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Corporate Mission and Highlights

Our mission is to develop a new class of small molecule drugs that reset muscle repair and regeneration, the *next horizon* in treating degenerative muscle conditions.

In doing so we **challenge** conventions, **break** scientific ground, and **urgently** advance new drug development because *time is muscle*.

Corporate Highlights

Initial focus on Duchenne muscular dystrophy ("DMD" or "Duchenne")

Lead drug SAT-3247 to enter clinical trials in mid-2024

Multibillion \$ market opportunities across multiple diseases.

Experienced Board and management (15% insider holding)

Two years of cash runway with strong holdings (>50%) by healthcare focused institutions



An enormous market opportunity in which the vast majority of patients go untreated!

70%

of Duchenne patients remain untreated²

7,000-9,500

Duchenne patients in the US¹

5

Approved drugs for DMD

>\$4B

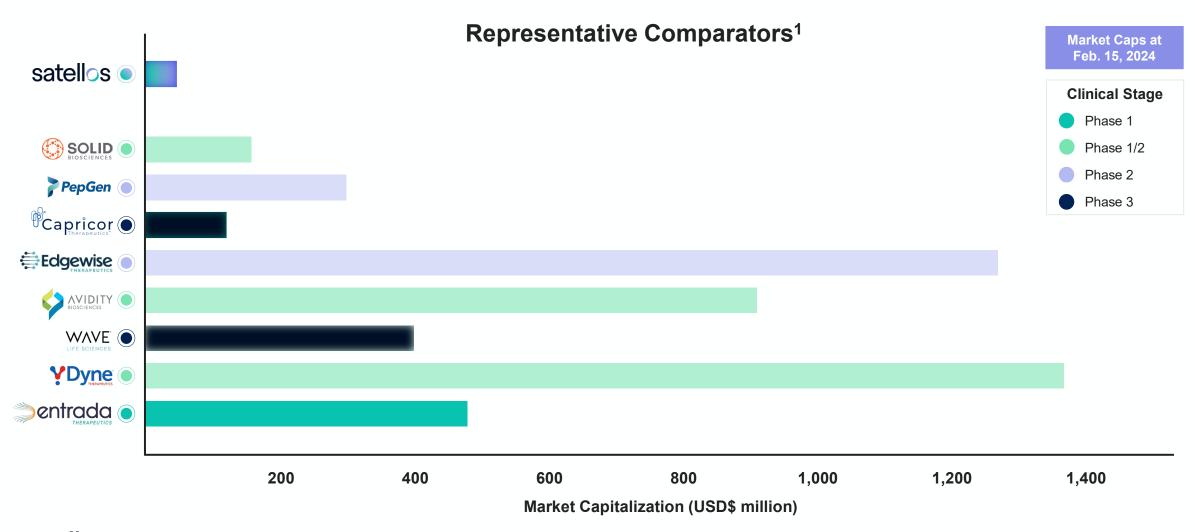
DMD market opportunity

Multiple

Indication expansion opportunities

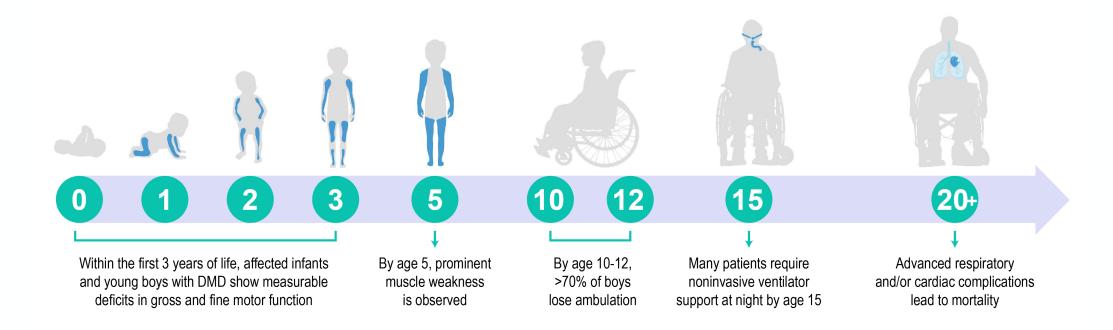


Satellos – attractive entrance point





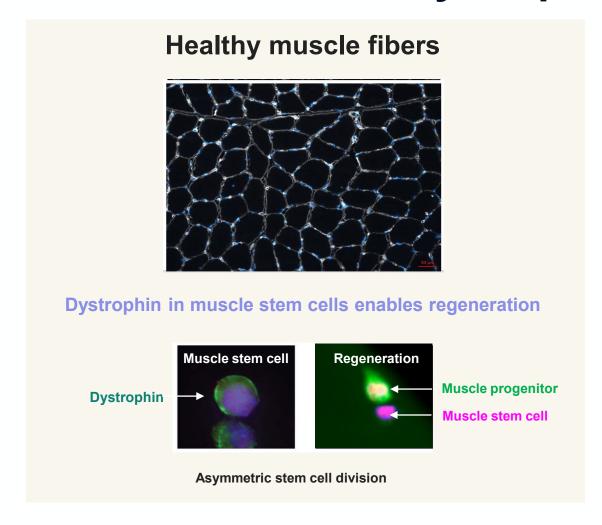
Duchenne muscular dystrophy: the patient journey

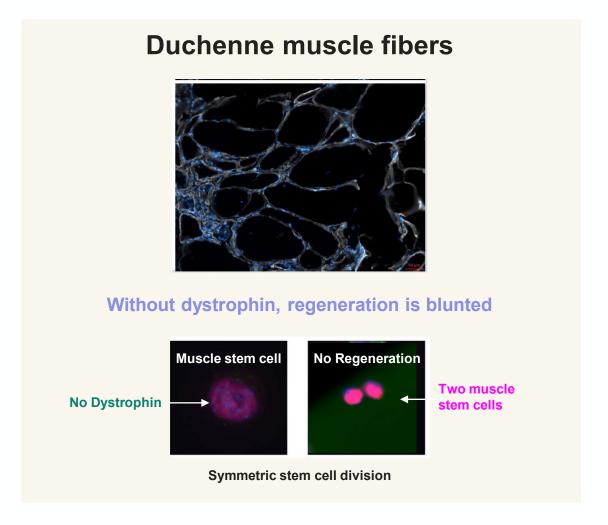


- 1:5,000 male babies born annually with Duchenne, a fatal genetic disease.
- Mutation(s) in the dystrophin gene leads to a loss or dysfunction of the dystrophin protein.
- Muscle regeneration is severely impaired in Duchenne leading to ongoing, progessive muscle loss.



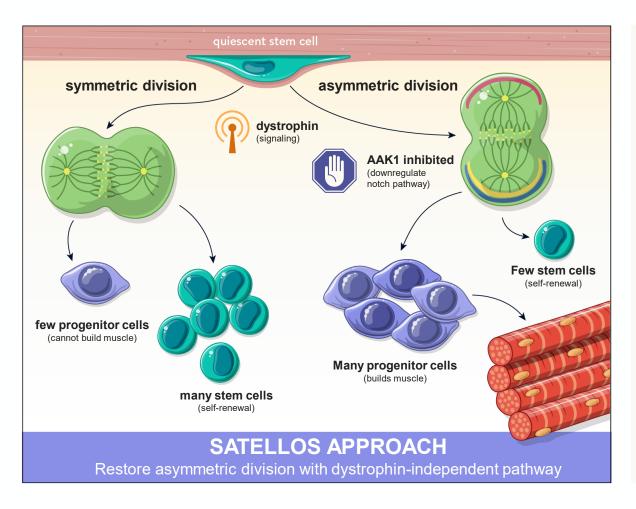
Satellos discovery: regeneration is compromised in DMD due to absence of dystrophin in muscle stem cells







