PARIS MASH MEETING

10th edition

Organized by

Arun Sanyal & Lawrence Serfaty

September 5 & 6 2024

Institut Pasteur, Paris

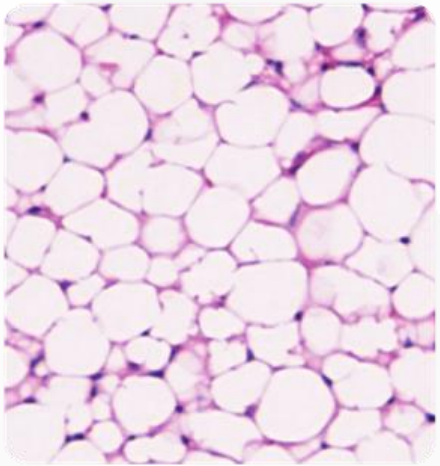
PARIS MASH MEETING

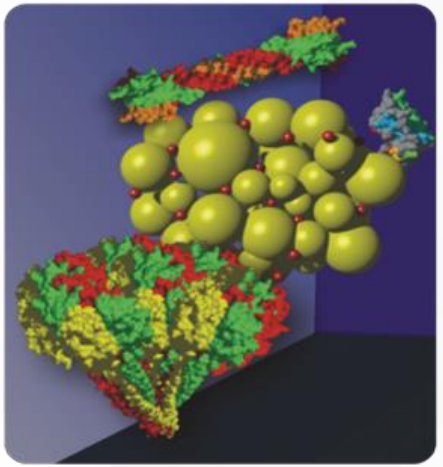
10th edition

September 5 & 6 2024
Institut Pasteur, Paris

BASIC SCIENCE LECTURE

Interplay between adipose tissue and GLP1, FGF21 in MASLD





Paris MASH Meeting
Institut Pasteur
Paris, France
June 17, 2024

Philipp E. Scherer, PhD
Touchstone Diabetes Center
University of Texas
Southwestern Medical Center
Dallas, Texas

COI

Philipp Scherer declares

- Sponsored Research Agreement with Eli Lilly

- NASH Merck Investigator Studies Program (MISP)
- Global NASH Group Input Advisor

- Scientific co-founder PriveBio

Cell Metabolism

Cell Metabolism *34*, January 4, 2022

Perspective

Why does obesity cause diabetes?

Samuel Klein,[1,2,*] Amalia Gastaldelli,[3] Hannele Yki-Järvinen,[4,5] and Philipp E. Scherer[6,7]

Weight Loss

Liver
Insulin sensitivity
Triglyceride content
NASH

Muscle
Insulin sensitivity
Triglyceride content

Adipose tissue
Insulin sensitivity
Adipose tissue mass
Adipose tissue biology

Pancreas
Insulin secretory function
Triglyceride content

Kidney
Kidney function
Albuminuria

Dyslipidemia
Triglyceride
HDL-C

Cardiovascular system
Blood pressure
Cardiovascular structure
and function

Cell
Leading Edge

Cell *187*, July 25, 2024

CellPress

Review

Transforming obesity: The advancement of multi-receptor drugs

Christine M. Kusminski,[1] Diego Perez-Tilve,[2] Timo D. Müller,[3,4] Richard D. DiMarchi,[5] Matthias H. Tschöp,[6,7] and Philipp E. Scherer[1,*]

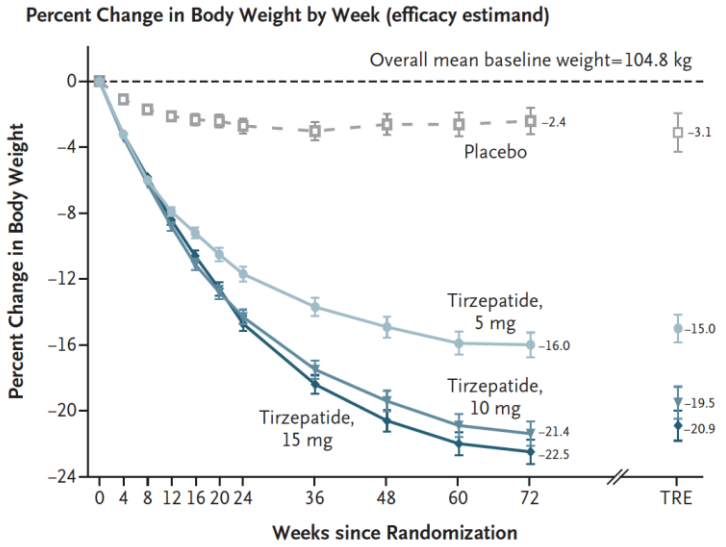
[1]Touchstone Diabetes Center, The University of Texas Southwestern Medical Center, Dallas, TX 75390, USA
[2]Department of Pharmacology and Systems Physiology, University of Cincinnati College of Medicine, Cincinnati, OH, USA
[3]Institute for Diabetes and Obesity, Helmholtz Diabetes Center, Helmholtz Munich, Munich, Germany
[4]German Center for Diabetes Research (DZD) and Walther-Straub Institute of Pharmacology and Toxicology, Ludwig-Maximilians-University (LMU) Munich, Munich, Germany
[5]Department of Chemistry, Indiana University, Bloomington, IN, USA
[6]Helmholtz Munich, Munich, Germany
[7]Division of Metabolic Diseases, Department of Medicine, Technische Universität, Munich, Germany
*Correspondence: philipp.scherer@utsouthwestern.edu

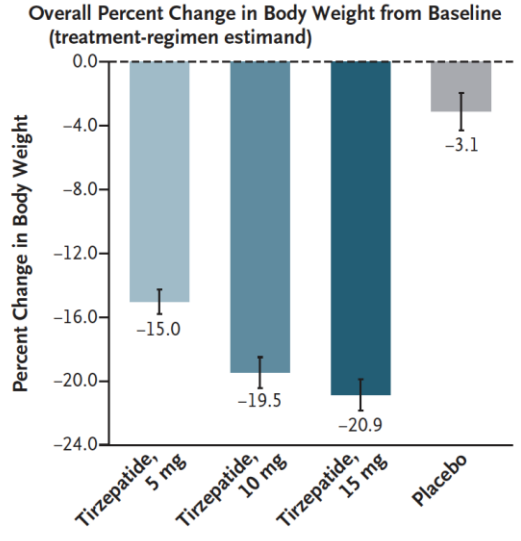
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JULY 21, 2022 VOL. 387 NO. 3

Tirzepatide Once Weekly for the Treatment of Obesity

Ania M. Jastreboff, M.D., Ph.D., Louis J. Aronne, M.D., Nadia N. Ahmad, M.D., M.P.H., Sean Wharton, M.D., Pharm.D., Lisa Connery, M.D., Breno Alves, M.D., Arihiro Kiyosue, M.D., Ph.D., Shuyu Zhang, M.S., Bing Liu, Ph.D., Mathijs C. Bunck, M.D., Ph.D., and Adam Stefanski, M.D., Ph.D., for the SURMOUNT-1 Investigators*





GLP1-R
GIP-R

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes

Vlado Perkovic, M.B., B.S., Ph.D., Katherine R. Tuttle, M.D., Peter Rossing, M.D., D.M.Sc., Kenneth W. Mahaffey, M.D., Johannes F.E. Mann, M.D., George Bakris, M.D., Florian M.M. Baeres, M.D., Thomas Idorn, M.D., Ph.D., Heidrun Bosch-Traberg, M.D., Nanna Leonora Lausvig, M.Sc., and Richard Pratley, M.D., for the FLOW Trial Committees and Investigators*

This article was published on May 24, 2024, at NEJM.org.

