



Overview

May 2026



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Targeting STAT3: Central Mediator of Inflammatory and Proliferative Diseases



Deep expertise in STAT3 biology

- Unlocking highly-validated target within inflammatory and proliferative diseases
- Demonstrated target engagement and disease modification across animal models



Potential to serve as a disease-modifying therapy

- Demonstrated enhanced biological activity in fibrotic cancers in P1
- Observed reductions in inflammation and fibrosis marker (IL-6) in IPF¹ P2
- Evaluating mono- and combination therapy in fibrosis-driven HCC² P2



TTI-109: designed to enhance delivery of STAT3 inhibitor & improve tolerability

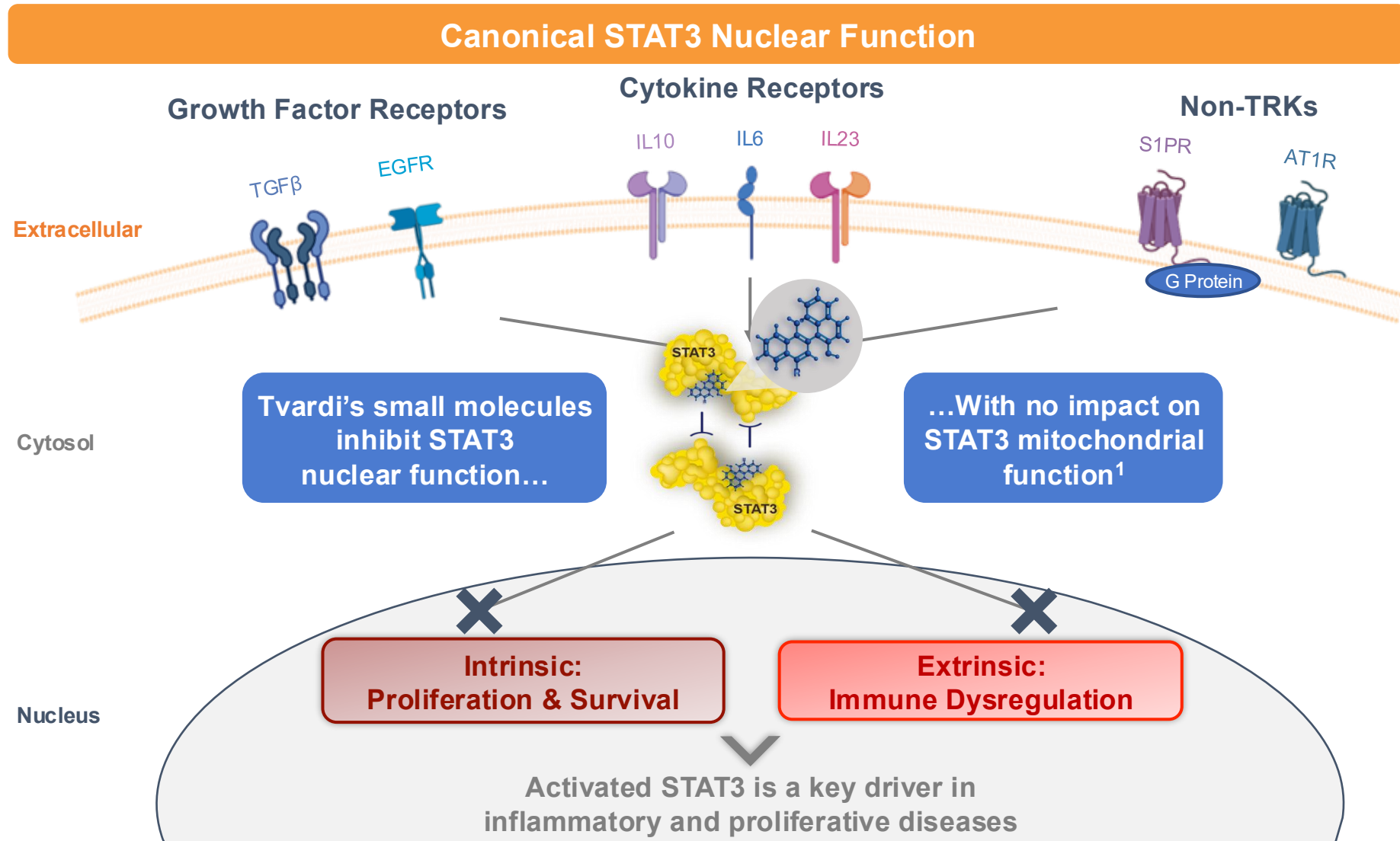
- Prodrug designed to rapidly convert to active TTI-101 in the blood - IND³ filed June 2025
- P1 trial in healthy volunteers ongoing to demonstrate PK⁴, bioequivalence and tolerability



Multiple near-term data catalysts expected

- TTI-109: P1 healthy volunteer topline data in 2Q:2026
- TTI-101: P1b/2 HCC² topline data in 2H:2026

STAT3's Canonical Function Plays a Central Role in Inflammatory and Proliferative Diseases

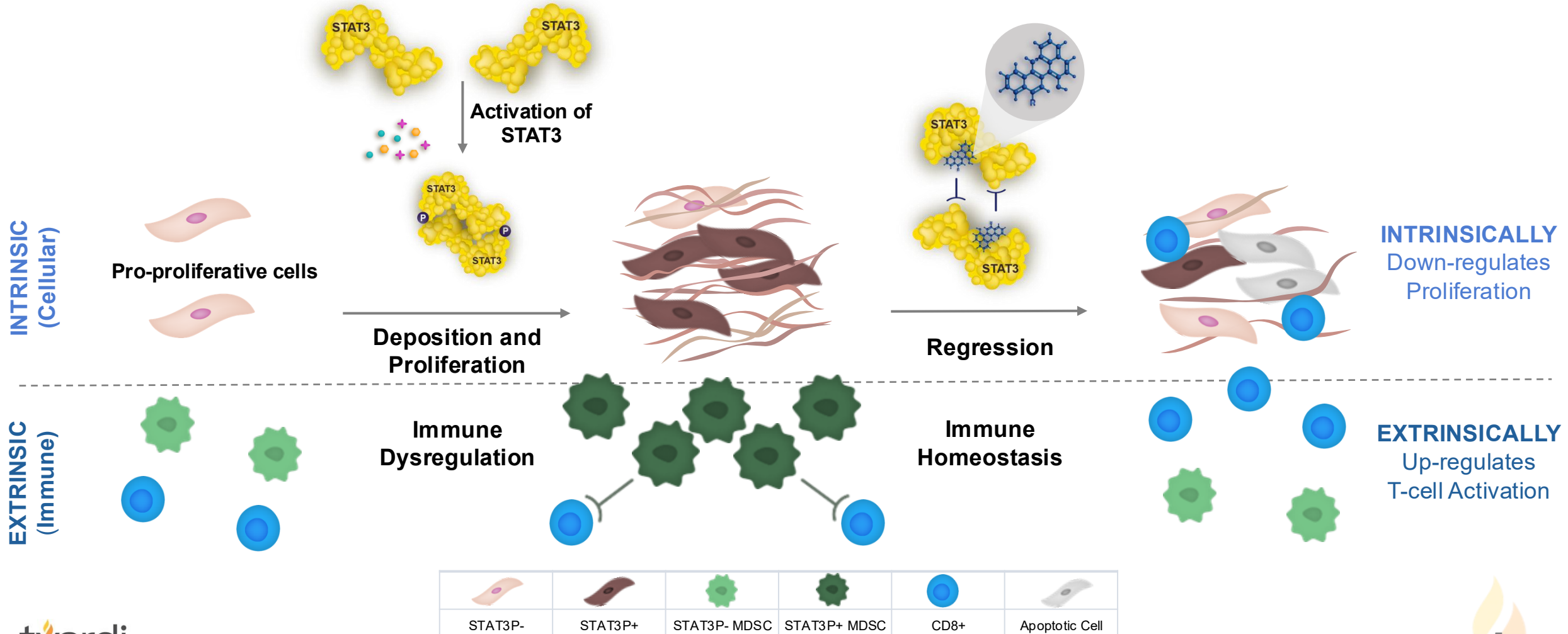


STAT3's Dual Mechanism of Action in Inflammatory and Proliferative Diseases

Mechanism of the Canonical Pathway

Tvardi's Approach

Tvardi's Impact



Seasoned Leadership: Deep R&D and Operational Expertise

Management Team



Imran Alibhai, PhD CEO & Director



Dan Conn, JD, MBA CFO



John Kauh, MD CMO



Scientific Advisory Board

David Tweardy, MD Founder & Advisor



Ron DePinho, MD Founder & Advisor



Keith Flaherty, MD Advisor



Board of Directors

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Our Pipeline

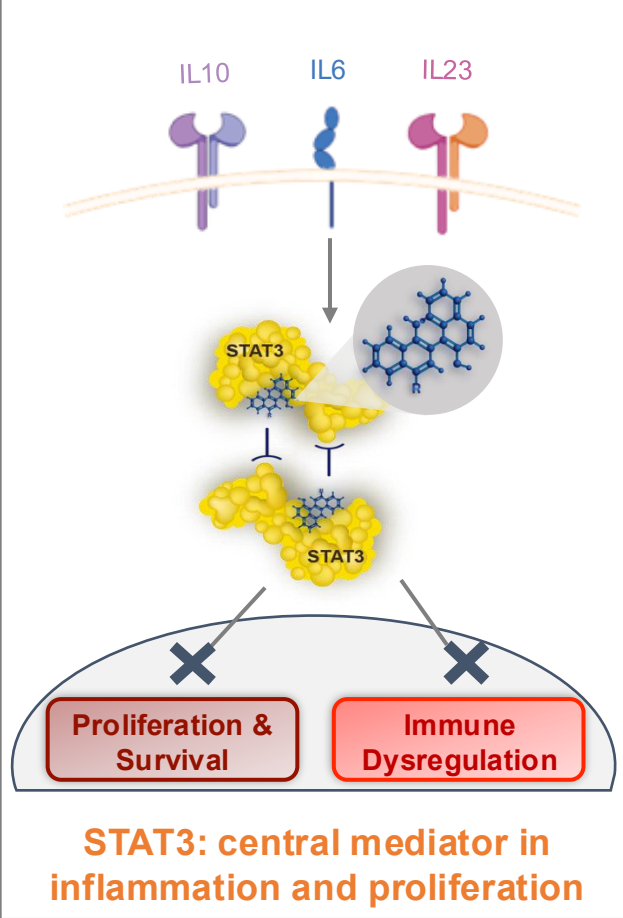
Program	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestone
TTI-109	Inflammatory / Proliferative Disease ¹		Phase 1			2Q:2026 Phase 1 Healthy Volunteer data
TTI-101	Hepatocellular Carcinoma		Phase 1b/2			2H:2026 Phase 1b/2 topline data
TTI-101	Idiopathic Pulmonary Fibrosis		Phase 2			Topline Data Reported Oct 2025 Analysis of Pooled Patient Subgroup Reported Jan 2026

1. We plan to commence clinical trials in inflammatory/proliferative disease driven by dysregulated STAT3 pending IND submission and FDA feedback.

TTI-101 in IPF

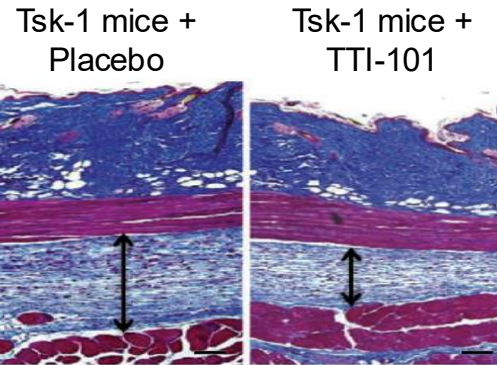
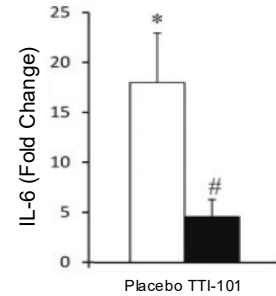
Our STAT3 Inhibitors are Designed to Address the Unmet Need in Inflammatory and Proliferative Diseases

Well-Established Target

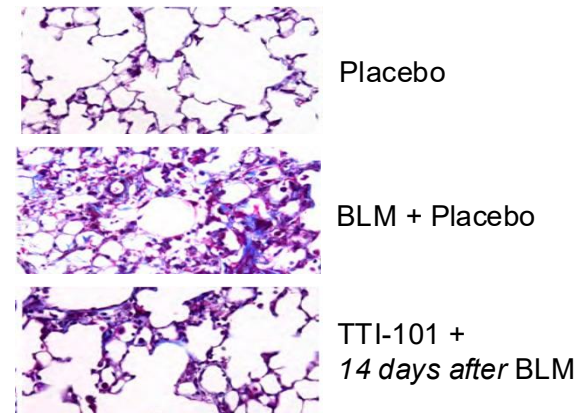
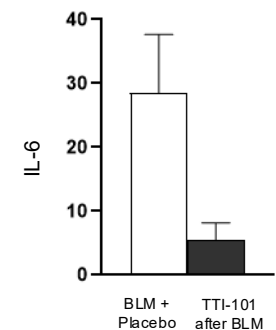


