Forward-Looking Statements

This presentation includes express and implied "forward-looking statements," including forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward looking statements include all statements that are not historical facts, and in some cases, can be identified by terms such as "may," "might," "will," "could," "would," "should," "expect," "intend," "plan," "objective," "anticipate," "believe," "estimate," "predict," "potential," "continue," "ongoing," or the negative of these terms, or other comparable terminology intended to identify statements about the future. Forward-looking statements contained in this presentation include, but are not limited to, statements about our product development activities, including our expectations around -the timing for filing our IND for our GSPT1 program advancement of additional programs to Lead Optimization, the continuing development of our QuEEN^{TM} platform and the expansion of our degron library and ability to identify additional molecular glue degraders. By their nature, these statements are subject to numerous risks and uncertainties, including the impact that the current COVID-19 pandemic will have on our development activities and operations, as well as those risks and uncertainties set forth in our Quarterly Report on Form 10-Q for the third quarter ended September 30, 2021 filed with the US Securities and Exchange Commission, and subsequent filings, that could cause actual results, performance or achievement to differ materially and adversely from those anticipated or implied in the statements. You should not rely upon forward looking statements as predictions of future events. Although our management believes that the expectations reflected in our statements are reasonable, we cannot guarantee that the future results, performance or events and circumstances described in the forward-looking statements will be achieved or occur. Recipients are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date such statements are made and should not be construed as statements of fact. We undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, future presentations or otherwise, except as required by applicable law. Certain information contained in this presentation and statements made orally during this presentation relate to or are based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, it has not independently verified, and makes no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research.

Monte Rosa Therapeutics Highlights
Taking molecular glue degraders (MGDs) to new heights

- **Next-generation molecular glue-based targeted protein degradation** platform developing breakthrough small molecule drugs that selectively degrade therapeutically-relevant proteins

- **Targeting the undruggable proteome** via AI-based degron prediction & rational design of highly selective MGDs

- **IND for lead program in 2022** with clinical development planned in Myc-addicted solid tumors and hematological malignancies

- **Five disclosed programs** targeting high unmet medical needs in oncology and non-oncology indications

- **World-class leadership & SAB** with deep drug discovery and development expertise and know-how

World Class Leadership

Deep expertise in molecular glue discovery and drug development

Senior Management

Markus Warmuth, M.D.
Chief Executive Officer
H3 / RELAY THERAPEUTICS / NOVARTIS

Ajim Tamboli, CFA
Chief Financial Officer
COLUMBIA THREADNEEDLE INVESTMENTS / LEHMAN BROTHERS

Sharon Townson, Ph.D.
Chief Technology Officer
KYMERA / Warp Drive Bio

Owen Wallace, Ph.D.
Chief Scientific Officer
Fulcrum Therapeutics / NOVARTIS

John Castle, Ph.D.
Chief Data Scientist
agenus / BIONTECH

Filip Janku, M.D., Ph.D.
Chief Medical Officer
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

Jullian Jones, Ph.D., J.D., MBA
SVP, Business Development
Lilly / Boehringer Ingelheim

Silvia Buonamici, Ph.D.
SVP, Drug Discovery Biology
H3 / NOVARTIS

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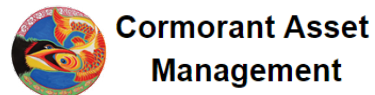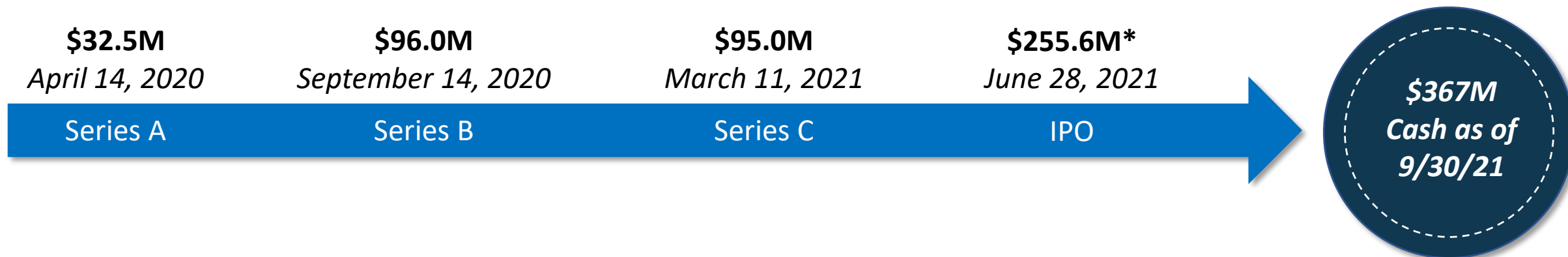**Kimberly Blackwell, M.D.**, *Chief Medical Officer, Tempus*

David Schenkein, M.D., *General Partner, GV*

Nir London, PhD, Assistant Professor, *Weizman Institute of Science, Israel*

Strong Cash Position and Investor Support

Over $455M raised since 2020 with top tier investors provides runway into late 2024

$32.5M	**$96.0M**	**$95.0M**	**$255.6M***
April 14, 2020	*September 14, 2020*	*March 11, 2021*	*June 28, 2021*
Series A	Series B	Series C	IPO

$367M Cash as of 9/30/21

*Aggregate IPO gross proceeds were approximately $255.6 million before deducting underwriting discounts and commissions and other offering expenses and include an additional $33.3 million in gross proceeds the company received as part of its IPO from the full exercise of the underwriters' option to purchase up to an additional 1,755,000 shares of common stock at the public offering price of $19.00 per share.

Introduction to Monte Rosa Therapeutics

Next-generation molecular glue-based targeted protein degradation platform developing breakthrough small molecule therapeutics

Molecular Glue Degraders (MGDs)
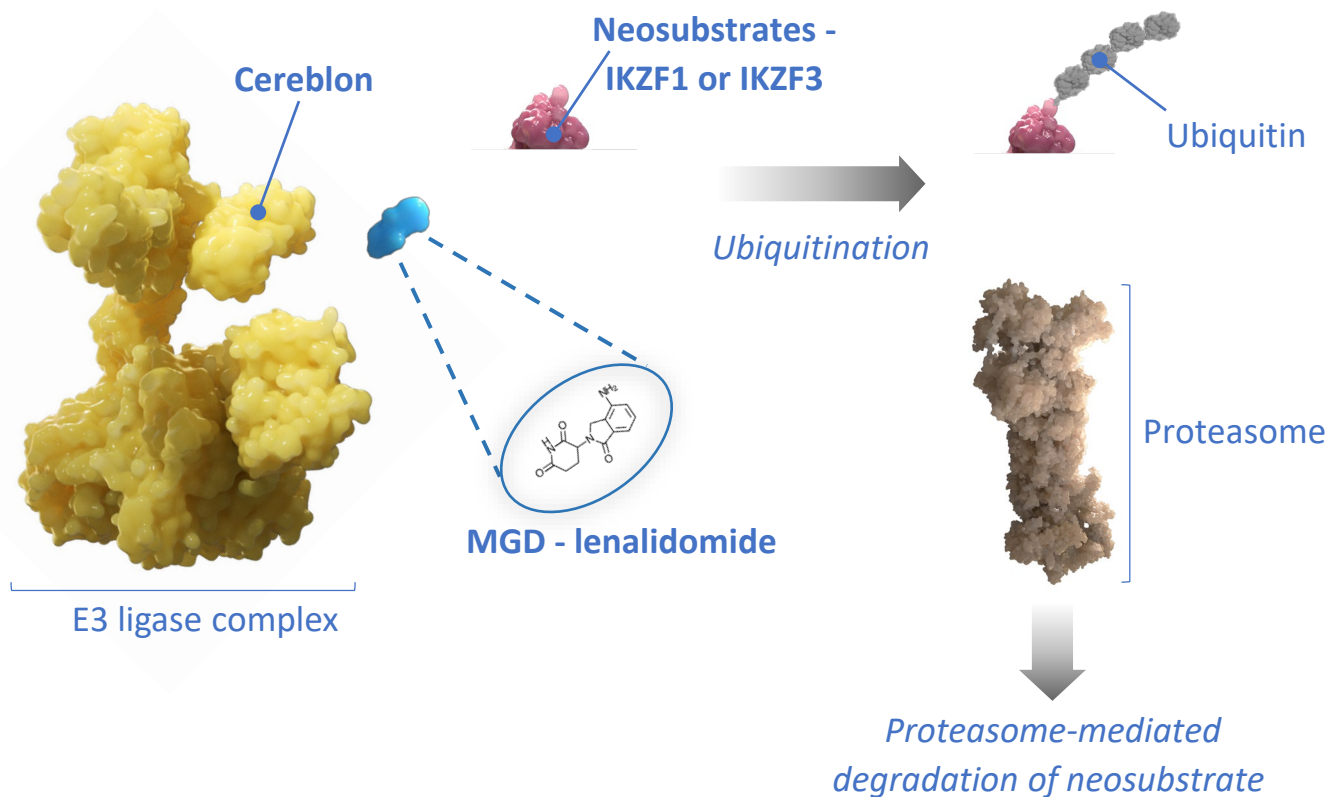## *Expanding target space, fostering a new generation of drugs*

INHIBITOR

TARGET

**Drugging
the Druggable**

PROTAC

TARGET

E3 LIGASE

**Redrugging
the Druggable**

MGDs

MAJORITY OF
TARGETS LACK
DRUGGABLE
POCKETS

E3 LIGASE

**Drugging
the Undruggable**
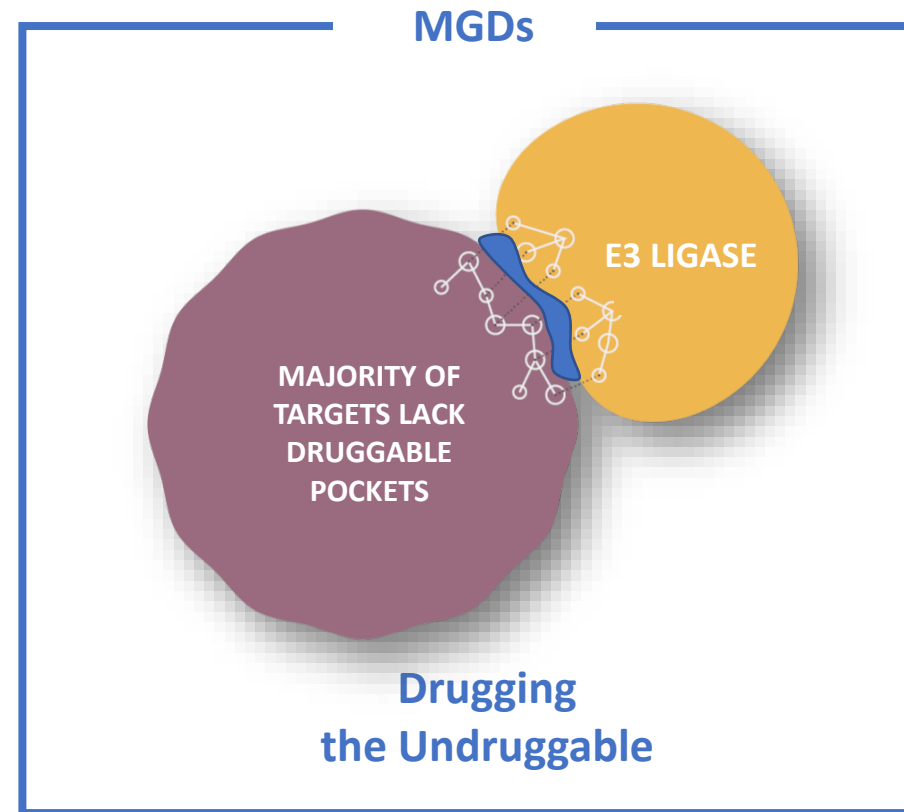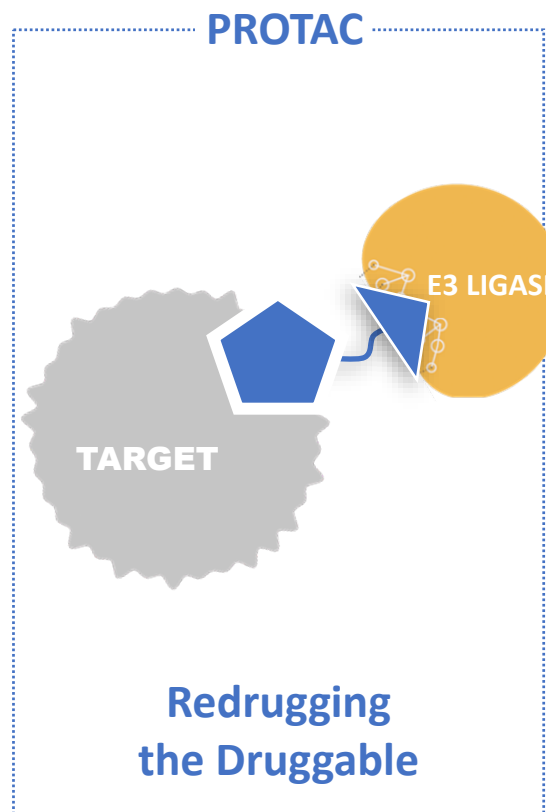
**3-10% OF
PROTEOME**

**UNCHARTED
CHEMICAL AND TARGET SPACES**

Molecular Glue Degraders (MGDs)
A powerful and differentiated approach to eradicate disease-causing proteins

Cereblon

Neosubstrates - IKZF1 or IKZF3

Ubiquitin

Ubiquitination

MGD - lenalidomide

E3 ligase complex

Proteasome

Proteasome-mediated degradation of neosubstrate

- ✓ **Undruggable** target space
- ✓ Favorable **drug-like** properties
- ✓ Clinically **validated**
- ✓ **Systematic** and **selective** reprogramming
- ✓ **Broad** therapeutic application

Systematic Chemical Reprogramming of E3 Ligases using MGDs

Cereblon (CRBN), the G-loop Degron and Beyond

A rational approach to unleash the full potential of MGDs

Target

degron*

MGD

Cereblon
(CRBN)

