THiogenesiS THerapeuticS

Strong Orphan Platform

Proven Biology + Rapid Clinical Path + Multiple Catalysts

(TSXV: TTI / OTCQX: TTIPF)

December - 2025

Forward Looking Statement

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Results in early-stage clinical trials may not be indicative of full results or results from later stage or larger scale clinical trials and do not ensure regulatory approval. You should not put undue reliance on these statement or the scientific data presented as a number of important factors, many of which are beyond our control, could cause our actual results to differ materially from the beliefs, plans, objectives, expectations, anticipations, estimates and intentions expressed in such forward-looking statements. We do not undertake to update any forward-looking statements, whether written or oral, that may be made from time to time by us or on our behalf; such statements speak only as of the date made. The forward-looking statements included herein are expressly qualified in their entirety by this cautionary language.

Experienced Leadership

Leadership with deep experience in thiol pharmacology, rare pediatric diseases and orphan approvals

Patrice Rioux, MD, PhD
Founder, CEO, Director

- Leading authority in mitochondrial metabolism
- Former CMO / Head of Regulatory at Raptor; *led approval of Procysbi®*
- Clinical development leadership across rare and metabolic disorders

Christopher M. Starr, PhD
Chairman of the Board

- Co-founder, BioMarin; co-founder & CEO of Raptor *(acquired for $800M),* Co-founder and Executive Chairman, Monopar
- 30+ years of experience building transformative orphan drug companies

BIOMARIN Biogen RepliGen raptor pharmaceutical corp. Naglazyme ALDURAZYME (LARONIDASE) KUVAN (sapropterin dihydrochloride) Tablets or Powder for Oral Solution PROCYSBI (cysteamine bitartrate) delayed-release capsules delayed-release oral granules

Thiogenesis - Summary

Clinical-stage biotech developing novel thiol-based prodrugs for high-need pediatric and orphan diseases

- **Lead Compound, TTI-0102:** improved cysteamine prodrug that targets mitochondrial dysfunction by increasing intracellular cysteine, GSH, and taurine
- **Leveraging decades of human data with** cysteamine across multiple diseases → de-risked mechanisms of action and well understood biology
- *Active clinical programs in MELAS, Leigh syndrome and pediatric MASH, plus planning for Phase 3 in cystinosis*
- **505(b)(2) regulatory pathway** reduces time and cost across all indications
- **Compelling Valuation:** < C$40m with multiple value-creating catalysts

