Overcoming challenges of tuberculosis drug discovery and development

Guest speakers: Jeremy Rock, Dirk Schnappinger & Laura Cleghorn

Moderator: Valerie Mizrahi

Host: Shirine Derakhshani

9 September 2025

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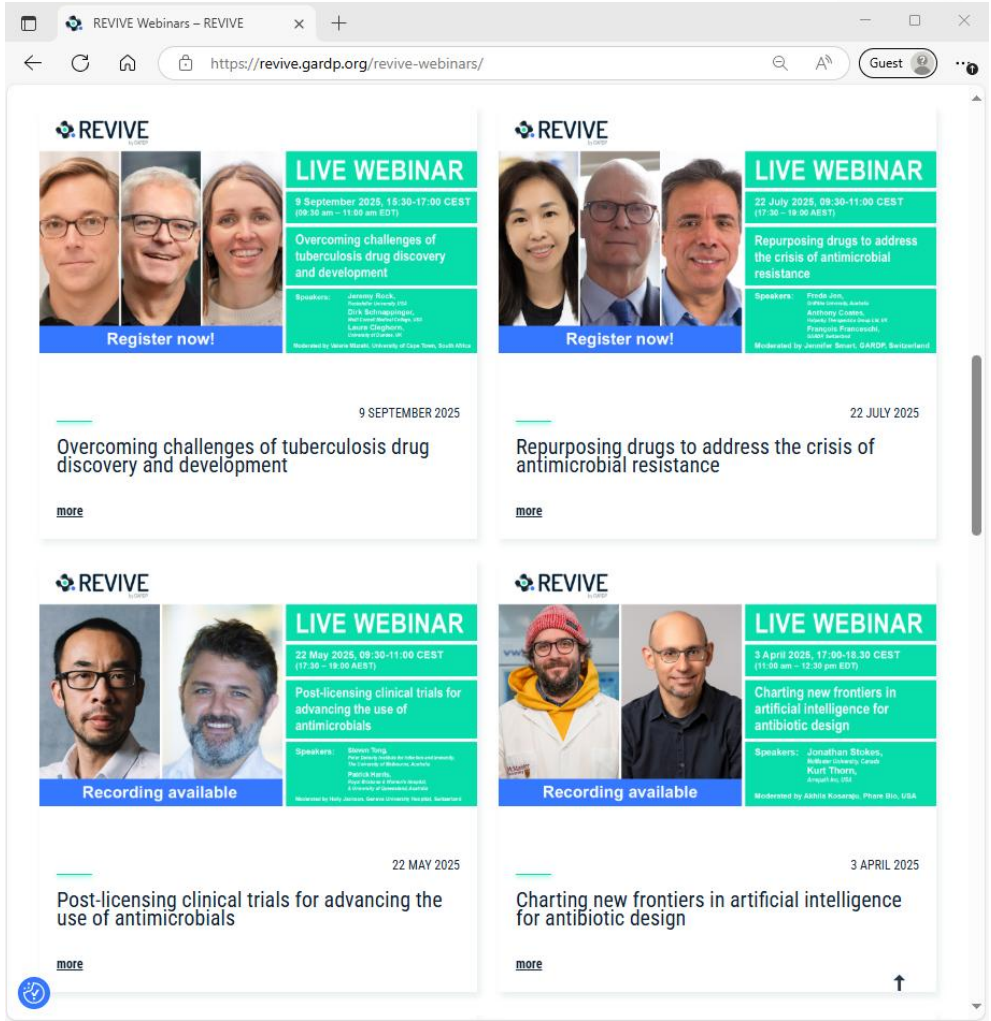

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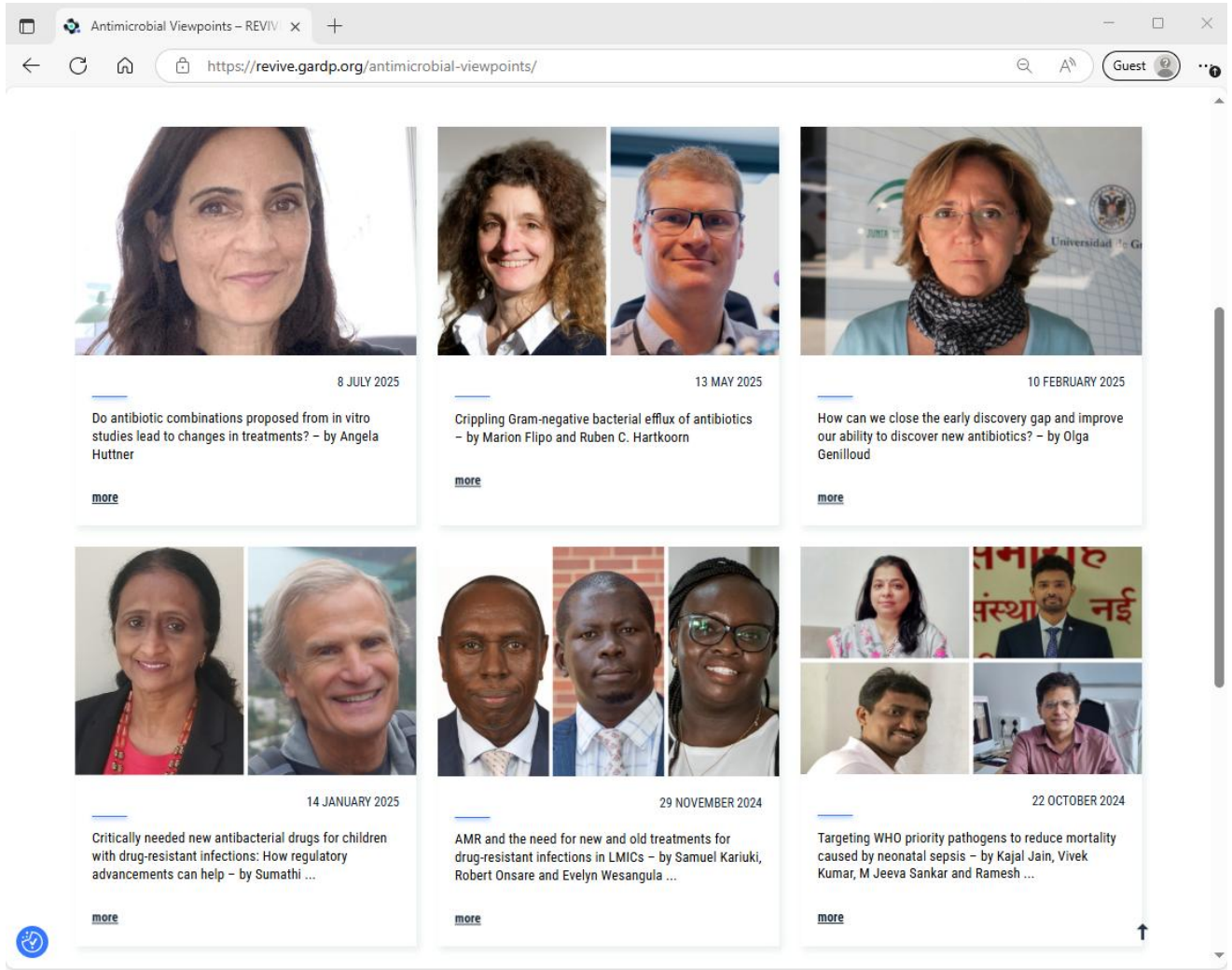

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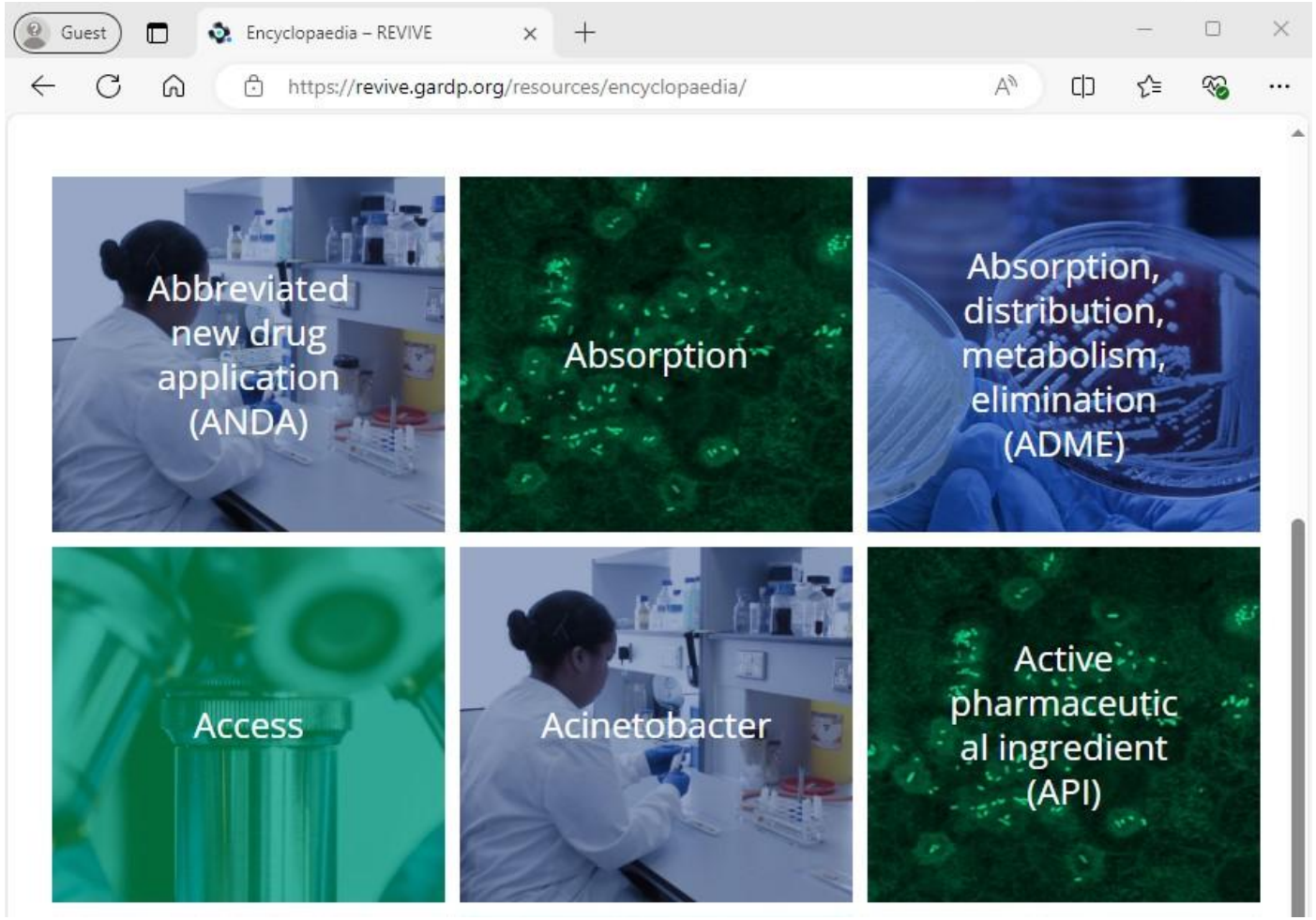

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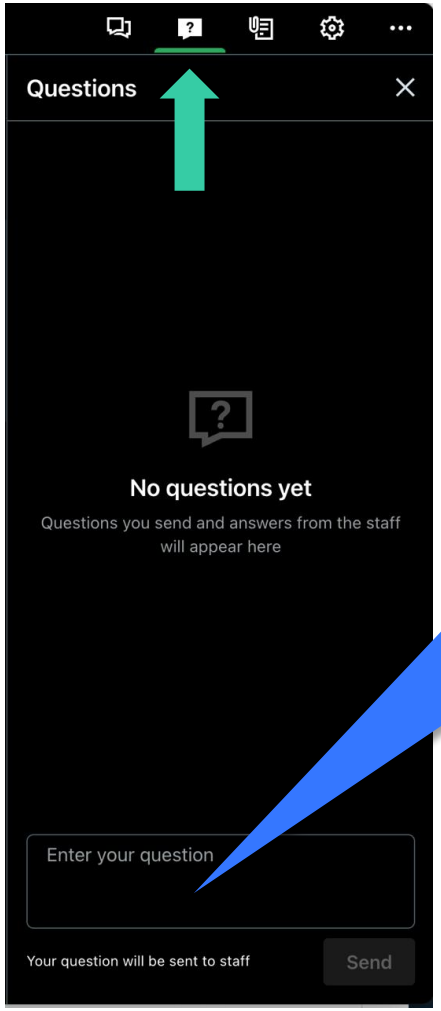
How to submit your questions

REVIVE by GARDP

If your question is addressed to a specific speaker, please include their name when submitting the question.



Please submit your questions through the box provided after clicking the ‘questions’ button. We will review all questions and respond to as many as possible after the presentation.

Today's speakers

REVIVE by GARDP

Overcoming challenges of tuberculosis drug discovery and development

Moderator:
Valerie Mizrahi
Former director, Institute of Infectious Disease and Molecular Medicine, University of Cape Town (South Africa)

Jeremy Rock
Associate Professor, Rockefeller University (USA)

Dirk Schnappinger
Professor, Department of Microbiology & Immunology, Weill Cornell Medical College (USA)

Laura Cleghorn
Reader, Drug Discovery Unit, School of Life Sciences, University of Dundee (UK)

Jeremy Rock

Jeremy Rock is an Associate Professor at the Rockefeller University and head of the Laboratory of Host-Pathogen Biology. His lab studies the human pathogen Mycobacterium tuberculosis, the leading cause of death due to infectious disease. The Rock lab uses functional and chemical genomics approaches to investigate the mechanisms by which these bacteria colonize their hosts and how they evade killing by antibiotics.

Jeremy received his undergraduate degrees in biochemistry and economics from the University of California, Berkeley. He then spent two years in the biotech industry at Sangamo Biosciences to develop new tools for genome editing. Following this, he earned his PhD from MIT where he studied cell cycle regulation with Angelika Amon. Jeremy found his calling in mycobacterial pathogenesis while performing postdoctoral studies at the Harvard School of Public Health with Sarah Fortune and Eric Rubin.

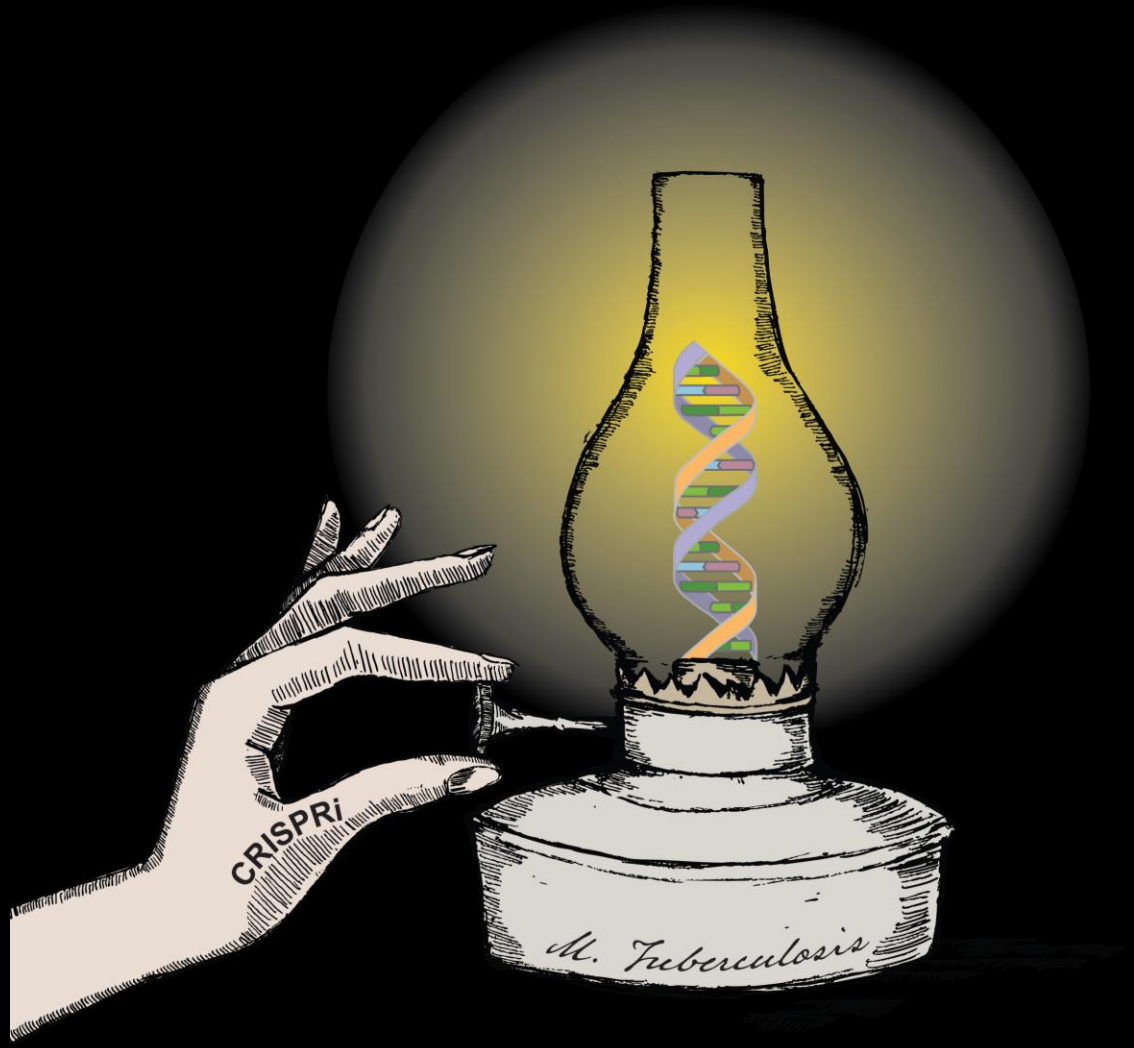
https://www.rocklaboratory.org/

Identifying vulnerable targets & pathways in *M. tuberculosis*

Jeremy Rock

Associate Professor
Head of the Laboratory
of Host-Pathogen Biology

The Rockefeller University
1901
SCIENTIA · PRO · BONO · HUMANI · GENERIS



Understand Mtb biology to build better therapies

Discover new biology that facilitates Mtb pathogenesis

Discover new drugs that perturb that biology and thereby inhibit Mtb pathogenesis

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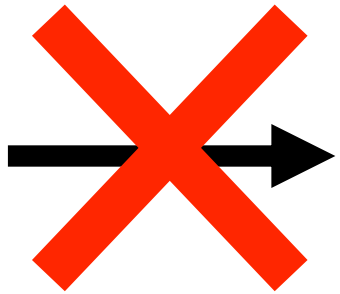
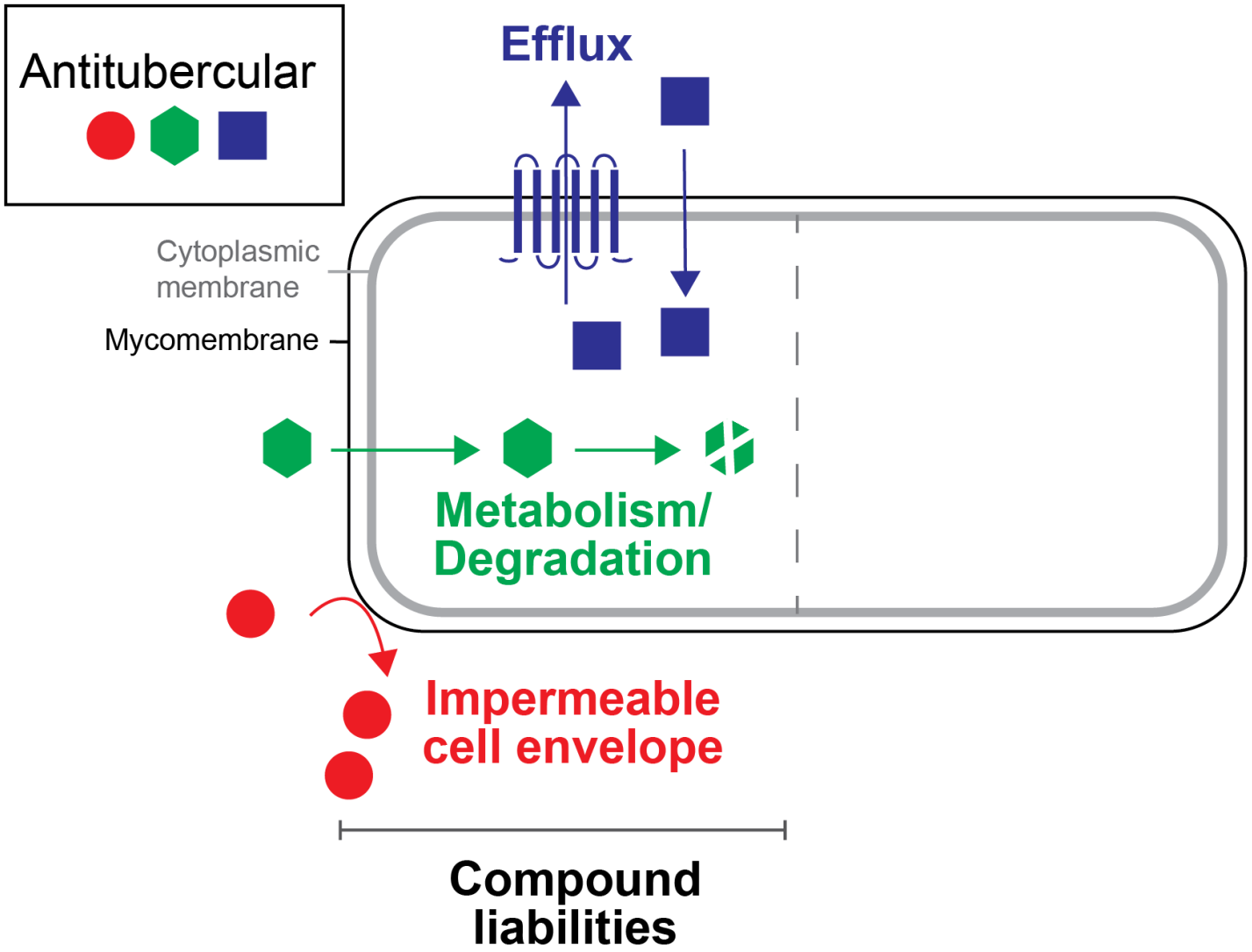


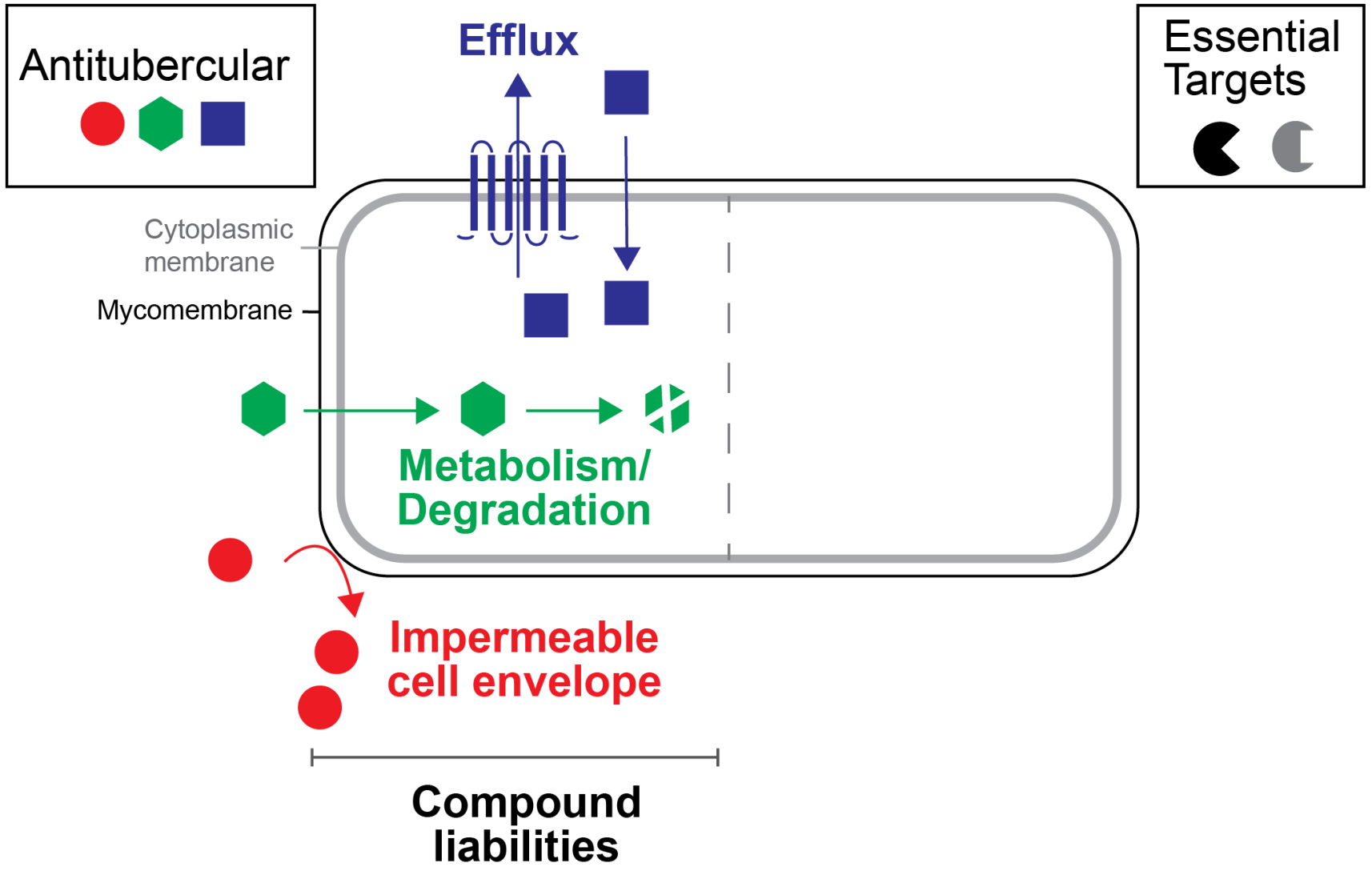
Really hard!

