

UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): July 14, 2026

SYNDAX PHARMACEUTICALS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware
(State or Other Jurisdiction
of Incorporation)

001-37708
(Commission File Number)

32-0162505
(IRS Employer
Identification No.)

730 THIRD AVENUE
FLOOR 9

NEW YORK, New York
(Address of Principal Executive Offices)

10017
(Zip Code)

Registrant's Telephone Number, Including Area Code: (781) 419-1400

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

- ☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- ☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- ☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- ☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock	SNDX	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01. Other Events.

On July 14, 2026, Syndax Pharmaceuticals, Inc. (the “*Company*”) issued a press release and presentation highlighting its late-stage programs and the next chapter of its R&D strategy, including new pipeline assets, during its R&D event held on July 14, 2026. A copy of the press release and presentation are furnished as Exhibits 99.1 and 99.2 to this Current Report on Form 8-K and are incorporated herein by reference.

The information contained in this Item 8.01 and in Exhibits 99.1 and 99.2 attached hereto is intended to be furnished and shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “*Exchange Act*”), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference into any of the Company’s filings under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

<u>Exhibit No.</u>	<u>Description</u>
99.1	Press Release, dated July 14, 2026
99.2	Corporate Presentation, dated July 14, 2026
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

SYNDAX PHARMACEUTICALS, INC.

By: /s/ Michael A. Metzger
Michael A. Metzger
Chief Executive Officer

Dated: July 14, 2026

Syndax to Highlight Late-Stage Programs and Unveil New Pipeline Assets at R&D Event

- *Expands pipeline with SNDX-4321, a novel, mutant-selective, allosteric EGFR inhibitor designed to address NSCLC patient populations with significant unmet needs –*
- *SNDX-62122, a next-generation menin inhibitor, selected for development in MF, building on compelling preclinical results observed with revumenib in MF –*
- *SNDX-62122 is the first candidate from the Company's library of internally developed and wholly owned next-generation menin inhibitors that the Company plans to deploy in new areas –*
- *Phase 2 trial of axatilimab in IPF on track for topline data in 4Q26 –*
- *R&D Event featuring three thought leaders and members of Syndax's leadership team to be held today at 8:30 a.m. ET –*

NEW YORK, July 14, 2026 (GLOBE NEWSWIRE) – Syndax Pharmaceuticals (Nasdaq: SNDX), a commercial-stage biopharmaceutical company advancing innovative cancer therapies, today announced that the Company will highlight its late-stage programs and the next chapter of its R&D strategy, including new pipeline assets, during its R&D Event being held today from 8:30 a.m. to 11:00 a.m. EDT.

"Today we are unveiling our new pipeline assets which reflect the continued evolution of our R&D capabilities, including our ability to leverage both external innovation and a library of internally developed next-generation menin inhibitors to expand our portfolio and create new growth opportunities," said Michael A. Metzger, Chief Executive Officer. "With a robust financial foundation, a track record of success, and multiple near-term catalysts, Syndax is positioned to deliver the next breakthroughs for patients and drive long-term value for shareholders."

"Syndax has established world-class R&D capabilities and strong partnerships with leading clinicians and scientists around the world to drive innovation and develop transformative new medicines for patients," said Nick Botwood, MBBS, Head of Research & Development and Chief Medical Officer at Syndax. "Today's event will highlight our strategy to expand our leadership in menin and CSF-1R inhibition and build our pipeline with two new differentiated assets supported by compelling preclinical data and mechanistic insights. With both Revuforj and Niktimvo, we demonstrated our ability to efficiently generate clinical data that validates new therapeutic targets, a strength we look forward to showcasing again as we advance the next chapter of our R&D strategy."

Guest speakers will include:

- **Toby M. Maher, MD, PhD**, Professor of Clinical Medicine and Director of Interstitial Lung Disease, Keck Medical School of University of Southern California, will present on idiopathic pulmonary fibrosis (IPF) and the potential for axatilimab in IPF.
- **John D. Crispino, PhD, MBA**, Director, Division of Experimental Hematology, St. Jude Children's Research Hospital, will present on the potential for menin inhibition in myelofibrosis (MF).
- **Michael J. Eck, MD, PhD**, Professor, Department of Cancer Biology, Dana-Farber Cancer Institute and Professor, Department of Biological Chemistry & Molecular Pharmacology, Harvard Medical School, will present on the development of SNDX-4321 for EGFR-mutated (EGFRm) non-small cell lung cancer (NSCLC).

Highlighted R&D Day Topics

SNDX-4321, a novel, mutant-selective, allosteric EGFR inhibitor for NSCLC

- SNDX-4321 is an EGFR inhibitor in development for NSCLC patient populations with significant unmet needs, such as those with L858R mutations, CNS metastases, atypical activating mutations, or acquired resistance to current therapies. In contrast to ATP-site directed third and fourth generation EGFR inhibitors, SNDX-4321 is a novel allosteric inhibitor which binds at a pocket adjacent to the ATP site that is only accessible in the presence of L858R and certain other EGFR mutations. The Company expects to submit an investigational new drug (IND) application for SNDX-4321 by the end of 2026 and to initiate a Phase 1 trial in EGFRm NSCLC in 2027. SNDX-4321 is an externally developed molecule that the Company has an exclusive worldwide license to develop and commercialize. During the R&D Event, the Company will highlight the potential advantages of an allosteric approach in EGFRm NSCLC and the supporting preclinical data.

SNDX-62122, a candidate from the Company's library of internally developed next-generation menin inhibitors, selected for development in MF

- SNDX-62122 is the first candidate from a library of wholly owned next-generation menin inhibitors that the Company intends to advance into new areas.
- SNDX-62122 is in development for MF, with submission of an IND and initiation of a Phase 1 trial in MF expected in 2027. During the R&D Event, the Company will highlight recently published revumenib preclinical data showing that menin is a novel dependency in

proliferative megakaryocytes, major drivers of MF. The Company expects the development of SNDX-62122 to be informed and de-risked by a Phase 1/2 proof-of-principal trial of revumenib in MF that is expected to initiate in the fourth quarter of 2026 with initial clinical data expected in the second half of 2027.

Ongoing late-stage trials of Revuforj® (revumenib) and Niktimvo™ (axatilimab-csfr)

- During the R&D event, the Company will highlight its late-stage revumenib and axatilimab programs in newly diagnosed acute leukemia and chronic GVHD, respectively, as well as the Phase 2 trial of axatilimab in IPF that is expected to readout in the fourth quarter of 2026. During the R&D event, Dr. Toby M. Maher will discuss the unmet patient needs, evolving treatment landscape, and the potential for axatilimab's mechanism in IPF.

Webcast

Syndax will host an R&D Event today, July 14, 2026, from 8:30 a.m. to 11:00 a.m. ET in person and via webcast. The live audio webcast and accompanying slides may be accessed through the Events & Presentations page in the Investors section of the Company's website. Alternatively, the conference call may be accessed through the following link: <https://sndx-rdevent-2026.open-exchange.net/>.

For those unable to join the live webcast, a replay will be available in the Investors section of the Company's website at <https://syndax.com/> after the event and will be available for a limited time.

About Syndax

Syndax Pharmaceuticals is a commercial-stage biopharmaceutical company advancing innovative cancer therapies. Highlights of the Company's pipeline include Revuforj® (revumenib), an FDA-approved menin inhibitor, and Niktimvo™ (axatilimab-csfr), an FDA-approved monoclonal antibody that blocks the colony stimulating factor 1 (CSF-1) receptor. Fueled by our commitment to reimagining cancer care, Syndax is working to unlock the full potential of its pipeline and is conducting several clinical trials across the continuum of treatment. For more information, please visit www.syndax.com or follow the Company on X and LinkedIn.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "anticipate," "believe," "could," "estimate," "expects," "intend," "may," "plan," "potential," "predict," "project," "should," "will," "would" or the negative or

plural of those terms, and similar expressions (as well as other words or expressions referencing future events, conditions or circumstances) are intended to identify forward-looking statements. These forward-looking statements are based on Syndax's expectations and assumptions as of the date of this press release. Each of these forward-looking statements involves risks and uncertainties. Actual results may differ materially from these forward-looking statements. Forward-looking statements contained in this press release include, but are not limited to, statements about the progress, timing, clinical development and scope of clinical trials, the reporting of clinical data for Syndax's product candidates, the acceptance of Syndax and its partners' products in the marketplace, sales, marketing, manufacturing and distribution requirements, and the potential use of its product candidates to treat various cancer indications and fibrotic diseases. Many factors may cause differences between current expectations and actual results, including: unexpected safety or efficacy data observed during preclinical or clinical trials; clinical trial site activation or enrollment rates that are lower than expected; changes to Revuforj's or Niktimvo's commercial availability; changes in expected or existing competition; changes in the regulatory environment; failure of Syndax's collaborators to support or advance collaborations or product candidates; and unexpected litigation or other disputes. Other factors that may cause Syndax's actual results to differ from those expressed or implied in the forward-looking statements in this press release are discussed in Syndax's filings with the U.S. Securities and Exchange Commission, including the "Risk Factors" sections contained therein. Except as required by law, Syndax assumes no obligation to update any forward-looking statements contained herein to reflect any change in expectations, even as new information becomes available.

Syndax Contact

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Syndax Pharmaceuticals, Inc.
sklahre@syndax.com
Tel 781.684.9827

SNDX-G



Reimagining Cancer Treatment

Exhibit 99.2

Syndax 2026 R&D Event

July 14, 2026



Forward-looking statements disclosure

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "may," "will," "expect," "plan," "anticipate" and similar expressions (as well as other words or expressions referencing future events, progress, timing or circumstances) are intended to identify forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding future operations, financial results and the financial condition of Syndax Pharmaceuticals, Inc. ("Syndax" or the "Company"), including financial position, strategy and plans, the progress, timing, clinical development and scope of clinical trials and the reporting of clinical data for Syndax's product candidates, the progress of regulatory submissions and approvals and subsequent commercialization and the potential use of Syndax's product candidates to treat various cancer indications and fibrotic diseases, and Syndax's expectations for liquidity and future operations, are forward-looking statements. Many factors may cause differences between current expectations and actual results, including unexpected safety or efficacy data observed during preclinical studies or clinical trials, clinical site activation rates or clinical trial enrollment rates that are lower than expected; changes in expected or existing competition; the impact of macroeconomic conditions (the Russia-Ukraine war, inflation, among others) on Syndax's business and that of the third parties on which Syndax depends, including delaying or otherwise disrupting Syndax's clinical trials and preclinical studies, manufacturing and supply chain, or impairing employee productivity; failure of our collaborators to support or advance collaborations or product candidates and unexpected litigation or other disputes. Moreover, Syndax operates in a very competitive and rapidly changing environment. Other factors that may cause our actual results to differ from current expectations are discussed in Syndax's filings with the U.S. Securities and Exchange Commission, including the "Risk Factors" sections contained therein. New risks emerge from time to time. It is not possible for Syndax's management to predict all risks, nor can Syndax assess the impact of all factors on its business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statement. In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated or implied. Except as required by law, neither Syndax nor any other person assumes responsibility for the accuracy and completeness of the forward-looking statements. Syndax undertakes no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in Syndax's expectations.

Syndax is delivering for patients and driving substantial long-term value



TRACK RECORD
OF SUCCESS

Proven ability to translate scientific discoveries into novel therapies, with 3 FDA approvals between 2 drugs and thousands of patients treated



WORLD-CLASS R&D
CAPABILITIES

Fully integrated biotech with deep expertise and strong partnerships with leading researchers



DEEP AND GROWING
PIPELINE

Robust late- and early-stage pipeline with multiple near-term catalysts in 2H26 and blockbuster opportunities



SOLID FINANCIAL
POSITION

Driving towards profitability with growing revenues; fully funded to advance late-stage trials and pipeline assets

Advancing the standard of care for R/R NPM1m AML and KMT2Ar acute leukemia and chronic GVHD with two first- and best-in-class medicines



First and only menin inhibitor
with multiple indications



First and only CSF-1R-blocking
antibody approved in R/R cGVHD

Revuforj® (revumenib) and Niktimvo™ (axatilimab-csfr) were both annualizing
at ~\$200M as of 1Q26, advancing the company towards profitability



Syndax is driving innovation for patients

Syndax

Expanding our pipeline to fuel the next phase of innovation and growth

Revuforj[®]
(revumenib) tablets
25 mg • 110 mg • 160 mg

Ongoing trials across
the acute leukemia
treatment continuum

Positioned to be the 1st
menin inhibitor approved
in frontline AML

Niktimvo[™]
(axatilimab-csfr)
50 mg/mL for injection, for
intravenous use

Ongoing trials in frontline
cGVHD and IPF

Opportunities in several
other inflammatory and
fibrosing diseases

+

SNDX-4321

Mutant-selective allosteric
EGFR inhibitor for NSCLC

Novel approach to
addressing EGFRm NSCLC
patient populations with
high unmet need

Asset aligns with our proven
in-license + develop strategy

Targeting IND
submission in 4Q26

SNDX-62122

Next-generation menin
inhibitor for MF

Novel, potentially disease-
modifying mechanism in MF

1st candidate from our
internally developed, wholly
owned library of next-gen
menin inhibitors

Targeting IND
submission in 2027

PIPELINE TARGETS MULTIPLE BLOCKBUSTER OPPORTUNITIES

Syndax 

R/R, relapsed or refractory; NSCLC, non-small cell lung cancer

Today's speakers

Syndax speakers



Michael A. Metzger
Chief Executive Officer and Director



Nick Botwood, MBBS
Head of R&D and Chief Medical Officer



Peter Ordentlich, PhD
Chief Scientific Officer and Founder

Thought leaders



John D. Crispino, PhD, MBA
Director, Division of Experimental Hematology,
St. Jude Children's Research Hospital



Toby M. Maher, MD, PhD
Professor of Clinical Medicine and Director of Interstitial Lung
Disease, Keck Medical School of University of Southern California



Michael J. Eck, MD, PhD
Professor, Department of Cancer Biology, Dana-
Farber Cancer Institute; Professor, Department of
Biological Chemistry & Molecular Pharmacology, ⁷
Harvard Medical School

Today's agenda

Introduction	
Driving long-term growth through innovation	Michael A. Metzger, Syndax Pharmaceuticals
An R&D engine built to deliver breakthroughs	Nick Botwood, MBBS, Syndax Pharmaceuticals
Revuforj (revumenib) & Next-Gen Menin Inhibitors	
Advancing our leadership in menin inhibition	Nick Botwood, MBBS, Syndax Pharmaceuticals
Menin inhibition in myelofibrosis	John D. Crispino, PhD, MBA, St. Jude Children's Research Hospital
Q&A	Syndax leadership and Dr. Crispino
Niktimvo (axatilimab)	
Unlocking the potential of CSF-1R inhibition	Peter Ordentlich, PhD, Syndax Pharmaceuticals
Clinical perspective on IPF and CSF-1R inhibition	Toby M. Maher, MD, PhD, Keck Medical School of University of Southern California
Q&A	Syndax leadership and Dr. Maher
SNDX-4321	
A novel mutant-selective, allosteric EGFR inhibitor for NSCLC	Nick Botwood, MBBS, Syndax Pharmaceuticals
	Michael J. Eck, MD, PhD, Dana-Farber Cancer Institute and Harvard Medical School
Q&A	Syndax leadership and Dr. Eck
Closing remarks	
	Michael A. Metzger, Syndax Pharmaceuticals

An R&D engine built to deliver breakthroughs



Nick Botwood, MBBS
Head of Research & Development and Chief Medical Officer

Syndax is built to deliver innovative therapies in areas of high unmet need

WORLD-CLASS R&D ORGANIZATION

- Talented team with extensive experience developing novel medicines and building leading franchises
- Trailblazers in menin, CSF-1R, and EGFR inhibition

PARTNERSHIPS WITH LEADING RESEARCHERS

- New discoveries catalyzed by long-standing collaborations with leading researchers