Preclinical Biological Activity

SSC (GEM)¹



IPF (BLM)^{2,3}

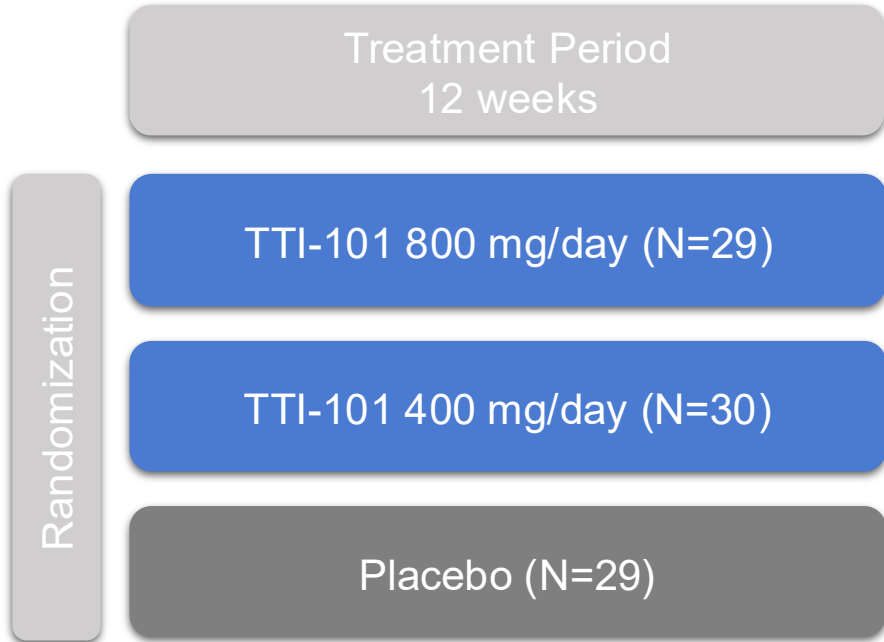


↓ IL-6 levels and ↓ fibrosis in models of proliferative disease

Initial Clinical Proof of Mechanism: IPF



REVERT_{IPF}: Preliminary Conclusions Released October 2025



- Placebo arm overperformed compared to historical data
- FVC change from baseline overlapped between treatment arms, with large variability within each cohort
- Treatment emergent adverse events resulted in early discontinuations leading to a small number of patients available for efficacy analysis at the 12-week time point

Patient Characteristics

Characteristic	Placebo (n=29)	TTI-101 400mg/day (n=30)	TTI-101 800mg/day (n=29)
Age	72.0 (9.3)	72.4 (6.9)	72.8 (7.7)
Sex, Male – no. (%)	23 (79.3)	25 (83.3)	21 (72.4)
Time since diagnosis of IPF – yrs., mean (SD)	2.4 (2.0)	2.6 (1.7)	2.5 (1.5)
Standard of care use, nintedanib – no. (%)	17 (58.6)	18 (60.0)	17 (58.6)
FVC – L, mean (SD)	2.59 (0.75)	2.84 (0.71)	3.03 (1.00)
Percent of predicted FVC (ppFVC), mean (SD)	70.08 (16.72)	74.05 (13.38)	81.12 (19.90)
GI comorbidity at baseline, mean (%) ¹	21 (72.4)	26 (86.7)	20 (69.0)

GI comorbidities were prevalent among the patient population

Summary of Treatment-Emergent Adverse Events

Characteristic	Placebo (n=29)	TTI-101 400mg/day (n=30)	TTI-101 800mg/day (n=29)
Any TEAEs	21 (72.4)	30 (100)	26 (89.7)
TEAE leading to death	1 (3.4)	1 (3.3) ¹	1 (3.4)
TEAE leading to discontinuation	3 (10.3)	17 (56.7)	18 (62.1)
Gastrointestinal disorders		6 (20.0)	11 (37.9)
Investigations (Labs) ²		4 (13.3)	6 (20.7)
Respiratory, thoracic and mediastinal disorders	1 (3.4)	5 (16.7)	3 (10.3)

Incidence of respiratory adverse events leading to discontinuation is consistent with adverse events expected in this patient population

Treatment-Emergent Adverse Events Resulting in Discontinuations by Concomitant Nintedanib Use

Dose	Placebo (n=29)		TTI-101 400mg/day (n=30)		TTI-101 800mg/day (n=29)	
	Nintedanib (n=17)	Placebo (n=12)	TTI-101 + Nintedanib (n=18)	TTI-101 alone (n=12)	TTI-101 + Nintedanib (n=16)	TTI-101 alone (n=13)
Reason for discontinuation by concomitant nintedanib use						
Gastrointestinal disorders			2	4	7	4
Investigations (Labs) ¹			4		4	2
Respiratory, thoracic and mediastinal disorders		1	3	2	1	2

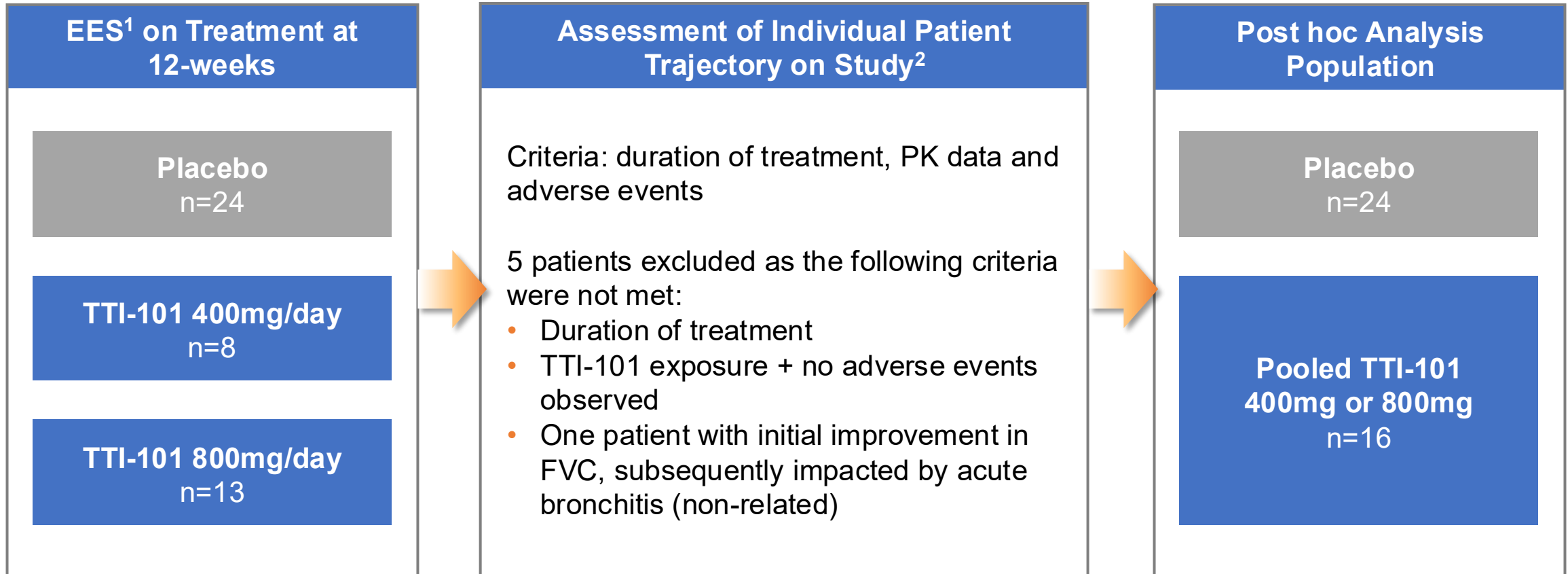
Majority of discontinuations were observed with concomitant nintedanib

Most Common Adverse Events Reported in INPULSIS vs REVERT_{IPF} Trials

	INPULSIS-1 and 2 ¹		Pooled TTI-101	
	Placebo (N=423)	Nintedanib (N=638)	TTI-101 alone (n=25)	TTI-101 + Nintedanib (n=34)
Any adverse event	379 (89.6)	609 (95.5)	23 (92.0)	33 (97.1)
Diarrhea	78 (18.4)	398 (62.4)	11 (44.0)	25 (73.5)
Nausea	28 (6.6)	156 (24.5)	4 (16.0)	10 (29.4)
Nasopharyngitis	68 (16.1)	87 (13.6)	1 (4.0)	0 (0.0)
Cough	57 (13.5)	85 (13.3)	3 (12.0)	4 (11.8)
Progression of IPF	61 (14.4)	64 (10.0)	2 (8.0)	2 (5.9)
Bronchitis	45 (10.6)	67 (10.5)	2 (8.0)	0 (0.0)
Upper respiratory tract infection	42 (9.9)	58 (9.1)	0 (0.0)	4 (11.8)
Dyspnea	48 (11.4)	49 (7.7)	1 (4.0)	4 (11.8)

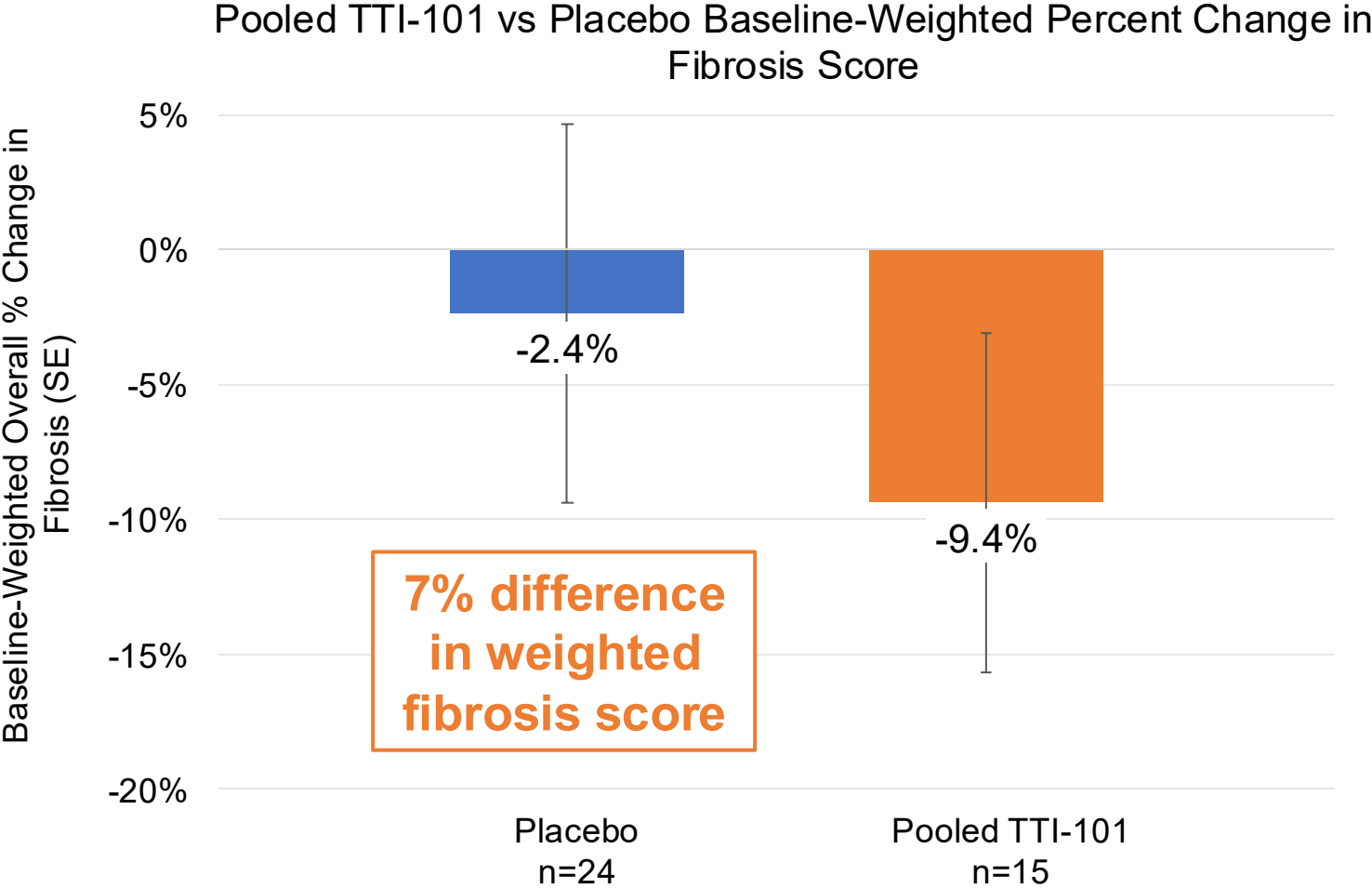
- TTI-101 treated patients reported adverse events consistent with overall expectations for an IPF population
- However, addition of nintedanib meaningfully increased incidence of diarrhea

Preliminary Conclusions and Additional Analyses



¹The Efficacy Evaluable Set (EES) consists of all patients defined as all randomized patients who received at least one dose of IP and have at least one post-baseline acceptable or borderline acceptable forced vital capacity (FVC) result while on-treatment. On treatment is defined as spirometry values obtained when actively receiving IP at protocol-defined collection time points and spirometry values when on IP hold at protocol-defined collection time points; excludes spirometry values collected > 14 days of last IP dosing after they have been permanently discontinued from IP. ² Post hoc analysis population: The additional analysis was limited to patients who were exposed to study drug for 12 weeks. One patient was not exposed to TTI-101 at 12 weeks; one patient was removed from the analysis due to receiving less than 60% of the expected dosing; two patients were removed due to no measurable TTI-101 observed in the blood as well as no reported adverse events; and one additional patient was removed as an outlier for the 12-week analysis as their pulmonary function initially improved on treatment, but was subsequently severely impacted by acute bronchitis deemed unrelated to study drug.