Thiogenesis - Investment Highlights

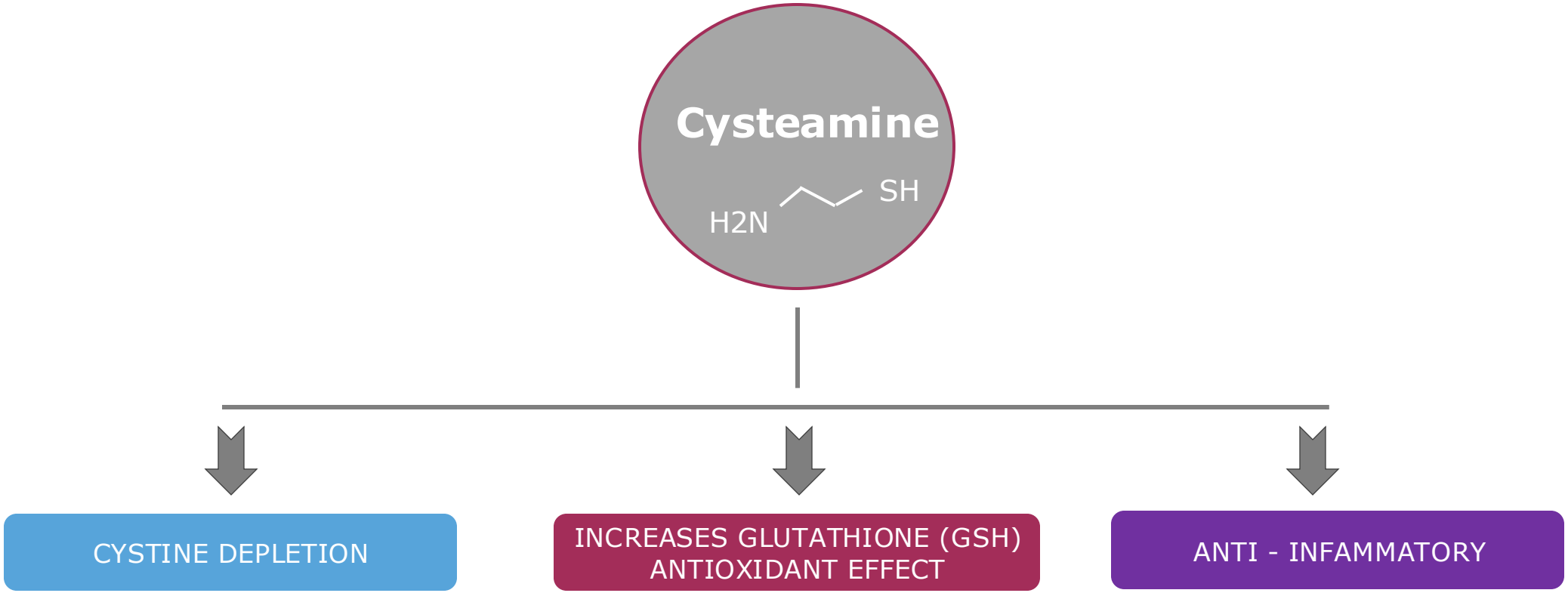
- **Clinically validated biology:** cysteamine-based drugs used in thousands of patients; strong GSH/antioxidant rationale
- **505(b)(2)** enables accelerated development with lower clinical and regulatory risk
- **Next generation cysteamine prodrug:** with improved GI tolerability + controlled sustained release → improves adherence and potential for once-daily dosing
- **Three de-risked indications:** with clear regulatory pathways (MELAS, Leigh syndrome, & Cystinosis) plus pediatric MASH expansion
- **Efficient capital plan:** multiple milestones achievable with modest spend
- **Leadership with multiple prior orphan-drug approvals** and successful commercial launches *(including Procysbi® - delayed release cysteamine)*

TTI-0102 Regulatory Pathway

505(b)(2) regulatory strategy accelerates development

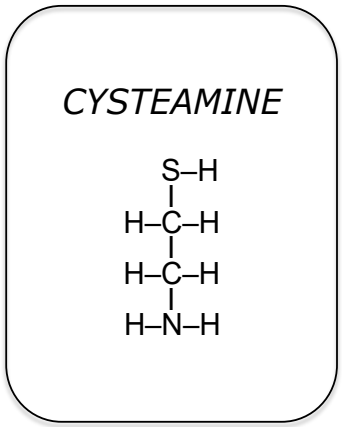
- TTI-0102 qualifies as a **New Chemical Entity** with **5 years of market exclusivity** (US/EU)
- ***As an oral prodrug,*** it leverages existing cysteamine toxicology and safety data
- ***505(b)(2) regulatory pathway*** reduces time and cost by avoiding redundant studies
- Enables streamlined path across multiple rare and metabolic indications

Cysteamine - Core Mechanisms of Action



These mechanisms are clinically validated in cystinosis and relevant to other mitochondrial disease models and metabolic disorders

