UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event Reported): June 24, 2024

Scholar Rock Holding Corporation
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-38501**	**82-3750435**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification Number)

301 Binney Street, 3rd Floor, Cambridge, MA 02142
(Address of Principal Executive Offices) (Zip Code)

(857) 259-3860
(Registrant's telephone number, including area code)

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.001 per share	SRRK	Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2). Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure.

On June 23, 2024, Scholar Rock Holding Corporation (the "Company") presented new data from its preclinical study of SRK-439 in combination with GLP-1 receptor agonist ("GLP1-RA") in an oral presentation during the American Diabetes Association ("ADA") 84th Scientific Sessions.

A copy of the press release relating to the ADA presentation is attached hereto as Exhibit 99.1 and a copy of the ADA presentation slides are attached hereto as Exhibit 99.2.

The information in this report furnished pursuant to Item 7.01 and Exhibits 99.1 and 99.2 shall not be deemed "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section nor shall such information be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, regardless of the general incorporation language of such filing, except as shall be expressly set forth by specific reference in such filing. It may only be incorporated by reference in another filing under the Exchange Act or the Securities Act of 1933, as amended, if such subsequent filing specifically references the information furnished pursuant to Item 7.01 and Exhibits 99.1 and 99.2 of this report.

Item 8.01. Other Events.

On June 23, 2024, the Company presented new data from its preclinical study of SRK-439 in combination with GLP-1RAs. The preclinical study tested a murine equivalent of SRK-439 in a diet-induced obesity ("DIO") mouse model. Mice were given either a high-fat diet plus semaglutide (0.04 mg/kg, daily) and an IgG control antibody (weekly, 10 mg/kg), or a high-fat diet plus semaglutide (0.04 mg/kg, daily) in combination with weekly injections of SRK-439 (10 mg/kg). Following four weeks of treatment, semaglutide was withdrawn from both treatment groups and mice remained on either the IgG control antibody weekly or on SRK-439. Treatment continued for another four weeks, for a total of eight weeks in the study. Quantitative nuclear magnetic resonance ("qNMR") was used to analyze change in lean mass at two weeks and again at four weeks of semaglutide treatment, and every two weeks after that until the end of the subsequent four-week withdrawal period.

The group that received SRK-439 maintained more favorable body composition than the group receiving IgG antibody. Key findings supporting the potential for SRK-439 in advancing healthier weight management include:

- Administration with SRK-439 attenuated the loss of lean mass during semaglutide treatment and significantly increased lean mass after semaglutide discontinuation as compared to IgG control;
- SRK-439 administration also attenuated the fat mass rebound after semaglutide discontinuation as compared to that in IgG control + semaglutide mice; and
- Body composition, i.e. proportion of lean mass or fat mass to total body weight, was more favorable in mice receiving SRK-439 as compared to IgG control: Mice administered SRK-439 had higher relative lean mass (65.8%) and lower relative fat mass (18.0%) at the end of the withdrawal period compared to IgG control (57.1% lean mass and 28.7% fat mass).

Shown below are results for body composition at baseline (6 days before semaglutide treatment), the end of semaglutide treatment (at 4 weeks), and at the end of the semaglutide withdrawal period (at 8 weeks):

Endpoint (units)	**IgG control + semaglutide**	**SRK-439 + semaglutide**	**P value**
Absolute lean mass (g) at baseline	24.8	25.5	n.s.
Absolute lean mass (g) at 4 weeks	22.3	26.4	P<0.001
Absolute lean mass (g) at 8 weeks	25.1	29.4	P<0.0001
Absolute fat mass (g) at baseline	11.8	10.3	n.s.
Absolute fat mass (g) at 4 weeks	5.9	3.8	n.s.
Absolute fat mass (g) at 8 weeks	12.7	8.3	n.s.
Relative lean mass (%) at 8 weeks	57.1%	65.8%	P<0.001
Relative fat mass (%) at 8 weeks	28.7%	18.0%	P<0.01

Additionally, the Company announced that the first participants were dosed in the Phase 2 EMBRAZE proof-of-concept trial, designed to assess the safety and efficacy of apitegromab, an investigational, highly selective myostatin inhibitor, to preserve lean muscle mass in individuals living with obesity and on background therapy of a GLP-1 RA.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press Release issued by the Company on June 24, 2024, furnished hereto.
99.2	Presentation Slides, furnished hereto.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Scholar Rock Holding Corporation

Date: June 24, 2024

By: /s/ Junlin Ho
Junlin Ho
General Counsel and Corporate Secretary

Scholar Rock Announces New SRK-439 Preclinical Data Showing Significant Lean Mass Preservation and Attenuation of Fat Mass Rebound Following GLP-1 Receptor Agonist Withdrawal

- *SRK-439, a myostatin inhibitor, is part of Scholar Rock's industry-leading anti-myostatin portfolio*
- *Obesity program continues to progress, with first participants dosed in Phase 2 EMBRAZE trial of apitegromab in obesity*

CAMBRIDGE, Mass. — June 24, 2024 — Scholar Rock (NASDAQ: SRRK), a late-stage biopharmaceutical company focused on advancing innovative treatments for spinal muscular atrophy (SMA), cardiometabolic disorders, and other serious diseases where protein growth factors play a fundamental role, today announced that the first participants were dosed in the Phase 2 EMBRAZE proof-of-concept trial, designed to assess the safety and efficacy of apitegromab, an investigational, highly selective myostatin inhibitor, to preserve lean muscle mass in individuals living with obesity and on background therapy of a GLP-1 receptor agonist (GLP-1 RA). The trial will also evaluate the effects of apitegromab on the durability of weight loss upon withdrawal of GLP-1 RA therapy. The results from this trial will inform the development of SRK-439, a novel investigational selective myostatin inhibitor optimized for the treatment of cardiometabolic disorders, including obesity.

The Company also presented new preclinical data that support the potential of SRK-439 to increase lean mass and contribute to a favorable body composition following withdrawal from GLP-1 RA treatment. These data were presented by Melissa Fulham, PhD, of Scholar Rock, at the American Diabetes Association's 84th Scientific Sessions on June 23rd in Orlando, Florida.

"We are happy to share the exciting news that we've dosed the first participants in our EMBRAZE clinical trial ahead of schedule and to have new preclinical data with SRK-439, our highly selective anti-myostatin, featured at the American Diabetes Association Scientific Sessions," said Jay Backstrom, M.D., MPH, President and Chief Executive Officer at Scholar Rock. "SRK-439 preclinical data to date have demonstrated preservation of lean mass with GLP-1 RA-induced weight loss, attenuation of fat mass regain following GLP-1 RA withdrawal, and greater potency compared to an anti-ACTRII antibody. Together, these data continue to support a best-in-class potential for healthy weight loss management and could be transformative for the management of weight loss. We are looking forward to providing additional updates on our cardiometabolic program as we advance SRK-439, as well as the EMBRAZE trial."