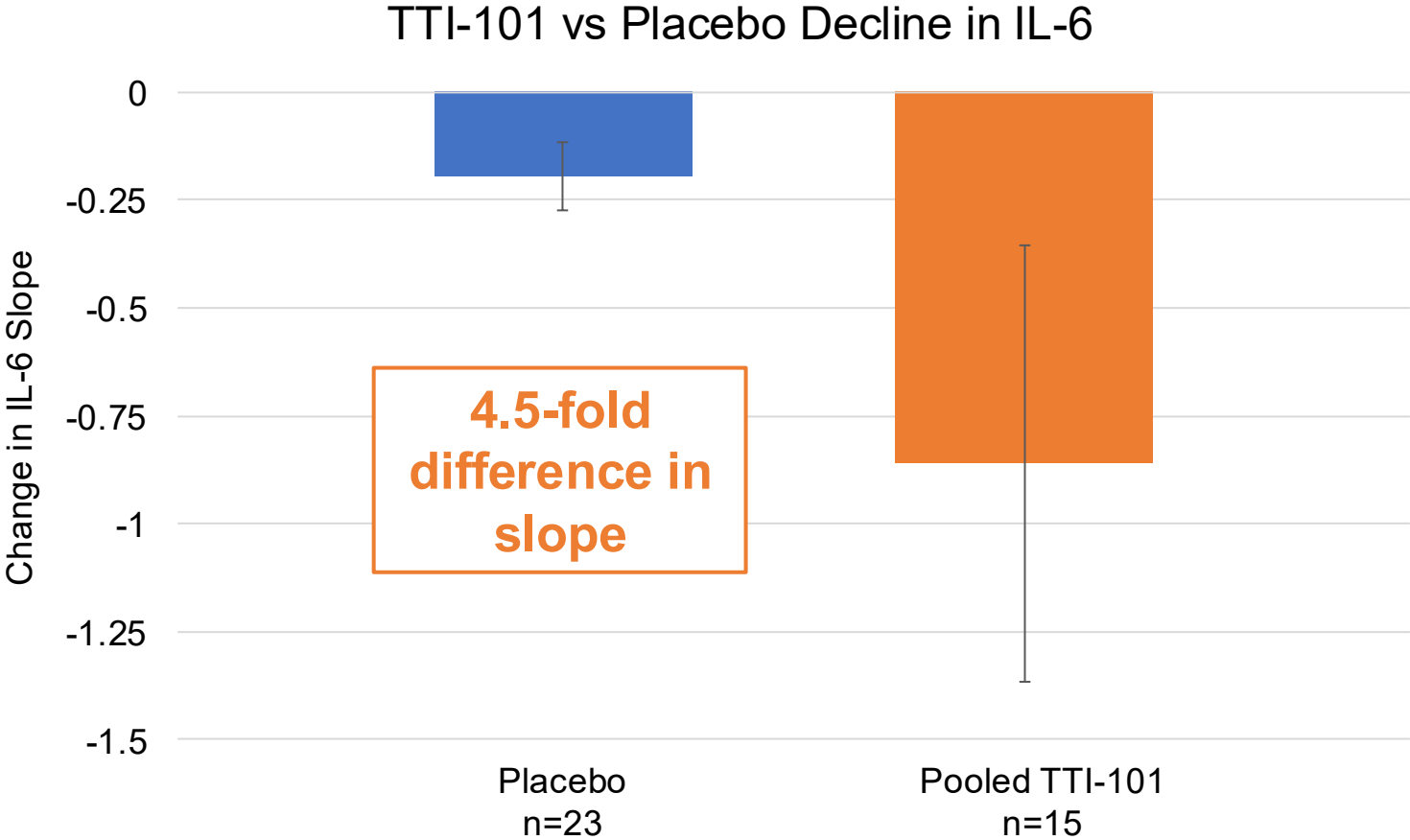
TTI-101-treated Patients Demonstrated Greater Decline in Fibrosis Score (Baseline to 12 Weeks) vs Placebo



Greater decrease in fibrosis score (improvement in fibrosis) among patients treated with TTI-101 (either arm) vs placebo

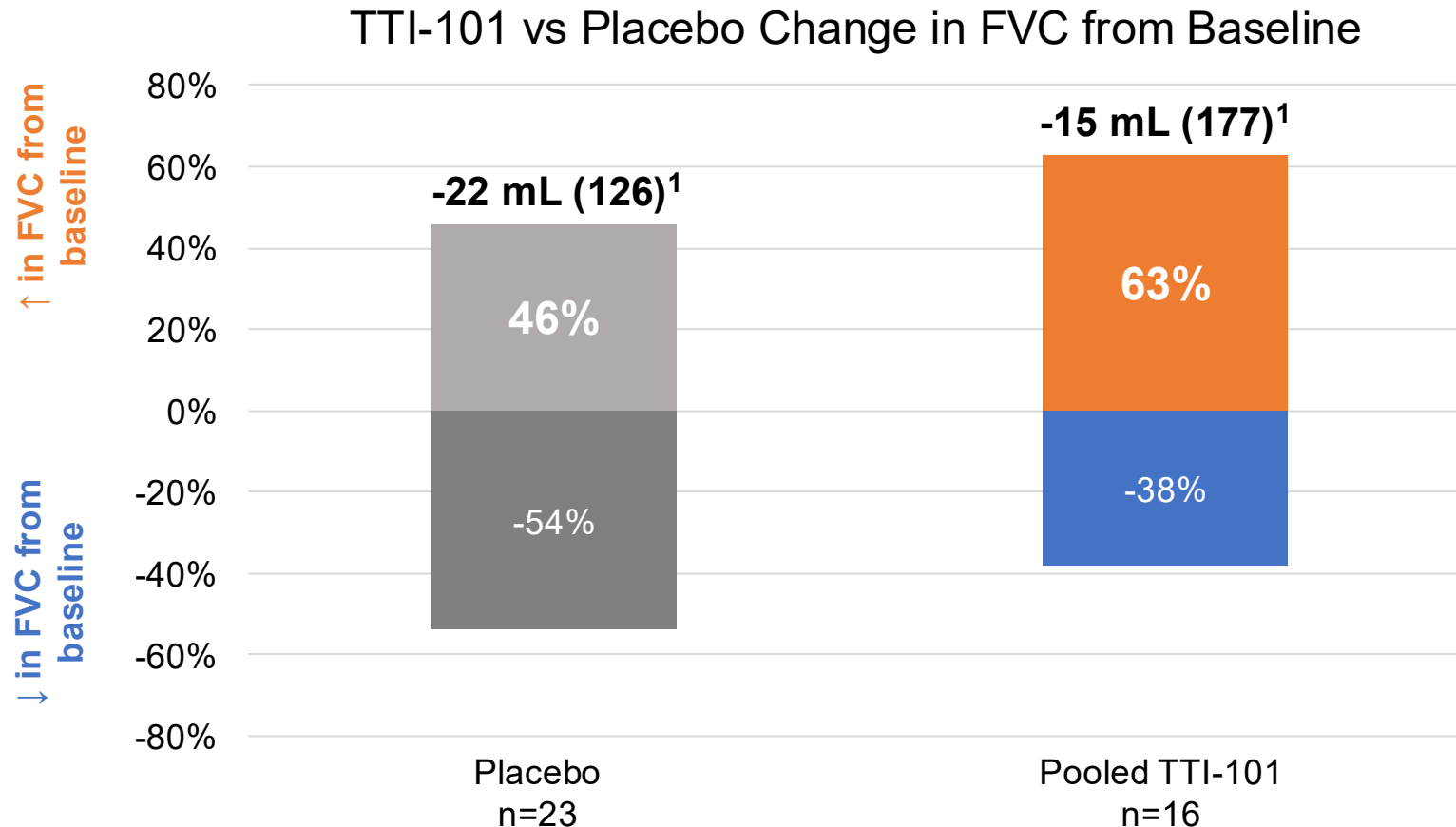
7% difference in weighted fibrosis score

TTI-101-treated Patients Demonstrated Greater IL-6 Decline vs Placebo



IL-6 is a key pro-inflammatory cytokine that signals through STAT3; inhibition of STAT3 is expected to reduce downstream inflammatory signaling associated with disease

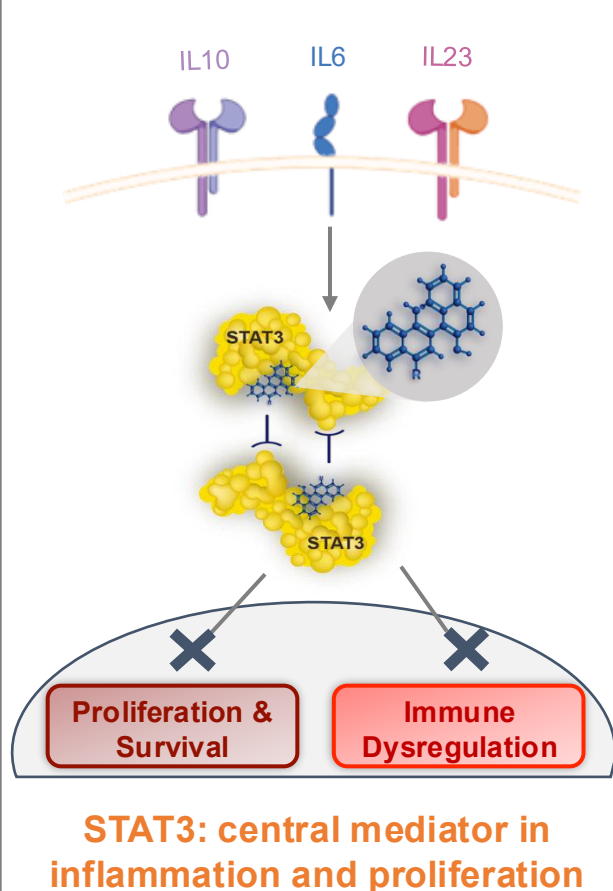
A Greater Proportion of TTI-101-treated Patients Demonstrated Increase in FVC vs Placebo



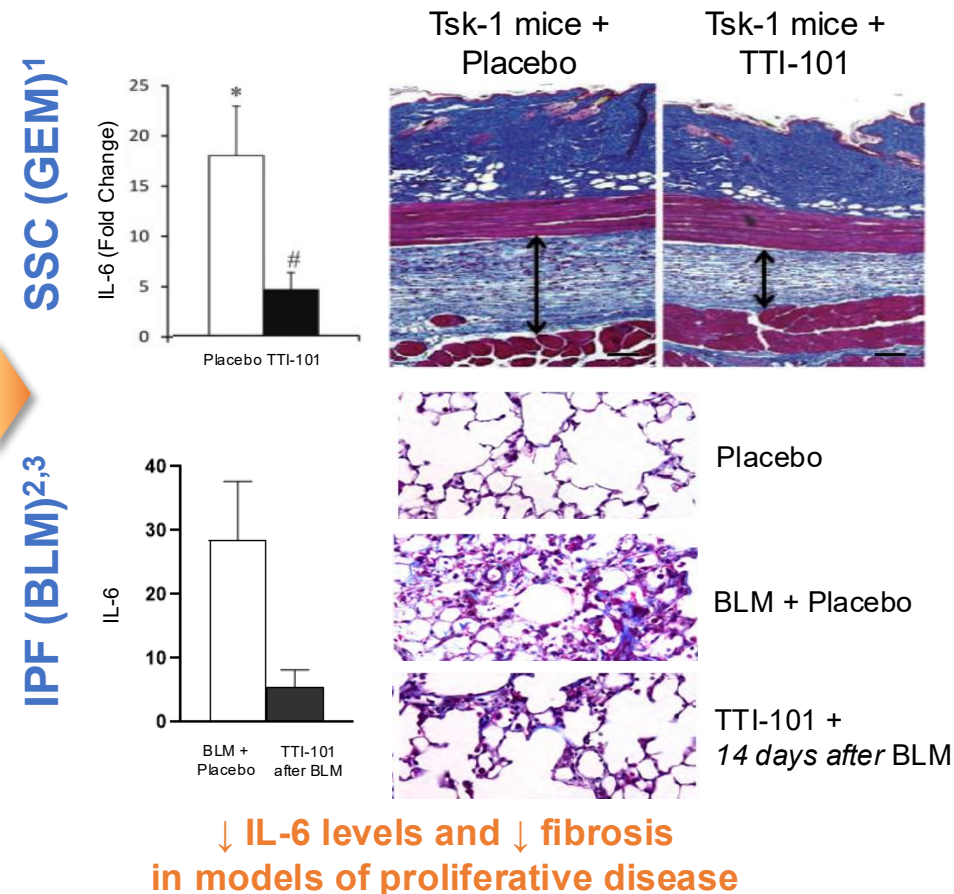
63% of patients treated with TTI-101 demonstrated an increase in FVC at 12 weeks, and less of a decline when compared to the REVERT and historical placebo groups

Our STAT3 Inhibitors are Designed to Address the Unmet Need in Inflammatory and Proliferative Diseases