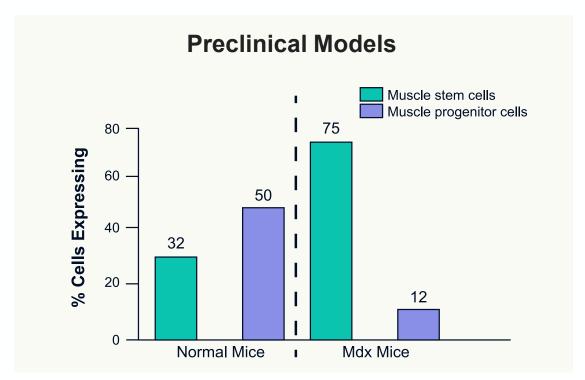
The role of dystrophin in muscle stem cells can be replaced to restore asymmetric divisions and regeneration

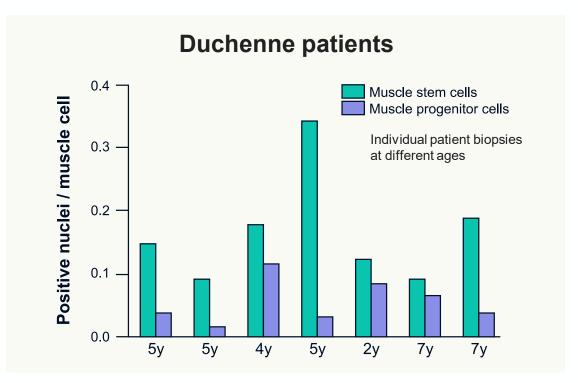


- Dystrophin promotes asymmetric divisions¹
- Asymmetric division produces progenitor cells
- Progenitors are required for repair and regeneration
- Satellos discovered an alternative: inhibiting AAK1,
 a regulator of Notch signaling
- Inhibition of AAK1 has the potential to restore asymmetric divisions and regeneration
- Satellos developed SAT-3247, a novel and purpose built AAK1 inhibitor



Muscle stem cell divisions are clearly compromised in preclinical models and Duchenne patients



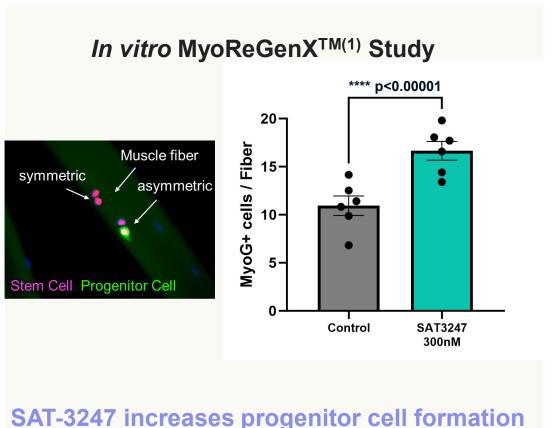


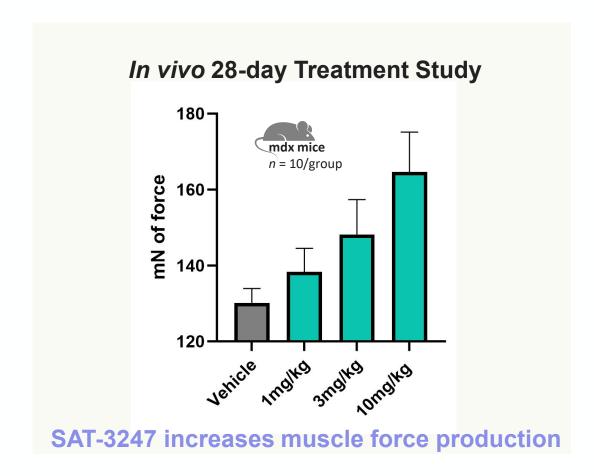
DMD patients and mice have abundant muscle stem cells. They lack sufficient muscle progenitor cells to form mature muscle fibers.



Inhibition of AAK1 corrects for loss of dystrophin signal in muscle stem cells, enabling regeneration process









Duchenne patients walk without any muscle dystrophin

Milder course in Duchenne patients with nonsense mutations and no muscle dystrophin

M. Zatz*, R.C.M. Pavanello, M. Lazar, G.L. Yamamoto, N.C.V. Lourenço, A. Cerqueira, L. Nogueira, M. Vainzof

This discovery of dystrophin-null, ambulatory Duchenne patients by Zatz et al provides genetic **proof of concept** that it is possible to have functional muscle in the absence of dystrophin.