TTI-0102 Lead Compound

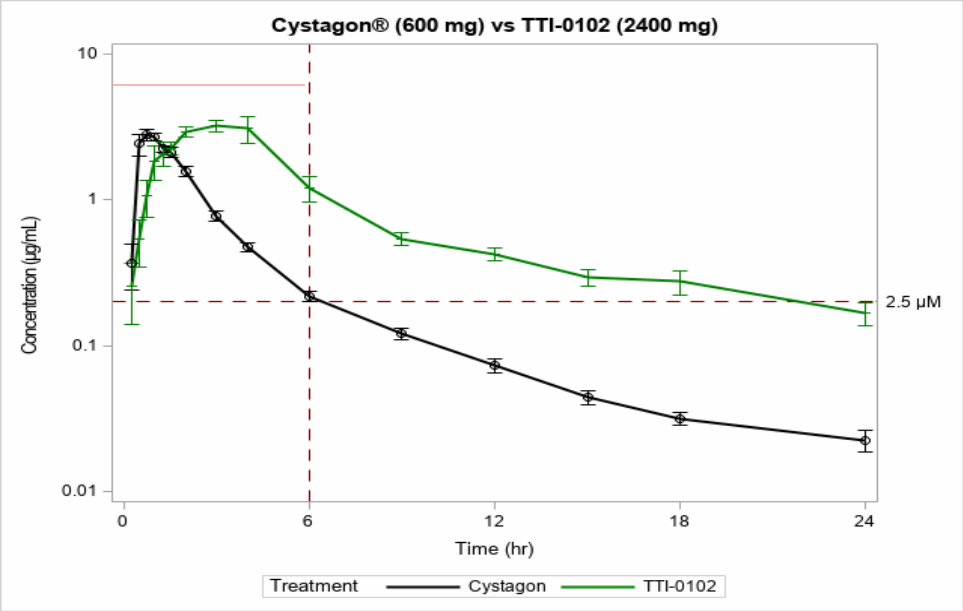


- **Asymmetric disulfide prodrug** that produces *2 cysteamine molecules + pantothenic acid (B5)*
- **Controlled metabolic activation** → reduces peak-related GI related side effects common with existing cysteamine-based compounds
- ***Extended PK*** *provides potential for once-daily dosing, improving adherence and real-world use in chronic pediatric diseases*
- **Boosts intracellular cysteine,** enabling increased GSH (master antioxidant) and taurine to support mitochondrial function & neuronal stability
- **Applicable across multiple indications** where oxidative stress, cysteine deficiency, and mitochondrial dysfunction are key drivers

TTI-0102 (Phase 1)

A controlled-release next-generation cysteamine prodrug

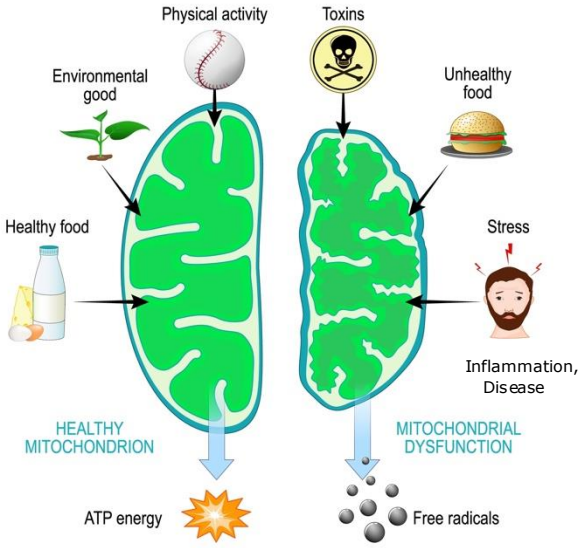
Phase 1 Study (PK/Safety)



Well-tolerated at 4x *therapeutic dosing level of generic cysteamine in Phase 1 (healthy adult volunteers)*