Preclinical experimental design

For the preclinical research study, the Company tested a murine equivalent of SRK-439 in a diet-induced obesity (DIO) mouse model. Mice were given either a high-fat diet plus semaglutide (0.04 mg/kg, daily) and an IgG control antibody (weekly, 10 mg/kg), or a high-fat diet plus semaglutide (0.04 mg/kg, daily) in combination with weekly injections of SRK-439 (10 mg/kg). Following four weeks of treatment, semaglutide was withdrawn from both treatment groups and mice remained on either the IgG control antibody weekly or on SRK-439. Treatment continued for another four weeks, for a total of eight weeks in the study. Quantitative nuclear magnetic resonance (qNMR) was used to analyze change in lean mass

at two weeks and again at four weeks of semaglutide treatment, and every two weeks after that until the end of the subsequent four-week withdrawal period.

Changes in body composition after semaglutide withdrawal

The group that received SRK-439 maintained more favorable body composition than the group receiving IgG antibody. Key findings supporting the potential for SRK-439 in advancing healthier weight management include:

- Administration with SRK-439 attenuated the loss of lean mass during semaglutide treatment and significantly increased lean mass after semaglutide discontinuation as compared to IgG control;
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Relative lean mass (%) at 8 weeks	57.1%	65.8%	P<0.001
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“These new preclinical data provide compelling evidence that SRK-439 contributed to lean muscle preservation during GLP-1 RA-induced weight loss and attenuated fat mass rebound following discontinuation of semaglutide,” said Mo Qatanani, PhD, Chief Scientific Officer at Scholar Rock. “Mice receiving SRK-439 treatment had significantly more lean mass at the end of the semaglutide withdrawal period. These exciting data continue to support the differentiated profile of SRK-439 and its potential to contribute to healthier weight management and long-term metabolic benefits during and after GLP-1 RA treatment.”

For conference information, visit https://professional.diabetes.org/scientific-sessions.

The slides from the presentation are available in the Publications & Posters section of Scholar Rock’s website.

About EMBRAZE

EMBRAZE is a randomized, double-blind, placebo-controlled, Phase 2 proof-of-concept trial evaluating the efficacy, safety and pharmacokinetics of apitegromab in adults with a body mass index (BMI) of >27 (overweight) or a BMI of >30 (obese) and taking a GLP-1 RA (tirzepatide or semaglutide). The target enrollment of EMBRAZE is 100 subjects aged 18-65 who are overweight or obese without diabetes. As part of the study design, the treatment period is 24 weeks, and all subjects will receive a GLP-1 RA. In addition, all subjects will be randomized 1:1 to receive either apitegromab or placebo by intravenous (IV) infusion every four weeks during the 24-week treatment period. The primary endpoint is change from baseline at Week 24 in lean mass assessed by dual-energy X-ray absorptiometry. Secondary endpoints include additional weight loss measures, safety and tolerability, and pharmacokinetic outcomes. Exploratory endpoints at Weeks 24 and 32 include cardiometabolic parameters (e.g. HbA1c), body composition, and physical function.

About SRK-439

SRK-439 is a novel, preclinical, investigational myostatin inhibitor that binds to pro- and latent myostatin with high affinity and is selective for myostatin (i.e., no GDF11 or Activin-A binding), and is initially being developed for the treatment of cardiometabolic disorders, including obesity. Based on preclinical data, SRK-439 has the potential to support healthier weight management by preserving lean mass during weight loss. The efficacy and safety of SRK-439 have not been established and SRK-439 has not been approved for any use by the FDA or any other regulatory agency.

About Apitegromab

Apitegromab is an investigational fully human monoclonal antibody inhibiting myostatin activation by selectively binding the pro- and latent forms of myostatin in the skeletal muscle. It is the first muscle-targeted treatment candidate to demonstrate clinical proof-of-concept in spinal muscular atrophy (SMA). Myostatin, a member of the TGFβ superfamily of growth factors, is expressed primarily by skeletal muscle cells, and the absence of its gene is associated with an increase in muscle mass and strength in multiple animal species, including humans. Scholar Rock believes that its highly selective targeting of pro- and latent forms of myostatin with apitegromab may lead to a clinically meaningful improvement in motor function in patients with SMA. The U.S. Food and Drug Administration (FDA) has granted Fast Track, Orphan Drug and Rare Pediatric Disease designations, and the European Medicines Agency (EMA) has granted Priority Medicines (PRIME) and Orphan Medicinal Product designations, to apitegromab for the treatment of SMA. The efficacy and safety of apitegromab have not been established and apitegromab has not been approved for any use by the FDA or any other regulatory agency.

About Scholar Rock

Scholar Rock is a biopharmaceutical company that discovers, develops, and delivers life-changing therapies for people with serious diseases that have high unmet need. As a global leader in the biology of the transforming growth factor beta (TGFβ) superfamily of cell proteins and named for the visual resemblance of a scholar rock to protein structures, the clinical-stage company is focused on advancing innovative treatments where protein growth factors are fundamental. Over the past decade, Scholar Rock has created a pipeline with the potential to advance the standard of care for neuromuscular disease, cardiometabolic disorders, cancer, and other conditions where growth factor-targeted drugs can play a transformational role.

Scholar Rock is the only company to show clinical proof-of-concept for a muscle-targeted treatment in spinal muscular atrophy (SMA). This commitment to unlocking fundamentally different therapeutic approaches is powered by broad application of a proprietary platform, which has developed novel monoclonal antibodies to modulate protein growth factors with extraordinary selectivity. By harnessing cutting-edge science in disease spaces that are historically under-addressed through traditional therapies, Scholar Rock works every day to create new possibilities for patients. Learn more about our approach at ScholarRock.com and follow @ScholarRock and on LinkedIn.

Availability of Other Information About Scholar Rock

Investors and others should note that we communicate with our investors and the public using our company website www.scholarrock.com, including, but not limited to, company disclosures, investor presentations and FAQs, Securities and Exchange Commission filings, press releases, public conference call transcripts and webcast transcripts, as well as on Twitter and LinkedIn. The information that we post on our website or on Twitter or LinkedIn could be deemed to be material information. As a result, we encourage investors, the media and others interested to review the information that we post there on a regular basis. The contents of our website or social media shall not be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended.