DEEP MECHANISTIC UNDERSTANDINGS

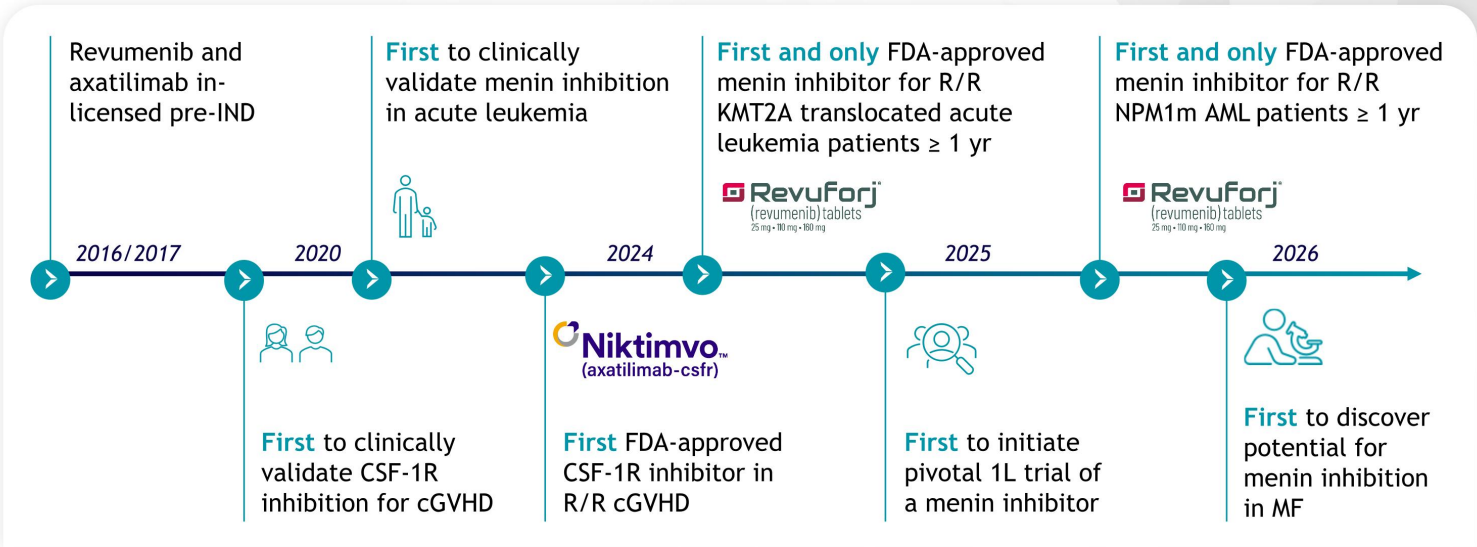
- Proven ability to follow the science to deliver groundbreaking new treatments

FULLY INTEGRATED CAPABILITIES

- In-house expertise spanning discovery, development, CMC, regulatory approval, and commercialization
- Right-sized for innovation and rapid decision making



Proven track record of delivering breakthroughs



Achieved three FDA approvals between revumenib and axatilimab; both drugs advanced from IND to FDA approval in ~5 years

PIONEERING MENIN & CSF-1R INHIBITION FOR PATIENTS

Multiple opportunities for expansion with revumenib and axatilimab

Asset	Setting	Pre-clinical	Ph 1	Ph 2	Ph 3	FDA approved
Revumenib	R/R acute leukemia					
	1L AML (with SOC drugs)					
	Post-HSCT maintenance					
	MRD relapse					
	Myelofibrosis (proof-of-principle)					
Axatilimab	R/R cGVHD					
	1L cGVHD (with steroids)*					
	1L cGVHD (with rux)*					
	IPF					

A GROWING PIPELINE OF DIFFERENTIATED ASSETS

At least 4 innovative
assets expected in the
clinic in 2027

Asset	Setting	Pre-clinical	Ph 1	Ph 2	Ph 3	FDA approved
Revumenib	R/R acute leukemia					
	1L AML (with SOC drugs)					
	Post-HSCT maintenance					
	MRD relapse					
	Myelofibrosis (proof-of-principle)					
Axatilimab	R/R cGVHD					
	1L cGVHD (with steroids)*					
	1L cGVHD (with rux)*					
	IPF					
NEW SNDX-4321	EGFRm NSCLC					
	SNDX-62122 (next-gen menin inhibitor)					
	Myelofibrosis					

Advancing our leadership in menin inhibition

Nick Botwood, MBBS
Head of Research & Development and Chief Medical Officer

Positioned to continue leading menin inhibition in acute leukemia and beyond



BROADEST INDICATION & CLINICAL ACTIVITY

- Only menin inhibitor approved for R/R **NPM1m** AML + **KMT2A** translocated acute leukemia in **adults and children**
- Only menin inhibitor with presented data showing clinical activity in **NUP98r**



INTEGRATED EVIDENCE GENERATION PLAN

- Rapidly growing body of **practice-informing**, potentially **guideline- and registration-enabling** data across multiple acute leukemia settings and genetic subtypes



LIBRARY OF NEXT-GEN MENIN INHIBITORS

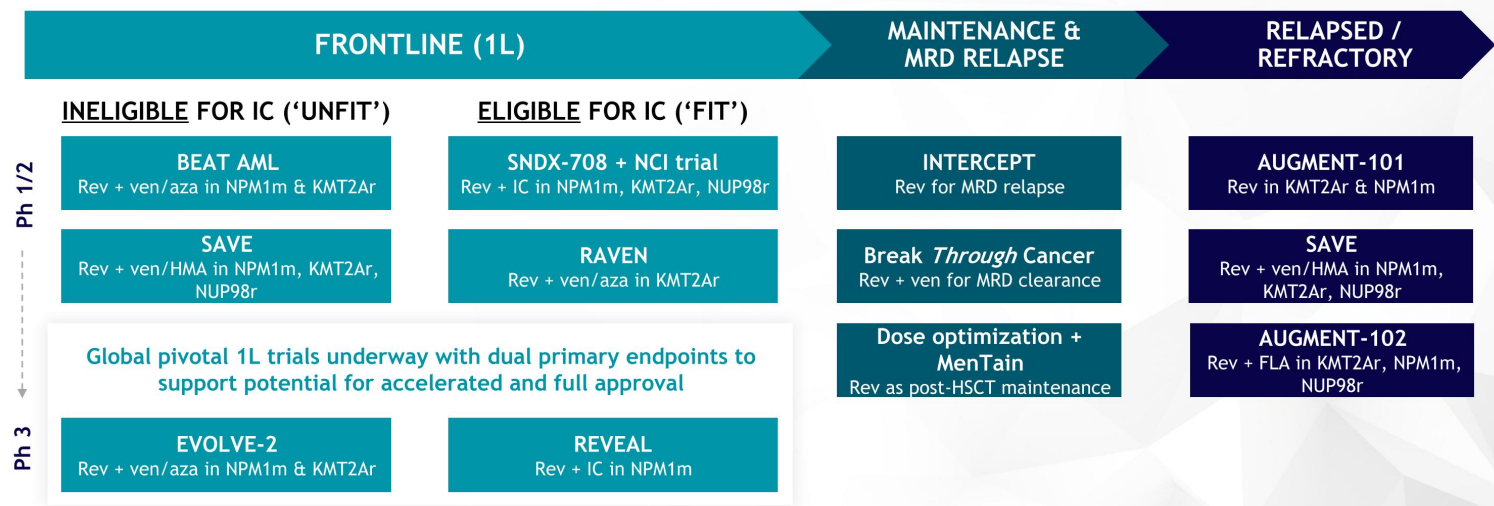
- Internally developed library coupled with the ability to leverage revumenib to inform and de-risk **development in promising new areas**



STRONG SCIENTIFIC PARTNERSHIPS

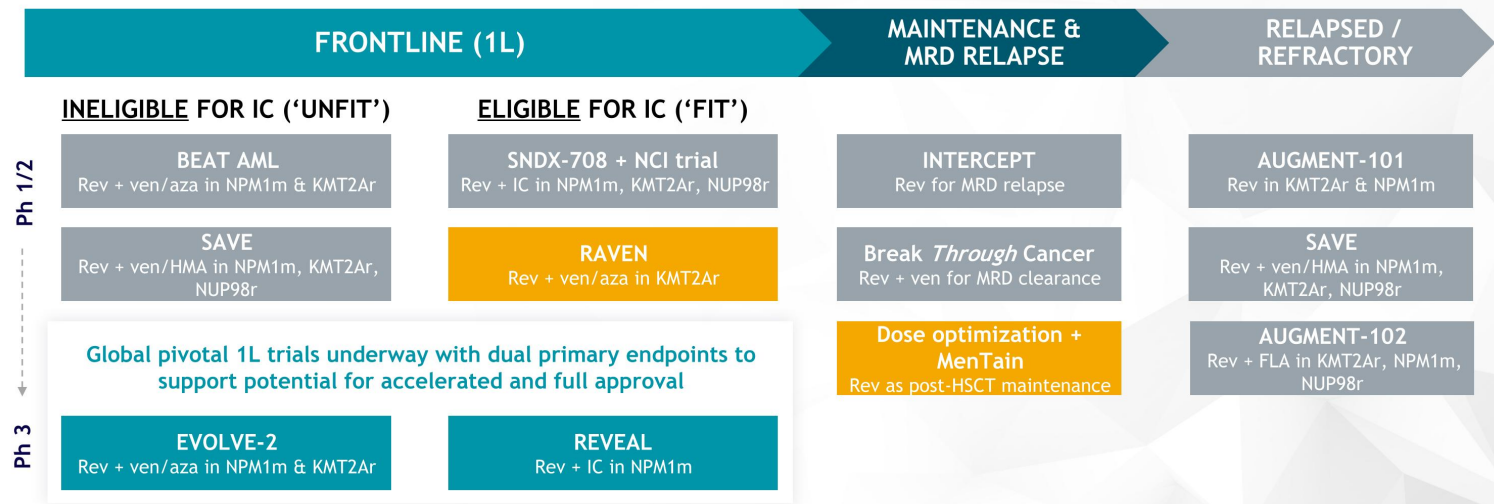
- Collaborations with **world-leading scientists and clinicians** catalyze translation of promising scientific discoveries into breakthroughs for patients

Syndax is pioneering menin inhibition across multiple acute leukemia settings and subtypes, including NPM1m, KMT2Ar, and NUP98r



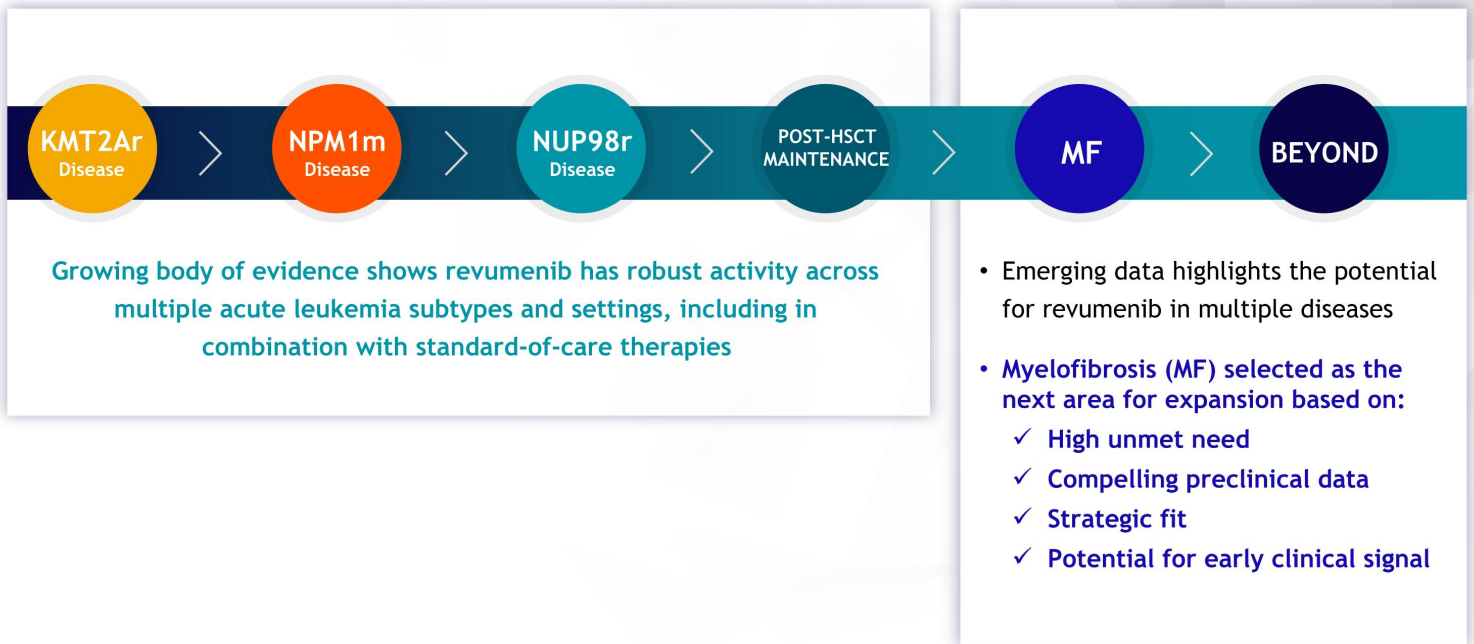
Rapidly growing body of practice-informing, potentially guideline- and registration-enabling data

Syndax is pioneering menin inhibition across multiple acute leukemia settings and subtypes, including NPM1m, KMT2Ar, and NUP98r



Pivotal 1L trials will be complemented by RAVEN (1st trial of a menin inhibitor + ven/aza in 1L fit KMT2Ar) + MenTain (1st placebo-controlled trial focused on revumenib as post-HSCT maintenance)

Following the science to unlock the full potential of menin inhibition

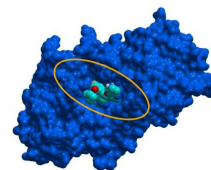


Leading menin inhibition into the future with a library of next-gen molecules plus the ability to leverage revumenib to de-risk development in new areas

Library of internally developed next-gen menin inhibitors



SNDX-62122 next-generation menin inhibitor



- Molecules will be deployed in new areas, starting with MF
- Rationally designed molecules informed by our deep molecular understanding of revumenib's interaction with menin
- Wholly owned assets with no financial encumbrances
- Multiple distinct scaffolds with composition of matter patents into late 2040s
- 1st molecule to emerge from our library
- IND submission in MF expected in 2027
- Profile:
 - ✓ Higher potency
 - ✓ Increased selectivity
 - ✓ Optimized PK
 - ✓ Active against common resistance mutations

Menin inhibition in myelofibrosis

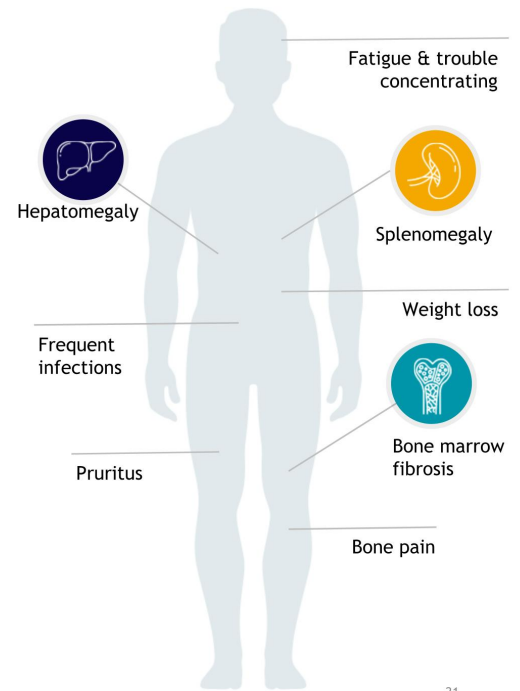


John D. Crispino, PhD, MBA
Director, Division of Experimental Hematology, St.
Jude Children's Research Hospital