DOI: 10.1056/NEJMoa2403347

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Semaglutide in Patients with Heart Failure with Preserved Ejection Fraction and Obesity

M.N. Kosiborod, S.Z. Abildstrøm, B.A. Borlaug, J. Butler, S. Rasmussen, M. Davies, G.K. Hovingh, D.W. Kitzman, M.L. Lindegaard, D.V. Møller, S.J. Shah, M.B. Treppendahl, S. Verma, W. Abhayaratna, F.Z. Ahmed, V. Chopra, J. Ezekowitz, M. Fu, H. Ito, M. Lelonek, V. Melenovsky, B. Merkely, J. Núñez, E. Perna, M. Schou, M. Senni, K. Sharma, P. Van der Meer, D. von Lewinski, D. Wolf, and M.C Petrie, for the STEP-HFpEF Trial Committees and Investigators*

Cell Metabolism

CellPress

Commentary

A big STEP for treatment of heart failure with preserved ejection fraction

Subodh Verma,[1,2,3,13,*] Barry A. Borlaug,[4] Javed Butler,[5,6,14] Melanie J. Davies,[7,8,15] Dalane W. Kitzman,[9] Mark C. Petrie,[10,16] Sanjiv J. Shah,[11,17] Nitish K. Dhingra,[1,18] and Mikhail N. Kosiborod[12,19,*]

Semaglutide and cardiovascular outcomes in patients with obesity and prevalent heart failure: a prespecified analysis of the SELECT trial

www.thelancet.com Vol 404 August 24, 2024

John Deanfield, Subodh Verma, Benjamin M Scirica, Steven E Kahn, Scott S Emerson, Donna Ryan, Ildiko Lingvay, Helen M Colhoun, Jorge Plutzky, Mikhail N Kosiborod, G Kees Hovingh, Søren Hardt-Lindberg, Ofir Frenkel, Peter E Weeke, Søren Rasmussen, Assen Goudev, Chim C Lang, Miguel Urina-Triana, Mikko Pietilä, A Michael Lincoff, for the SELECT Trial Investigators

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Tirzepatide for Metabolic Dysfunction–Associated Steatohepatitis with Liver Fibrosis

R. Loomba, M.L. Hartman, E.J. Lawitz, R. Vuppalanchi, J. Boursier, E. Bugianesi, M. Yoneda, C. Behling, O.W. Cummings, Y. Tang, B. Brouwers, D.A. Robins, A. Nikooie, M.C. Bunck, A. Haupt, and A.J. Sanyal, for the SYNERGY-NASH Investigators*

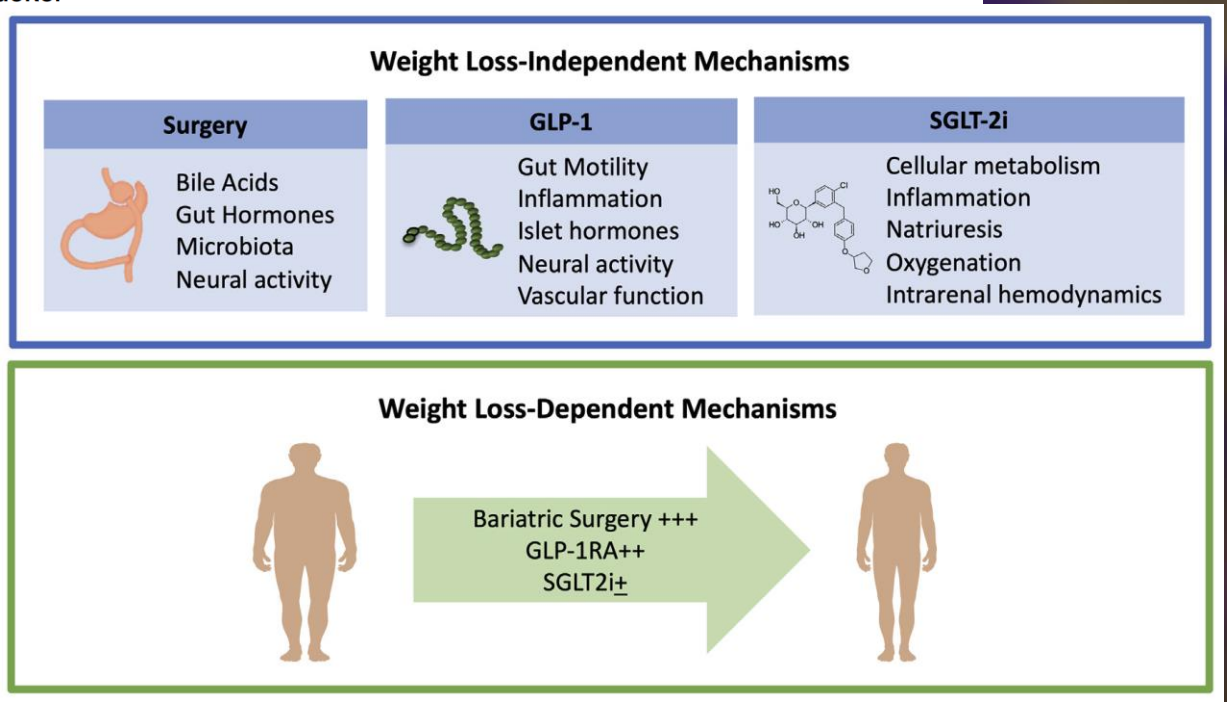
In this phase 2 trial involving participants with MASH and moderate or severe fibrosis, treatment with tirzepatide for 52 weeks was more effective than placebo with respect to resolution of MASH without worsening of fibrosis. Larger and longer

This article was published on June 8, 2024, at NEJM.org.

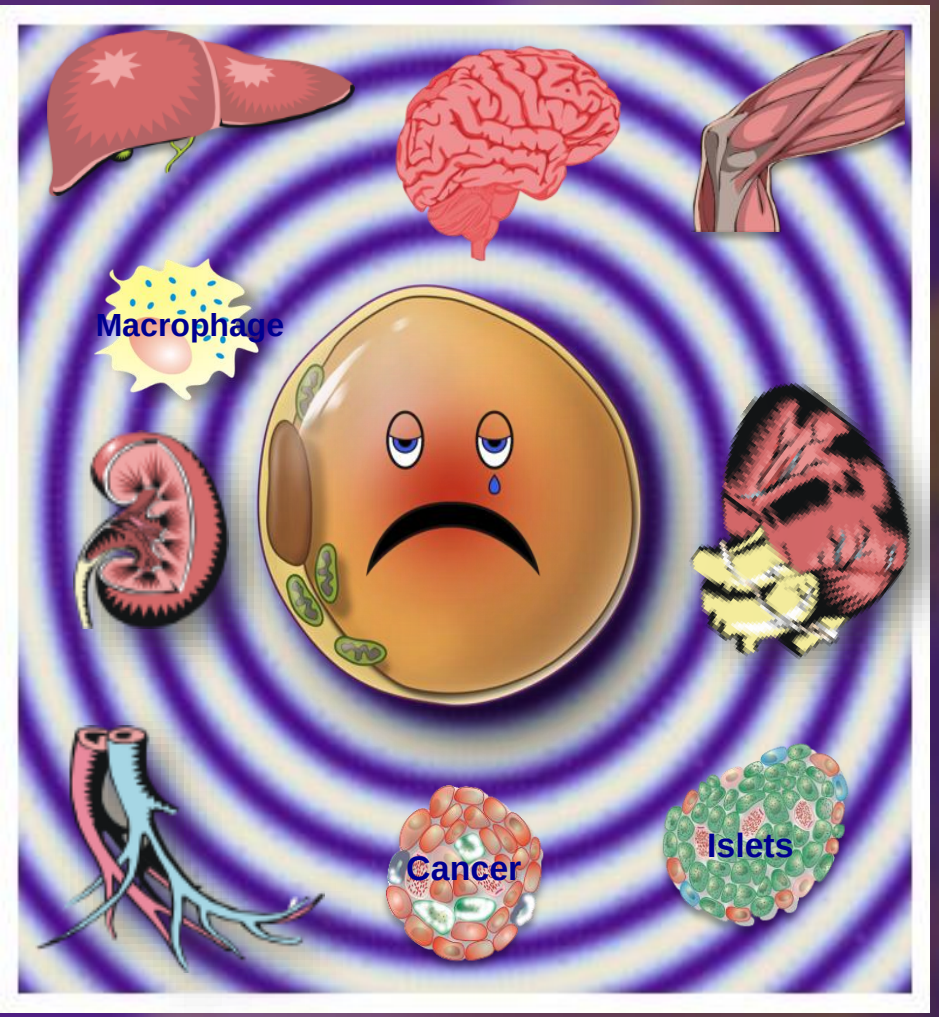
Review

Prevention of cardiorenal complications in people with type 2 diabetes and obesity

Daniel Joshua Drucker[1,*]



Cell Metabolism *36*, February 6, 2024


Macrophage
Cancer
Islets


Macrophage
Cancer
Islets

Are we done?

What EXACTLY is it about the adipocyte that is so bad for the liver?

