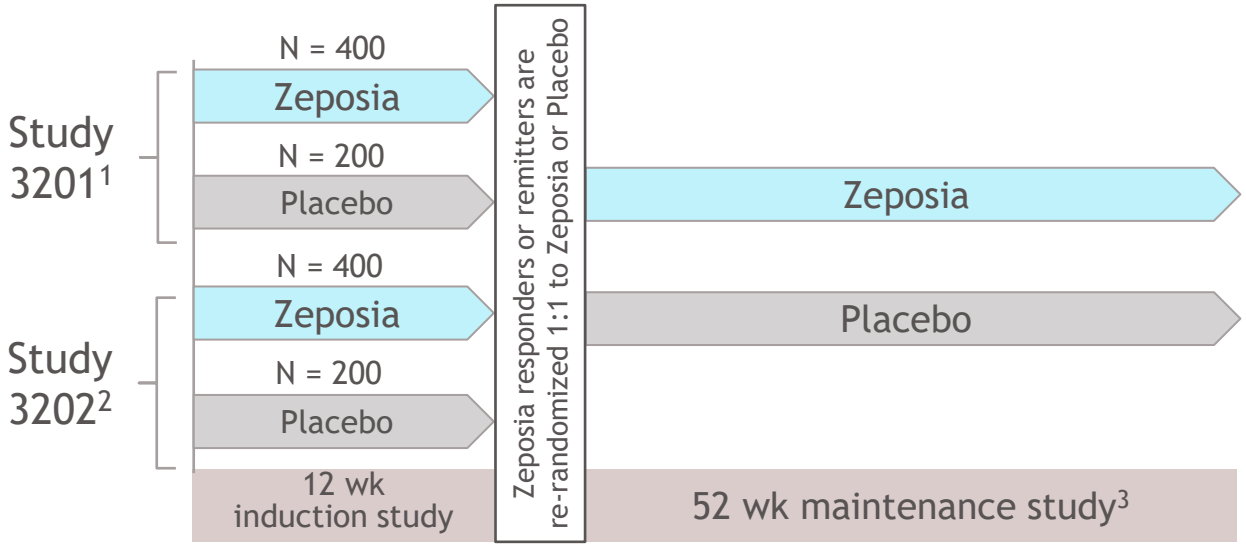
Crohn's Disease

Phase 3 YELLOWSTONE program ongoing

Maintenance study data anticipated 2026

Primary endpoints:

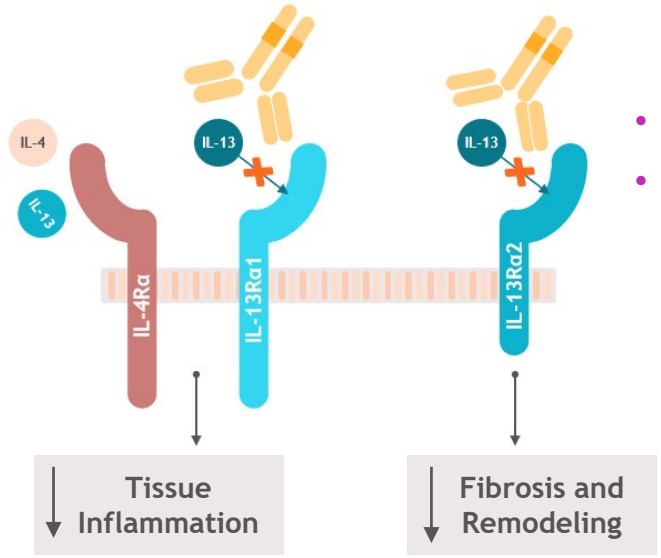
- Induction studies: Week 12 clinical remission
- Maintenance study: Co primary @ Week 52 clinical remission and endoscopic response



1. NCT03440372; 2. NCT03440385; 3. NCT03464097

Cendakimab: High-Affinity IL-13 Neutralizing Antibody for EoE

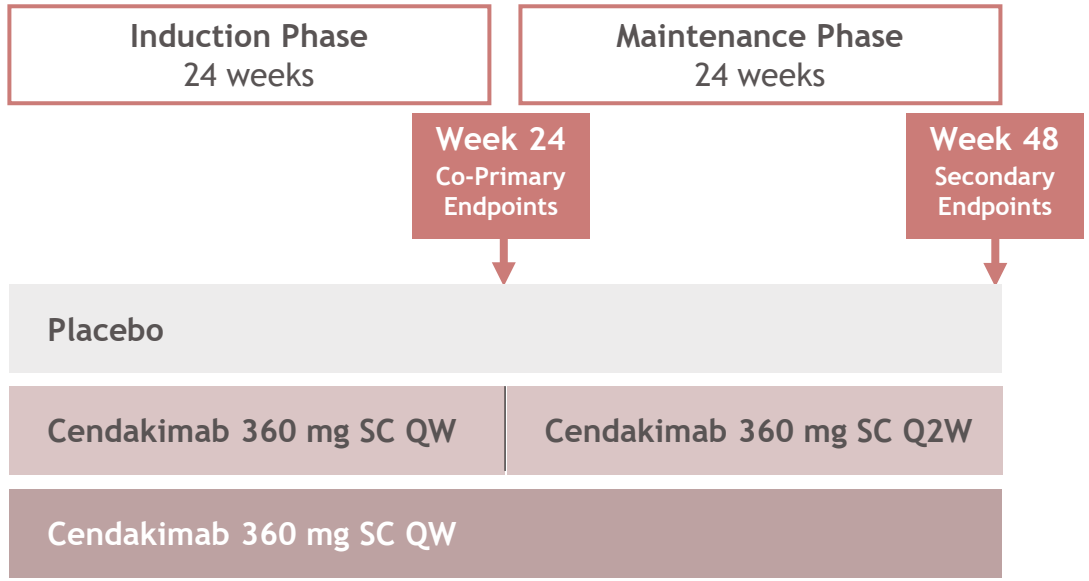
Eosinophilic Esophagitis + Cendakimab



- Binds to IL-13 ligand
- Blocks IL-13 binding to both IL-13Rα1 & IL-13Rα2 subunits

- EoE is a life altering disease affecting ~700k[1] prevalent patients (combined U.S./EU5)
- Potentially differentiated MoA addressing a significant unmet need for a highly efficacious treatment that improves both inflammation & fibrosis/remodeling

EoE: Currently Enrolling Phase 3 study[2]



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