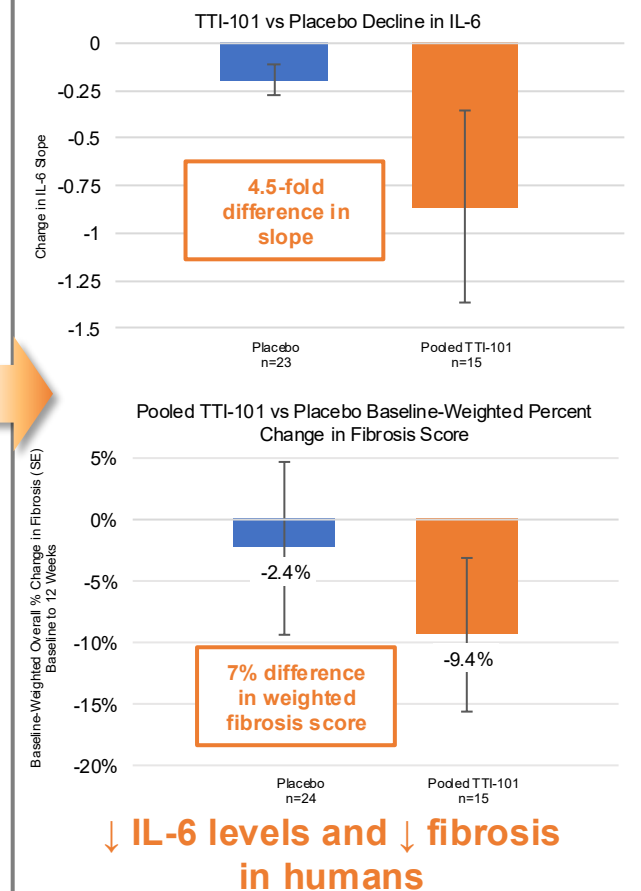
Well-Established Target



Preclinical Biological Activity



Clinical Proof of Mechanism



REVERT_{IPF} Provided Additional Proof of Mechanism for STAT3 Inhibition in Inflammatory and Proliferative Diseases

Fibrosis

Patients treated with TTI-101 demonstrated a ~9% vs ~2% placebo decrease from baseline fibrosis score

IL-6 Biomarker

IL-6, a key pro-inflammatory cytokine that signals through STAT3, was observed to have a greater decline among TTI-101-treated patients vs placebo

Pulmonary Function

Placebo overperformed vs historical controls; TTI-101-treated patients demonstrated less of an FVC decline vs REVERT and historical trial placebo groups

Safety

Adverse events in TTI-101-treated patients were consistent with IPF population; however, addition of nintedanib resulted in increased diarrhea incidence

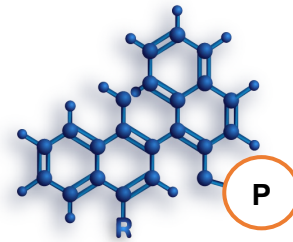
TTI-109 is designed to enhance delivery of STAT3 inhibitor and improve tolerability for future studies

TTI-109

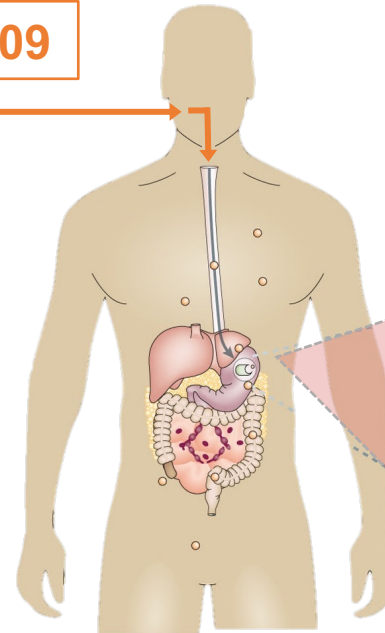
TTI-109 Designed as a Prodrug to Enhance Delivery of STAT3 Inhibitor

TTI-109 Design

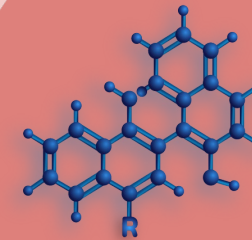
- Preserve mechanism of action
 - Clinical activity and tolerability evaluated in >300 patients with TTI-101
- Improve drug delivery
- Diminish GI exposure
- Patent protection



TTI-109



Phosphate prodrug
designed to rapidly
convert to active
TTI-101 in the blood

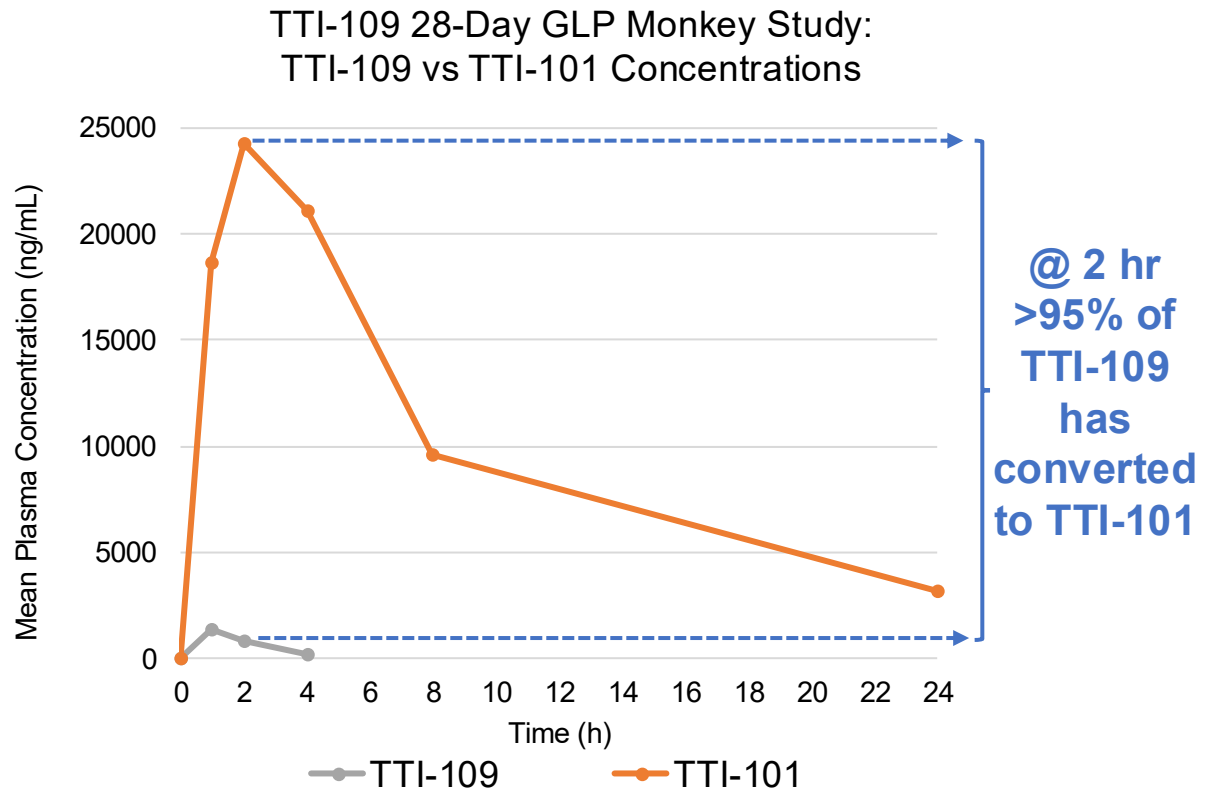


TTI-101

TTI-109 Possesses Desired Characteristics of Next STAT3 Inhibitor

IND-Enabling GLP Results

- ✓ **No toxicology findings with TTI-109;** consistent with absence of findings with TTI-101
- ✓ Systemic exposures provided a **large safety margin** for anticipated clinical doses
- ✓ At equal molar doses, **TTI-109 gave equal exposures of TTI-101**
- ✓ **Rapidly converted** to active moiety TTI-101



IND Filed June 2025

Phase 1 Healthy Volunteer Study of TTI-109 Relative to TTI-101

Part A: Single Ascending Dose (SAD)

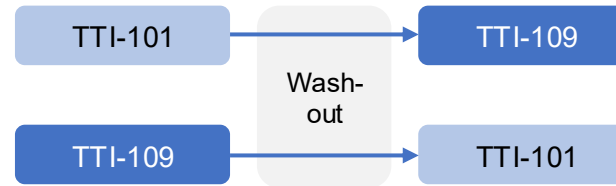


Single dosing of TTI-109 vs placebo

Objectives:

- Confirm rapid PK conversion of TTI-109 to TTI-101 in humans
- Demonstrate dose-dependent increases in TTI-101 exposures from TTI-109 administration
- Characterize safety/tolerability

Part B: Bioequivalence Crossover Study



Randomized sequence of treatment

Objectives:

- Evaluate TTI-109 vs TTI-101: within-subject comparison
- Confirm equivalent exposures of active moiety with TTI-101 and TTI-109 dosing

Part C: Multiple Ascending Dose (MAD)



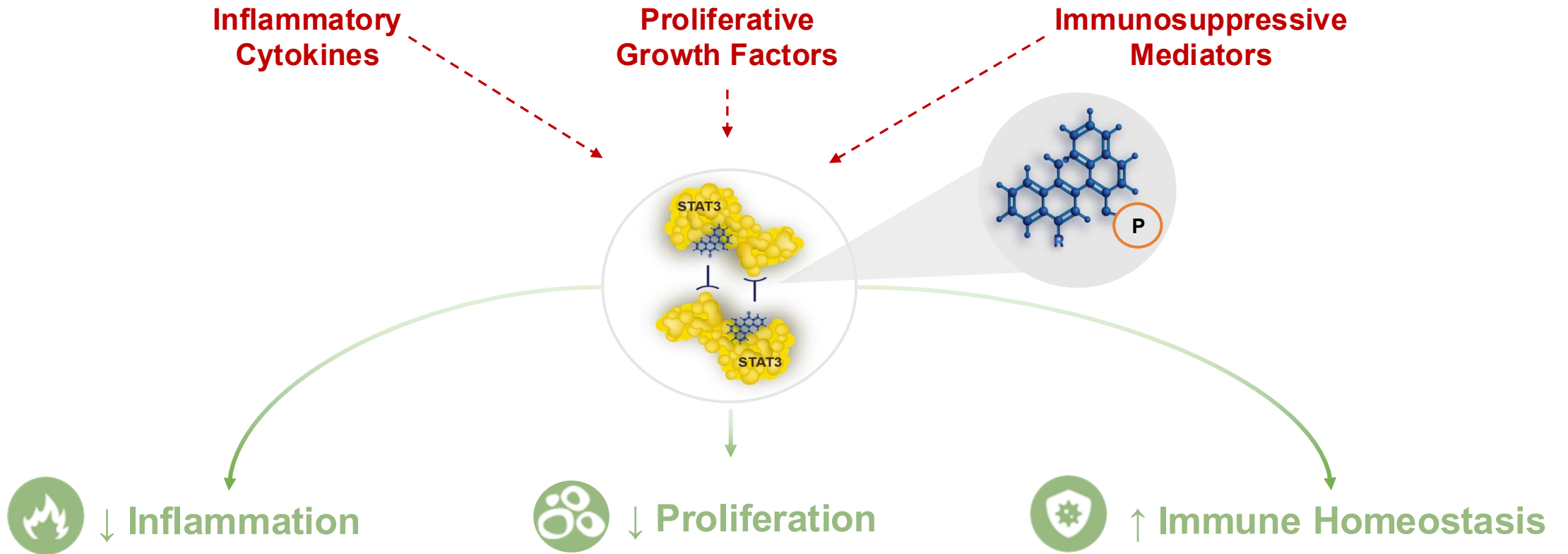
Repeat dosing of TTI-109 vs placebo; TTI-109 vs TTI-101 at RP2D

Objectives:

- Characterize safety/tolerability of TTI-109 relative to TTI-101 after repeated doses
- Evaluate steady state PK parameters

Phase 1 Data Expected 2Q 2026

TTI-109: Potential Reach Across STAT3-Driven Diseases



— Broad Relevance Across Inflammatory and Proliferative STAT3-Driven Conditions —

Dermatologic | Gastrointestinal | Hemopoietic | Autoimmune

TTI-101 in HCC

TTI-101 Reversed Multiple Pathogenic Steps of Liver Cancer in a NASH-induced HCC Model