Discover new drugs that perturb that biology and thereby inhibit Mtb pathogenesis

Common failure modes for target-based drug discovery



Common failure modes for target-based drug discovery



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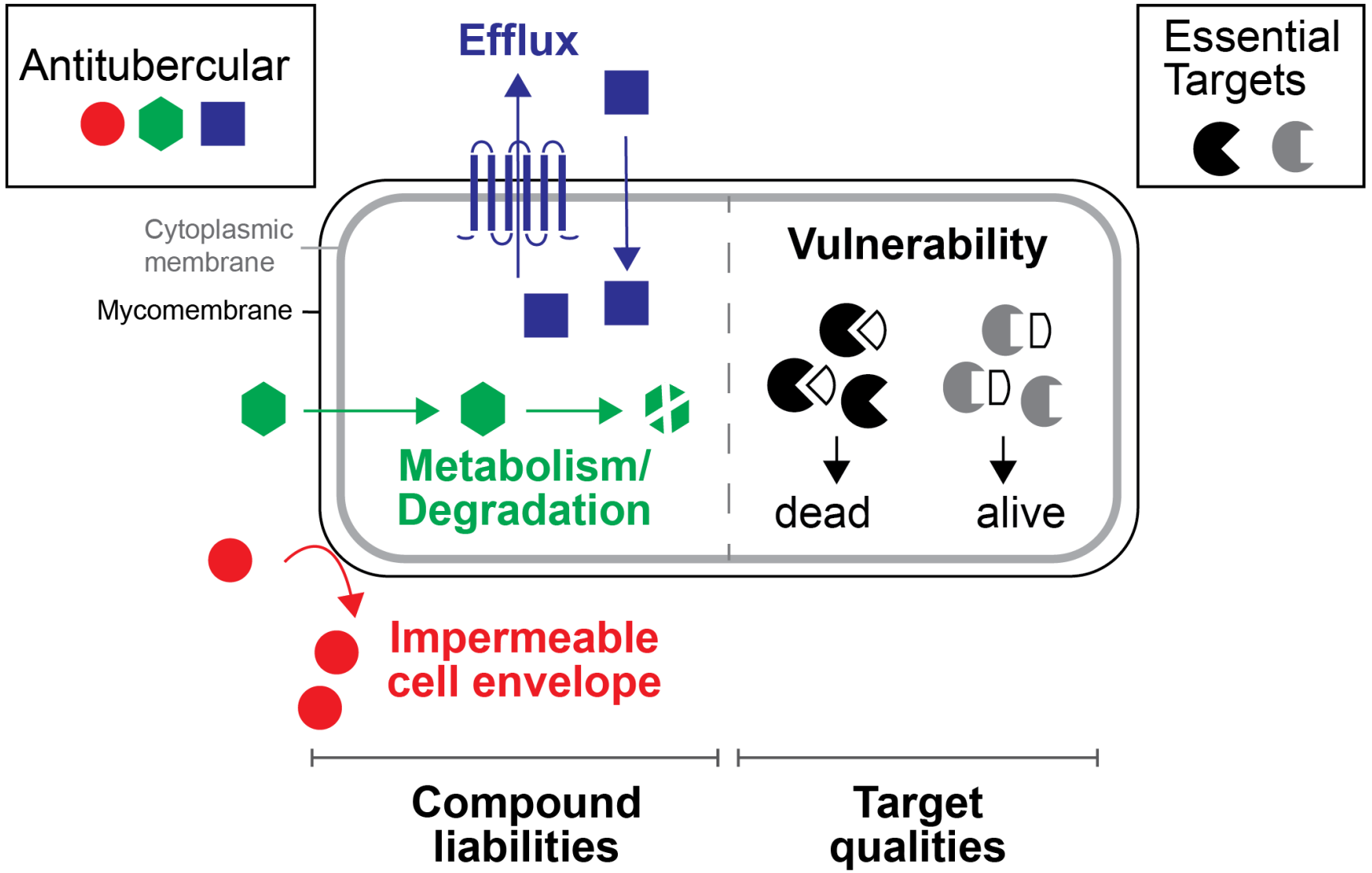

Defining target vulnerability

VULNERABILITY: CONTINUOUS VARIABLE

relates magnitude of gene inhibition with cell fitness

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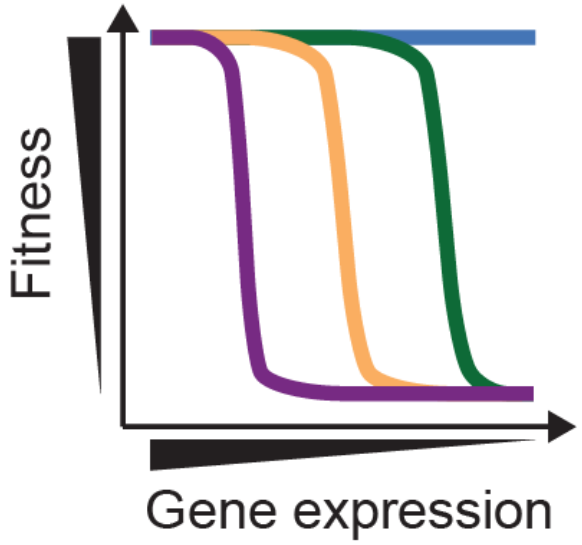

Gene 1	Essential
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Gene 3	Essential
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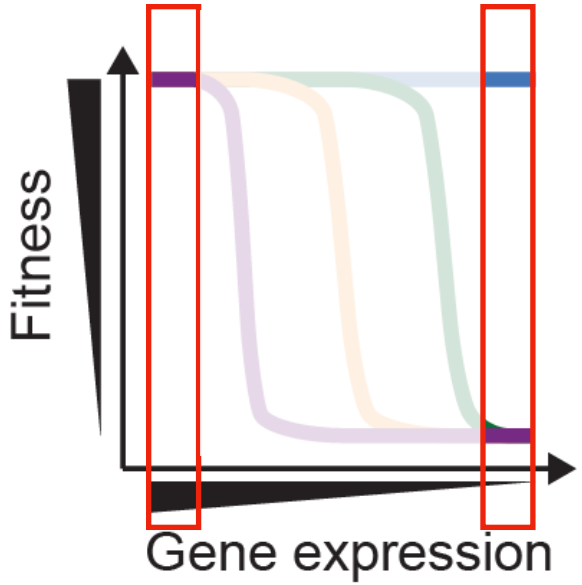

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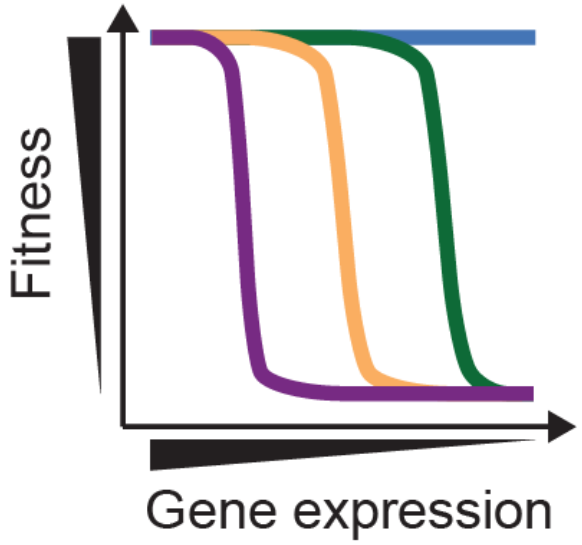

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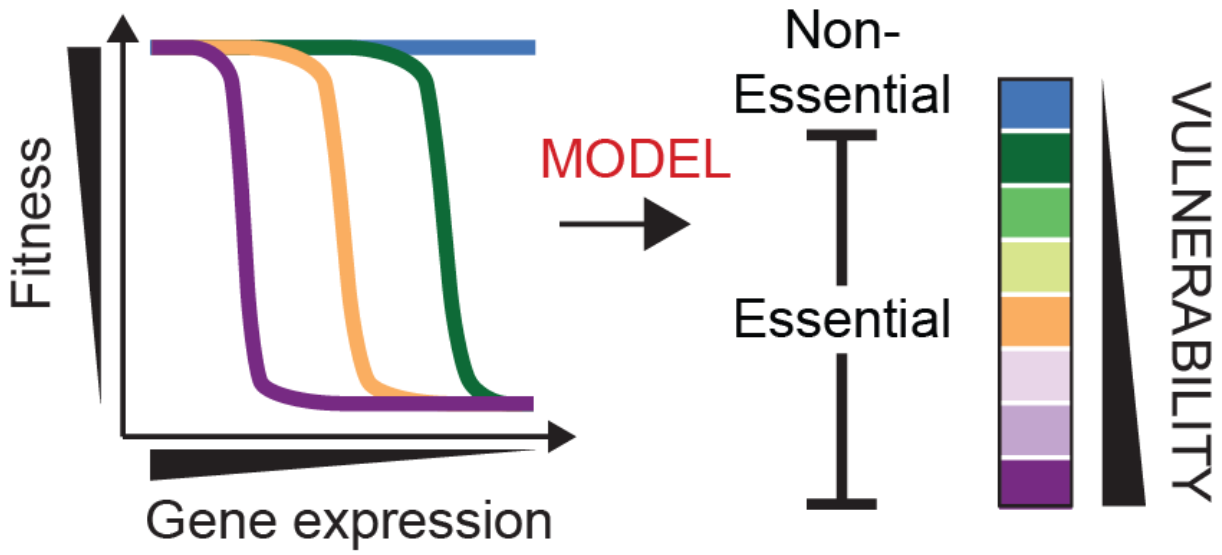

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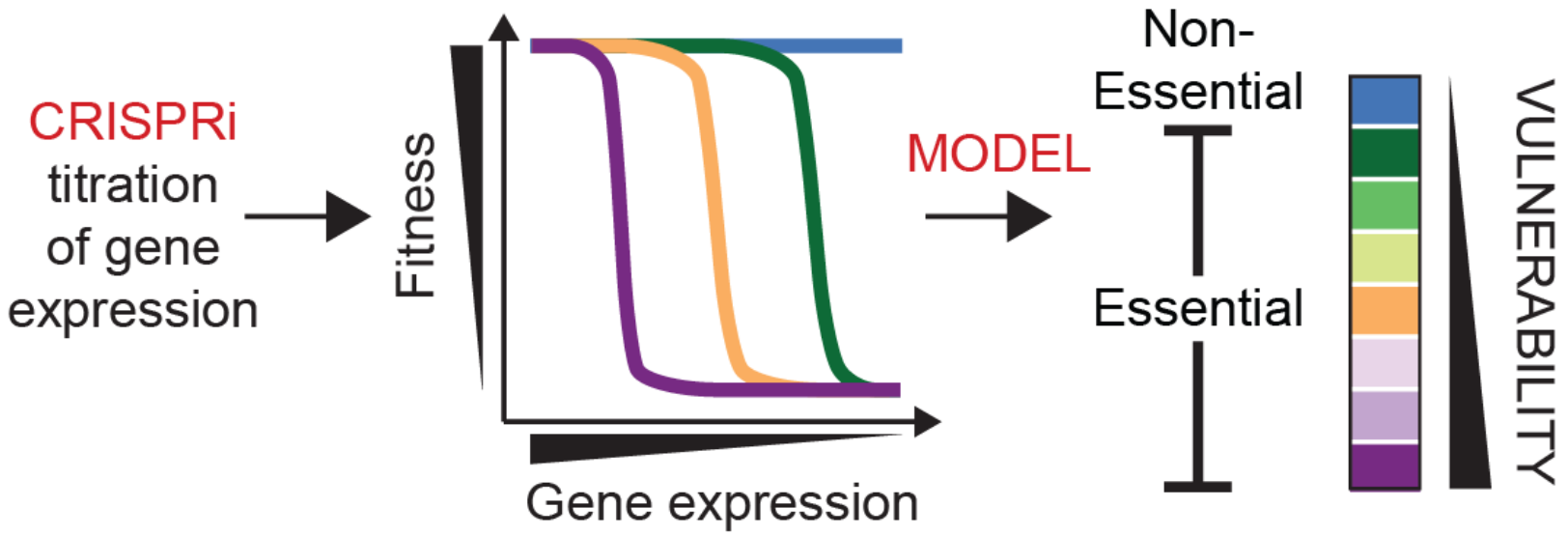


Defining target vulnerability

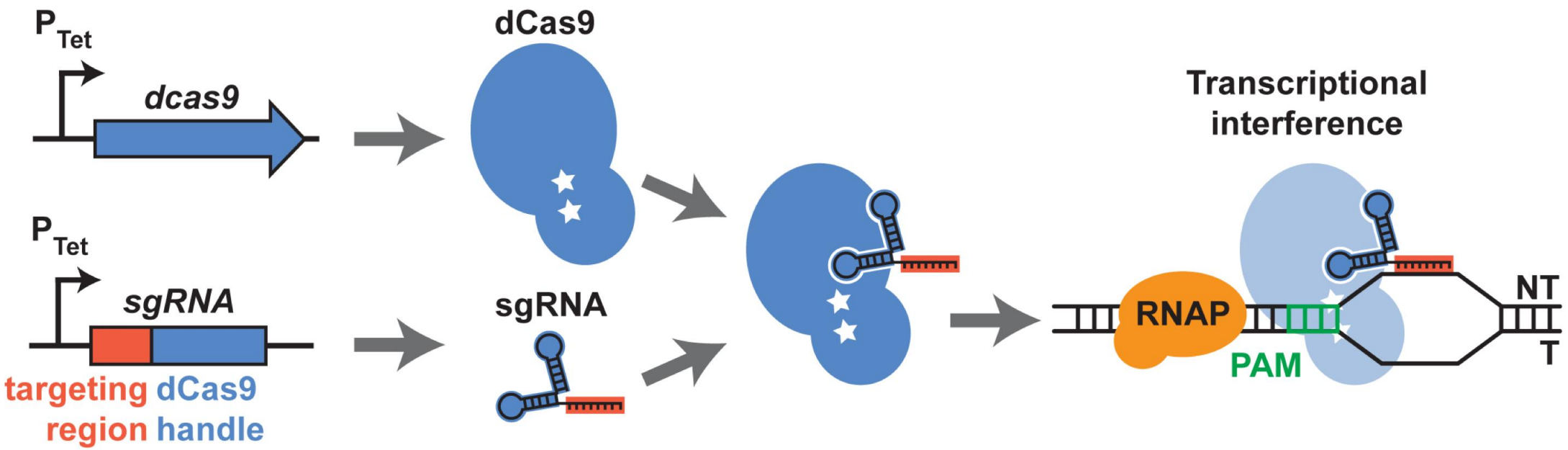
Barbara Bosch
(Former PhD student)

Michael DeJesus
(Senior Computational Scientist)

Dirk Schnappinger
(PI @ WCM)

Bosch & DeJesus et al, *Cell*, 2021

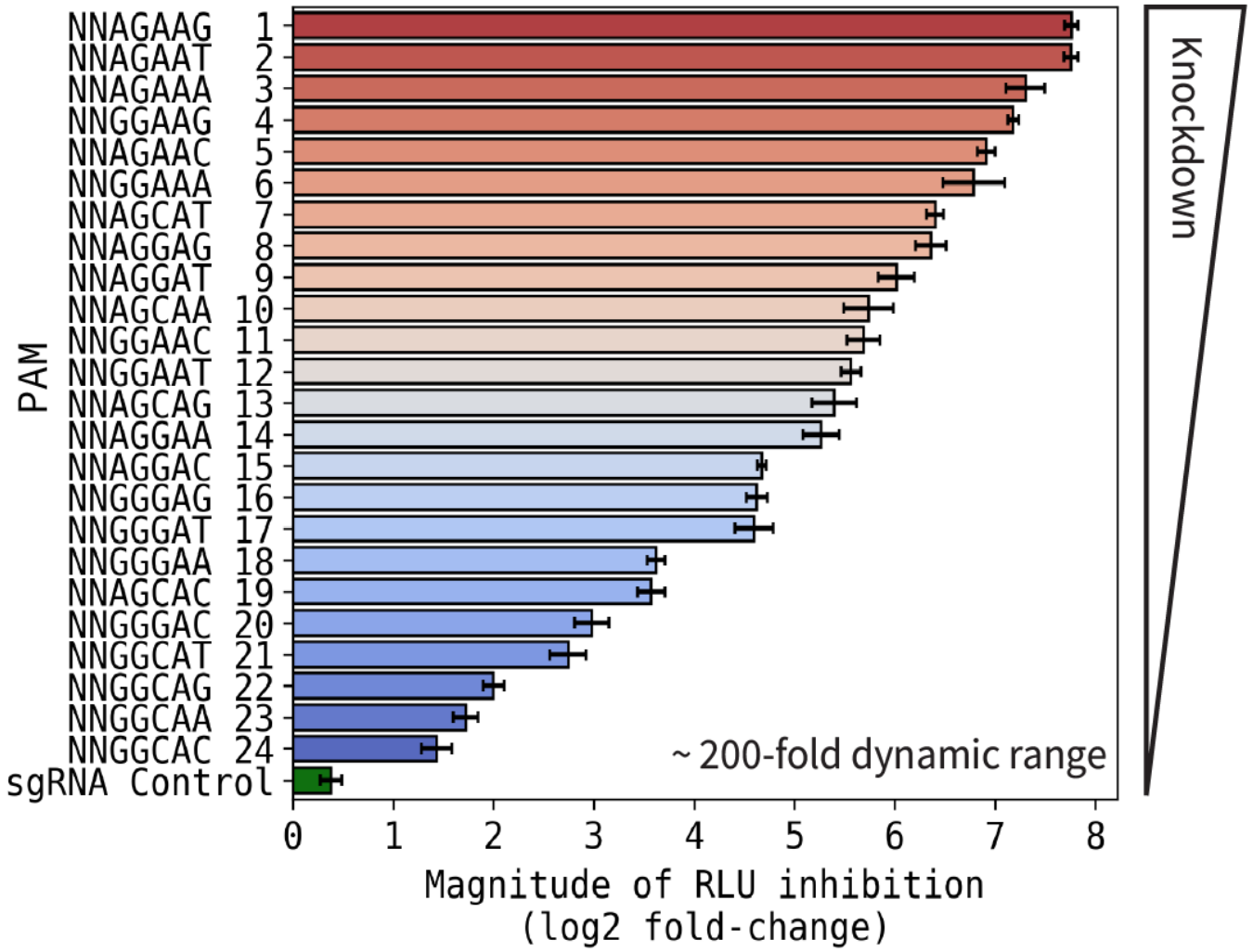
CRISPR interference for programmable target knockdown



Sth1 Cas9 consensus PAM:
5'-NNAGAAW-3'

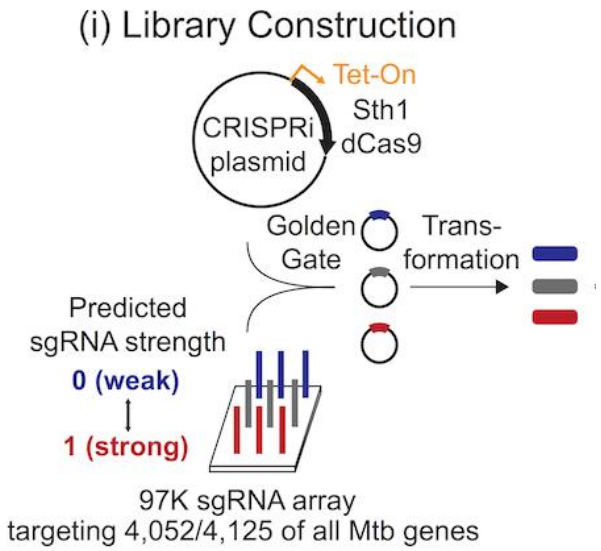
Rock et al, *Nat Micro*, 2017

Tunable CRISPRi with Sth1 dCas9



Rock et al, *Nat Micro*, 2017

Defining target vulnerability in Mtb



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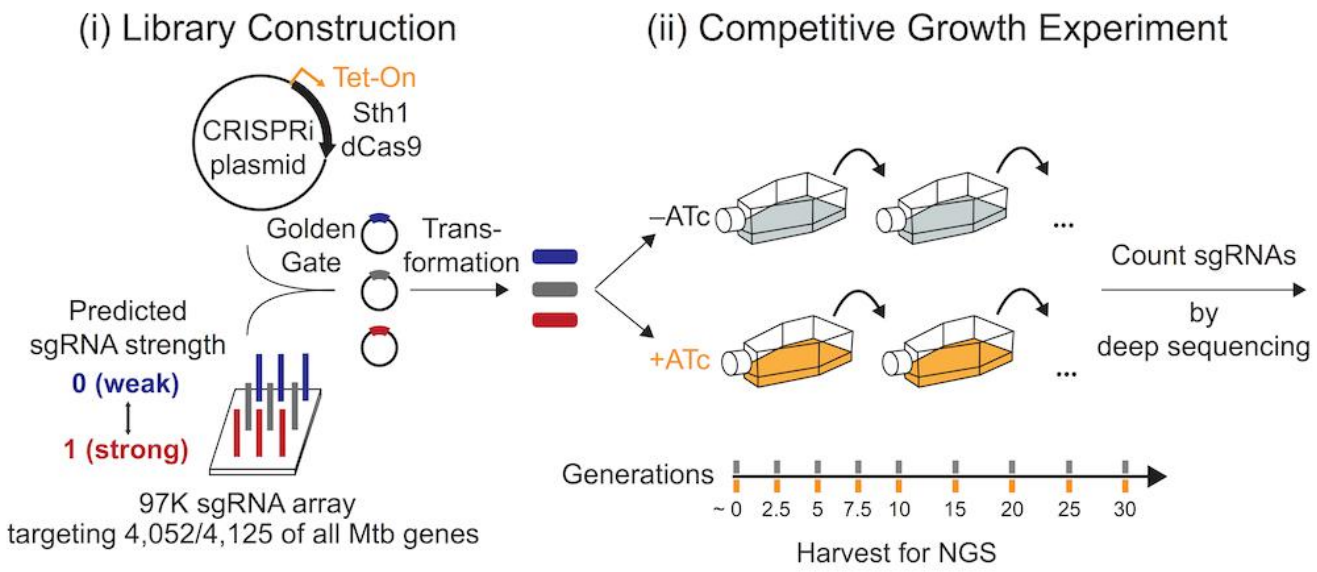

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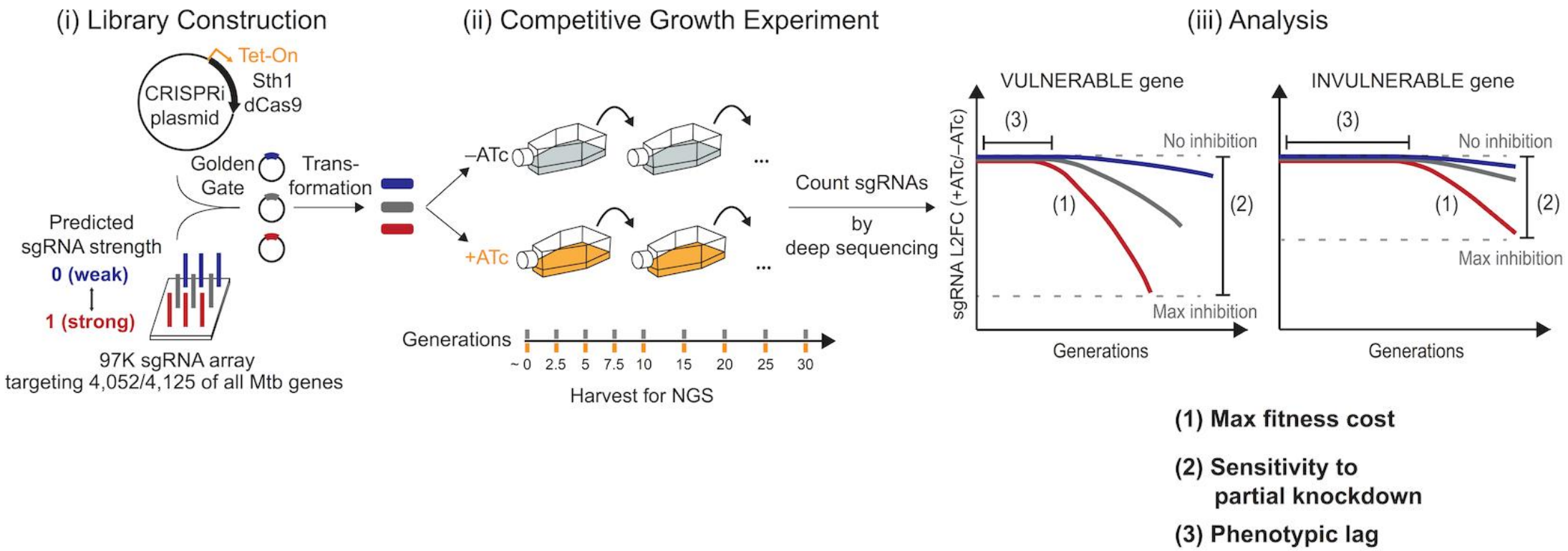
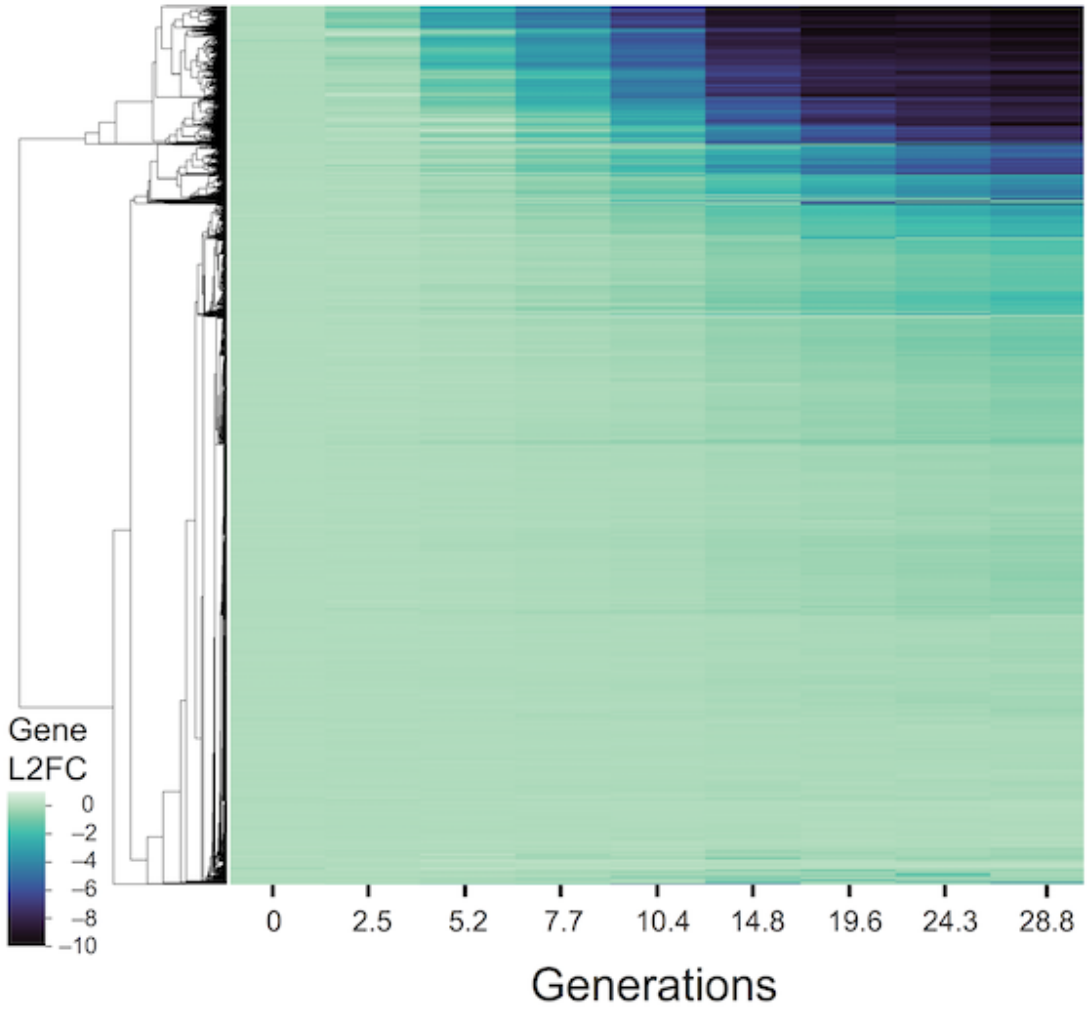
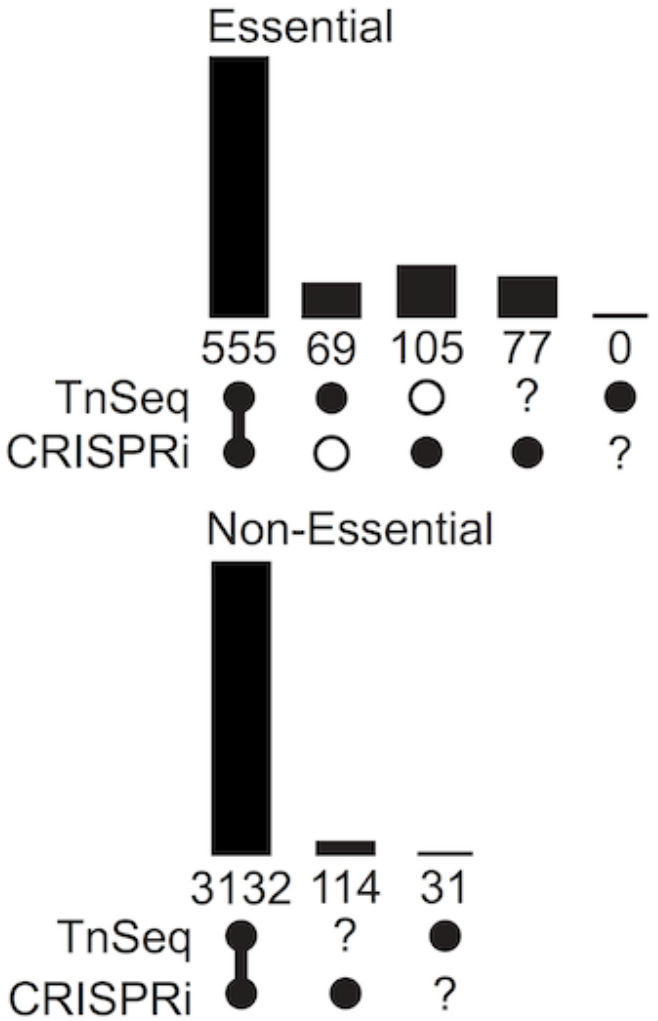
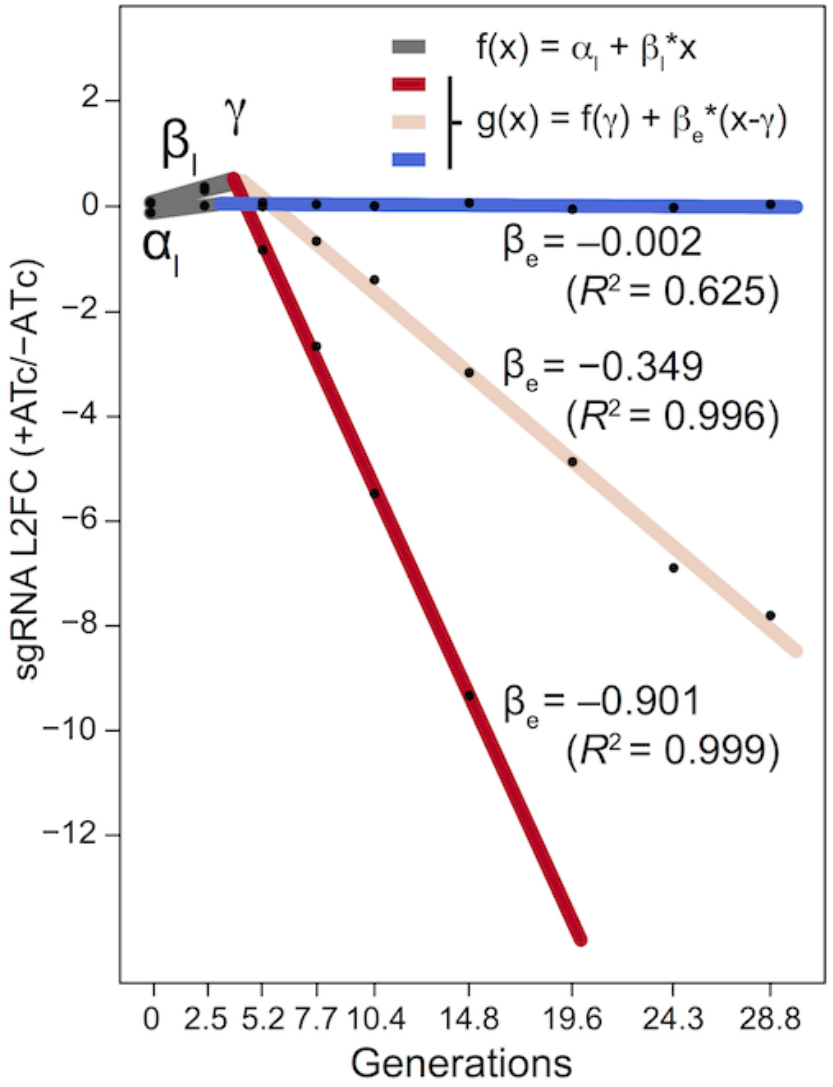