Mito Dysfunction/Oxidative Stress & GSH

Oxidative stress & GSH deficiency drive mitochondrial disease

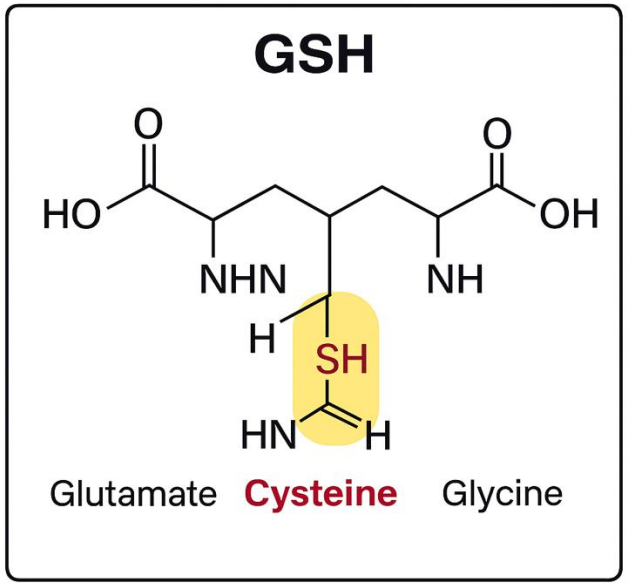


- *Mitochondria generate the energy required to power cells*
- Damaged mitochondria generate excess ROS (free radicals):

  ROS → oxidative stress → inflammation
- **GSH deficiency** is a key feature across *MELAS, Leigh syndrome, and related mitochondrial disorders*
- Low cysteine → low GSH → impaired redox balance and mitochondrial dysfunction
- Pathology drives symptoms: seizures, fatigue, motor decline, neurodegeneration
- Restoring cysteine/GSH is a **validated therapeutic strategy**

TTI-0102 Restores the GSH Pathway

TTI-0102 increases intracellular cysteine - the rate-limiting factor in GSH



- TTI-0102 increases intracellular **cysteine,** the rate-limiting precursor for GSH
- GSH is the **master antioxidant; detoxifies ROS,** supports immune function, and regulates inflammation
- Higher GSH → **reduces oxidative stress,** improves redox balance
- Supports **mitochondrial function,** neuronal stability, and cellular energy production
- Cysteamine also a precursor to **taurine,** important for neuronal protection and an antioxidant
- Mechanisms supported by **Phase 2 MELAS interim biomarkers**

MELAS

(Mitochondrial Encephalomyopathy, Lactic Acidosis & Stroke Like Episodes)

MELAS: Strong biological rationale + Phase 2 underway

- Severe pediatric mitochondrial disease with rapid neurodegeneration; ~4.1/100,000 prevalence
- Driven by oxidative stress, low GSH, impaired energy metabolism
- TTI-0102 increases **GSH + taurine,** reduces oxidative stress, and supports neuronal stability
- Phase 2 EU trial (NL + FR): randomized, blinded, placebo-controlled
- *Interim analysis: biological proof-of-concept, mitochondrial biomarker improvements, dosing insights*
- **Final 6-month data expected January 2026**

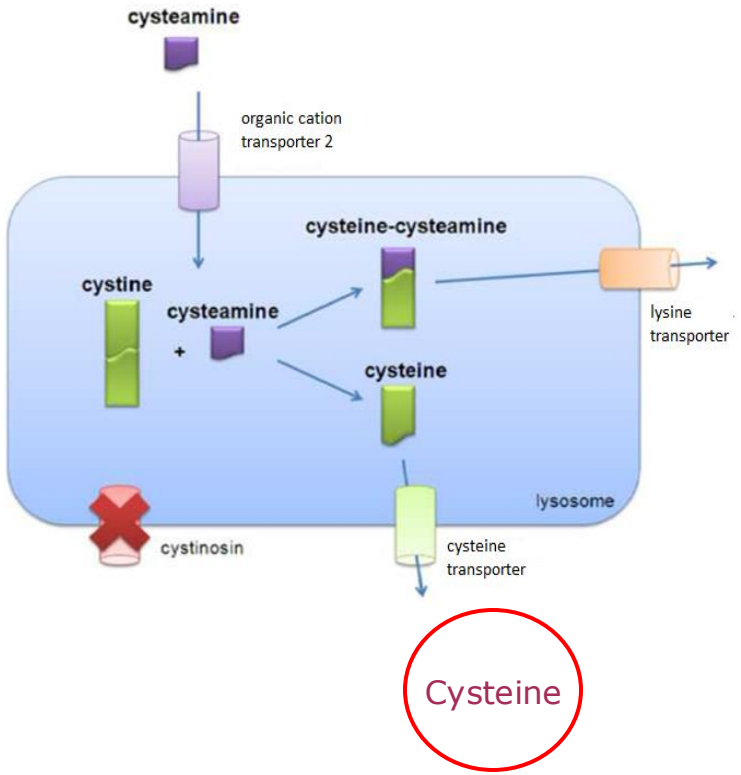
Leigh Syndrome Spectrum (LSS)