**PLATFORM BUILD
AND VALIDATION**

Unraveling the
canonical Degrome

**PLATFORM
EXPANSION**

Beyond the
canonical Degrome

**PLATFORM
DIVERSIFICATION**

Beyond
Cereblon
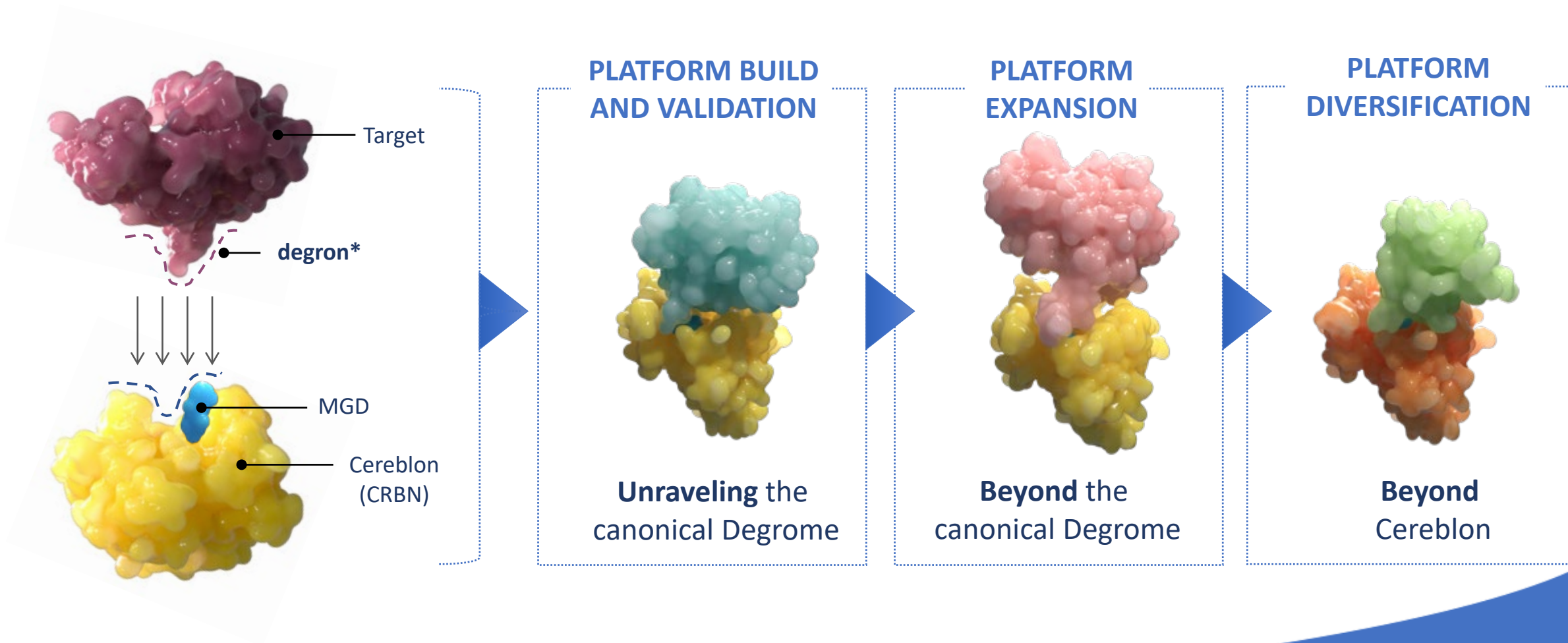
**EXPANDING THE DEGRADABLE
PROTEOME BY RATIONAL DESIGN**

* Structural feature (β-hairpin loop) allowing target recruitment to CRBN

9

The Next Generation of Precision Medicine-based Small Molecule Drugs
Challenges with undruggable vs druggable proteins

	Traditional small molecule inhibitors	Therapeutic Antibodies	RNAi, RNA Editing	CRISPR/Gene Therapy	MGDs
Ability to access undruggable space	✗	✓	✓	✓	✓
Cellular permeability	✓	✗	✓	✓	✓
Oral bioavailability	✓	✗	✗	✗	✓
Systemic distribution	✓	✓	✗	✗	✓
CNS Penetration	✓	✗	✗	✗	✓
Manufacturing scalability	✓	✓	✗	✗	✓

QuEEN™ Discovery Platform: Transformational Approach to MGDs

Building a unique portfolio of precision medicines addressing high unmet medical needs

Degron Encyclopedia

degron

Degron discovery using our AI-powered algorithm

Glueomics Toolbox

Specialized suite of *in vitro* and *in silico* assays to discover, optimize and advance MGDs as clinical candidates

Proprietary Library

Rationally designed, diverse and growing library with drug-like properties
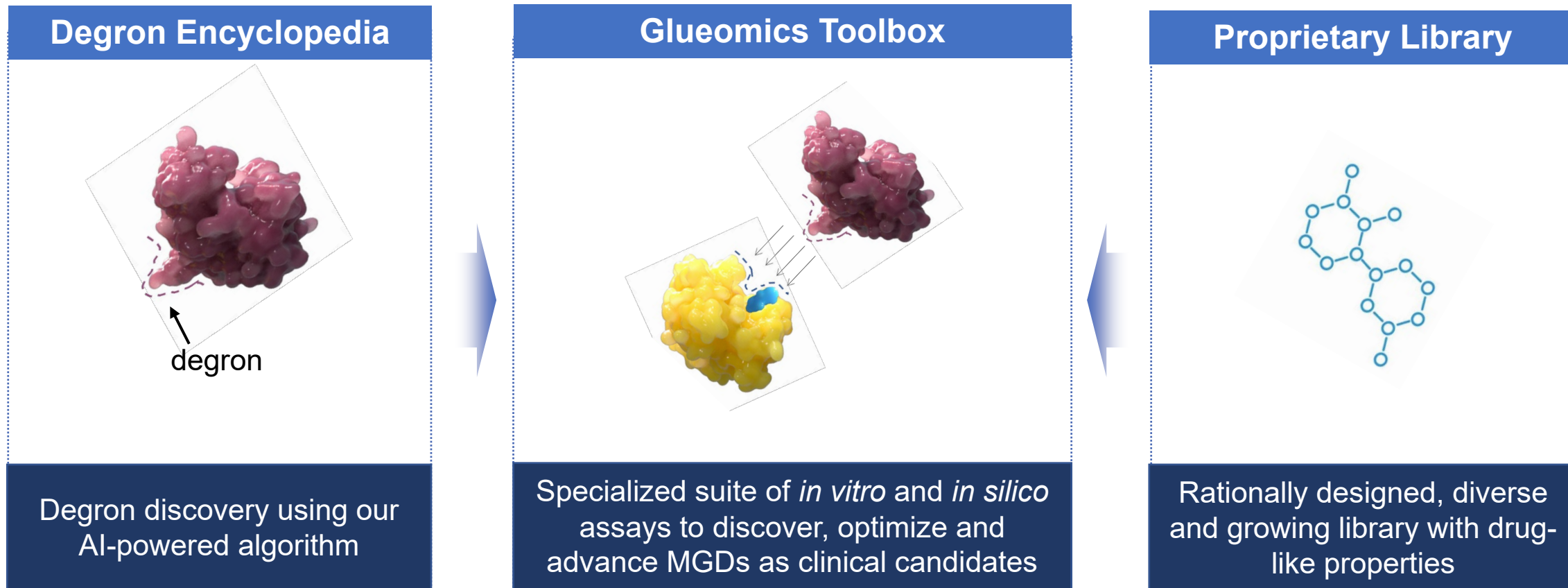
The Degron Encyclopedia
A rich, differentiated target space across protein domains and diseases

Integrated Degron Mining

Sequence

Deep Neural Net

Topology

Loop scoring

Surface

Surface geometry
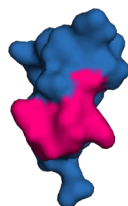
Degron Encyclopedia

>3000 predicted degron-containing proteins

Broad disease landscape

- Genetic disease
- Nervous system
- Musculoskeletal
- Urinary system
- Psychiatric disorder
- Respiratory system
- Neoplasm
- Endocrine system
- Metabolic disease
- Immune system

0   500

Top protein classes

- Catalytic activity
- Reductase
- Transporter
- Nucleic acid binding
- Receptor
- Hydrolase
- Transcription factor
- Transferase
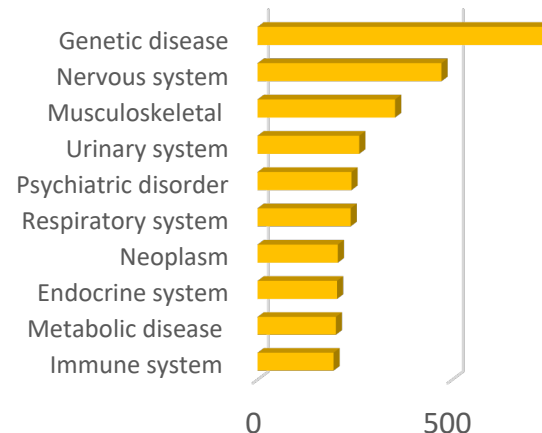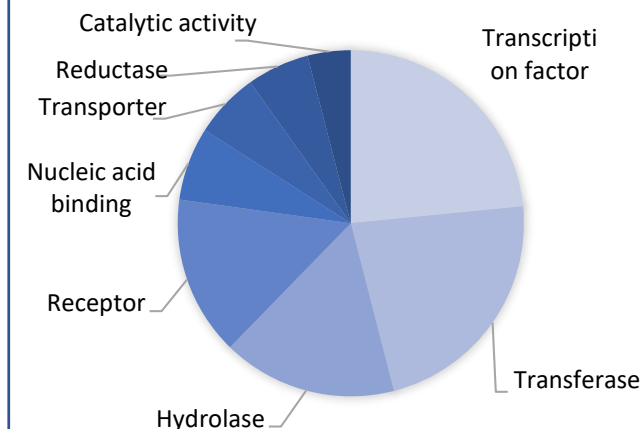
Many highly credentialed targets

>75% undruggable

>85% degrons have unique sequence

Expanding the Target Space by Identifying More Degrons

Example of degron-containing proteins in different diseases
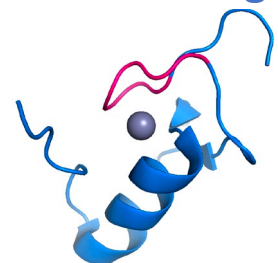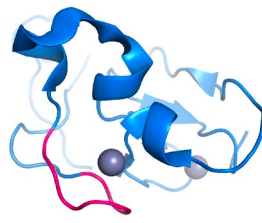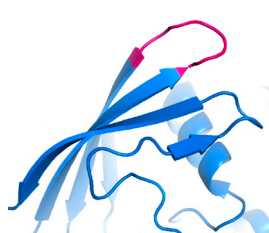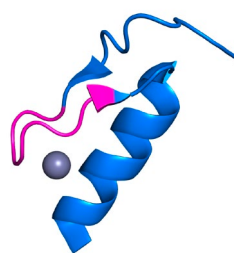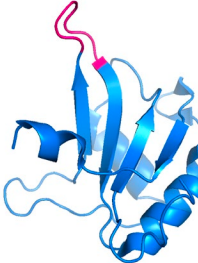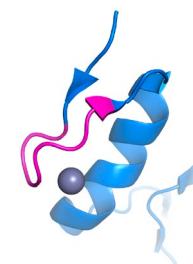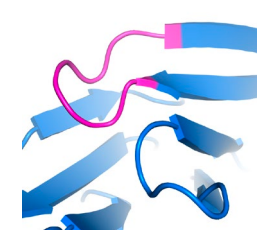
Immunology/ Inflammation	Cancer	I-O	Neurology	Genetic Diseases
Kinase	**Demethylase**	**Phosphatase**	**DNA binding**	**DNA binding**
Inflammation	*Solid tumors*	*T-cell exhaustion*	*ALS*	*Blood*
DNA binding	**GEF**	**DNA binding**	**RNA binding**	**WD repeat**
T-cell development	*Hematological cancers*	*Tregs*	*ALS*	*Spastic Paraplegia*

>95% of degrons have a unique sequence, providing a unique handle to engage MGD chemical matter

GEF - Guanine nucleotide exchange factor

New Chemical Space: MGD Anatomy and Evolving MGD Library

Increasing novelty and structural diversity to match the target space

MGD Anatomy

Core
Degron domain

Warhead
Cereblon binding domain
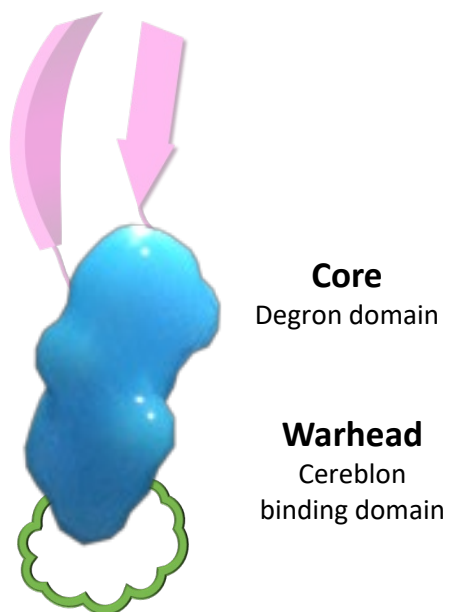
Increasing the Core-Warhead Chemical Diversity