Benchmarking genome-scale CRISPRi to TnSeq

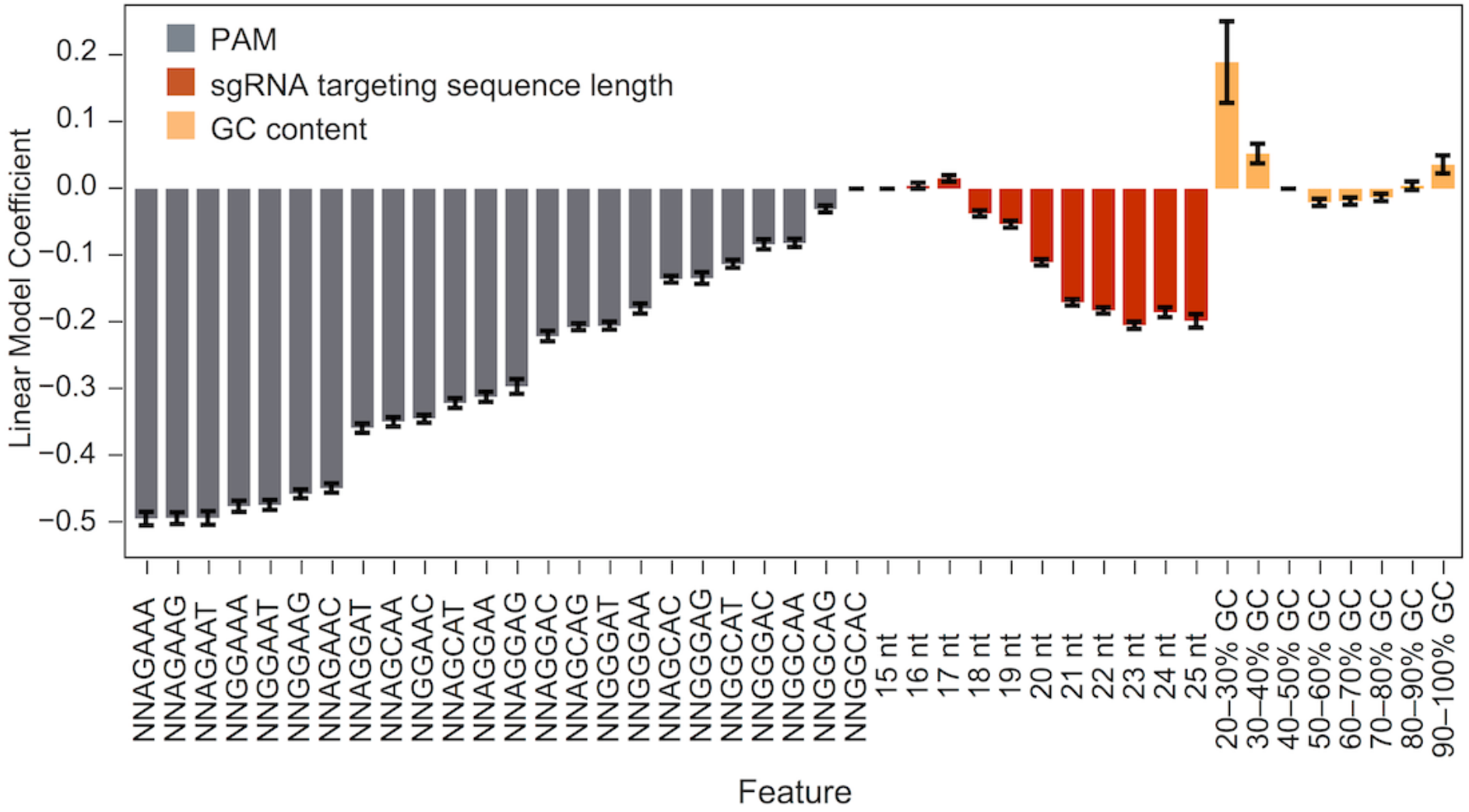




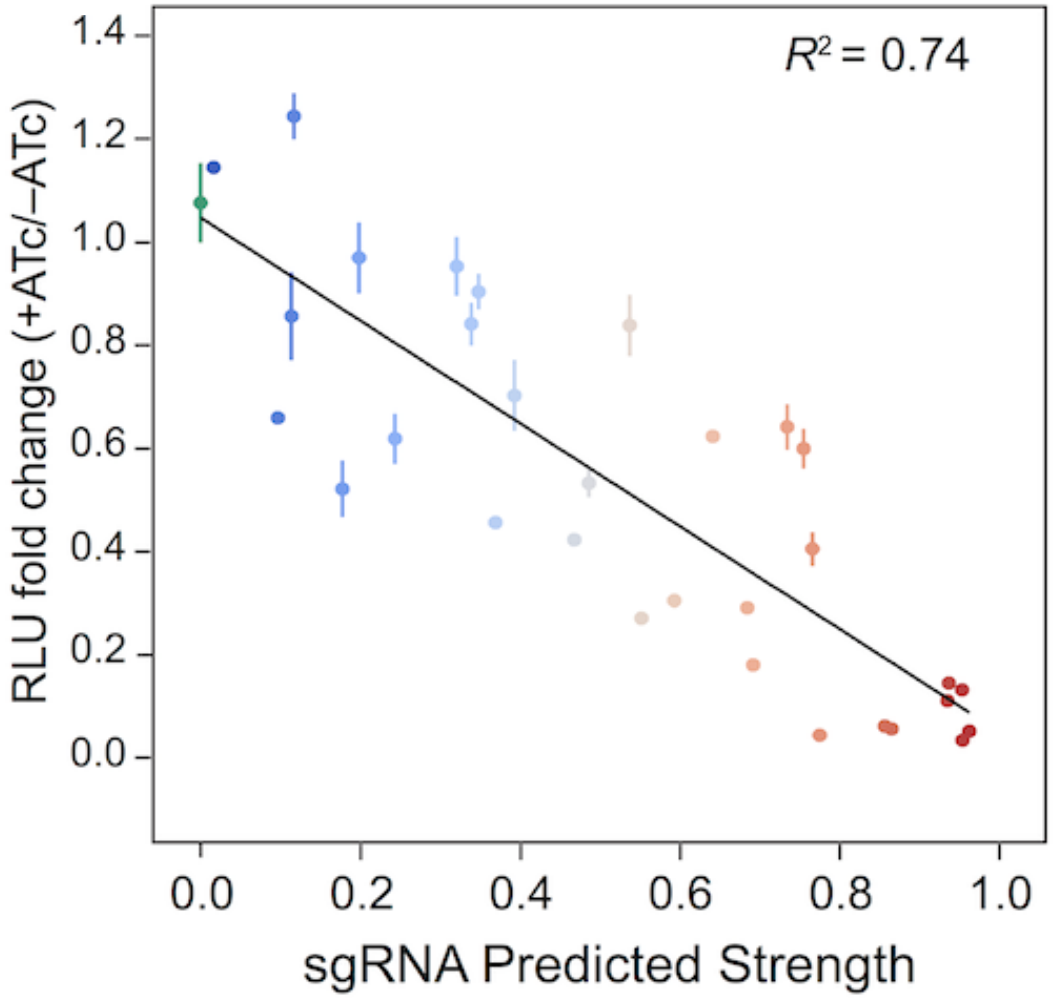
Quantifying sgRNA “strength”



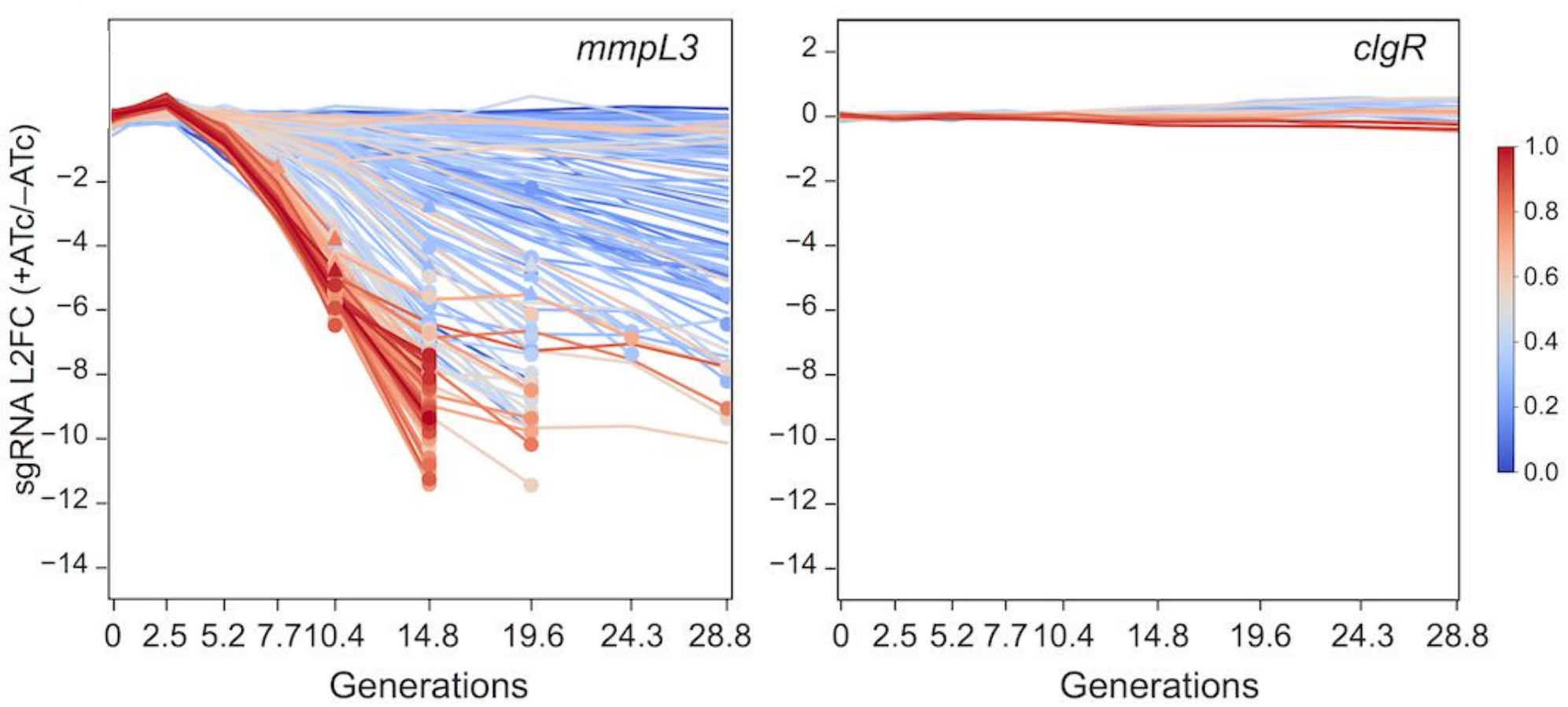
Identification of features that dictate sgRNA strength



sgRNA strength predictions are reasonable



Predictably tunable gene knockdown



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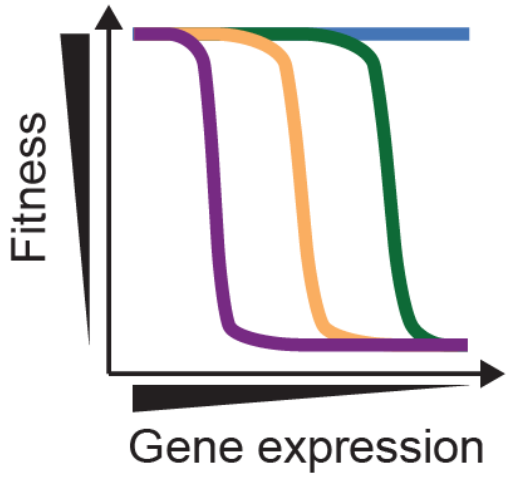

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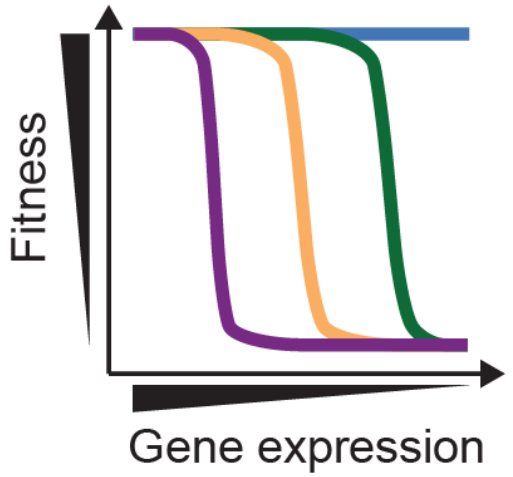

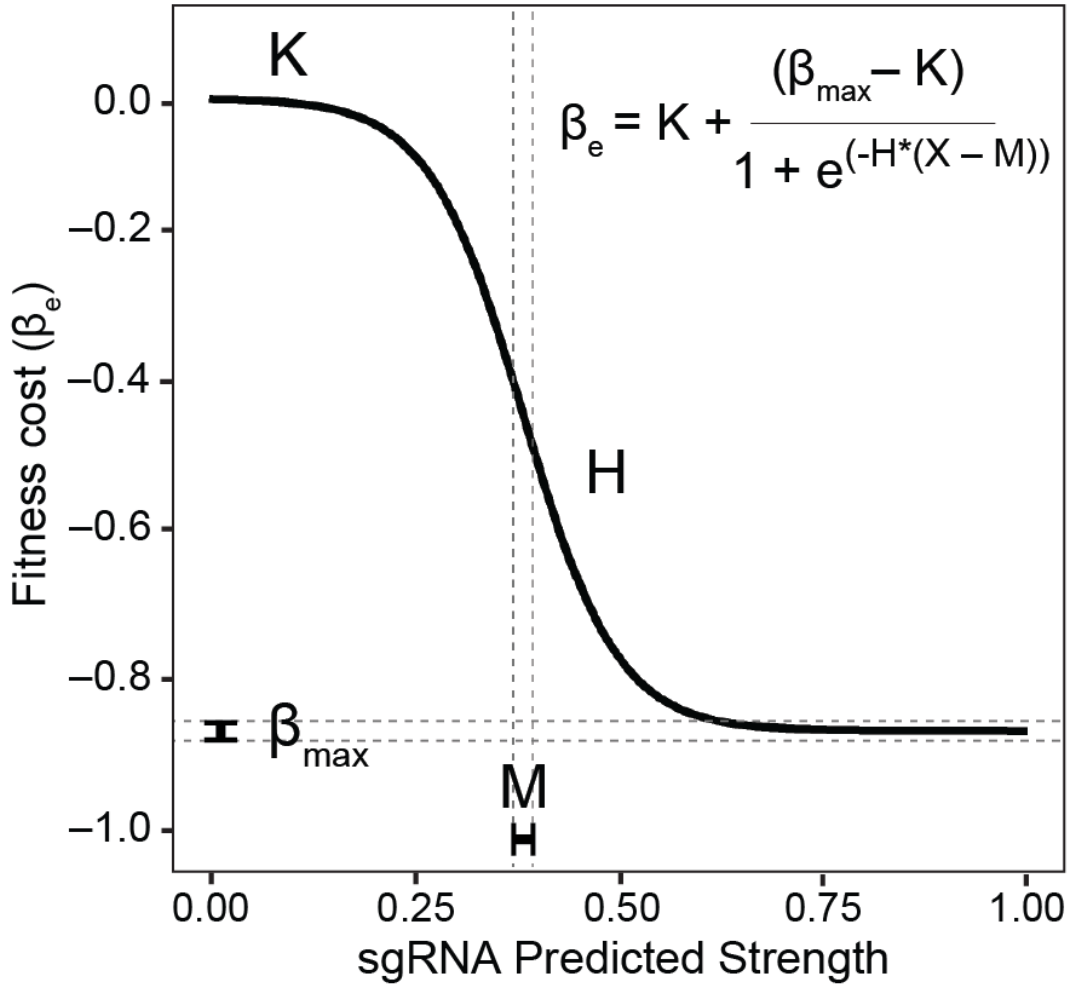

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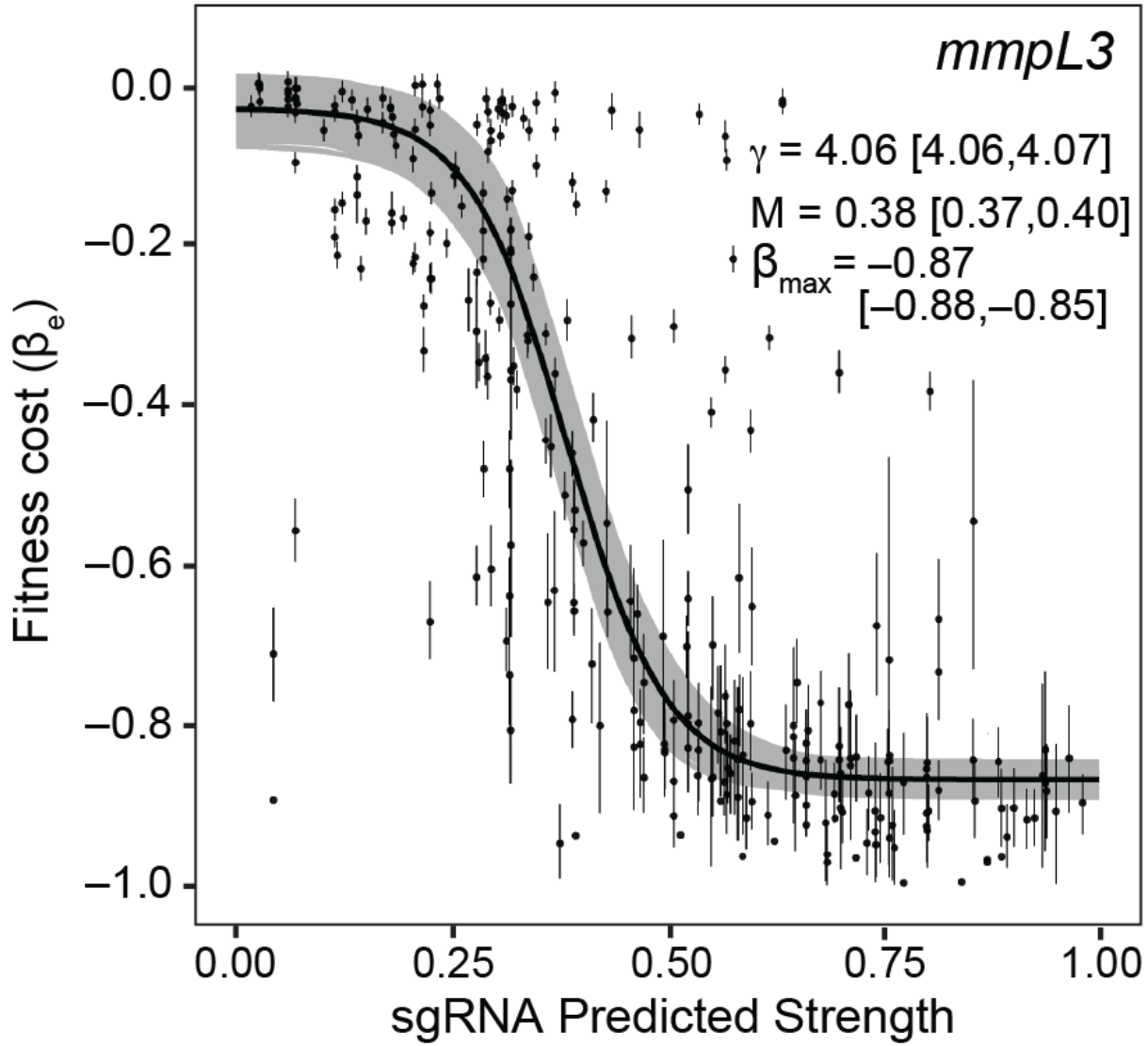
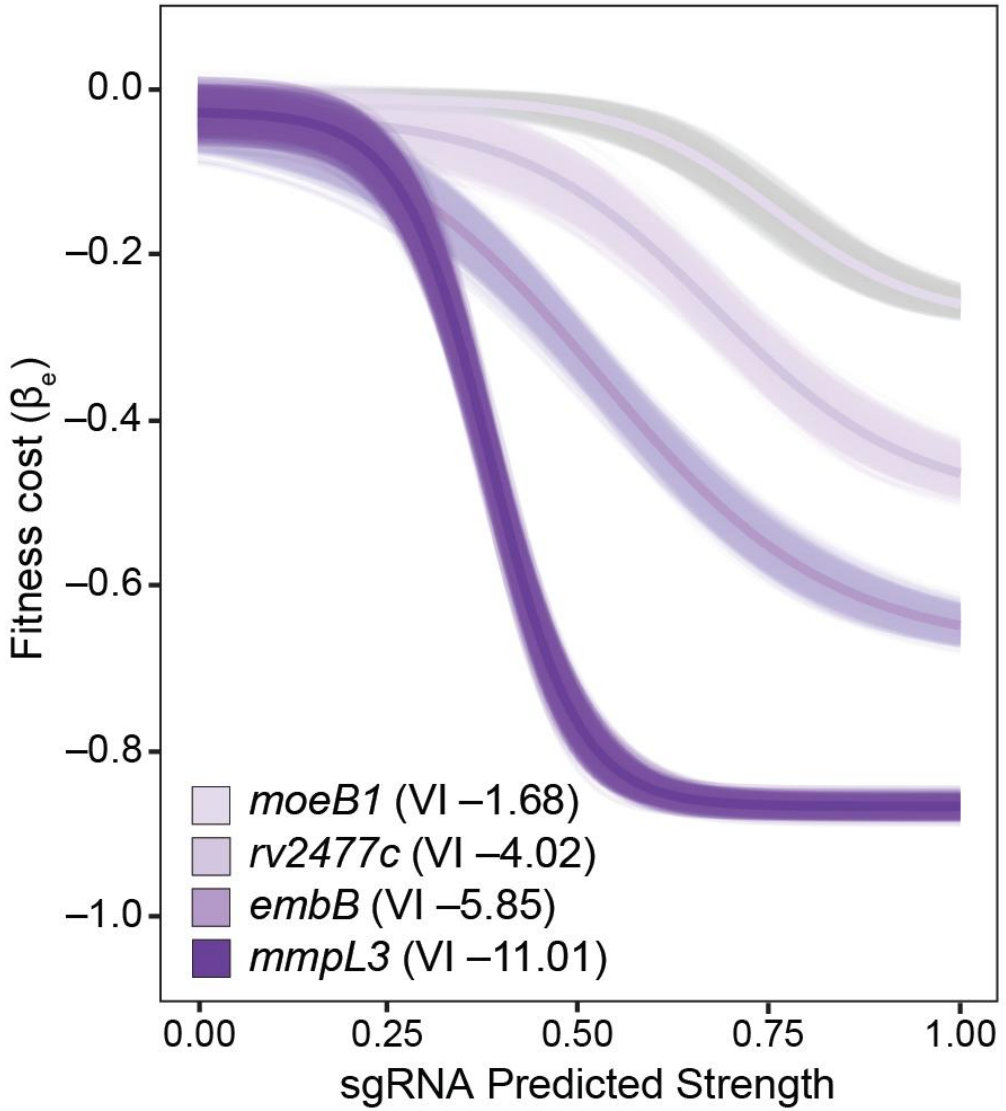
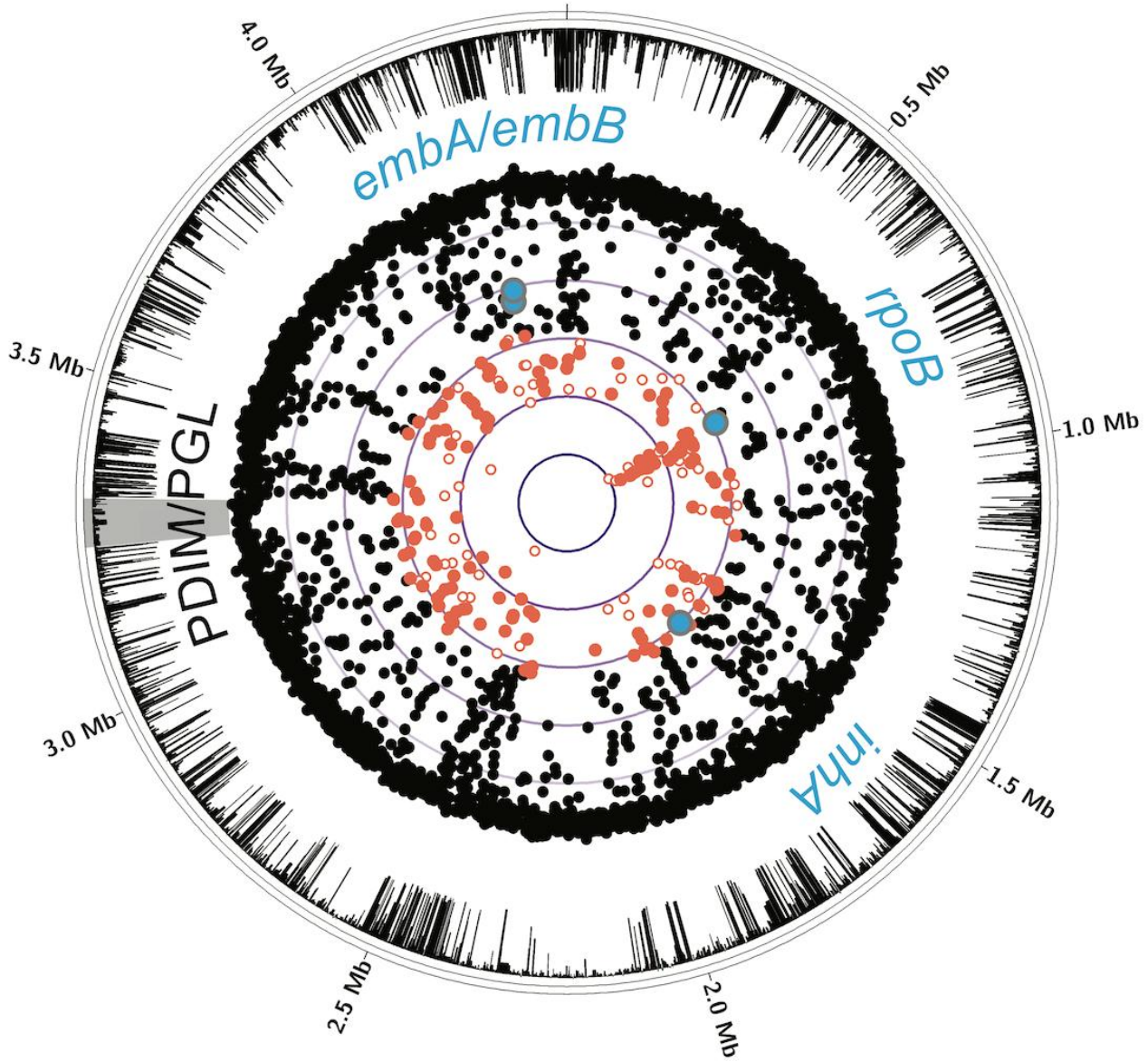