One of the most severe pediatric mitochondrial diseases

- Devastating early-onset mitochondrial disorder; ~1:40,000 live births
- Characterized by energy failure → developmental regression, motor loss, seizures
- Oxidative stress + low GSH/taurine implicated in neuronal and metabolic dysfunction
- **FDA-cleared IND (June 2025)** for a two-stage Phase 2a trial
- ***Two-stage Phase 2a:***
  - Stage 1: randomized, placebo-controlled (9 pts)
  - Stage 2: pediatric open-label extension (6 pts)
- Dosing strategy incorporates ***MELAS interim biomarker data***

Nephropathic Cystinosis

Cystinosis: Established biology with clear Phase 3 path



Besouw M - Adapted from et al. Drug Discovery Today, 2013

Disease Biology

- Defective lysosomal transporter → toxic cystine crystal accumulation
- Progressive kidney, muscle, eye and multi-organ damage
- Cysteamine is clinically proven but limited by **GI intolerance and strict multi-dose schedules**

TTI-0102 Rationale

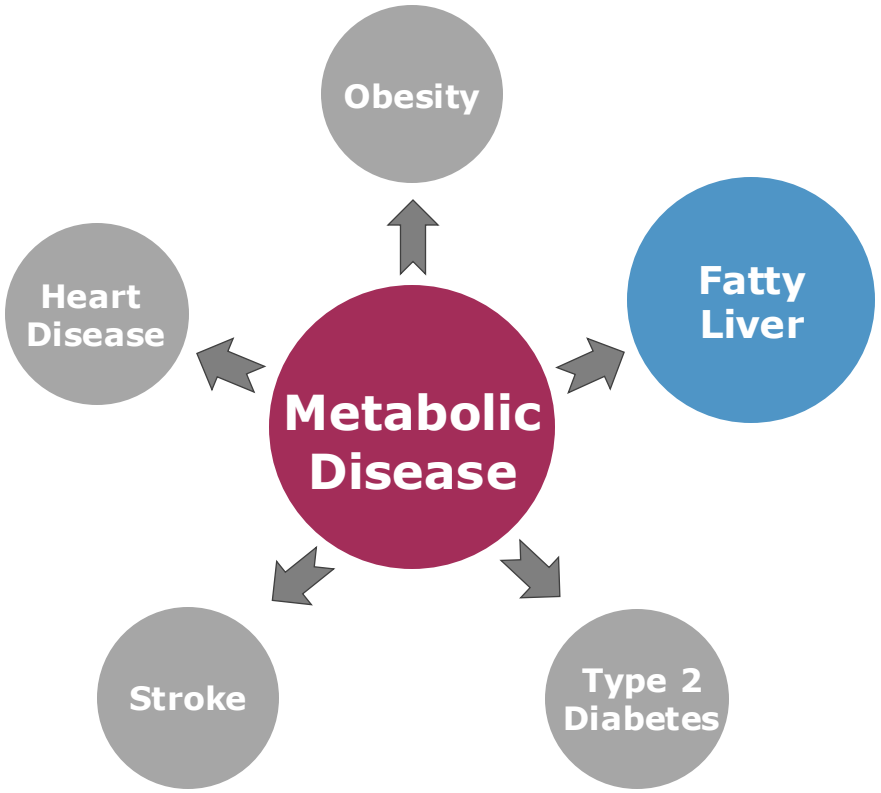
- Preserves cysteamine's dual mechanisms **(cystine depletion + antioxidant pathways)**
- Designed for **improved tolerability** and **once-daily potential**

Commercial Opportunity

- ~2,000 patients globally; >$300M market driven by >$200k/year therapy cost
- ***Near-term Phase 3: IND planned for early 2026***

