Tackling metabolic disorders with non-coding RNAs

Resalis Therapeutics' transformative metabolic disease approach targets a master regulator of multiple pathways underlying obesity and fatty liver disease. The company is applying its profound understanding of the non-coding RNA drug modality and lipid metabolism to develop its lead program, RES-010, into a safe and convenient treatment providing disease-modifying therapeutic impact including durable weight loss and reduction of hepatic steatosis. Building on robust preclinical evidence, Resalis will rapidly bring RES-010 into clinical trials for a range of metabolic disorders.

Founded: 2021 by pioneers in ncRNA drug discovery and development

HQ: Torino, Italy

Scientific roots: Harvard Medical School (USA) and Aalborg University (DK)

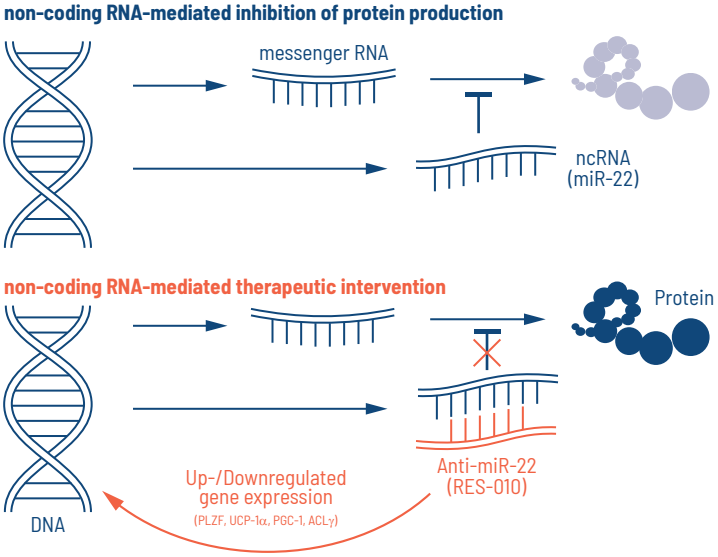
Therapeutic focus: Metabolic diseases

Funding to date: Seed round led by Claris Ventures with support from angel investors

Leadership: Experienced Big Pharma management and high-level RNA medicine advisors

Lead Program

Resalis has elucidated the central role of ncRNA miRNA-22 (miR-22) at the intersection of a range of molecular pathways underlining metabolic disorders. By applying a deep understanding of the ncRNA modality together with these insights, the company has developed its lead candidate, RES-010. RES-010 is an antisense oligonucleotide that targets miR-22 and is designed to become a safe and convenient treatment option with durable disease-modifying therapeutic impact.



RES-010 inhibits miR-22 to:

- Restore regulation of lipid biosynthesis
- Promote metabolic rewiring towards higher energy expenditure
- Transform white adipose tissue into brown adipose tissue
- Reduce and prevent hepatic steatosis, inflammation and fibrosis
- Induce weight loss

Resalis has completed multiple proof-of-concept studies in animal models including rodents and non-human primates and is now focused on IND-enabling studies to achieve entry into the clinic in early 2024.

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Clinical Develoment Strategy

New marketed treatments for obesity have shown the ability to reduce weight in many patients, providing the first medical success for an indication that has significant impact on a range of health concerns.

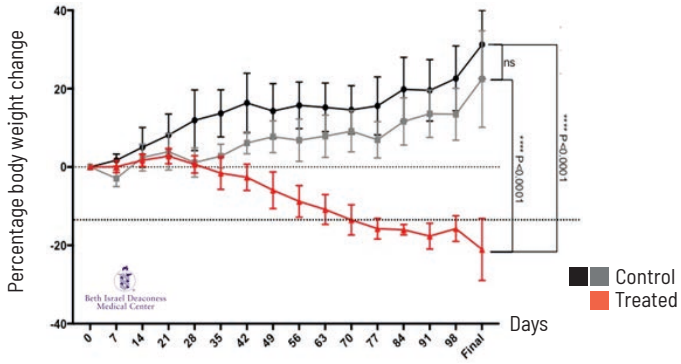
Nevertheless, these new drugs have limitations with durability of effect and in addressing other molecular pathways.

Resalis' RES-010 has to date exhibited a unique profile that may enable it to provide longer-lasting weight reduction, alone or in combination with approved drugs, by providing an orthogonal approach to other therapeutics.

In addition, its mechanism of action shows promise in addressing liver fibrosis and inflammation, opening a door to meeting the major need for effective fatty liver disease treatments.

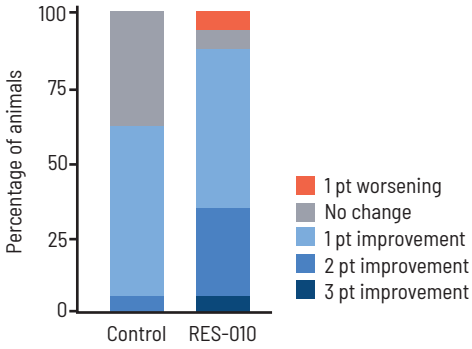
Obese mice on high fat diet

- Up to 40% weight reduction vs control
- Weight loss starts 3 weeks after treatment start
- Trend continued as of study termination



NAFLD score

- RES-010 shows benefit vs control
- Total improvement 81% vs 44%



Resalis will focus initially on bringing RES-010 into the clinic for obesity and fatty liver disease, while further investigating other indications, formulations and assets that will establish the company with a strong position in treating metabolic diseases by leveraging the ncRNA modality.

Team

Leadership

Alessandro Toniolo
Chief Executive Officer
Riccardo Panella
CSO & Founder
Sakari Kauppinen
CTO & Founder

Board of Directors

Pietro Puglisi *Chairman*
Michael Hodges
Antonio Leone

Investors

CLARIS VENTURES

15 Anni 2007-2022 Italian Angels for Growth

CLUB degli INVESTITORI

Publications

Assessment of immunostimulatory responses to the antimiR-22 oligonucleotide compound RES-010 in human peripheral blood mononuclear cells. Panella, R et al., Front. Pharmacol. Vol. 14, 2023

miR-22 modulates brown adipocyte thermogenesis by synergistically activating the glycolytic and mTORC1 signaling pathways. Lou, P et al., Theranostics, 11(8), 3607, 2021

Metabolic and energetic benefits of microRNA-22 inhibition. BMJ Open Diabetes Research and Care, Thibonnier, M et al., 8(1), e001478, 2020

miR-22 inhibition reduces hepatic steatosis via FGF21 and FGFR1 induction. Hu, Y et al., JHep Reports, 2(2), 100093, 2020

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