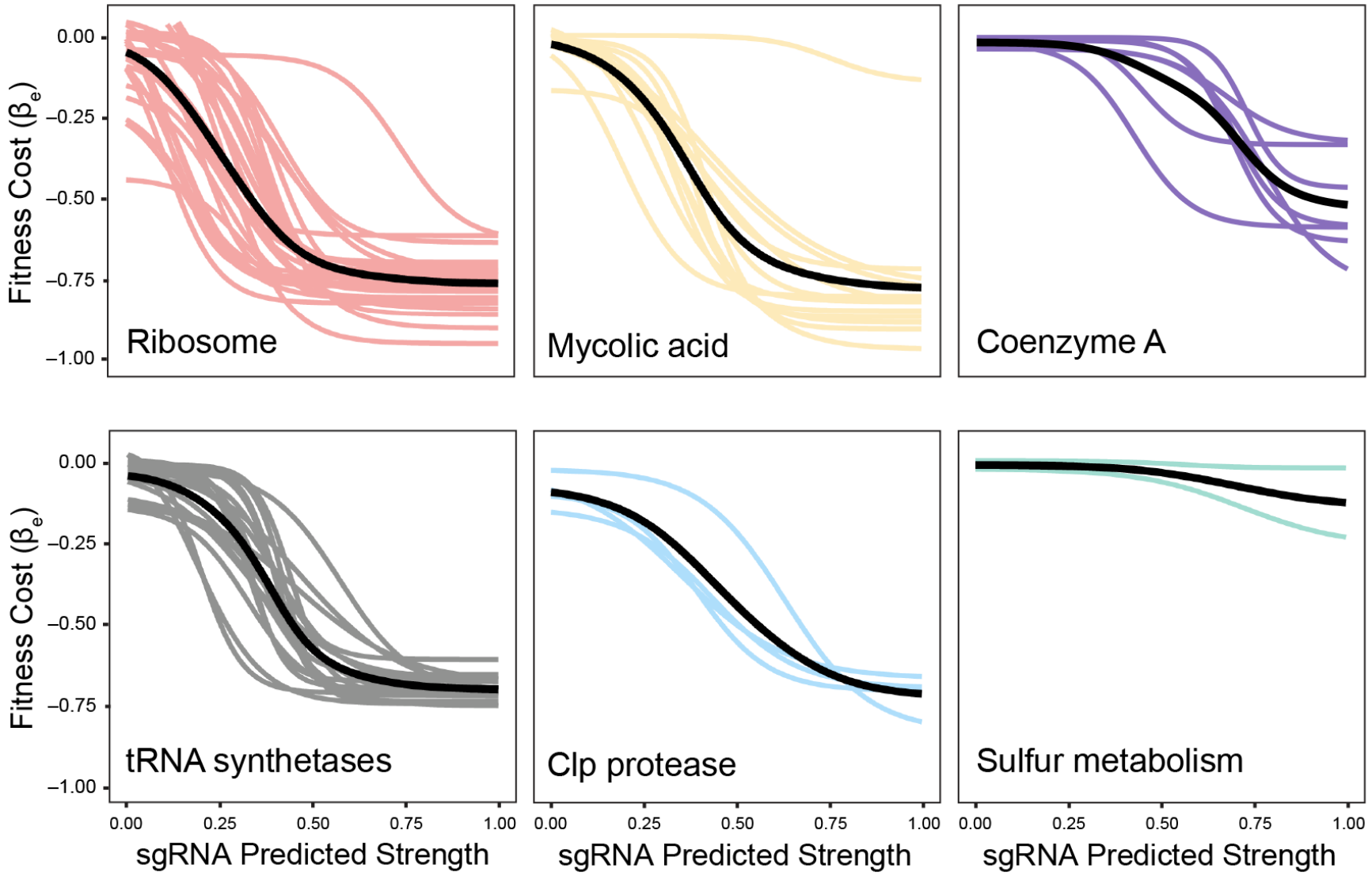
Essential genes have different vulnerabilities



Gene vulnerability varies widely across the Mtb genome



Pathway analysis of vulnerability



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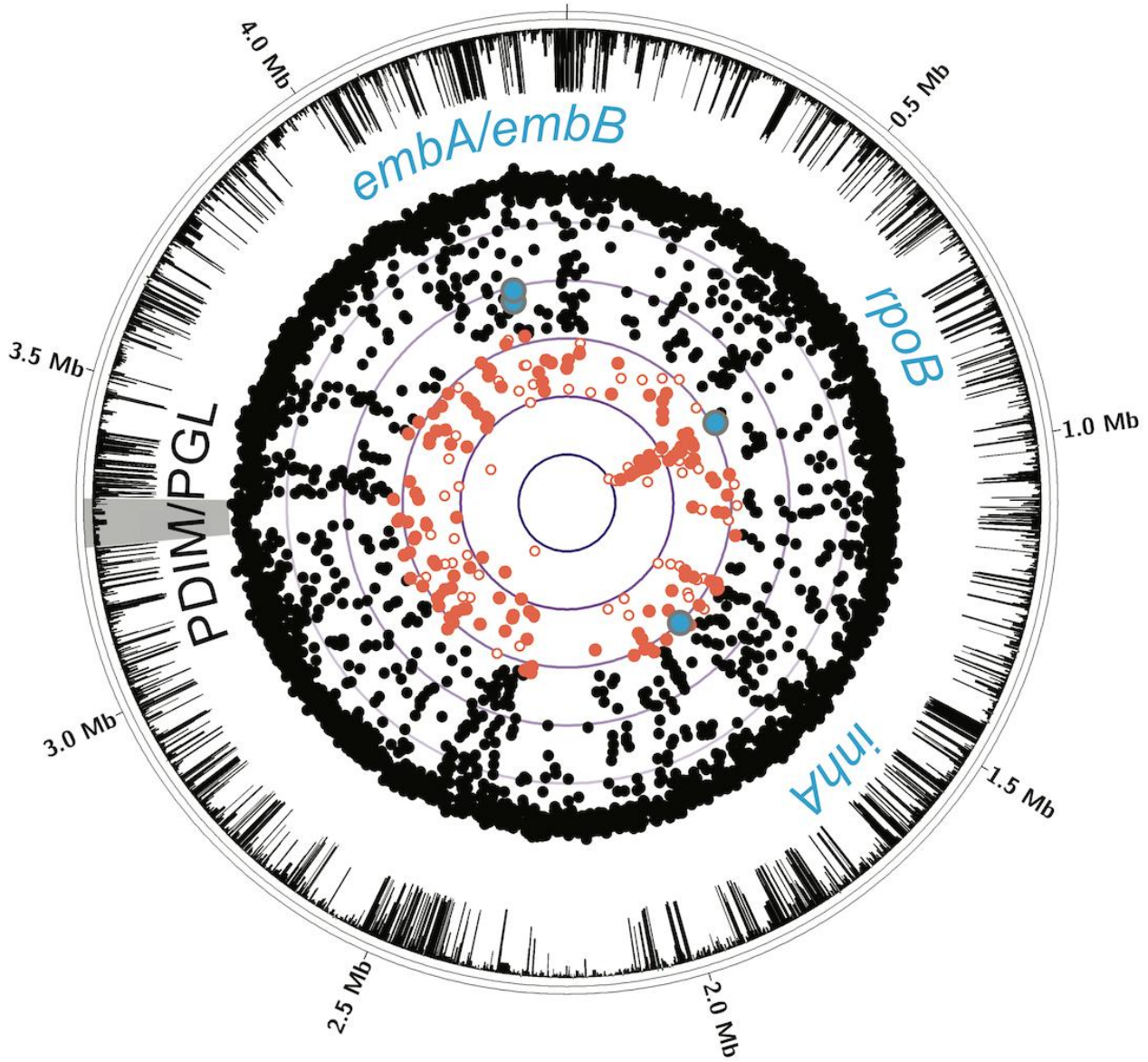
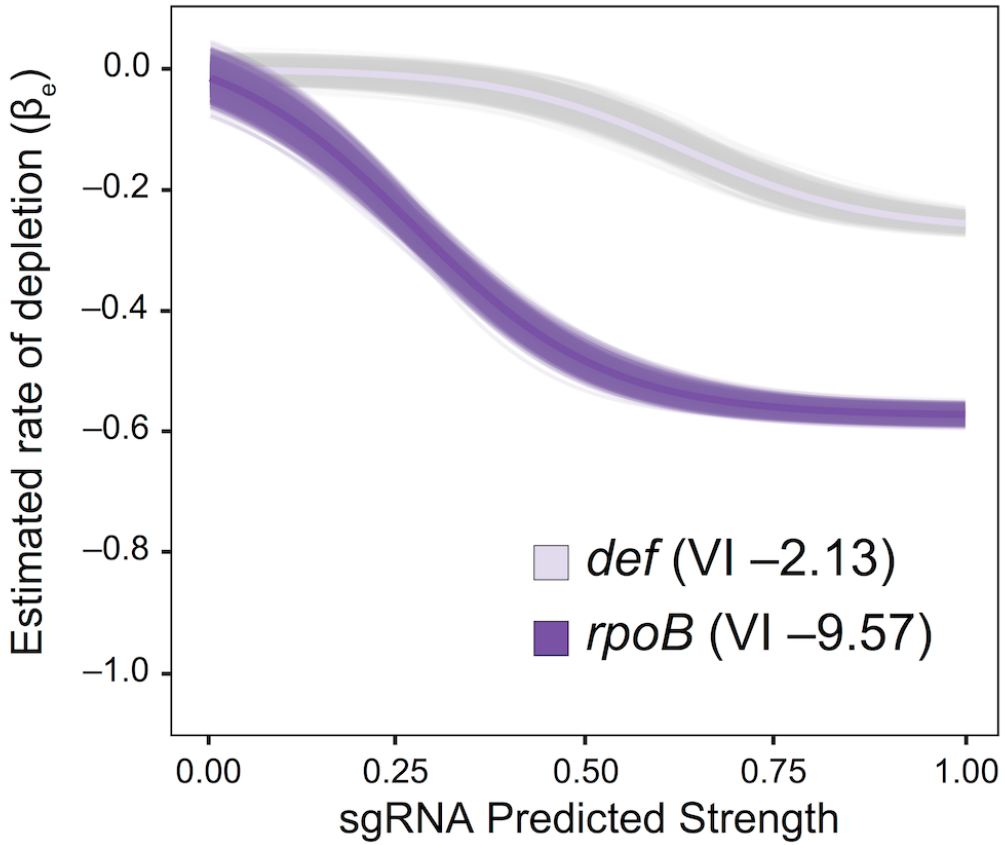
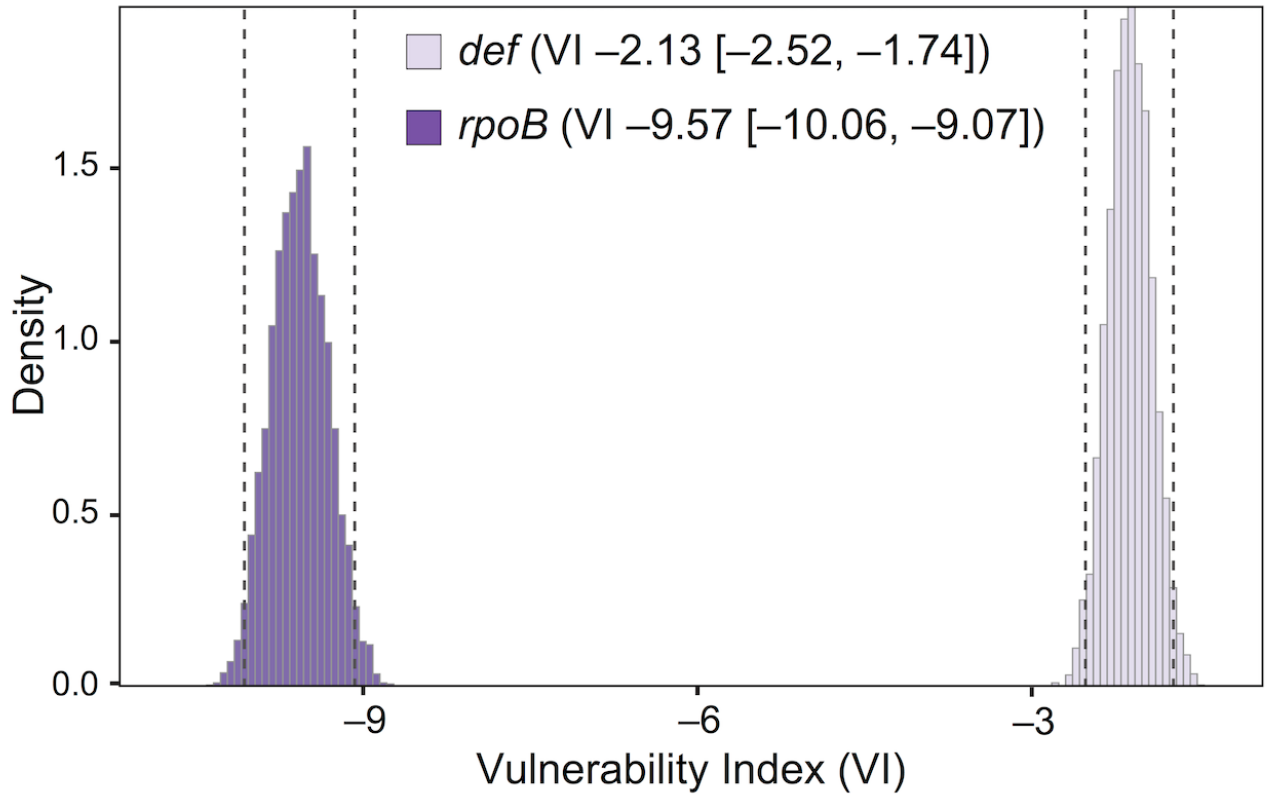


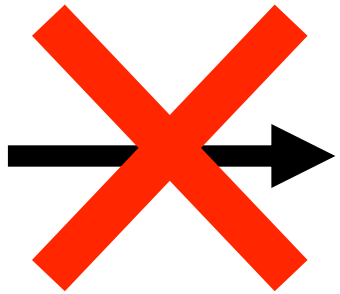
Not all drug targets are made equal





Understand Mtb biology to build better therapies

Discover new biology that facilitates Mtb pathogenesis



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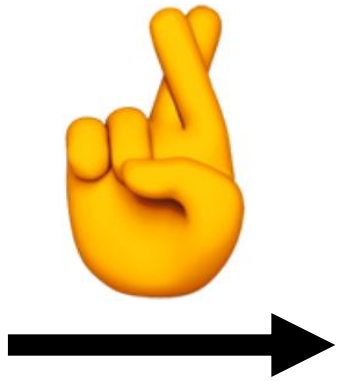


Discover new drugs that perturb that biology and thereby inhibit Mtb pathogenesis

The TB Drug Accelerator

Acknowledgements – Rock Lab

Dirk Schnappinger

Dirk Schnappinger joined Weill Cornell Medical College in 2001, where he currently holds the position of Professor in the Department of Microbiology & Immunology. He received his Ph.D. from the Friedrich-Alexander University of Erlangen-Nürnberg, Germany, in 1998 for work on the repressor controlling tetracycline resistance in Gram negative bacteria. After his graduate work Dr. Schnappinger began to study the human pathogen Mycobacterium tuberculosis (Mtb), first at UC Berkeley, in the lab of Dr. Lee Riley, and then at Stanford under the guidance of Dr. Gary Schoolnik, where he helped to adapt microarray-based RNA profiling to the analysis of bacterial pathogens.

His current research aims to help develop new medicines for the treatment and prevention of Tuberculosis (TB), an infectious disease that still claims over a million lives each year. This work began with developing a regulatory system that allows to turn Mtb genes on and off, both in vitro and during infections. His lab now applies this and other genetic approaches to evaluate Mtb gene products as new targets for TB drug development by documenting the impact of their genetic inactivation on growth and persistence of Mtb in vitro and in mice, help elucidate the mechanisms by which small molecules inhibit the growth of Mtb, improve safety of the M. bovis BCG vaccine and develop a human challenge model for TB.

https://www.ehrtschnappingerlabs.org/

The application of chemical-genetic tools in TB drug discovery

Dirk Schnappinger, PhD

Department of Microbiology and Immunology

Weill Cornell Medicine

New York, USA

Applications

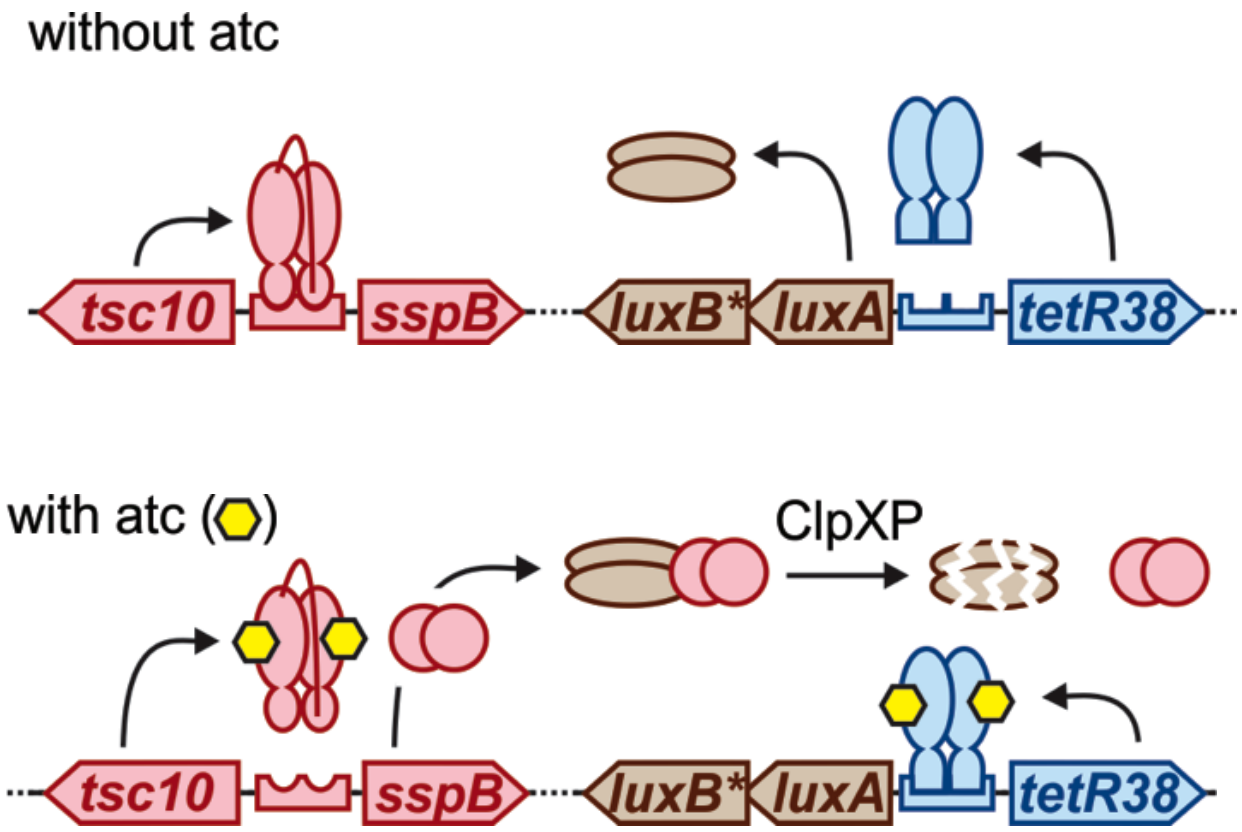
1. Validation of Mtb targets in mice
2. Mechanistic analysis of hit and lead compounds
3. Discovery of new chemical matter: target-directed whole cells screens

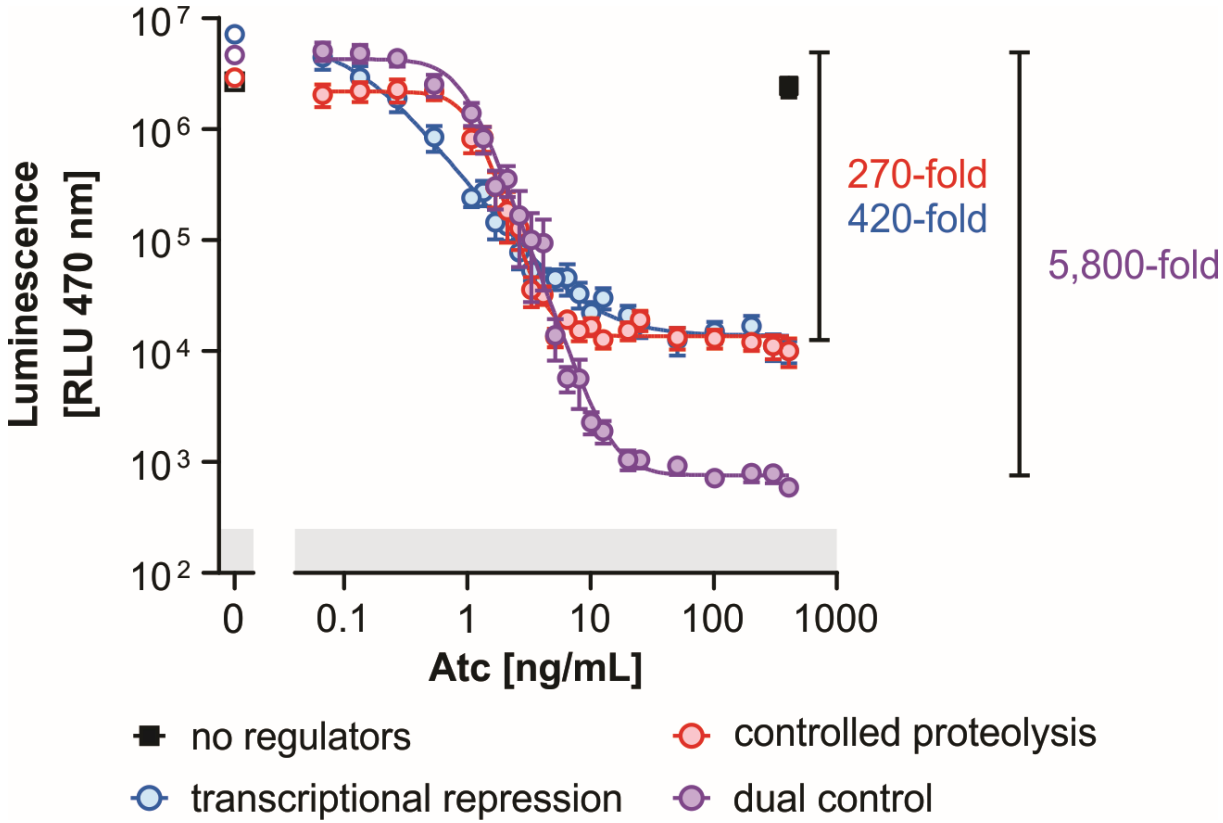
Why conditional gene silencing?

- Most antibiotics target in vitro essential gene products/processes, which are difficult to study using traditional genetic approaches and screens (such as transposon-based methods).
- Successful treatment of an infection requires targeting gene products/processes required by a pathogen to maintain an infection.
- Conventional mutants (transposon mutants, deletion mutants) often only allow to assess the importance of a gene product for establishing an infection.

Goal: Develop a conditional gene silencing system that allows to efficiently suppress gene activity in vitro and during infection.

