Childhood Cancer Data Initiative Virtual Symposium Series

Frederic G. Barr, Corinne M. Linardic, and Ann Ramer

NIH NATIONAL CANCER INSTITUTE

March 18, 2026

Today's Moderator and Speakers

GREGORY REAMAN, M.D.

Moderator

Advisor, Childhood Cancer Data Initiative

National Cancer Institute

FREDERIC G. BARR, M.D., PH.D.

- *Deputy Chief*
- *Medical Director*
- *Senior Investigator*

Laboratory of Pathology
National Cancer Institute

CORINNE M. LINARDIC, M.D., PH.D.

- *Associate Professor of Pediatrics*
- *Associate Professor of Pharmacology and Cancer Biology*
- *Associate Professor of Cell Biology*

Duke University

ANN RAMER, M.P.H.

Regional Health Officer

Ohio Department of Health

Agenda

1. *Application of DNA Methylation Analysis to the Classification of Rhabdomyosarcoma*
   - *Q&A with Dr. Frederic G. Barr*
2. *Findings From the FusOnC2 Consortium: Focus on Rhabdomyosarcoma*
   - *Q&A with Dr. Corinne Linardic*
3. *Opportunities to Expand Patient/Family Engagement in the Genomic Characterization of Pediatric Malignancies*
   - *Q&A with Ann Ramer*

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Application of DNA methylation analysis to the classification of rhabdomyosarcoma

Frederic G. Barr, MD PhD
Laboratory of Pathology
National Cancer Institute

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Rhabdomyosarcoma

- Family of soft tissue cancers
- Striated muscle differentiation
- Clinical and biological heterogeneity

Subtype	Fusion-negative	Fusion-positive
Age		
Site		
Outcome		
Genetics		 PAX3::FOXO1 PAX7::FOXO1

Epigenetic mechanisms – DNA methylation

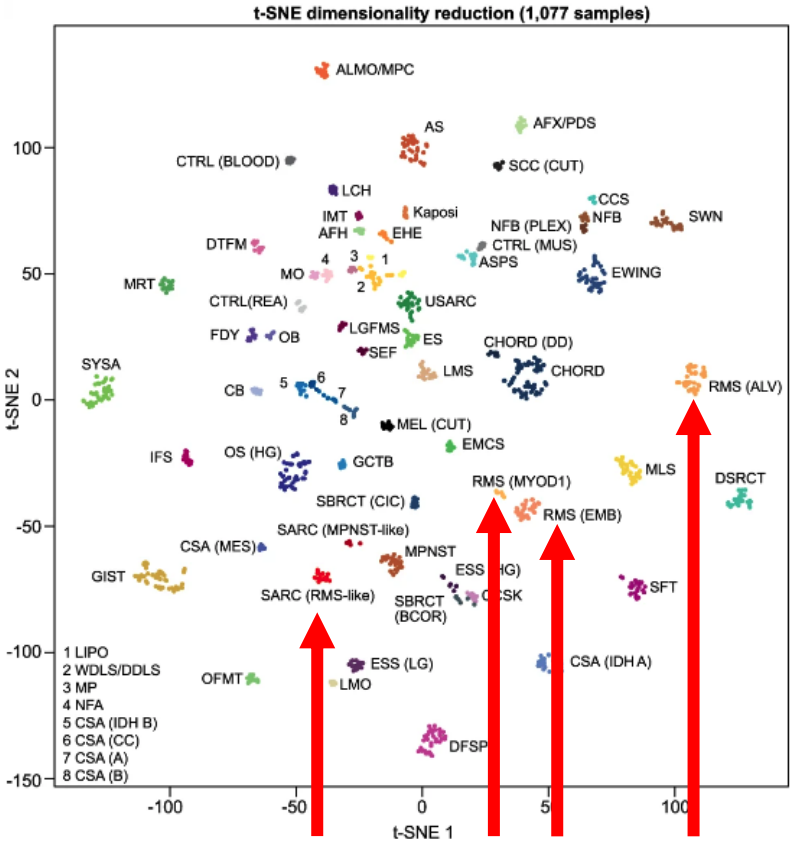
Role of DNA methylation:

- Regulates nuclear organization and chromatin structure
- Impact on gene expression

DNA methylation pattern in cancer cells:

- Partly reflects cell of origin
- Partly reflects acquired changes during tumorigenesis
- Potentially useful for differential diagnosis and management

DNA methylation-based classification of sarcomas



1,077 bone & soft tissue sarcomas
- Unsupervised analysis of DNA methylation data
- Defined 62 tumor methylation classes

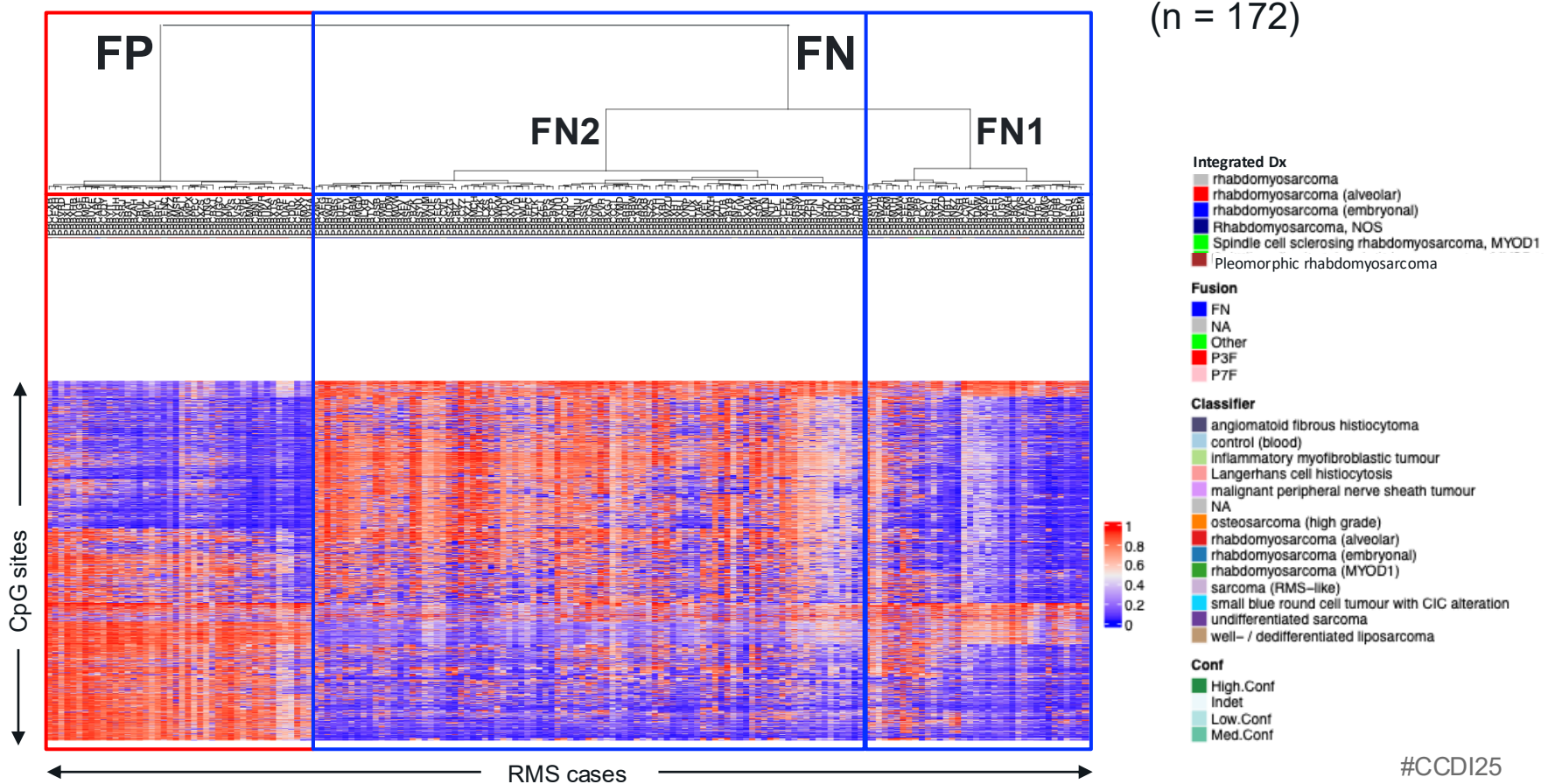
4 classes corresponding to RMS categories:
- RMS (ALV) - alveolar RMS
- RMS (EMB) - embryonal RMS
- RMS (MYOD1) - MYOD1-mutant RMS
- SARC (RMS-like) – RMS-like sarcoma (DICER1)

DNA methylation-based classifier
- Algorithm developed by machine learning
- Individualized specimen analysis
  - ➢ Predict tumor class
  - ➢ Calculate confidence level (0 to 1.0)

(Koelsche et al. Nature Comm 12:498, 2021)

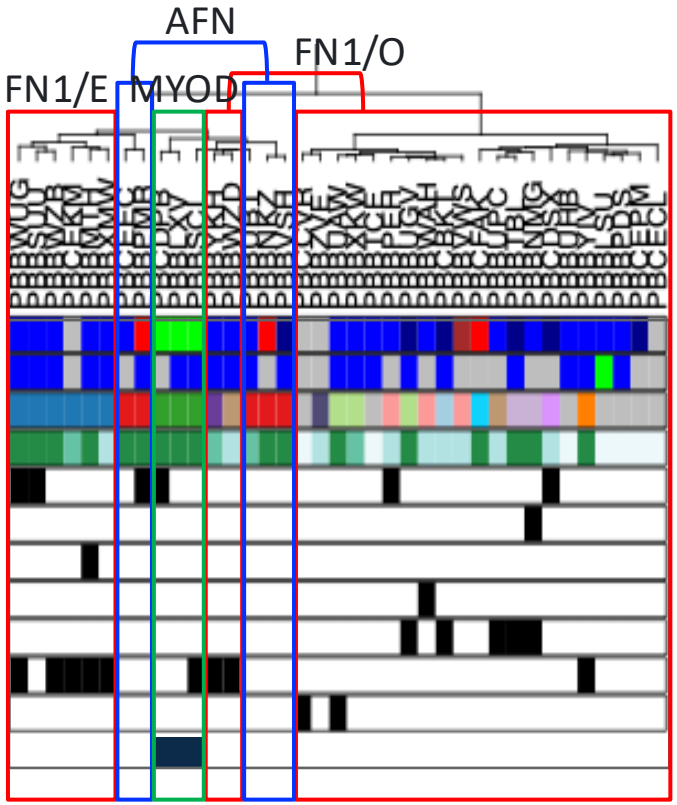
DNA methylation analysis of MCI cohort of RMS

(n = 172)



#CCDI25

Composition of FN1 subset in MCI RMS cohort



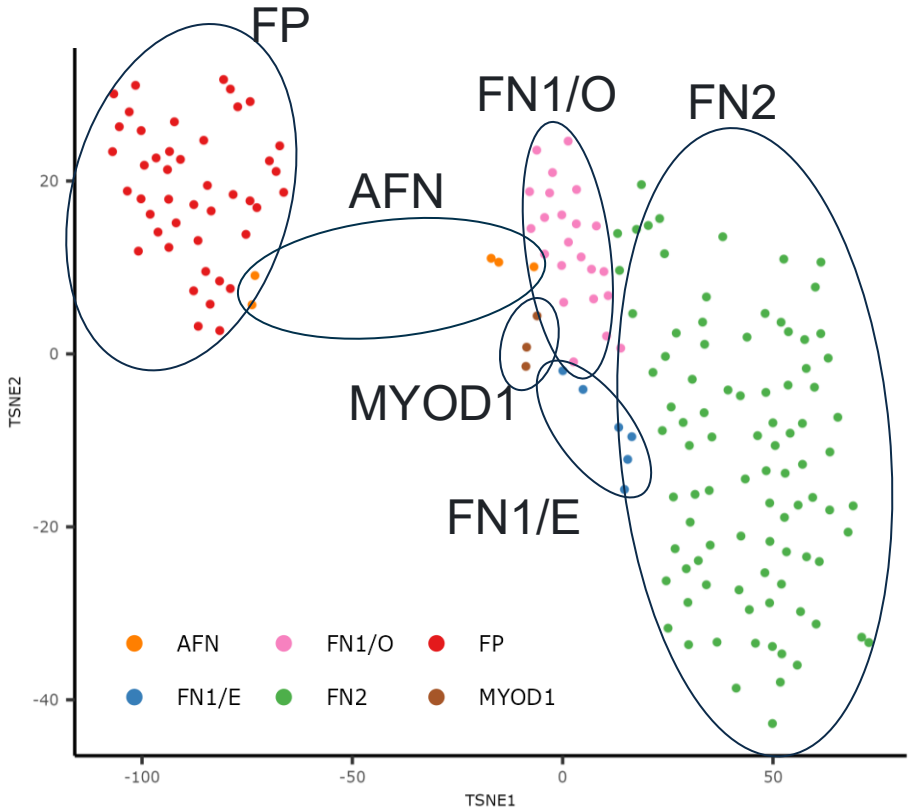
Integrated Dx
- rhabdomyosarcoma
- rhabdomyosarcoma (alveolar)
- rhabdomyosarcoma (embryonal)
- Rhabdomyosarcoma, NOS
- Spindle cell sclerosing rhabdomyosarcoma, MYOD1
- Pleomorphic rhabdomyosarcoma