The fibro-inflammatory milieu is a key contributor to disease and the adipocyte is one of the engines fueling that milieu

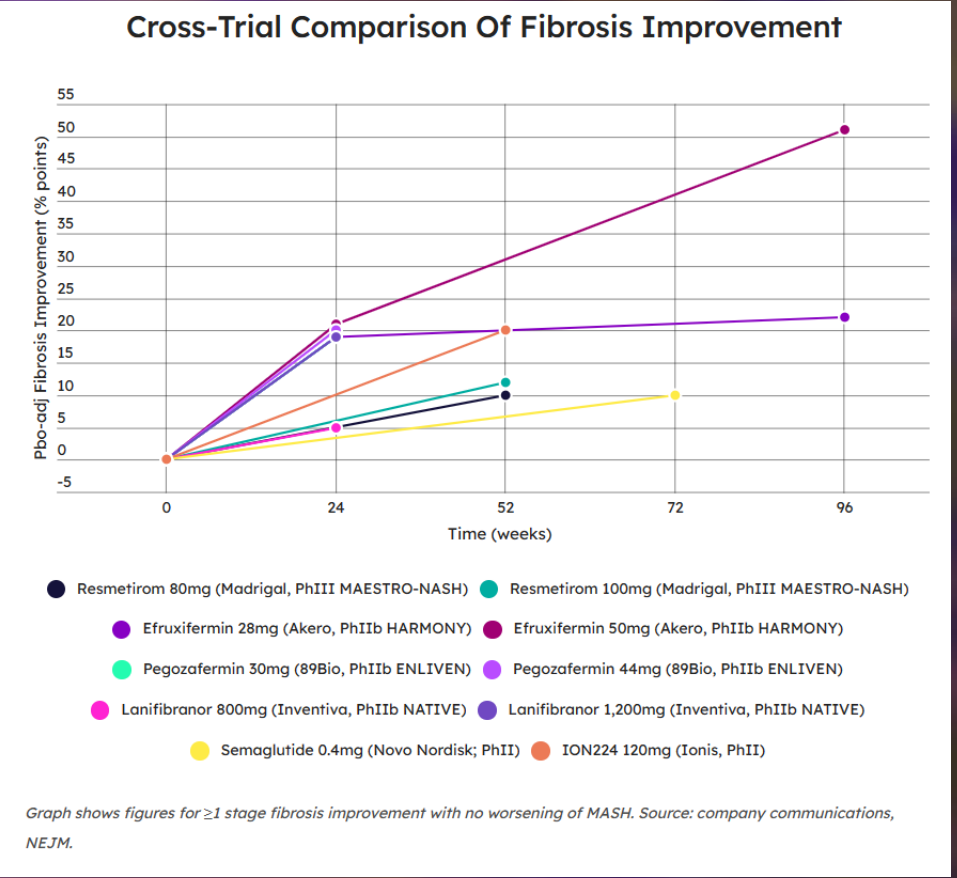
“Residual disease” post treatment with single, dual and triagonists

“Residual disease” is a term used in the context of statin exposure, when despite an overall reduction in LDL-cholesterol, there still remains an increased CVD risk

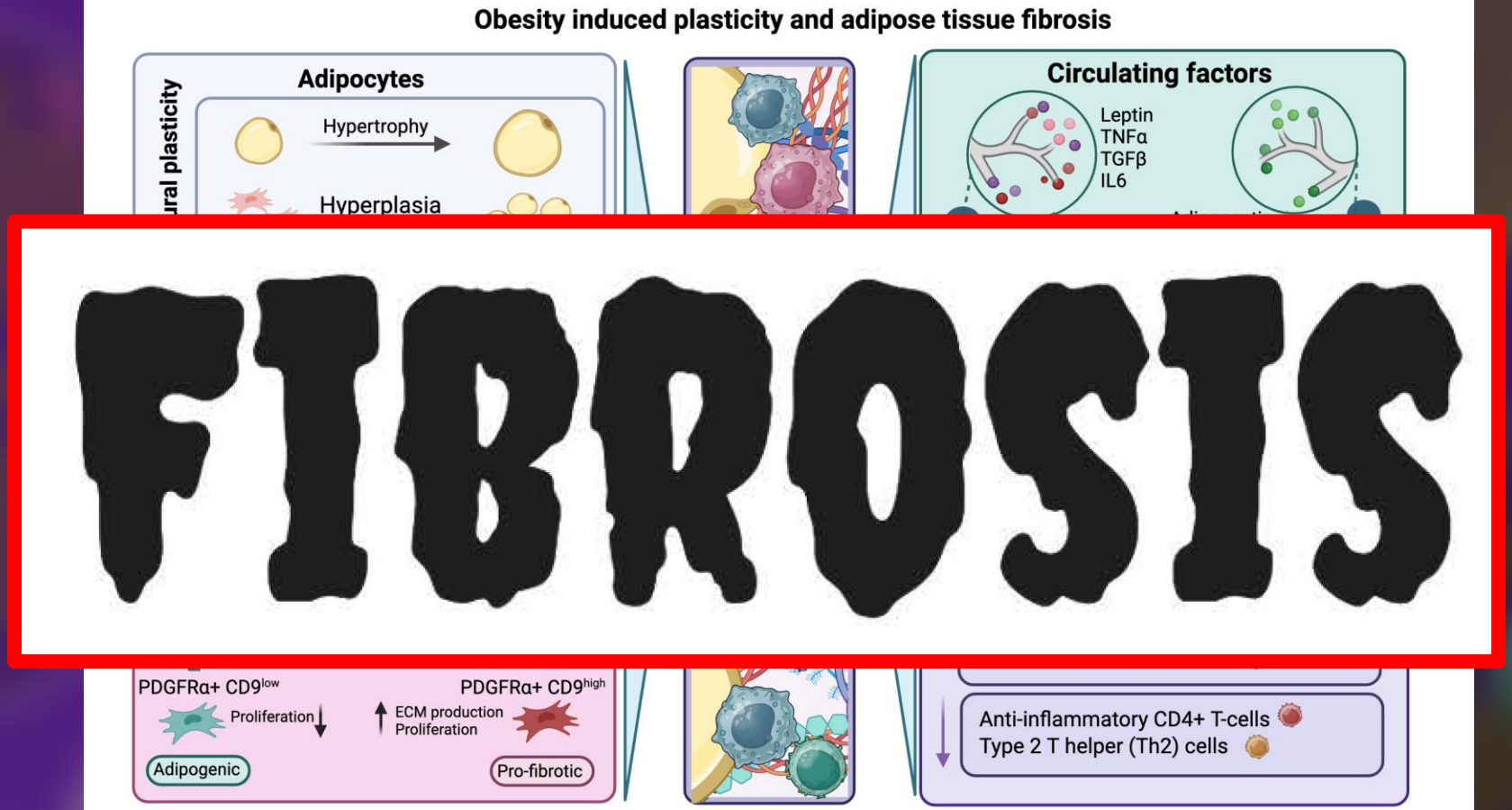
“Residual disease” post GLP1R/GIPR/GcgR agonist treatment refers to the remaining tissue fibrosis post weight loss

Prominently observed in the liver

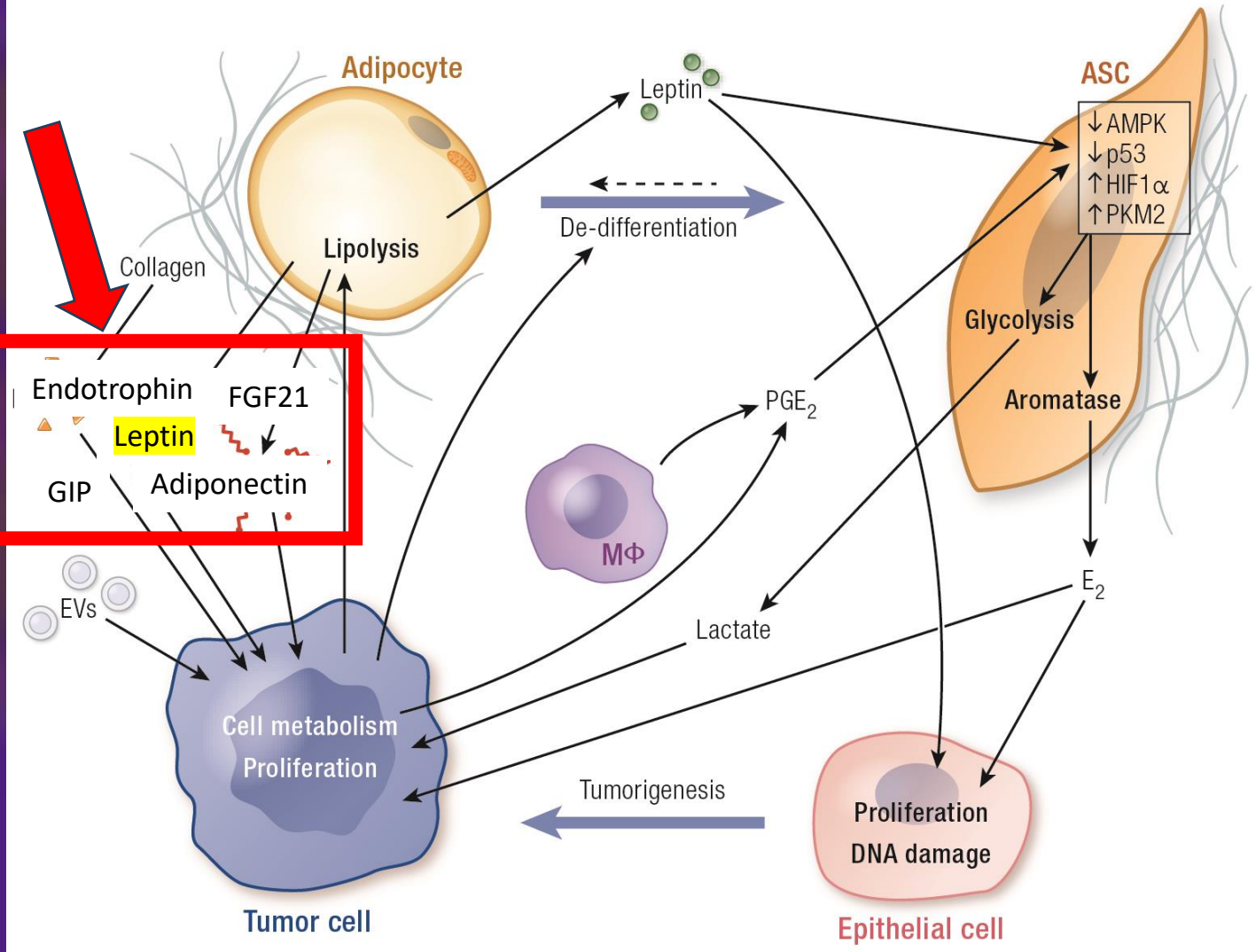
Steatosis resolution, but remaining fibrosis in the liver