Dual-control (DUC) system

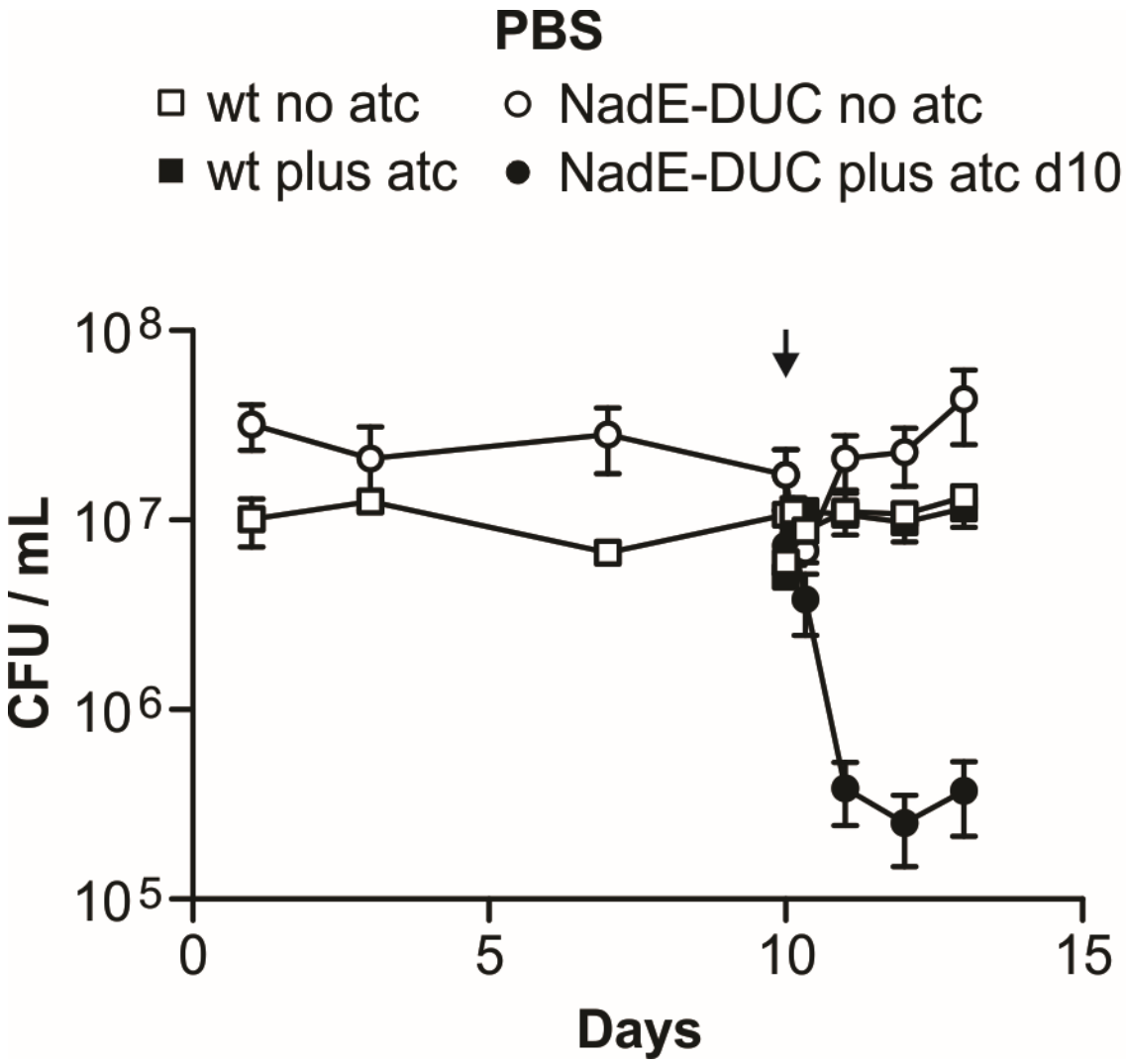


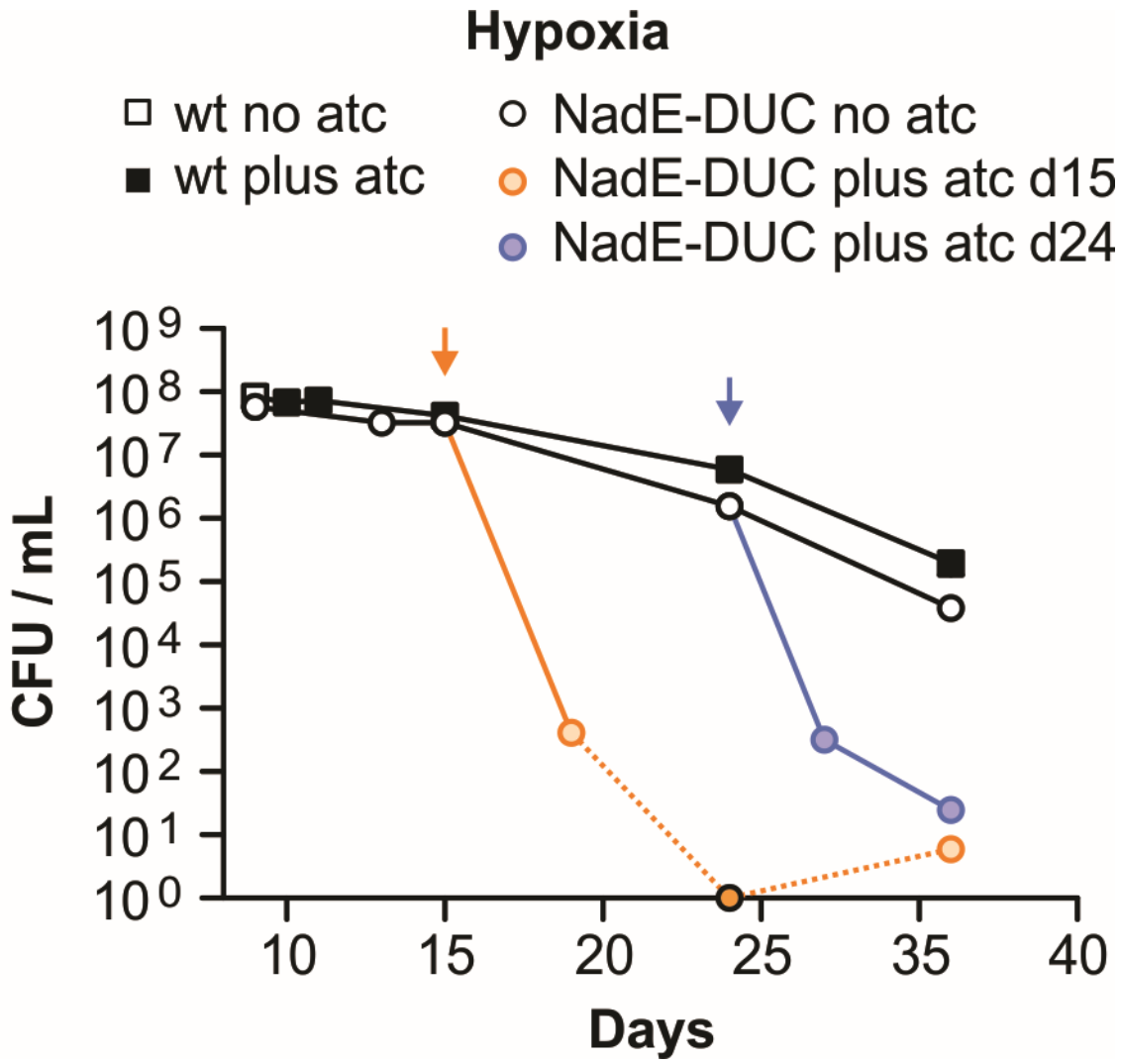


Jee-Hyun Kim, Kathryn O'Brien et al. PMID: 24191058

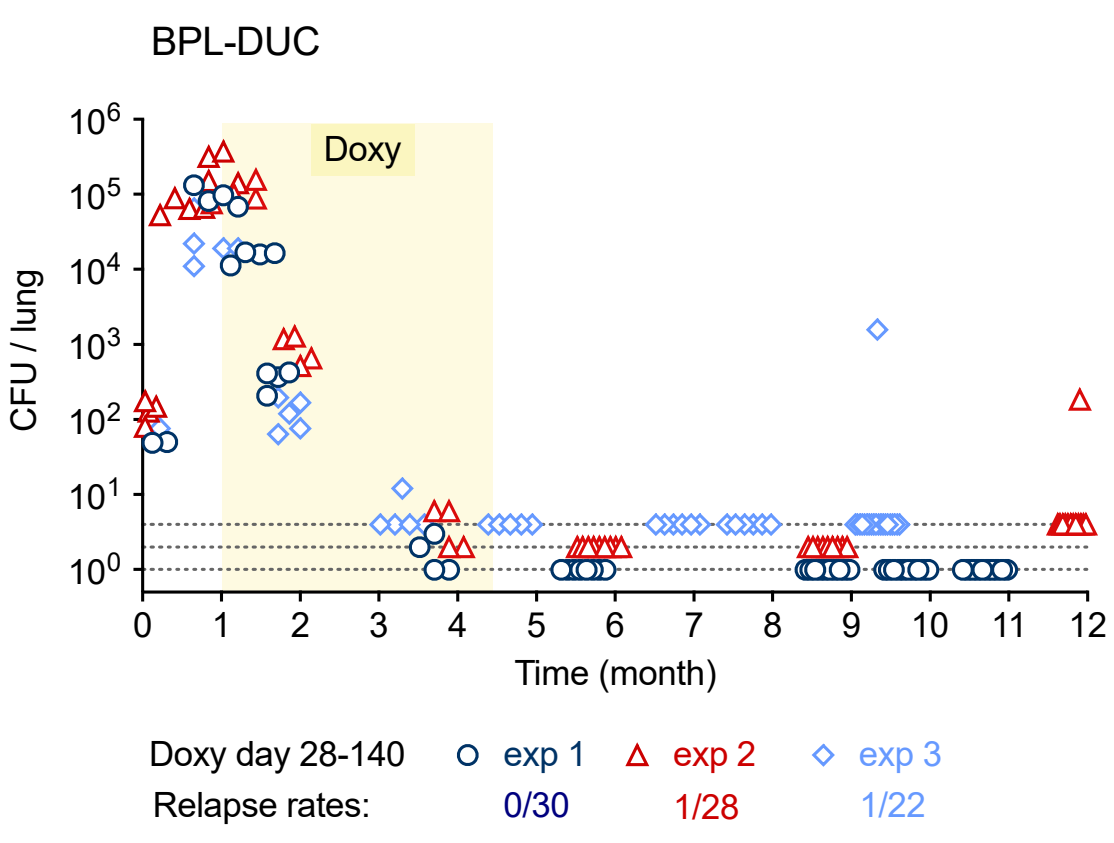
Impact of interfering with NAD synthesis

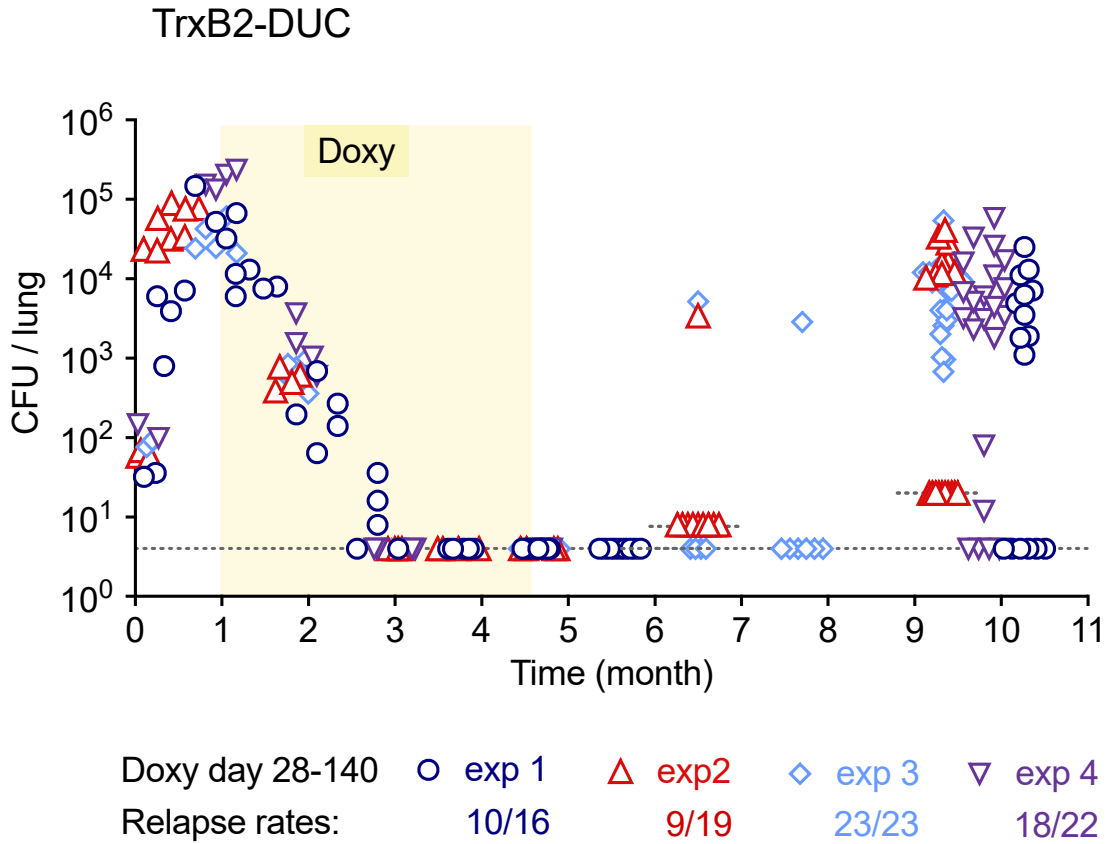
Impact of interfering with NAD synthesis