New therapeutic strategies are needed in myelofibrosis (MF)

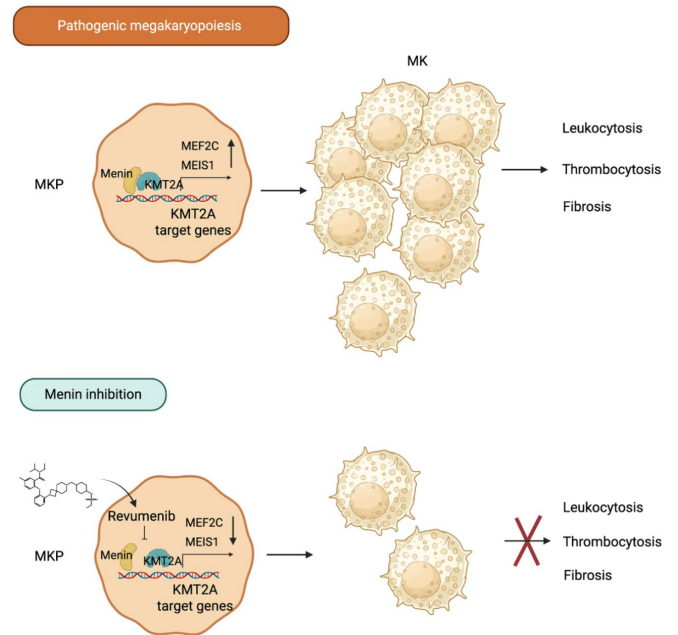
- MF, a type of myeloproliferative neoplasm (MPN), affects ~20,000 people in the U.S.
- MF is associated with:
 - Extramedullary hematopoiesis contributing to hepatosplenomegaly
 - Increased levels of inflammatory cytokines mediating debilitating symptom burden
 - Bone marrow fibrosis accompanying cytopenias
- Stem cell transplant is the only potentially curative option, but >90% of MF patients are not candidates for a transplant; JAK inhibitors (e.g., ruxolitinib) are the current SOC for the vast majority of patients

- While they reduce symptom burden and splenomegaly, JAK inhibitors do not appreciably reduce fibrosis or the mutant allele burden
- Most patients stop responding to JAK inhibitors within 2-3 years with poor outcomes



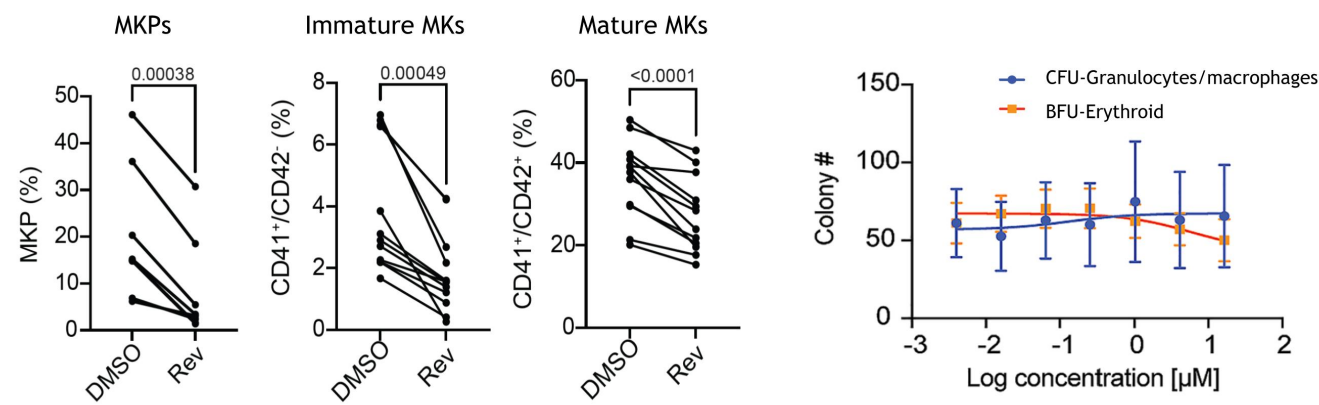
Menin inhibition is a promising new target in MF

- MF is characterized by an accumulation of atypical megakaryocytes (MKs), which contribute to fibrosis by secreting factors that increase collagen deposition
- Emerging data reveal that menin is a novel dependency in proliferative MKs
 - Menin inhibition suppresses megakaryopoiesis via downregulation of *MEF2C* and *MEIS1*
 - Menin inhibition selectively affects megakaryocyte progenitors (MKPs)
 - Revumenib showed striking anti-tumor activity in preclinical models of MPNs, supporting further investigation of menin inhibition for MF
 - Menin inhibition targets pathways independent of the JAK/STAT pathway



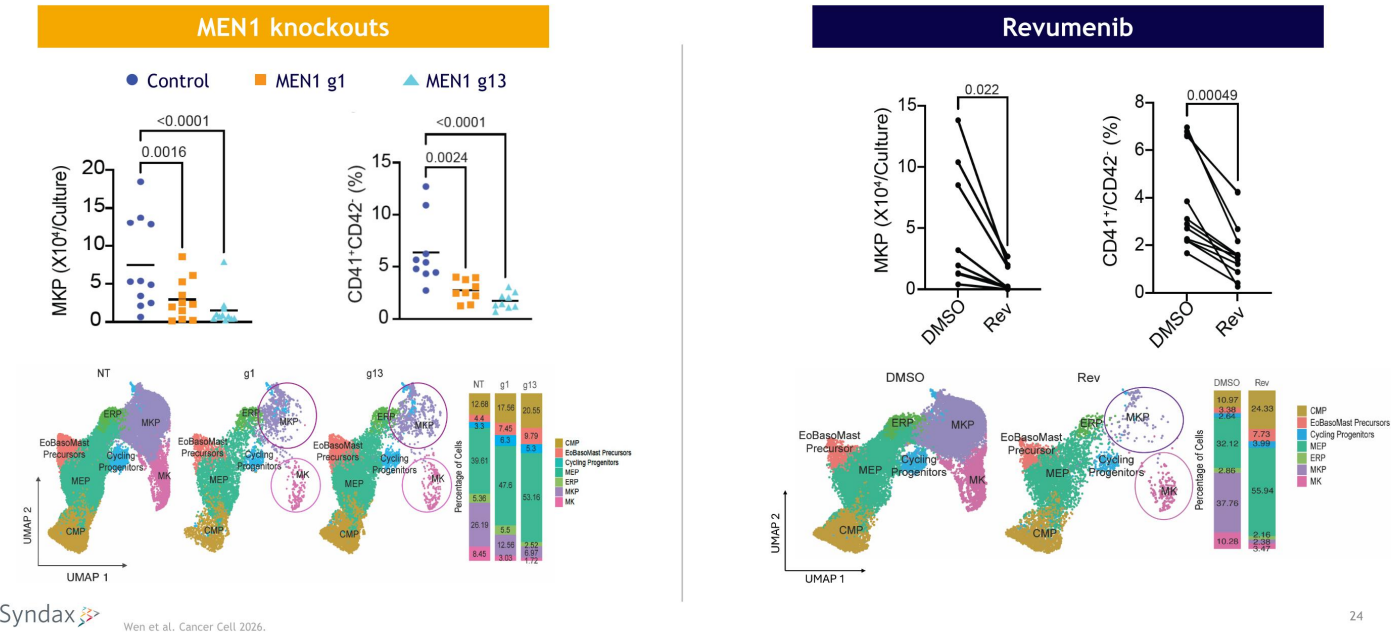
Our recently published preclinical data show revumenib selectively inhibits megakaryopoiesis

Revumenib reduced megakaryocyte progenitors (MKPs) and the formation of immature and mature megakaryocytes (MKs), without affecting myeloid or erythroid colonies

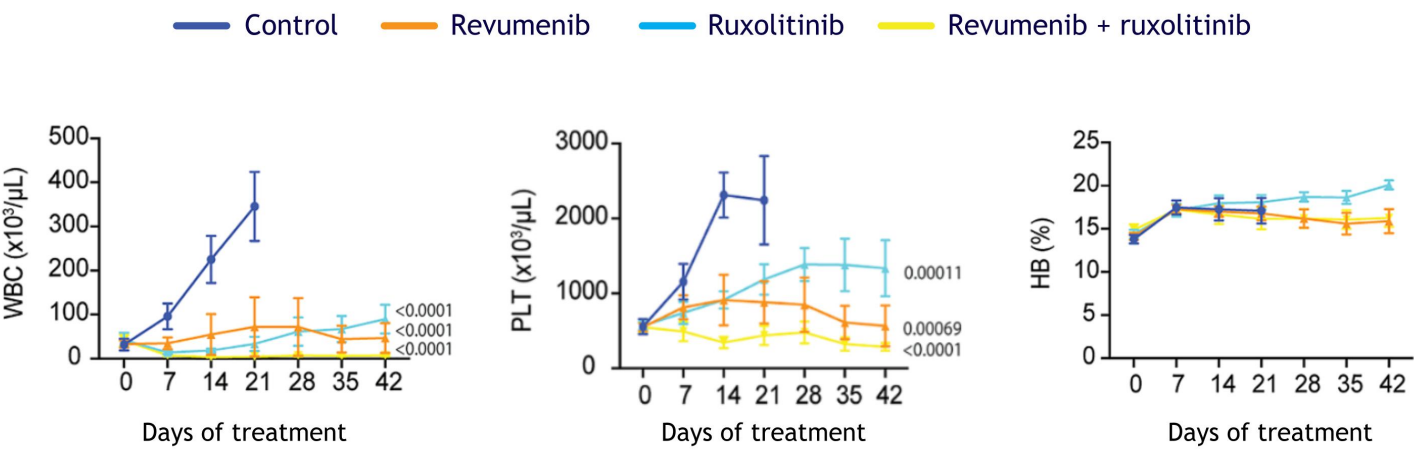


Loss of menin (MEN1) phenocopies revumenib, confirming on-target effect of the drug

Both revumenib and MEN1 loss substantially decreased MKPs and immature MKs



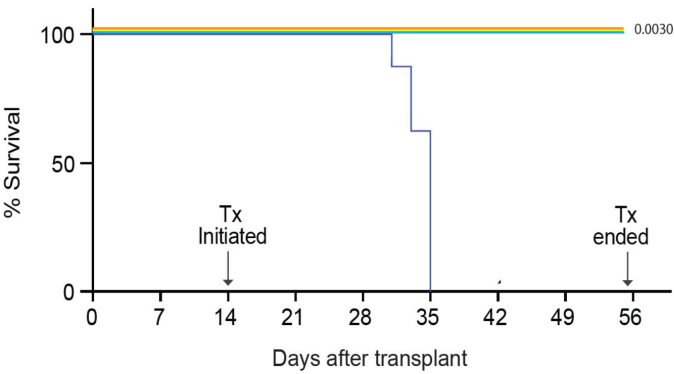
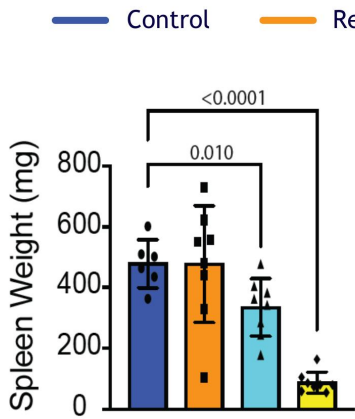
Revumenib alone and in combo with ruxolitinib improves peripheral blood counts in MPN animal models



Revumenib increases survival and normalizes spleen weight in combination with ruxolitinib in MPN animal models

Rev + rux combo notably reduced spleen weight

Both single agent arms and the rev + rux combo showed extended survival compared to the control

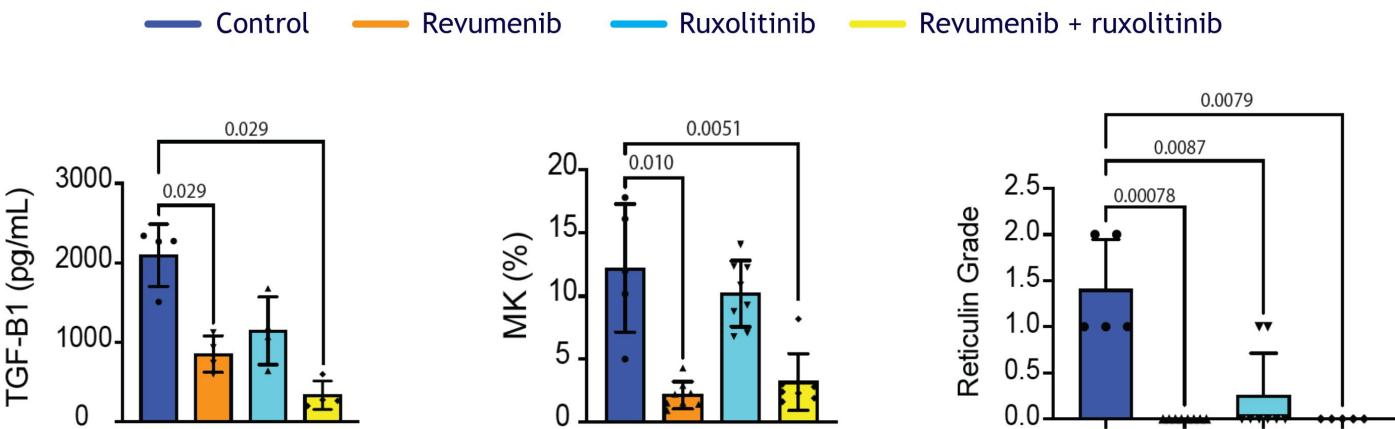


Revumenib alone and in combo with ruxolitinib suppressed levels of TGF- β , megakaryocyte accumulation, and fibrosis more than ruxolitinib alone

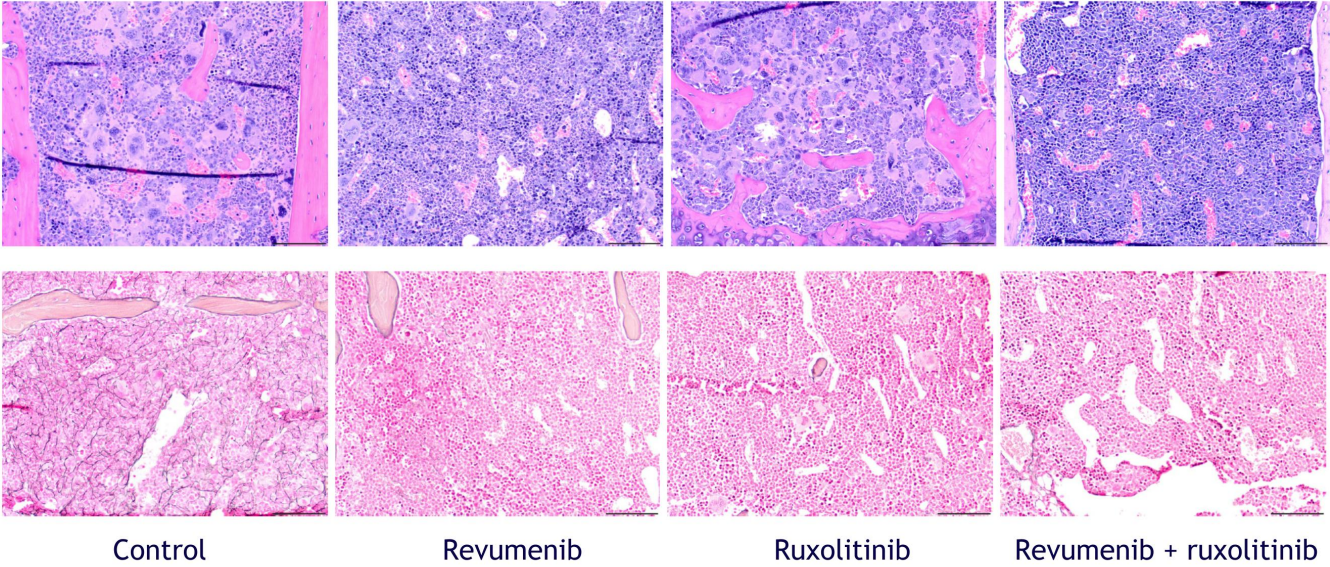
Both rev arms decreased TGF- β , an inflammatory & profibrotic cytokine