Color – Molecular Weight

190 Da   300 Da

Size – pIC$_{50}$ CRBN binding*

4.7   6.8

Tanimoto similarity — 0.2, 0.4, 0.6, 0.8, 1

Lenalidomide

Scaffold evolution
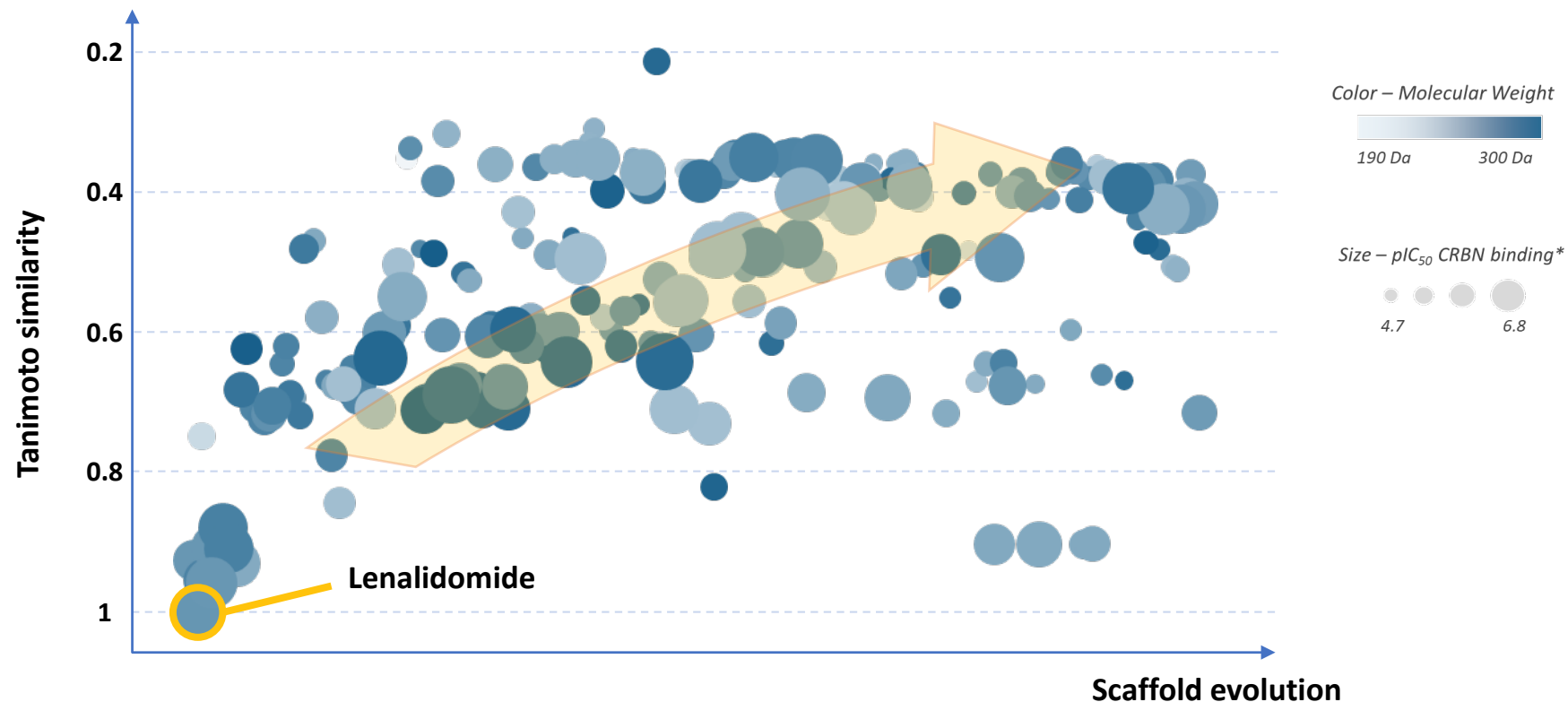
>200 unique scaffolds validated with increasing diversity, confirmed binding and structural insights

Utilizing Diverse Geometries to Selectively Engage Neosubstrates

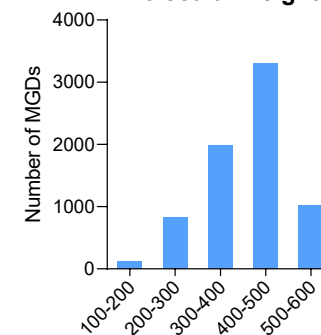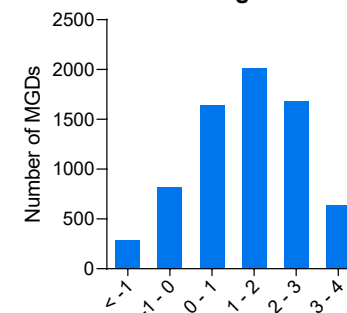*Increasing structural diversity while maintaining drug like properties*

Expanding MGD Anatomy

Side Chains

PPI domain

200+ Scaffolds
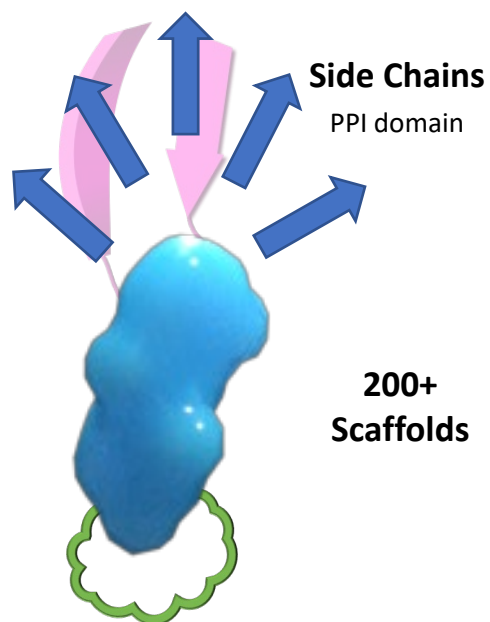
Exponential Growth and Properties

# of library compounds

3Q18 — 4Q21

Molecular weight

Number of MGDs

100-200, 200-300, 300-400, 400-500, 500-600

clogP

Number of MGDs

< -1, -1 - 0, 0 - 1, 1 - 2, 2 - 3, 3 - 4

Polar surface area

Number of MGDs

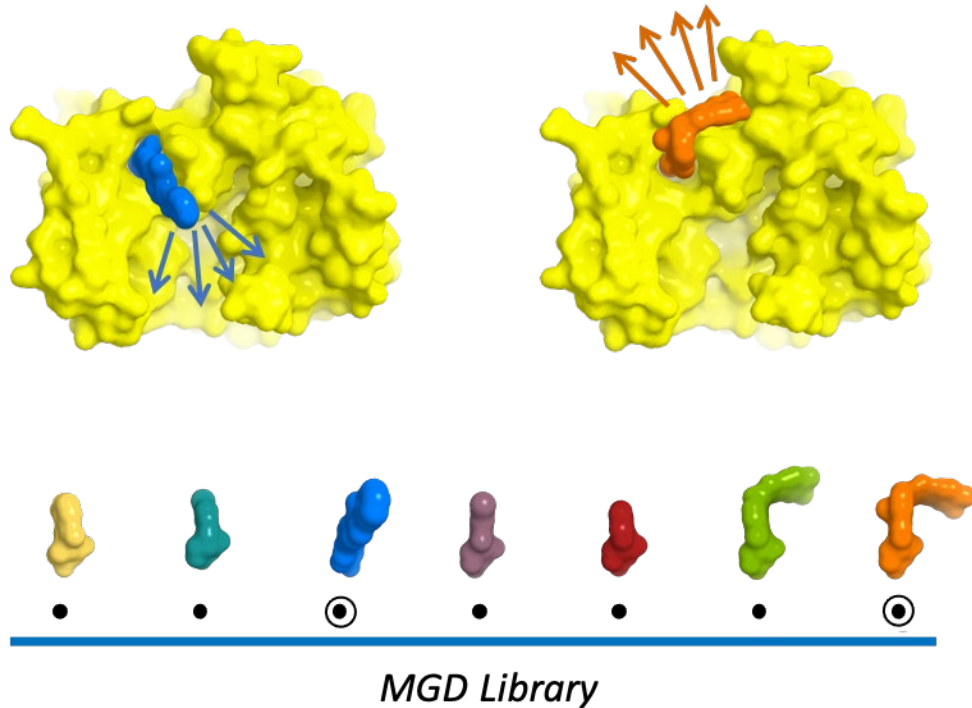< 60, 60 - 80, 80 - 100, 100 - 120, 120 - 140, 140 - 160, > 160
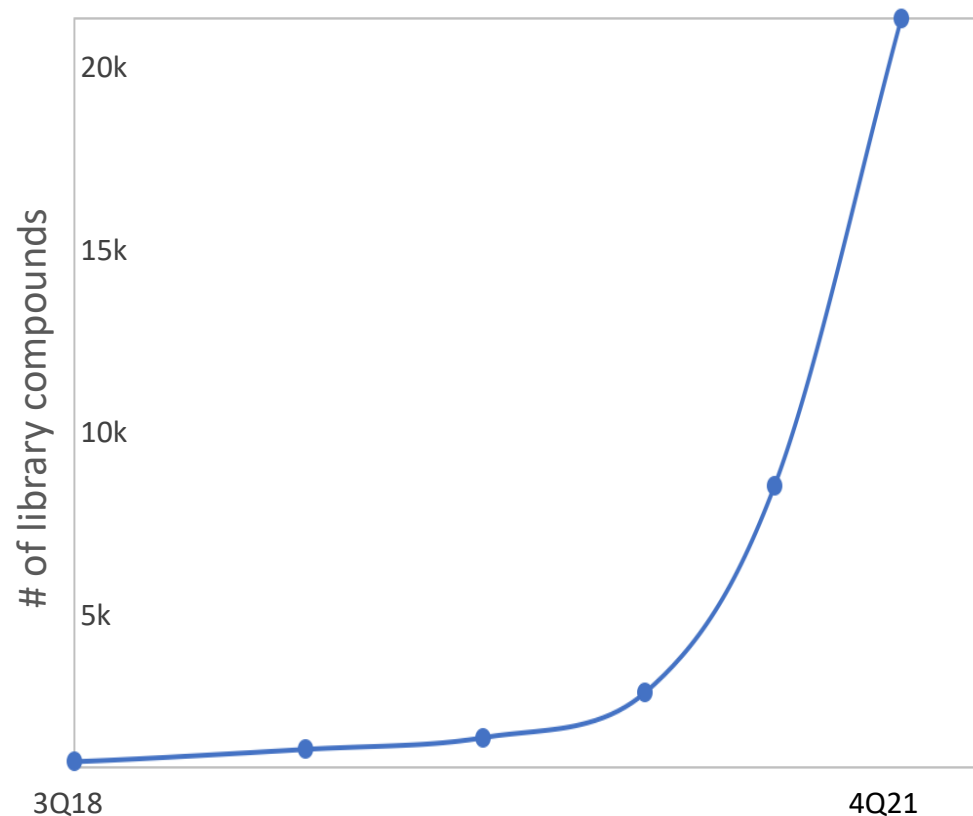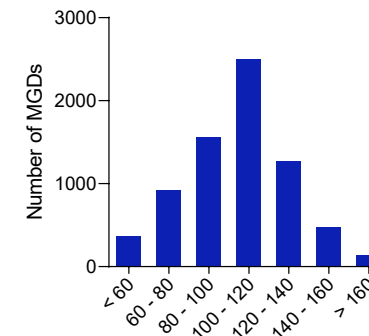
Expanding Chemical Library to >20K unique molecules in 2021

MGDs Designed to Remodel and Reprogram Cereblon Surface Interaction
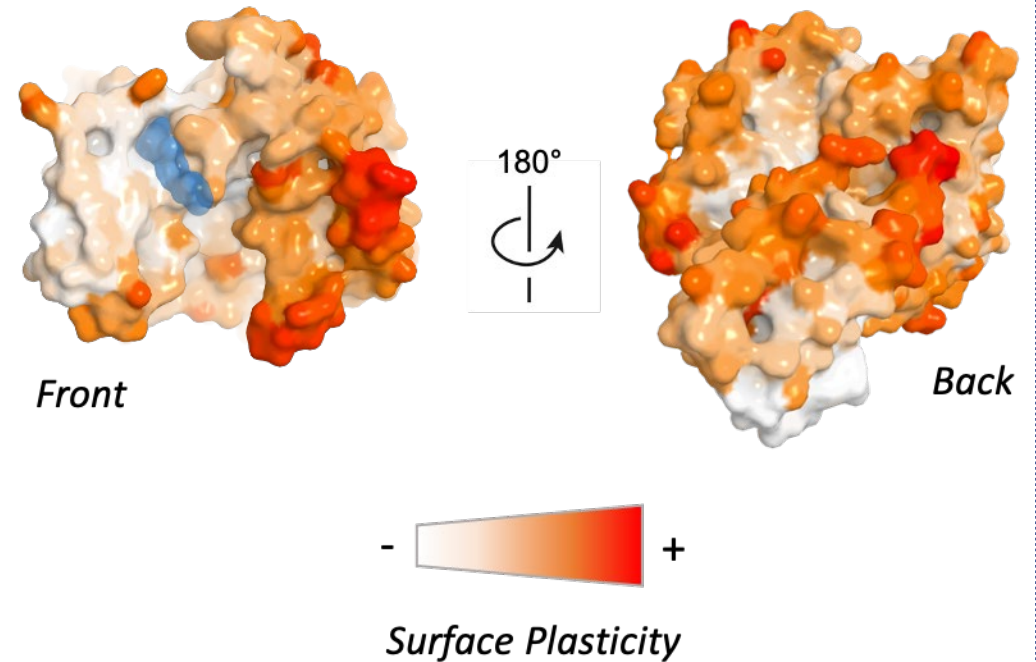*Proximal and distal changes facilitate selective binding to neo-substrates*

Proximal remodeling of cereblon

MGD Library

✓ **Creates unique interactions with degrons**

Distal remodeling of cereblon
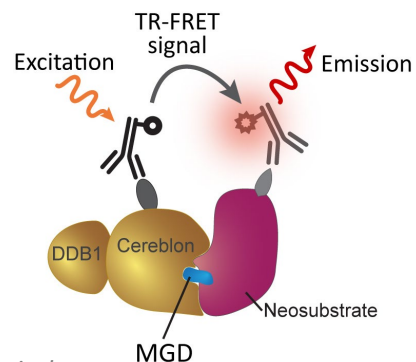
Front

180°

Back

- ⟶ +

Surface Plasticity

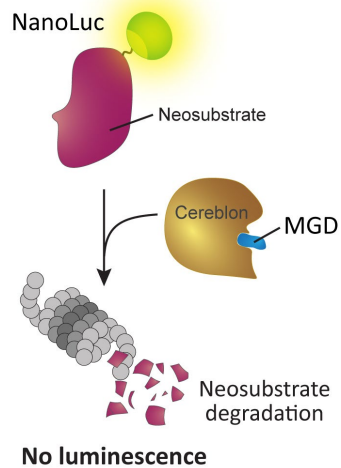✓ **Facilitates selective neosubstrates engagement**