Forward-Looking Statements

This press release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, including, but not limited to, statements regarding Scholar Rock's future expectations, plans and prospects, including without limitation, Scholar Rock's expectations regarding its growth, strategy, progress and timing of its clinical trials for apitegromab and its preclinical programs, including SRK-439, and indication selection and development timing, including the therapeutic potential, clinical benefits and safety thereof, expectations regarding timing, success and data announcements of current ongoing preclinical and clinical trials, the ability of any product candidate to perform in humans in a manner consistent with earlier nonclinical, preclinical or clinical trial data, and the potential of its product candidates and proprietary platform. The use of words such as "may," "might," "could," "will," "should," "expect," "plan," "anticipate," "believe," "estimate," "project," "intend," "future," "potential," or "continue," and other similar expressions are intended to identify such forward-looking statements. All such forward-looking statements are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, without limitation, that preclinical and clinical data, including the results from the Phase 2a clinical trial of apitegromab, or its preclinical data with respect to SRK-439, are not predictive of, may be inconsistent with, or more favorable than, data generated from future or ongoing clinical trials of the same product candidates, including, without limitation, the Phase 3 clinical trial of apitegromab in SMA or the Phase 2a EMBRAZE clinical trial; Scholar Rock's ability to provide the financial support, resources and expertise necessary to identify and develop product candidates on the expected timeline; the data generated from Scholar Rock's nonclinical and preclinical studies and clinical trials; information provided or decisions made by regulatory authorities; competition from third parties that are developing products for similar uses; Scholar Rock's ability to obtain, maintain and protect its intellectual property; Scholar Rock's dependence on third parties for development and manufacture of product candidates including, without limitation, to supply any clinical trials; and Scholar Rock's ability to manage expenses and to obtain additional funding when needed to support its business activities and establish and maintain strategic business alliances and new business initiatives; as well as those risks more fully discussed in the section entitled "Risk Factors" in Scholar Rock's Quarterly Report on Form 10-Q for the quarter ended March 31, 2024, as well as discussions of potential risks, uncertainties, and other important factors in

Scholar Rock's subsequent filings with the Securities and Exchange Commission. Any forward-looking statements represent Scholar Rock's views only as of today and should not be relied upon as representing its views as of any subsequent date. All information in this press release is as of the date of the release, and Scholar Rock undertakes no duty to update this information unless required by law.

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The Anti-Myostatin Antibody SRK-439 Promotes Healthy Body Composition in Combination with GLP-1RAs in a Mou Model of Obesity

Melissa A. Fulham, Christopher D. Chapron, Francis T. Danehy, Jr., Frederick C. Streich, Jr., Brian Liang, Christopher J. Boston, Justin W. Jackson, Yan Huang, Samantha B. Nicholls, Leslie K. Cortes, Mo Qatanani

Presenter disclosure

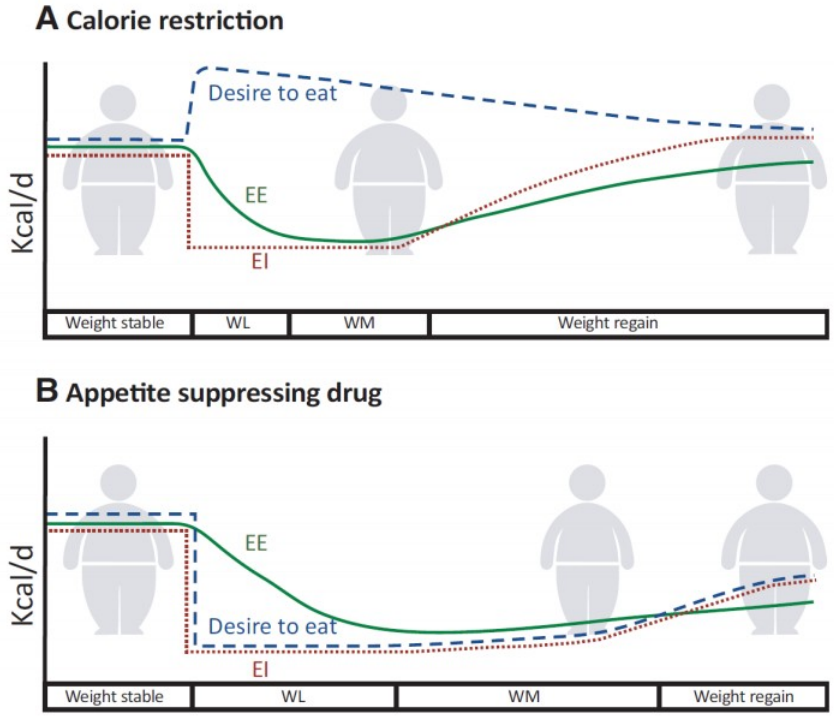
Melissa Fulham

Employee and Stock/Shareholder of Scholar Rock, Inc.

PHOTOGRAPHY PROHIBITED

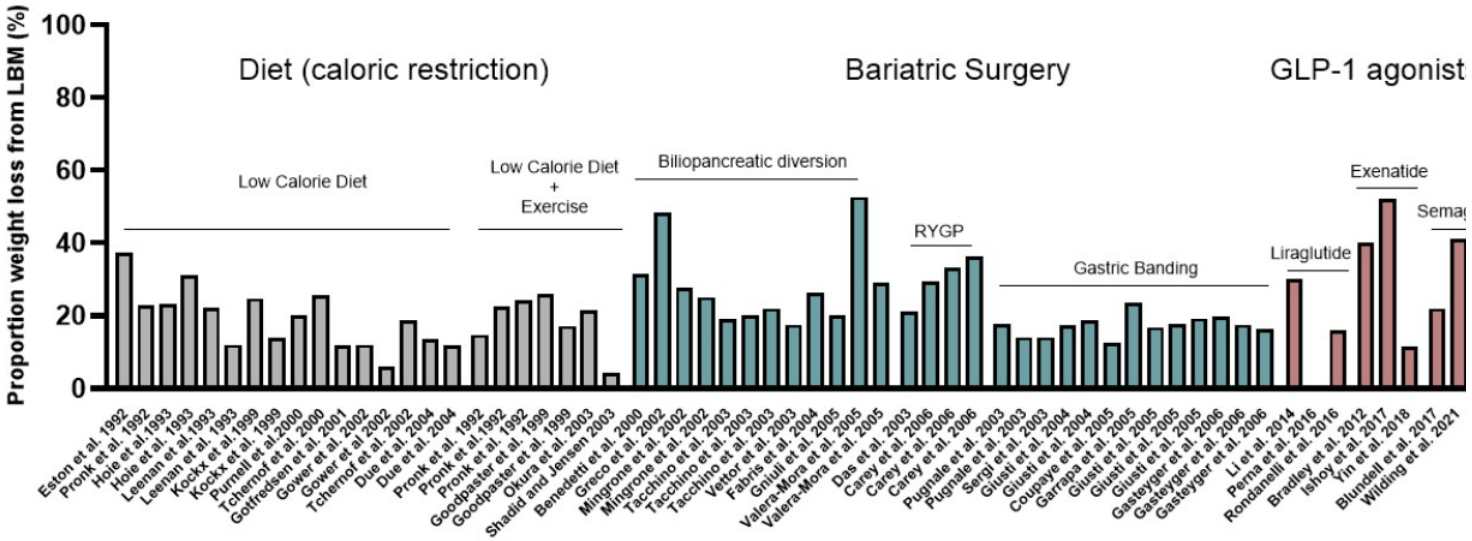
PLEASE DO NOT take photos of this presentation