Graph shows figures for ≥1 stage fibrosis improvement with no worsening of MASH. Source: company communications, NEJM.



Gliniak, Pedersen and Scherer, J Endocrinol. 2023; 259(3):e230180



Adapted from:

Kristy A. Brown
Philipp E. Scherer
Endocrine Reviews, 2023
44(6):961-974

Diabetes Volume 69, May 2020

Leptin: Less Is More

Shangang Zhao,[1] Christine M. Kusminski,[1] Joel K. Elmquist,[2] and Philipp E. Scherer[1]

Diabetes 2020;69:823–829 | https://doi.org/10.2337/dbi19-0018

The successful use of leptin for the treatment of individuals with lipodystrophy and leptin deficiency is well established. However, pharmacological approaches of leptin therapy for the treatment of diet-induced obesity have been ineffective. There is ample room for a better understanding of the much famed "leptin resistance" phenomenon. Our recent data in this area prompt us to call for a conceptual shift. This shift entails a model in which a reduction of bioactive leptin levels in the context of obesity triggers a high degree of leptin sensitization and improved leptin action, both centrally and peripherally. Put another way, hyperleptinemia per se causes leptin resistance and associated metabolic disorders. In this perspective, we briefly discuss the underlying conceptual steps that led us to explore partial leptin reduction as a viable therapeutic avenue. We hope this discussion will contribute to potential future applications of partial leptin reduction therapy for the treatment of obesity and type 2 diabetes.

Any intervention that either directly leads to a downregulation of leptin

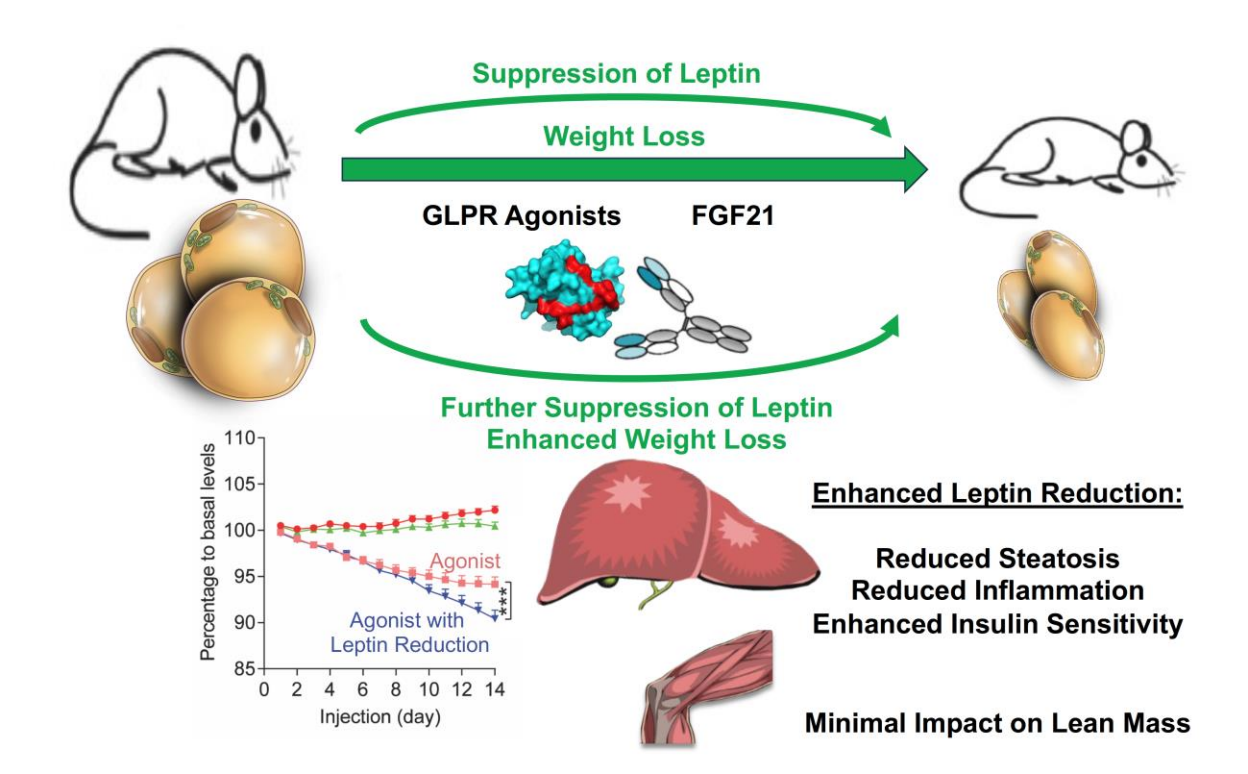
or

prevents an obesity-induced upregulation of leptin

triggers metabolic improvements

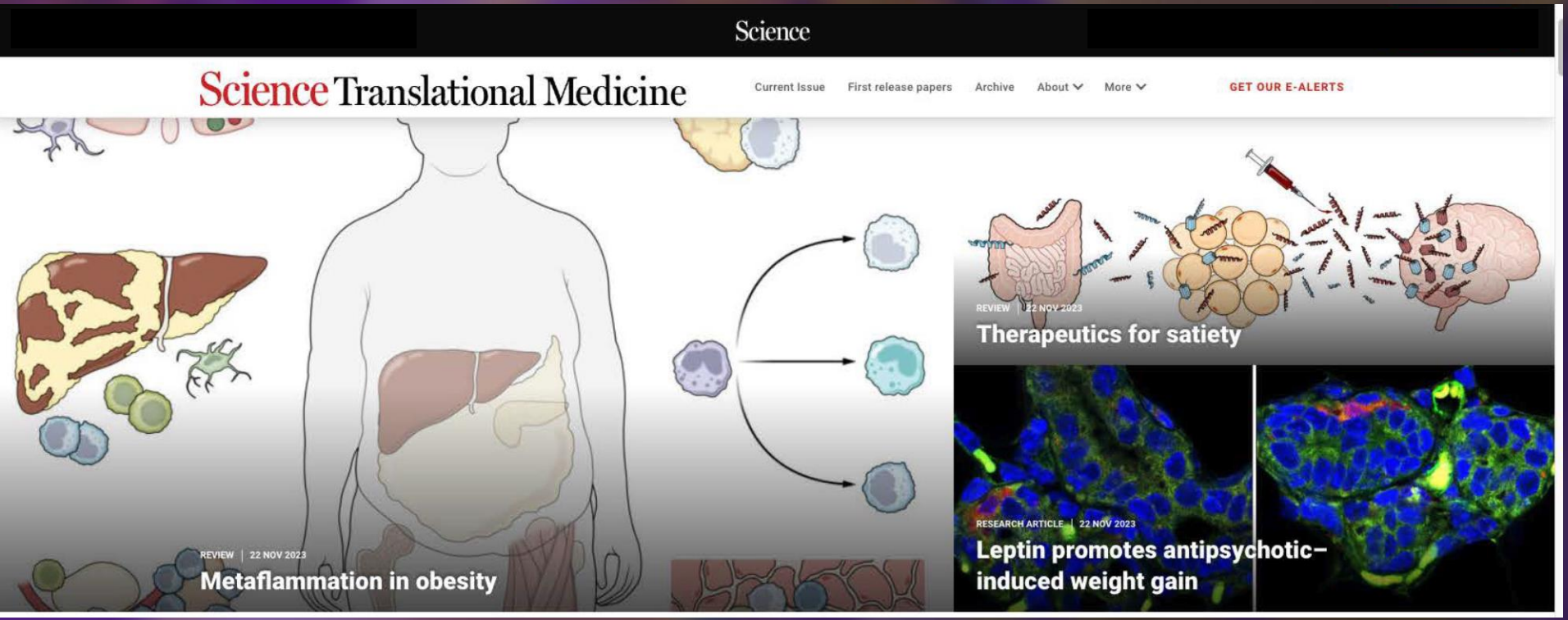
Leptin as a driver, not as a passenger to obesity!