Glueomics™ Toolbox Accelerates Identification of MGDs
Multiple screening formats enable rapid identification and validation of MGDs for novel targets
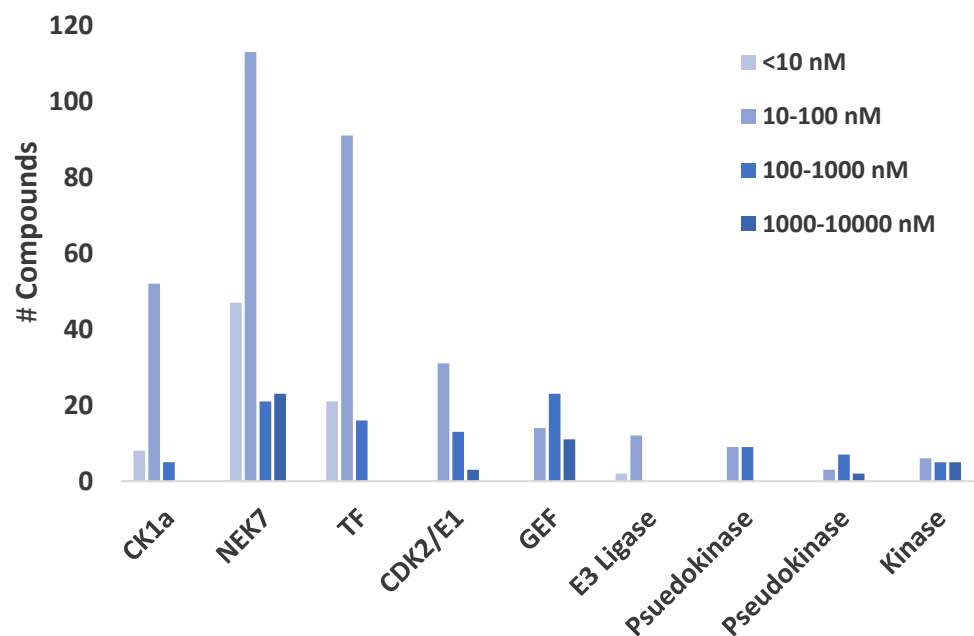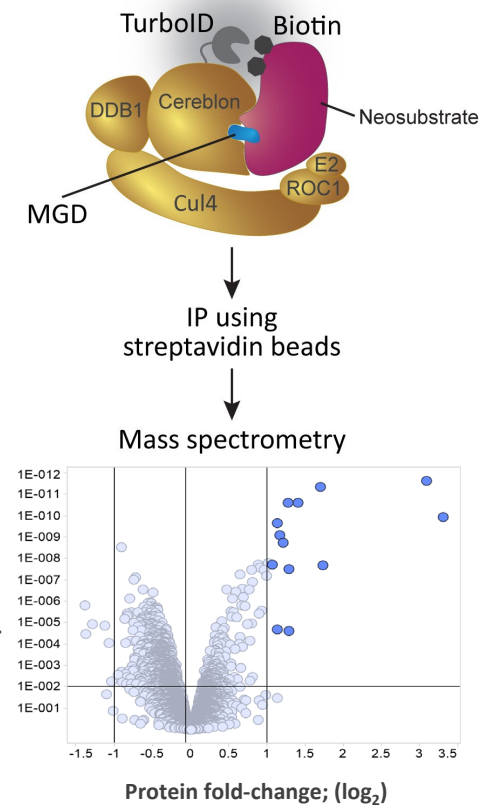
in vitro screens

Biochemical

TR-FRET signal
Excitation
Emission
DDB1 | Cereblon | Neosubstrate
MGD

Cellular

NanoLuc
Neosubstrate
Cereblon — MGD
Neosubstrate degradation
No luminescence

Results

EC$_{50}$ values in a biochemical ternary complex HTRF assay

Compounds

Legend:
- <10 nM
- 10-100 nM
- 100-1000 nM
- 1000-10000 nM

Categories: CK1a, NEK7, TF, CDK2/E1, GEF, E3 Ligase, Psuedokinase, Pseudokinase, Kinase

Chemoproteomics

TurboID | Biotin
DDB1 | Cereblon | Neosubstrate
MGD | Cul4 | E2 ROC1

IP using streptavidin beads

Mass spectrometry
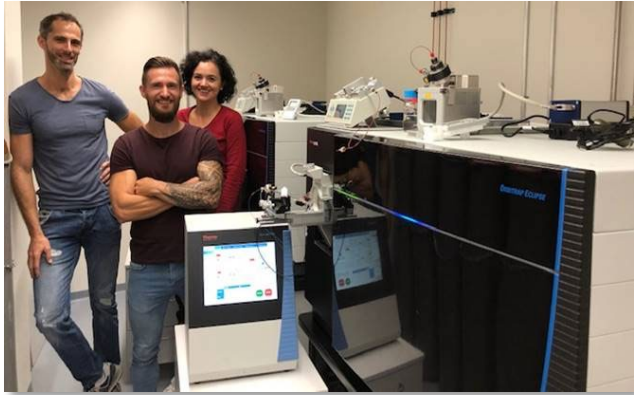
p-value
Protein fold-change; (log$_2$)

Turbo-ID

18

Proteomics Platform is Scaled for Success

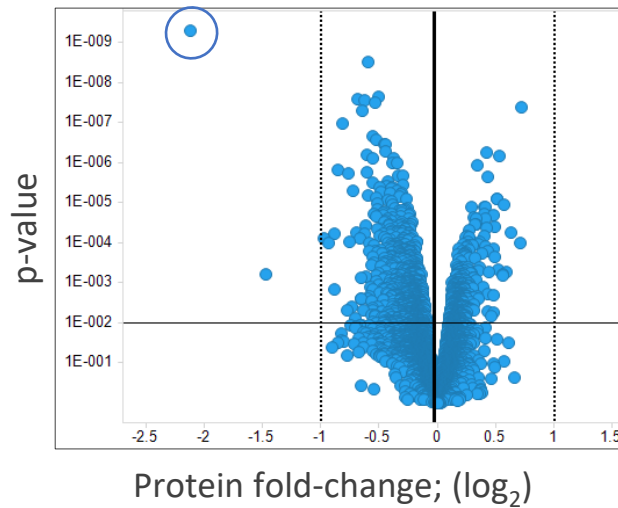*Purpose-built assay suite for selectivity profiling and identification of novel neo-substrates*

In-house Capabilities

✓ 3 Orbitraps
✓ Multiple assay formats
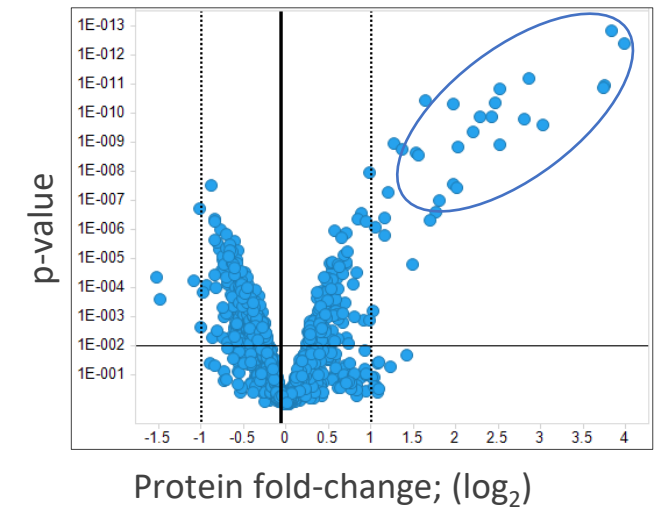✓ Double capacity in 2021

Degradation

Global Protein expression - TMT Proteomics

p-value vs Protein fold-change; $(\log_2)$

✓ Degradation
✓ Selectivity

Proximity

Turbo ID - TMT Proteomics

p-value vs Protein fold-change; $(\log_2)$

✓ Complex formation
✓ Neosubstrate identification

Multiple screening formats enable rapid identification and validation of MGDs for novel degron targets
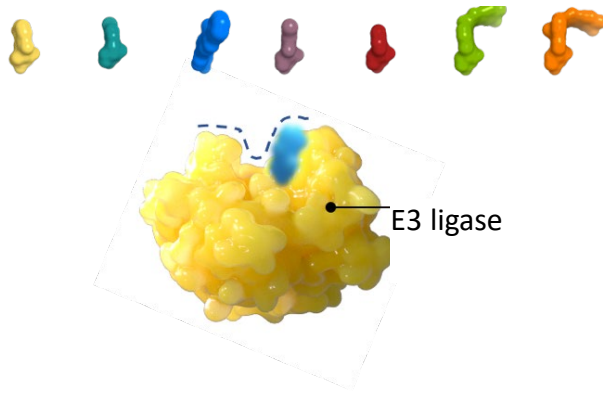
Rhapsody, QuEEN's *in silico* MGD Engine

*A suite of proprietary AI-powered algorithms to design, discover and develop MGDs*

in silico library generation

Creation and e3 ligase docking of novel MGDs, expanding our library to engage more targets

E3 ligase

in silico ternary complex models

Ternary complex models enabling MRT scientists to engineer and optimize selective MGDs

MGD

Neosubstrate

E3 ligase
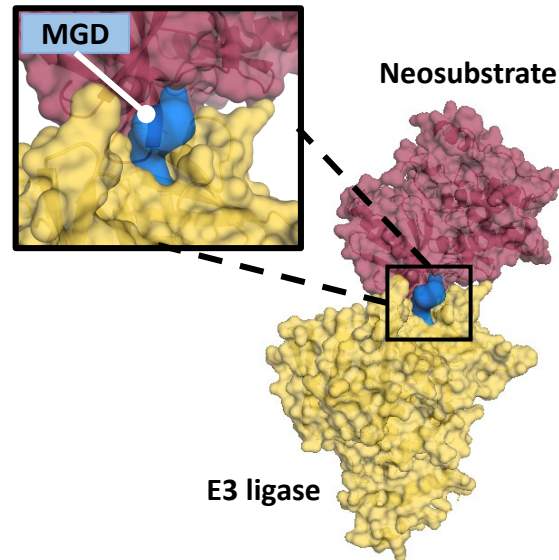
in silico MGD screening

Computational screening identifying and prioritizing hits inducing binding and selective degradation

Neosubstrate

Degron

MGD library

E3 ligase
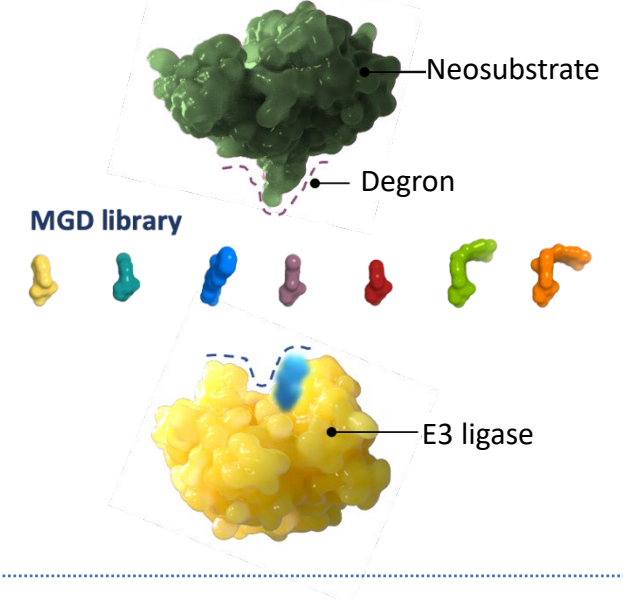
Powered by MTR highly customized AWS infrastructure

Monte Rosa's Pipeline of MGDs

Leveraging a Leading Drug Discovery Platform
Purpose-built to discover and develop a wide landscape of therapeutically-relevant MGDs

Towards a High-Value Proprietary Pipeline

Targets

Undruggable and inadequately drugged degron-containing proteins

High level of target validation, preclinically and clinically

Clinical Path

Programs with biomarker-based patient selection strategy and clear path to the clinic

Opportunity for rapid clinical PoC for MOA and efficacy

Patient Benefit

Address high unmet needs

Potential to address a wide range of therapeutically-relevant proteins in oncology and beyond

Create synergies within therapeutic areas

Monte Rosa Pipeline
Rapidly advancing wholly owned MGD programs

Target / Program	Indication(s)	Discovery	IND-Enabling	Clinical	Next Anticipated Milestones	Ownership
GSPT1	NSCLC, SCLC, Heme Malignancies				IND filing mid-2022	
NEK7	Inflammatory Diseases					
CDK2	Ovarian Cancer, Breast Cancer				**Advance at least one program in addition to NEK7 into Lead Optimization in 2021**	
VAV1	T and B Cell Malignancies, Autoimmune Disease					
BCL11A	SCD, β-Thalassemia					
Undisclosed	Multiple					

● Oncology   ● Autoinflammation   ● Oncology / immunology   ● Genetic diseases

Identification and Development of GSPT1 MGDs

From identifying that GSPT1 is a key regulator and vulnerability of Myc-induced translational addiction to a biomarker-driven program in Myc-addicted tumors

Targeting Myc-Driven Tumors and Their Addiction to Protein Translation
GSPT1 is a key regulator and vulnerability of Myc-induced translational addiction

Myc hijacks the cellular protein translation machinery creating a vulnerability to GSPT1