Fusion
- FN
- NA
- Other
- P3F
- P7F

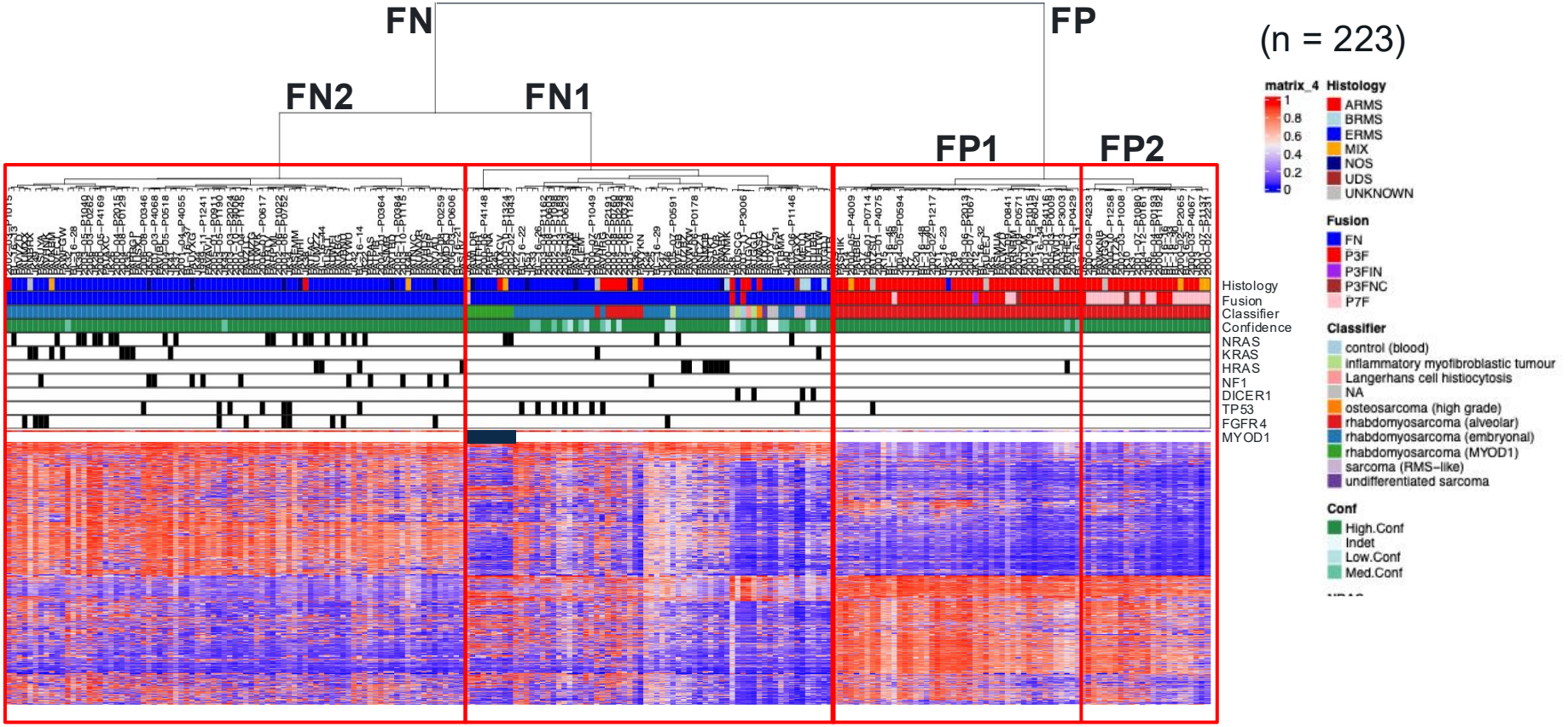
Classifier
- angiomatoid fibrous histiocytoma
- control (blood)
- inflammatory myofibroblastic tumour
- Langerhans cell histiocytosis
- malignant peripheral nerve sheath tumour
- NA
- osteosarcoma (high grade)
- rhabdomyosarcoma (alveolar)
- rhabdomyosarcoma (embryonal)
- rhabdomyosarcoma (MYOD1)
- sarcoma (RMS–like)
- small blue round cell tumour with CIC alteration
- undifferentiated sarcoma
- well– / dedifferentiated liposarcoma

Conf
- High.Conf
- Indet
- Low.Conf
- Med.Conf

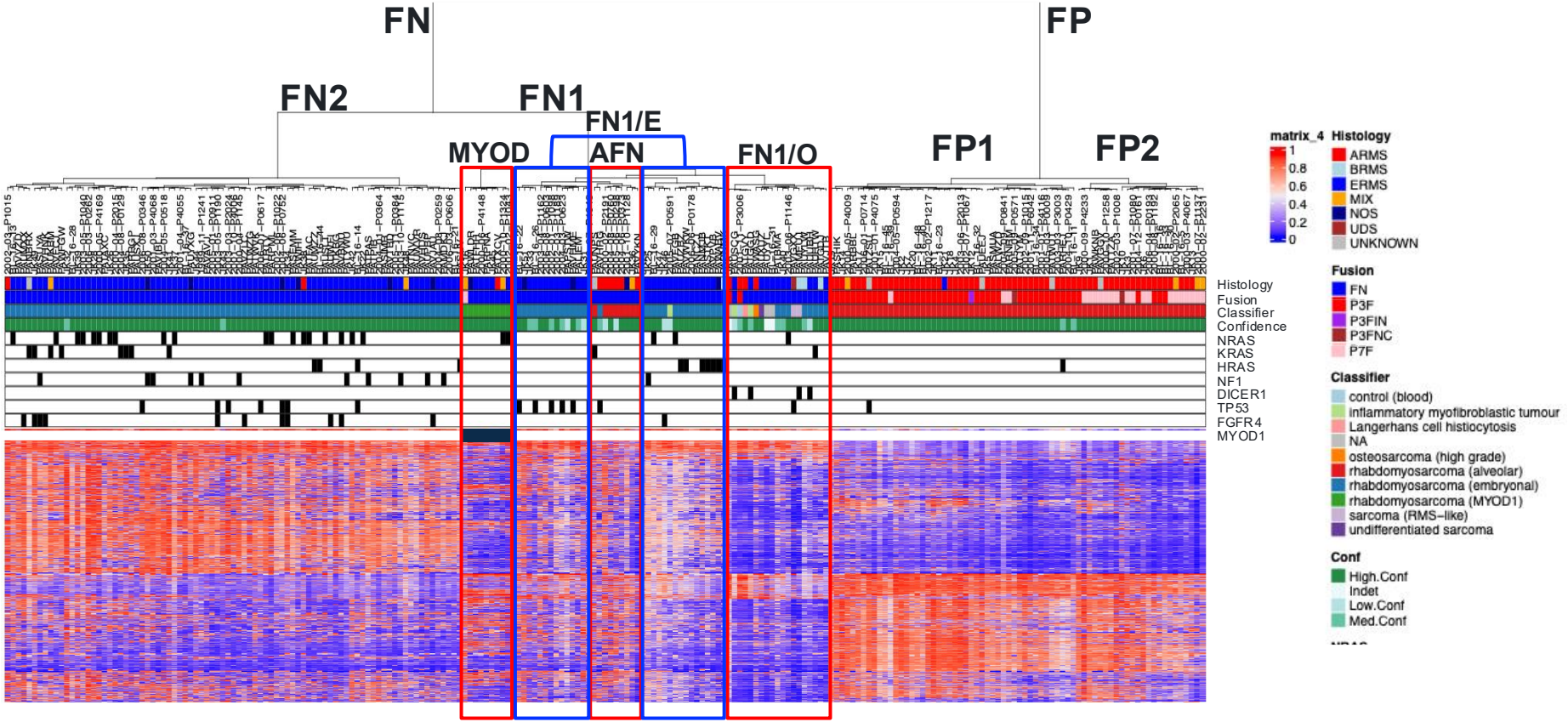
tSNE plot of RMS cases from MCI cohort



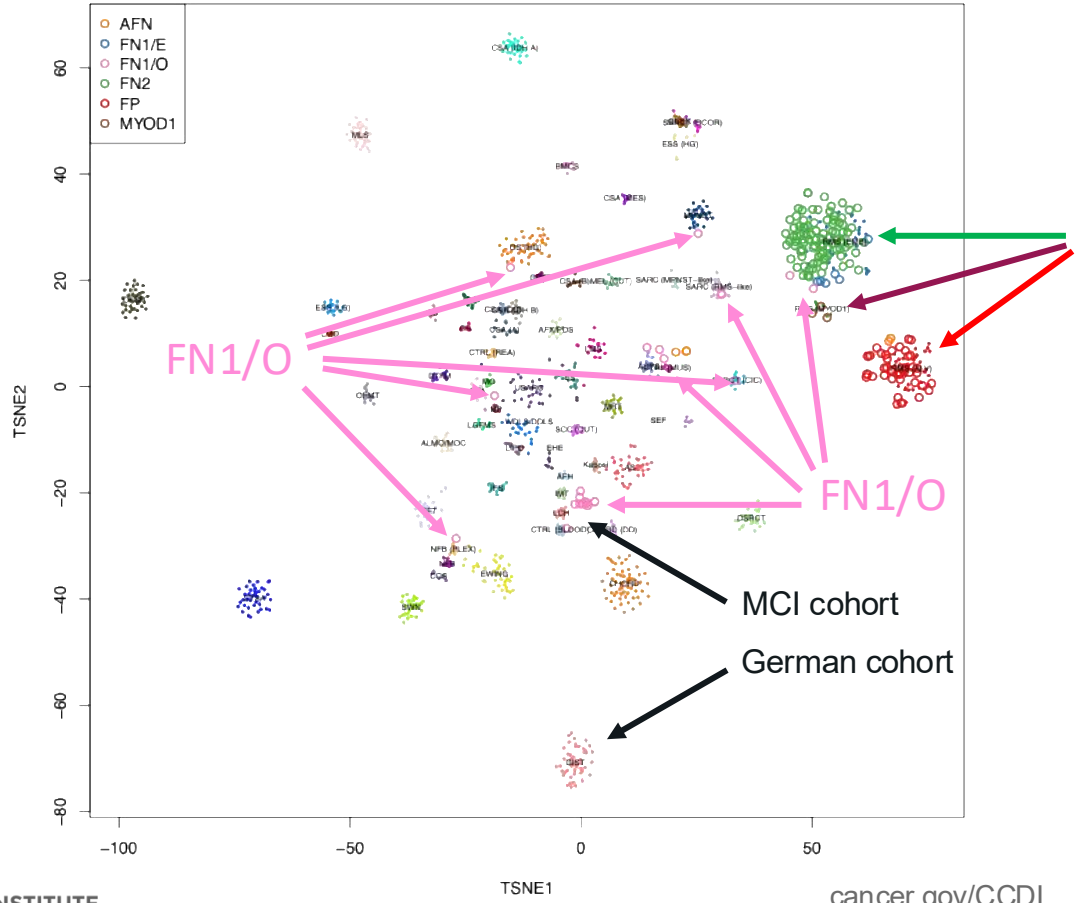
DNA methylation analysis of archival cohort of RMS



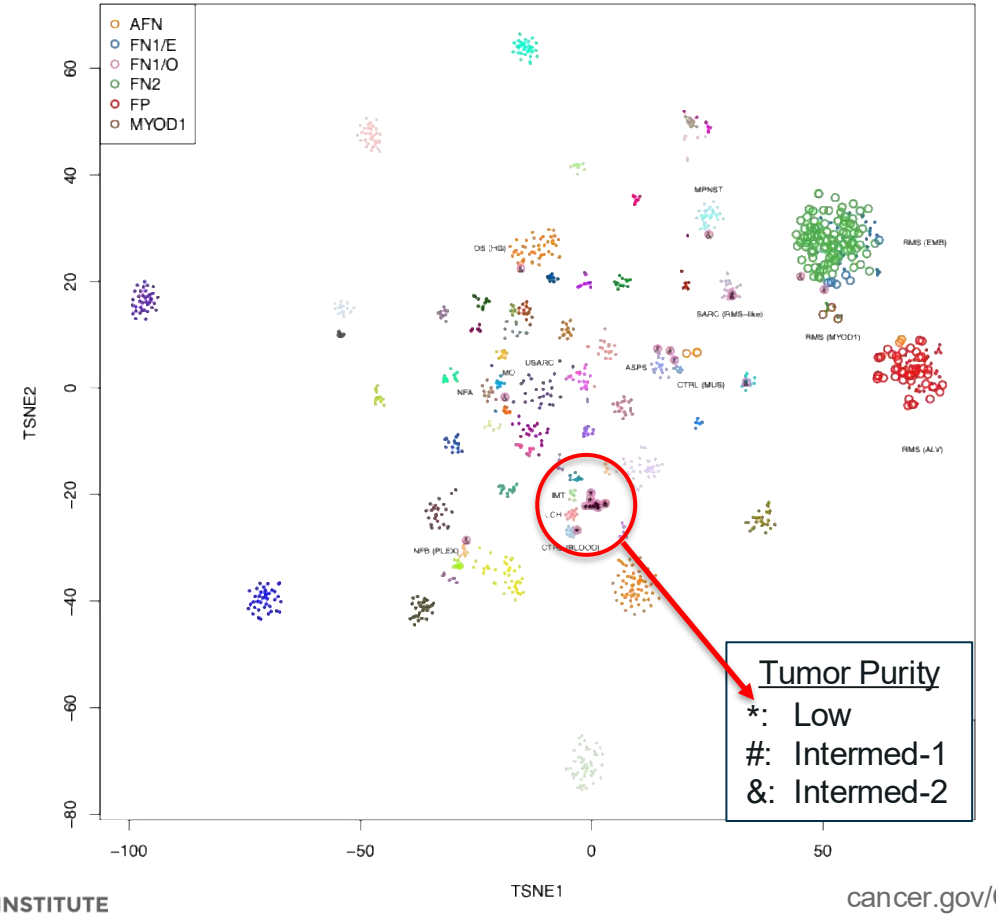
Composition of FN1 subset in archival RMS cohort



Relationship of outliers to other sarcoma categories



Tumor purity in outlier tumor samples



DNA methylation-defined RMS subsets

Fusion-positive RMS tumors - relatively homogeneous/alveolar histology

- PAX3::FOXO1-enriched subset (FP1)
- PAX7::FOXO1-enriched subset (FP2)

Fusion-negative RMS tumors - heterogenous

- Embryonal histology
  - FN2 subset - most abundant
  - FN1/E subset - less abundant
- MYOD1-mutant - sclerosing spindle cell histology
- Alveolar histology (AFN) - heterogeneous
- Outliers (FN1/O) - multiple subsets
  - Rare RMS subsets
  - Non-RMS subsets
  - Substantial normal cell component

Next challenge – biological & clinical significance of these RMS subsets

Research team and collaborators

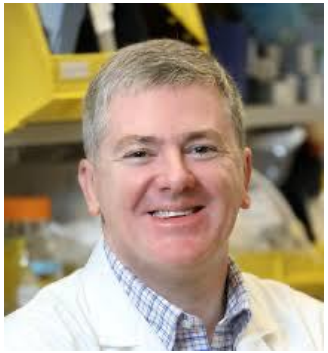
National Cancer Institute
Laboratory of Pathology

Wenyue Sun, PhD
Staff Scientist

Jorge Lopez-Nava
Postbac Trainee

Children's Oncology Group
Soft Tissue Sarcoma Committee

Jack Shern, MD
NCI



Philip Lupo, PhD
Emory Univ.

Erin Rudzinski, MD
Indiana Univ.

Sapna Oberoi, MD
Univ. Manitoba

Q&A

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Findings from the FusOnC2 Consortium: Focus on Rhabdomyosarcoma

Corinne M. Linardic M.D., Ph.D.