Both rev arms decreased MK numbers, while rux alone did not

Both rev arms reduced fibrosis more significantly than rux alone

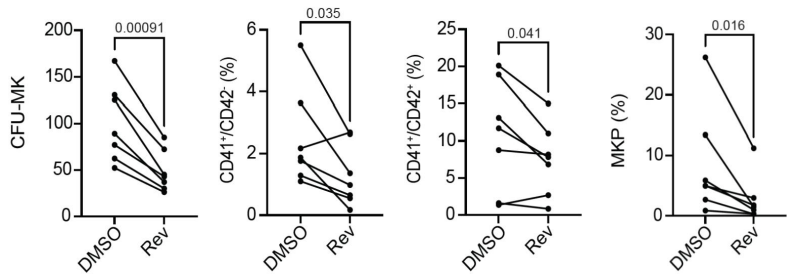


Revumenib as a single agent and in combo with ruxolitinib normalizes bone marrow cellularity and abrogates fibrosis in MPN animal models

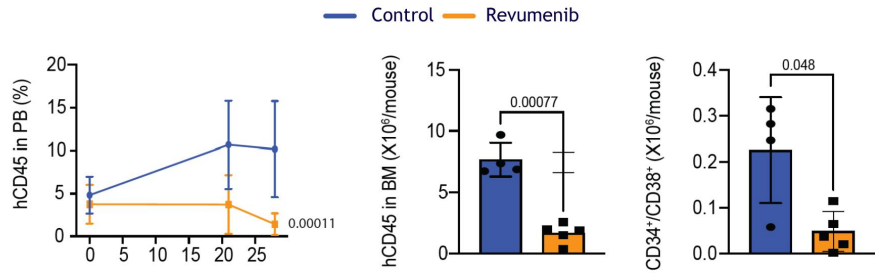


Revumenib targets malignant megakaryocyte progenitors (MKPs) in MF patient samples

In vitro, revumenib reduced MKs and MKPs derived from human MF samples



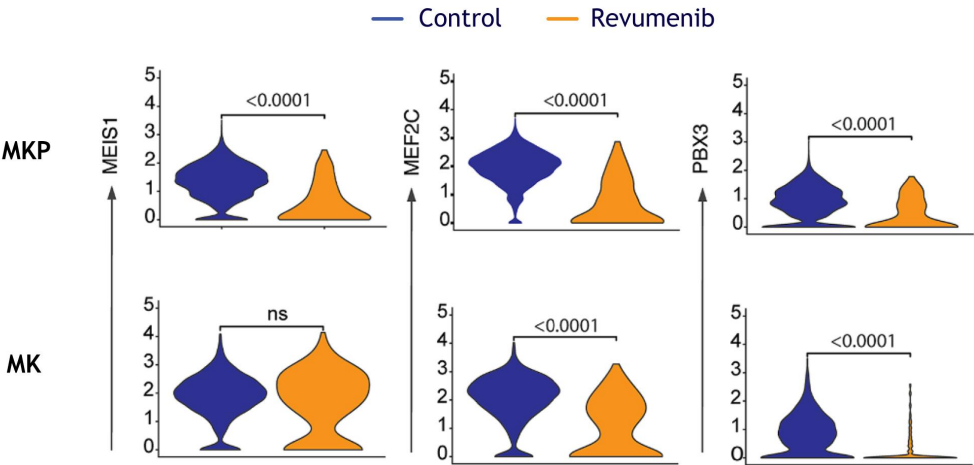
In vivo, revumenib reduced the proportion of human cells in the peripheral blood (PB) and bone marrow (BM) and the number of CD34⁺CD38⁻ HSPCs in the BM



Revumenib could provide a unique and complementary mechanism of action in myelofibrosis

Revumenib's effects are mediated by downregulation of key MKP genes, such *MEF2C* and *MEIS1*

Revumenib reduced expression of key menin-KMT2A target genes in MKPs and MKs



Summary

- **Menin inhibition suppresses megakaryopoiesis in times of stress or disease** when progenitors are highly proliferative; on-target effect in MF is driven in part by downregulation of *MEF2C* and *MEIS1*
- **Revumenib has anti-tumor effects in multiple MPN mouse models** as a single agent and in combination with ruxolitinib
- **Revumenib and ruxolitinib appear to synergistically suppress MF cell growth** by targeting regulation of key MKP genes and JAK/STAT signaling, respectively
- **Menin inhibition may be complimentary to other emerging targeted therapies for MF** (e.g., mutCALR-targeted, JAK2 selective) which target activated JAK/STAT pathway
- Our study provides rationale for **further clinical investigation of menin inhibition in MPNs**

Menin inhibition in MF - Next steps

Nick Botwood, MBBS
Head of Research & Development and Chief Medical Officer

Leveraging revumenib to inform and de-risk development of SNDX-62122 in MF

Anticipated path forward

✓ Publish
preclinical MF data



Initiate revumenib
proof-of-principle
MF trial in 4Q26



Submit IND for
SNDX-62122
(next-gen menin
inhibitor) in 2027

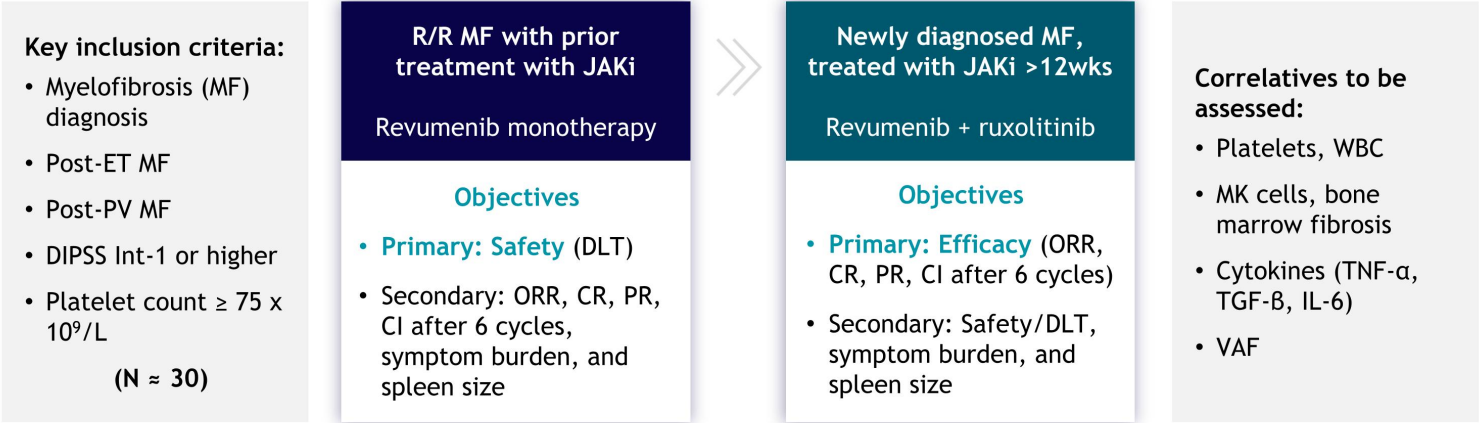


Initiate Ph 1 of
SNDX-62122 in MF
in 2027, leveraging
learnings with
revumenib

**Proof-of-principle trial with revumenib expected to generate data that will
inform the trial design and dosing of SNDX-62122 in MF**

Phase 1/2 proof-of-principle trial of menin inhibition in MF expected to initiate in 4Q26 in partnership with the MPN-RC consortium

The Myeloproliferative Neoplasm Research Consortium (MPN-RC) is a group of leading researchers from 15 institutions



Validation of revumenib clinical activity in MF anticipated in 2H27

Q&A - Revumenib and Next-Gen Menin Inhibitors



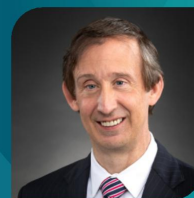
Michael A. Metzger
Chief Executive Officer and Director



Nick Botwood, MBBS
Head of R&D and Chief Medical Officer



Peter Ordentlich, PhD
Chief Scientific Officer and Founder



John D. Crispino, PhD, MBA
Director, Division of Experimental Hematology,
St. Jude Children's Research Hospital

Unlocking the potential of CSF-1R inhibition



Peter Ordentlich, PhD
Chief Scientific Officer and Founder

Axatilimab development driven by deep mechanistic understandings of the CSF-1R pathway

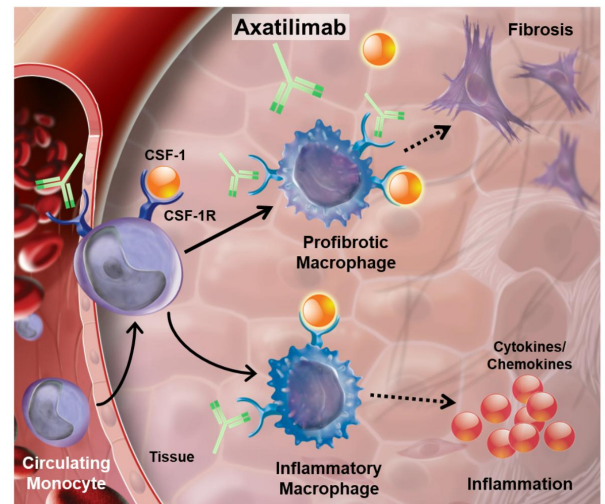
- Colony stimulating factor 1 receptor (CSF-1R) signaling is essential for the activation, proliferation, and differentiation of monocytes and monocyte-derived macrophages
- Axatilimab is a monoclonal antibody that blocks the activation of CSF-1R by its two ligands, CSF-1 and IL-34, which:

1 Inhibits the inflammatory process by:

- Suppressing the activity of monocytes and macrophages, reducing proinflammatory cytokine secretion

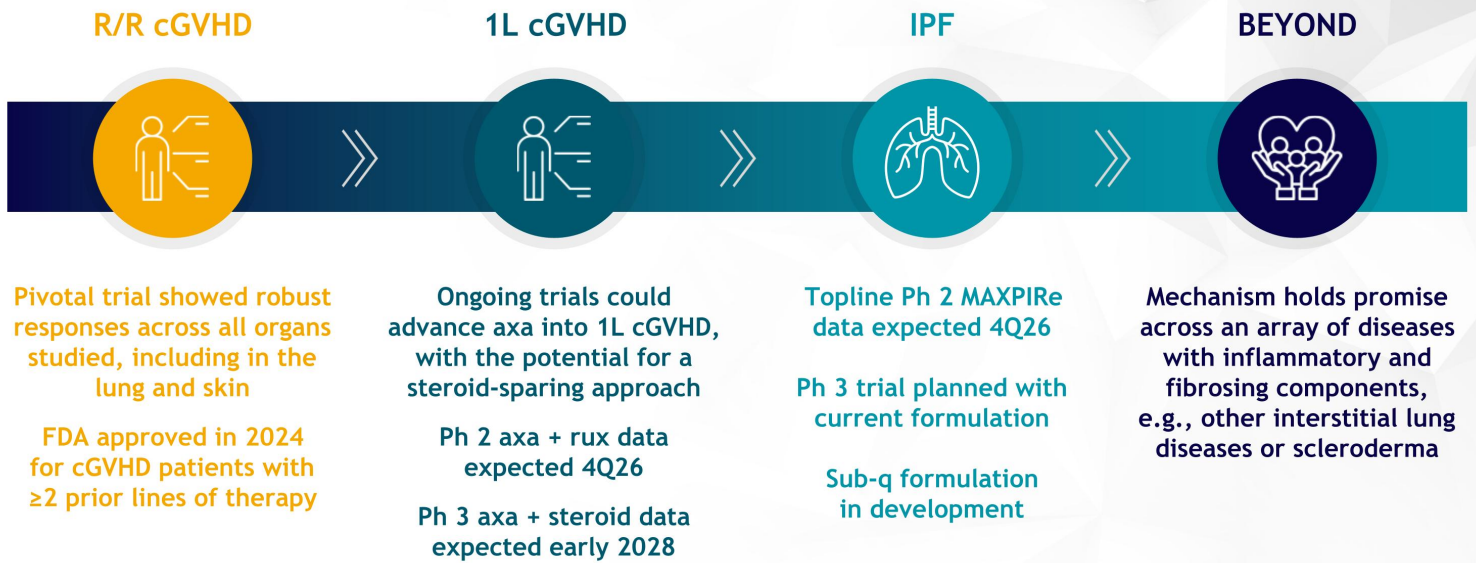
2 Inhibits the fibrotic process by:

- Reducing monocyte levels, suppressing their differentiation into profibrotic macrophages
- Inhibiting macrophages, disrupting the TGF- β -mediated development of fibrosis



CSF-1R inhibition has the potential to impact an array of inflammatory and fibrosing diseases

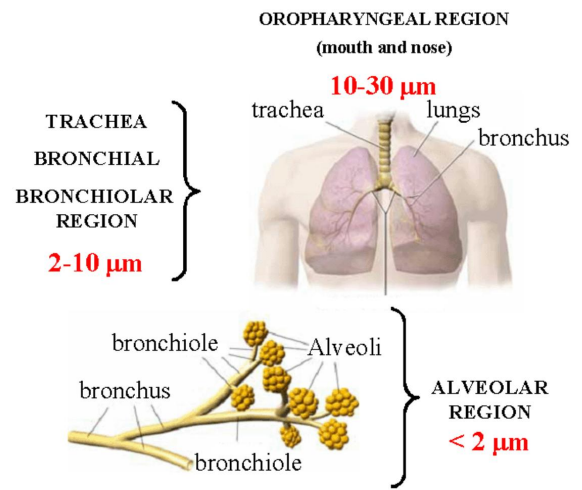
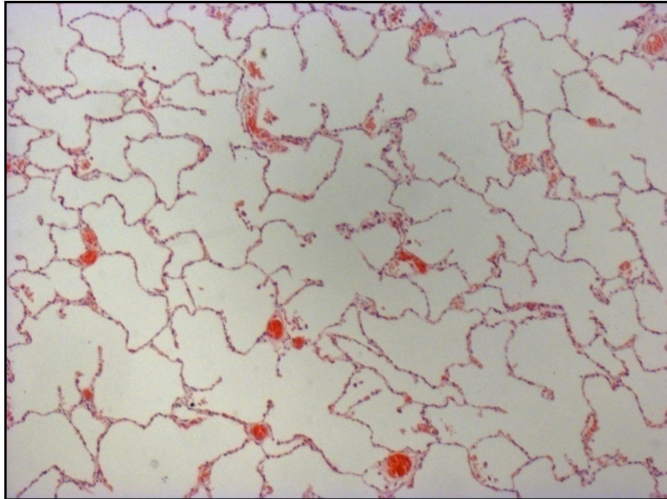
Following the science to unlock the full potential of CSF-1R inhibition