Genes involved in protein synthesis e.g. eIF4E, 4EBP1 and 4EBP2
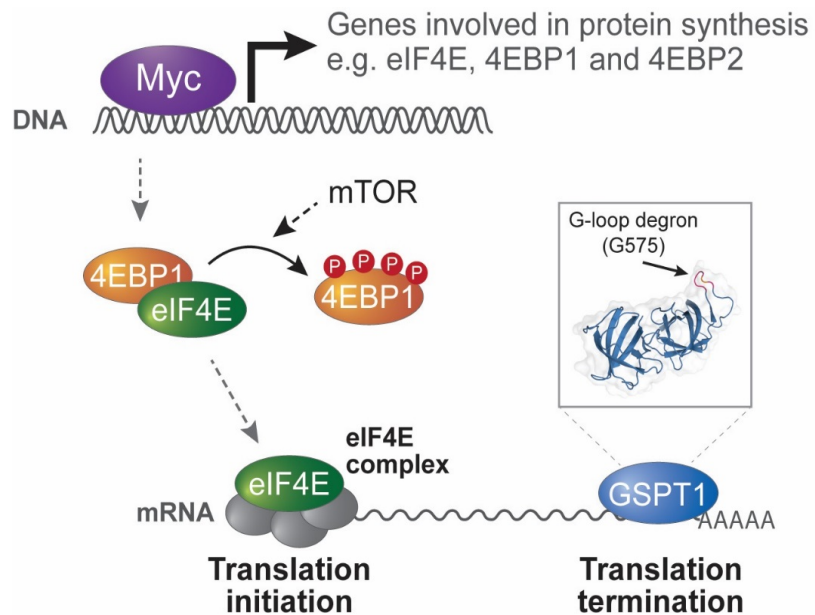
Target profile

To sustain growth, Myc-driven tumors are **addicted to protein translation**

- Myc regulates the expression of key genes related to protein translation, including the master regulator 4EBP1 and eIF4E

This addiction to protein translation creates a possible **dependency** to the termination translation factor GSPT1, a degron-containing protein
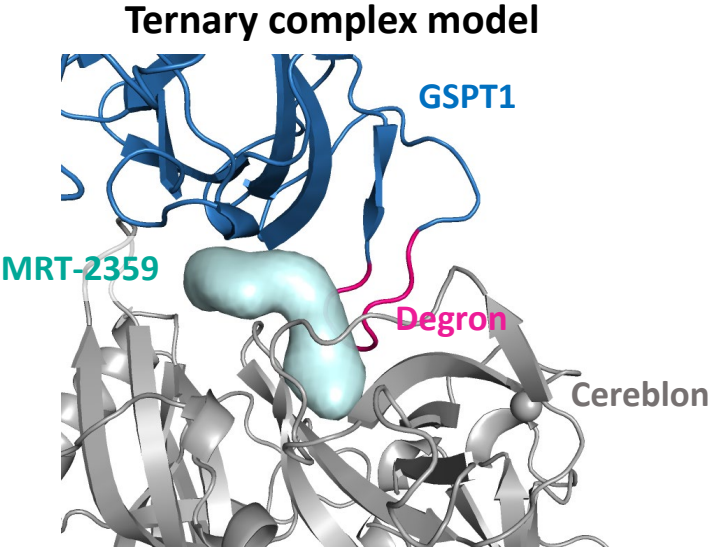
Hypothesis - GSPT1 MGDs exploit this **vulnerability** by:
- Disrupting protein translation output
- Reducing Myc-oncogenic signaling

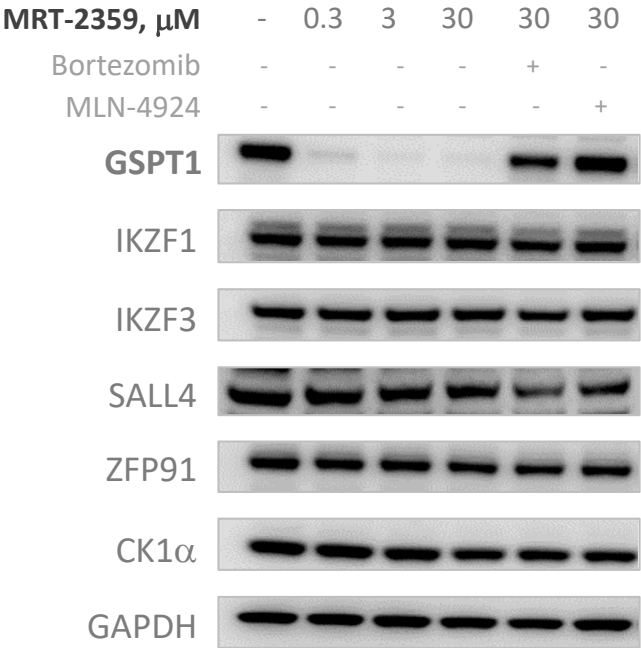# MRT-2359 is a Potent and Selective GSPT1-directed MGD

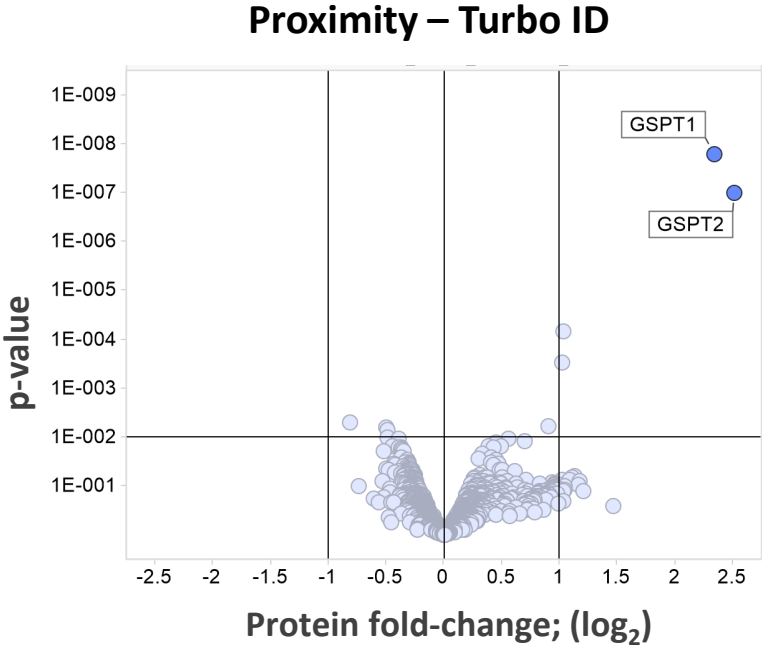## MRT-2359 is a potent inducer of GSPT1-cereblon proximity

Ternary complex model

GSPT1
MRT-2359
Degron
Cereblon

in vitro data

CRBN binding, K_i	113 nM
Ternary complex, EC_{50}	< 7 nM
Degradation, DC_{50}	80 nM

MRT-2359 is a selective GSPT1-directed MGD

MRT-2359, μM	-	0.3	3	30	30	30
Bortezomib	-	-	-	-	+	-
MLN-4924	-	-	-	-	-	+

GSPT1
IKZF1
IKZF3
SALL4
ZFP91
CK1α
GAPDH

6hr post treatment in MM1S and Kelly (SALL4)

Proximity – Turbo ID

GSPT1
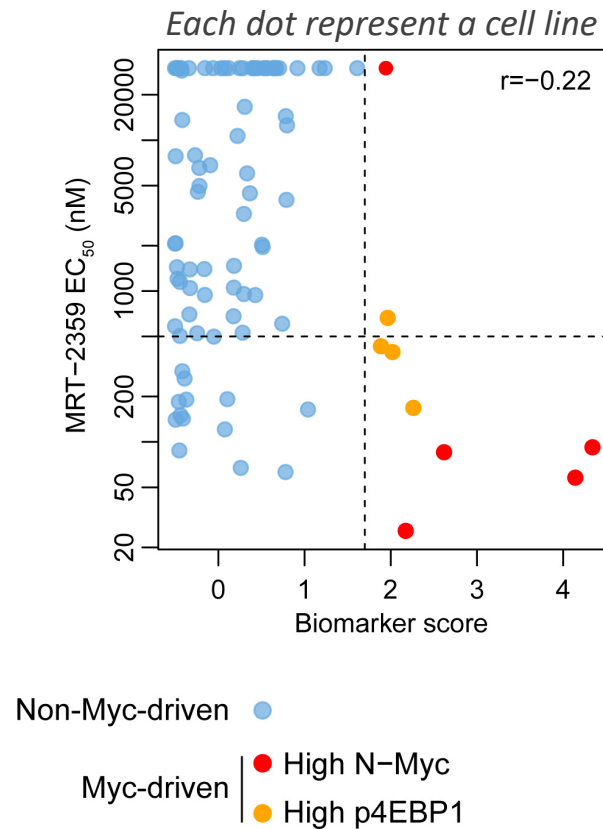GSPT2

p-value

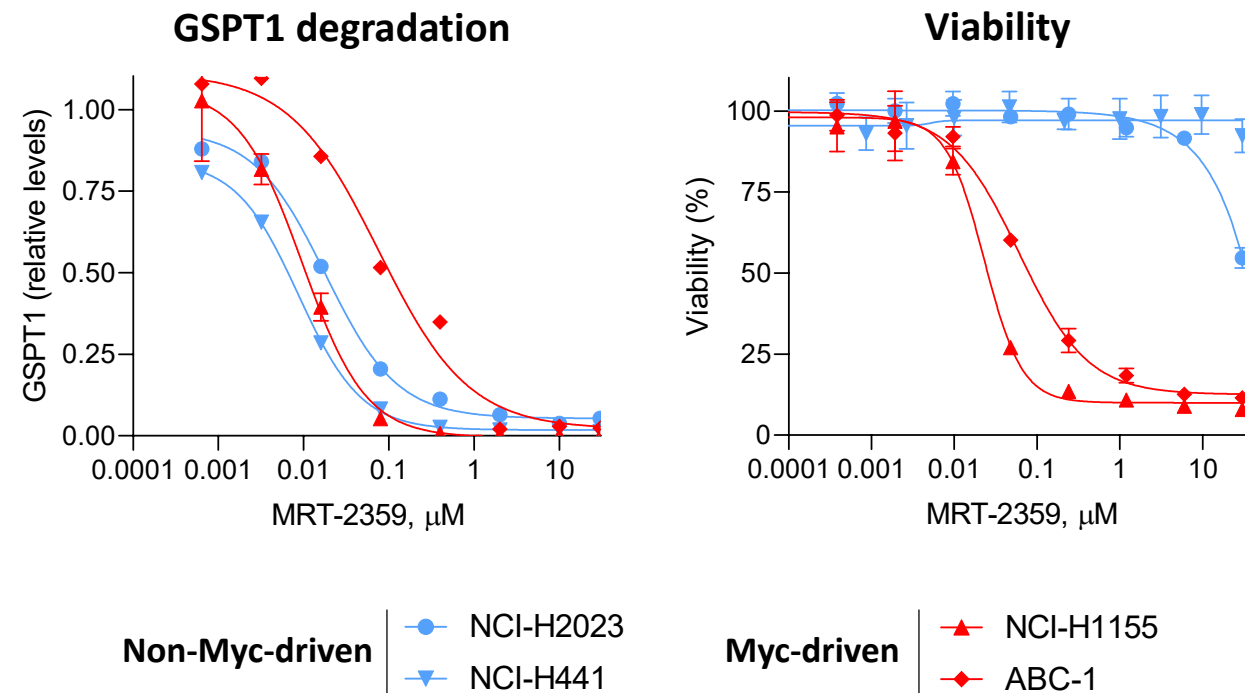**Protein fold-change; (log$_2$)**

1hr post treatment

Myc-Driven NSCLC lines are Highly Sensitive to MRT-2359
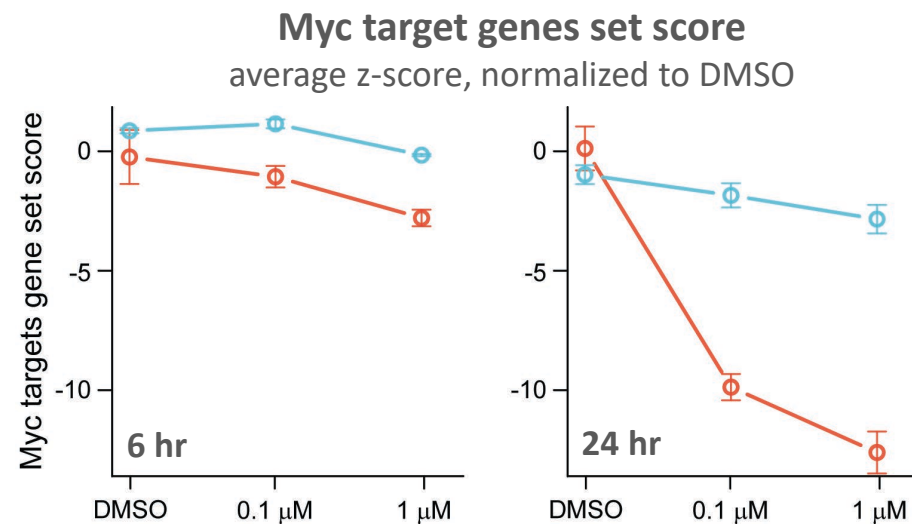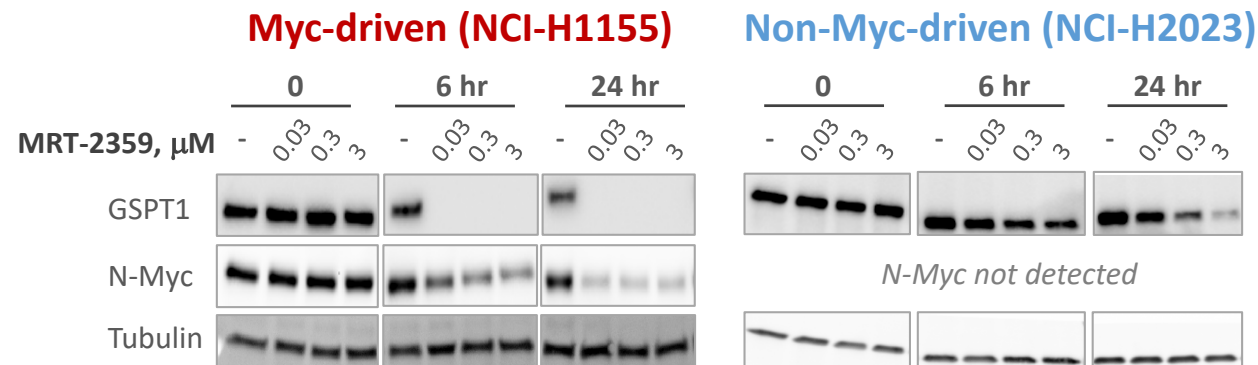
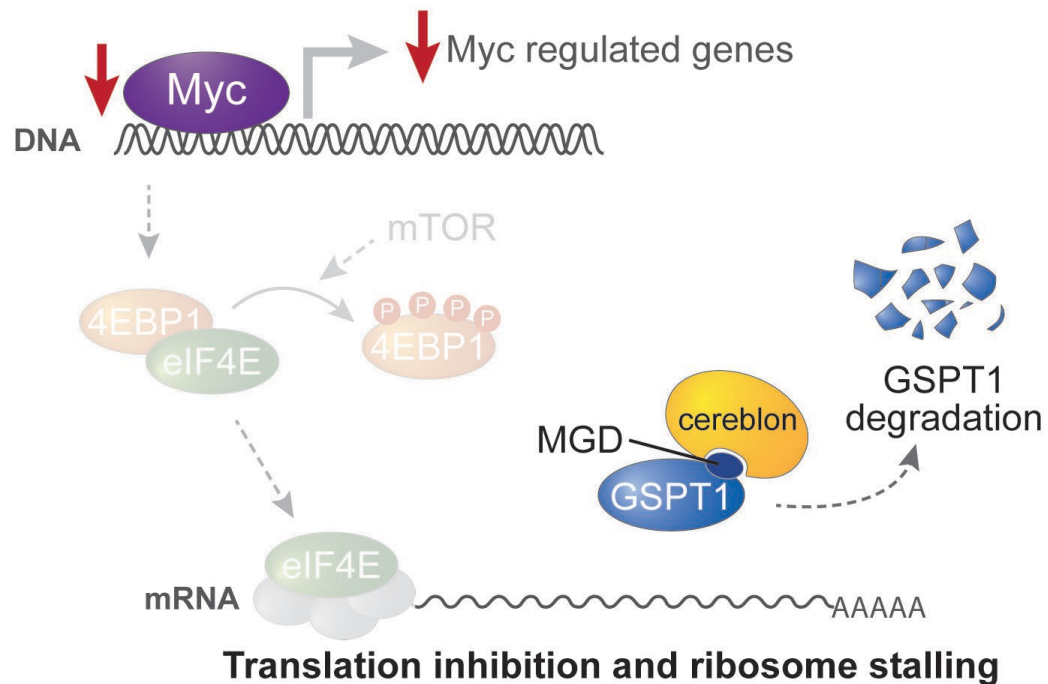## Myc-driven NSCLC cell lines are sensitive to MRT-2359