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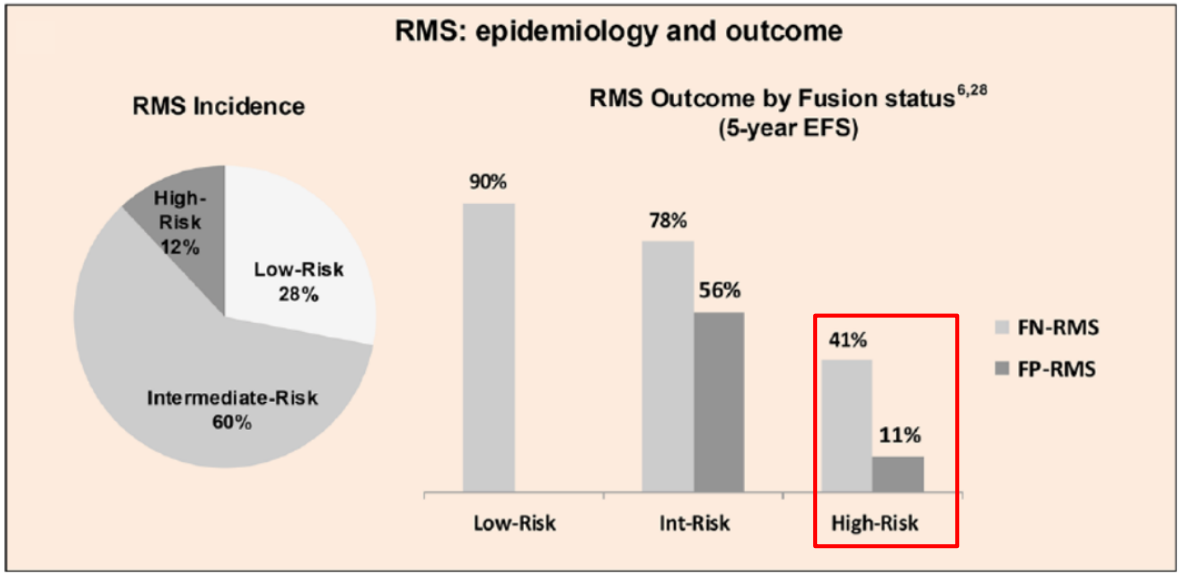
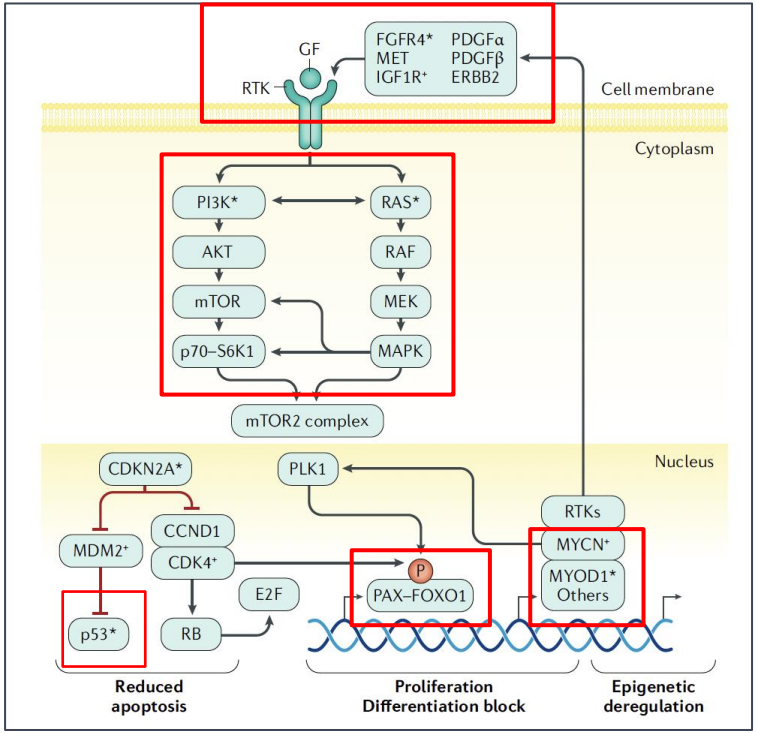
Agenda

- Rationale for focus on rhabdomyosarcoma (RMS)
- Introduce FusOnC2 consortium
- Share insight into molecular vulnerabilities of RMS
  - CDK8
  - TYMS
  - FANCM

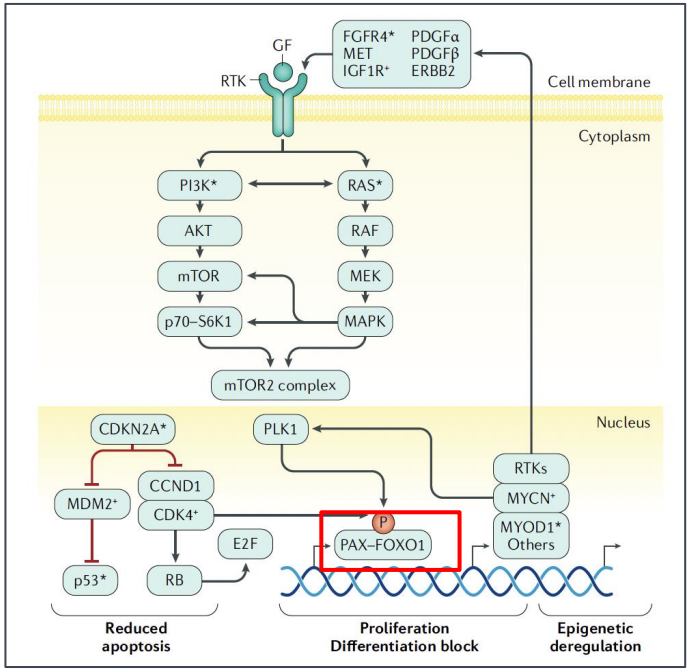
Rationale for focus on RMS

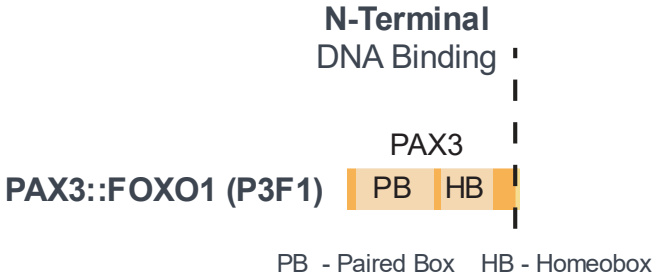


Rationale for focus on RMS



The PAX3::FOXO1 Oncofusion





Hyperactive transcription factor
- retains DNA-binding specificity of PAX3, reactivating embryonic myogenic programs, but has altered transactivation

Excessive growth and motility

Deficient apoptosis and differentiation

Recalcitrant pharmacologic target

AlphaFold (Beta) prediction

NCI FusOnC2 Program for Fusion-Positive Childhood Cancers


Corinne Linardic
Duke


Chris Counter
Duke


Kris Wood
Duke


Dave Root
Broad Institute


Dave Langenau
Mass General


Angela Koehler
MIT-Koch Institute


Jack Shern
NCI


Kim Stegmaier
Dana Farber

Fusion Oncoproteins in Childhood Cancers

Long-Term

Chemical Probe Discovery

Small molecule microarrays; Development of chemical probes and PROTACs

Catalyze ligand discovery to probe biological and therapeutic hypotheses

Short-Term

Fusion Interactome Mapping

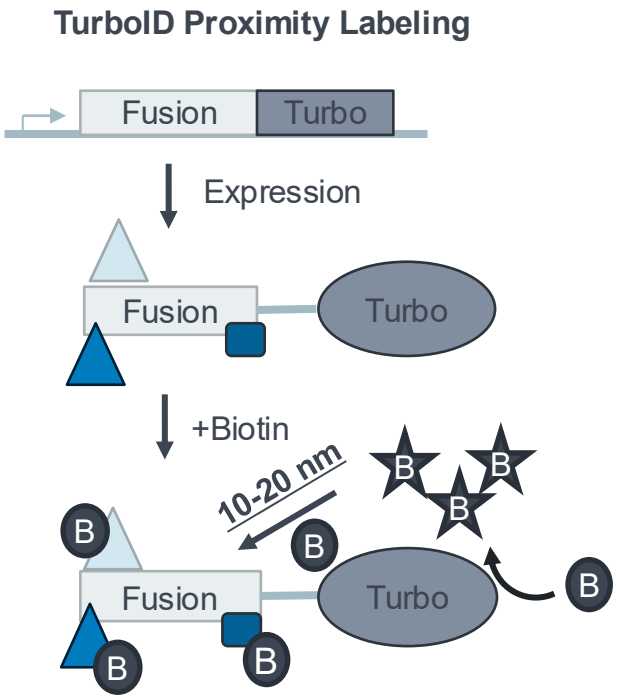
Proximity labelling; Saturation mutagenesis; Functional genomic screens

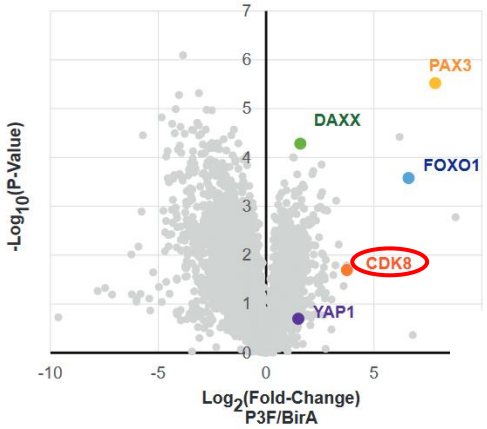
Map physiologically relevant interactions; Define critical interactions and molecular interfaces

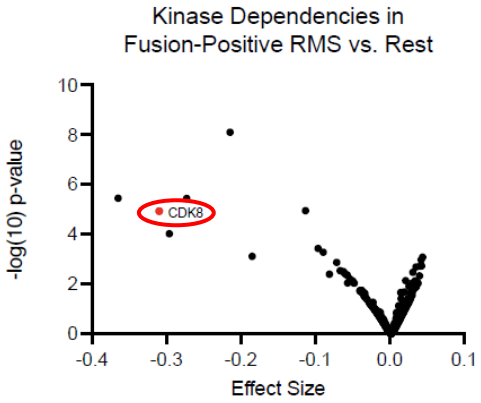
CDK8

A transcriptional vulnerability

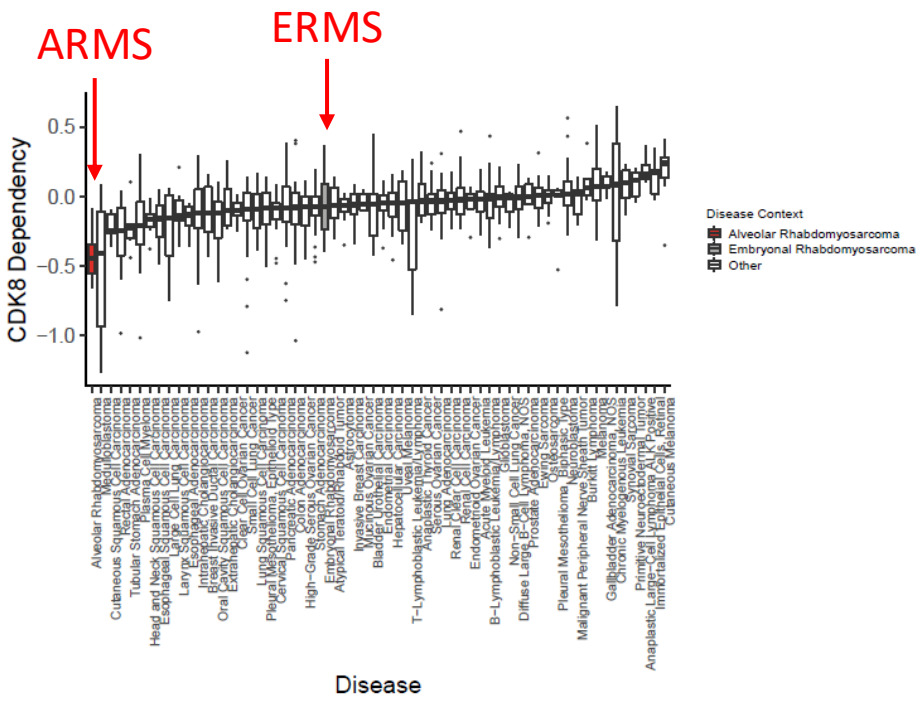
Project Premise: Proximity Labelling Proteomics Will Identify PAX3::FOXO1 Interactors and RMS Vulnerabilities



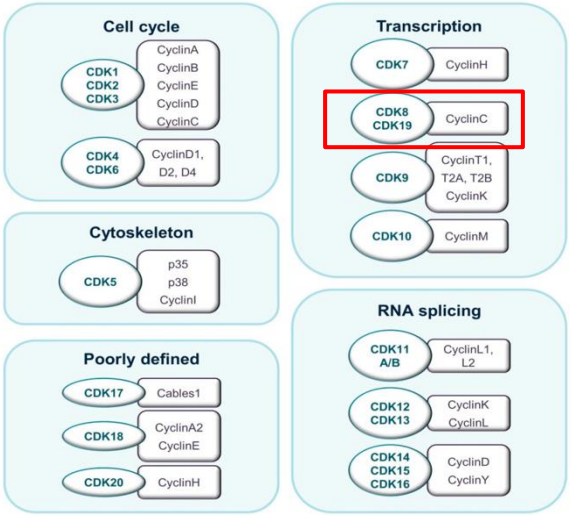




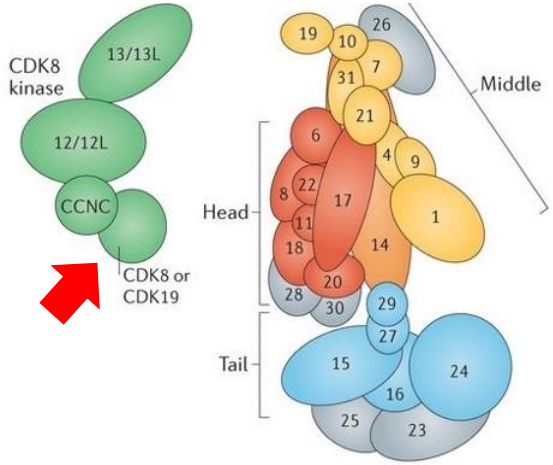
Proximity Labelling Proteomics Identifies CDK8 as a PAX3::FOXO1 Interactor and RMS Vulnerability



What Is CDK8?