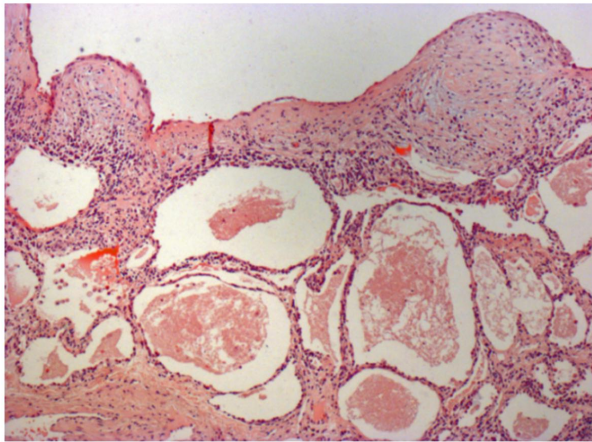
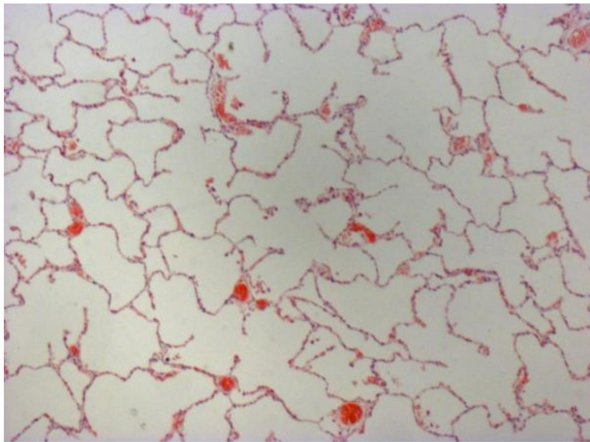
Clinical perspective on IPF and CSF-1R inhibition



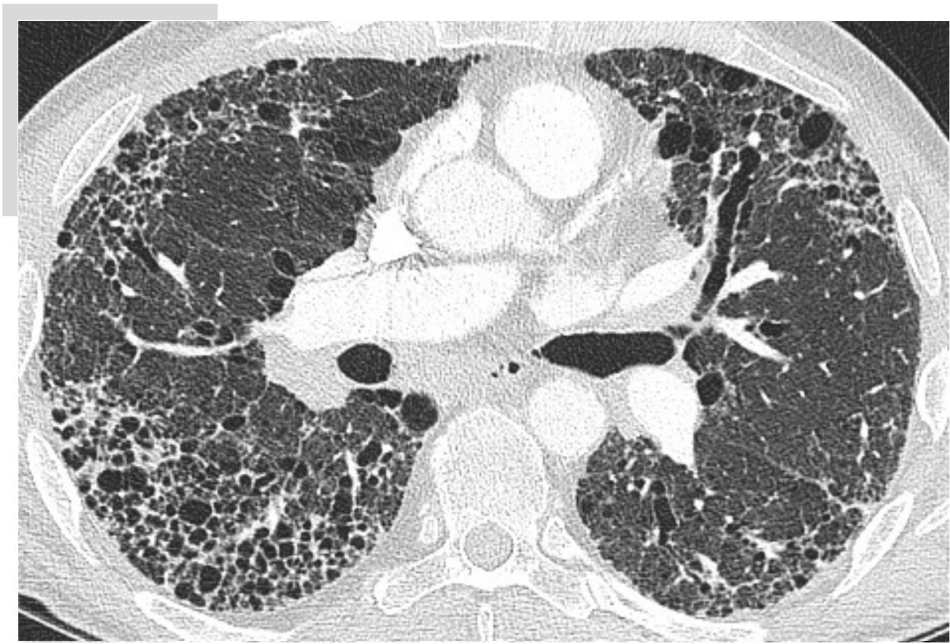
Toby M. Maher, MD, PhD
Professor of Clinical Medicine and Director of Interstitial Lung Disease,
Keck Medical School of University of Southern California



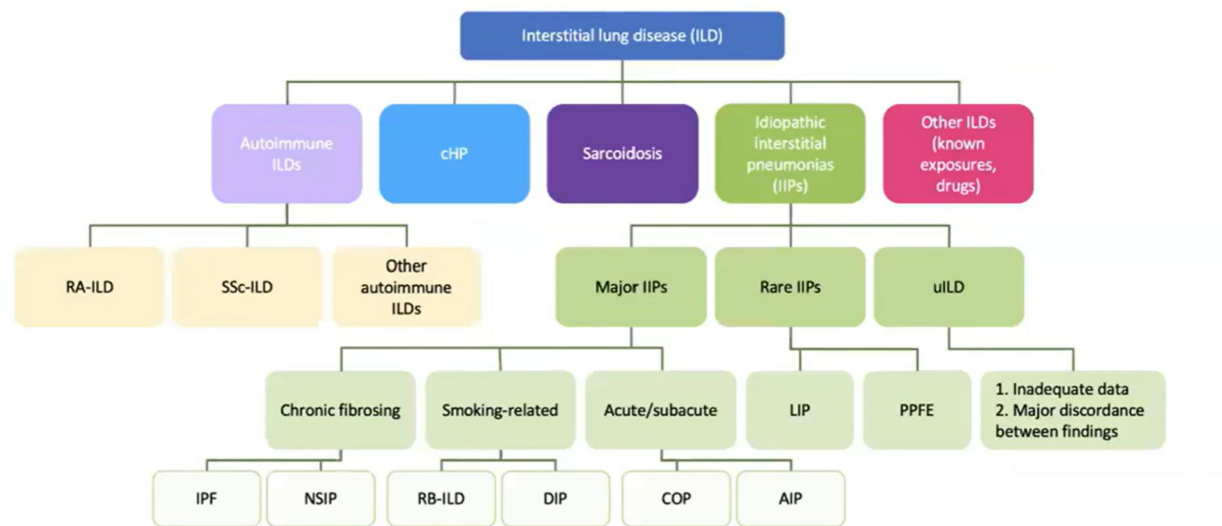
IPF - Histology



Idiopathic pulmonary fibrosis



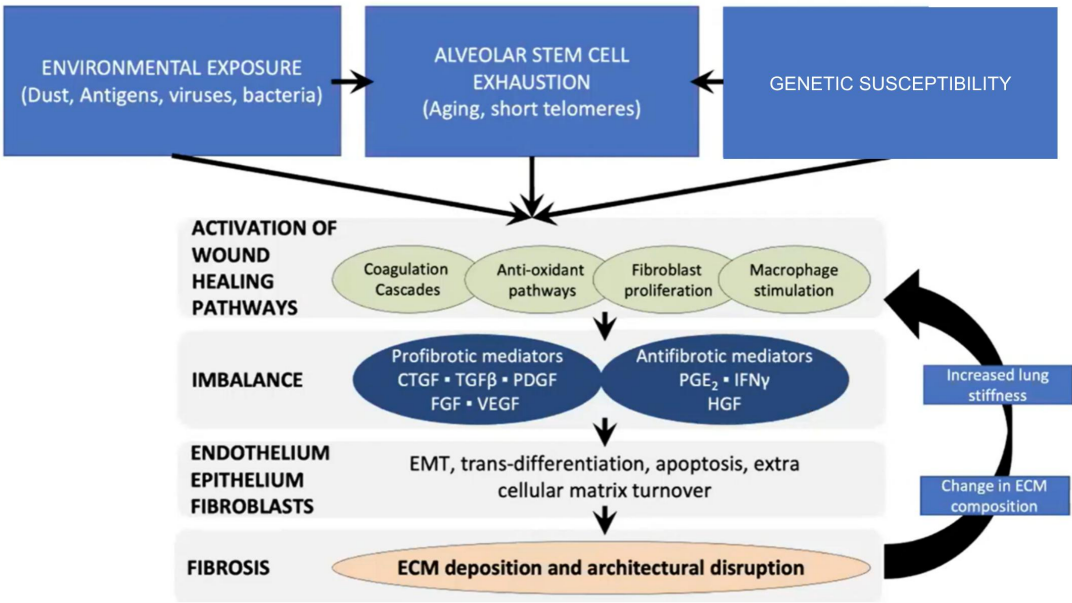
IPF is one of several interstitial lung diseases with shared pathophysiology



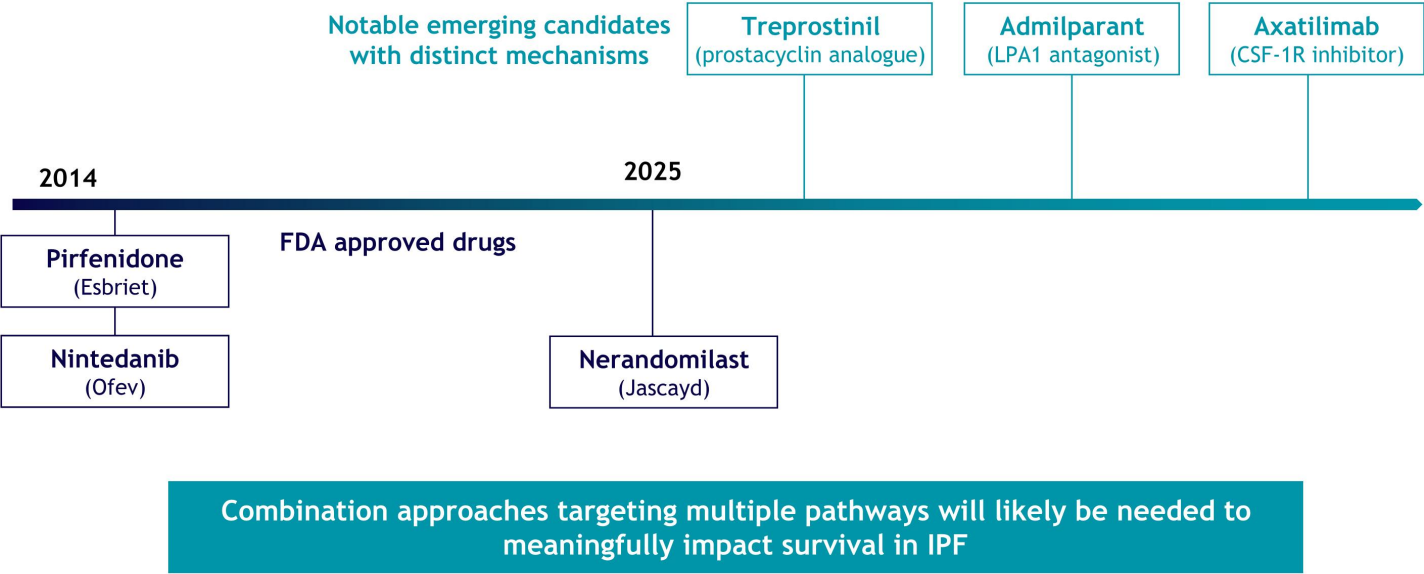
AIP, acute interstitial pneumonia; ATS, American Thoracic Society; COP, cryptogenic organising pneumonia; ERS, European Respiratory Society; IPF, idiopathic pulmonary fibrosis; LAM, lymphangioleiomyomatosis; LCH, Langerhans cell histiocytosis; LIP, lymphocytic interstitial pneumonia; PPFE, pleuroparenchymal fibroelastosis

Adapted from: ATS/ERS. *Am J Respir Crit Care Med.* 2002;165:277–304; Ryerson CJ, Collard HR. *Curr Opin Pulm Med.* 2013;19:453–459; Travis WD, et al. *Am J Respir Crit Care Med.* 2013;188:733–748; Cottin V, et al. *Eur Respir Rev.* 2018;27:pil180076

IPF is a complex, multi-factorial disease



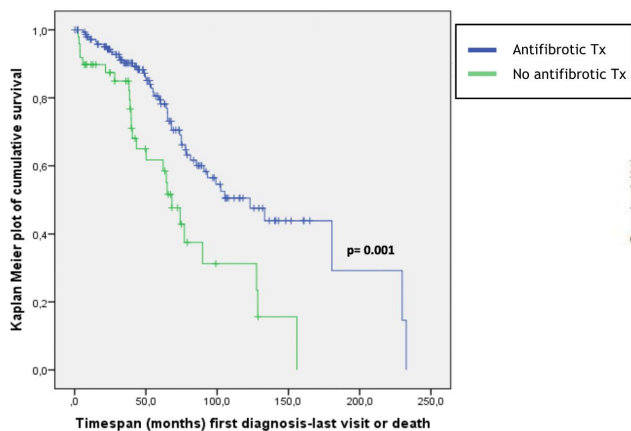
Evolving Tx landscape and multiple emerging modalities could advance patient outcomes in IPF



Real-world outcomes with first generation antifibrotics underscore need for new therapeutic approaches in IPF

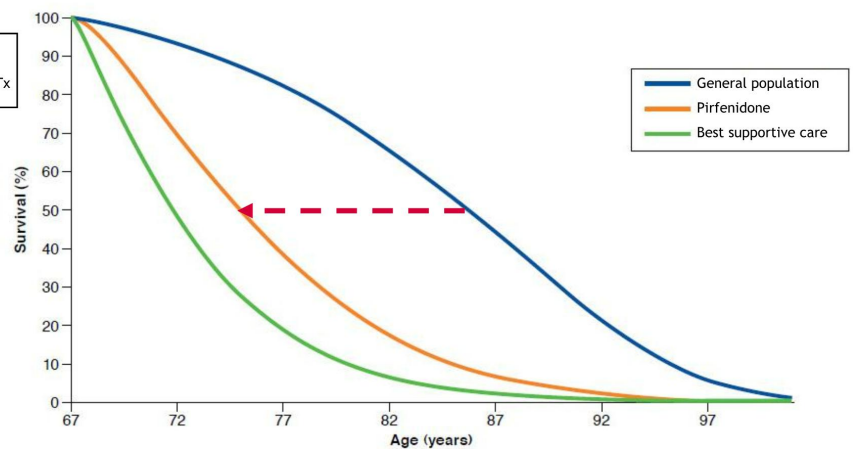
The European IPF registry (eurlPFreg): baseline characteristics and survival of patients with idiopathic pulmonary fibrosis

Andreas Guenther^{1,2,3,4,5*}, Eleonora Krauss^{1,2}, Silke Tello³, Jasmin Wagner^{1,2}, Bettina Paul^{1,2}, Stefan Kuhn^{1,2}, Olga Maurer^{1,4}, Sabine Heinemann^{1,2}, Ulrich Costabel^{1,6}, Maria Asuncion Nieto Barbero^{1,8}, Veronika Muller^{1,10}, Philippe Bonniaud^{1,11}, Carlo Vancheri^{1,9}, Athol Wells^{1,7}, Marina Vasakova^{1,11}, Alberto Pesci^{1,12}, Matteo Sofia^{1,13}, Walter Kleecko^{1,14}, Werner Seeger^{1,15}, Fotios Drakopanagiotakis^{1,9} and Bruno Crestani^{1,16}



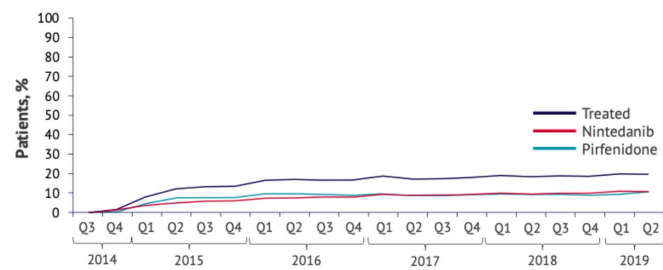
Predicting Life Expectancy for Pirfenidone in Idiopathic Pulmonary Fibrosis

Mark Fisher, MSc; Steven D. Nathan, MD; Christian Hill, BSc; Jade Marshall, MSc; Fred Dejonckheere, MD; Per-Olof Thuresson, MSc; and Toby M. Maher, MD