Each dot represent a cell line

r=−0.22

MRT−2359 EC$_{50}$ (nM) vs Biomarker score

Non-Myc-driven ●

Myc-driven | ● High N−Myc
          | ● High p4EBP1

MRT-2359 induces GSPT1 degradation in all cell models, but selective killing in high N-Myc lines only

GSPT1 degradation

GSPT1 (relative levels) vs MRT-2359, μM

Viability

Viability (%) vs MRT-2359, μM

Non-Myc-driven | ● NCI-H2023
                   | ▼ NCI-H441

Myc-driven | ▲ NCI-H1155
               | ◆ ABC-1

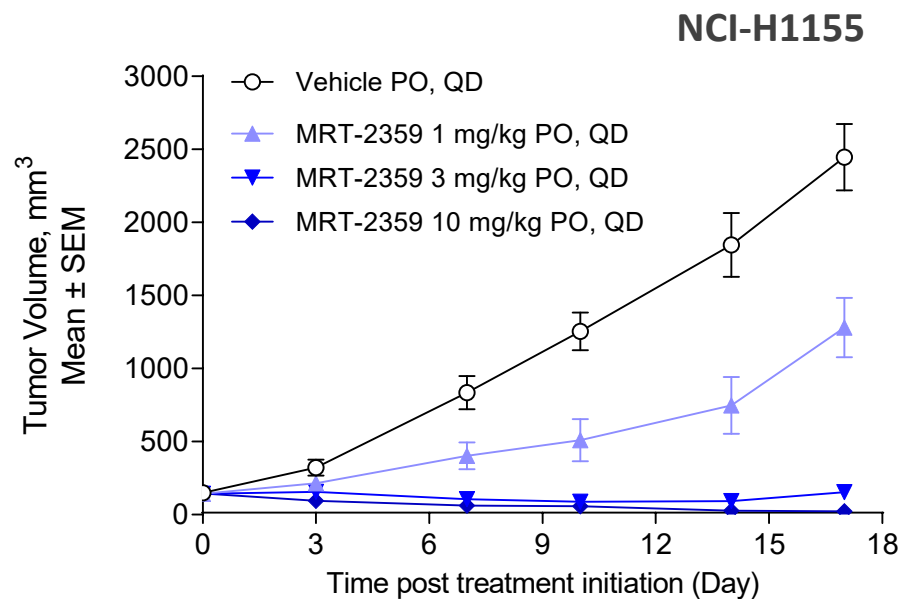*GSPT1 western blot at 6 hr (N-Myc high) and 24 hr (low). 72 hr viability assay (CTG)*

MRT-2359 Only Effects N-Myc Pathway in Myc-driven Cells

GSPT1-directed MGD degradation affect translation, a critical vulnerability of Myc-driven cells

Myc → Myc regulated genes

DNA — Myc

mTOR
4EBP1 / eIF4E → P P P P 4EBP1

MGD — cereblon — GSPT1 → GSPT1 degradation

mRNA — eIF4E — AAAAA

Translation inhibition and ribosome stalling

Myc-driven (NCI-H1155)   **Non-Myc-driven (NCI-H2023)**

| | 0 | 6 hr | 24 hr | 0 | 6 hr | 24 hr |

MRT-2359, µM: - 0.03 0.3 3

GSPT1
N-Myc
Tubulin

N-Myc not detected

Myc target genes set score
average z-score, normalized to DMSO

Myc targets gene set score

6 hr: DMSO, 0.1 µM, 1 µM

24 hr: DMSO, 0.1 µM, 1 µM

Oral dosing of MRT-2359 Induces Regressions in Myc-driven Xenograft Model

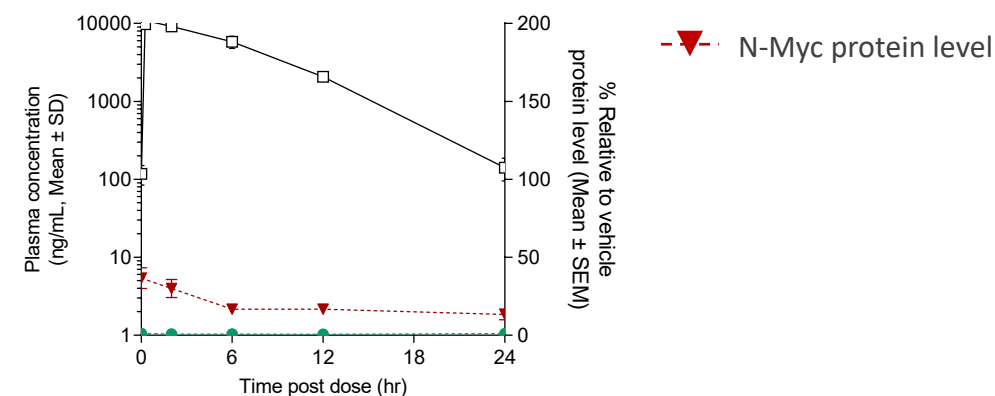## Oral dosing of MRT-2359 shows strong activity in Myc-driven Xenograft NSCLC model

NCI-H1155

- Vehicle PO, QD
- MRT-2359 1 mg/kg PO, QD
- MRT-2359 3 mg/kg PO, QD
- MRT-2359 10 mg/kg PO, QD

Dose- and time-dependent degradation of GSPT1 is associated with N-Myc downregulation

Day 5

1 mg/kg

10 mg/kg

- MRT-2359 plasma concentration
- GSPT1 protein level
- N-Myc protein level
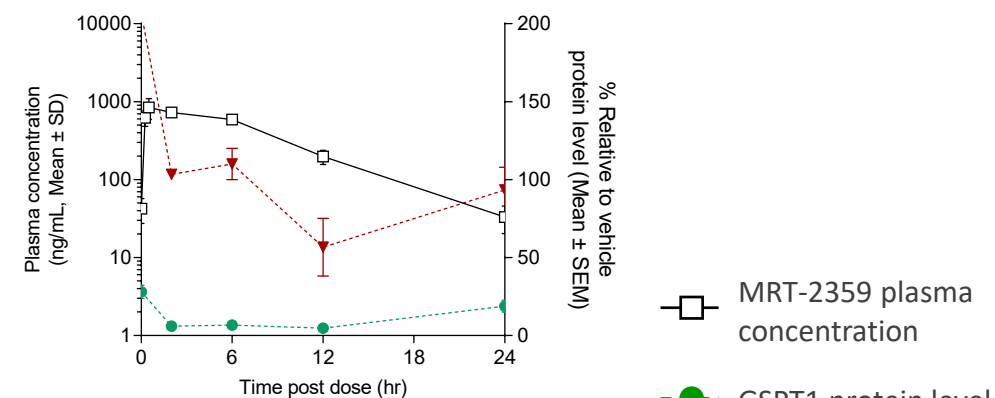
Targeting Myc-driven Tumors with GSPT1-directed MGDs

Potential indications and patient stratification hypotheses

Indications considered for Phase I/II trial
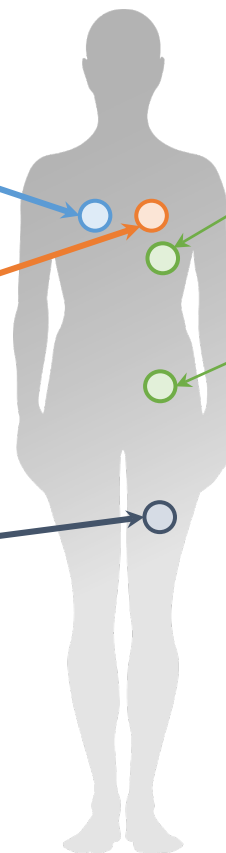
Additional possible indications

Small cell lung cancer – 66K patients
50%

Non-small cell lung cancer – 352K patients (LUAD + SCC)
7 - 10%

Multiple myeloma – 61K patients
36%

Lymphomas – 8K patients
30 - 100%

Triple-neg breast cancer – 68K patients
30%

Ovarian cancer – 63K patients
64%

Tumors with neuroendocrine features

Neuroblastoma

Retinoblastoma

Medulloblastoma

Neuroendocrine lung cancer, Lu-NET

Neuroendocrine prostate cancer, NEPC

L-Myc overexpression/amplification N-Myc overexpression/amplification c-Myc amplification c-Myc translocation/rearrangement

Patient diagnosed incidence #s, major markets (US, EU and JP): Decision Resources Group (DRG)
Patient stratification %s: Schaub - Cell Systems 2018; Massó-Vallés – Exp. Op. therapeutic targets 2020; Sesques and Johnson - Blood 2016

Targeting Myc-addicted Tumors with MRT-2359

IND-enabling activities have been initiated

- Rationally designed **potent and selective** GSPT1-directed MGD

- **Orally bioavailable** development candidate

- Favorable **drug-like properties** and ADMET profile

- Robust **antitumor activity** *in vivo* in **multiple tumor models**

- **IND-enabling** activities initiated

- **Patient stratification hypothesis** developed and being validated

IND filing
in mid-2022

Discovery Stage Programs

Discovery Stage Pipeline
Advancing multiple programs into Lead Optimization

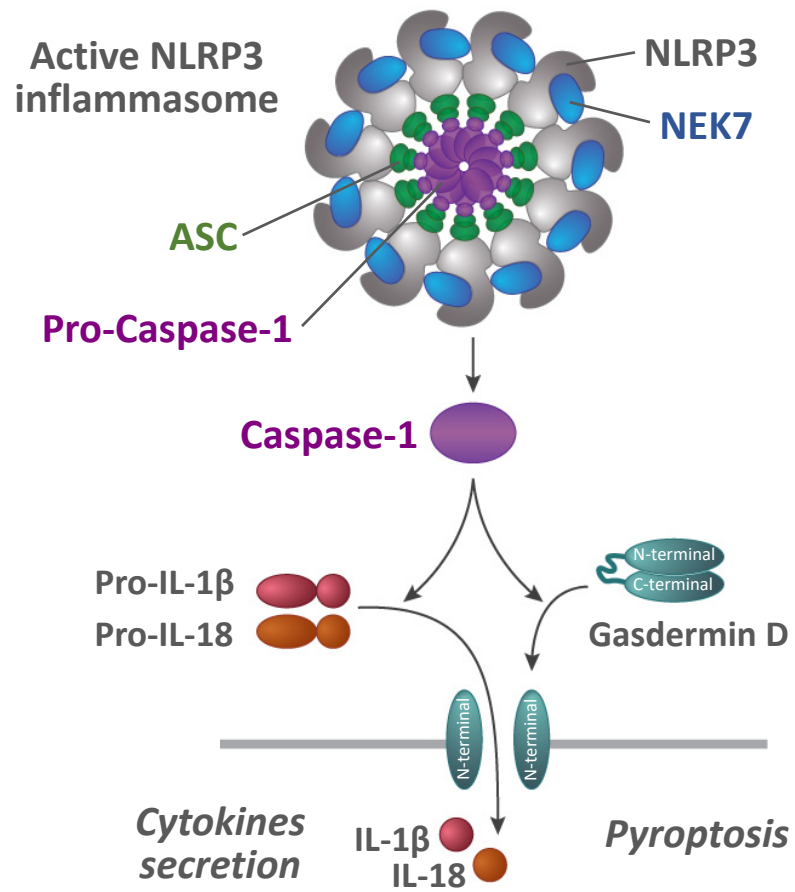| Target / Program	Indication(s)	Discovery	Lead Optimization
NEK7	Inflammatory Diseases		
CDK2	Ovarian Cancer, Breast Cancer		
VAV1	T and B Cell Malignancies, Autoimmune Disease		
BCL11A	SCD, β-Thalassemia		
Undisclosed	Multiple		