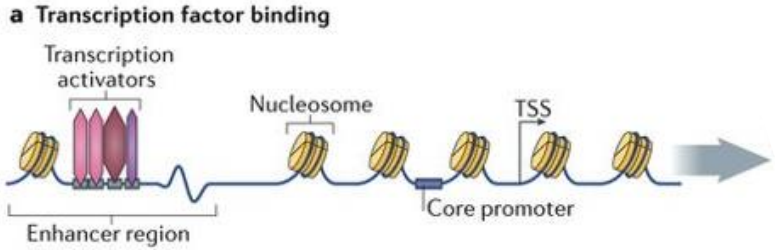


Mediator complex

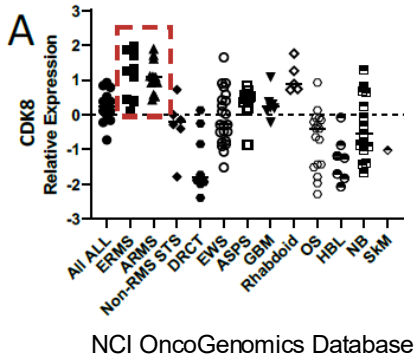


Menzl et al. *Pharmaceuticals*, 2019
Soutourina. *Nature Reviews MCB*, 2018

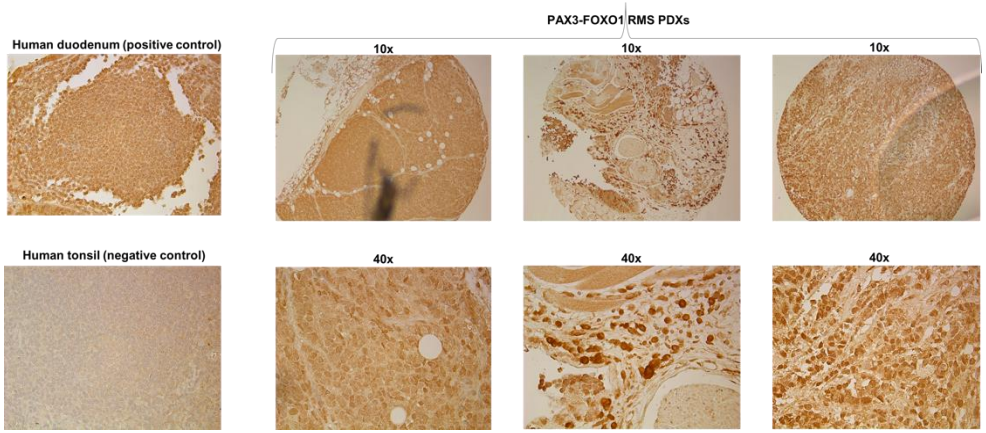
What Is CDK8?


a Transcription factor binding
Transcription activators
Nucleosome
TSS
Core promoter
Enhancer region

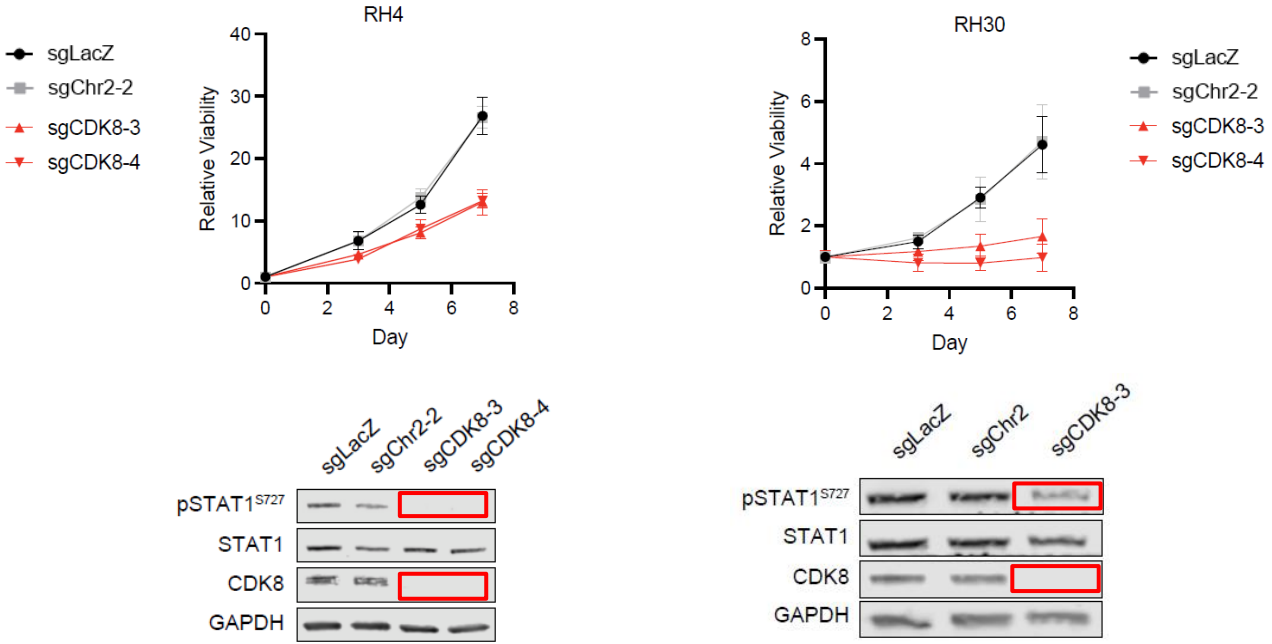
Expression of CDK8 in RMS



NCI OncoGenomics Database

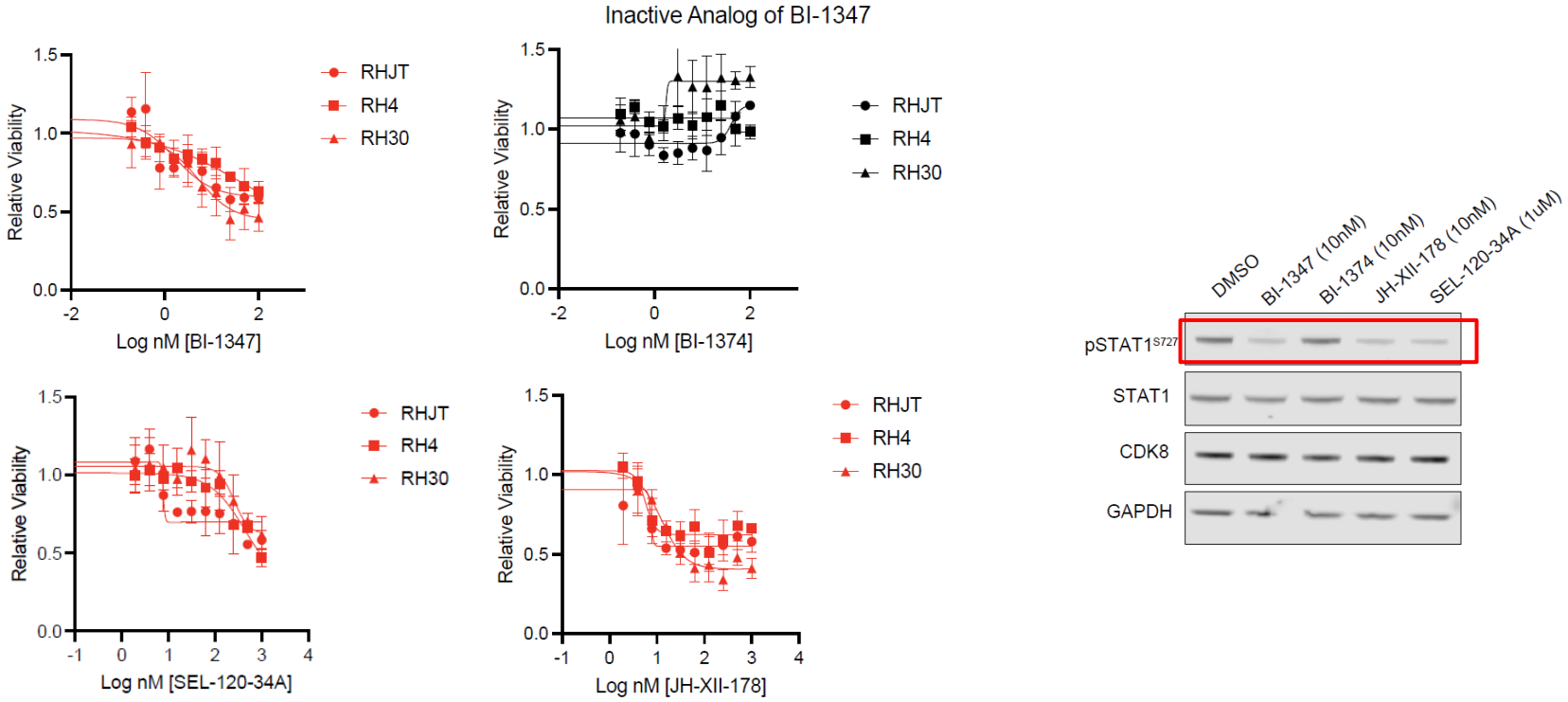


Genetic Loss of Function of CDK8 (CRISPR)

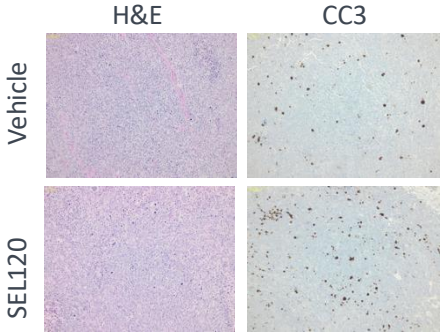


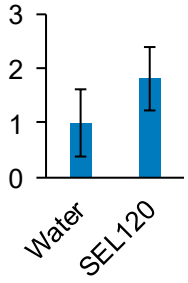
Phenocopied with RNAi

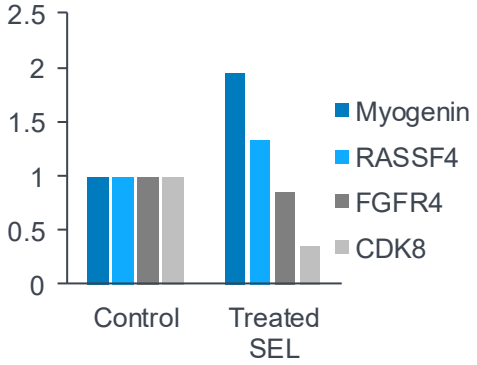
Pharmacologic Loss of Function of CDK8



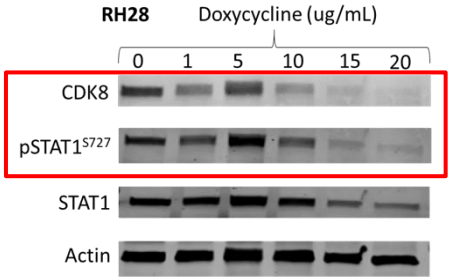
In Vivo Pharmacologic Inhibition

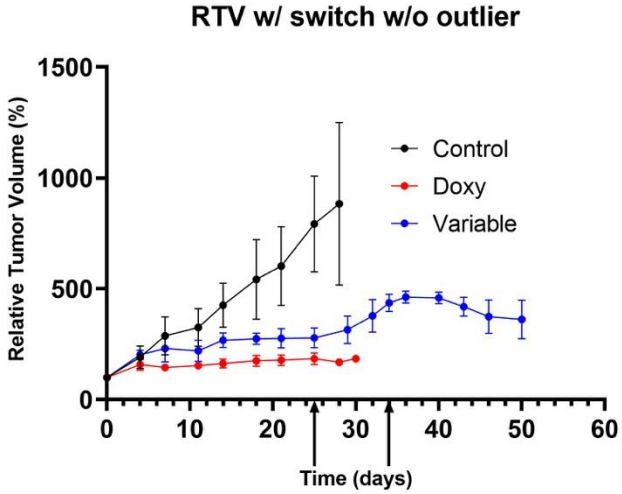


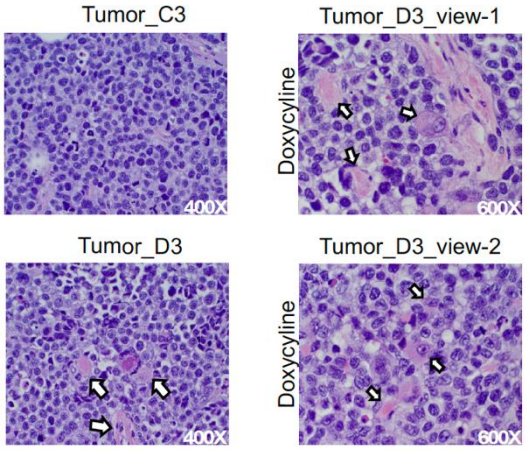




In Vivo Conditional Genetic Inhibition

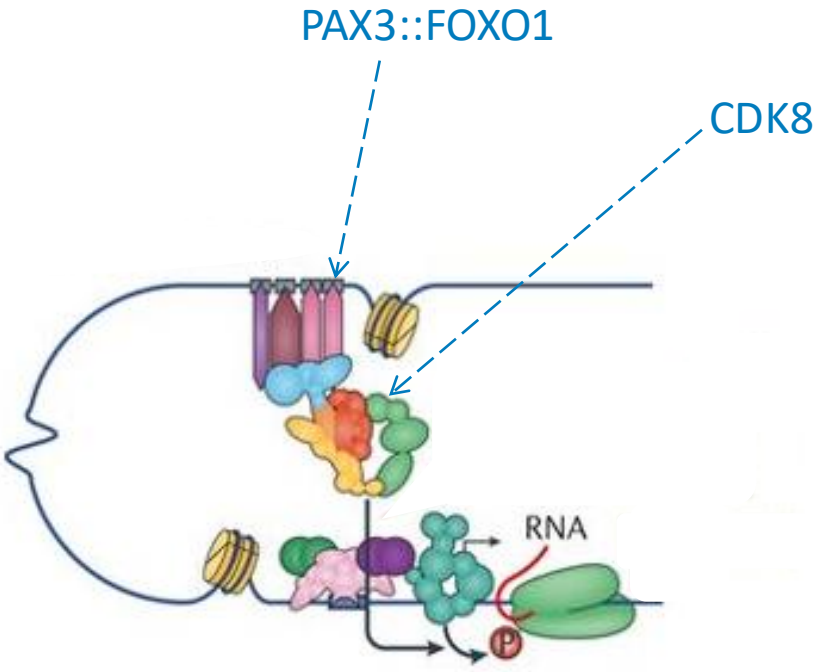


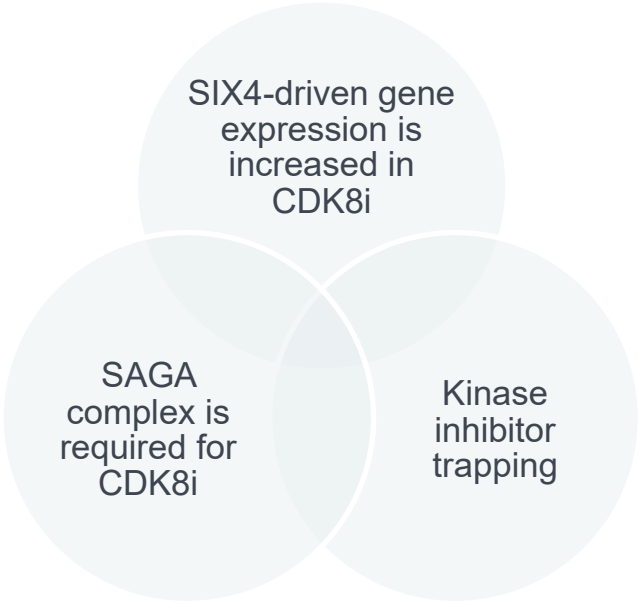




In Vitro Genetic Inhibition (RNA-seq)

Mechanism?





