Thiogenesis Presents Phase 2 (EU) MELAS Data at Mitocon 2026

Highlighting Optimized Once-Daily Weight-Based Dosing and Clinically Meaningful Fatigue Improvement

San Diego, California – January 23, 2026 – Thiogenesis Therapeutics, Corp. (TSXV: TTI) (OTCQX: TTIPF) ("Thiogenesis" or the "Company"), a clinical-stage biotechnology company developing next-generation thiol-based prodrugs for rare mitochondrial and pediatric diseases, today announced the presentation of interim data from its Phase 2 (EU) clinical study of TTI-0102 in patients with Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-like Episodes (MELAS) at Mitocon 2026 - a leading international conference dedicated to mitochondrial medicine being held in Pisa, Italy.

The poster, titled ***"TTI-0102 in MELAS: PK/PD Results from a Phase 2 Study – Preliminary Evidence of Safety and Efficacy in Mitochondrial Disease,"*** was presented on January 23 as part of Mitocon's Late-Breaking News session, a distinction reserved for data of high scientific and clinical relevance selected by the conference's Scientific Committee.

Phase 2 (EU) MELAS - Study Highlights

The randomized, single-blind, high fixed-dose, placebo-controlled exploratory Phase 2 (EU) study enrolled **nine (n=9) patients** with genetically confirmed MELAS, a devastating mitochondrial disorder with limited therapeutic options. Results presented at Mitocon demonstrated that **weight-based dosing is critical to achieving optimal exposure and tolerability**, consistent with long-standing clinical experience using cysteamine in concentrations comparable to approved formulations (Cystagon® or Procysbi®) in cystinosis.

Key clinical, pharmacokinetic, and pharmacodynamic findings included:

Clinical Outcome (Fatigue):

- **Statistically significant improvement in patient-reported fatigue** as measured by the total score of Modified Fatigue Impact Scale (MFIS)
- At an average dose of approximately **60 mg/kg/day**, TTI-0102-treated patients demonstrated a **mean reduction of up to 10% in total MFIS score** compared to placebo (**$p < 0.001$**)
- Fatigue is a cardinal and functionally limiting symptom affecting patients with mitochondrial disease

Pharmacokinetics (PK):

- **Sustained 24-hour cysteamine exposure** achieved with once-daily, weight-based dosing
- Identification of an **optimal dose of approximately 60 ± 5 mg/kg of TTI-0102** to achieve target exposure

- **Lower peak plasma cysteamine concentrations (Cmax)** at this dose compared with fixed dosing and approved cysteamine formulations, supporting improved gastrointestinal tolerability

Pharmacodynamics (PD):

- **Increased plasma pyruvate without lactate elevation**, suggesting enhanced glycolytic flux; *a metabolic state in which glucose is more efficiently converted into usable cellular energy rather than diverted into lactate production, consistent with improved mitochondrial energy handling*
- **Decreased plasma tryptophan**, potentially reducing oxidative stress associated with neurotoxic kynurenine pathway metabolites
- These pharmacodynamic effects were **dose-dependent**, negligible below 40 mg/kg/day, optimal at approximately 60 mg/kg/day, and slightly reduced at higher doses

Taken together, these pharmacodynamic findings are consistent with reduced oxidative stress and improved cellular energy metabolism, supporting the proposed mechanism of TTI-0102 in mitochondrial disease, based on thiol-disulfide exchange equilibrium, and providing biological rationale for the observed clinical improvement in fatigue.

The study further demonstrated that fixed dosing resulted in gastrointestinal adverse events and treatment discontinuations in lower-weight patients, whereas weight-adjusted dosing mitigated these effects, supporting its use in future clinical trials. Weight-based dosing at 60 ± 5 mg/kg TTI-0102 (~26 mg/kg cysteamine base equivalent) achieved sustained 24-hour cysteamine exposure with half the daily dose of Cystagon® or Procysbi® administered to patients with cystinosis, and lower peak concentrations.

“The results presented at Mitocon confirm that TTI-0102 achieves meaningful biological and clinical proof-of-concept in MELAS when administered using appropriate weight-based dosing,” said Patrice Rioux, M.D., Ph.D., Chief Executive Officer and Co-Founder of Thiogenesis. “Notably, the statistically significant improvement in fatigue measured by MFIS is highly relevant, as fatigue represents a core functional component captured within the comprehensive MM-COAST outcome framework that will be used in our upcoming Phase 2a study in Leigh syndrome spectrum. Together with a well-characterized pharmacokinetic and pharmacodynamic profile, these findings directly inform the design of our Leigh program.”

About MFIS

The Modified Fatigue Impact Scale (MFIS) is a validated, patient-reported outcome measure widely used in neurological and mitochondrial disorders to assess the impact of fatigue on physical, cognitive, and psychosocial functioning. Fatigue affects an estimated 71–100% of patients with mitochondrial disease and is frequently reported as one of the most debilitating symptoms. MFIS has been shown to be sensitive to clinically meaningful change and is considered an appropriate endpoint for evaluating therapeutic benefit in mitochondrial disorders.

About Mitocon

Mitocon – *Insieme per lo studio e la cura delle malattie mitocondriali OdV* – is Italy’s leading patient advocacy organization dedicated to mitochondrial diseases. Mitocon supports research, education, and collaboration among clinicians, scientists, and patient communities, and its annual international conference is widely recognized as a premier forum for the presentation of emerging scientific and clinical advances in mitochondrial medicine.

About MELAS

Mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (MELAS) is a rare, inherited mitochondrial disorder most caused by a mutation in mitochondrial DNA (m.3243A>G). Symptoms include seizures, muscle weakness, fatigue, headaches, and stroke-like episodes. Oxidative stress and impaired mitochondrial energy metabolism are key contributors to disease progression. MELAS affects an estimated 4.1 per 100,000 individuals worldwide and is considered an orphan disease.

About Leigh Syndrome Spectrum

Leigh syndrome spectrum is a rare, inherited mitochondrial disorder typically diagnosed in infancy or early childhood and affecting approximately 1 in 40,000 live births. The disease is characterized by progressive neurological decline, developmental regression, respiratory dysfunction, and seizures. There are currently no approved therapies for Leigh syndrome spectrum.

About TTI-0102

TTI-0102 is Thiogenesis' lead product candidate and a next-generation cysteamine-based prodrug designed to overcome limitations of first-generation thiol therapies, including short half-life, gastrointestinal side effects, and dosing constraints. Through controlled metabolic activation, TTI-0102 enables sustained cysteamine exposure with the potential for improved tolerability and simplified dosing. The compound is being evaluated across multiple indications associated with mitochondrial dysfunction and oxidative stress, including MELAS, Leigh syndrome spectrum, and nephropathic cystinosis.

About Thiogenesis Therapeutics

Thiogenesis Therapeutics, Corp. (TSXV: TTI) (OTCQX: TTIPF) is a clinical-stage biopharmaceutical company headquartered in San Diego, California. The Company is developing sulfur-containing prodrugs designed to act as precursors to previously approved thiol-active compounds, with the potential to address serious pediatric diseases with significant unmet medical need. Thiogenesis' lead program, TTI-0102, is being evaluated in MELAS, an IND-cleared Phase 2a study planned in Leigh syndrome spectrum, and a Phase 3 study planned in nephropathic cystinosis.

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Forward Looking Statements

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