Leptin Reduction as a Required Component for Weight Loss

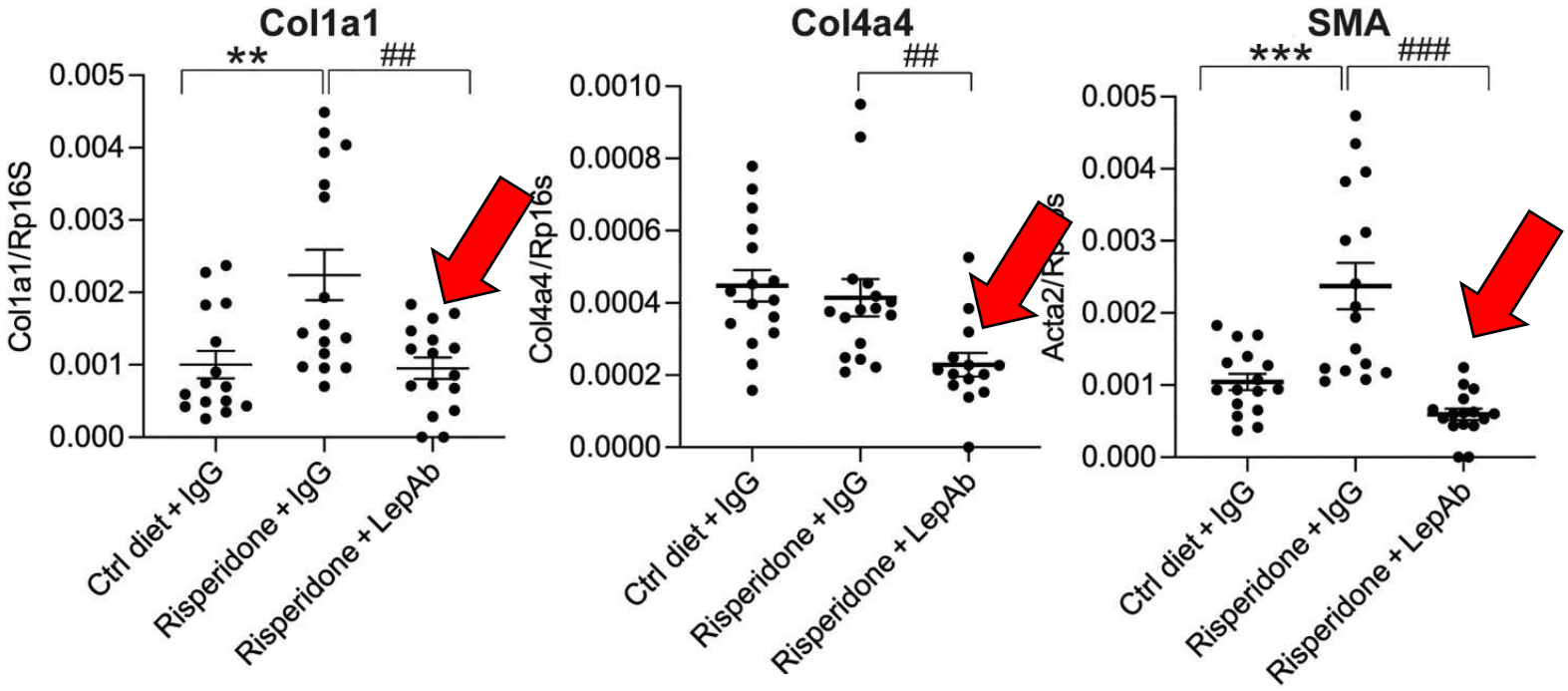


Diabetes. 2024 Feb 1;73(2):197-210

A novel MASH / MAFLD Model



Zhao *et al.*, *Sci. Transl. Med.* **15**, eade8460 (2023) 22 November 2023



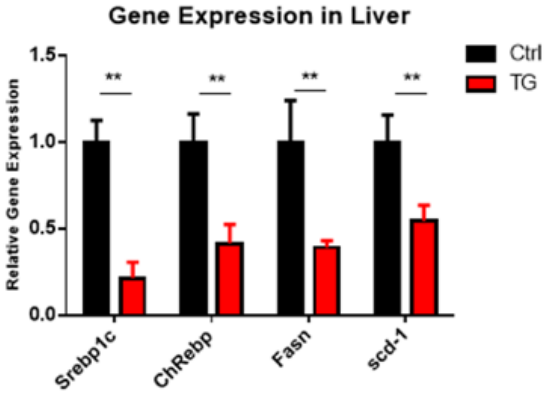
Zhao *et al.*, *Sci. Transl. Med.* **15**, eade8460 (2023) 22 November 2023

Is reducing leptin to maintain leptin sensitivity unique?

Insulin uses the same concept!