SAGA complex = large transcriptional coactivator complex, primarily functioning through histone acetylation and deubiquitylation

SIX4 = myogenic differentiation transcription factor

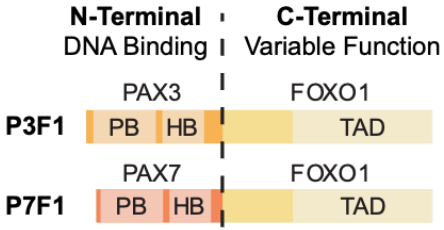
Translational Relevance

Drug	NCI Identifier	Disease target	Phase	Status	Single agent or combined?	Location
RVU120	NCT04021368	AML, MDS	I	Active, not recruiting	Single	USA
TSN084	NCT06386705	Malignant neoplasm	1	Recruiting	Single	China
TSN084	NCT05300438	Advanced malignant tumors	I	Active, not recruiting	Single	USA
RVU120	NCT06191263	Relapsed/refractory AML	2	Recruiting	Combo with venetoclax	Europe
RVU120	NCT06243458	Low risk MDS	2	Recruiting	Single	Europe
RVU120	NCT06268574	Relapsed/refractory AML	2	Recruiting	Single	Europe
RVU120	NCT06397313	Myelofibrosis	2	Recruiting	Combo with ruxolitinib	Europe

TYMS

A metabolic vulnerability

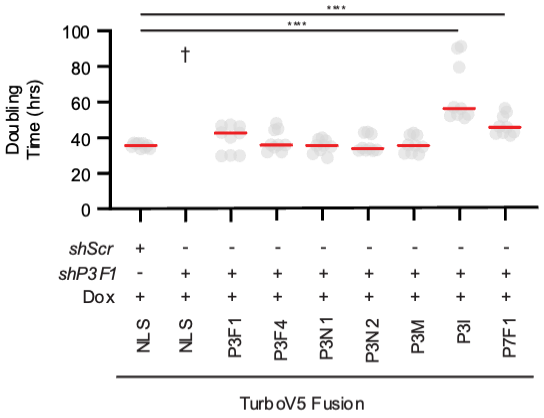
Project Premise: We Can Use RMS Oncofusion Interactions to Nominate Oncogenic Mechanisms



PB – Paired Box
HB – Homeobox
TAD – Transactivation Domain
HAT- Histone Acetyltransferase

Can Different Oncofusions Interchangeably Drive RMS?

Proliferation

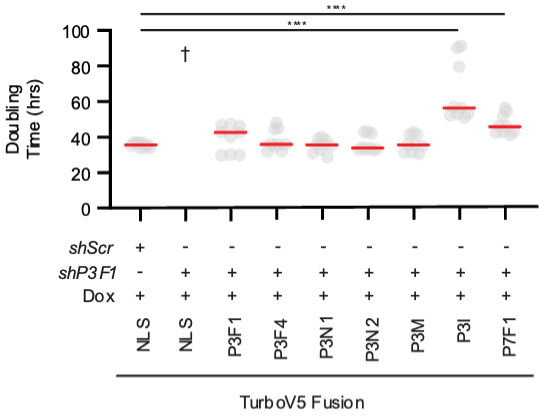


Chromatin interactions

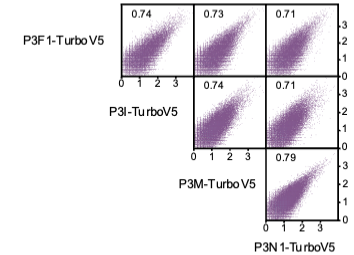
Transcription

Can Different Oncofusions Interchangeably Drive RMS?

Proliferation



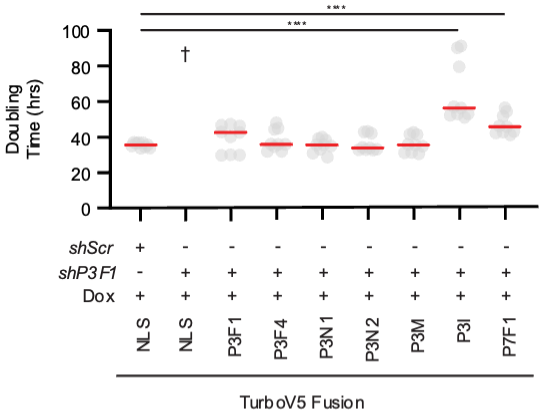
Chromatin interactions



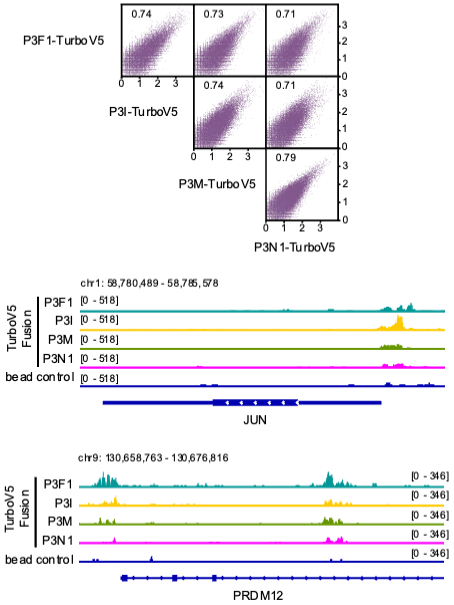
Transcription

Can Different Oncofusions Interchangeably Drive RMS?

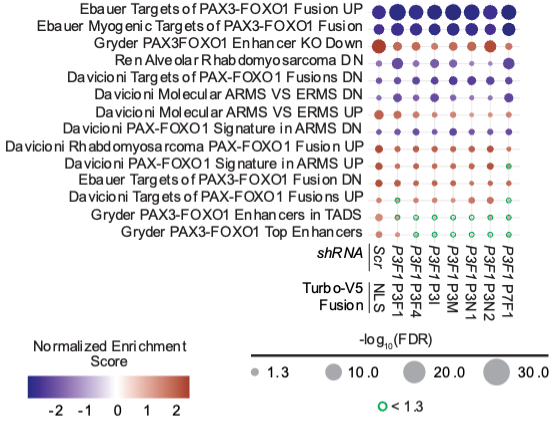
Proliferation



Chromatin interactions

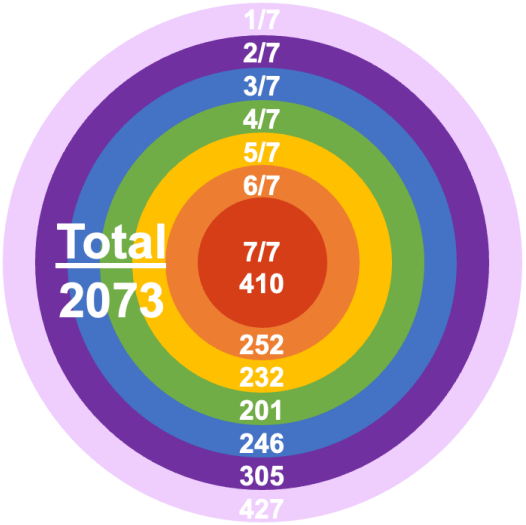


Transcription

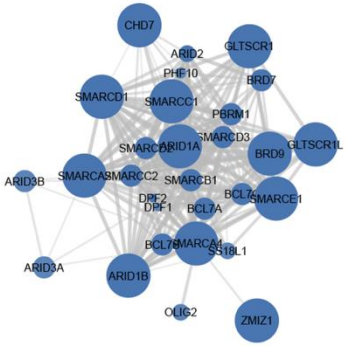


FP-RMS Fusion Proteins Share a Common Interactome

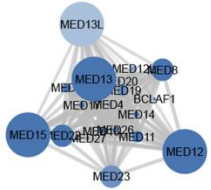
FC > 1.5 and P<0.05



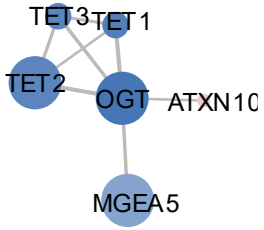
SWI/SNF Complex



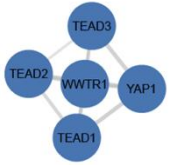
Mediator Complex

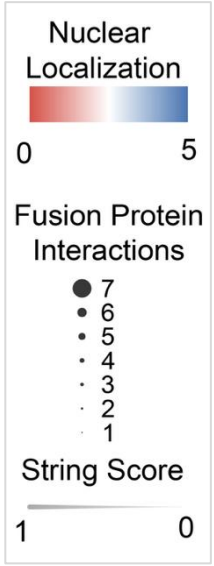


O-GlcNAcylation
DNA Demethylation



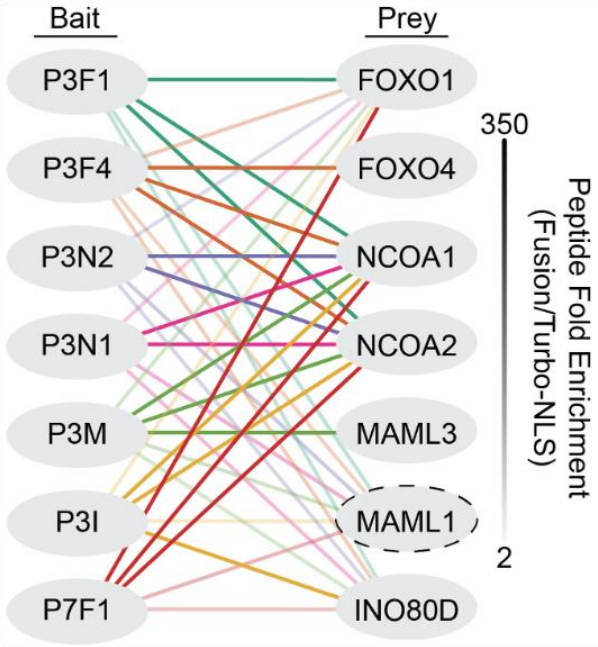
Hippo Signaling



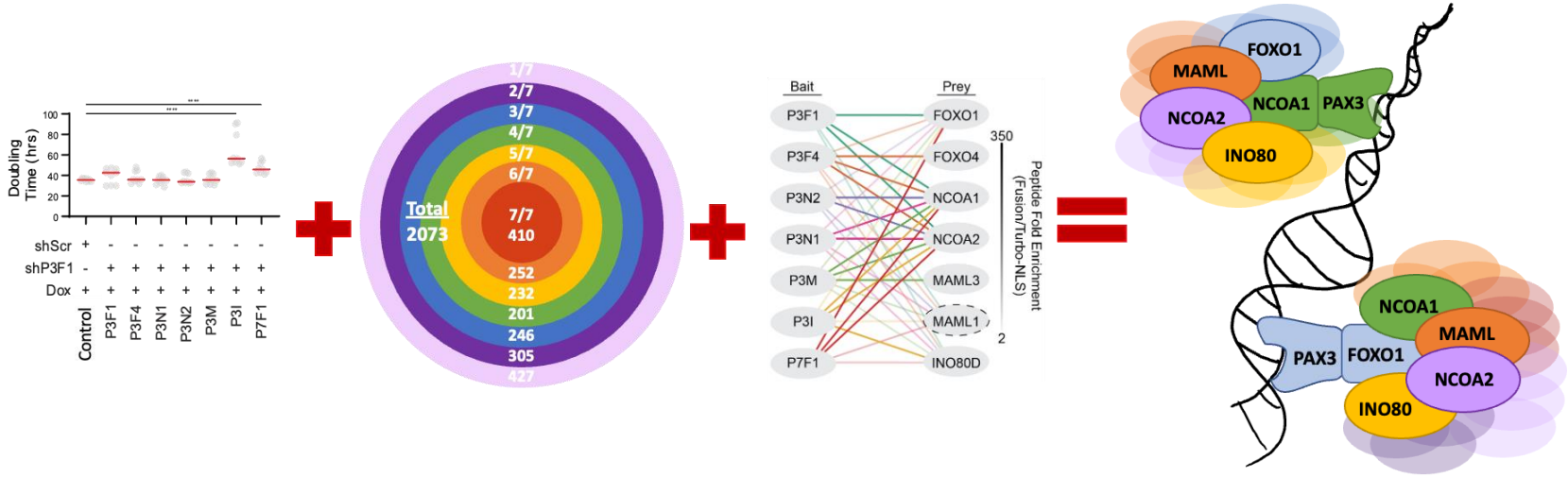


Seth Zimmerman et al, under review
Taulli R et al, *Oncogene*, 2014
Laubscher et al, *Nat Commun*, 2021
Bharathy N et al, *Oncogene*, 2022
Zhang et al, *Mol Cell*, 2022

FP-RMS Fusion Proteins Interact with Full-Length C-Terminal Fusion Partners

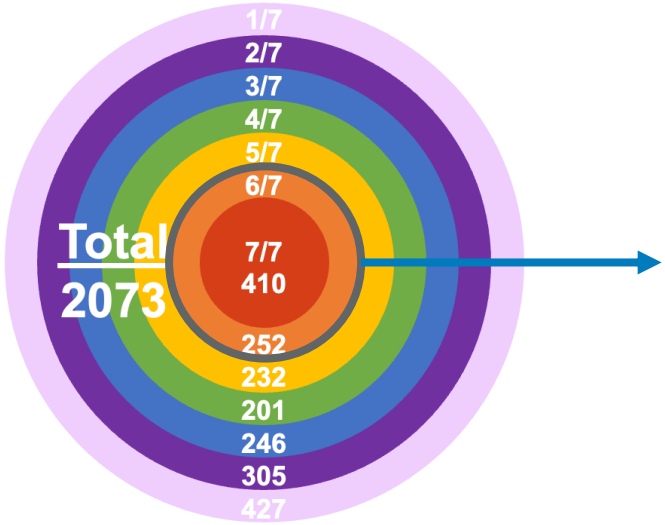


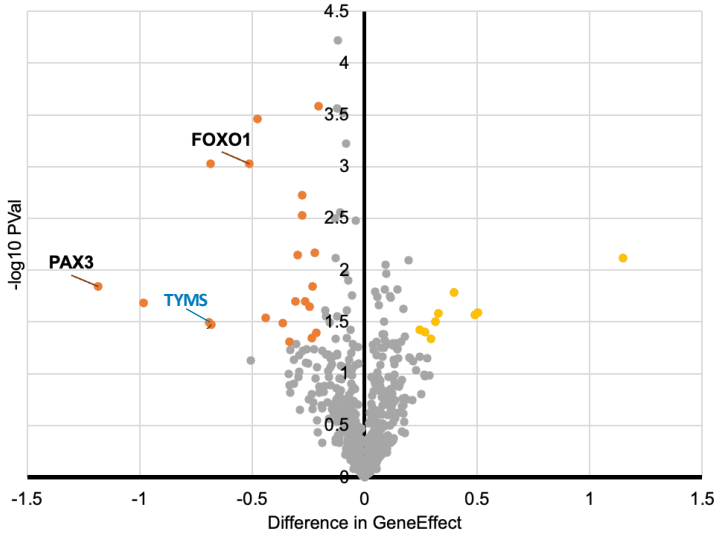
RMS Oncofusions Arise from PAX3/7 Fused to a Common Interactome Member Because They Mediate Common Interactions