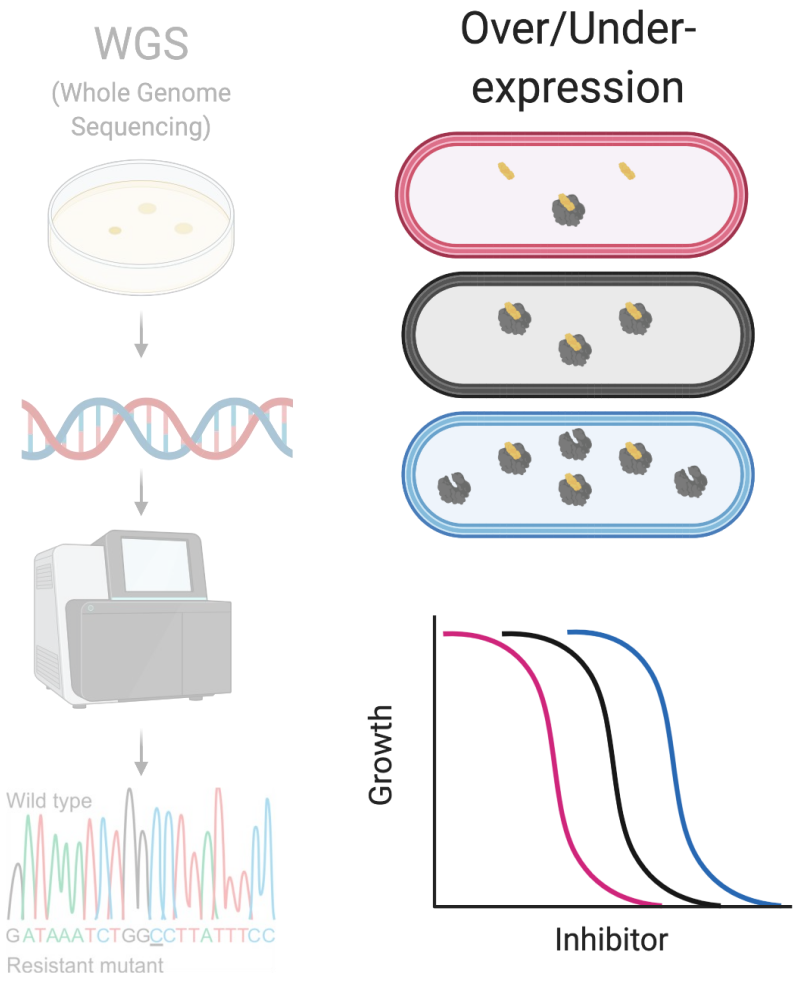
Identification of sterilizing targets

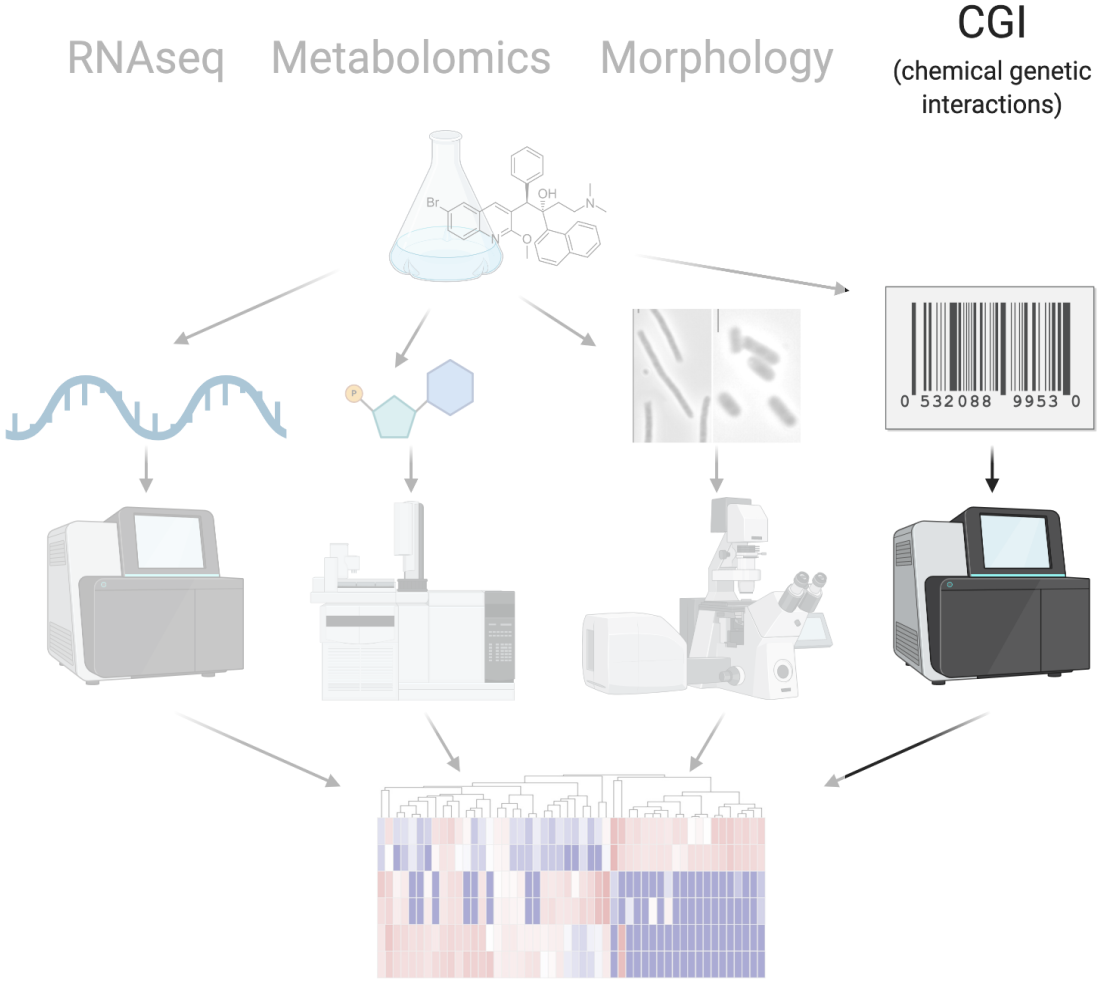




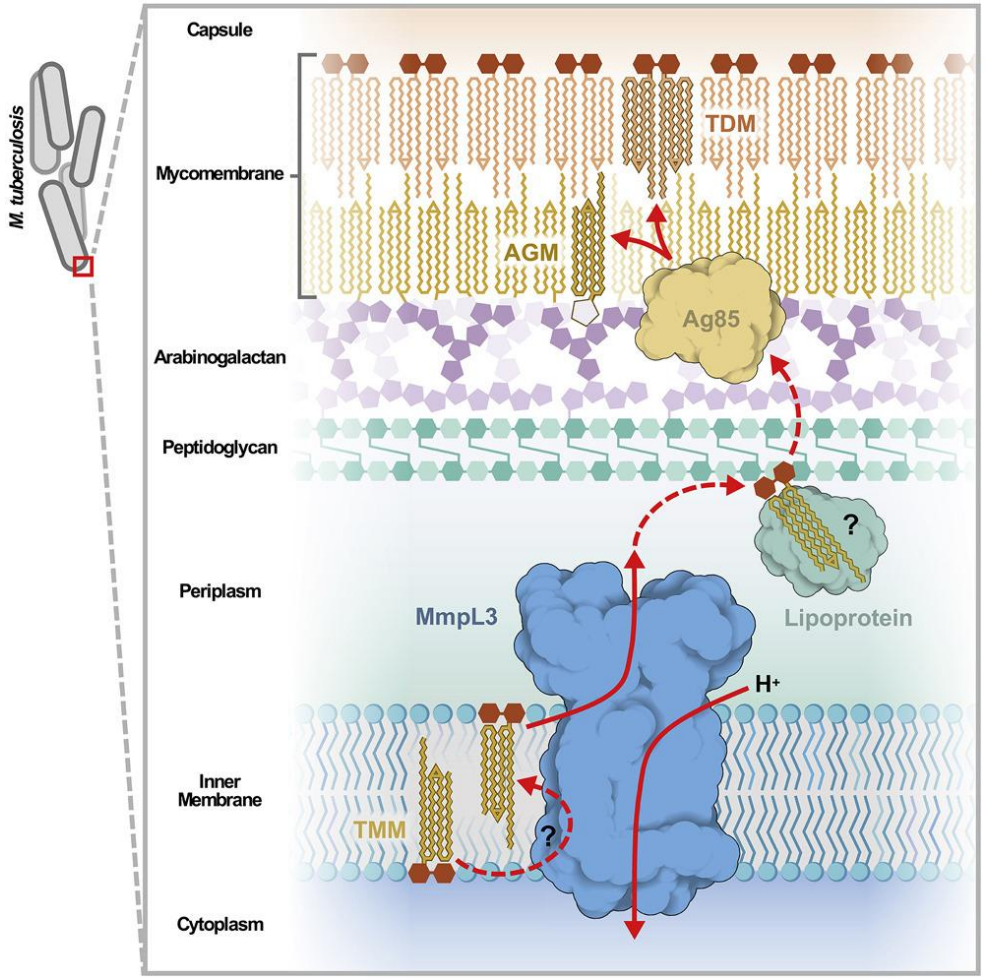
Su et al. PMID: 34269789

Mechanistic analyses of hits and leads



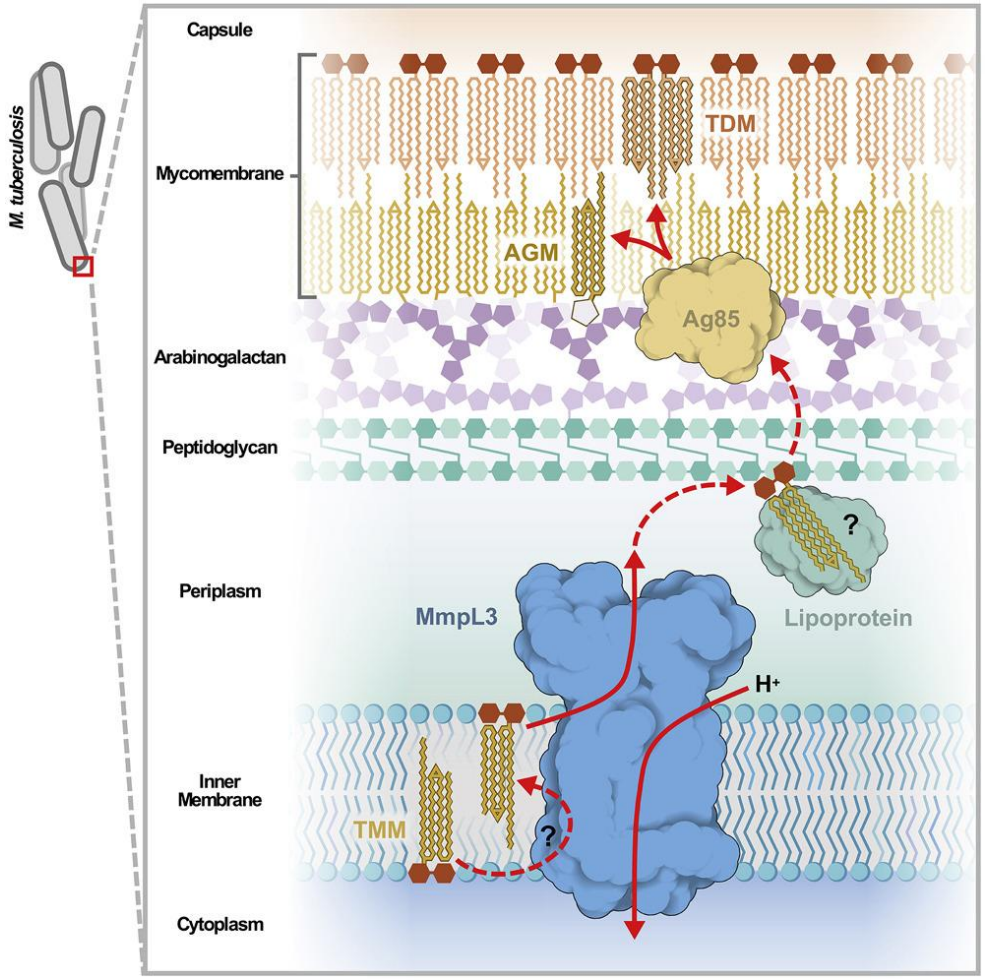


MmpL3: TMM transporter

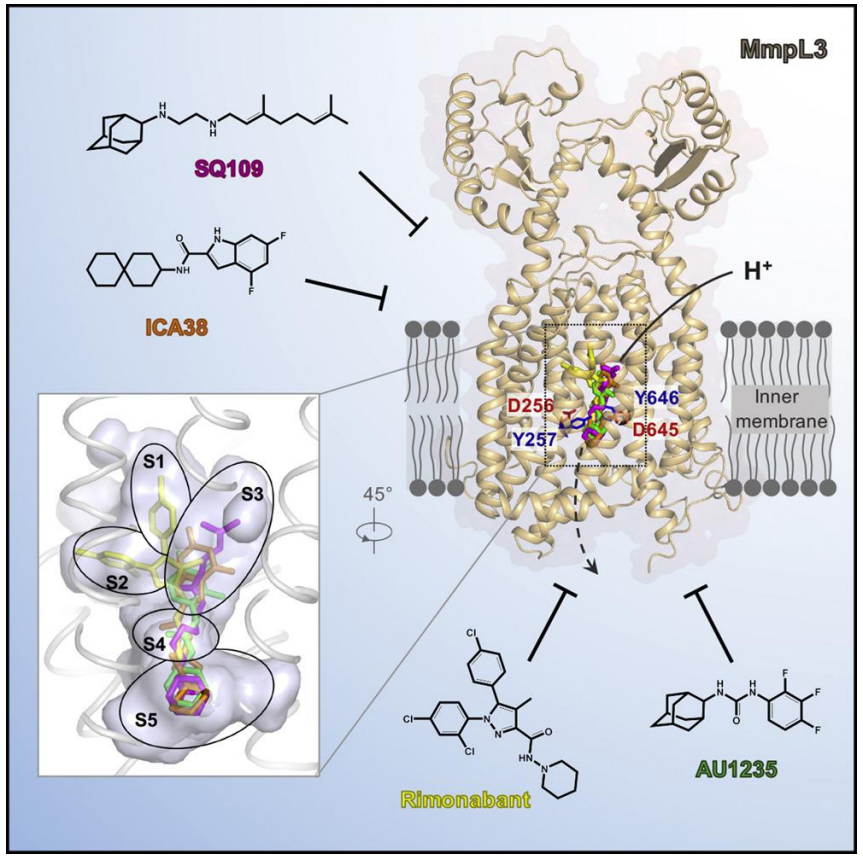


Adams et al, PMID 34242558

MmpL3: frequent target in phenotypic screens

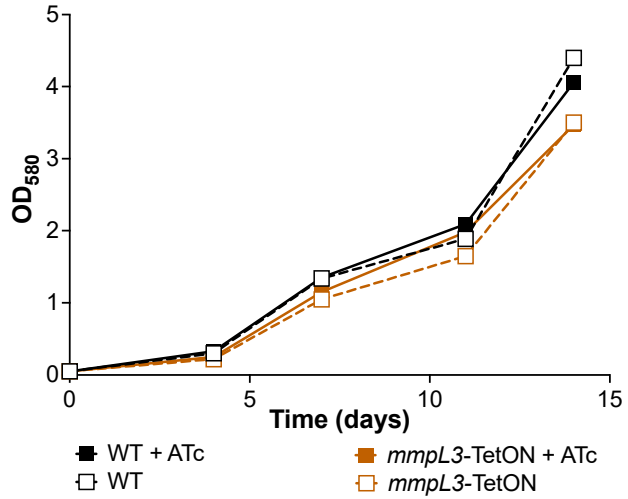


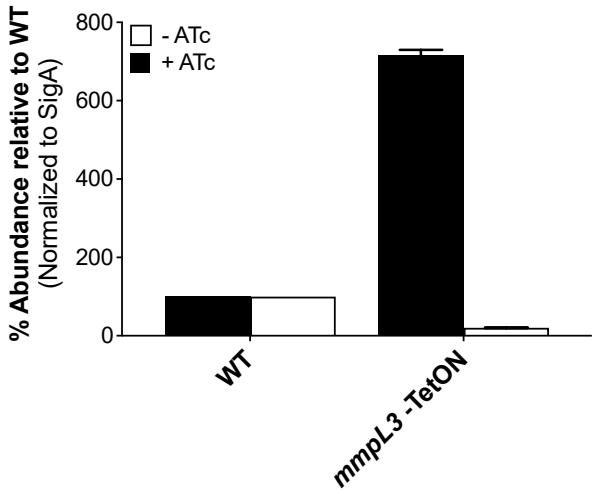
Adams et al, PMID 34242558

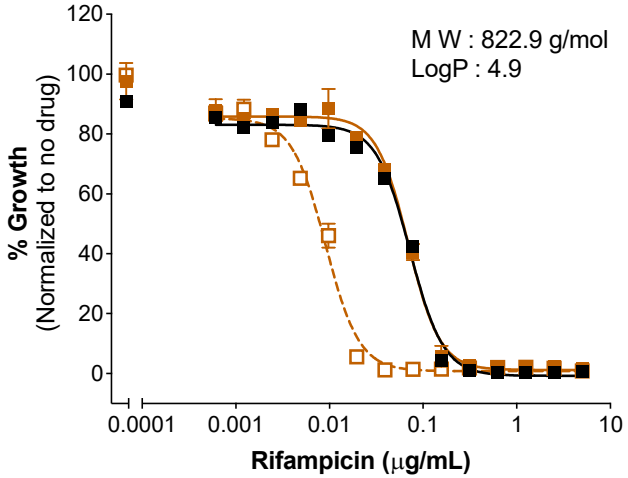


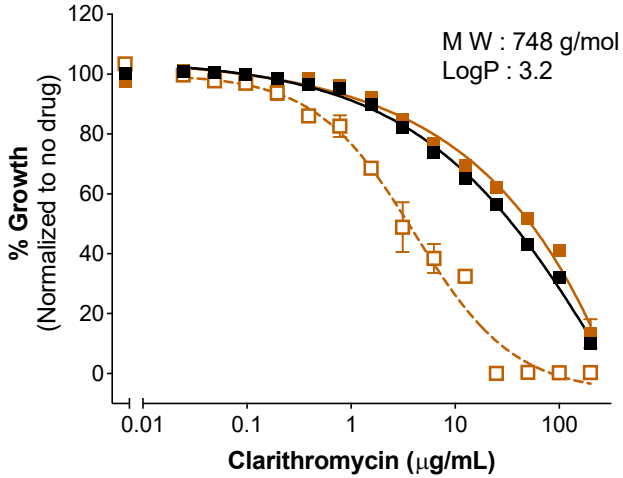
Zhang et al, PMID 30682372

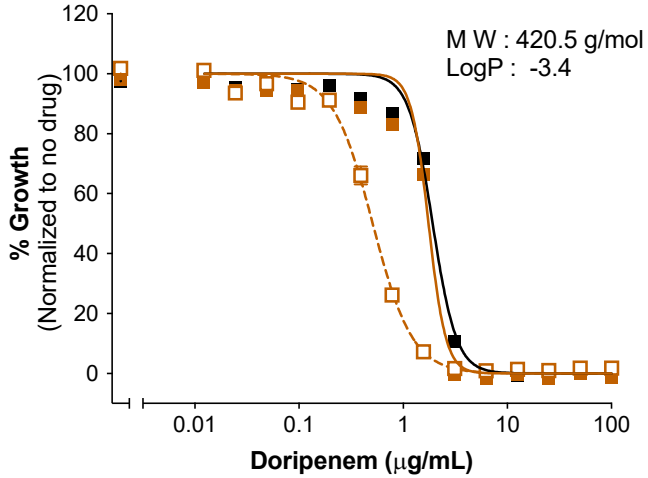
Two-way regulation of *mmpL3*



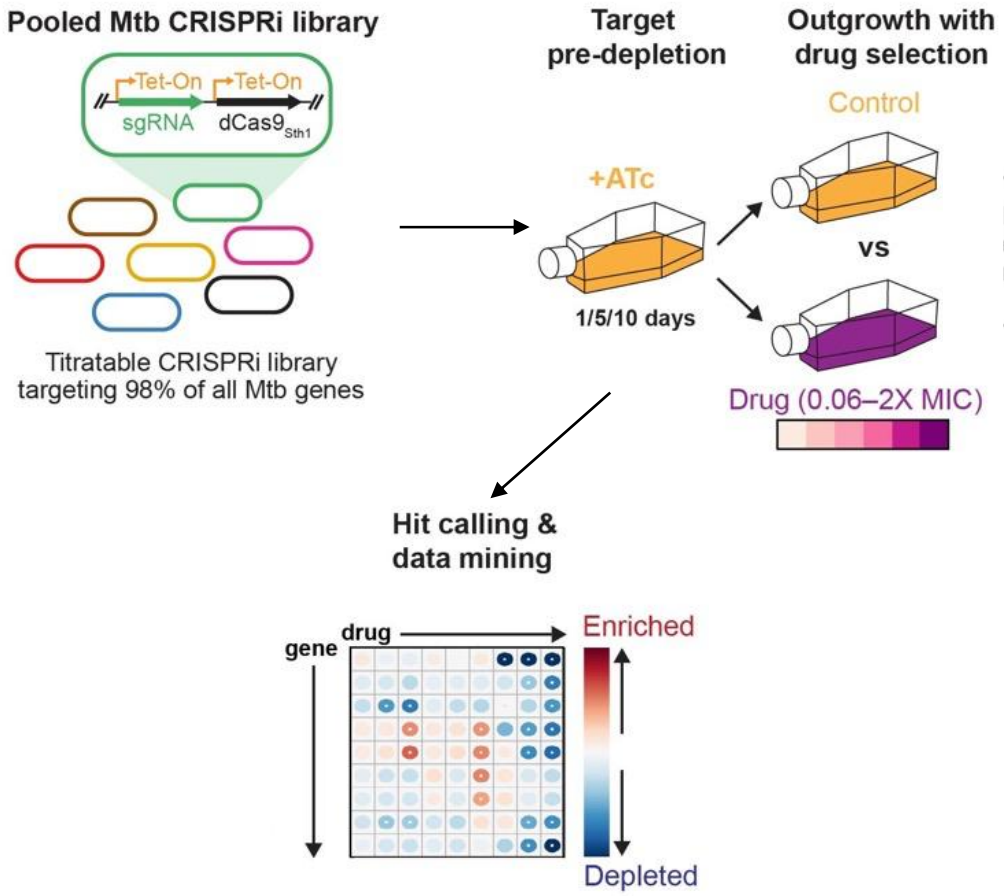


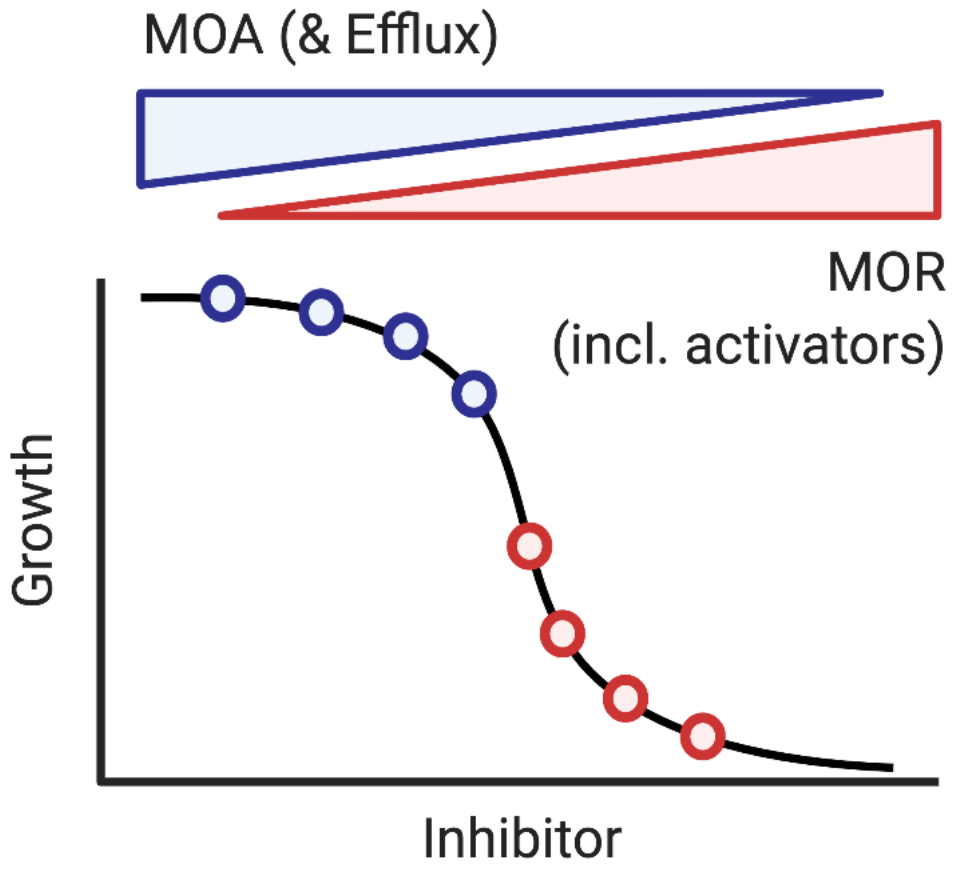






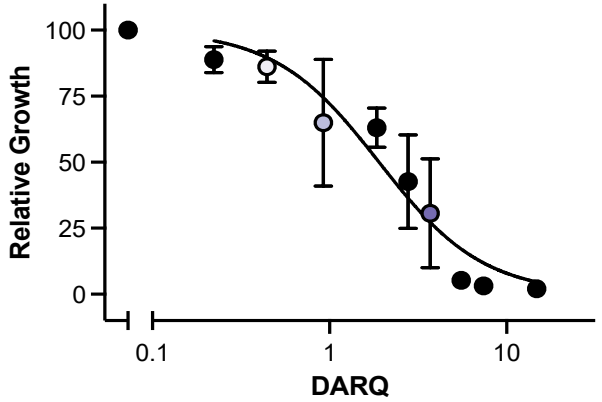
Chemical genomics

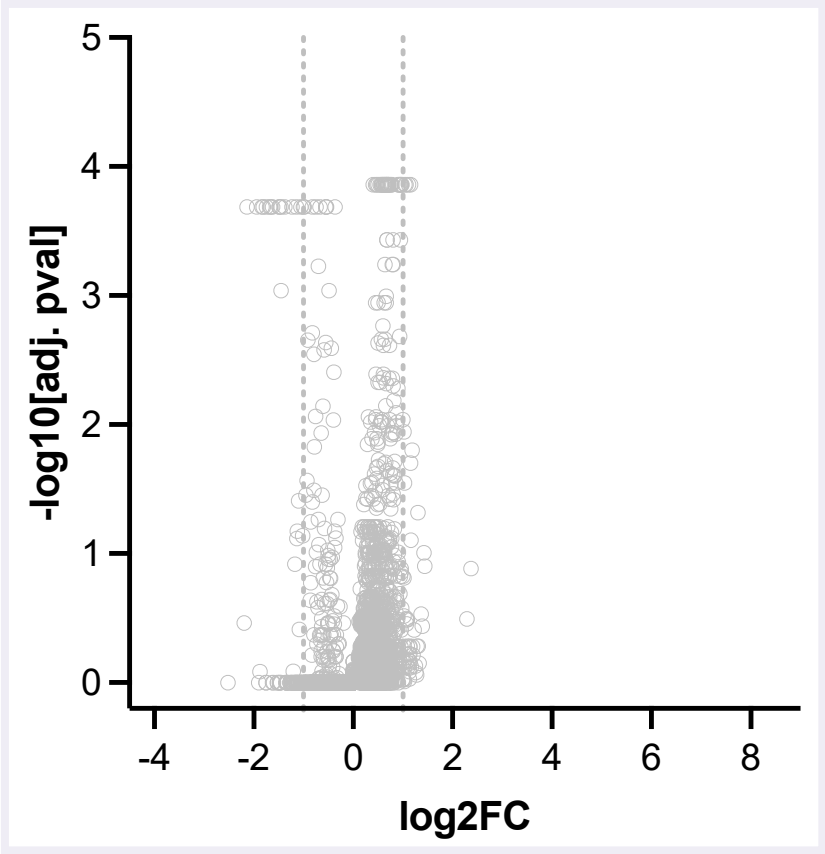


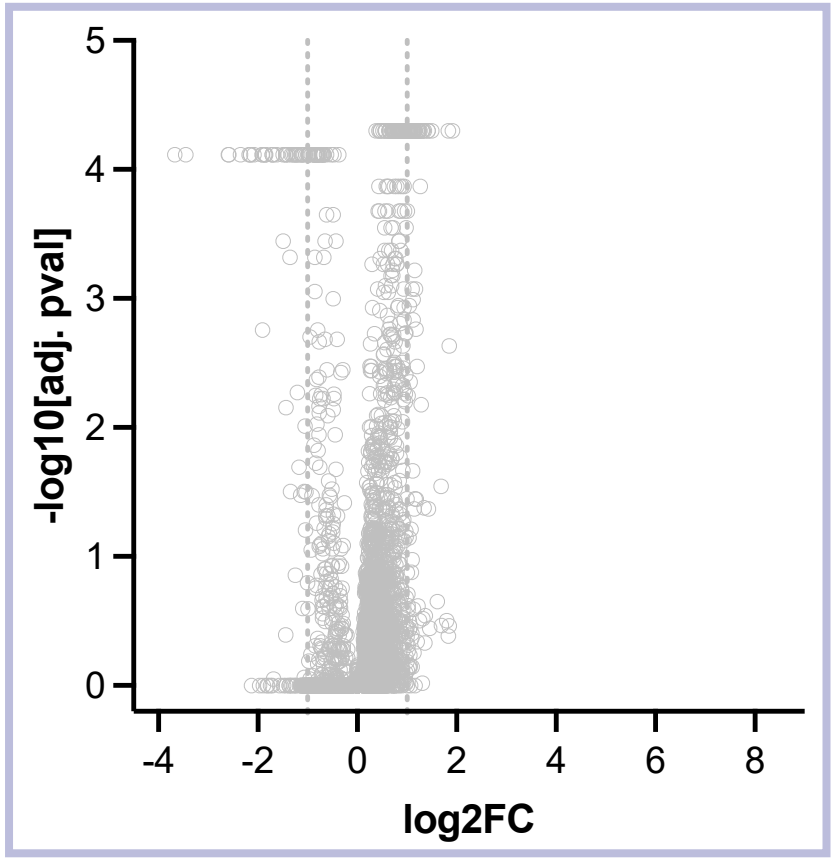


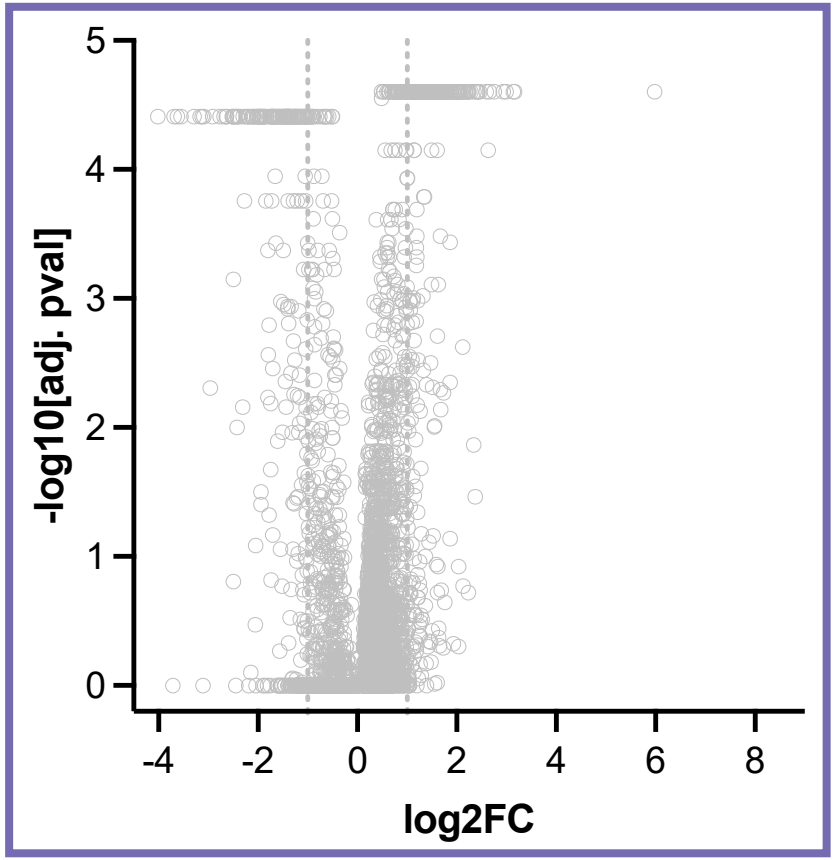
Li, Poulton et al. PMID 35637331

Chemical genomics



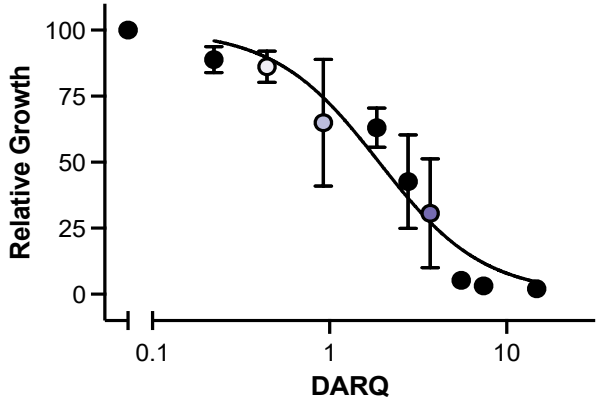


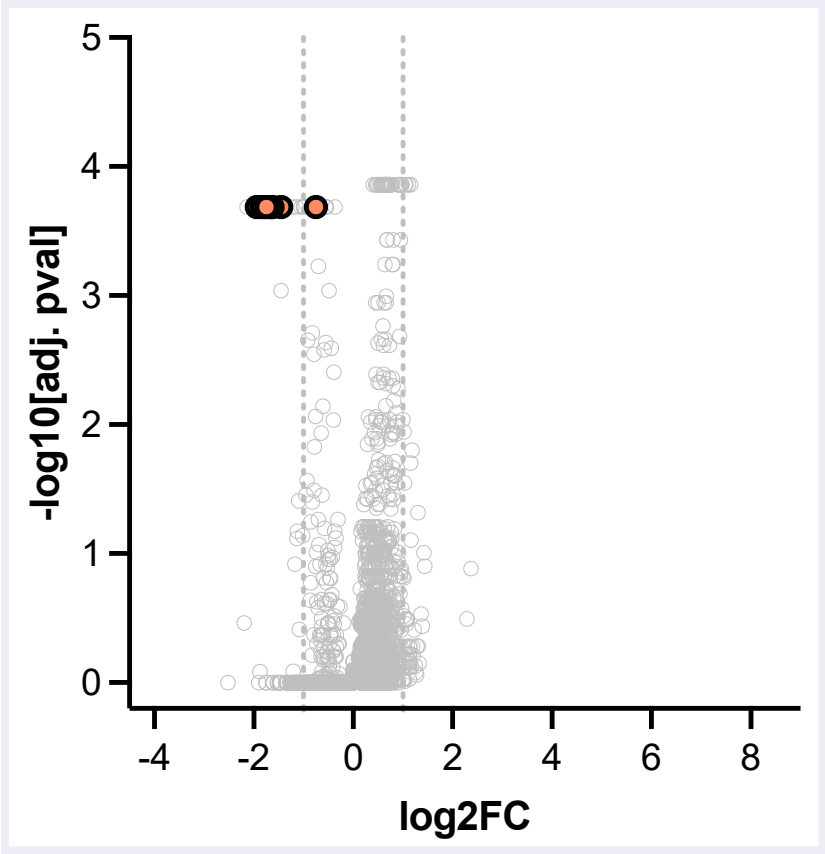


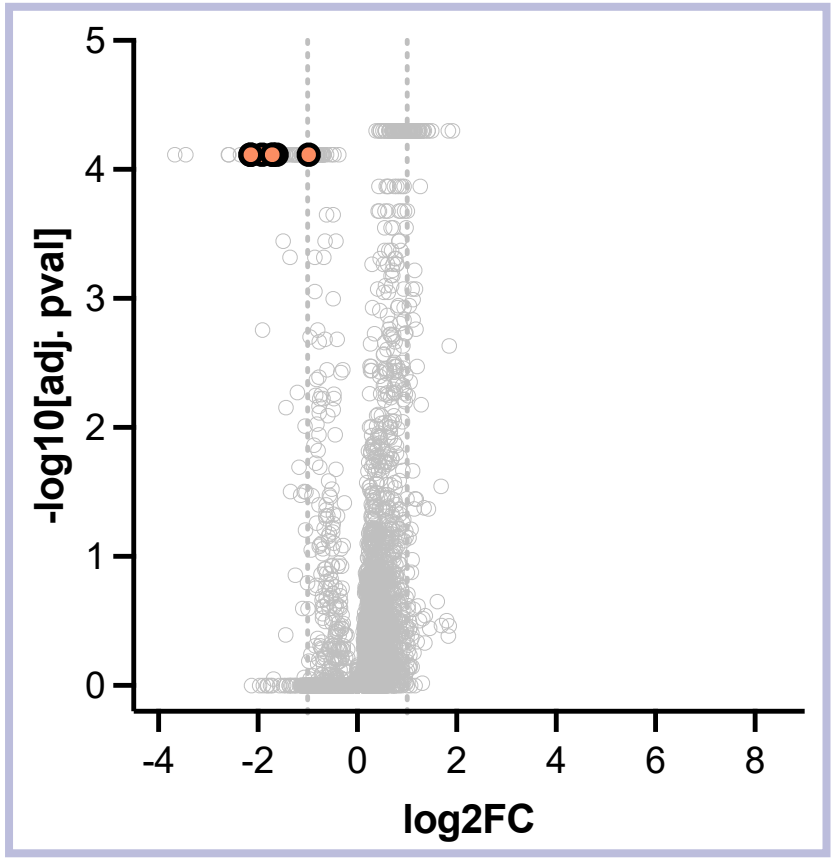


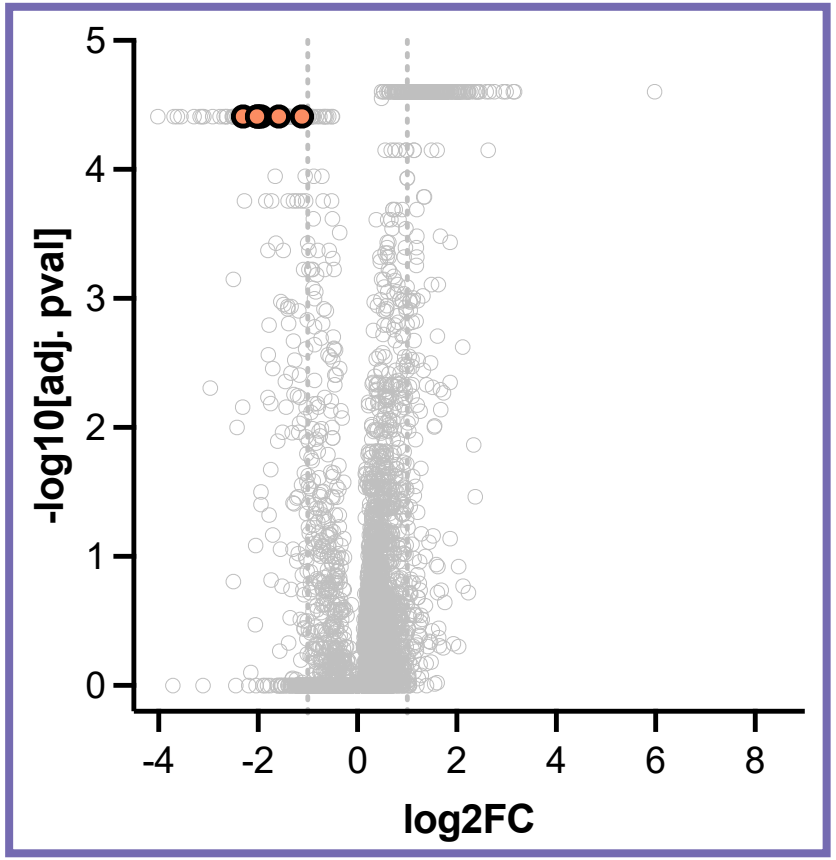
Chemical genomics

- ATP synthase

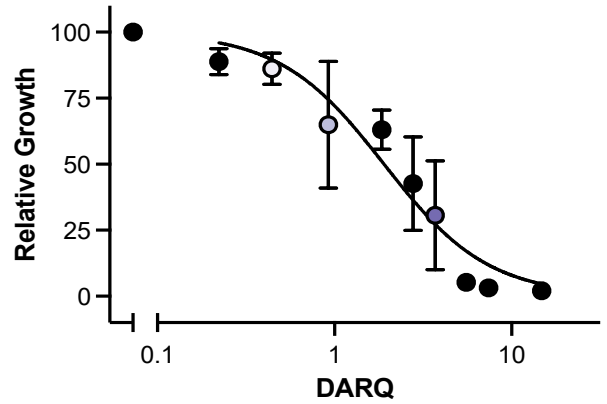




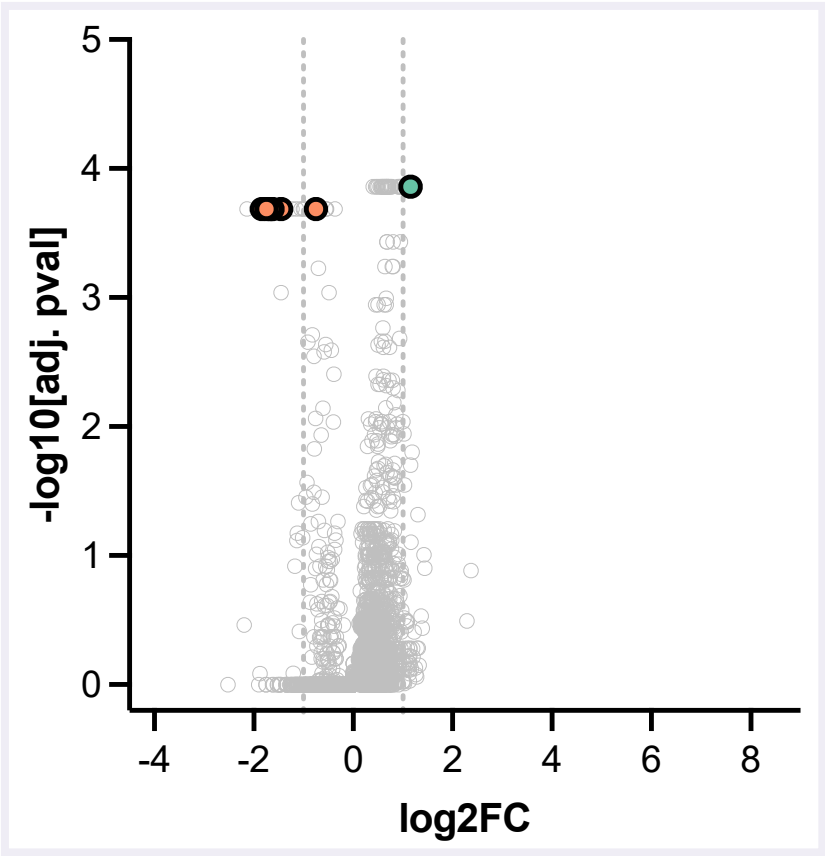


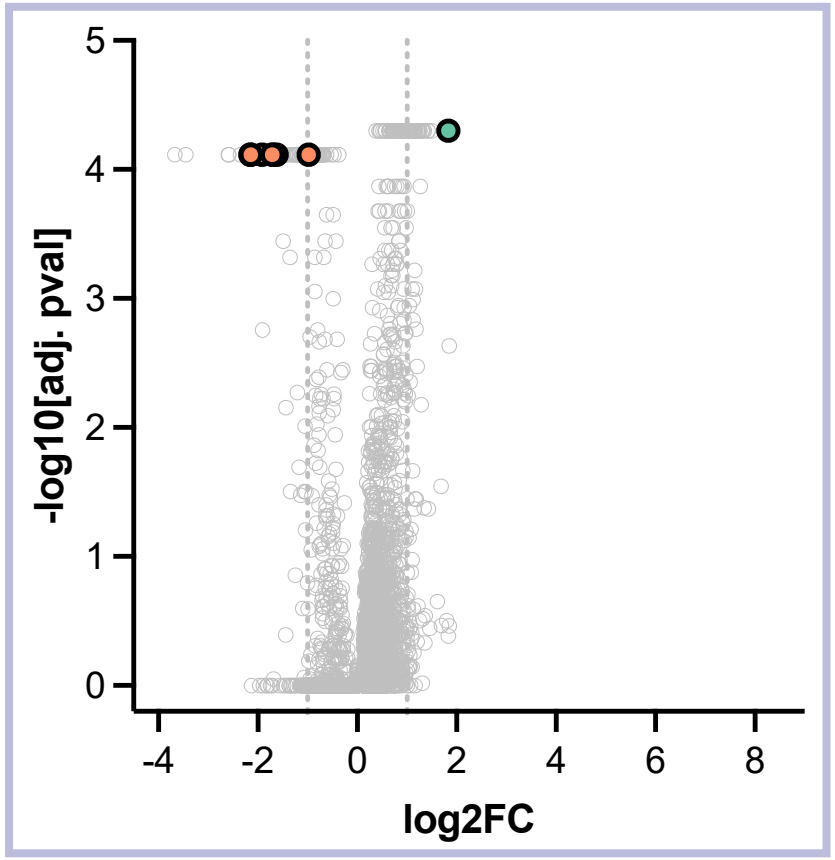


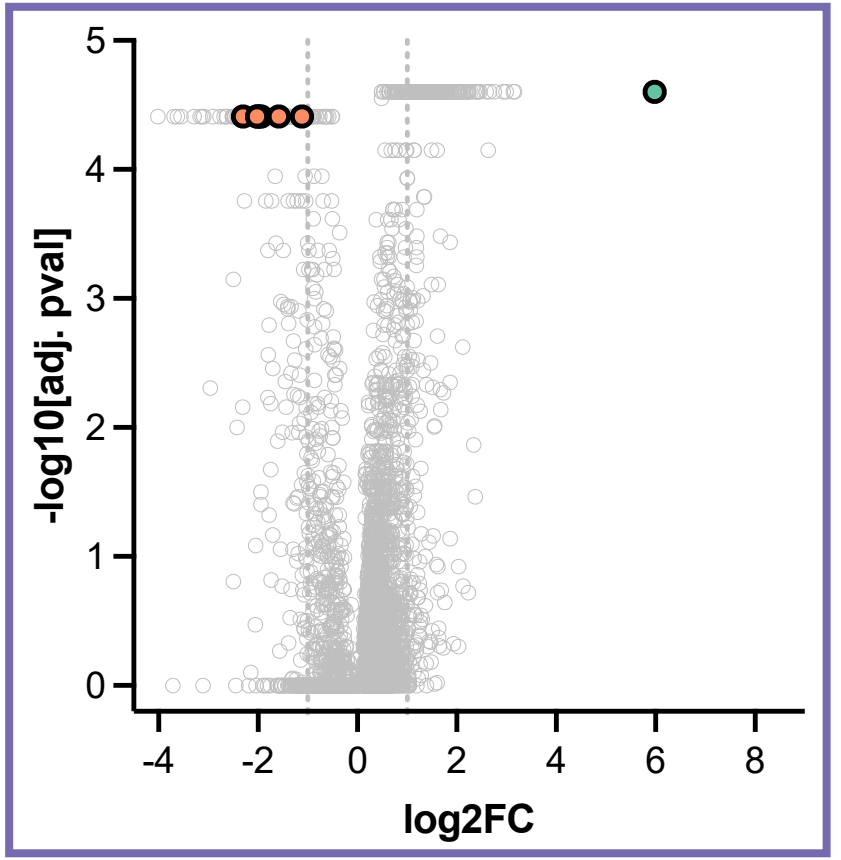
Chemical genomics



ATP synthase *mmpR5* (*rv0678*)

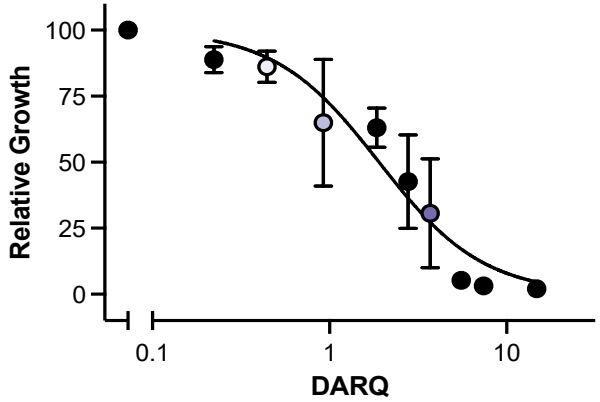


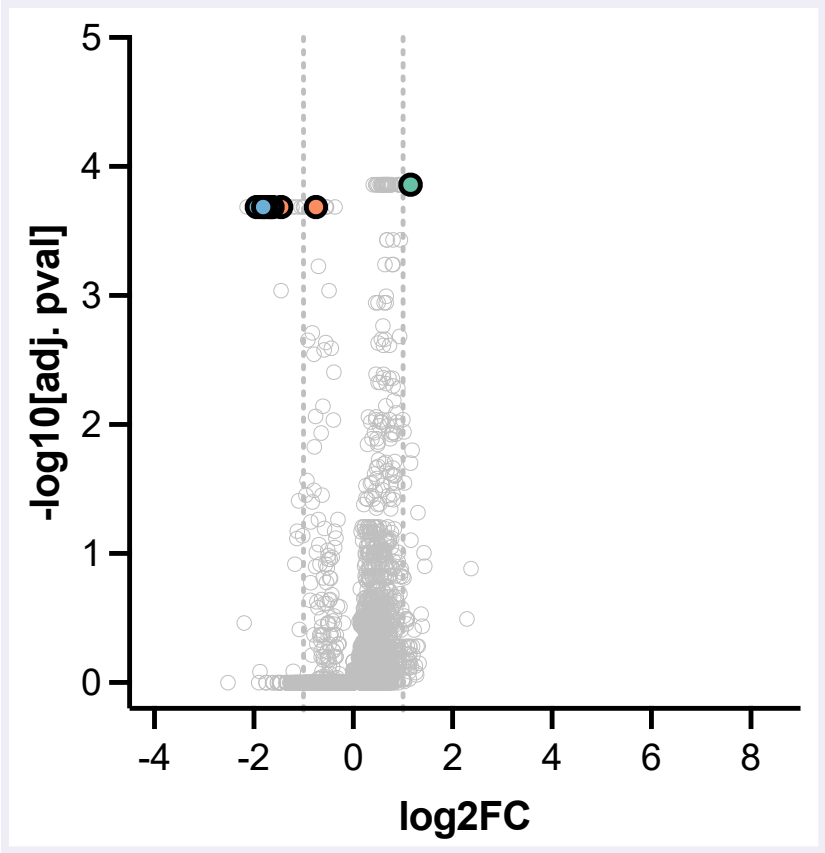


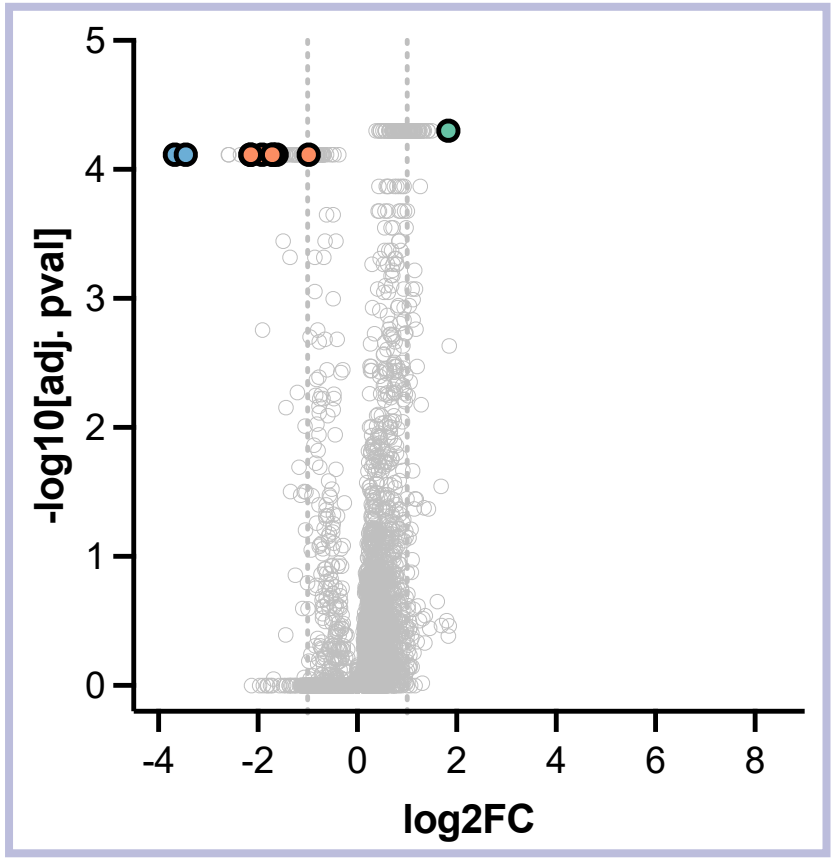


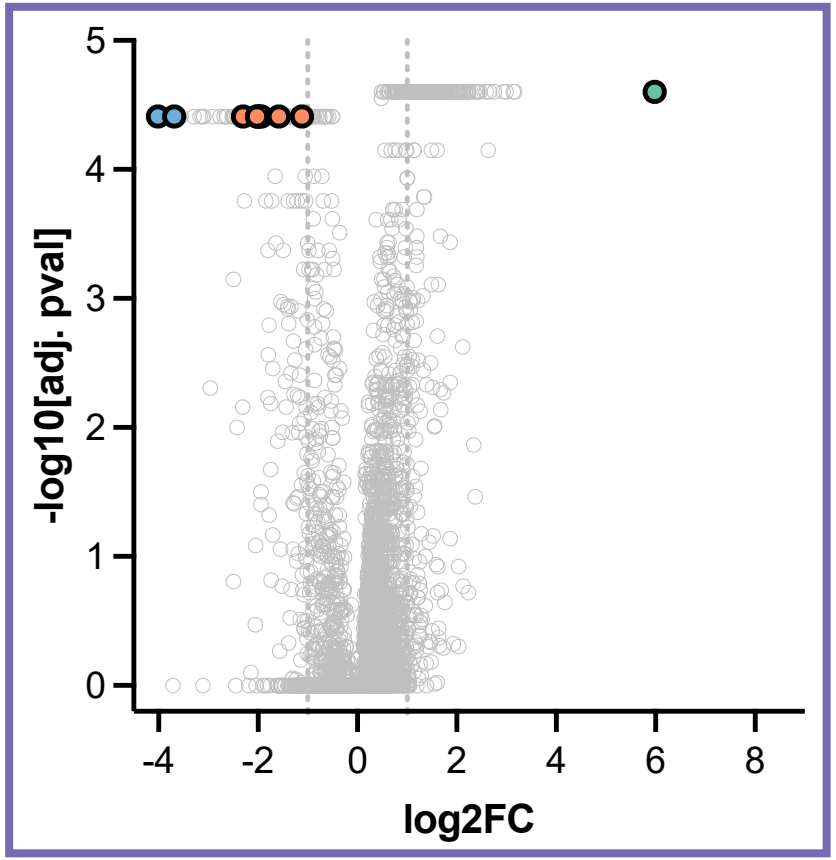
Chemical genomics

ATP synthase *mmpR5* (*rv0678*) *mmpL5S5*









Target-directed whole cells screens

nature Vol 441|18 May 2006|doi:10.1038/nature04784

LETTERS

Platensimycin is a selective FabF inhibitor with potent antibiotic properties

Jun Wang[1]*, Stephen M. Soisson[1]*, Katherine Young[1], Wesley Shoop[1]†, Srinivas Kodali[1], Andrew Galgoci[1], Ronald Painter[1], Gopalakrishnan Parthasarathy[1], Yui S. Tang[1], Richard Cummings[1], Sookhee Ha[1], Karen Dorso[1], Mary Motyl[1], Hiranthi Jayasuriya[1], John Ondeyka[1], Kithsiri Herath[1], Chaowei Zhang[1], Lorraine Hernandez[1], John Allocco[1], Ángela Basilio[1], José R. Tormo[1], Olga Genilloud[1], Francisca Vicente[1], Fernando Pelaez[1], Lawrence Colwell[1], Sang Ho Lee[1], Bruce Michael[1], Thomas Felcetto[1], Charles Gill[1], Lynn L. Silver[1]†, Jeffery D. Hermes[1], Ken Bartizal[1], John Barrett[1]‡, Dennis Schmatz[1], Joseph W. Becker[1], Doris Cully[1] & Sheo B. Singh[1]

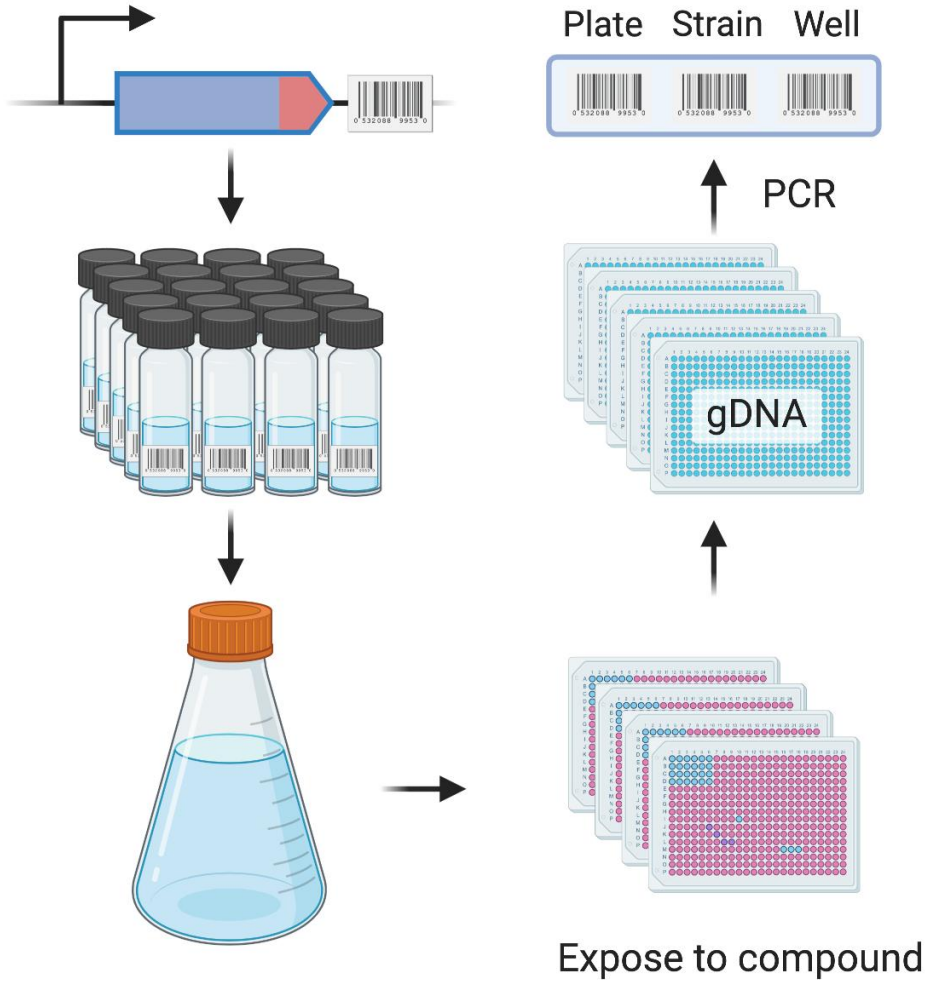
Chemistry & Biology
Article

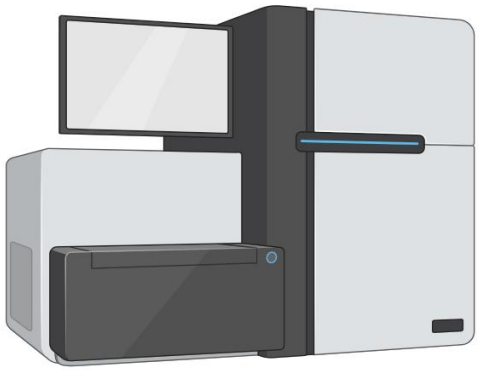
Pathway-Selective Sensitization of *Mycobacterium tuberculosis* for Target-Based Whole-Cell Screening