STAT3-mediated pathogenesis

TTI-101 STAT3-inhibition in NASH-induced HCC

Inflammation



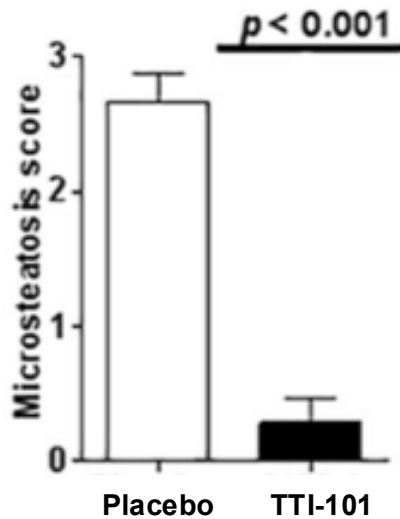
Fibrosis/Cirrhosis



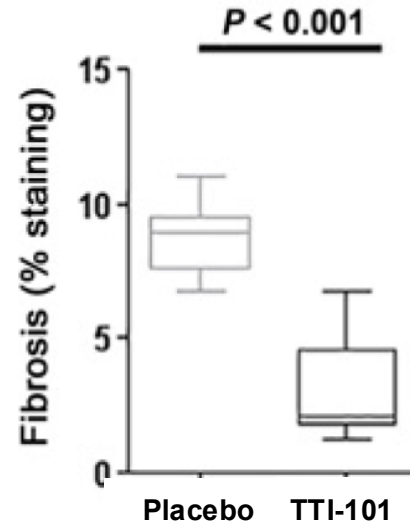
HCC



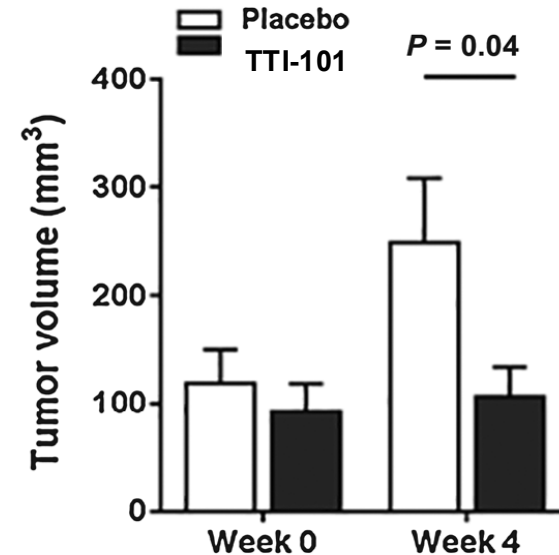
Microsteatosis



Fibrosis

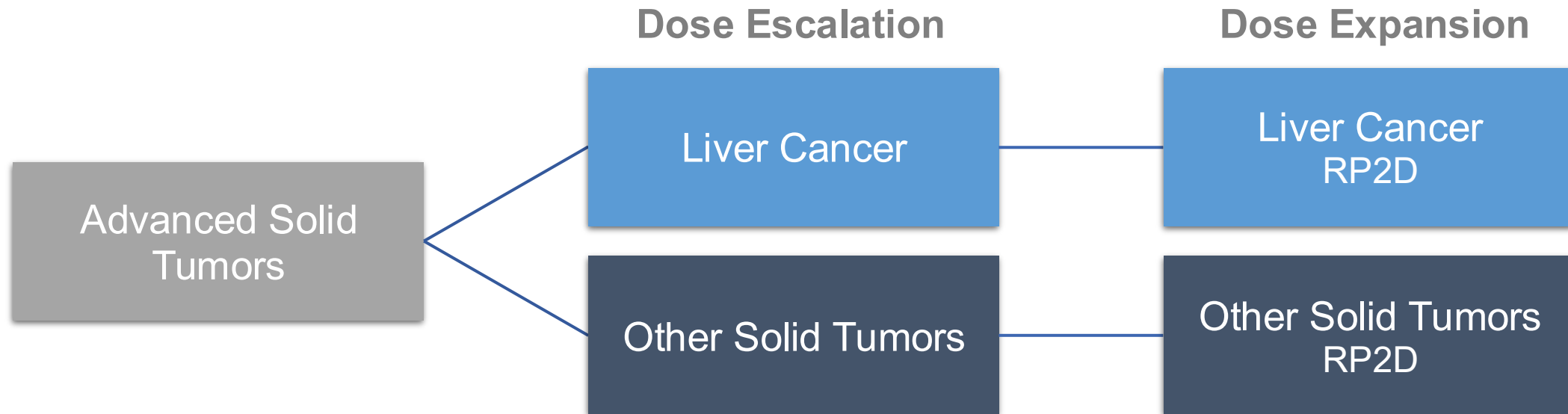


Tumor volume



After formation of tumors at 11 months, we observed treatment with TTI-101 therapeutically reduced inflammation, fibrosis, and tumor growth

Phase 1 Clinical Trial: First in Human TTI-101 Monotherapy Study Design



Objectives:

- **Primary:** Maximum tolerated dose, safety, and pharmacokinetics
- **Secondary:** Clinical efficacy and pharmacodynamics

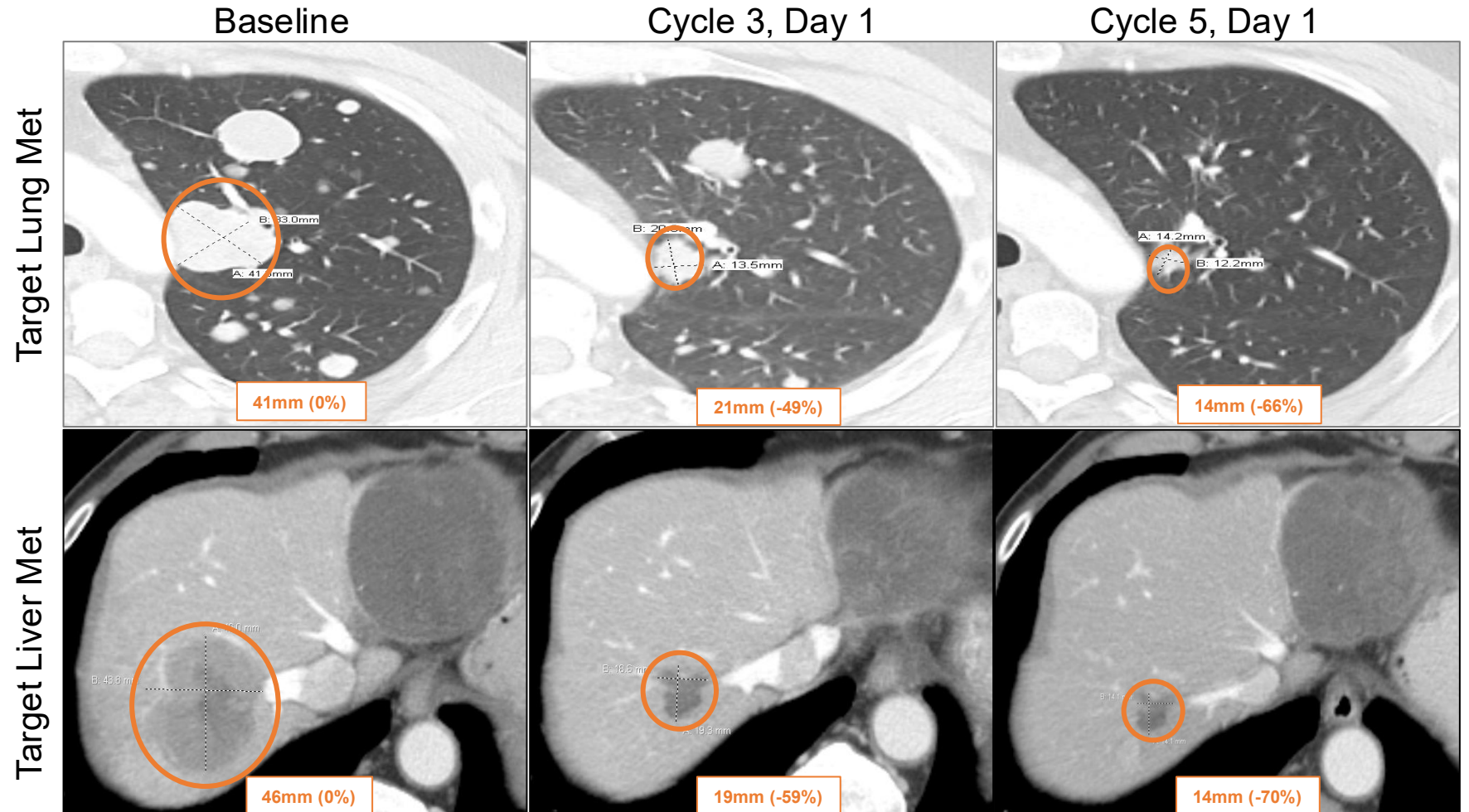
Phase 1 Clinical Trial: TTI-101 Monotherapy Led to Durable Partial Responses in Fibrotic Tumors

Partial Responder A: HCC

- Failed sorafenib, pembro, nivo, nivo+bev
- Best Response: **42% Reduction in Sum of Targets Overall**
- Sustained PR for 10 months

Partial Responder B: HCC

- Failed lenvatinib, nivo
- Best Response: **66% Reduction in Sum of Targets Overall**
- Sustained PR for 14 months



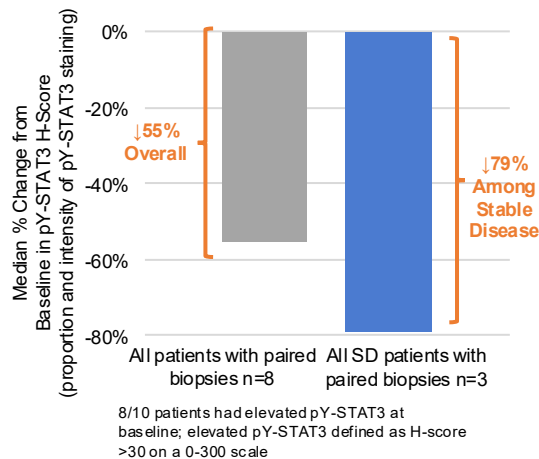
Diameter Length in mm (% Change from Baseline)

Phase 1: TTI-101 Monotherapy Clinical Trial Summary

PK / PD

- Exposures in humans above the level required for efficacy in preclinical inflammatory and proliferative models
- Linear PK from DL1-3
- Exposures above IC_{50} for STAT3 induced growth*
- Exposure plateaued at DL3, resulting in a RP2D of 12.8mg/kg/day
- 100% of patients with elevated pY-STAT3 levels at baseline demonstrated decrease within ~6 weeks of TTI-101 therapy
- 55% decrease in pY-STAT3 overall; 79% in SD

Paired Biopsies after ~6 weeks of TTI-101



Tolerability

- Well-tolerated BID oral dosing
- No DLTs

TRAEs Occurring in >10% of Patients

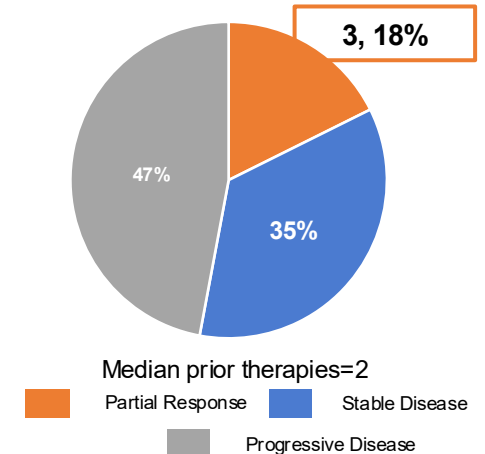
Formulation	F1 N=15		F2 N=47		F3 N=7**	
	G1/2	G3	G1/2	G3	G1/2	G3
Diarrhoea	3 (20)	3 (20)	16 (34)	6 (13)	2 (29)	0 (0)
Nausea	4 (26)	0 (0)	6 (13)	1 (2)	0 (0)	1 (14)
Fatigue	6 (40)	0 (0)	4 (8)	0 (0)	0 (0)	0 (0)
Elevated ALT/AST***	1 (7)	1 (7)	1 (2)	4 (8)	1 (14)	1 (14)
Dose reduction	3 (20)		2 (4)		0 (0)	
Dose discont.	0 (0)		2 (4)		0 (0)	

*Most severe AE counted per subject by grade (G1/2=grade 1 or 2, G3=grade 3) **5 subjects started on F2 and transitioned to F3 ***Elevated alanine aminotransferase/aspartate aminotransferase (ALT/AST) is the sum of elevated ALT and AST events

Biological Activity

- Enhanced biological activity in fibrotic cancers with ORR that exceeds current standard of care in HCC
- Current expected ORR in 2L HCC is <5%

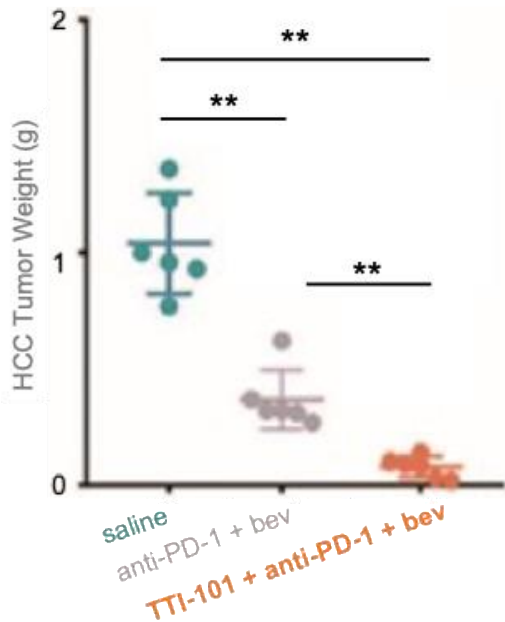
Best Overall Response Among HCC Patients, N=17



Strong Rationale for TTI-101 and ICI Combination Therapy

Preclinical Model

TTI-101 additive to 1L SoC (ICI + Bev)¹



POC Established for STAT3 Inhibition + ICI

Ph 2: Danvatirsen (STAT3 ASO) + Durvalumab (ICI) in 2L HNSCC²

	Durva ³		Dan+Durva ²
ORR	9%	→	23%
CR	0%	→	7%

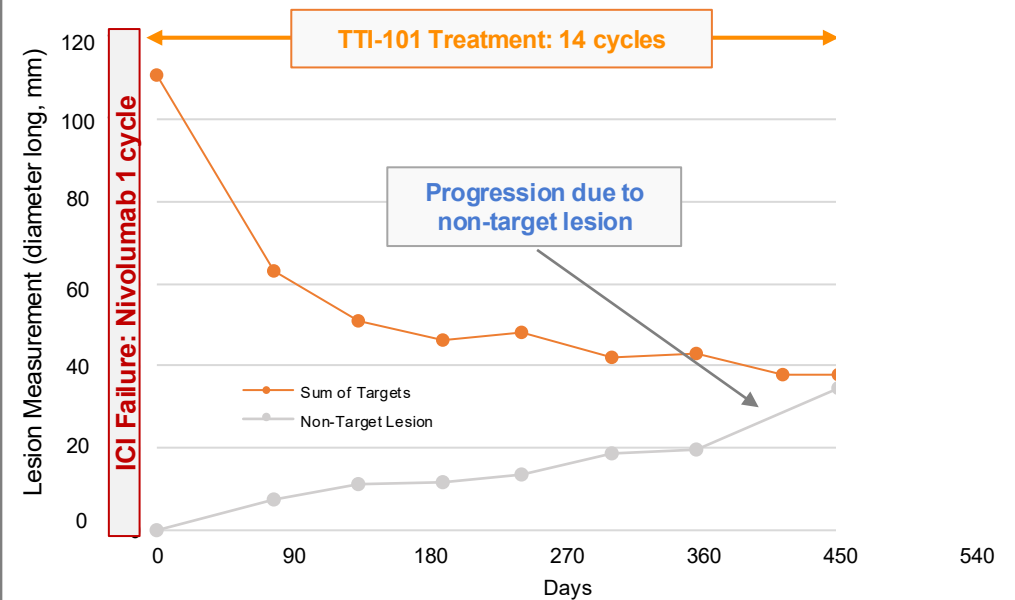
Danvatirsen key limitations:

- Observed AEs: Thrombocytopenia and transaminitis
- Onerous dosing: IV 3x week 1 then Q weekly
- Poor PD: Inhibition of STAT3 not observed in tumor, only in stroma

Danvatirsen development suspended by AZN/Ionis

Phase 1 Trial Responder Overcame ICI Resistance After TTI-101 Monotherapy

Sum of Tumor Responses After ICI Failure, On TTI-101 Therapy and After ICI+Bev Rechallenge⁴

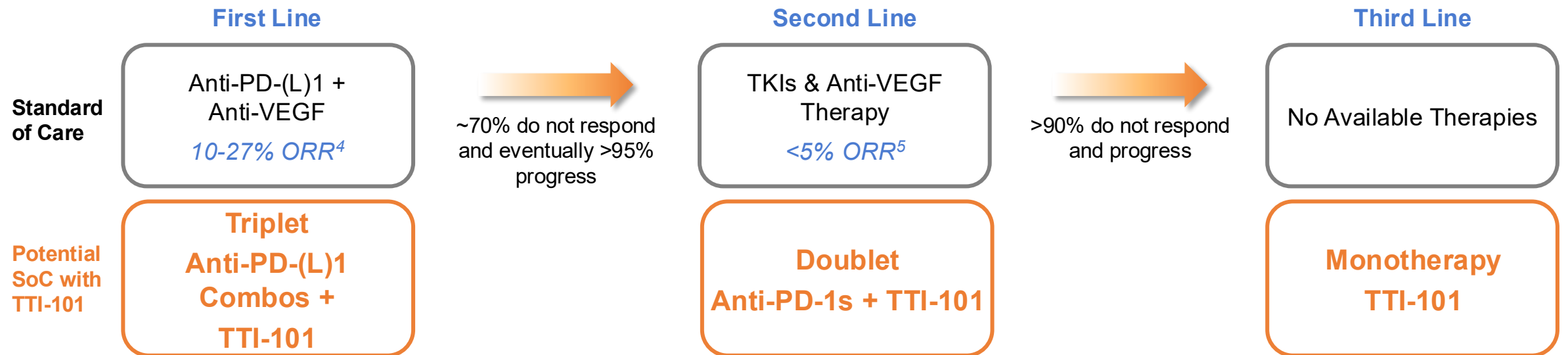


TTI-101 is Designed to Provide a Distinct and Synergistic Mechanism for Unmet Need in HCC

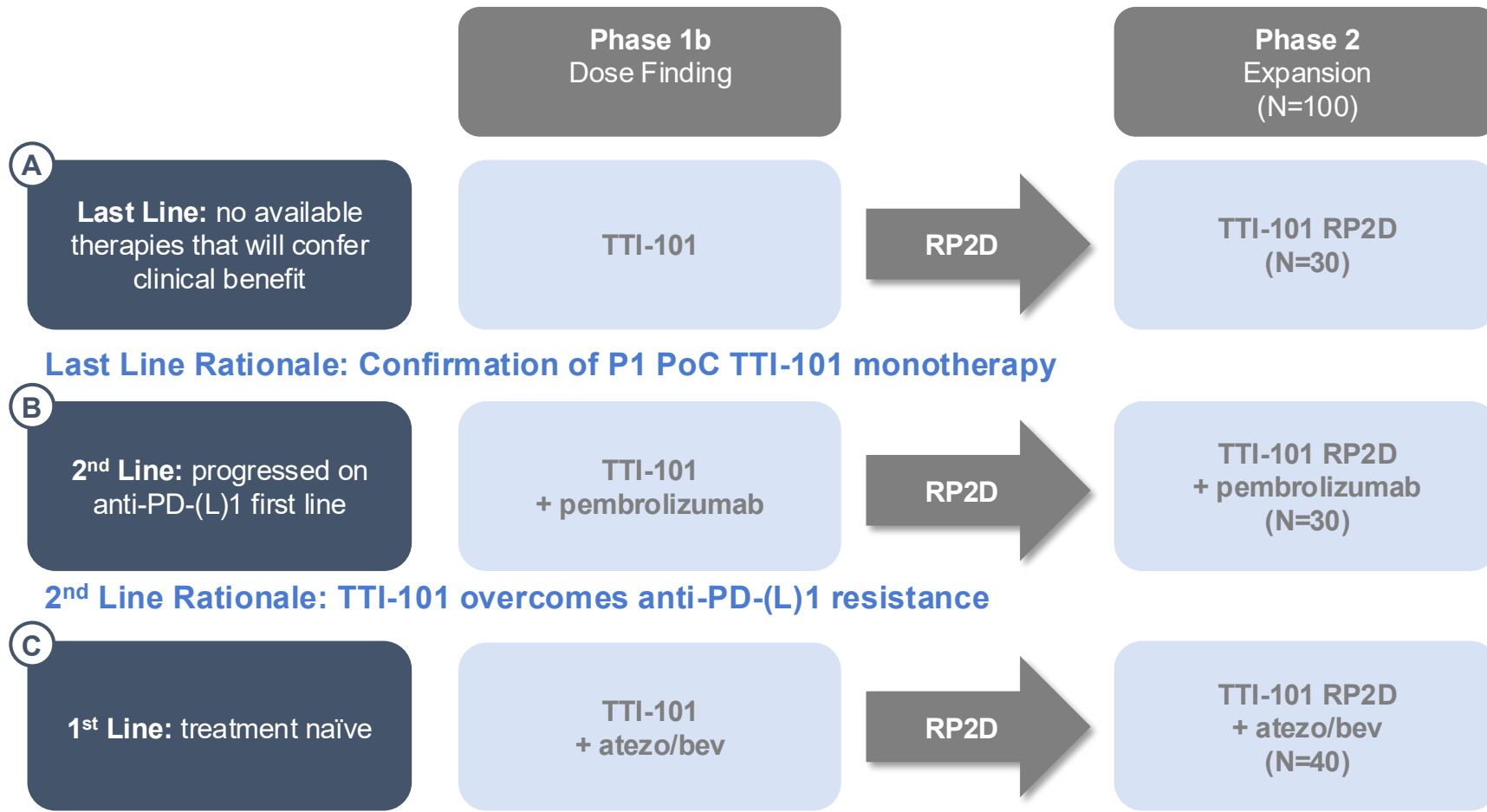
HCC Disease Overview

- HCC is 3rd leading cause of cancer deaths in the world¹
- Annually in the US, >42,000 new cases of HCC and ~32,000 deaths recorded²
- Incidence has more than tripled since 1980³

Overview of Current Treatment Landscape + Role of TTI-101



REVERT_{Liver Cancer}: Phase 2 Study of TTI-101 in HCC



Last Line Rationale: Confirmation of P1 PoC TTI-101 monotherapy

2nd Line Rationale: TTI-101 overcomes anti-PD-(L)1 resistance

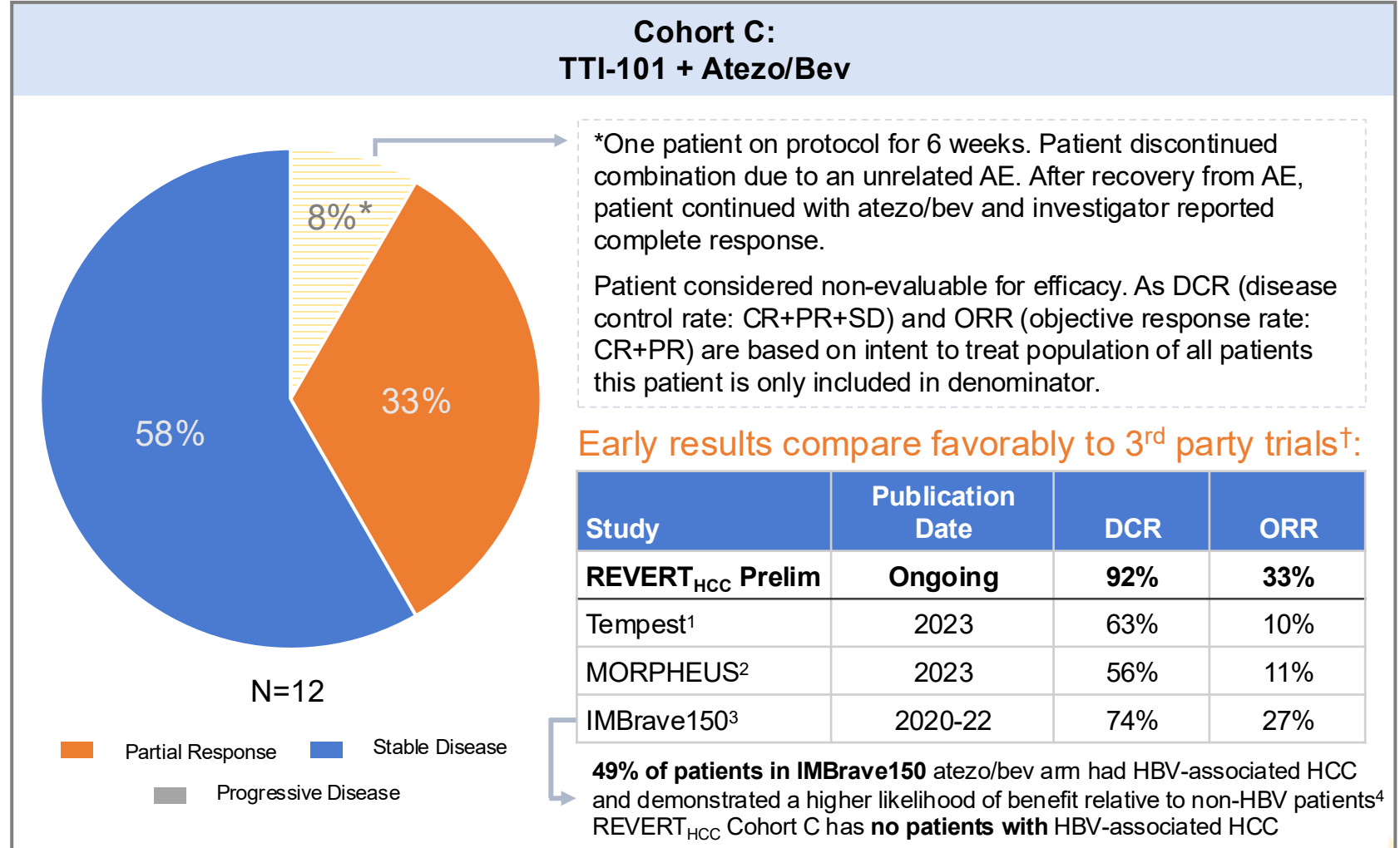
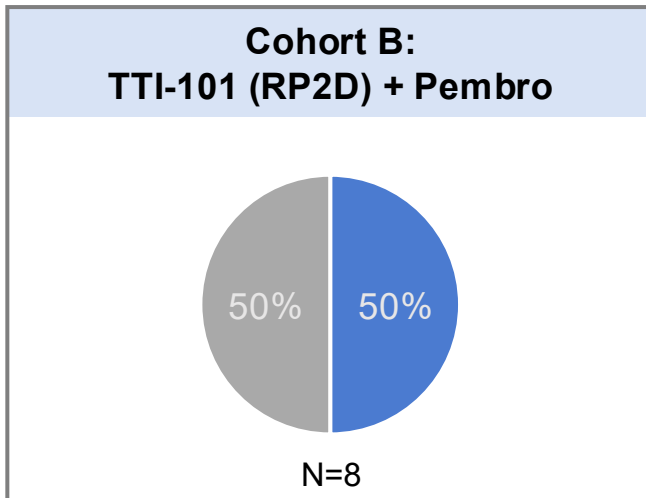
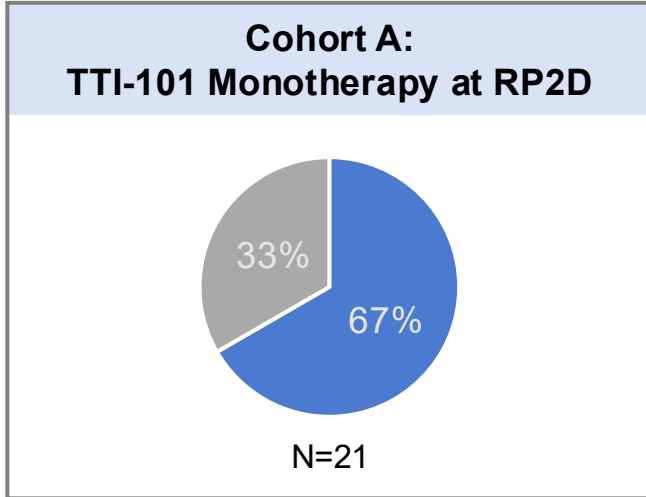
1st Line Rationale: TTI-101 is synergistic with anti-PD-L1 and anti-angiogenic inhibition

Early clinical data suggests clinical benefit across treatment lines



- Overall Response Rate (ORR)
- Duration of Response (DoR)
- Progression-free survival
- Liver stiffness (elastogram)
- Biomarkers (IL-6/AFP)
- pY-STAT3 in tumor