● Oncology   ● Autoinflammation   ● Oncology / immunology   ● Genetic diseases

NEK7 (NIMA-Related Kinase 7) as a Target for Inflammatory Disease

An opportunity to expand into the non-oncology disease space

NEK7 is an essential regulator of the inflammasome

Active NLRP3 inflammasome

NLRP3

NEK7

ASC

Pro-Caspase-1

Caspase-1

Pro-IL-1β
Pro-IL-18

Gasdermin D

N-terminal
C-terminal

Cytokines secretion

IL-1β
IL-18

Pyroptosis

Target profile

✓ Therapeutic hypothesis: Diseases with over-activated or mutated NLRP3 inflammasome
  - NEK7 licenses NLRP3 assembly in a kinase independent manner
  - NEK7-deficient macrophages are severely impaired in IL-1β and IL-18 secretion

✓ Clinical opportunity: First-in-class NEK7 degraders for
  - Over-activated NLRP3 inflammasome: metabolic pathologies, cardiovascular diseases, inflammatory and neurologic disorders (e.g., Gout 17.1M patients, NASH 2.3M patients, acute HF 8.2M patients)
  - NLRP3 activating mutations: Cryopyrin-associated periodic syndromes (1-2 patients per million in EU and US)

✓ Differentiation: Novel target within the inflammasome and mechanism of action

Patient diagnosed prevalence #s, major markets (US, EU and JP): DRG; www.rheumatologyadvisor.com

Rationally Designed NEK7-Directed MGDs are Selective Degraders

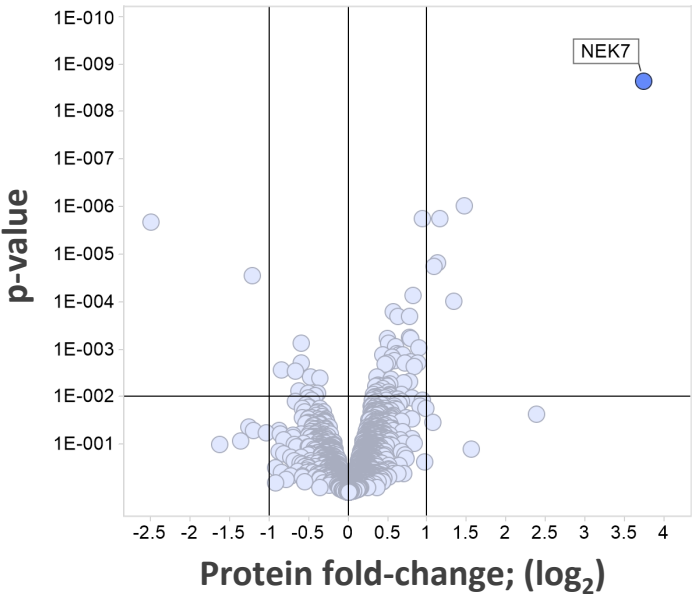*Demonstration of selective NEK7 degradation with MGD treated cells*

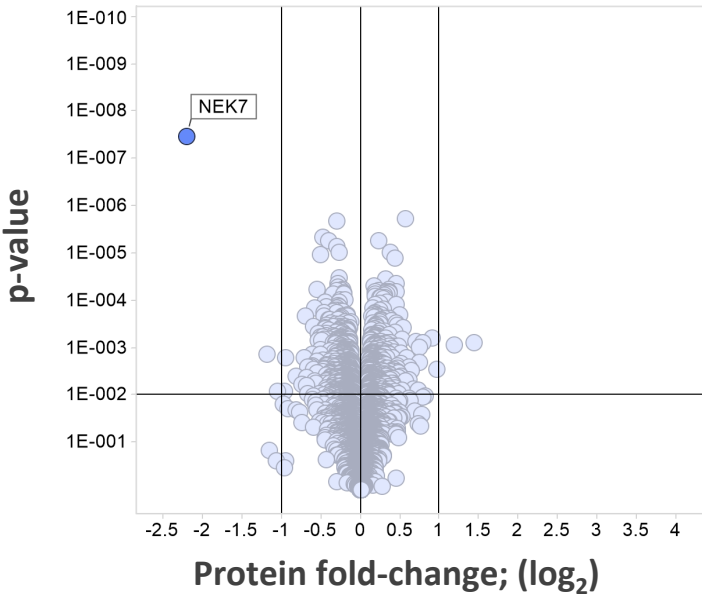### Rhapsody™ model enables rapid chemistry optimization

NEK7

MGD

Cereblon

degron
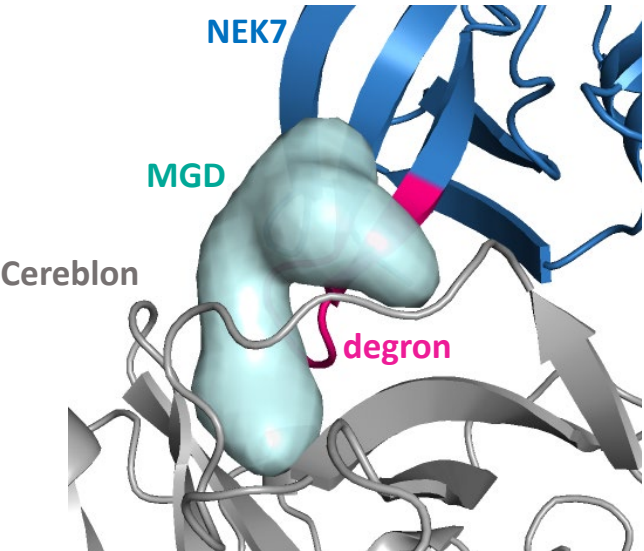
***in vitro* data**	
CRBN binding, K_i	48 nM
Ternary complex, EC_{50}	20 nM
Degradation, DC_{50}	10 nM

Rationally designed MGDs promote selective CRBN proximity

Turbo-ID – 6hr post treatment

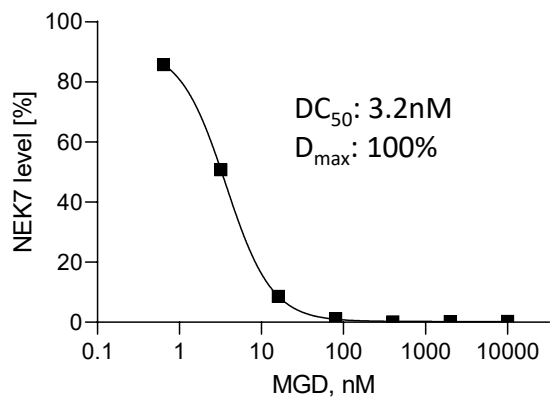### NEK7-directed MGD promotes selective degradation of NEK7
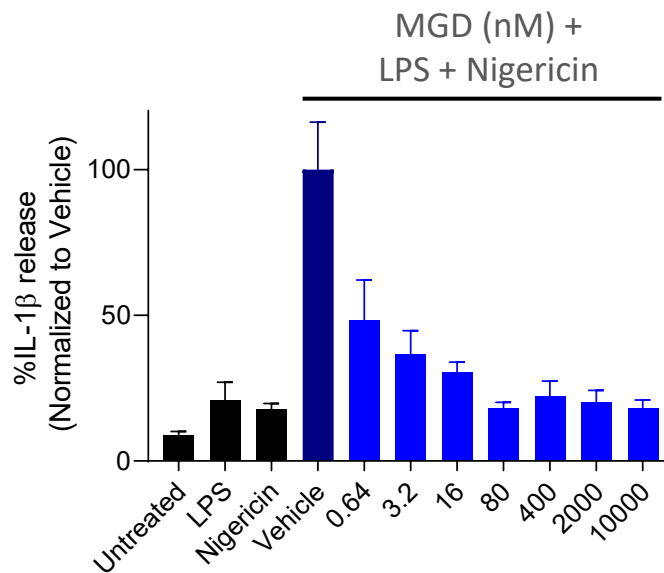

TMT-Proteomics – 24hr post treatment

35

Inhibition of NLRP3 Pathway in Human Monocyte-derived Macrophages

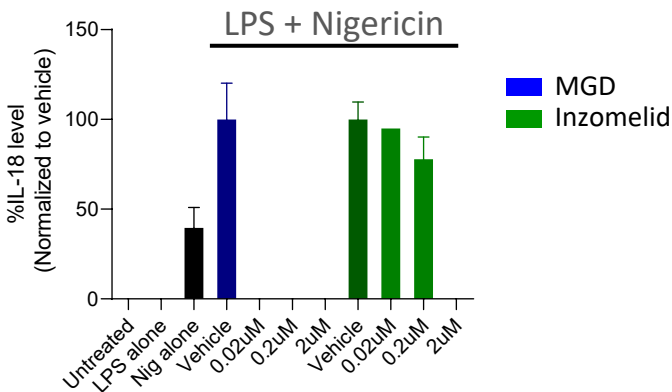## NEK7-directed MGD promotes NEK7 degradation and pathway engagement in hMDMs

DC$_{50}$: 3.2nM
D$_{max}$: 100%

MGD (nM) +
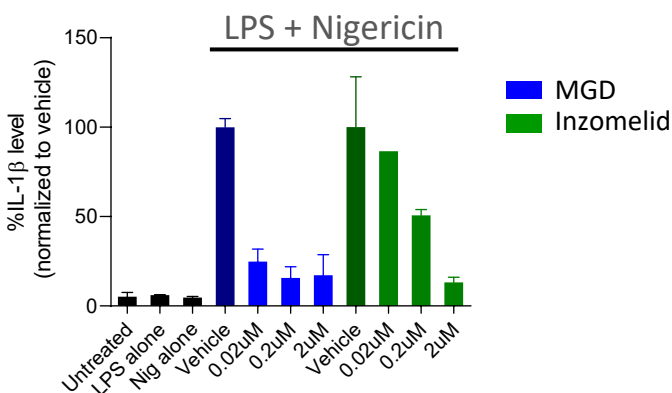LPS + Nigericin

Western blot – 24 hr

Treatment (20 hr) of primed hMDMs

NEK7-directed MGD compared to NLRP3 inhibitor

IL-18

IL-1β

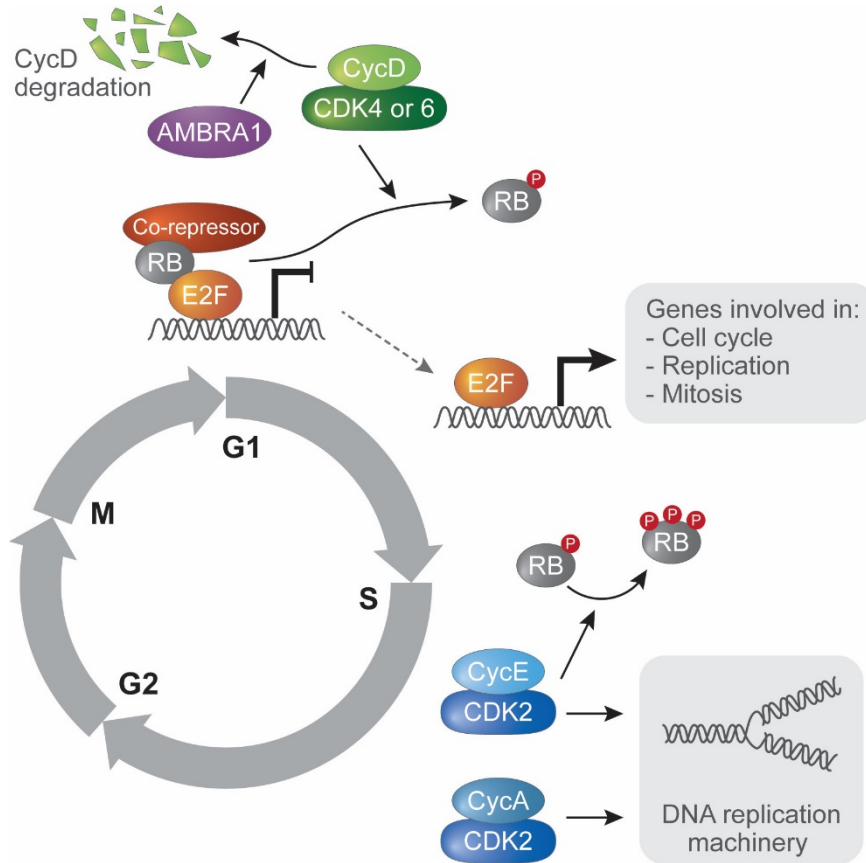*Treatment (20 hr) of primed hMDMs*

CASP1 and LDH showed similar profile

CDK2 as a Target for Solid Tumors

Unlocking the potential to achieve target selectivity through recognition of the degron

CDK2 is one of the key regulators of the cell cycle

Target profile

✓ **Therapeutic hypothesis:** Tumors with CDK2 pathway activation by:
- CyclinE1/E2 amplification or loss of AMBRA1
- Loss of RB

✓ **Clinical Opportunity:** CDK2 driven cancers: ER positive breast cancer (444K patients), ovarian cancer (63K patients), and endometrial cancer (118K patients), as well as breast cancer post treatment with CDK4/6 inhibitors