Improving insulin sensitivity reduces liver steatosis

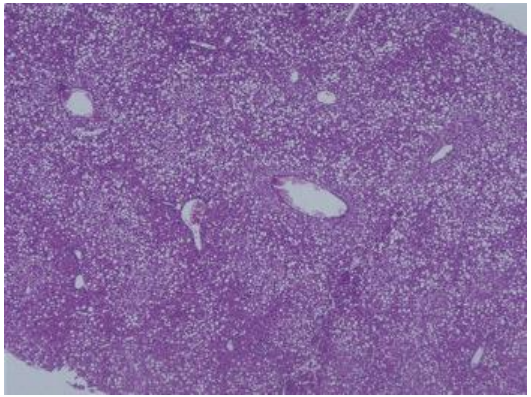
Kir6.2 Female



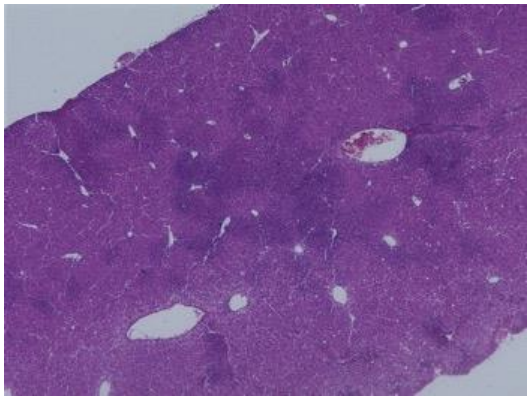
9 weeks HFD DOX 3
FEMALE

Liver Histology

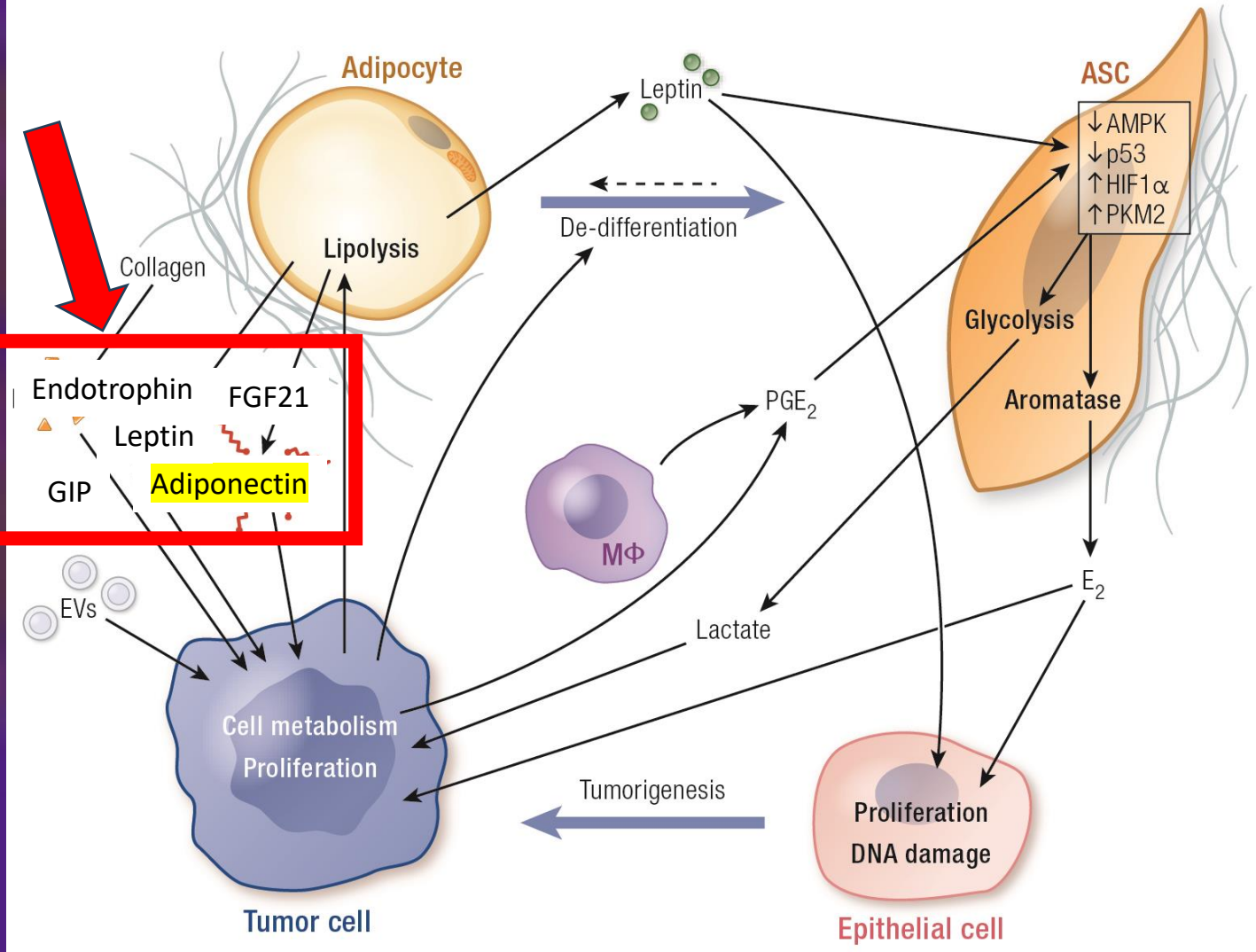
Control



TG



May Wang, unpublished



Adapted from:

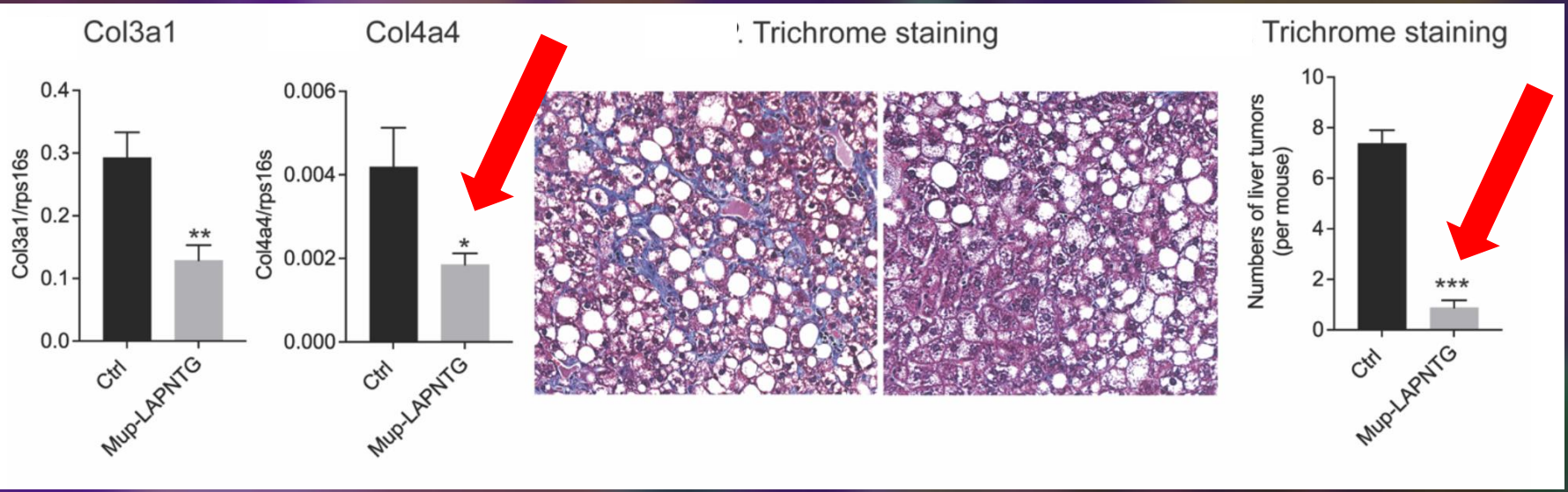
Kristy A. Brown
Philipp E. Scherer
Endocrine Reviews, 2023
44(6):961-974

The Adiponectin-PPARγ Axis in Hepatic Stellate Cells Regulates Liver Fibrosis

Shangang Zhao[1,2#], Qingzhang Zhu[1#], Jan-Bernd Funcke[1], Zhuzhen Zhang[1,3], May-Yun Wang[1], Bianca Field[1], Xuenan Sun[1], Toshiharu Onodera[1], Na Li[1,4], Yi Zhu[1,5], Christine M Kusminski[1] and Philipp E. Scherer[1*]

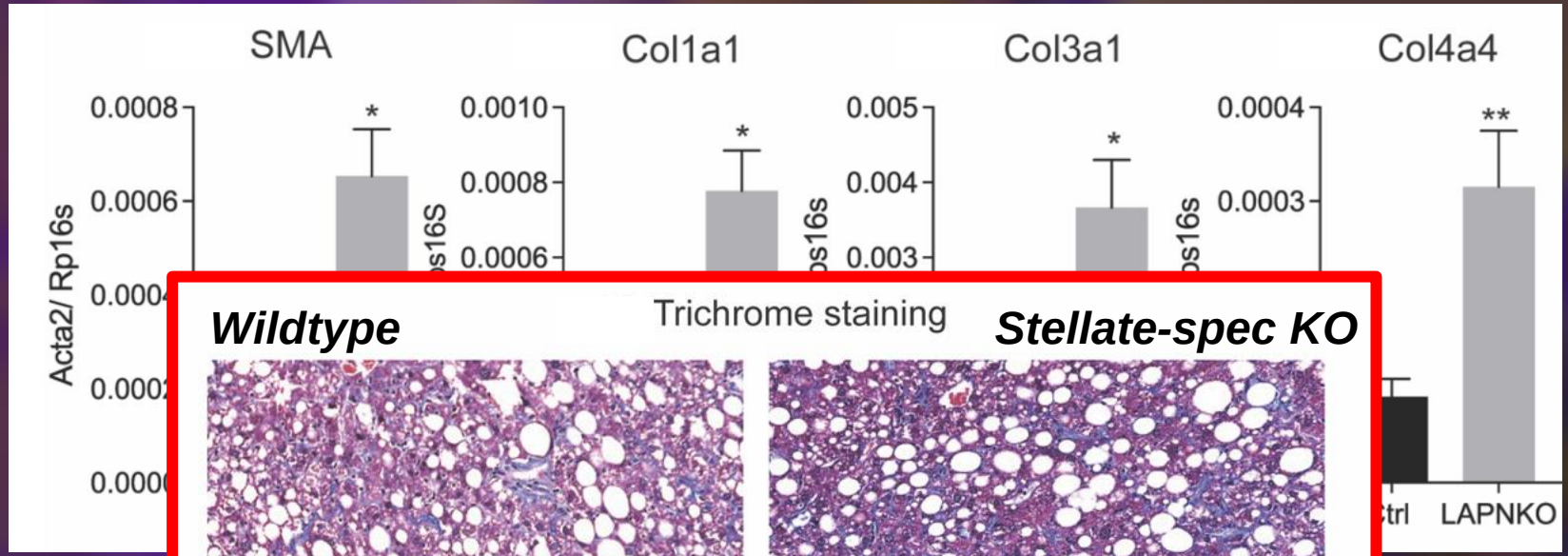
Zhao and Scherer, under revision
Merck MISP