REVERT_{Liver Cancer}: Interim Phase 1b/2 Data



Key Takeaways: TTI-101 in HCC

STAT3: Well-Established Biology

STAT3 long recognized as prime target in oncology; >95% of patients with HCC have activated STAT3 in their tumors

Differentiated Approach

Inhibition of STAT3 activation to have dual therapeutic effect on cancer cells – overcoming tumorigenesis and immune suppression

Encouraging Clinical Activity

Clinically meaningful activity in both monotherapy and combination therapy in areas of unmet need

Near-Term Clinical Milestone

Topline results from ongoing Phase 2 REVERT_{LIVER CANCER} trial expected in 2H:2026

Targeting STAT3: Central Mediator of Inflammatory and Proliferative Diseases



Deep expertise in STAT3 biology

- Unlocking highly-validated target within inflammatory and proliferative diseases
- Demonstrated target engagement and disease modification across animal models



Potential to serve as a disease-modifying therapy

- Demonstrated enhanced biological activity in fibrotic cancers in P1
- Observed reductions in inflammation and fibrosis marker (IL-6) in IPF¹ P2
- Evaluating mono- and combination therapy in fibrosis-driven HCC² P2



TTI-109: designed to enhance delivery of STAT3 inhibitor & improve tolerability

- Prodrug designed to rapidly convert to active TTI-101 in the blood - IND³ filed June 2025
- P1 trial in healthy volunteers ongoing to demonstrate PK⁴, bioequivalence and tolerability



Multiple near-term data catalysts expected

- TTI-109: P1 healthy volunteer topline data in 2Q:2026
- TTI-101: P1b/2 HCC² topline data in 2H:2026

Financial Overview and Upcoming Milestones

Select Corporate Information	
Ticker	TVRD
HQ	Houston, TX
Shares Outstanding¹	9,381,344
Cash / Cash Equivalents / ST Investments¹	\$25.0 M

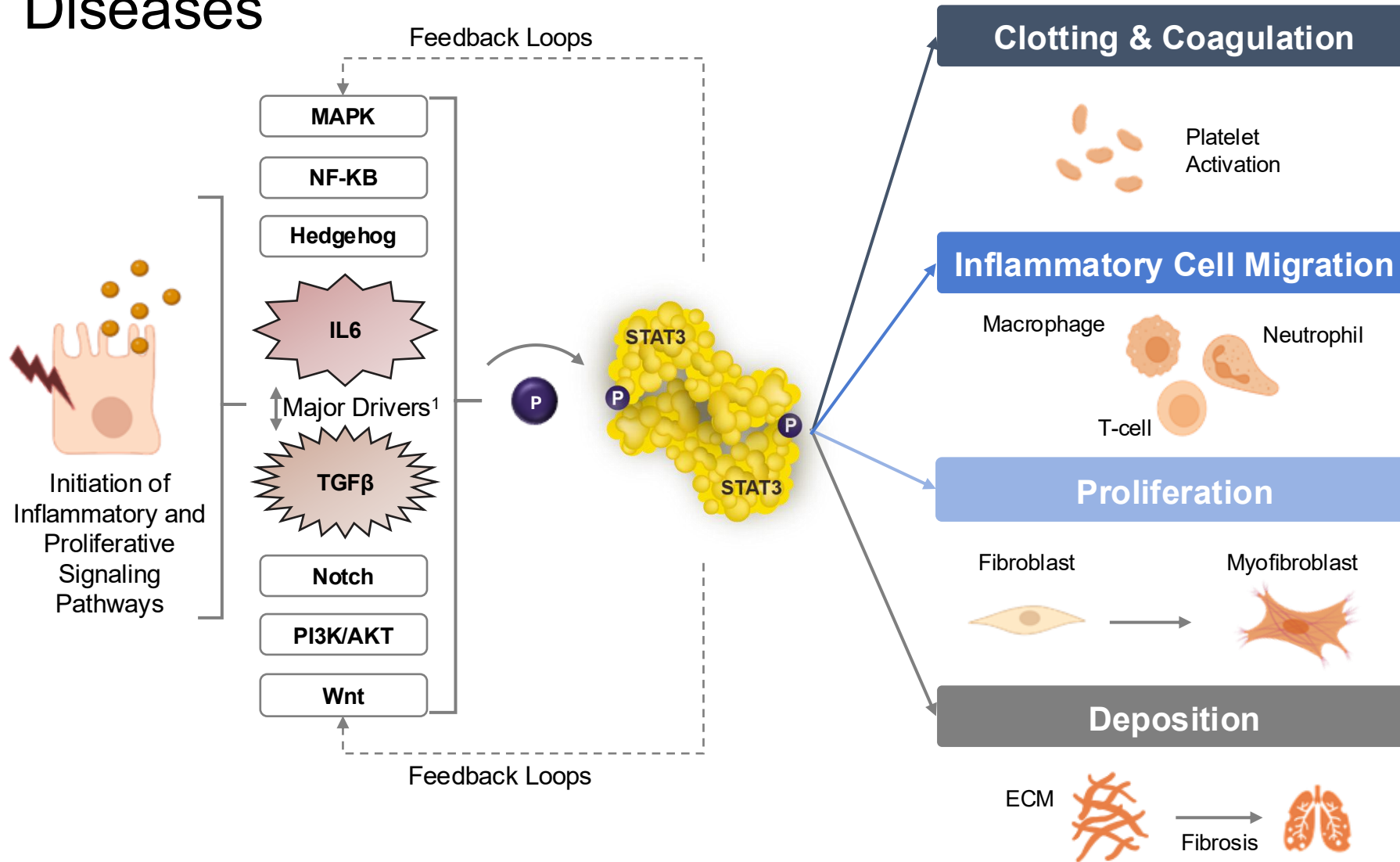
Upcoming Milestones	
2Q 2026	Topline Data from TTI-109 Phase 1 Healthy Volunteer Trial
2H 2026	Topline Data from TTI-101 Phase 1b/2 HCC Trial

**Anticipated Cash Runway Expected to Fund
Tvardi's Operating Plan into Q4 2026**

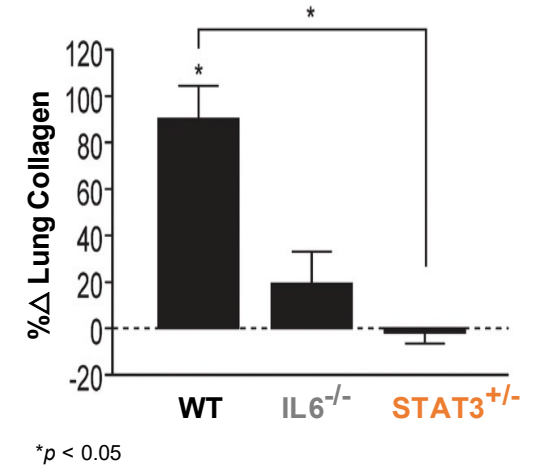
Appendix



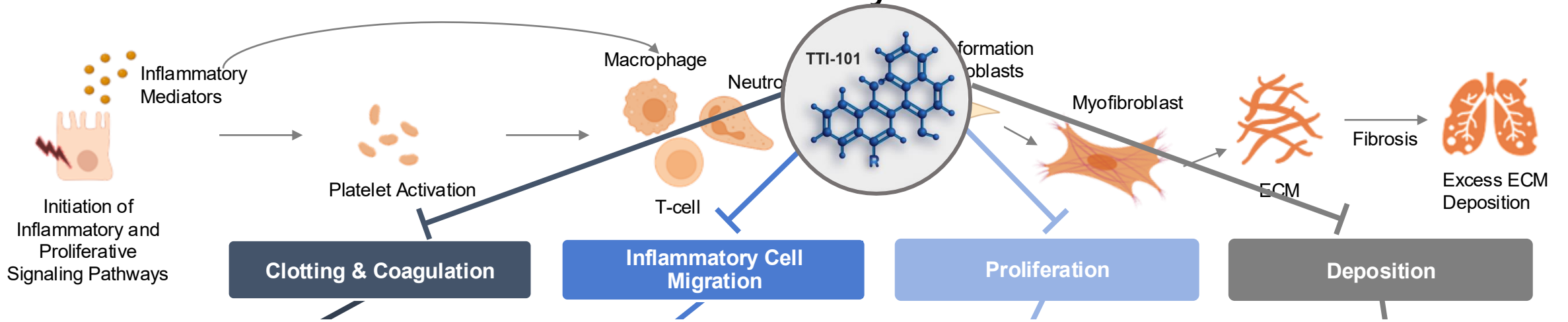
STAT3 Activation is a Central Catalyst in Inflammatory and Proliferative Diseases



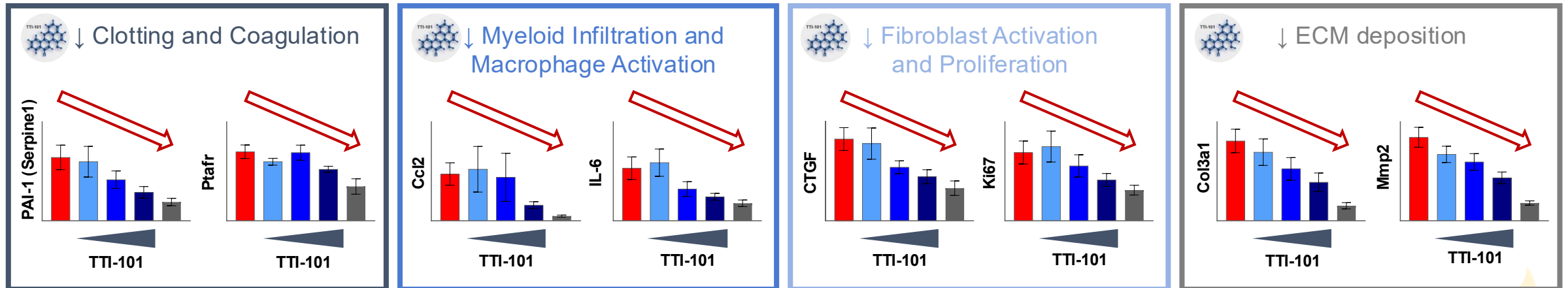
Haploinsufficiency of STAT3 (STAT3^{+/-}) protects mice from development of lung fibrosis vs. knockout of IL6² or TGFβR³ which still results in fibrosis



TTI-101 Simultaneously Modulates **Intrinsic (Cellular)** and Extrinsic Cascades of STAT3-driven Inflammatory and Proliferative Diseases

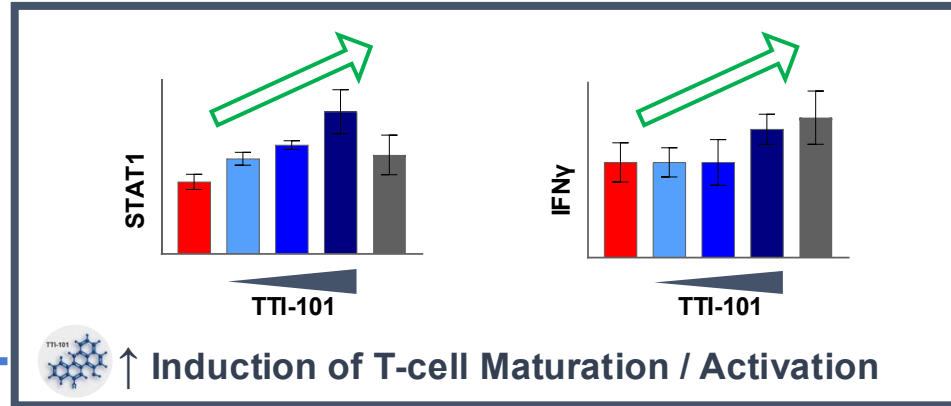


Dose-Dependent Decrease in Validated Targets Associated with Inflammation and Proliferation (Intrinsic)

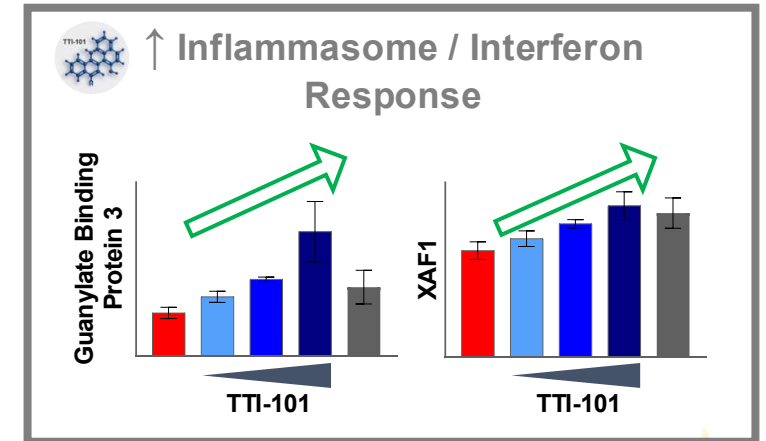
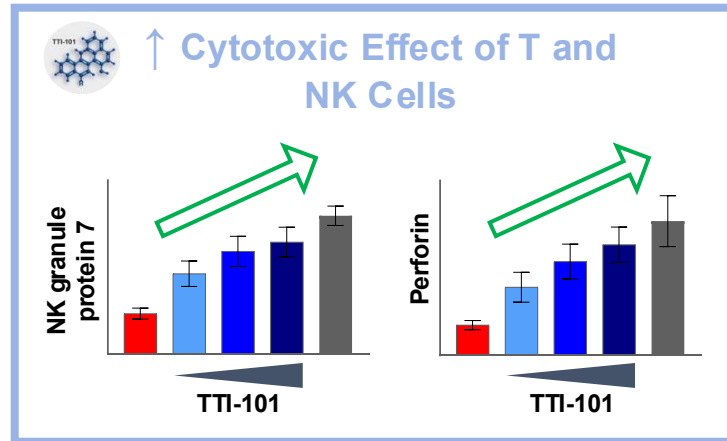
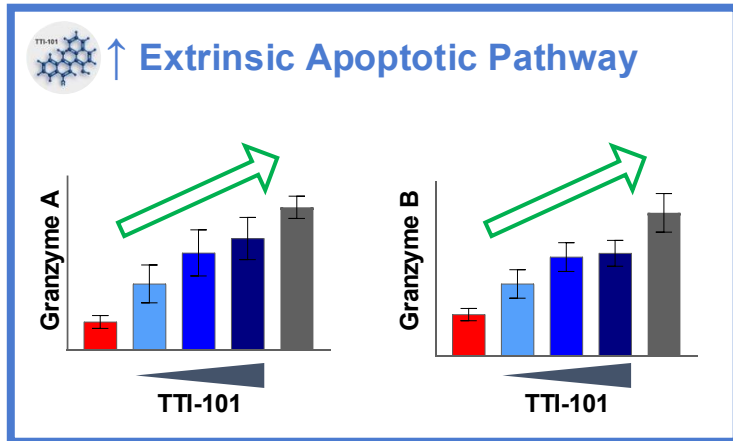