✓ **Differentiation:** Opportunity to selectively target CDK2 over other CDKs

Patient diagnosed incidence #s, major markets (US, EU and JP): DRG

Rationally Designed CDK2-Directed MGDs are Selective Degraders
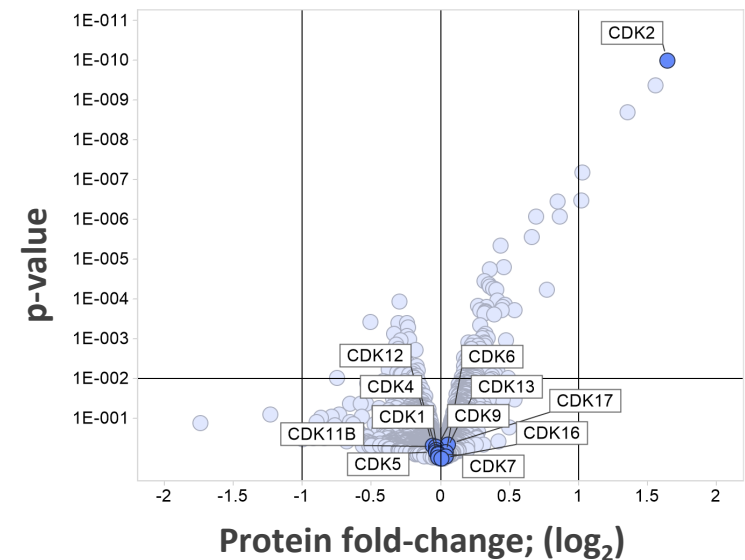## *Demonstration of selective CDK2 degradation with MGD treated cells*

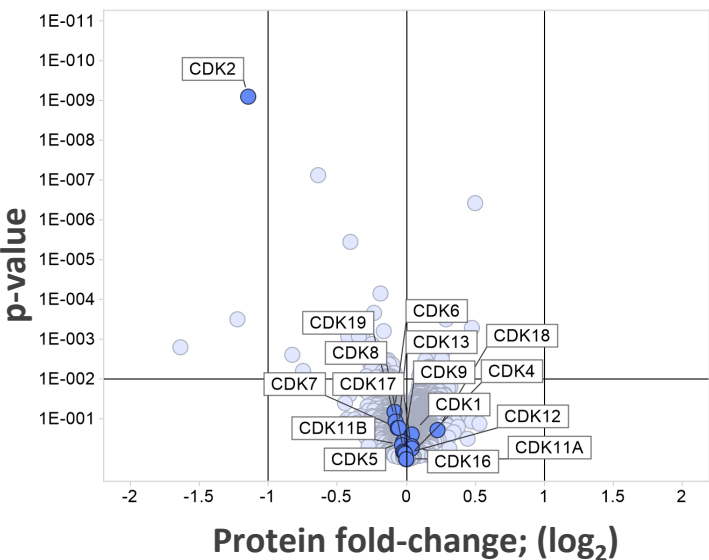### Rhapsody™ model enables rapid chemistry optimization
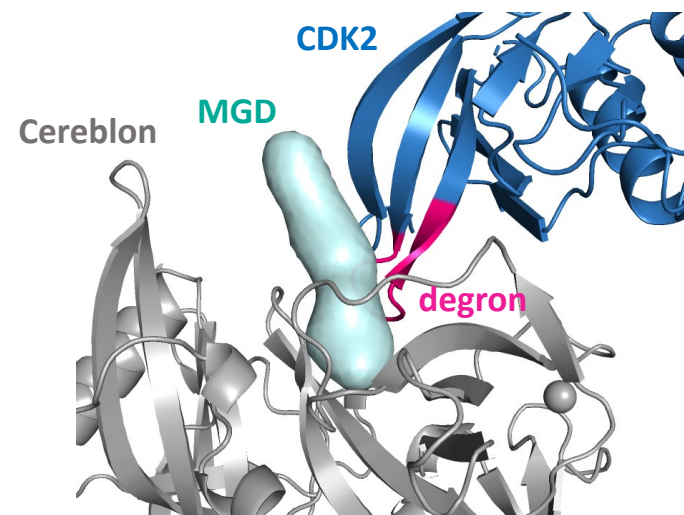
in vitro data	
CRBN binding, K_i	163 nM
Ternary complex, EC_{50}	9 nM

Rationally designed MGDs promote more selective CRBN proximity

Turbo-ID – 6hr post treatment
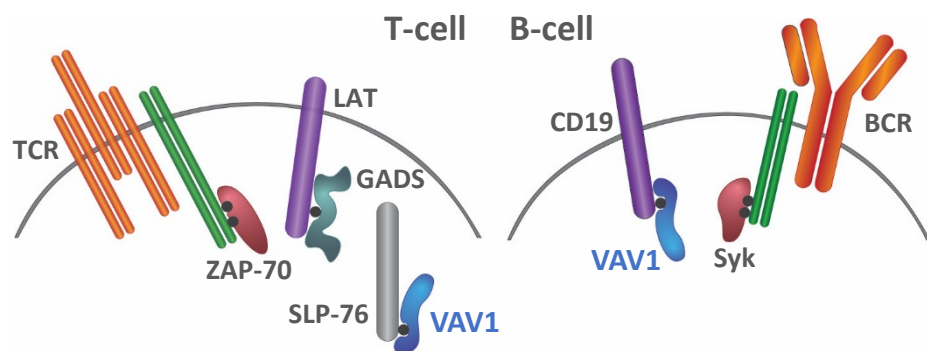
Rationally designed MGDs selectively degrade CDK2
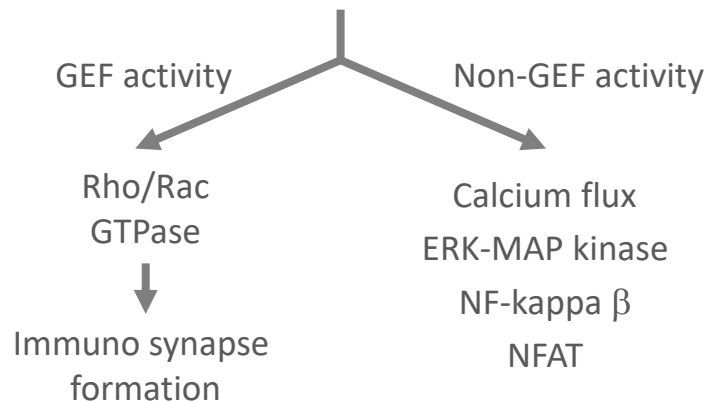

TMT-Proteomics – 24hr post treatment

VAV1 as a Target for Cancer and Autoimmune Disease
Potential to address an undruggable target

VAV1 plays a key role in T-cell and B-cell development and activation

T-cell   B-cell

LAT

TCR

CD19   BCR

GADS

ZAP-70

VAV1   Syk

SLP-76   VAV1

Activated VAV1

GEF activity   Non-GEF activity

Rho/Rac GTPase   Calcium flux

ERK-MAP kinase

Immuno synapse formation   NF-kappa β

NFAT

Target profile

✓ Therapeutic hypothesis: Diseases with VAV1 activating mutations or autoimmune disorders
  - VAV1 activation mutations identified in leukemia, lymphoma and lung cancer
  - VAV1 KO mice improved multiple autoimmune models

✓ Clinical Opportunity: First-in-class VAV1 degraders for
  - T-cell and B-cell lymphomas: DLBCL (66K patients) and Burkitt lymphoma
  - Autoimmune disorders including MS (1.2M patients), myasthenia gravis (36K - 60K patients in US), and acute graft-versus-host disease (10K patients)

✓ Differentiation: VAV1 is currently considered undruggable

Patient diagnosed prevalence and incidence #s, major markets (US, EU and JP): DRG; myasthenia.org

BCL11A as a Target for Hemoglobinopathies (SCD and β–Thalassemia)

BCL11A is the zinc finger transcription repressor of the fetal globin genes
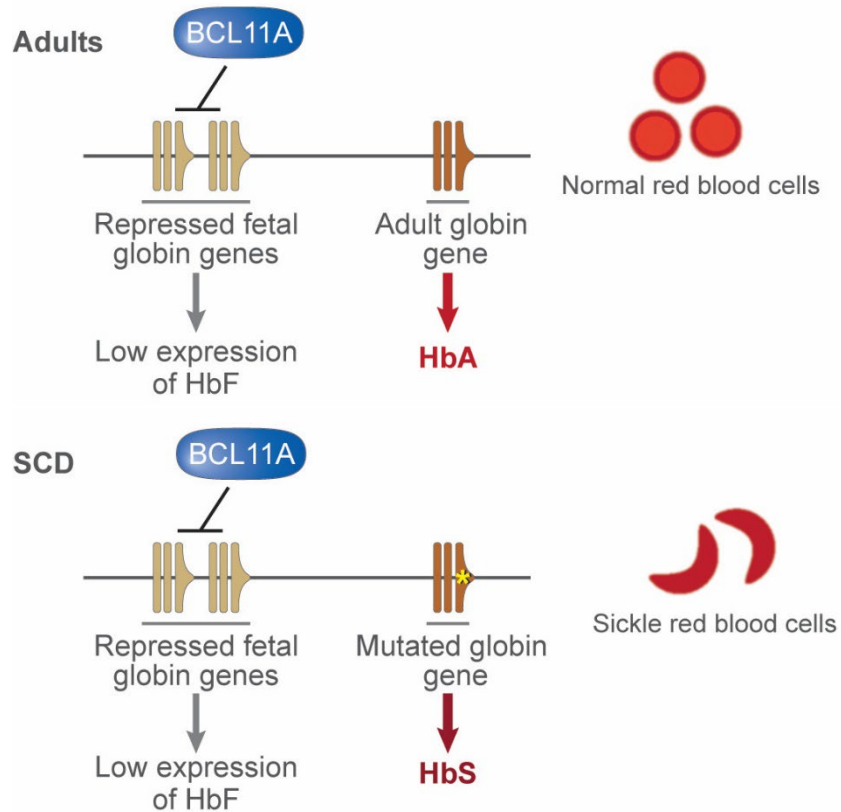

Target profile

- ✓ Therapeutic hypothesis: Reactivate expression of fetal hemoglobin (HbF) to compensate for mutated adult globin

- ✓ Clinical Opportunity: First-in-class BCL11A degraders for
  - Sickle cell disease (SCD)
    - 155,000 patients (US and EU)
    - >6M patients (ROW)
  - β-thalassemia
    - 17,000 patients (US and EU)

- ✓ Differentiation: BCL11A is currently considered undruggable

Patient diagnosed prevalence #s: DRG; www.notaloneinsicklecell.com

Unlocking the Full Potential of Protein Degradation with MGDs

Quantitative and engineered elimination of proteins across a broad spectrum of diseases

Target Space
(Degron)

Chemical Space
(MGD Library)

E3 ligases

Cereblon
(CRBN)
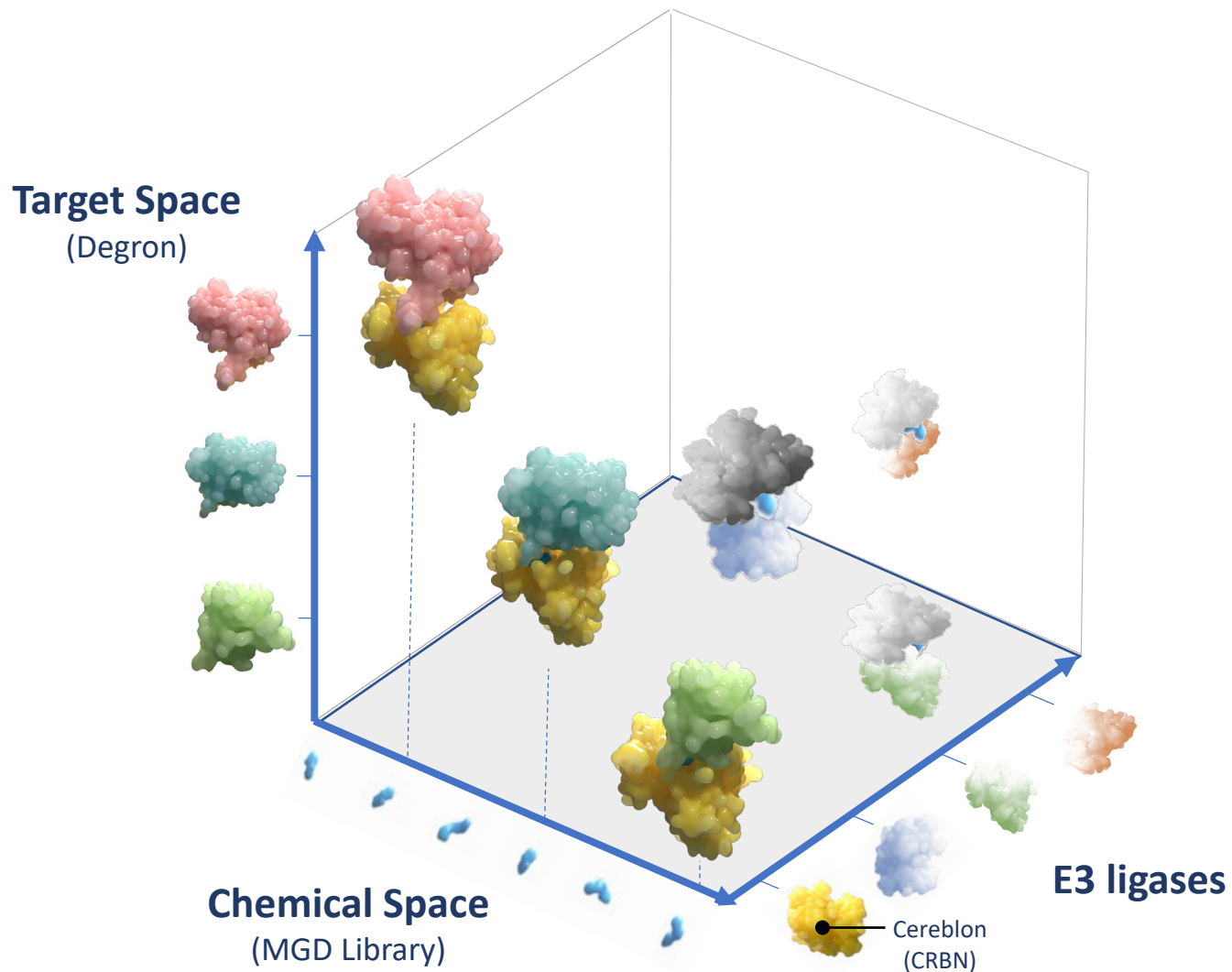
- Oncology
- Immunology
- Inflammation
- Metabolic disorders
- Neurodegeneration
- Genetic diseases

Monte Rosa
THERAPEUTICS

Thank You

MONTE ROSA 4634 M / 45°56'12.6"N 07°52'01.4"E / SWITZERLAND