Maintaining weight loss is challenging



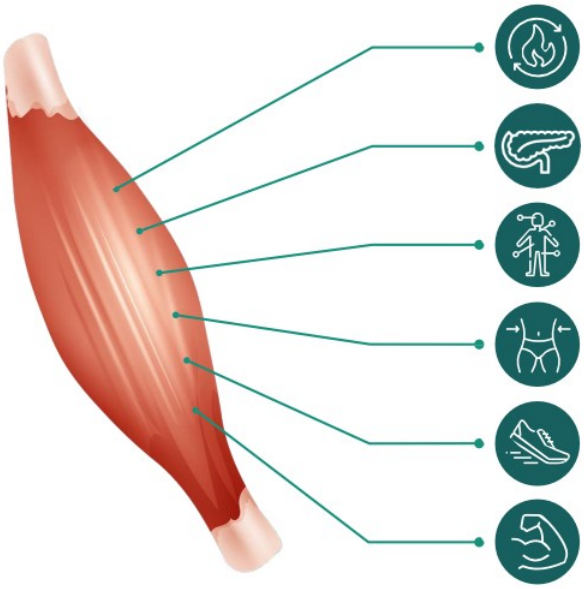
Christoffersen, B.Ø., *et al*. *Obesity (Silver Spring)*. 2022. PMID: 35333444

Lean mass is reduced during weight loss regardless of intervention



Chaston, T. B., *et al*. *Int J Obes*. 2007. PMID: 17075583
Sargeant, J. A., *et al. Endocrinol Metab*. 2019. PMID: 31565876
Wilding J.P.H., *et al*. *Diabetes Obes Metab*. 2022. PMID: 35441470

Muscle is critical for overall health

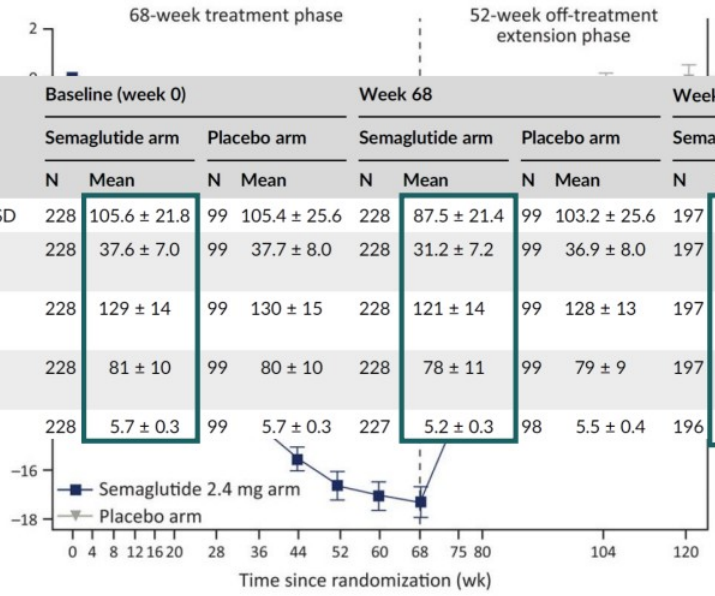


- Increase basal metabolic rate (BMR)[1]
- Enhance glucose homeostasis[2] and better insulin sensitivity and lower risk of prediabetes[3]
- Signal to other systems as an endocrine organ impacting overall health[4]
- Reduce visceral fat[5]
- Increase caloric expenditure post-exercise[6]
- Increase bone density, strength, function, and longevity and decreased risk of injury and disability[7-9]

1. Aristizabal, J.C., *et al. Eur J Clin Nutr*. 2015. 2. Lindegaard, B., *et al. J Clin Endocrinol Metab*. 2008. 3. Srikanthan, P. and Karlamangla, A.S. *J Clin Endocrinol Metab*. 2011. 4. Severinsen, and Pedersen, B.K. *Endocr Rev*. 2020. 5. Wewege, M.A., *et al. Sport Med*. 2022. 6. Zurlo, F., *et al. J Clin Investl*. 1990. 7. Fukushima, Y., *et al. Diabetes Metab J*. 2016. 8. Roh, E. and Choi, *Front. Endocrinol (Lausanne)*. 2020. 9. Volpi, E., *et al. Curr Opin Clin Nutr Metab Care*. 2004.

GLP-1RA withdrawal leads to weight regain and loss of cardiometabolic benefit

STEP 1 trial extension: Participants regained weight after semaglutide withdrawal



	Baseline (week 0)				Week 68				Week 120			
	Semaglutide arm		Placebo arm		Semaglutide arm		Placebo arm		Semaglutide arm		Placebo arm	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
Body weight (kg), mean ± SD	228	105.6 ± 21.8	99	105.4 ± 25.6	228	87.5 ± 21.4	99	103.2 ± 25.6	197	99.0 ± 22.5	93	105.5 ± 26.2
Body mass index (kg/m^2), mean ± SD	228	37.6 ± 7.0	99	37.7 ± 8.0	228	31.2 ± 7.2	99	36.9 ± 8.0	197	35.0 ± 7.1	93	37.6 ± 8.2
Systolic blood pressure (mmHg), mean ± SD	228	129 ± 14	99	130 ± 15	228	121 ± 14	99	128 ± 13	197	131 ± 15	93	132 ± 15
Diastolic blood pressure (mmHg), mean ± SD	228	81 ± 10	99	80 ± 10	228	78 ± 11	99	79 ± 9	197	82 ± 10	93	81 ± 11
HbA1c (%), mean ± SD	228	5.7 ± 0.3	99	5.7 ± 0.3	227	5.2 ± 0.3	98	5.5 ± 0.4	196	5.6 ± 0.3	91	5.7 ± 0.5