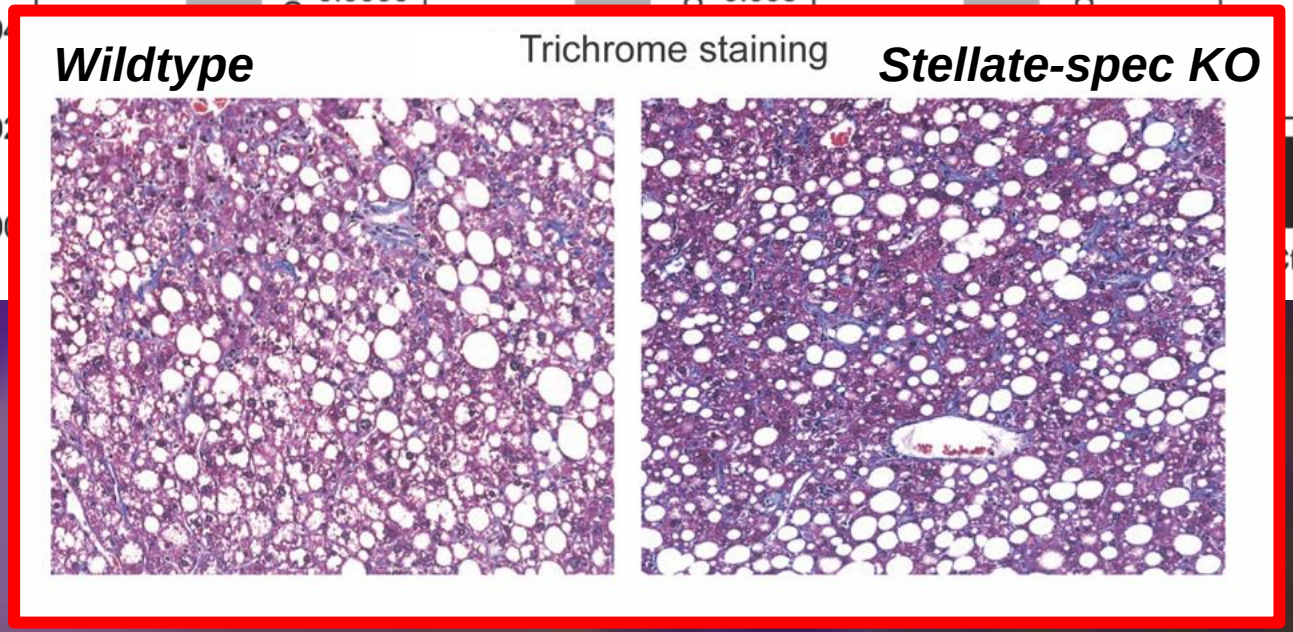
Stellate cell-specific adiponectin overexpression is potently anti-fibrotic MuP-uPA mouse



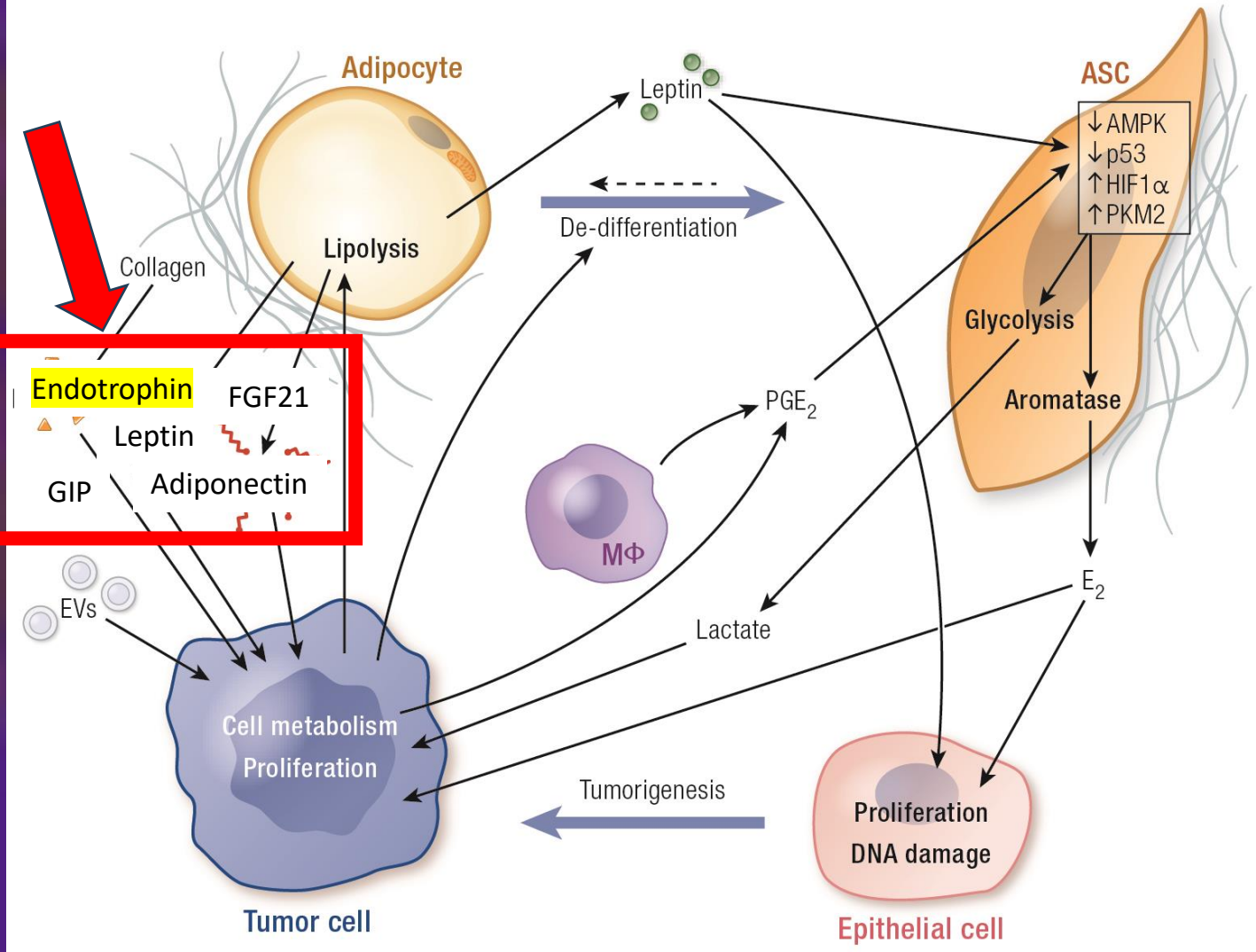
Zhao and Scherer, under revision
Merck MISP

Stellate cell-specific elimination of adiponectin is potently pro-fibrotic





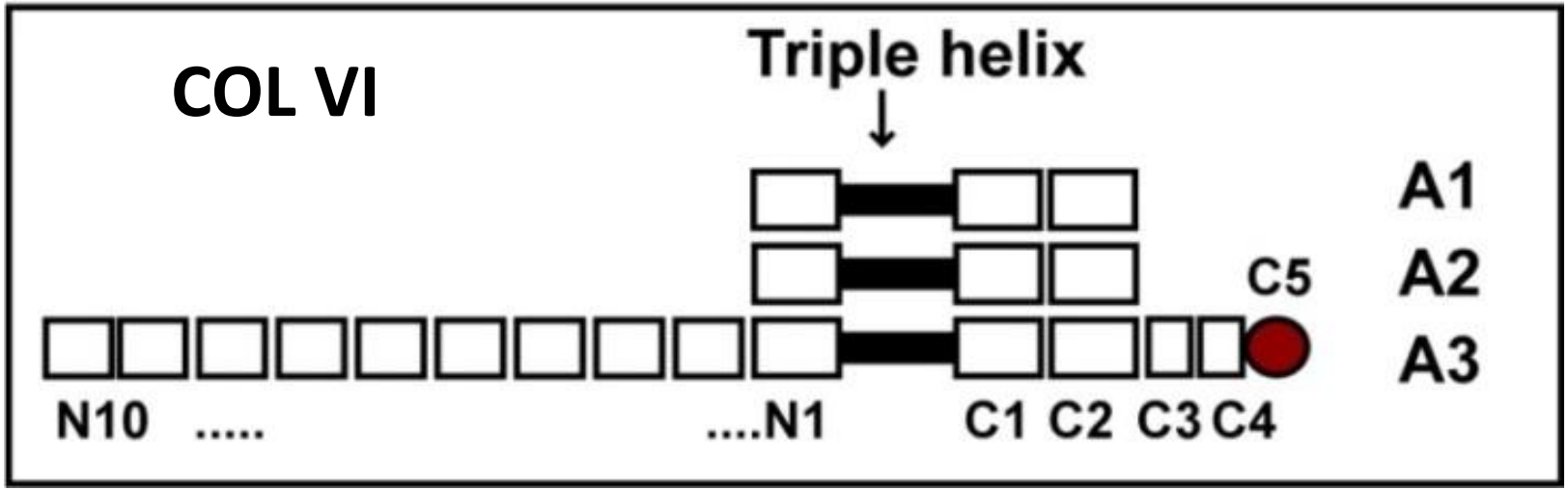
Zhao and Scherer, under revision



Adapted from:

Kristy A. Brown
Philipp E. Scherer
Endocrine Reviews, 2023
44(6):961-974

Endotrophin



- Cleavage product of COL VI: “Endotrophin”