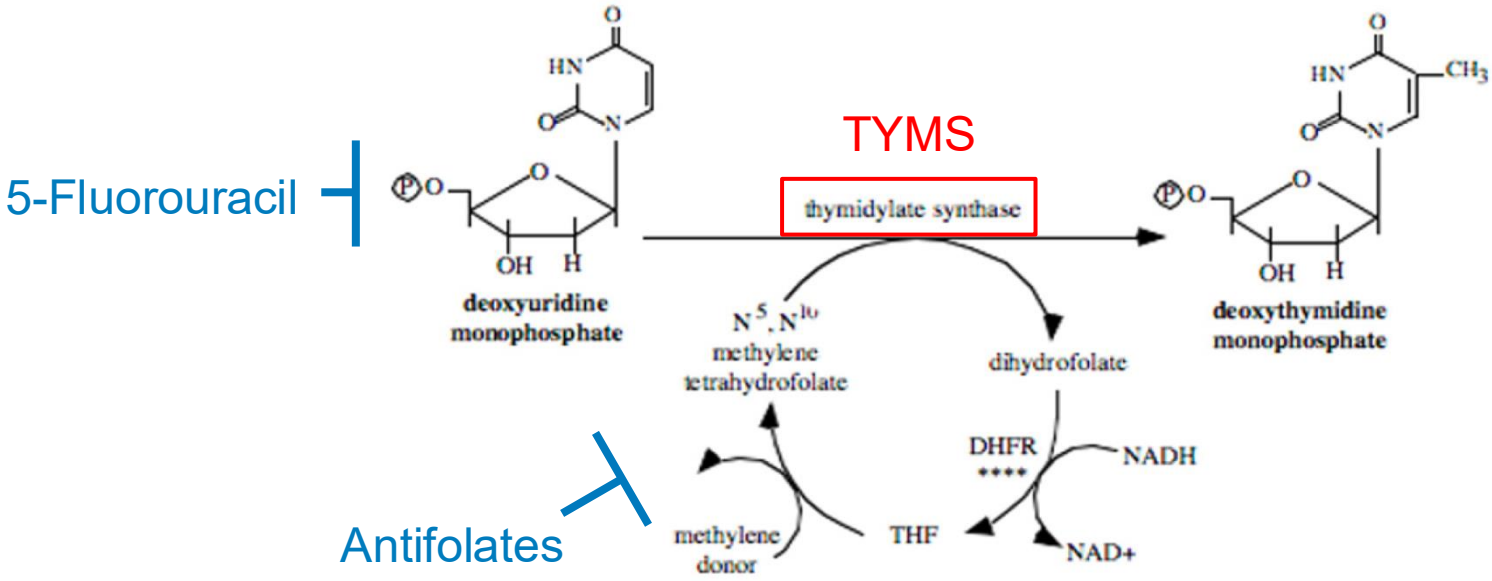
It's not the function of the C-terminal fusion that matters – it's the proteins that it interacts with

The 410 Common Proteins are Likely Most Relevant to Fusion Oncogenesis, and Therefore Linchpin Targets





Translational Relevance



History of Antifolates in Treatment of RMS

4 relapsed RMS patients, range from 25% tumor reduction to complete response

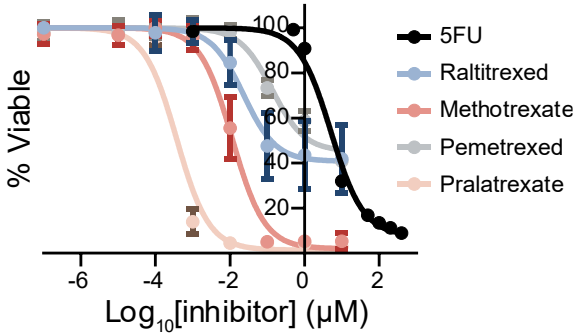
2 out of 4 ARMS showed stable disease or partial response

no responses in 8 RMS patients

Reasons for variable response?

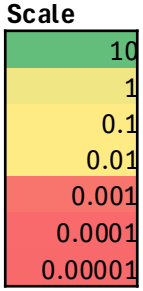
- Route of cellular entry: folate receptors vs channels
- Cell retention: polyglutamylation
- Affinity: for folate dependent enzymes

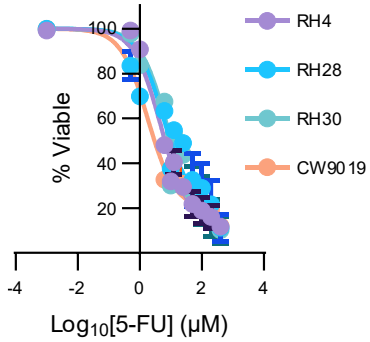
FP-RMS Cells are Sensitive to 5-FU and Antifolates In Vitro

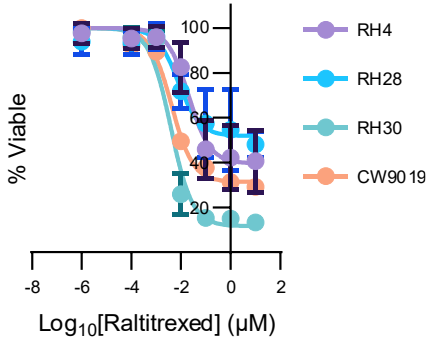


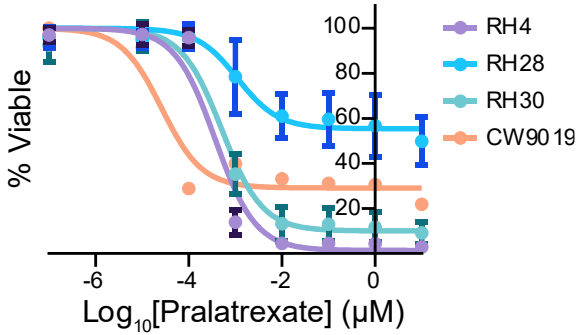
IC50 (μM)

Cell Line	5-FU	Raltitrexed	Pralatrexate
RH4	4.207	0.02085	0.00097118
RH28	5.8725	0.0115277	0.003859
RH30	7.085	0.003902	0.0008609
CW9019	1.831	0.005101	0.00002357



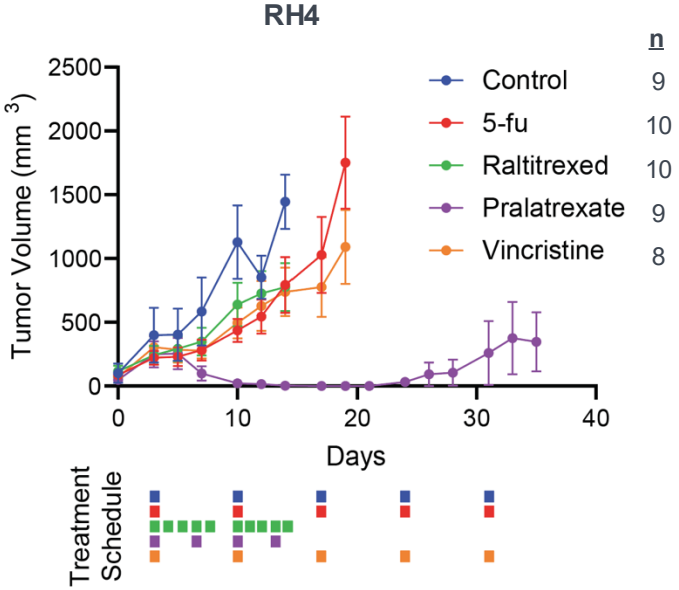


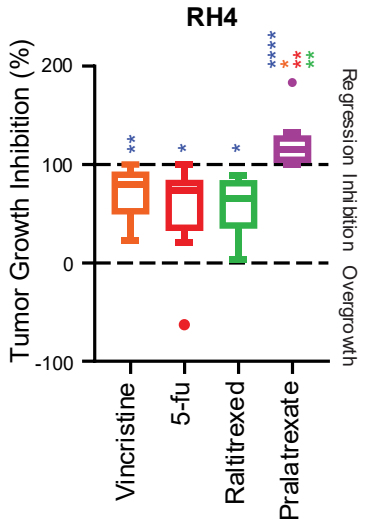


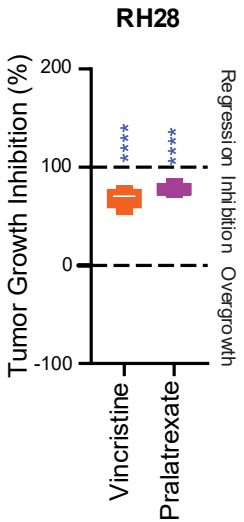


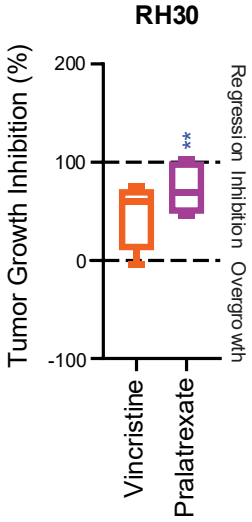
DO NOT POST

Pralatrexate Induces Tumor Growth Inhibition and Regression In Vivo







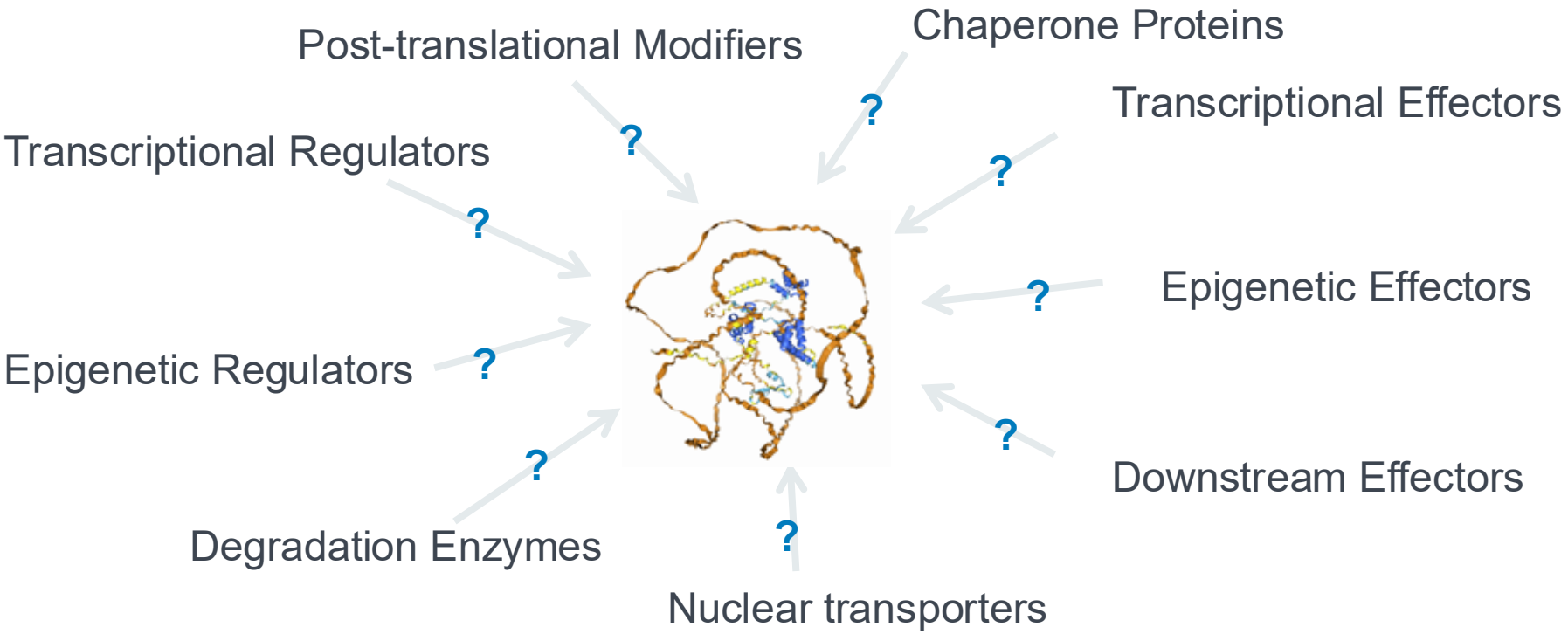


Pralatrexate: 2nd generation antifolate designed to get into cells better and stay in cells better
Approved by FDA in 2009 for peripheral T-cell lymphoma

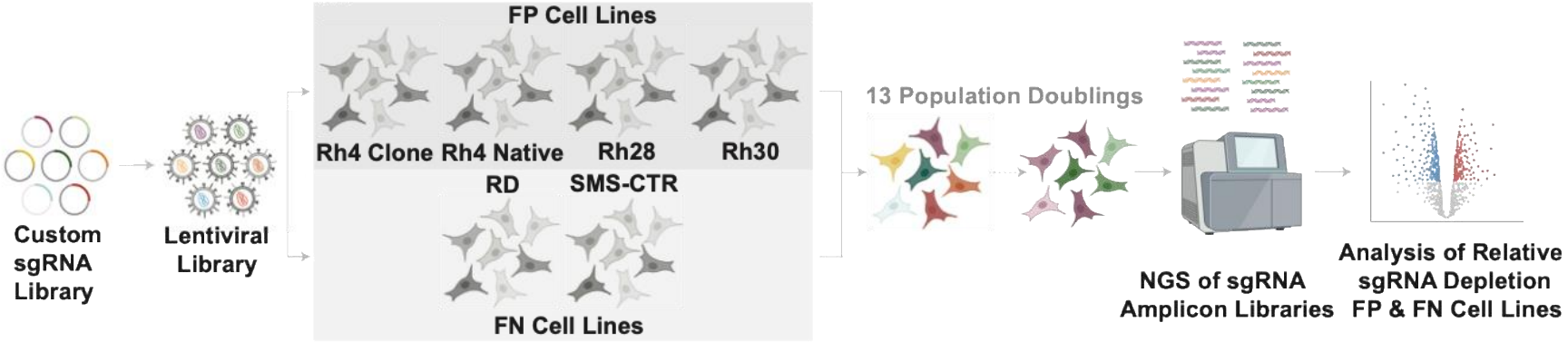
FANCM

A DNA damage vulnerability

Project Premise: We Can Use Unbiased Genomic Screens to Identify PAX3::FOXO1 Regulators



Custom sgRNA Sub-Library Screening Across FP and FN Cell Lines Identifies FP-Specific Genetic Dependencies



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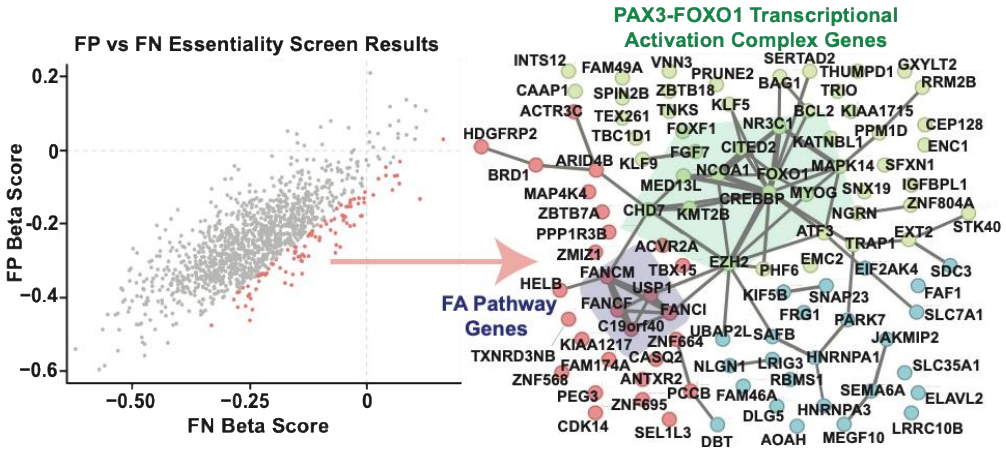
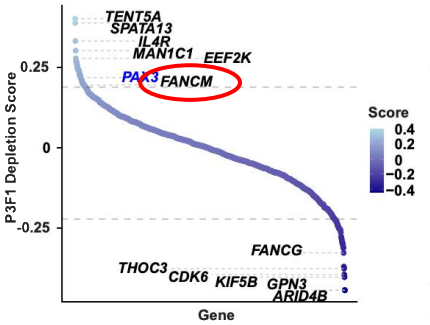
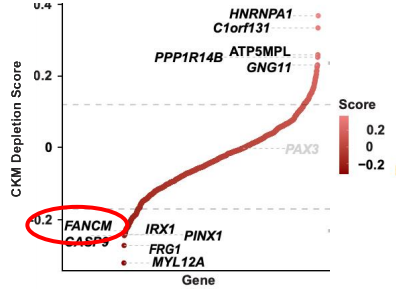