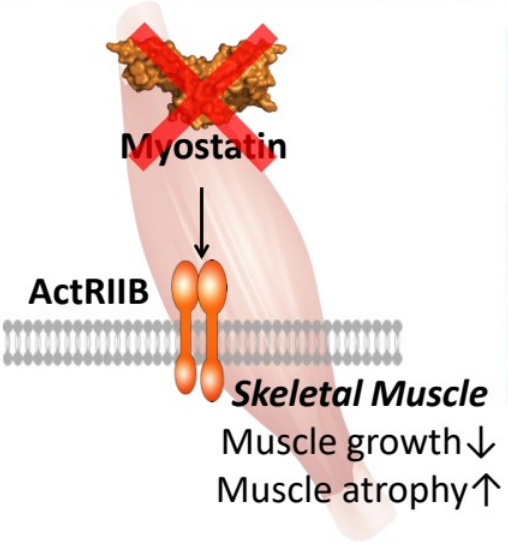
Wilding J.P.H., *et al*. *Diabetes Obes Metab*. 2022. PMID: 35441470

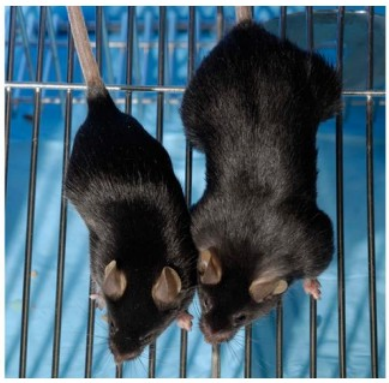
Can we mitigate weight regain and loss of metabolic benefit?

- This is not unique to GLP-1RAs; weight is regained with all weight loss methods

- How can we maintain or increase lean mass during weight loss?

Belgian blue cattle;
MSTN-null



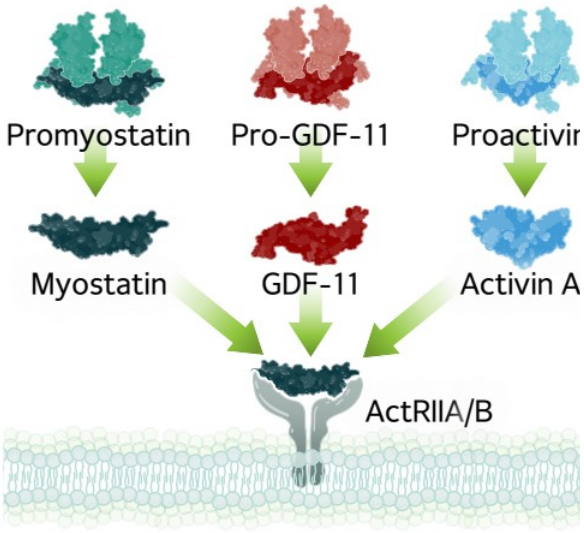


Mstn$^{-/-}$ mice

Lee, S.J. *PLoS One*. 2007. PMID: 1

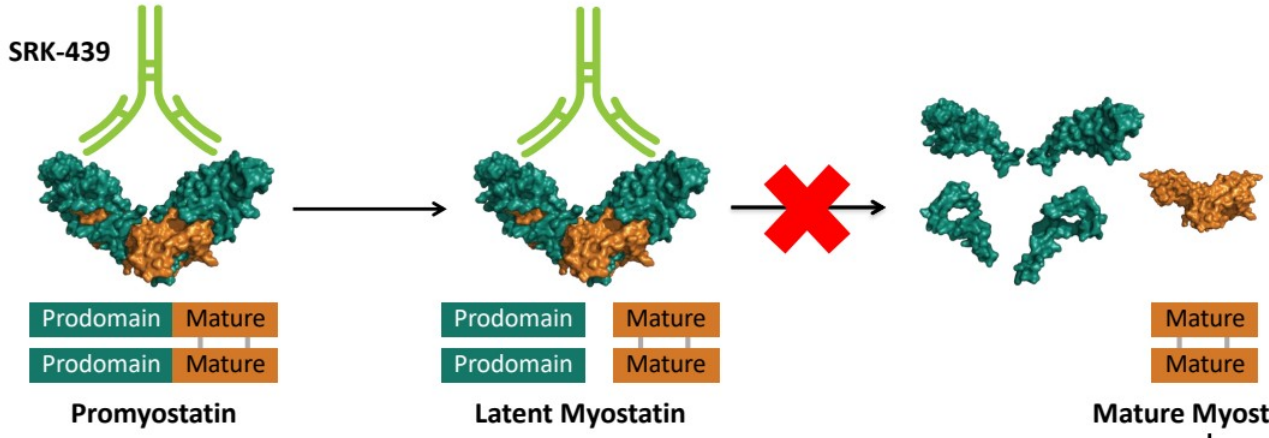
Selectively targeting myostatin is challenging but critical

- Developing an anti-myostatin agent is difficult due to a high degree of similarity with Activin A and GDF-11
- Selectively targeting myostatin is important:
  - ActRIIB/Activin A/GDF11 KO mice all have perinatal lethality due to developmental defects in multiple organ systems
  - *GDF11* LOF variants are associated with severe craniofacial, neurological, and skeletal phenotypes in humans
  - Inhibition of ActRII or Activin A in adult humans is associated with several health risks, including significant reduction in follicle-stimulating hormone levels

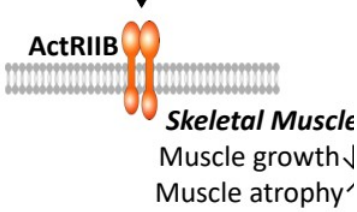


Oh, S.P. and Li, E. *Genes Dev*. 1997.; Matzuk, M. M., *et al*. *Nature*. 199
McPherron, A.C., *et al*. *Nat Genet*. 1999.; Garito, T., *et al*. *Clin Endocri*
(Oxf). 2018.; Bloise, E. *et al*. *Physiol Rev*. 2019.; Ravenscroft, T.A., *et a*
Genet Med. 2021.

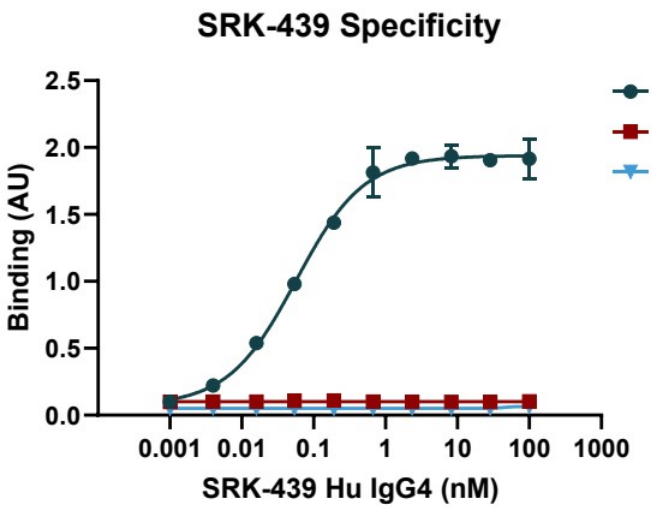
SRK-439 binds to pro- and latent myostatin and enables muscle growth