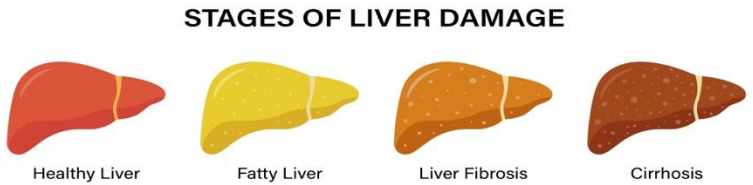
Oxidative Stress → Metabolic Disease

Metabolic Disease: Targeting Oxidative Stress Beyond GLP-1 Pathways



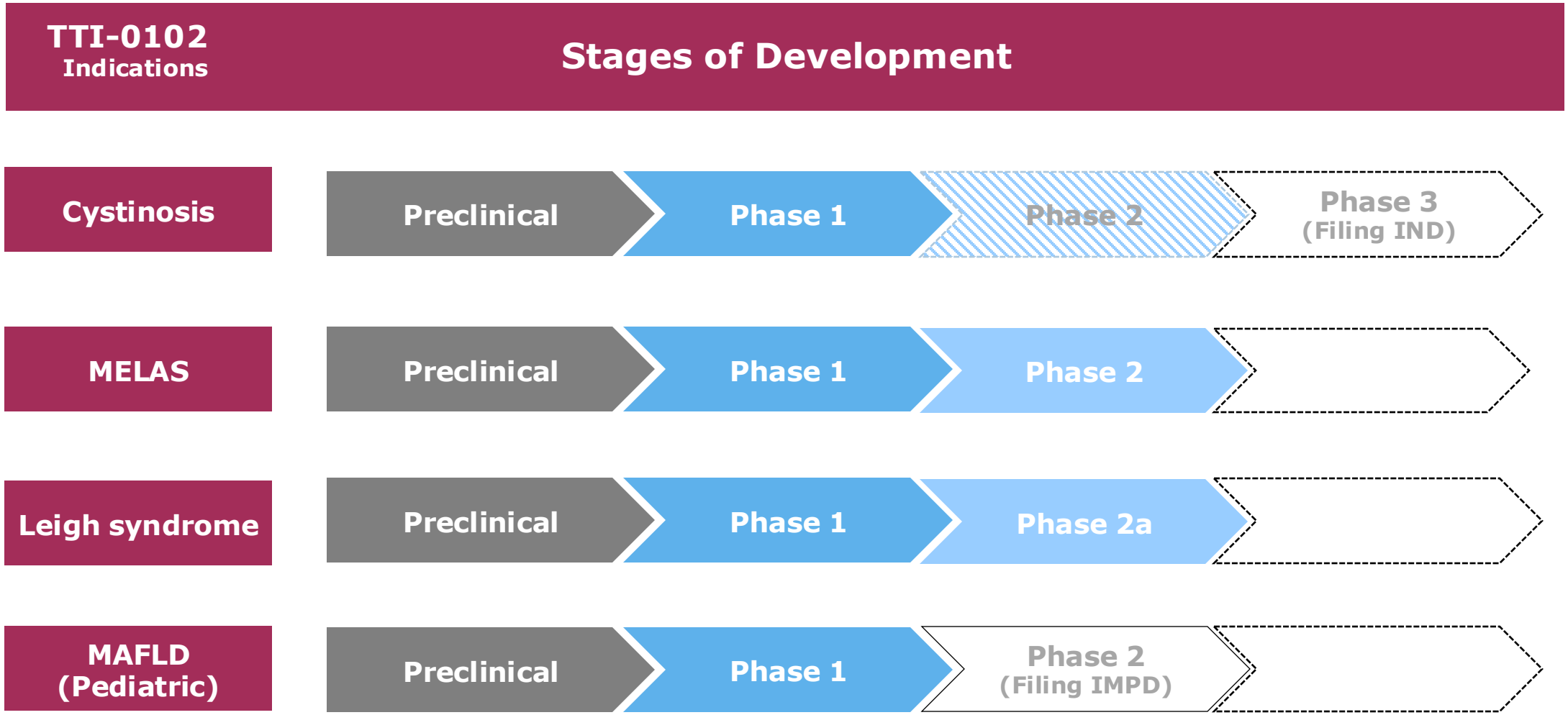
- Mitochondrial dysfunction & oxidative stress are also central in metabolic disorders
- TTI-0102 generates high intracellular cysteine – precursor to compounds that reduce oxidative stress *(GSH/Taurine)*
- Mechanisms are **distinct from GLP-1** and may be complimentary
- Potential **best-in-class compound for increasing intracellular cysteine/GSH** for pediatric and adult metabolic disease
- *Expands partnering + M&A optionality in large metabolic markets*