Additional reports of ambulatory Duchenne patients lacking dystrophin¹ by Flanigan et al provide strong support for Satellos' therapeutic strategy to restore regeneration in Duchenne.

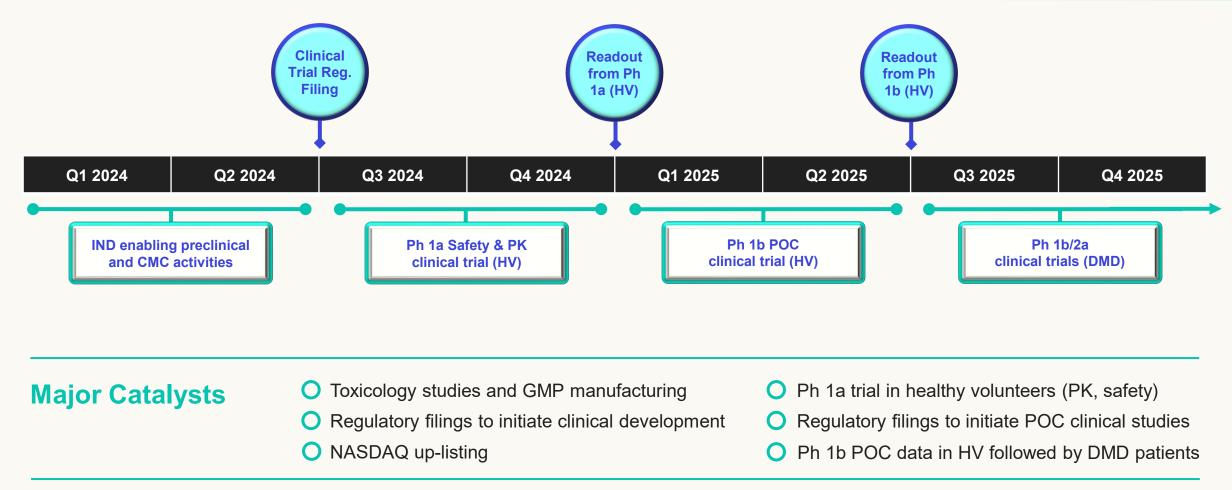


Dystrophin null siblings where one continued to ambulate.



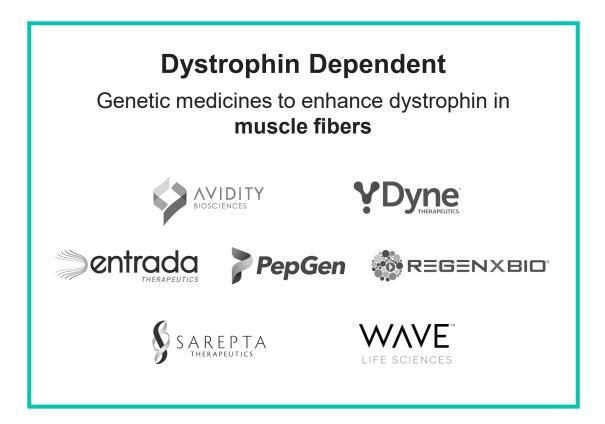
Robust, value creating plan through 2024/25







Satellos' approach is dystrophin independent and of potential benefit to all Duchenne patients