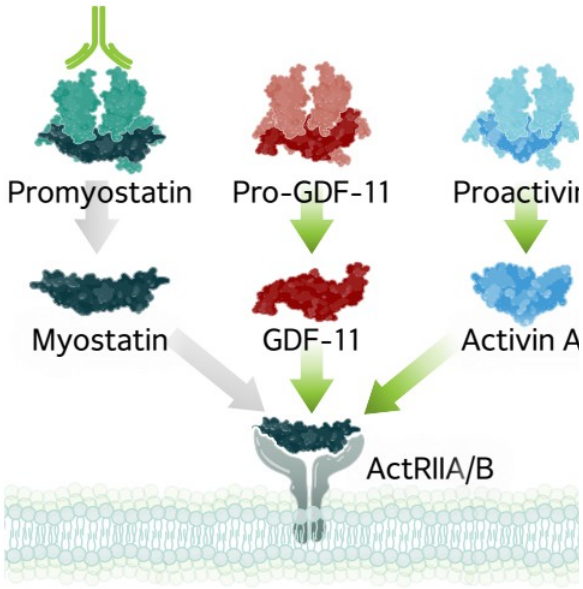
- SRK-439 binds to pro- and latent myostatin and blocks the conversion of the latent form to mature myostatin
- By inhibiting the release of mature myostatin, SRK-439 prevents myostatin from interacting with receptors which results in the inhibition of signaling and promotes muscle growth



SRK-439 is exquisitely selective for pro-and latent-myostatin



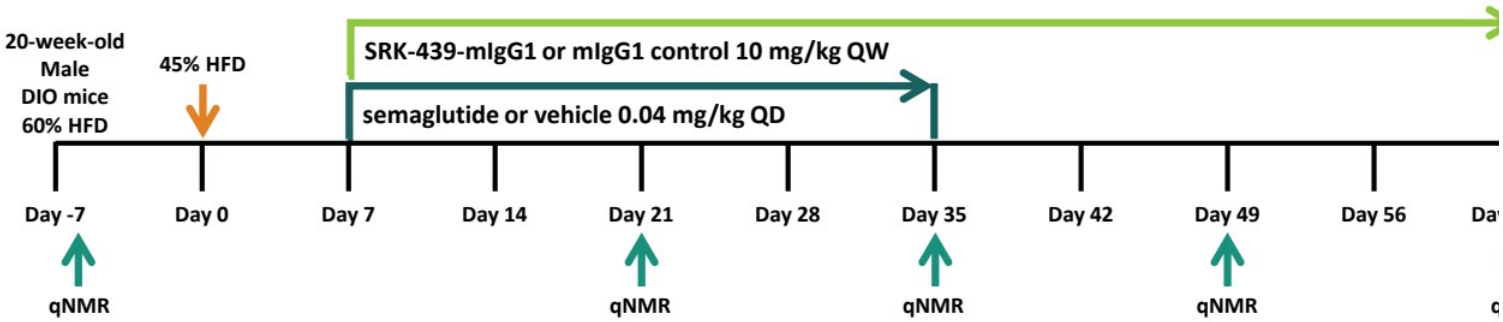
K_D = 0.579 nM



Therapeutic hypothesis

Inhibiting myostatin, a negative regulator of muscle mass, during GLP-1RA-induced weight loss will maintain lean mass and result in a favorable body composition after GLP-1RA withdrawal

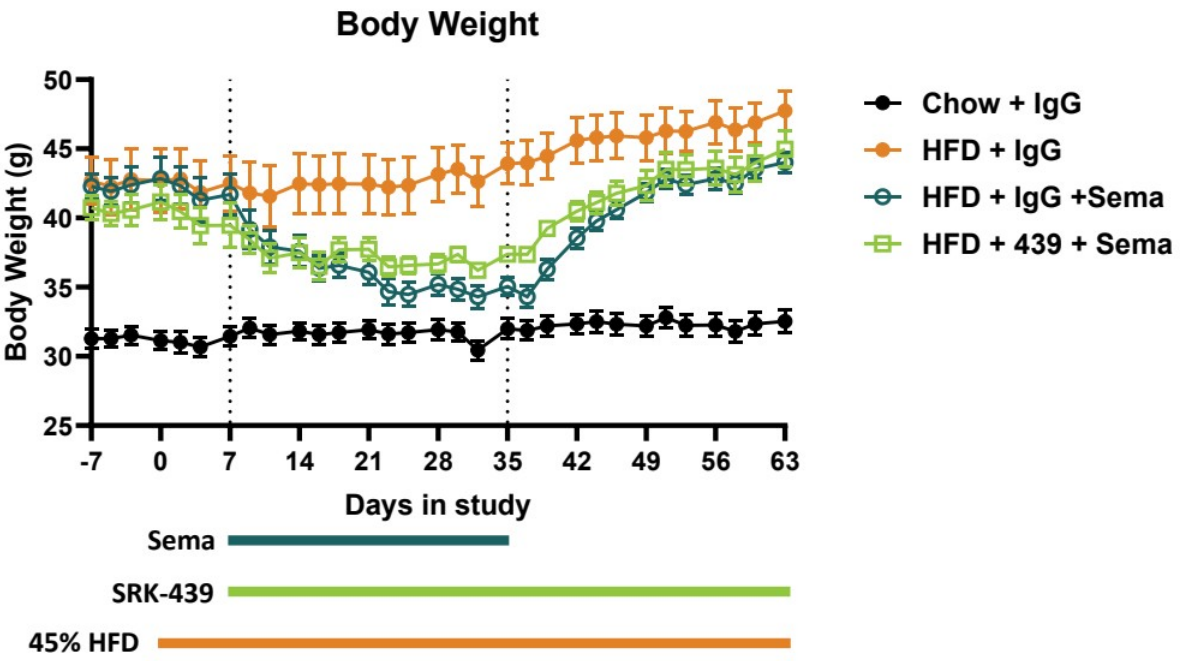
Administering SRK-439-mIgG1 to DIO mice during semaglutide treatme and after discontinuation