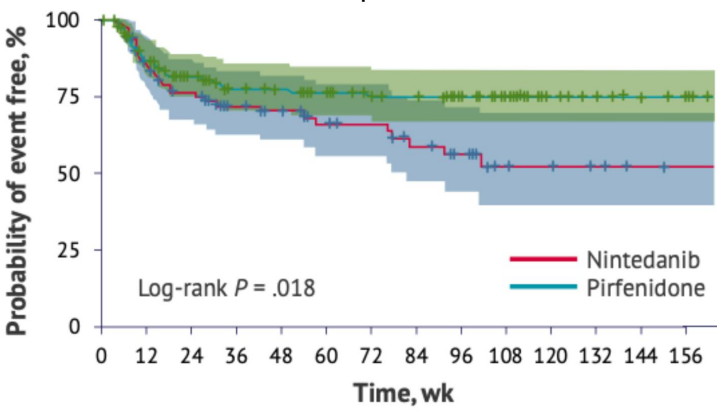


Pirfenidone and nintedanib use and persistence in the US

Anti-fibrotic use in US



Anti-fibrotic persistence in US



The monocyte-derived alveolar macrophages is a promising target in IPF



BRIEF DEFINITIVE REPORT

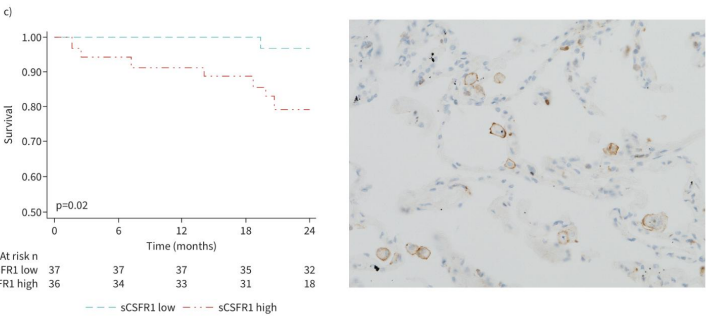
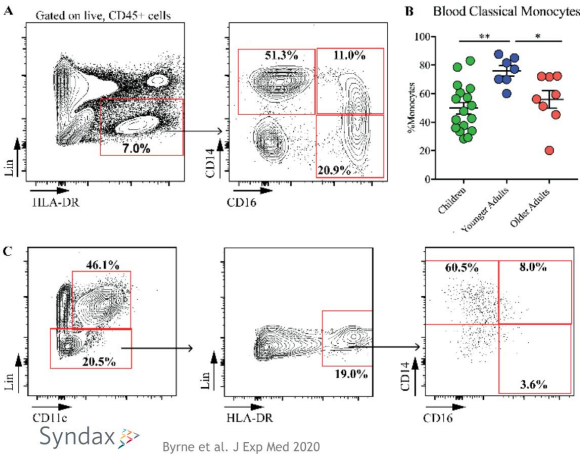
Dynamics of human monocytes and airway macrophages during healthy aging and after transplant

Adam J. Byrne^{1,2}, Joseph E. Powell^{1,4}, Brendan J. O'Sullivan^{1,6}, Patricia P. Oggeri¹, Ashley Hoffland^{1,2}, James Cook^{1,2}, Katie L. Bonner^{1,2}, Richard J. Hewitt^{1,2}, Simone Wolf¹, Poonam Ghai¹, Simone A. Walker¹, Samuel W. Lukowski¹, Philip L. Molyneaux^{1,2}, Sejal Saglam^{1,2,7}, Daniel C. Chambers^{1,2}, Toby M. Maher^{1,2,8}, and Clare M. Lloyd^{1,2,9}



ERJ OPEN RESEARCH
RESEARCH LETTER
J.M. OLDHAM ET AL.

Airway soluble CSF1R predicts progression in patients with idiopathic pulmonary fibrosis

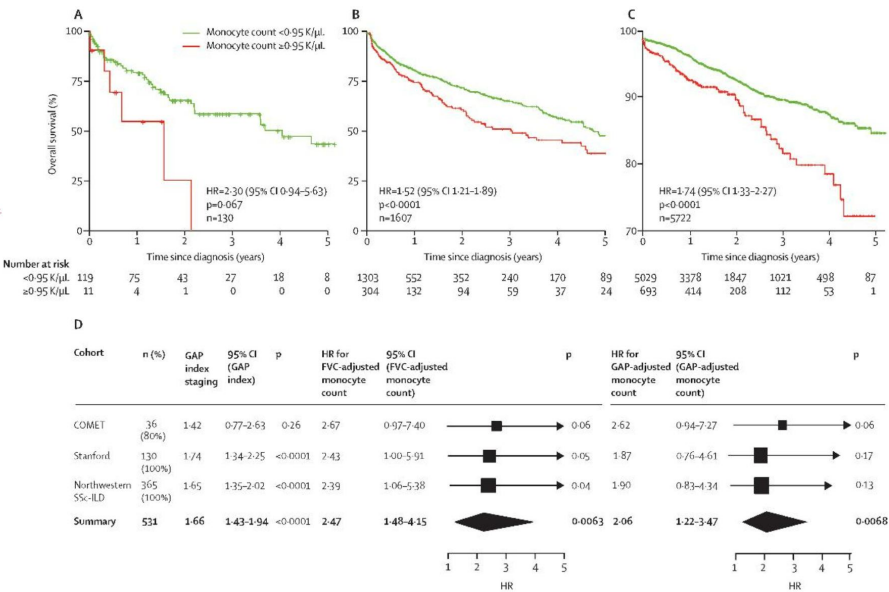


Oldham et al ERJ 2022

Higher monocyte levels are associated with shorter survival in IPF and other fibrotic diseases

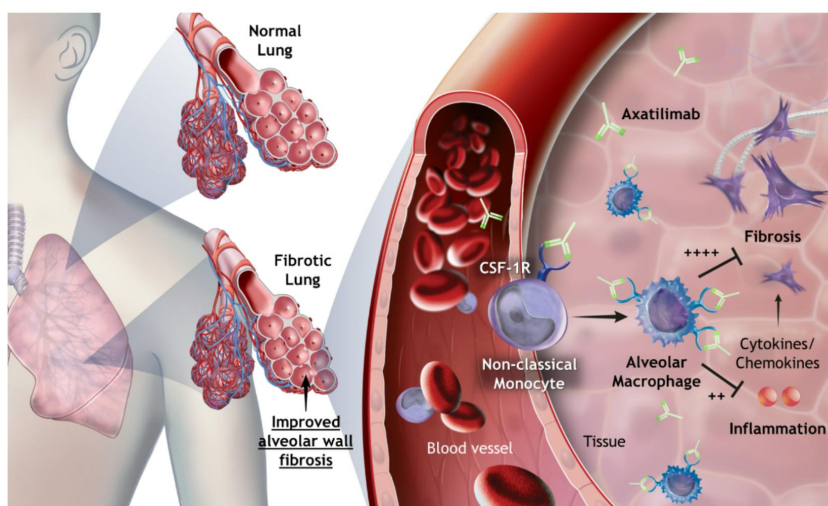
Increased monocyte count as a cellular biomarker for poor outcomes in fibrotic diseases: a retrospective, multicentre cohort study

Maddalena K D Scott, Katie Quinn, Qin Li, Robert Carroll, Hayley Wozniak, Francesca Vallerio, Shirley Chen, Mary A Carr, Kathleen Aron, Jiehua Sun, Kimberly Kofron, Junghwa Lee, Jessica Baral, Jonathan Krupski, Hongyu Zhao, Erica Herzog, Fernando J Martinez, Bethany B Moore, Monique Hirschhoff, Joshua Denny, Neelam Kaminski, Jose D Hernandez-Maya, Nigam H Shah*, Puneesh Khatri*



Axatilimab targets a novel and potentially complementary pathway in IPF

Axatilimab is a CSF1R-blocking antibody targeting monocyte-derived alveolar macrophages



Axatilimab has shown strong activity across multiple organs in cGVHD

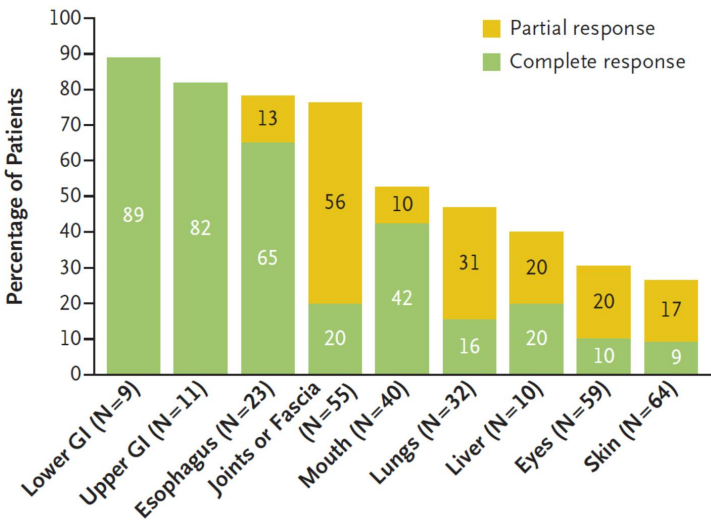
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Axatilimab in Recurrent or Refractory Chronic Graft-versus-Host Disease

D. Wolff, C. Cutler, S.J. Lee, I. Pusic, H. Bittencourt, J. White, M. Hamadani, S. Arai, A. Salhotra, J.A. Perez-Simon, A. Alousi, H. Choe, M. Kwon, A. Bermúdez, I. Kim, G. Socié, S. Chhabra, V. Radojcic, T. O'Toole, C. Tian, P. Ordentlich, Z. DeFilipp, and C.L. Kitko, for the AGAVE-201 Investigators*

Overall Response in the 0.3-mg Dose Group



The NEW ENGLAND JOURNAL of MEDICINE

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Syndax

Wolff et al. NEJM 2024.

Table 2. Safety Overview.*

Adverse Event	0.3-mg Dose Group (N = 79)	1-mg Dose Group (N = 81)	3-mg Dose Group (N = 79)
	number of patients (percent)		
Any adverse event	76 (96)	80 (99)	78 (99)
Grade ≥3	39 (49)	49 (60)	56 (71)
Serious	30 (38)	33 (41)	38 (48)
Fatal	1 (1)	7 (9)	6 (8)
Any grade event in ≥20% of any dose group			
Aspartate aminotransferase level increased	11 (14)	31 (38)	43 (54)
Blood creatine kinase level increased	9 (11)	26 (32)	49 (62)
Lipase level increased	9 (11)	21 (26)	39 (49)
Amylase level increased	3 (4)	10 (12)	34 (43)
Blood lactate dehydrogenase level increased	11 (14)	22 (27)	32 (41)
Alanine aminotransferase level increased	10 (13)	18 (22)	31 (39)
Periorbital edema	2 (3)	19 (23)	23 (29)
Fatigue	18 (23)	16 (20)	21 (27)
γ-Glutamyltransferase level increased	8 (10)	16 (20)	21 (27)
Covid-19	13 (16)	18 (22)	11 (14)
Diarrhea	13 (16)	18 (22)	7 (9)
Blood alkaline phosphatase level increased	5 (6)	4 (5)	17 (22)
Headache	15 (19)	14 (17)	16 (20)
Any grade infection	58 (73)	59 (73)	55 (70)
Any grade key infection or reactivation			
Covid-19	15 (19)	19 (23)	14 (18)
Pneumonia	9 (11)	12 (15)	8 (10)
Aspergillus	0	3 (4)	0
Cytomegalovirus	0	1 (1)	2 (3)
Epstein-Barr virus	0	0	4 (5)
Any grade infusion-related reaction	6 (8)	4 (5)	1 (1)
Any grade ≥3 event in ≥5% of any dose group			
Pneumonia	8 (10)	7 (9)	5 (6)
Blood creatine kinase level increased	1 (1)	6 (7)	12 (15)
Covid-19	3 (4)	5 (6)	5 (6)
Hypertension	3 (4)	5 (6)	4 (5)
γ-Glutamyltransferase level increased	1 (1)	2 (2)	4 (5)
Lipase level increased	1 (1)	1 (1)	4 (5)
Periorbital edema	0	1 (1)	5 (6)
Any serious event in ≥5% of any dose group			
Covid-19	2 (3)	4 (5)	5 (6)
Pneumonia	7 (9)	8 (10)	4 (5)
Any adverse event leading to dose interruption	30 (38)	34 (42)	25 (32)
Any adverse event leading to dose reduction	5 (6)	6 (7)	13 (16)
Any adverse event leading to discontinuation of axatilimab	5 (6)	18 (22)	14 (18)†

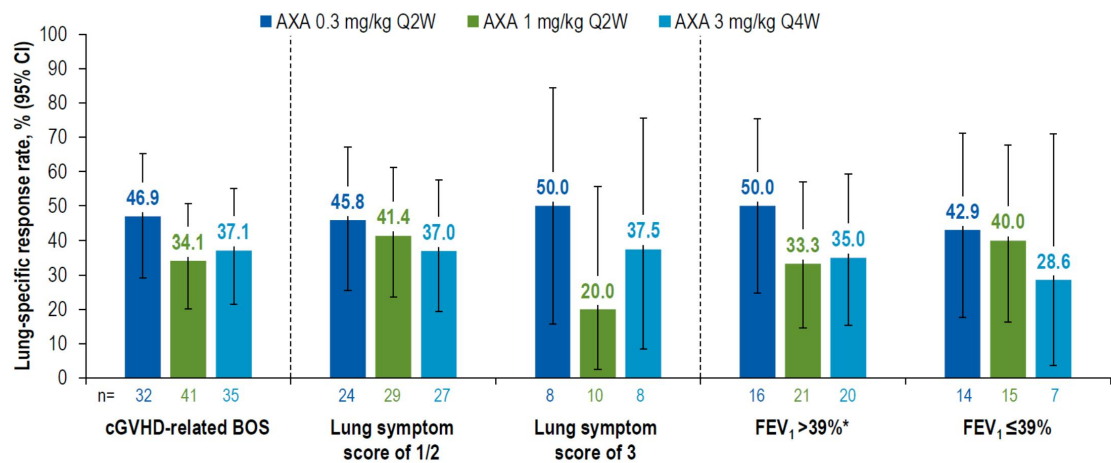
Data in cGVHD-related bronchiolitis obliterans syndrome (BOS) shows axatilimab has antifibrotic activity in the lung

Axatilimab in Patients With Lung Chronic Graft-Versus-Host Disease and Related Bronchiolitis Obliterans Syndrome: Results From the AGAVE-201 Study

Amandeep Salhotra, MD,¹ Vaikal Bhatt, PharmD,² Britnie Thomas, PhD,² Shuyuan Lou, PhD,² Vedran Radojic, MD,³ Zachariah DeFilipp, MD⁴

¹City of Hope National Medical Center, Duarte, CA, USA; ²Inocyte
³Syndax Pharmaceuticals, Inc., New York, NY, USA; ⁴Massachusetts

American Thoracic Society International Conference, May 17–21, 2023; San Francisco



Salhotra et al. Axatilimab in Patients With Lung Chronic Graft-Versus-Host Disease and Related Bronchiolitis Obliterans Syndrome: Results From the AGAVE-201 Study. Oral presentation at ATS 2025.