— No Bleomycin
 — Bleomycin + Placebo
 — Bleomycin + 50 mg/kg TTI-101
 — Bleomycin + 25 mg/kg TTI-101
 — Bleomycin + 12.5 mg/kg TTI-101

TTI-101 Simultaneously Modulates Intrinsic and **Extrinsic (Immune)** Cascades of STAT3-driven Inflammatory and Proliferative Diseases

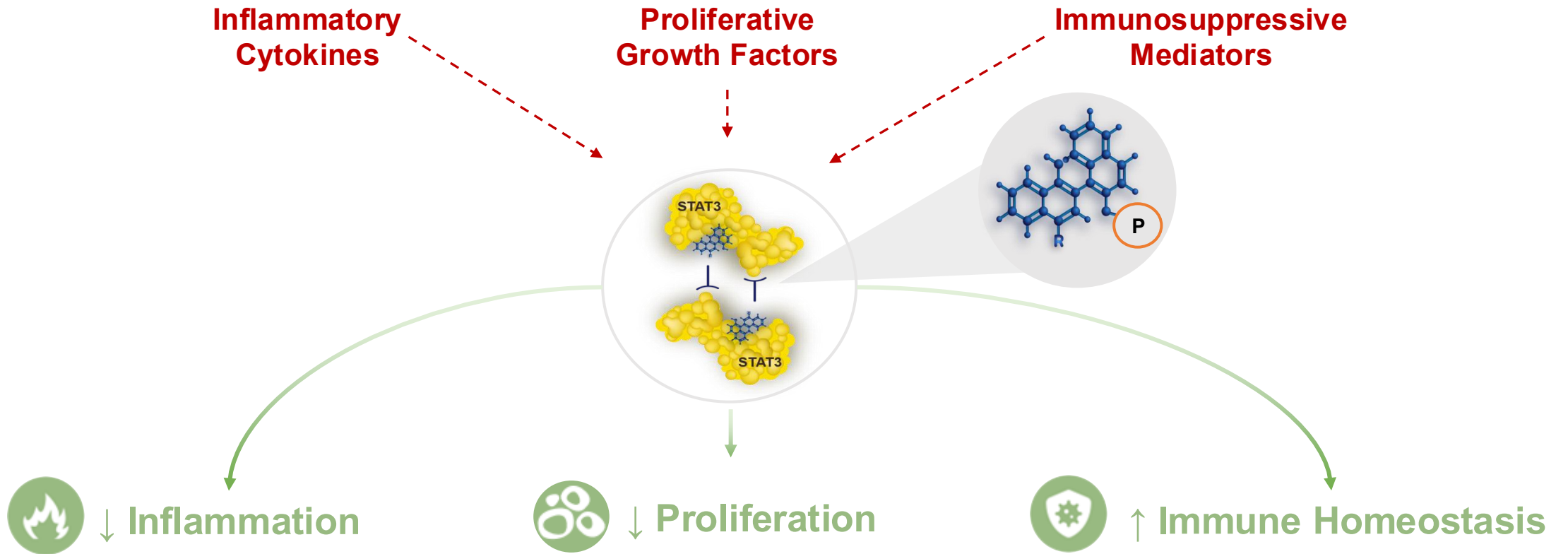


Dose-Dependent Increase in the Modulation and Activity of T-cells (Extrinsic):



— No Bleomycin — Bleomycin + Placebo — Bleomycin + 50 mg/kg TTI-101 — Bleomycin + 25 mg/kg TTI-101 — Bleomycin + 12.5 mg/kg TTI-101

TTI-109: Potential Reach Across STAT3-Driven Diseases



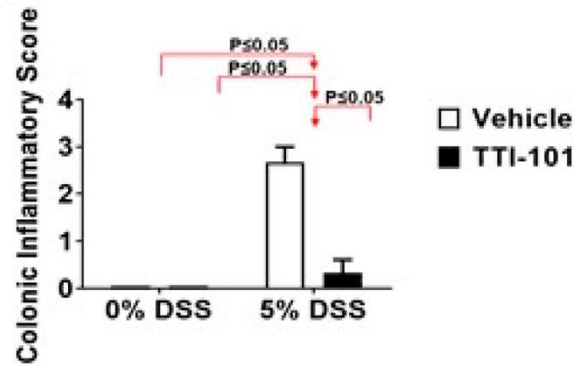
— Broad Relevance Across Inflammatory and Proliferative STAT3-Driven Conditions —

Dermatologic | Gastrointestinal | Hemopoietic | Autoimmune

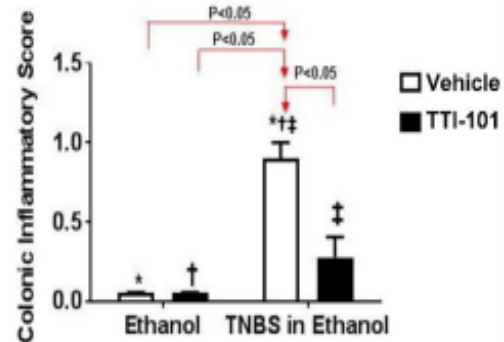
Across Disease Models, Tvardi's Inhibitors Modulate STAT3 Hallmarks

Inflammation

IBD-UC (5% DSS)¹



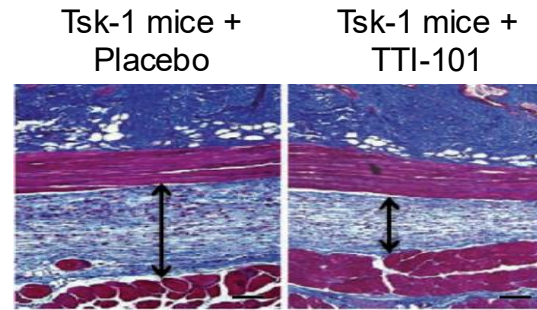
IBD-UC (TNBS)²



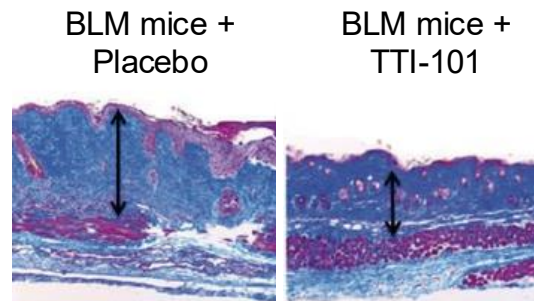
TVRD inhibitors reduce inflammatory indices

Proliferation

SSC (GEM)³



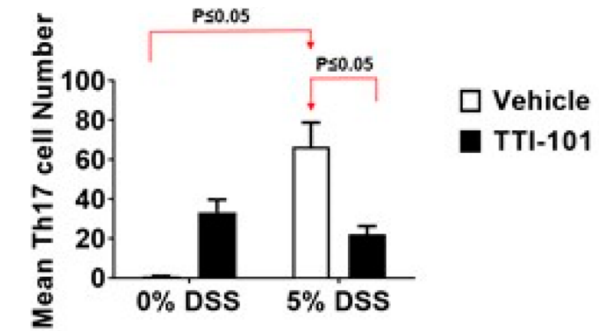
SSC (BLM)³



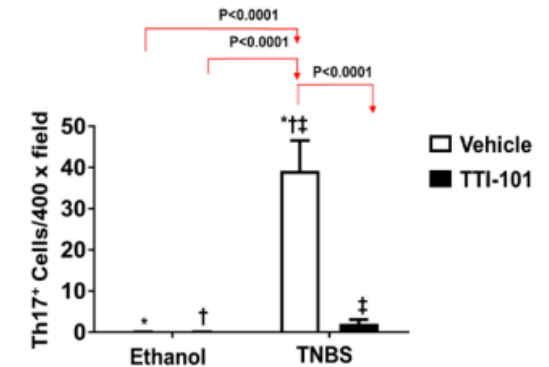
TVRD inhibitors reduce fibro-proliferative indices

Immune Modulation

IBD-UC (5% DSS)¹



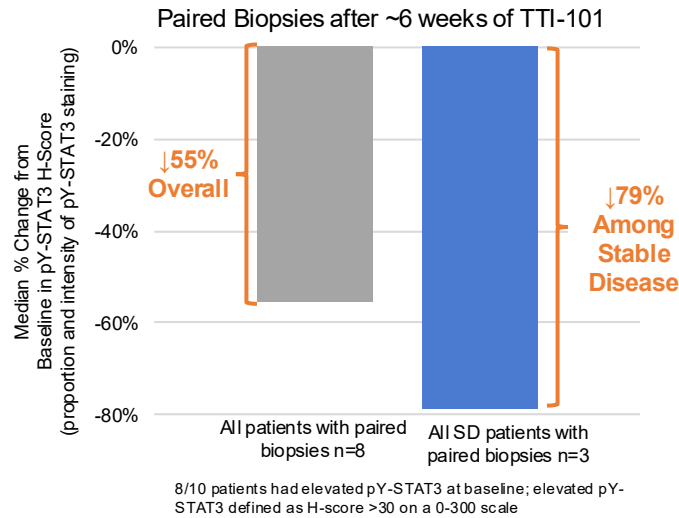
IBD-UC (TNBS)²



TVRD inhibitors reduce key immune modulators including Th17

Preclinical-to-Clinical Translation: Proof of Mechanism

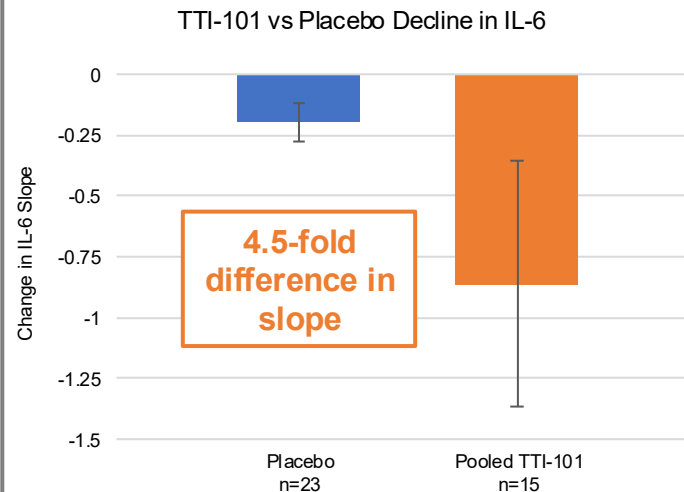
pY-STAT3 Target Engagement Oncology Phase 1



- 100% of patients with elevated baseline pY-STAT3 demonstrated a decrease within ~6 weeks; greater decrease in pY-STAT3 among patients with SD¹

Consistent with STAT3 pathway suppression observed in preclinical models

IL-6 Reduction REVERT IPF Phase 2

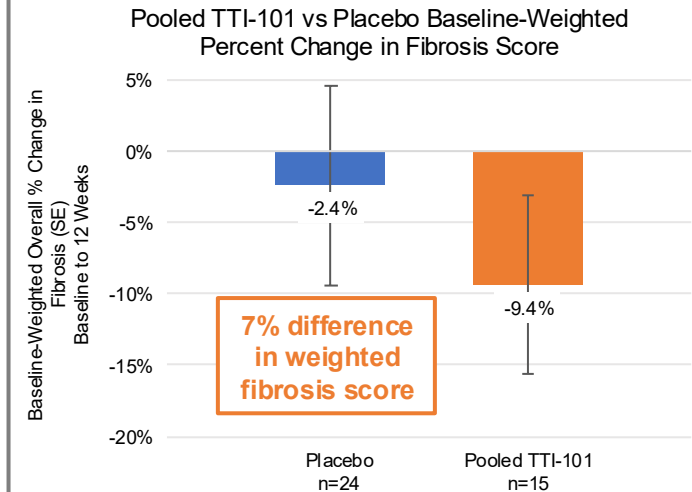


IL-6: a key pro-inflammatory cytokine that signals via STAT3

- Greater decrease in IL-6 slope among pooled patients treated with TTI-101 vs placebo²

Replicating IL-6 reductions observed in preclinical models

Fibrosis Reduction REVERT IPF Phase 2



- Greater decrease in fibrosis score (improvement in fibrosis) among pooled patients treated with TTI-101 vs placebo³

Consistent with histologic fibrosis reversal observed in preclinical models