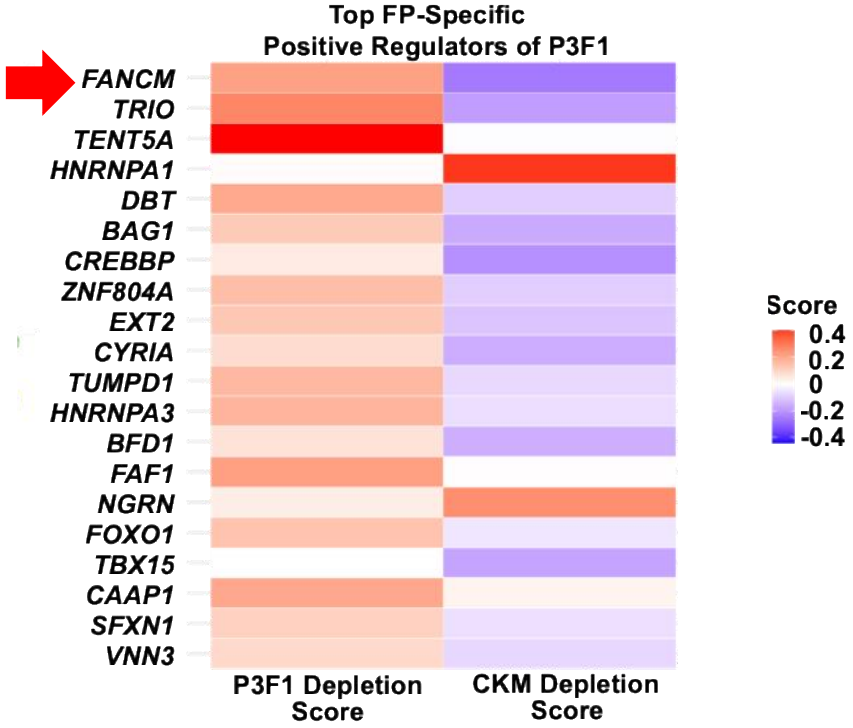
PAX3::FOXO1 expression flow screen



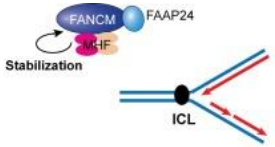
PAX3::FOXO1 differentiation screen



FANCM Is a Lead FP-Specific Positive Regulator of PAX3::FOXO1 Levels and a Negative Regulator of Myogenic Differentiation



FANCM Functions and the FA Pathway in RMS



> Clin Cancer Res. 2014 Jul 15;20(14):3884-95. doi: 10.1158/1078-0432.CCR-13-0556. Epub 2014 Apr 30.

FANCD2 is a potential therapeutic target and biomarker in alveolar rhabdomyosarcoma harboring the PAX3-FOXO1 fusion gene

Mamata Singh [1], Justin M Leasure [1], Christopher Chronowski [1], Brian Geier [2], Kathryn Bondra [1], Wenrui Duan [1], Lauren A Hensley [1], Miguel Villalona-Calero [1], Ning Li [1], Anthony M Vergis [1], Raushan T Kurmasheva [2], Changxian Shen [2], Gary Woods [2], Nikhil Sebastian [1], Denise Fabian [1], Rita Kaplon [1], Sue Hammond [2], Kamalakannan Palanichamy [1], Arnab Chakravarti [1], Peter J Houghton [3]

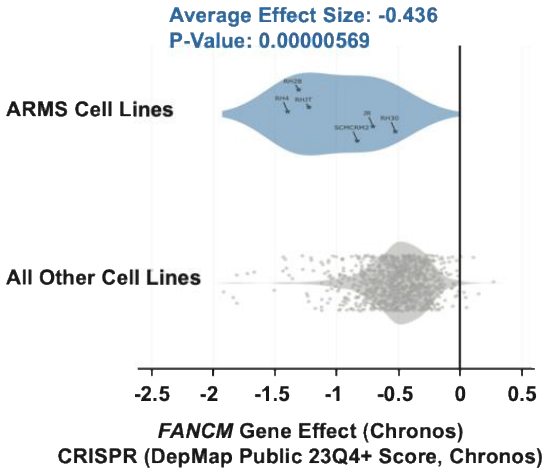
> Nat Commun. 2022 Jul 25;13(1):4297. doi: 10.1038/s41467-022-32023-7.

Therapeutic targeting of ATR in alveolar rhabdomyosarcoma

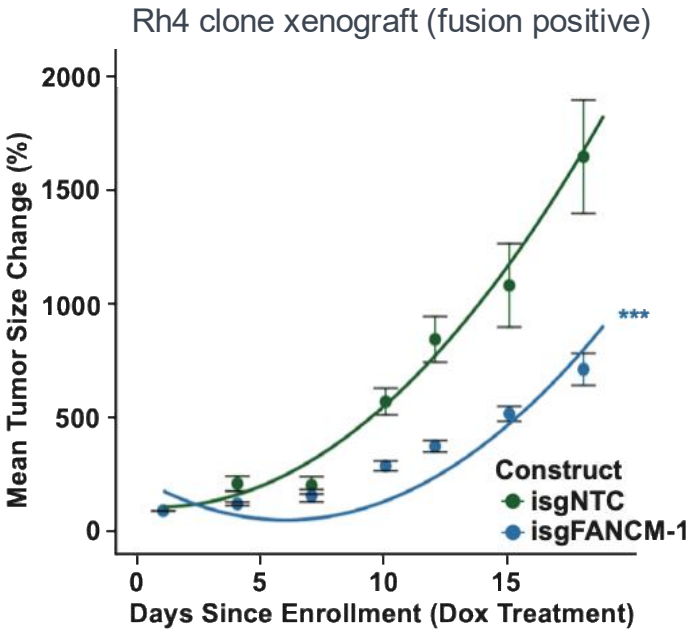
Heathcliff Dorado García [1 2 3], Fabian Pusch [1], Yi Bei [1 2 3], Jennifer von Stebut [1 2], Glorymar Ibáñez [4], Kristina Guillan [4], Koshi Imami [3], Dennis Gürgen [5], Jana Rolff [5], Konstantin Helmsauer [1 2], Stephanie Meyer-Liesener [1 3 6], Natalie Timme [2], Victor Bardinet [1 2], Rocío Chamorro González [1 2 3], Ian C MacArthur [2], Celine Y Chen [1 2], Joachim Schulz [2], Antje M Wengner [7], Christian Furth [2], Birgit Lala [2], Angelika Eggert [2], Georg Seifert [2], Patrick Hundsoerfer [2], Marieluise Kirchner [3 8], Philipp Mertins [3 8], Matthias Selbach [3], Andrej Lissat [2], Frank Dubois [9 10], David Horst [9], Johannes H Schulte [2], Simone Spuler [1 3 6 8 11], Daoqi You [4], Filemon Dela Cruz [4], Andrew L Kung [4], Kerstin Haase [1], Michela DiVirgilio [3], Monika Scheer [2], Michael V Ortiz [4], Anton G Henssen [12 13 14 15 16]

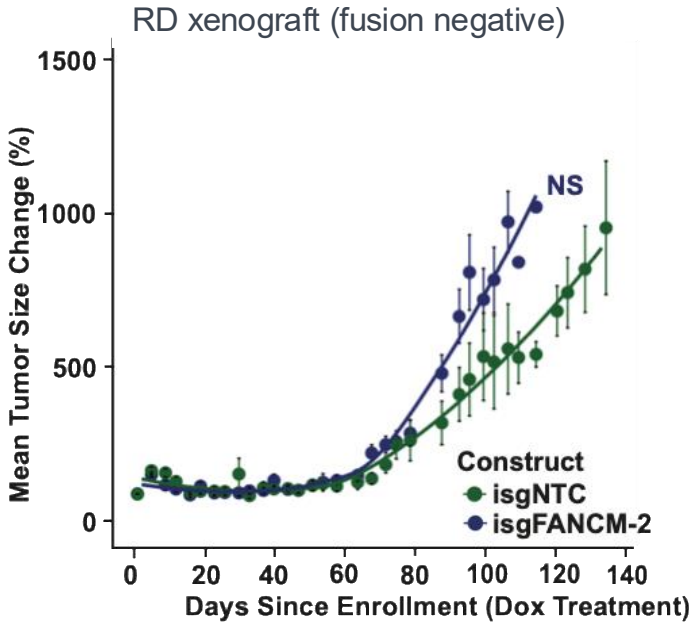
ICL = inter-strand crosslinks

FANCM Validates as a FP-Specific Dependency In Vitro



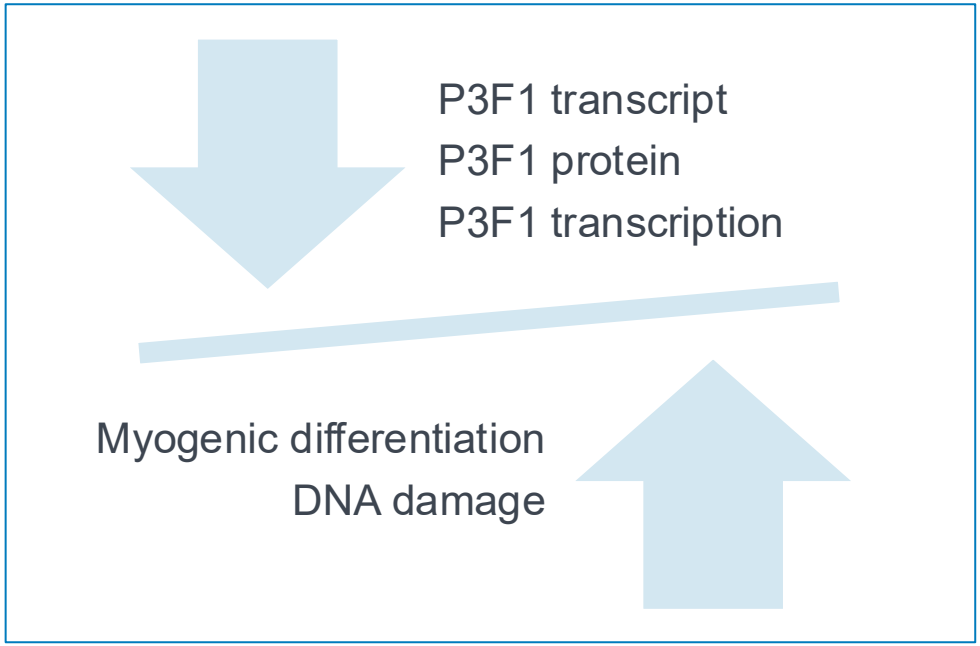
FANCM Validates as a FP-Specific Dependency In Vivo



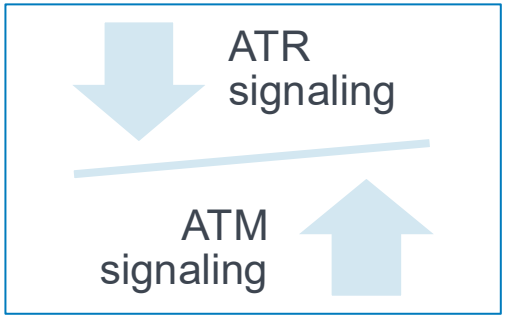


Additional Findings for FANCM Loss of Function

Observations



Mechanism



PAX3::FOXO1 Expression Induces Baseline Increase in DNA Damage, but also Replication Stress (RS)

› Nat Commun. 2022 Jul 25;13(1):4297. doi: 10.1038/s41467-022-32023-7.

Therapeutic targeting of ATR in alveolar rhabdomyosarcoma

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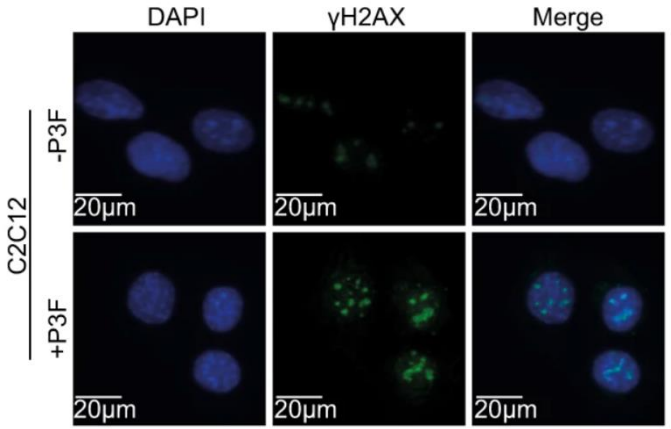
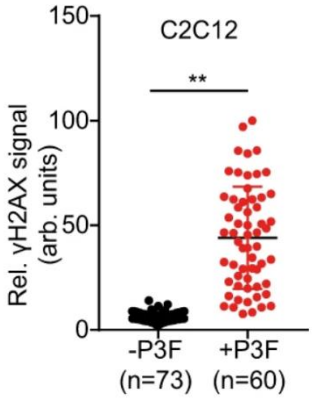
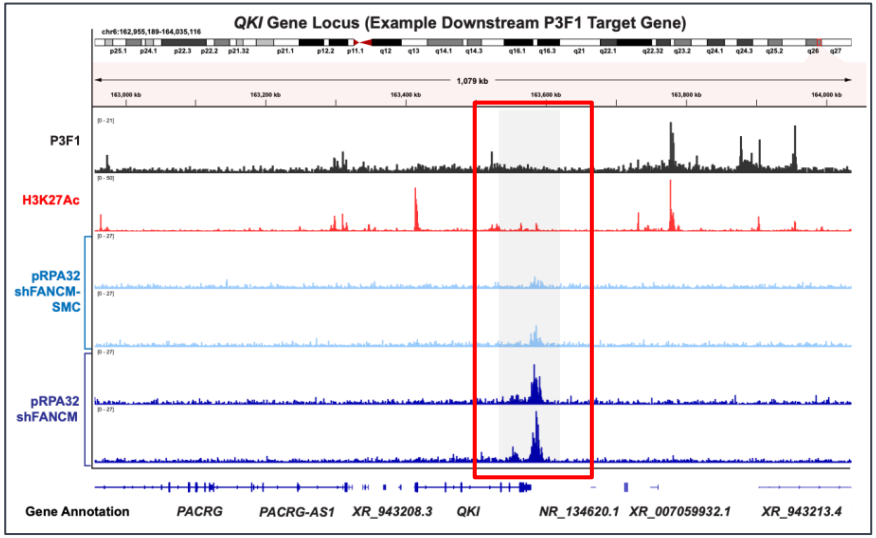
Ectopic PAX3::FOXO1 induces DNA damage