MAXPIRe is a robust Phase 2 trial of axatilimab in IPF

Randomized, double-blind, placebo-controlled, multi-center international trial



Key eligibility criteria:

- ≥ 40 yrs of age
- HRCT confirming IPF diagnosis
- FVC $\geq 45\%$ of predicted normal (PN)
- $FEV_1/FVC \geq 0.7$
- $DL_{CO} \geq 30\%$ and $\leq 90\%$ PN
- Stable background use of pirfenidone or nintedanib allowed

(N=135)

26-week treatment

Axatilimab 0.3 mg/kg Q2W
(n≈90)

Placebo Q2W
(n≈45)

Randomized 2:1 to axatilimab or placebo; stratified by background antifibrotic therapy (pirfenidone, nintedanib, or none)

PRIMARY ENDPOINT:

- Annualized rate of decline in FVC over 26 weeks (ml)

SECONDARY ENDPOINTS:

- Disease progression, SGRQ (quality of life measures), change in FVC % predicted, DL_{CO}

Summary

- IPF is a deadly and progressive disease
- Current therapies slow but do not prevent progression of fibrosis
- There is an increasing drive to combine therapies in IPF
- CSF-1R positive macrophages play an important role in fibrogenesis
- Axatilimab has been shown to be effective in GVHD - a disease of small airway fibrosis
- The MAXPIRe trial will provide important proof-of-concept in IPF

Q&A - Axatilimab



Michael A. Metzger
Chief Executive Officer and Director



Nick Botwood, MBBS
Head of R&D and Chief Medical Officer



Peter Ordentlich, PhD
Chief Scientific Officer and Founder

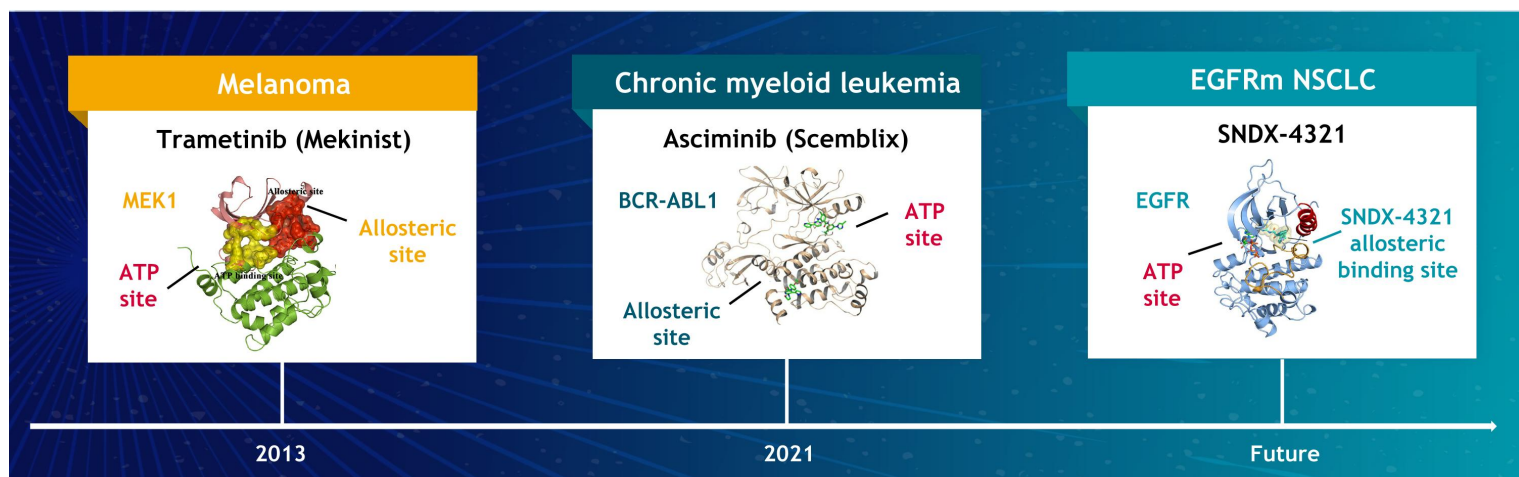


Toby M. Maher, MD, PhD
Professor of Clinical Medicine and Director
of Interstitial Lung Disease, Keck Medical
School of University of Southern California

Expanding our pipeline into EGFRm NSCLC with SNDX-4321

Nick Botwood, MBBS
Head of Research & Development and Chief Medical Officer

Allosteric inhibitors are increasingly reshaping the standard of care for difficult-to-treat cancers



Allosteric inhibitors bind to non-active sites rather than the ATP-binding site on tyrosine kinases; their binding induces a conformational change that 'locks' the kinase in an inactive state

Allosteric approaches can modulate kinase activity with high specificity, optimizing on-target efficacy while reducing off-target toxicity

Syndax aims to address significant unmet needs in EGFRm NSCLC with a novel approach

Currently approved and investigational drugs primarily target the EGFR ATP-binding site

3rd gen approved EGFRi

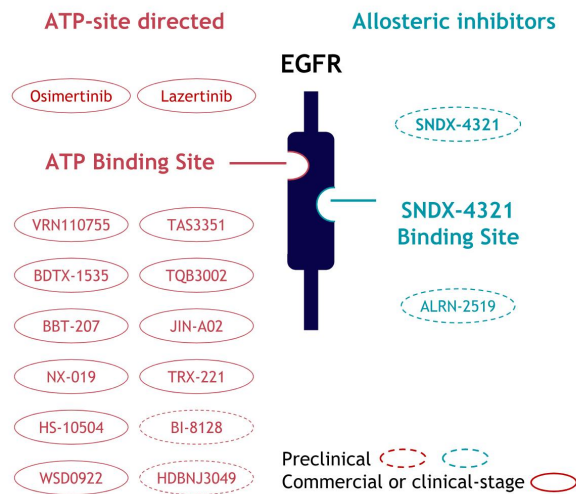
- Reduced benefit for patients with L858R, CNS metastases, or atypical activating mutations

4th gen investigational EGFRi

- Primarily target osimertinib on-target resistance (C797S), limiting benefit to a small population

Bispecific antibodies and ADCs

- Limited by significant toxicities and route of administration



SNDX-4321, a mutant-selective, allosteric EGFR inhibitor

- Allosteric approach allows for:
 - ✓ 'Double drugging' of the target (may enhance efficacy and delay resistance)
 - ✓ High selectivity (supports combinability + tolerability)
- First- and best-in-class potential
- Broad therapeutic window expected
- Oral administration

SNDX-4321, a mutant-selective, allosteric EGFR inhibitor for non-small cell lung cancer (NSCLC)

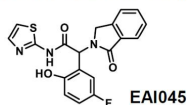


Michael J. Eck, MD, PhD

Professor, Department of Cancer Biology, Dana-Farber Cancer Institute
Professor, Department of Biological Chemistry and Molecular Pharmacology,
Harvard Medical School

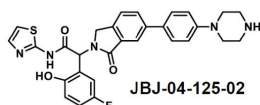
SNDX-4321 builds on Dana-Farber Cancer Institute's pioneering work on allosteric EGFR inhibitors

Overcoming EGFR(T790M) and EGFR(C797S) resistance with mutant-selective allosteric inhibitors



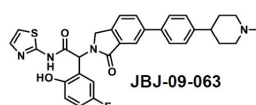
Nature 2016, 534, 129

Single and Dual Targeting of Mutant EGFR with an Allosteric Inhibitor



Cancer Disc 2019, 9, 926

An allosteric inhibitor against the therapy resistant mutant forms of EGFR in non-small cell lung cancer

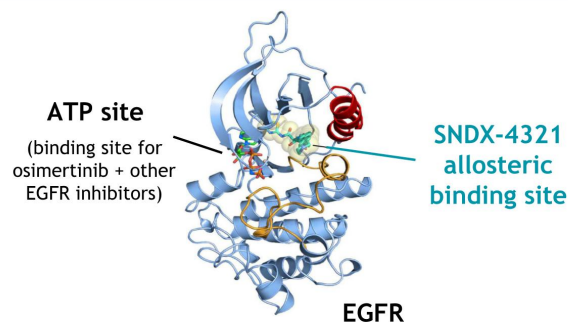


Nat Cancer. 2022, 3, 402

SNDX-4321
mutant-selective
allosteric EGFR
inhibitor

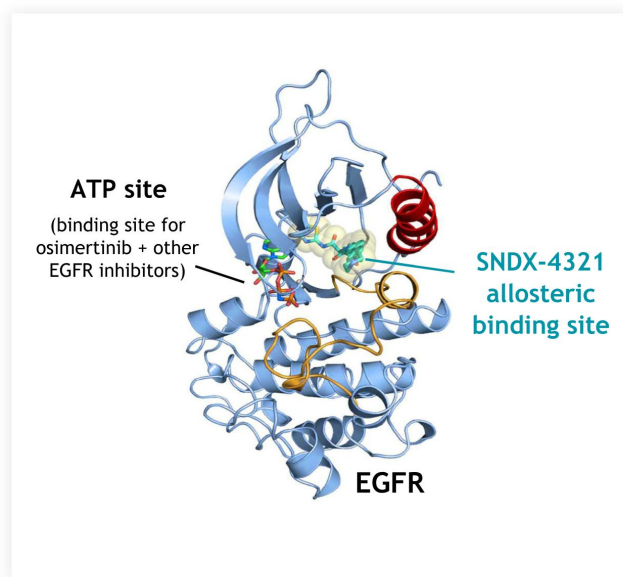
SNDX-4321:

- Binds at a pocket adjacent to the active (ATP) site that is only accessible with L858R and certain other EGFR mutations
- Structurally distinct from published compounds and addresses their key liabilities (prior compounds were potent and selective, but lacked suitable PK and chemical properties)
- Developed specifically for L858R with or without acquired resistance mutations



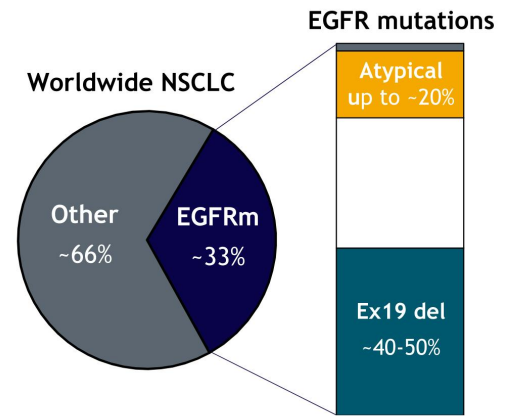
SNDX-4321 has the potential to advance the Tx paradigm for EGFRm NSCLC

- Excellent selectivity, oral bioavailability, and brain penetration
- Efficacy in multiple tumor models as a single-agent or in combination with osimertinib, and in intracranial model
- **Allosteric mechanism provides multiple benefits:**
 - ✓ **Ability to co-bind with ATP-site inhibitors enables 'double drugging'**, which could enhance efficacy and delay resistance
 - ✓ **Improved selectivity** for mutant vs. wild-type EGFR may improve safety
 - ✓ **Single-agent activity could enable treatment of osimertinib-resistant cancers** and patients who progress after frontline therapy



Significant unmet needs remain in EGFRm NSCLC despite recent advances

- ~200,000 people are diagnosed with NSCLC annually in the U.S. alone¹
- **Worldwide ~1/3 of NSCLC patients have EGFR mutations²**
 - The most common EGFR mutations are exon 19 deletions and L858R point mutations
- Osimertinib +/- chemotherapy is the most frequently used treatment for EGFR-mutated (EGFRm) NSCLC with an exon 19 deletion or L858R mutation



While three generations of EGFR inhibitors have transformed the SOC, significant unmet needs remain for patients with L858R mutations, CNS disease, atypical mutations, or resistance to osimertinib

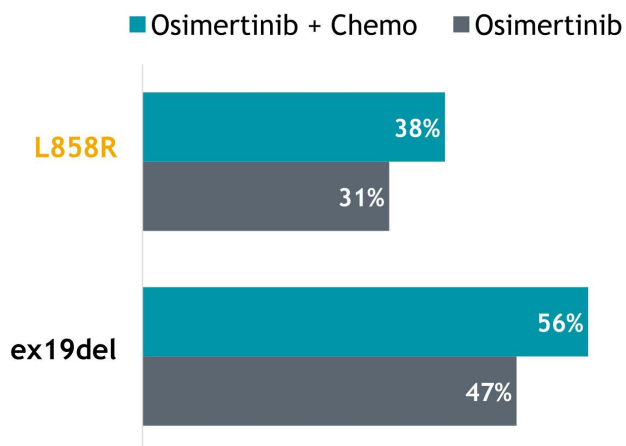
SNDX-4321 has the potential to address several areas of high unmet need in EGFRm NSCLC

UNMET NEEDS		SNDX-4321 PROFILE	
1	Patients with L858R have poorer outcomes than those with ex19 deletions	»	High activity as a single agent and in combination with osimertinib in L858R models
2	Patients with CNS metastases fair worse than those without	»	Highly CNS penetrant; demonstrated efficacy in intracranial model
3	Patients with atypical mutations have poorer outcomes than those with classical mutations	»	Preclinical data show activity against certain atypical mutants
4	Resistance emerges to osimertinib and all other 3 rd gen EGFR inhibitors	»	Unique binding site - no single mutation confers resistance to both SNDX-4321 and osimertinib

1 Patients with L858R NSCLC have poorer outcomes than those with ex19del

FLAURA2 showed that even with the addition of chemotherapy, L858R patients continue to have shorter OS than ex19del patients

48-month OS in 1L EGFRm advanced NSCLC



2 EGFRm NSCLC patients with CNS metastases fair worse than those without

FLAURA2 showed that even with the addition of chemotherapy, patients with CNS metastases continue to have shorter OS than those without

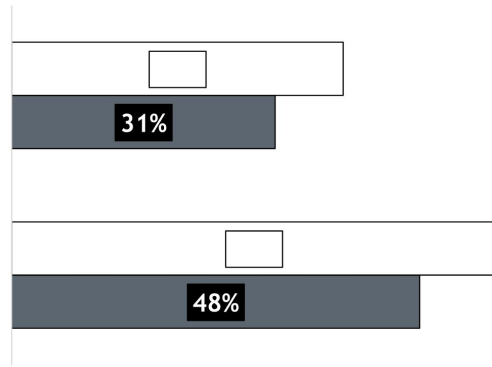
48-month OS in 1L EGFRm advanced NSCLC

□ Osimertinib + Chemo ■ Osimertinib

~40% of patients have CNS metastases at time of initial diagnosis

With CNS metastases

Without CNS metastases

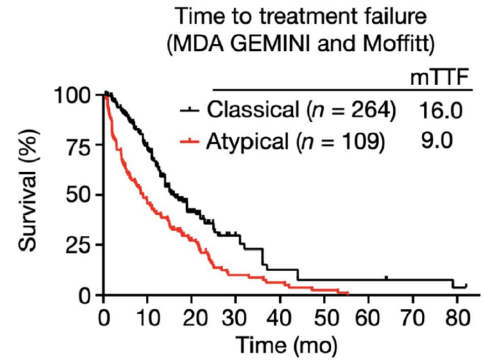
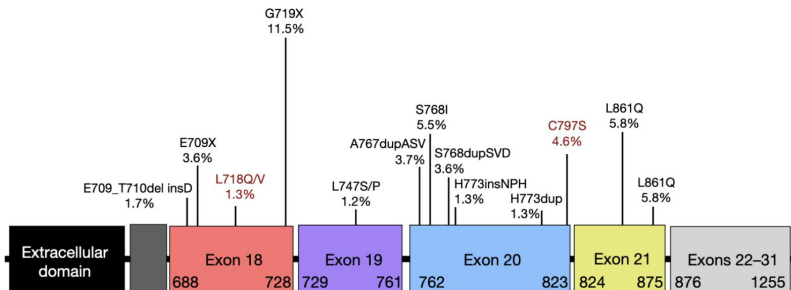


(Includes both L858R and ex19del patients)

3 Patients with atypical activating EGFR mutations have shorter time to treatment failure as compared with classical mutations

Atypical variants account for up to ~20% of EGFRm NSCLC (excluding exon20 insertions); most common atypical mutations are G719X, S768I, and L861Q

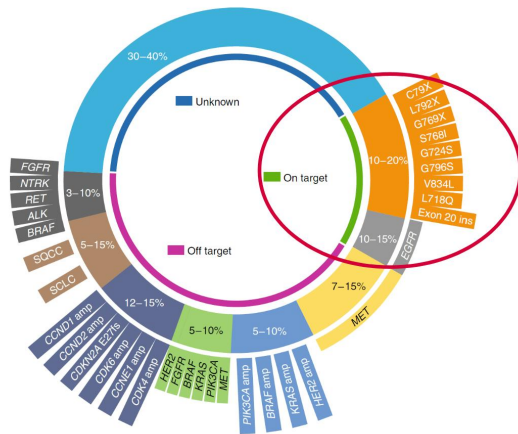
Frequency of atypical EGFR mutations >1%