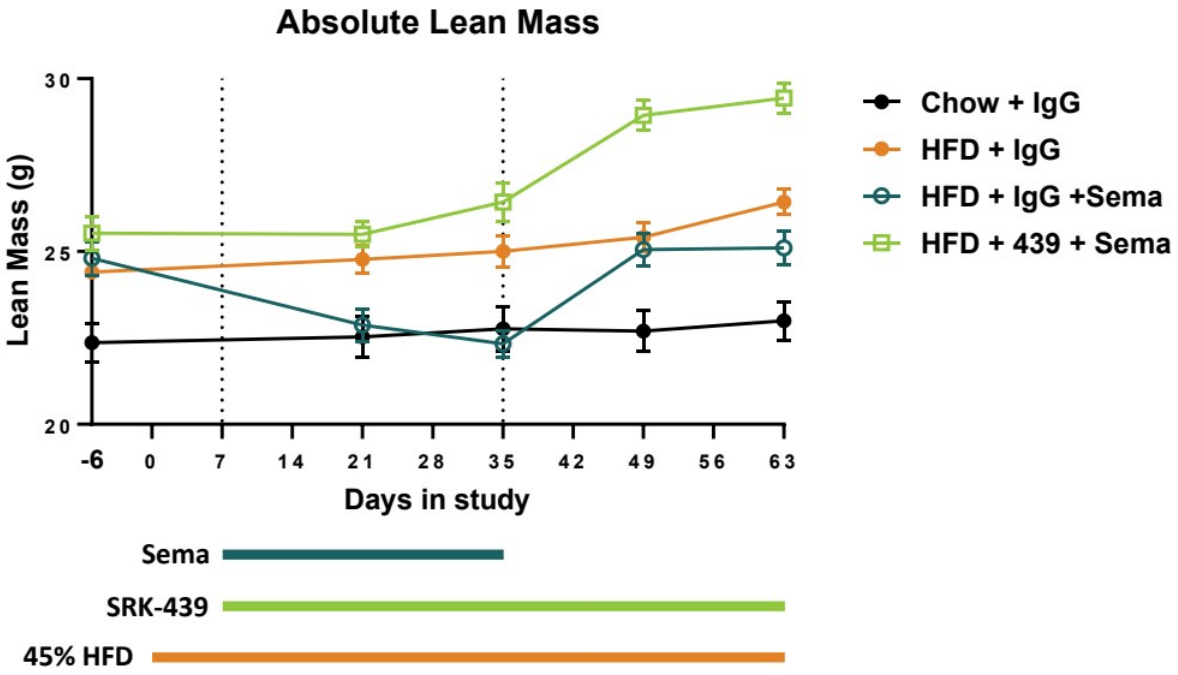
Diet	Ab	GLP-1RA
Chow	IgG	vehicle
HFD	IgG	vehicle
HFD	IgG	sema
HFD	SRK-439	sema

*IgG = mIgG1
*SRK-439 or 439 = SRK-439-mIgG1

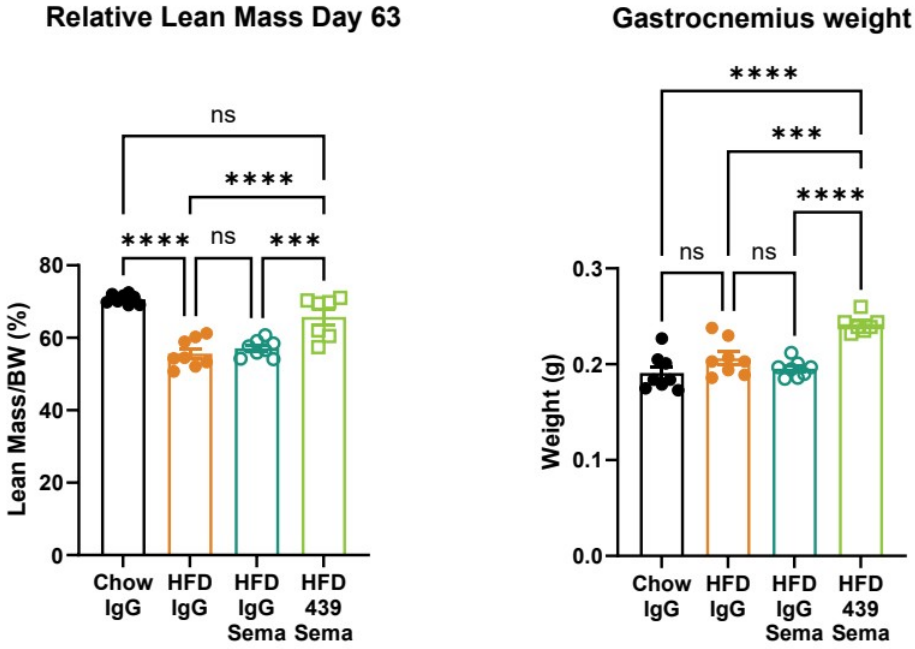
Semaglutide discontinuation caused weight regain