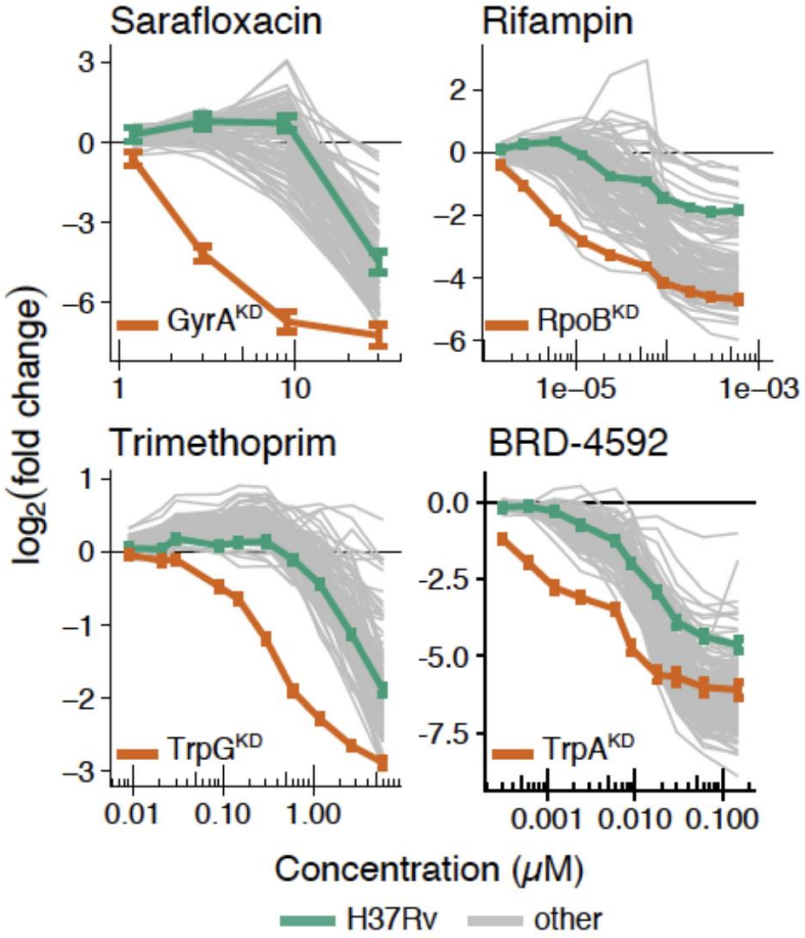
Garth L. Abrahams,[1,2,*] Anuradha Kumar,[3] Suzana Savvi,[1] Alvin W. Hung,[4] Shijun Wen,[4] Chris Abell,[4] Clifton E. Barry III,[5] David R. Sherman,[3] Helena I.M. Boshoff,[5] and Valerie Mizrahi[1,2,*]

Johnson et al. PMID: 31217586

Multiplexed target-directed whole cells screens







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Jeremy Rock

Barbara Bosch

Michael DeJesus

Nicholas Poulton

Shuqi Li

Broad Institute

Deborah Hung

Eachan Johnson

James Gomez

NIAID, BMGF, DOD, Potts Memorial Foundation, Boehringer Ingelheim Fonds, Wellcome Trust

Laura Cleghorn

Laura Cleghorn is the Tuberculosis Portfolio Leader in the Dundee Drug Discovery Unit (DDU) where she leads a team of multi-disciplinary researchers focused on identifying novel inhibitors with the potential to be progressed toward pre-clinical candidate selection and evaluated as a new therapy for TB.

Laura obtained a BSc(Hons) in Chemistry from the University of Edinburgh then worked at Organon Laboratories as a graduate medicinal chemist before moving to the University of Leeds where she obtained a PhD in Organic Chemistry in the lab of Prof. Ron Grigg. In 2006, Laura joined the newly formed Drug Discovery Unit as a medicinal chemist, initially working on Human African Trypanosomiasis, before moving to the Tuberculosis group in 2013, where she became the Portfolio Leader in 2020, and was afterwords promoted to the position of Reader in 2022.

https://www.dundee.ac.uk/people/laura-cleghorn | www.linkedin.com/in/laura-cleghorn-391822231/

Opportunities and challenges in TB drug discovery:

Targeting Pks13 as a case study

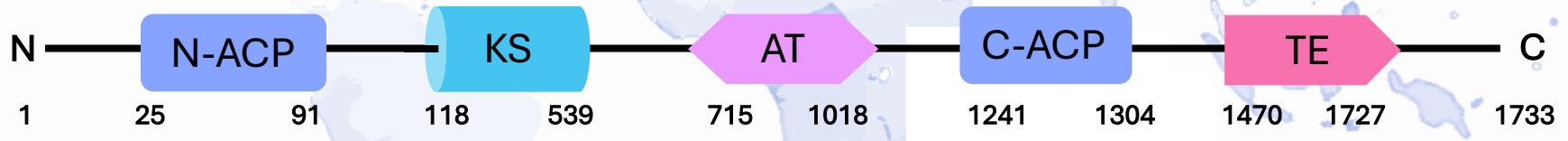
Laura Cleghorn

REVIVE webinar

9th September 2025

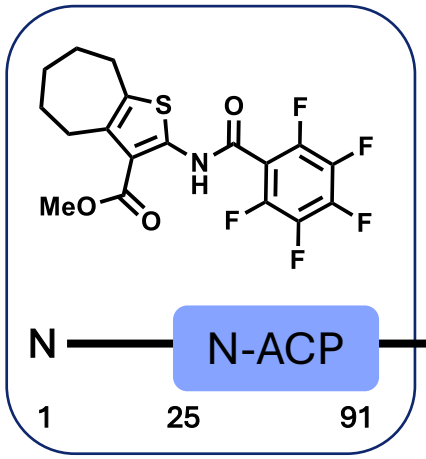
Polyketide Synthase (Pks13)

- Pks13 is an essential gene
  - Role: Condense 2 different fatty acid chains to produce cell wall mycolic acids
    - Only found in mycobacteria and is essential for its survival
- Pks13 protein contains1733 amino acids
  - 5 domains are known to have function

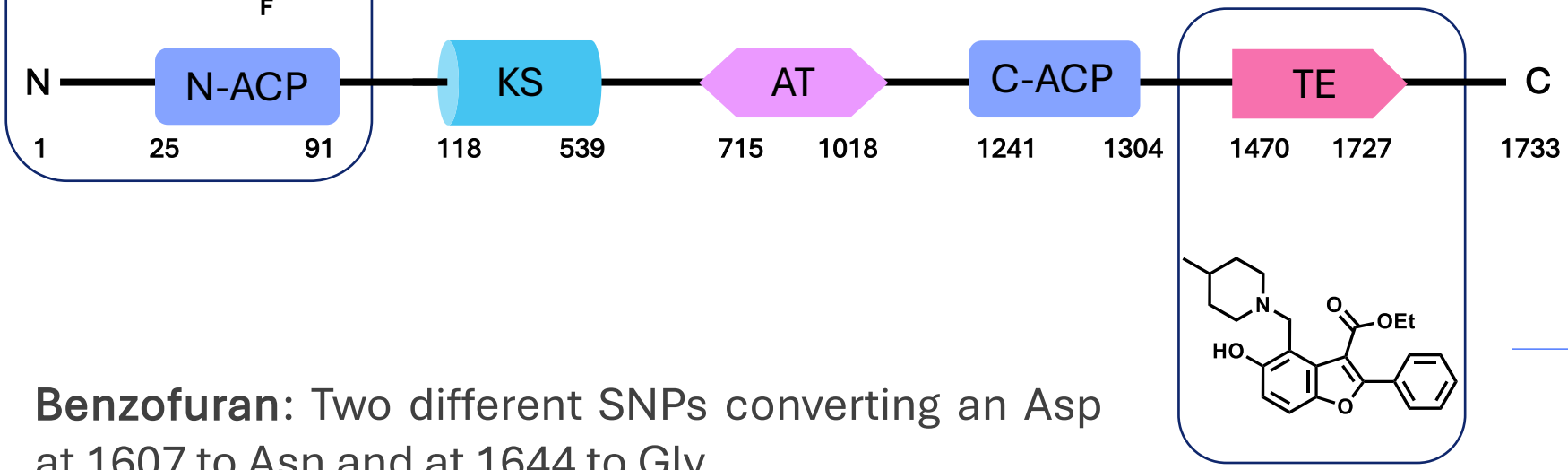


- Today's webinar will summarise drug discovery efforts to target Pks13

1st Discovery of Pks13 inhibitors



TP2: A single nucleotide polymorphism (SNP) converting Phe 79 to Ser conferred resistance

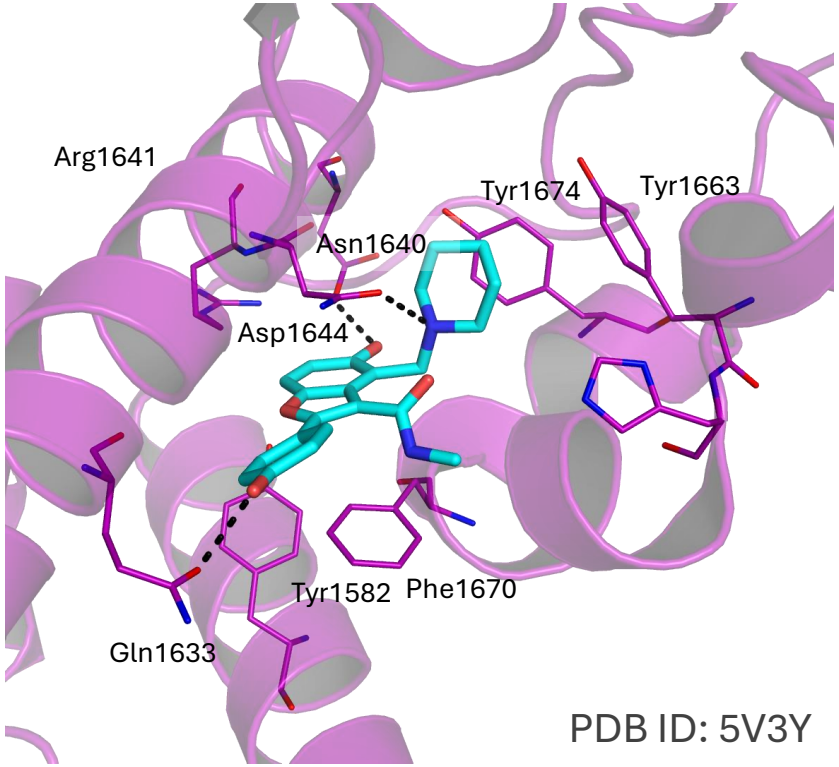


TAM16

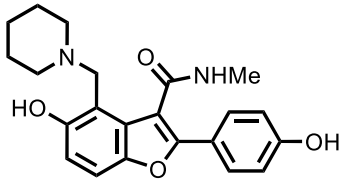
Benzofuran: Two different SNPs converting an Asp at 1607 to Asn and at 1644 to Gly

TP2: R. Wilson *et al.*, Antituberculosis thiophenes define a requirement for Pks13 in mycolic acid biosynthesis. *Nat Chem Biol* **2013,** *9* (8), 499-506.
Benzofuran: T. R. Ioerger *et al*, Identification of new drug targets and resistance mechanisms in *Mycobacterium tuberculosis*. *PLoS One* **2013,** *8* (9), e75245.