Dystrophin Independent

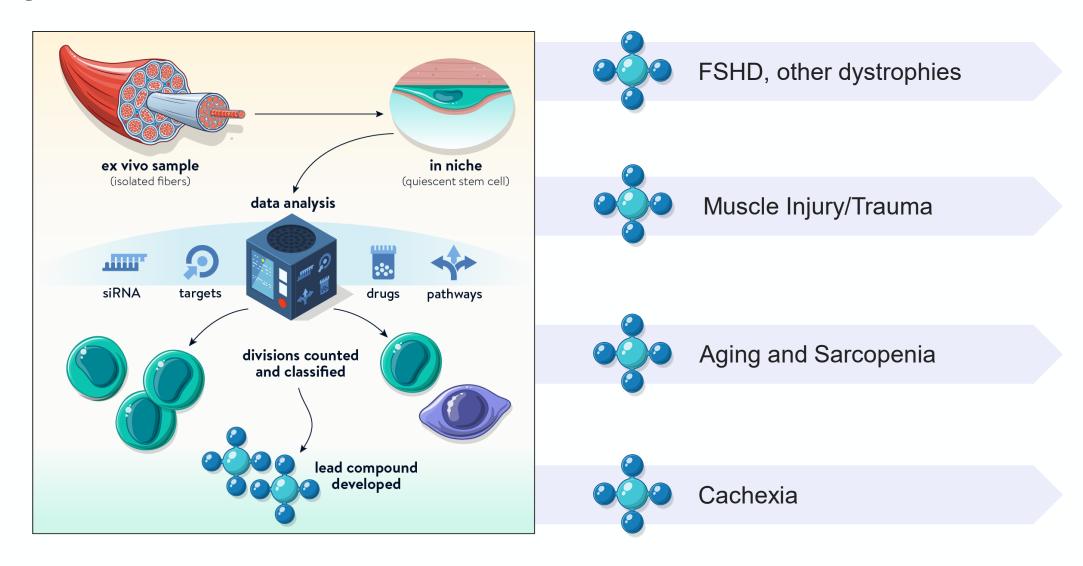
Small molecule drug to restore dystrophin signal in muscle stem cells



Potential to treat all patients regardless of genetics or prior treatment history



MyoReGenX™ platform powers pipeline expansion





Current Financials



Capital Table	
Common Share	152.5 M
Warrants	12.3 M
Stock Options	14.4 M
Fully Diluted	179.2 M
Insiders: ~15% (fully diluted) Cash at 12/31/23 ~CA\$39.6M	
TSY: MSCL LOTCOR: MSCLE	





Experts in muscle stem cell biology, drug development and biotechnology



Frank Gleeson, MBA Co-Founder & CEO



Michael Rudnicki, PhD, OC, FRS, FRSC Co-Founder & Chief

Discovery Officer



Philip Lambert, PhD
Chief Scientific Officer



Liz Williams, CPA, CA
Chief Financial Officer



Michael Cross, PhD, MBA
Chief Business Officer



Courtney Wells

SVP Clinical Development
Operations

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Frank Gleeson, MBA Director & CEO

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William McVicar, PhD Director

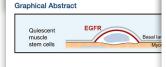
medicine

Dystrophin expression in muscle stem cells regulates their polarity and asymmetric division

Nicolas A Dumont^{1,2,4}, Yu Xin Wang^{1,2,4}, Julia von Maltzahn^{1,3}, Alessandra Pasut^{1,2}, C Florian Bentzinger^{1,3}, Caroline E Brun^{1,2} & Michael A Rudnicki^{1,2}

Cell Stem Cell

EGFR-Aurka Signaling Rescues Polarity and Regeneration Defects in Dystrophin-Deficient Muscle Stem Cells by Increasing Asymmetric Divisions



Cell Stem Cell
Review

Orienting Muscle Stem Cells for Regeneration in Homeostasis, Aging, and Disease

Peter Feige, 1-2 Caroline E. Brun, 1-2 Morten Ritso, 1-2 and Michael A. Rudnicki 1-2-15prott Centre for Stem Cell Research, Regenerative Medicine Program, Ottawa Hospital Research Institute, Ottawa, ON K1H 8L6, Canada *Department of Cellular and Molecular Medicine, Faculty of Medicine, University of Ottawa, Ottawa, ON K1H 8M5, Canada *Correspondence: mudnicidifications.ca *Institutes/fois.oru? Olifist stem 2018.10.008



REIMAGINE how degenerative muscle diseases are treated.

REGENERATE with novel small molecule medicines.

REALIZE the next horizon to improve lives.



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