SRK-439 maintained lean mass in combination with semaglutide administratio

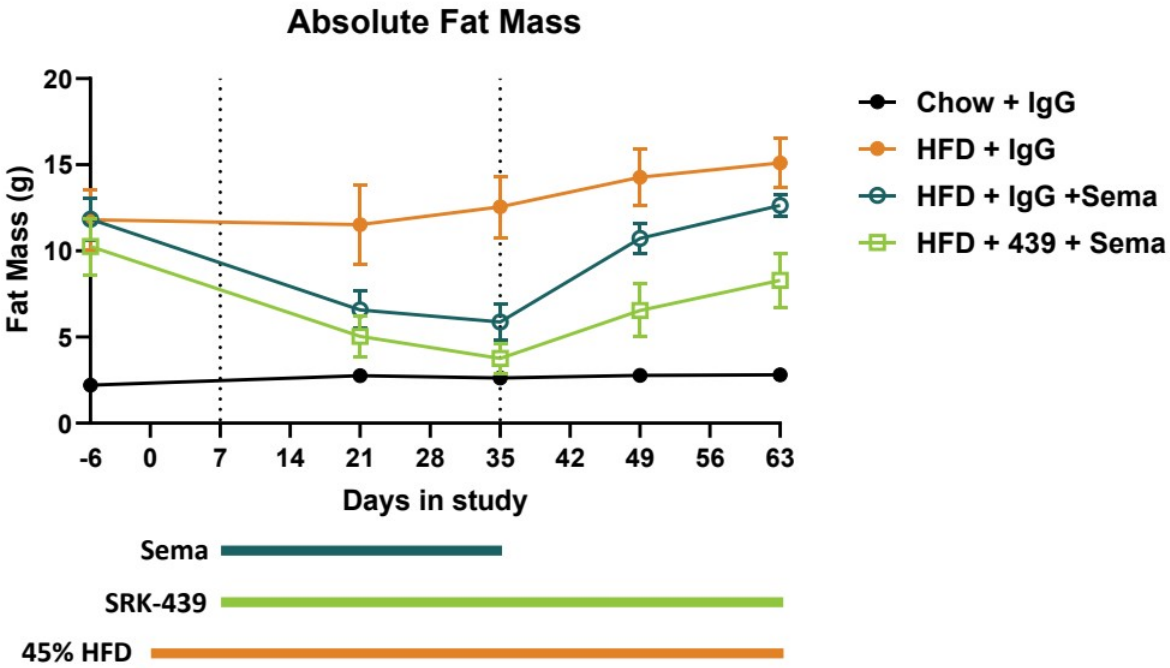


SRK-439 increased relative lean mass and skeletal muscle weight

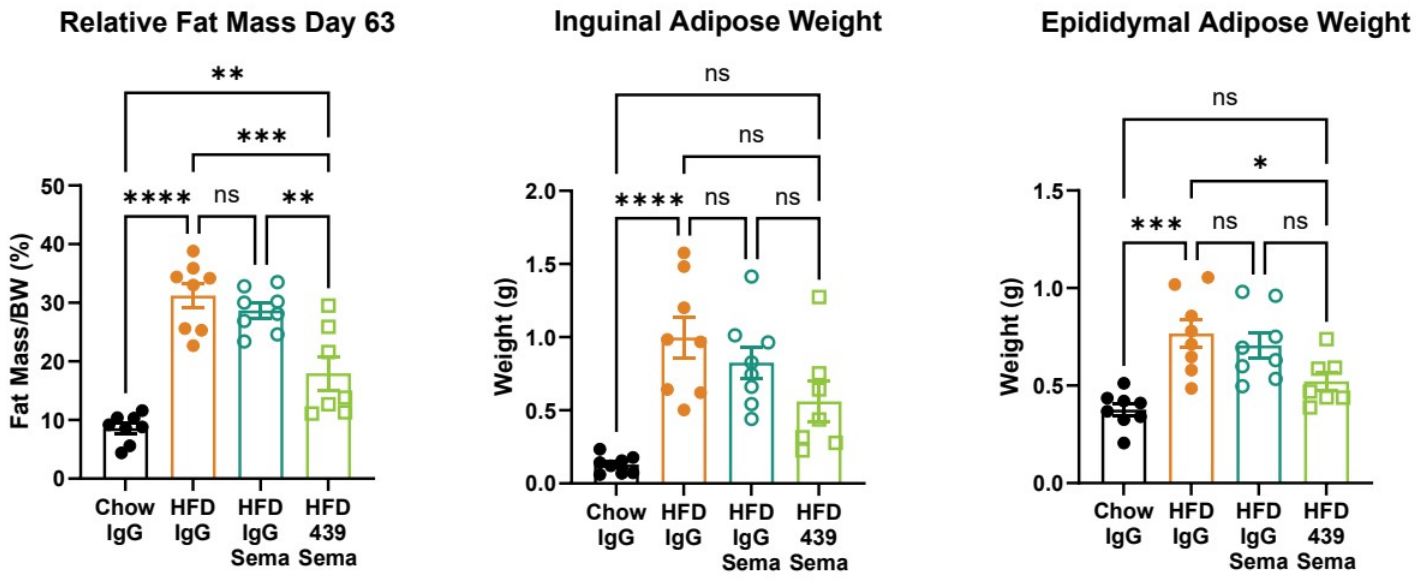


SRK-439 administration in combination with and after withdrawal of semaglutide results in lean mass composition similar to chow-fed animals

SRK-439 attenuated fat mass regain after discontinuation of semaglutic

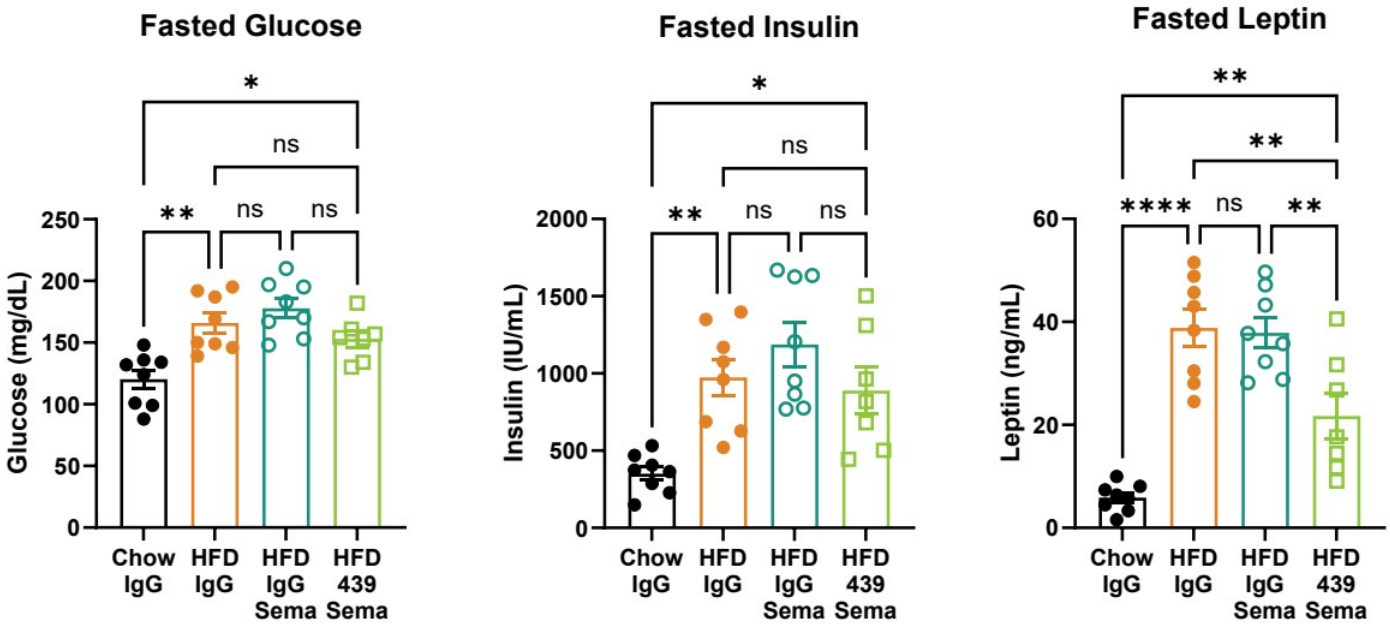


SRK-439 treatment improved body composition



SRK-439 administration in combination with and after withdrawal of semaglutide lowers percent fat mass and reduces adipose depot size

SRK-439 improved circulating metabolic biomarkers



SRK-439 administration in combination with and after withdrawal of semaglutide reduces circulating leptin which confirms reduced adiposity

Summary

- Lean mass decreases during weight loss, regardless of the intervention
- Selectively inhibiting myostatin increases lean mass without the potential liabilities of no selective targeting of the broader family
- SRK-439-mIgG1 administration prevented lean mass loss during semaglutide-induced weight loss and increased lean mass after semaglutide discontinuation
- SRK-439-mIgG1 administration resulted in lower body fat composition and lower circulat leptin during weight regain

SRK-439-mIgG1 maintains a healthy body composition during GLP-1RA-induce weight loss and subsequent regain following discontinuation

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