Pediatric-Metabolic Dysfunction-Associated Steatohepatitis (MASH)



- Pediatric MASH is driven by oxidative stress, mitochondrial dysfunction, and abnormal fat metabolism, it affects ~2–3% of children
- **Primary mechanism:** TTI-0102 reduces mitochondrial oxidative stress, improving lipid metabolism and reducing liver fat
- **Secondary mechanisms (fibrosis):**
  - **Cystine depletion** may down-regulate hepatic stellate cell activation
  - **Pantothenic acid (B5)** supports CoA pathways to metabolize fatty acids & aid tissue repair
- **NIH-CyNCh Trial** (DR-cysteamine) showed fat lowering signals in lighter children, effect on larger children limited by dosing constraints
- **TTI-0102 enables higher, better-tolerated dosing,** overcoming prior limitations
- **IMPD submission planned for 2025** to initiate a Phase 2 (EU)

Thiogenesis Pipeline



Thiogenesis – Upcoming Milestones

Potential milestones (6 months):

MELAS	→	Final Phase 2 data – Jan 2026
Leigh syndrome	→	Phase 2a patient enrollment Q1-26/data Q3-26
Cystinosis	→	Phase 3 IND clearance Q1-26
Pediatric MASH	→	Phase 2 IMPD cleared Q1-26

Scientific Advisory Board

World-class experts in mitochondrial, metabolic & lysosomal disease

Dr. David Housman

- MIT, award winning professor of biology, known for his contribution to the study of Huntington's disease and as a co-founder of 5 biotech companies

Dr. Gregory Enns

- Stanford University, professor of Medical Genetics and Director of Biochemical Genetics Program; focus on mitochondrial and lysosomal disorders

Dr. Miriam Vos

- Emory University, professor of Pediatrics and Division of Gastroenterology, Hepatology and Nutrition, and Director of Pediatric Fatty Liver Program at Children's Healthcare of Atlanta

Company Info

Thiogenesis Therapeutics	(TSXV: TTI / OTCQX: TTIPF)
Shares Issued	51.8 million
Shares Fully Diluted	56.9 million
Insiders (32%)	16.7 million
Share Price (11/11/2025)	$0.72
52 week high/low	$0.88/C$0.51
Market Cap.	$37.3 million
Cash (09/30/2025)	$3.3 million
Contact	info@thiogenesis.com

- Currency in Canadian dollars

Companies of Interest

Name	Symbol	Disease	Stage	Market Cap	Notes
Thiogenesis	***TTI***	***MELAS***	***Ph.2***	***C$34 mn***	***Anti-ox, Anti-inflam.***
Spruce	SPRB	MPS IIIb	Ph. 2	US$107 mn	Enz. Replacement
Sagimet	SGMT	Obesity/NASH	Ph 2	US$224 mn	FASN
Larimar	LRMR	F. Ataxia	Ph. 2	US$302 mn	Protein Replacement
Zevra	ZVRA	NP-C	NDA	US$475mn	Enz. Signaling
Monopar	MNPR	Wilson's	NDA	US$575 mn	Copper chelation
Reata	*RETA*	*F. Ataxia*	*Approved*	*US$7.0 bn*	*Anti-ox & Anti-inflam.*