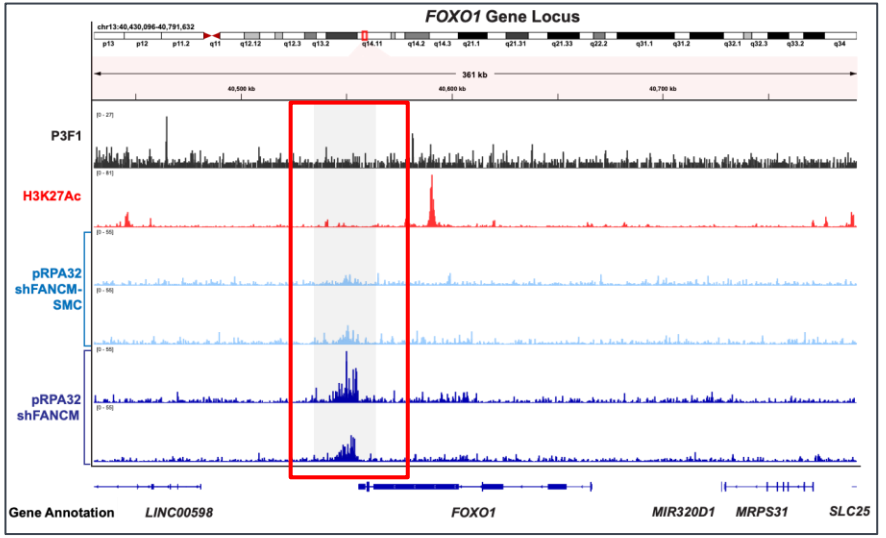


FANCM Loss Induces Increased RS via pRPA32 Signal at PAX3::FOXO1 Target Genes and PAX3::FOXO1 Itself!

pRPA32 occupancy at PAX3::FOXO1 target gene

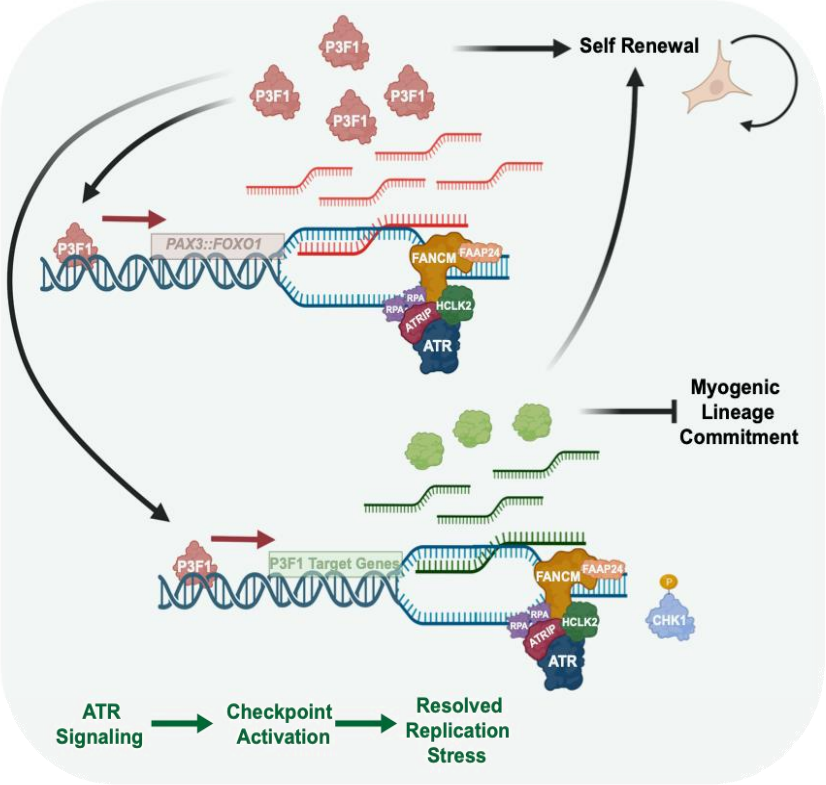


pRPA32 occupancy at PAX3::FOXO1 itself



H3K27Ac = active enhancers and promoters

Proposed Model of FANCM Regulation of FP-Specific Biology

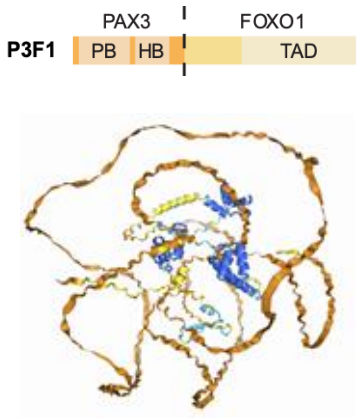


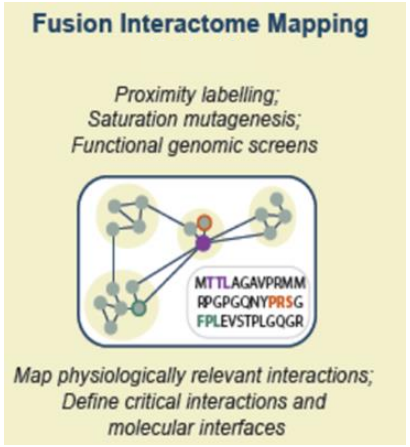
Translational Relevance

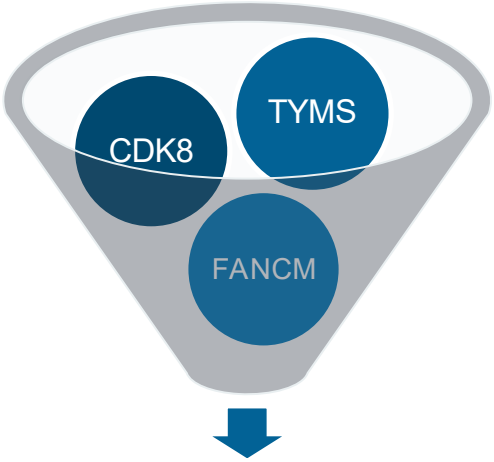
Novelty	• First time a FA pathway member is implicated as a vulnerability in FP RMS
Feasibility	• Therapeutic potential of targeting FANCM is promising, albeit limited at this stage (no specific compounds) • Normal human and mouse tissues tolerate biallelic *FANCM* loss
Future	• Need to study FANCM inhibitors in FP-RMS and how these future compounds may synergize with clinically viable ATR and ATM inhibitors

Takeaway

FusOnC2 Initiative for fusion-positive childhood cancers







CCDI initiative

Collect
Integrate
Share
Translate

Acknowledgements

Duke Research collaborations

- Chris Counter lab
- Kris Wood lab
- Lee Zou lab
- Rex Bentley (Pathology)
- Becca Morecci (U54 coordinator)

Additional NCI and Moonshot collaborations

- Keren Witkin (U54 PO)
- Angela Koehler lab (MIT)
- Dave Root lab (Broad)
- Dave Langenau lab (MGH)
- Jack Shern lab (NCI)
- Javed Khan lab (NCI)
- Fred Barr lab (NCI)
- Craig Thomas (NCATS)
- Kim Stegmaier lab (DFCI)

COG collaborations

- COG Soft Tissue Sarcoma Committee
- COG STS PathBio Subcommittee
- APEC14B1-MCI Committee
- Erin Rudzinski
- Mike Arnold
- Philip Lupo
- Nationwide Biopathology Center
- Nationwide Institute of Genomic Medicine

International RMS collaborations

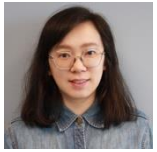
- Simone Hettmer lab
- Rosella Rota lab
- Janet Shipley lab
- Anna Kelsey pathology
- INSTRuCT Committee
- Suzi Birz INSTRuCT coordinator

RMS interactome project and subproject leads

- Seth Zimmerman (Counter)
- Susu Zhang (Stegmaier)
- Chris Delaney (Wood)

Duke Cancer Institute

Patients and their families



Q&A

Childhood Cancer Data Initiative Virtual Symposium Series

Opportunities to Expand Patient/Family Engagement in the Genomic Characterization of Pediatric Malignancies

Ann Ramer, M.P.H.

NIH NATIONAL CANCER INSTITUTE

Why Does Improving Patient Engagement in Molecular Characterization Matter?

- **Advanced diagnostics support clinical care for the patient.**
  - Targeted therapies: both U.S. Food and Drug Administration (FDA)-approved drugs and those in clinical trials.
  - Identify high risk patients and potentially modify clinical treatment.

- **Additional patient and family benefits from the use of molecular characterization.**
  - Identify those with familial cancer syndromes.
  - Initiate screening protocols and prevention.

Why Does Improving Patient Engagement in Molecular Characterization Matter?

- **Molecular Characterization Initiative (MCI) supports research and drug development:**
  - MCI linked with Project:EveryChild.
  - Data is centralized and available to researchers through CCDI.

How Can We Increase the Number of Patients That Receive Molecular Characterization Testing? Key Components:

- **Education.**
- **Referral.**
- **Access.**
  - Ensure that clinical trials and research initiatives include Clinical Laboratory Improvement Amendments (CLIA)-approved testing and return of results.
  - Advocate for insurance coverage for commercial testing.

Key Partners: Providers

- **Single most important partner for education and referrals.**
- **Without a referral, none of the patient barriers can be identified or addressed.**
- **Most trusted source of information.**

Key Partners: Disease-Specific Support Organizations

- **Online support that is provided by nonprofit organizations is often a place where the newly diagnosed gather.**
  - We can do a better job of educating the newly diagnosed through partnerships with the nonprofit organizations that moderate these trusted forums.
  - Many organizations also support registries or research initiatives.
  - Examples: Making It Better (MIB) Agents, Living LFS, Pediatric Brain Tumor Foundation (PBTF).

Key Partners: Centers for Disease Control and Prevention (CDC) Comprehensive Cancer Control Program

- **Focus on preventing and controlling cancer**
  - Reduce incidence and mortality using evidence-based strategies that promote healthy behaviors, increase screening rates and access to care.
- **Community-specific plans**
  - Address the unique cancer burdens in a geographic area, using data to inform strategies.
- **Collaboration**
  - The grant supports collaboration between public health agencies, providers and community groups.

Comprehensive Cancer Control
Collaborating to Conquer Cancer

CDC Comprehensive Cancer Control State Cancer Plans: Challenges for Pediatrics

- **Content, structure, timeframe and strategies vary widely from state to state.**
- **Childhood cancer is mentioned in only about half of state cancer plans.**
- **Only 15 states have robust language and well-defined goals and objectives that address the needs of children with cancer.**

State Cancer Plans: Still an Effective Option

- **There are common goals that can be on-ramps for pediatric representation in plans that do not have specific language for children:**
  - Increasing clinical trial participation or research studies.
  - Improving survivorship.
  - Supporting palliative care in cancer.
  - Improving quality of life for those diagnosed with cancer.

Health Resource and Service Administration (HRSA) Title V Grants

- **Like the CDC Comprehensive Cancer Control Program, all states have a HRSA Title V Maternal Child Health Services block grant.**
  - 30% of the block grant is earmarked to serve Children and Youth with Special Health Care Needs (CYSHCN).
  - There is existing state infrastructure for CYSCHN.
  - The work supported by Title V grant, can be aligned with the pediatric work done under the CDC state cancer grant.

Title V Maternal and Child Block Grant

CDC Comprehensive Cancer Control Grant

Children and Youth with Special Health Care Needs (CYSHCN) 30%

State Cancer Plans: Ohio Strategies

Ohio has focused on no or low-cost strategies for improving clinical care, increasing clinical trial participation, and supporting research.

- **Awareness and Education** about Molecular Characterization.
  - 2023 Summit speakers: Dr. Elaine Mardis, Dr. Katherine Janeway, Dr. Adam Resnick.
- **Data Collection:** How often are children receiving molecular characterization?
  - 2025 Needs Assessment Survey.
  - Data from MCI to track Ohio's participation.
  - Exploring ways to capture referral data from hospitals about other avenues for advanced diagnostics (via commercial testing and other research protocols).

State Cancer Plans: Ohio Strategies

- **Support research and clinical trial participation.**
  - MCI and Project:EveryChild are clinical trials at baseline.
  - Advanced diagnostics are necessary to determine eligibility for targeted therapy whether in a clinical trial or with FDA-approved drug.

State Cancer Plans: Ohio Strategies

- **Identify and support those with germline findings.**
  - **Complex Medical Help (CMH) Program**

    Supported in part by Title V funding, CMH was leveraged in Ohio to address the financial barrier for screening protocols for children found to have a genetic predisposition. With CMH as the payer of last resort, families are found financially harmless.
  - **Early and Periodic Screening, Diagnostic and Treatment (EPSDT)** is a Medicaid benefit that provides comprehensive and preventive healthcare services for children under age 21 who are enrolled in Medicaid. EPSDT is key to ensuring that children and adolescents receive appropriate preventive, dental, mental health, and specialty services.

Conclusion

- **Identify essential needs**
  - Education, referrals, and access
- **Engage with key partners**
  - Providers, nonprofit organizations, state cancer collaboratives
- **Combine existing resources**
  - Title V funding and infrastructure
  - CDC National Comprehensive Cancer Control Program
  - Medicaid EPSDT
- **Collect data to measure progress and impact**

Q&A

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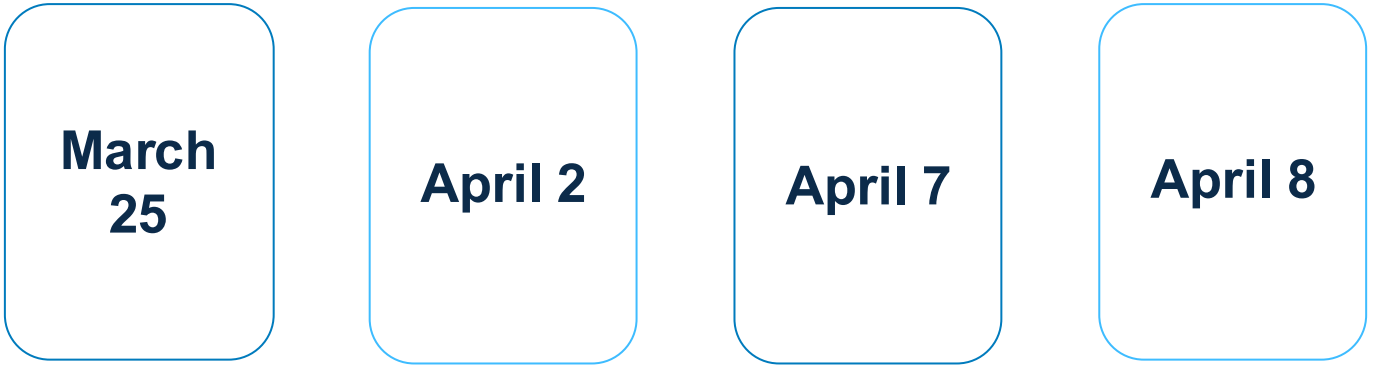

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