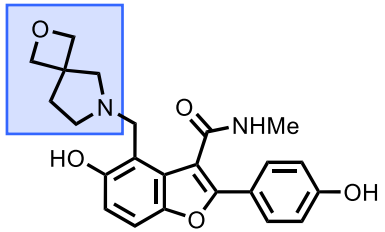
Benzofuran ‘TE’ series



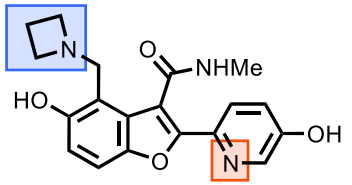
PDB ID: 5V3Y



	TAM16
Pks13 IC_{50}	0.3 µM
H37Rv MIC	0.08 µM
hERG IC50	7 µM
Microsomal clearance	3 mL/min/g

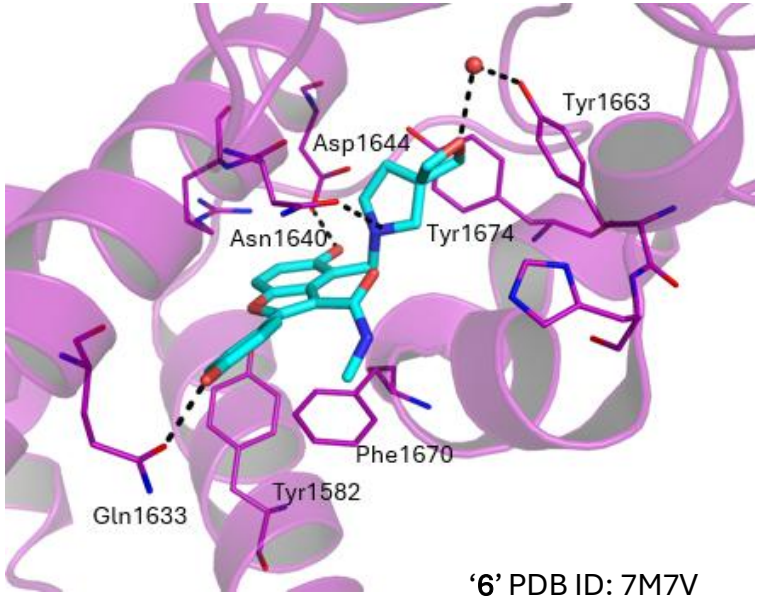


	6
Pks13 IC_{50}	0.3 µM
H37Rv MIC	0.6 µM
hERG IC50	> 30 µM
Microsomal clearance	3 mL/min/g

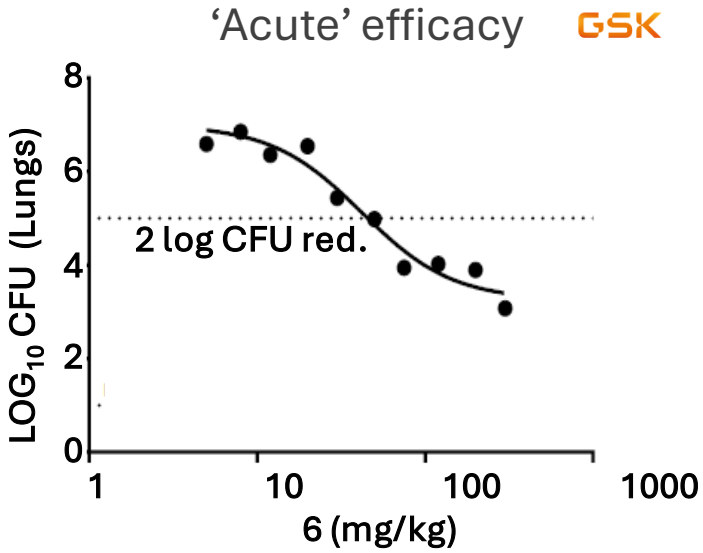


	12
Pks13 IC_{50}	0.2 µM
H37Rv MIC	0.3 µM
hERG IC50	> 30 µM
Microsomal clearance	0.9 mL/min/g

Benzofuran ‘TE’ series efficacy



‘**6**’ PDB ID: 7M7V



Follow up cardiotoxicity study on **6** highlighted the hERG liability remained

- Series halted

Major Challenge:

- Lipophilic amine essential & responsible for off-target cardiovascular toxicity

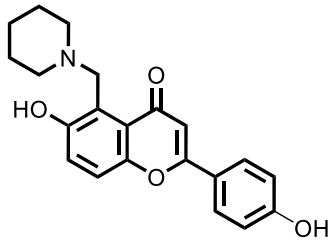
Target based screening strategy

Next a screening campaign to identify alternative chemical start points was initiated

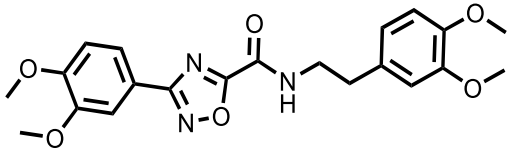
- ~183K compounds screened from a variety of libraries
- ~ 1,500 progressed to hit confirmation

1. Pks13 Enzyme Assay
2. Pks13 Counter screen
3. BLI direct binding assay
4. Pks13 hypomorph strain

Weill Cornell Medicine



	105*
Pks13 IC_{50}	0.5 µM
H37Rv MIC	0.08 µM
hERG IC50	0.8 µM
Microsomal clearance	3 mL/min/g

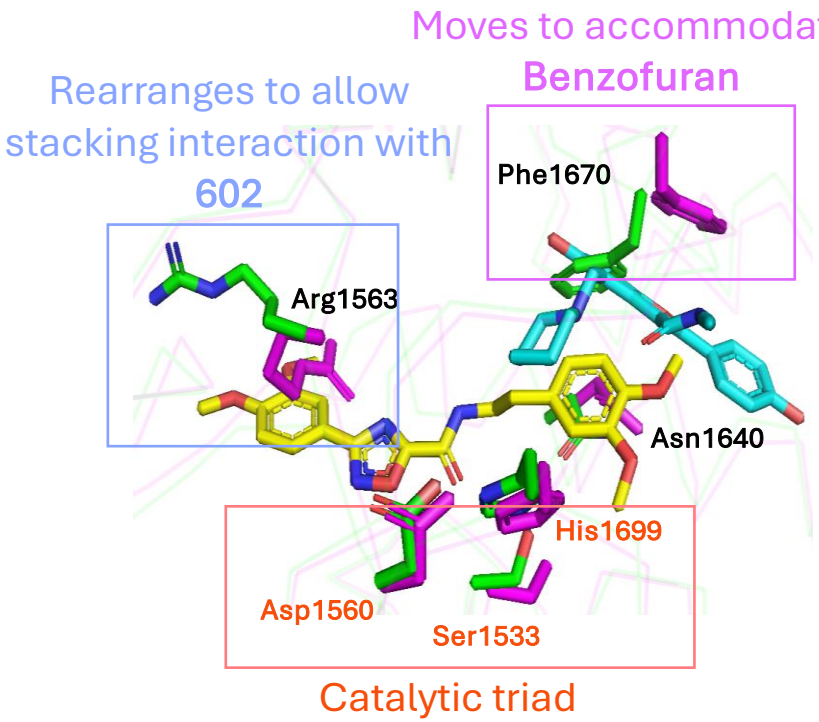


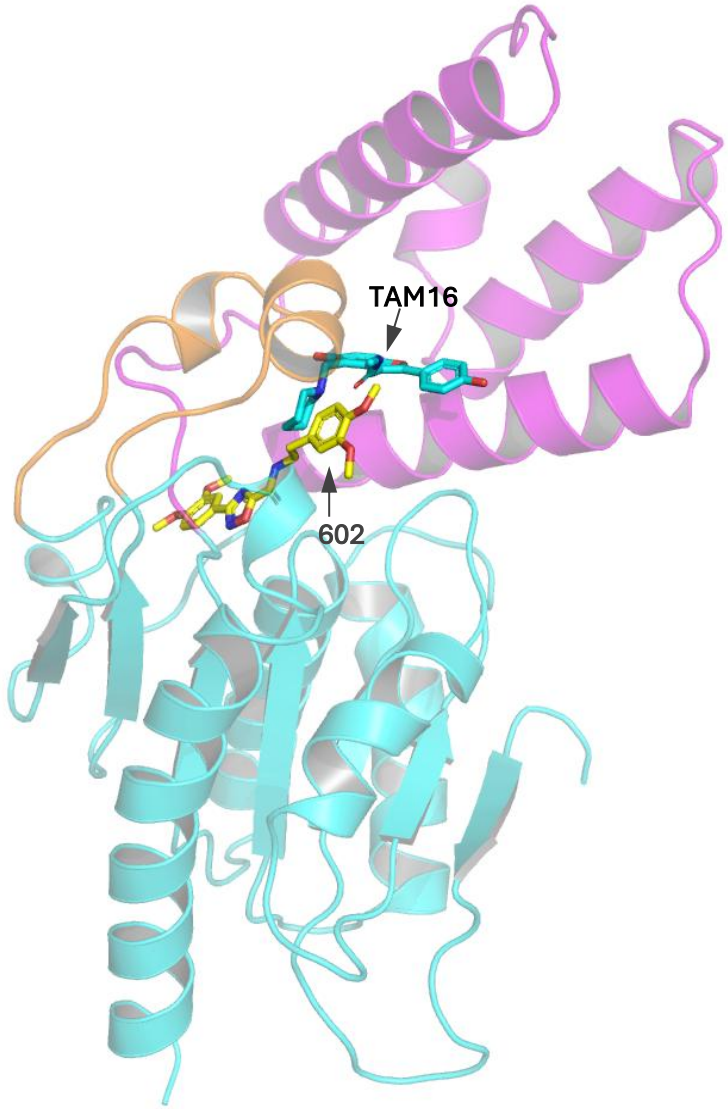
	602
Pks13 IC_{50}	0.3 µM
H37Rv MIC	11 µM
hERG IC50	> 20 µM
Microsomal clearance	20 mL/min/g

→ '602' Series

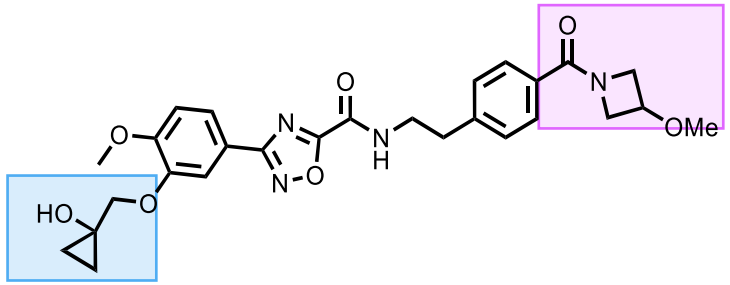
*X.Wang *et al.,* Identification of inhibitors targeting polyketide synthase 13 of *Mycobacterium tuberculosis* as antituberculosis drug leads. *Bioorg Chem* **2021,** *114*, 105110,

602 ‘TE’ series

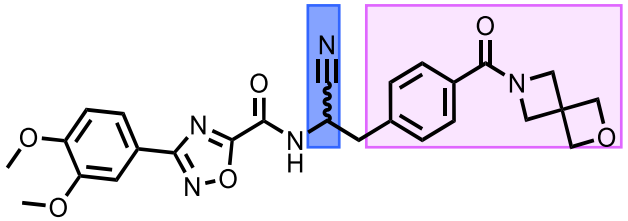




TAM16 (PDB ID 5V3Y: cyan and purple)
50 (PDB ID 8Q0T: yellow and green)



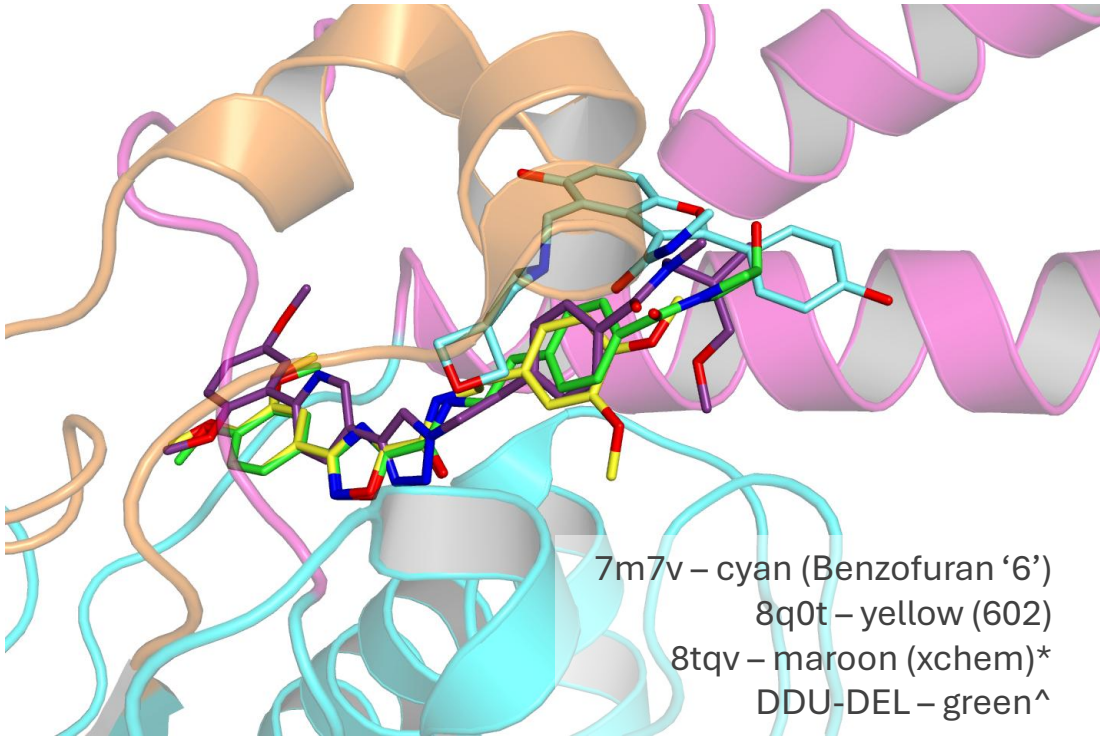
	80
Pks13 IC_{50}	0.3 µM
H37Rv MIC	1.2 µM
hERG IC50	ND
Microsomal clearance	1 mL/min/g



	44
Pks13 IC_{50}	0.4 µM
H37Rv MIC	0.7 µM
hERG IC50	> 70 µM
Microsomal clearance	1 mL/min/g

‘TE’ series from DEL screening

An alternative binding mode to inhibit the Pks13 ‘TE’ domain identified



DNA encoded library (DEL) screen

- Xchem/TAMU*
- DDU

DDU DEL

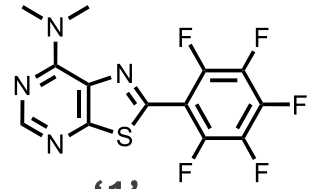
- Challenges
  - Balancing optimisation of ADME and retention of potency
  - hERG liability
  - Limited *in vivo* efficacy

*I. V. Krieger *et al.*, Inhibitors of the Thioesterase Activity of *Mycobacterium tuberculosis* Pks13 Discovered Using DNA-Encoded Chemical Library Screening, *ACS Infectious Diseases* **2024** *10* (5), 1561-1575, DOI: 10.1021/acsinfecdis.3c00592 ^Unpublished results

Phenotypic start point

Phenotypic screening of a large library of ~225K compounds - identified a potent singleton

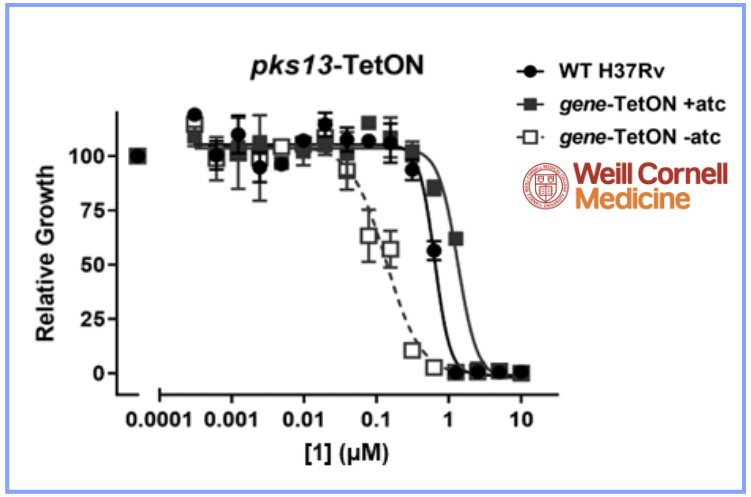
- Screening against a P*iniB*-LUX strain indicated the compound targeted the cell wall
  - Mmpl3, DprE1, InhA, Fad32, KasA, Pks13



'1'

Resistant mutants generated

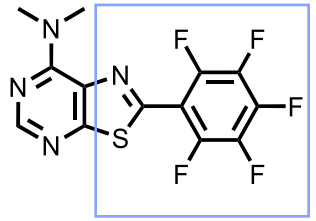
- FoR 10^{-8} (10 x MIC)
- N-ACP and KS domain
  - F79L like F79S mutation reported for **TP2**



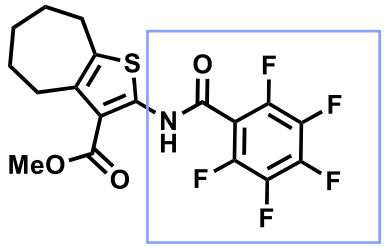
Both '**1**' and '**TP2**' contain a pentafluorophenyl moiety

- 'Potential covalent binder'
- Cys287 is the sole cysteine in 1st 800 residues of Pks13

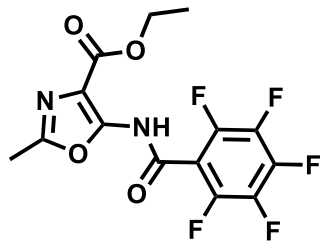
‘N-ACP/KS’ series development



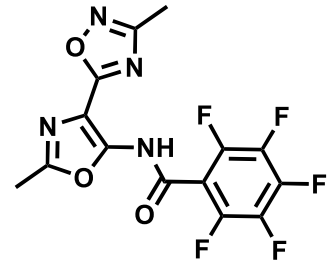
	1
H37Rv MIC	0.1 µM
hERG IC50	> 30 µM
Kin. Sol (µM)	2 µM
Microsomal clearance	> 50 mL/min/g



	TP2
H37Rv MIC	0.1 µM
hERG IC50	ND
Kin. Sol (µM)	0.2 µM
Microsomal clearance	15 mL/min/g



	24
H37Rv MIC	0.3 µM
hERG IC50	ND
Kin. Sol (µM)	204 µM
Microsomal clearance	> 0.5 mL/min/g
Hepatocyte clearance	12 mL/min/g



	43
H37Rv MIC	0.8 µM
hERG IC50	ND
Kin. Sol (µM)	196 µM
Microsomal clearance	> 0.5 mL/min/g
Hepatocyte clearance	3 mL/min/g

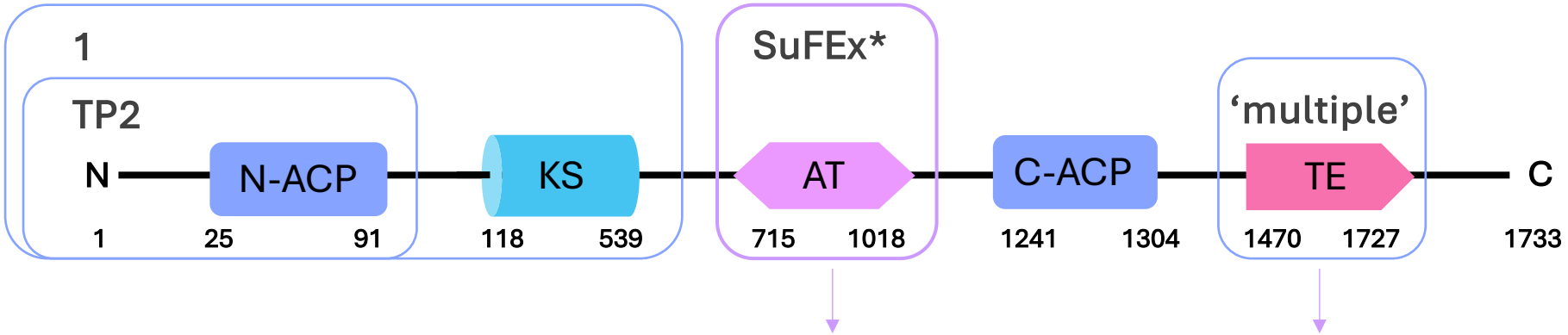
PK study ’**43**’ allowed progression to an ‘acute’ in vivo efficacy

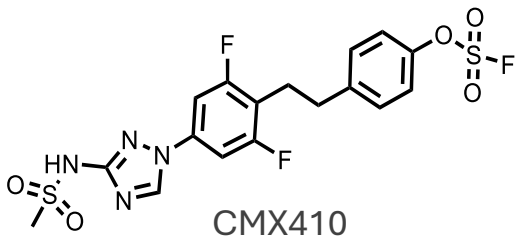
- No reduction in bacteria load (8-day study @ 200 mg/kg) observed
- High conc^{n} over 7 days required to see cidal effect in macrophages
  - Not an issue with other series

Domain coverage summary

Inhibitors across the Pks13 domains have been investigated

- Data available in public domain

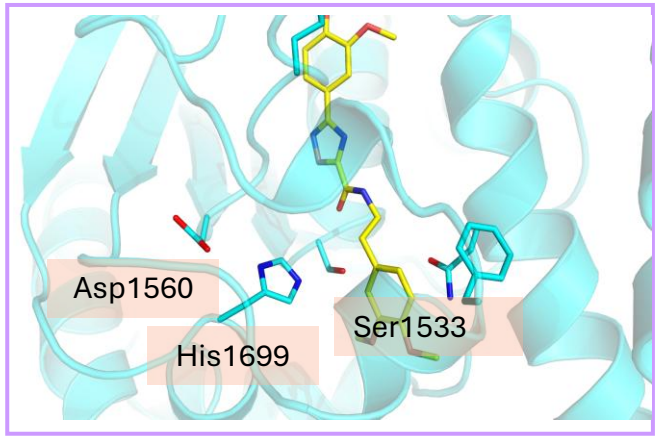




CMX410

Preclinical covalent compound

- Aryl fluorosulfate warhead



*Krieger, I.V., Sukheja, P., Yang, B. *et al.* SuFEx-based antitubercular compound irreversibly inhibits Pks13. *Nature,* **2025**, https://doi.org/10.1038/s41586-025-09286-3

Lessons learnt and challenges

Challenges:

- hERG (3/4 TE series needed to mitigate this risk)
- Limited PK exposure
- Identifying relevant chemical matter

Tools:

- Access to hypomorph has been essential to confirm on target
- Structural biology

Key question yet to be addressed:

- Will Pks13 contribute towards a superior regimen?

Acknowledgements

Benzofuran: Caroline Wilson, Peter Ray, Fabio Zuccotto, Jorge Hernandez, Anup Aggarwal, Claire Mackenzie, Nicola Caldwell, Malcolm Taylor, Margaret Huggett, Michael Mathieson, Dinakaran Murugesan, Alasdair Smith, Susan Davis, Mattia Cocco, Maloy K. Parai, Arjun Acharya, Fabio Tamaki, Paul Scullion, Ola Epemolu, Jennifer Riley, Laste Stojanovski, Eva Maria Lopez-Román, Pedro Alfonso Torres-Gómez, Ana Maria Toledo, Laura Guijarro-Lopez, Isabel Camino, Curtis A. Engelhart, Dirk Schnappinger, Lisa M. Massoudi, Anne Lenaerts, Gregory T. Robertson, Chris Walpole, David Matthews, David Floyd, James C. Sacchettini, Kevin D. Read, Lourdes Encinas, Robert H Bates, Simon R Green and Paul G Wyatt. J. Med. Chem. 2022, 65, 409−423

602: Simon R. Green, Caroline Wilson, Thomas C. Eadsforth, Avinash S. Punekar, Fabio K. Tamaki, Gavin Wood, Nicola Caldwell, Barbara Forte, Neil R. Norcross, Michael Kiczun, John M. Post, Eva Maria Lopez-Román, Curtis A. Engelhart, Iva Lukac, Fabio Zuccotto, Ola Epemolu, Helena I. M. Boshoff, Dirk Schnappinger, Chris Walpole, Ian H. Gilbert, Kevin D. Read, Paul G. Wyatt and Beatriz Baragaña *J. Med. Chem.* 2023, 66, 15380−15408

N-ACP/KS domain: Simon R Green, Justin R Harrison, Stephen Thompson, Dinakaran Murugesan, M Daben J Libardo, Curtis A Engelhart, Jaclynn Meshanni, Daniel Fletcher, Paul Scullion, Darren Edwards, Ola Epemolu, Nicole Mutter, Yoko Shishikura, Jennifer Riley, Thomas R Ioerger, Jose Juan Roca Guillén, Laura Guijarro López, Kevin D Read, Clifton E Barry 3rd, Dirk Schnappinger, Paul G Wyatt, Helena I M Boshoff, and Laura A T Cleghorn. *ACS Infect. Dis.* 2025, 11, 715−726

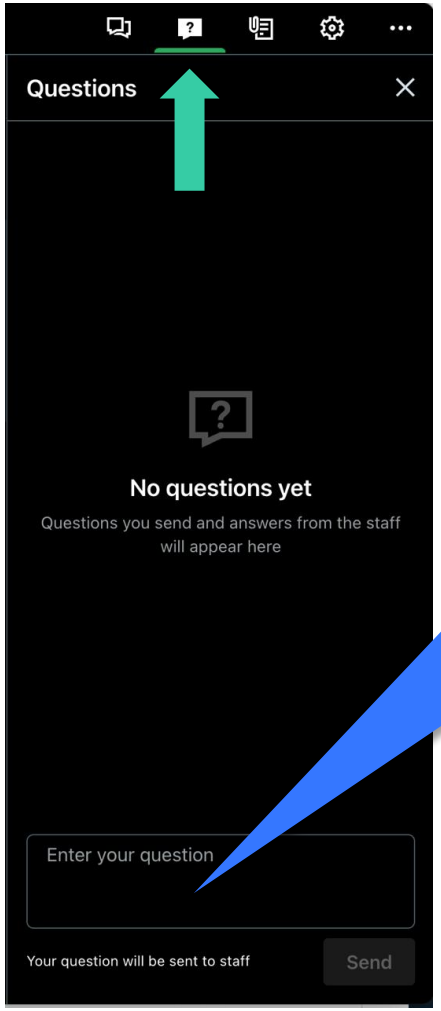
DEL series: Barbara Forte, Peter Campbell, Gary Knox, Avinash Punekar, Laura Simpson, Ian H. Gilbert, Kevin D. Read, Beatriz Baragaña and collaborators

Alice Dawson

How to submit your questions

REVIVE by GARDP

If your question is addressed to a specific speaker, please include their name when submitting the question.



Please submit your questions through the box provided after clicking the ‘questions’ button. We will review all questions and respond to as many as possible after the presentation.

Thank you to today's speakers

REVIVE by GARDP

Overcoming challenges of tuberculosis drug discovery and development

Moderator:
Valerie Mizrahi
Director, Institute of Infectious Disease and Molecular Medicine, University of Cape Town (South Africa)

Jeremy Rock
Associate Professor, Rockefeller University (USA)

Dirk Schnappinger
Professor, Department of Microbiology & Immunology, Weill Cornell Medical College (USA)

Laura Cleghorn
Reader, Drug Discovery Unit, School of Life Sciences, University of Dundee (UK)

Upcoming webinars

REVIVE by GARDP

Using artificial intelligence to analyse and predict susceptibility to antimicrobials

With Adrian Egli & Javier Fernández Domínguez

28 October 2025, 17:00-18:30 CEST

Thank you for joining us