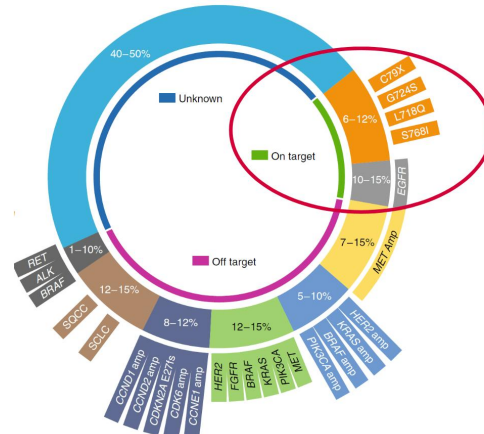
4 On-target mutations in EGFR confer resistance to osimertinib

C797S (most common), L718X, L792F, G796S and others

Up to ~20% after 2nd line Tx



Up to ~12% after 1st line Tx

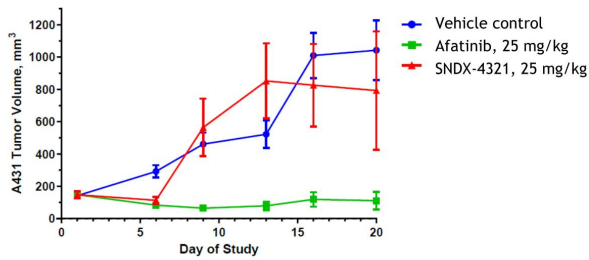


SNDX-4321 is a mutant-selective and potent allosteric EGFR inhibitor

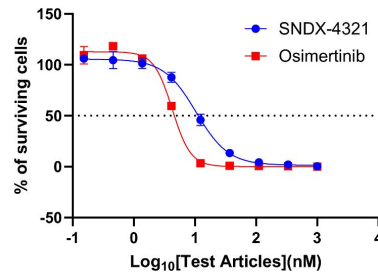
Preclinical studies show:

1. Negligible activity against wild-type EGFR
2. Activity against L858R, with or without common resistance mutations to osimertinib (e.g., C797S)
3. Activity against certain atypical mutations including L861Q, one of the most common atypical variants
4. No activity against exon 19 deletions or exon 20 insertions

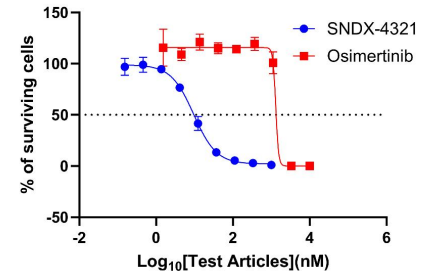
1. A431 (WT harboring tumor)



2. Ba/F3 EGFR L858R

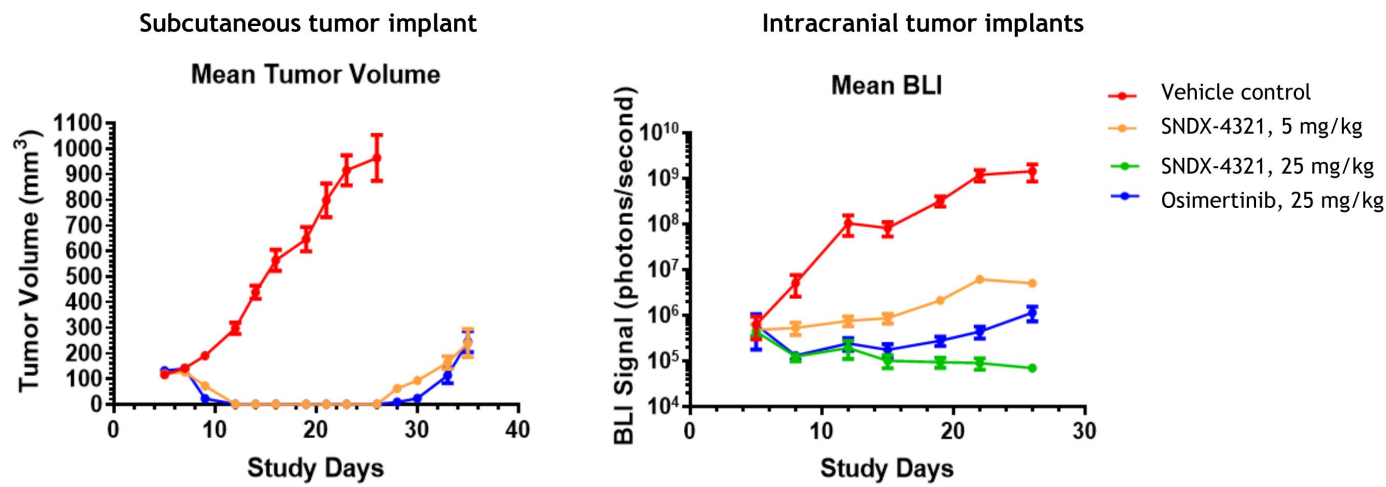


Ba/F3 EGFR L858R C797S



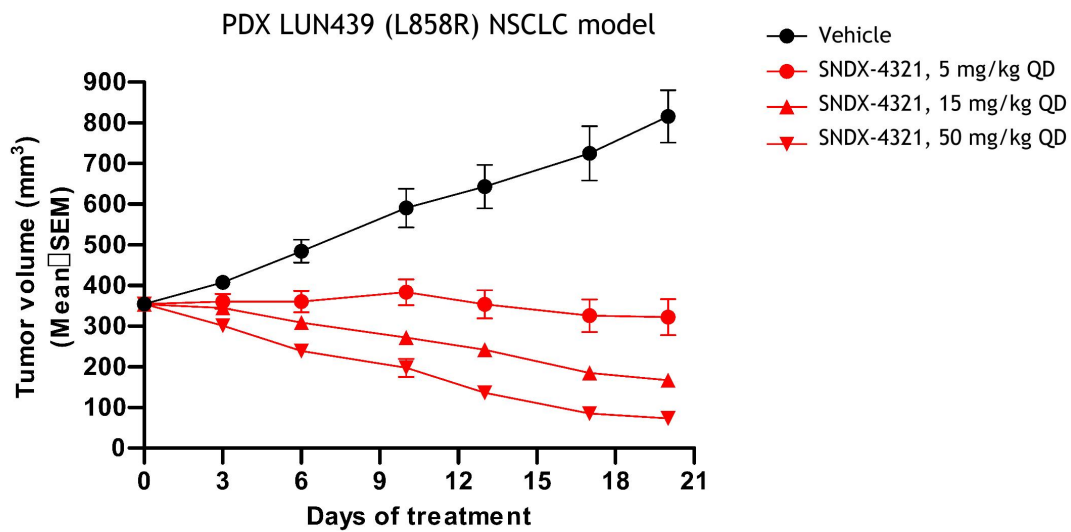
SNDX-4321 is effective in the CNS in intracranial xenograft model

H1975 (L858R/T790M) dual subcutaneous/intracranial model, 21 days dosing, QD



SNDX-4321 is highly active as a single agent in L858R NSCLC PDX models

SNDX-4321 inhibits growth of L858R containing tumors in a dose-dependent fashion

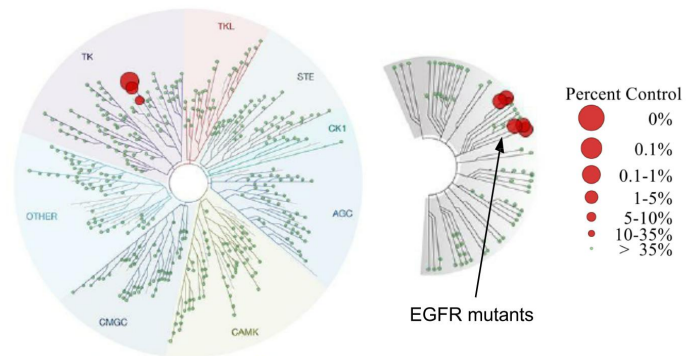


While SNDX-4321 is highly active as a single agent, the more significant opportunity is for combination therapy

Advantages of allosteric approach, especially for combination therapy:

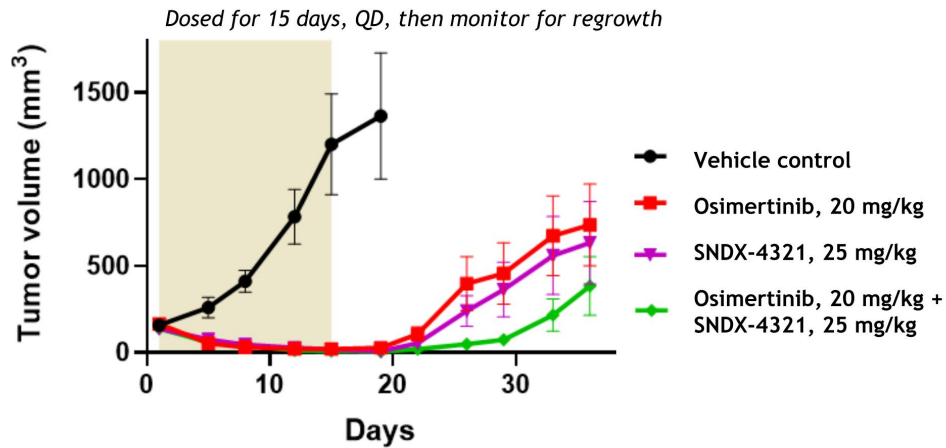
- Bind outside ATP site, yielding excellent kinome-wide selectivity
- Better/different tox profile as compared with ATP-site inhibitors
- Distinct resistance mechanisms - retain activity against ATP-site mutations
- Potential for co-binding with ATP-site compound (e.g., 3rd gen EGFR inhibitor) to deliver enhanced efficacy and delay resistance

DiscoverX KinomeScan, SNDX-4321 @ 1 μ M



In vivo efficacy studies show benefit of combining SNDX-4321 with osimertinib

Delayed tumor regrowth and improved survival in H1975 (L858R/T790M) model



- Combination of SNDX-4321 and osimertinib was well-tolerated
- Clear combination benefit in terms of delayed regrowth and survival

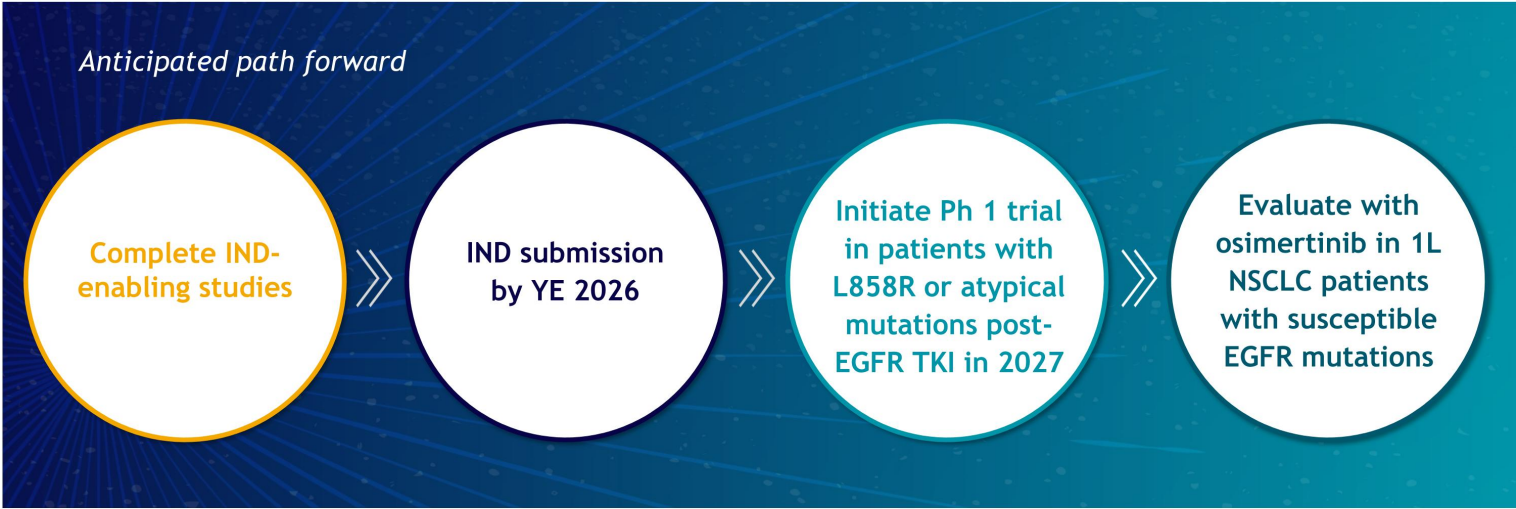
Summary - SNDX-4321

- **Novel, mutant-selective allosteric EGFR inhibitor that could address EGFRm NSCLC populations with significant unmet needs**, such as those with L858R mutations, CNS metastases, atypical mutations, or resistance to osimertinib
- **Highly active as a single agent and in combination with osimertinib** in L858R models with and without common resistance mutations
- **Allosteric approach makes SNDX-4321 an ideal combination partner** with osimertinib or other ATP-site directed EGFR inhibitors
 - Unique binding site - no single mutation confers resistance to both SNDX-4321 and osimertinib; not affected by ATP-site mutations that confer resistance to osimertinib
 - Lack of activity on wild-type EGFR and other kinases bode well for combinability
- **Highly CNS penetrant** with demonstrated efficacy in a brain metastasis model
- **Active against L861Q and other atypical mutations** in preclinical models

SNDX-4321 - Next steps

Nick Botwood, MBBS
Head of Research & Development and Chief Medical Officer

SNDX-4321 clinical development plan designed to efficiently generate safety and efficacy data that support allosteric approach in EGFRm NSCLC



Demonstration of monotherapy activity anticipated in early 2028

Q&A - SNDX-4321



Michael A. Metzger
Chief Executive Officer and Director



Nick Botwood, MBBS
Head of R&D and Chief Medical Officer



Peter Ordentlich, PhD
Chief Scientific Officer and Founder



Michael J. Eck, MD, PhD
Professor, Department of Cancer Biology, Dana-Farber Cancer Institute; Professor, Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School

Closing remarks

Michael A. Metzger
Chief Executive Officer and Director

Syndax is delivering for patients and driving substantial long-term value



TRACK RECORD
OF SUCCESS

Proven ability to translate scientific discoveries into novel therapies, with 3 FDA approvals between 2 drugs and thousands of patients treated



WORLD-CLASS R&D
CAPABILITIES

Fully integrated biotech with deep expertise and strong partnerships with leading researchers



DEEP AND GROWING
PIPELINE

Robust late- and early-stage pipeline with multiple near-term catalysts in 2H26 and blockbuster opportunities



SOLID FINANCIAL
POSITION

Driving towards profitability with growing revenues; fully funded to advance late-stage trials and pipeline assets

Continued focus on driving revenue growth and pipeline progress, with multiple near-term catalysts and blockbuster opportunities

Anticipated milestones

Revuforj (revumenib)
& SNDX-62122

Acute Leukemia

- Advance global enrollment in pivotal 1L trials of revumenib
- New 1L, maintenance, real-world, and R/R revumenib data in 2H26 + beyond
- Publish R/R NUP98r AML data in 4Q26

Myelofibrosis

- Initiate Ph 1/2 proof-of-principle trial with revumenib in 4Q26; initial data 2H27
- Submit IND and initiate Ph 1 SNDX-62122 trial in 2027

Niktimvo
(axatilimab-csfr)

Chronic GVHD

- Ph 2 axatilimab + ruxolitinib data in 4Q26
- Ph 3 axatilimab + corticosteroid data early 2028

Idiopathic pulmonary fibrosis

- Ph 2 MAXPIRe data in 4Q26

SNDX-4321

EGFRm NSCLC

- Submit IND by YE 2026
- Initiate Ph 1 trial in 2027; initial data early 2028

2

Ph 2 readouts
in 4Q26

Multiple

revumenib readouts
in acute leukemia
in 2H26 + beyond

4

assets in the
clinic in 2027