Endotrophin is upregulated in fibrotic human breast tissue

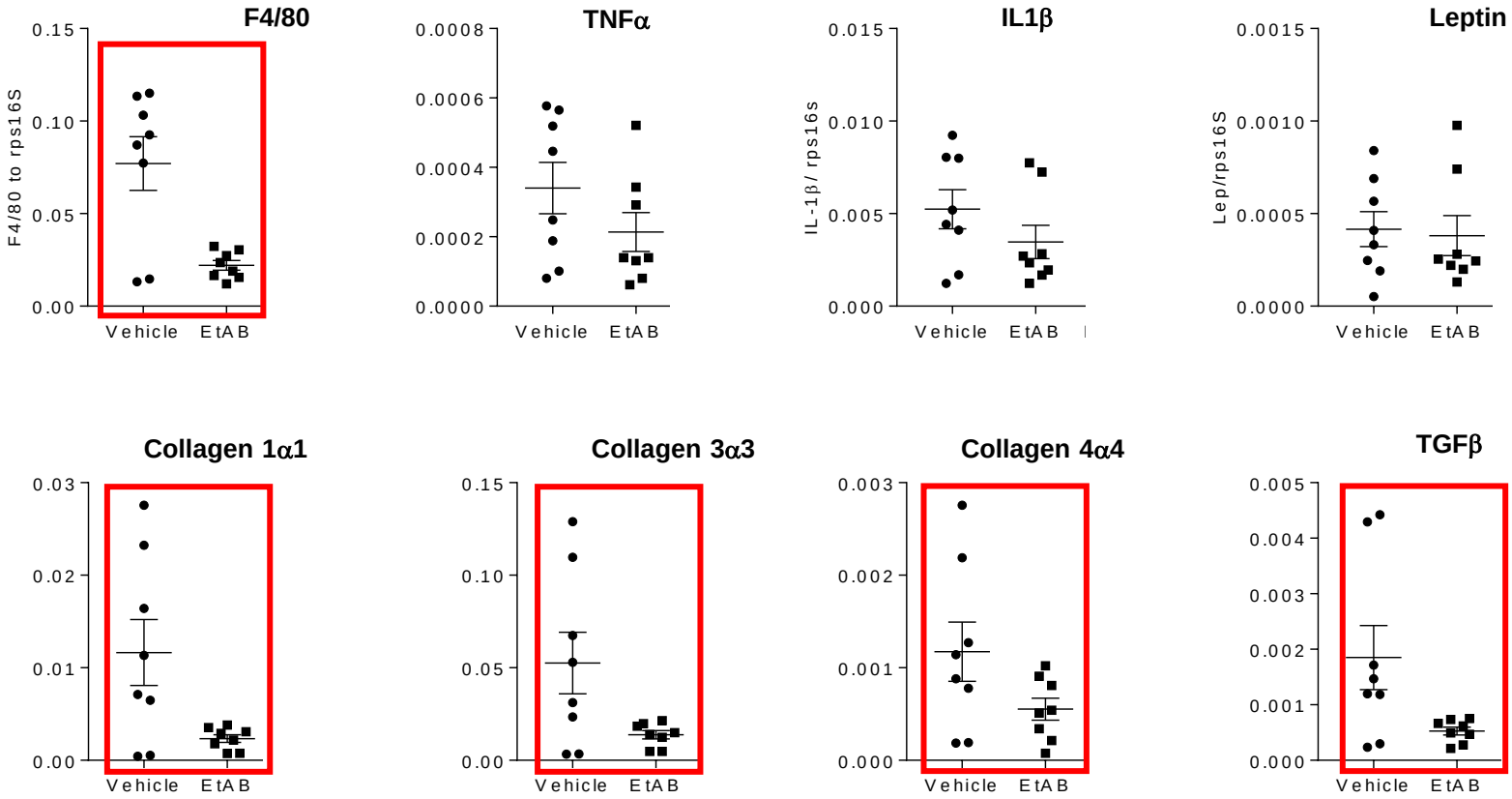
Non-Fibrotic Human Breast Tissue

Fibrotic Human Breast Tissue

Endotrophin Neutralization and Liver Fibrosis

(in the Mup-uPA mouse model)

Neutralizing Endotrophin Antibody: Gene Expression in Liver



Zhao, unpublished

SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY
https://doi.org/10.1080/00365521.2021.1879249

Taylor & Francis
Taylor & Francis Group

RESEARCH ARTICLE

OPEN ACCESS

Check for updates

Serum levels of endotrophin are associated with nonalcoholic steatohepatitis

Hannes Hagström[a,b,c], Dawei Bu[d], Patrik Nasr[e], Mattias Ekstedt[e], Hannes Hegmar[a,c], Stergios Kechagias[e], Ningyan Zhang[f], Zhiqiang An[f], Per Stål[a,c] and Philipp E. Scherer[d,g]

[a]Department of Upper GI, Division of Hepatology, Karolinska University Hospital, Stockholm, Sweden; [b]Department of Medicine, Solna, Clinical Epidemiology Unit, Karolinska Institutet, Stockholm, Sweden; [c]Department of Medicine, Huddinge, Karolinska Institutet, Stockholm, Sweden; [d]Department of Internal Medicine, Touchstone Diabetes Center, University of Texas Southwestern Medical Center, Dallas, TX, USA; [e]Department of Gastroenterology and Hepatology, Department of Health, Medicine and Caring Sciences, Linköping University, Linköping, Sweden; [f]Texas Therapeutics Institute, Brown Foundation Institute of Molecular Medicine, University of Texas Health Science Center at Houston, Houston, TX, USA; [g]Department of Cell Biology, University of Texas Southwestern Medical Center, Dallas, TX, USA

iScience

CellPress
OPEN ACCESS

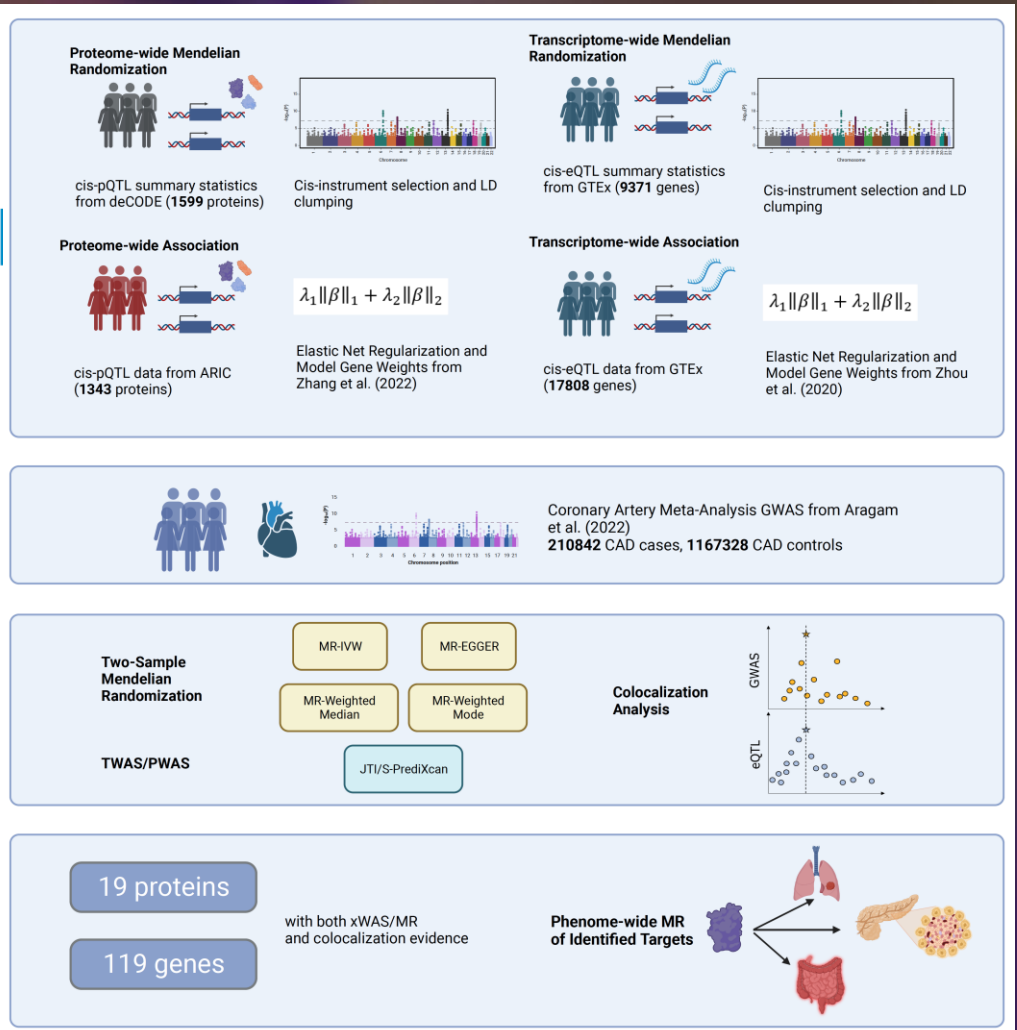
Article

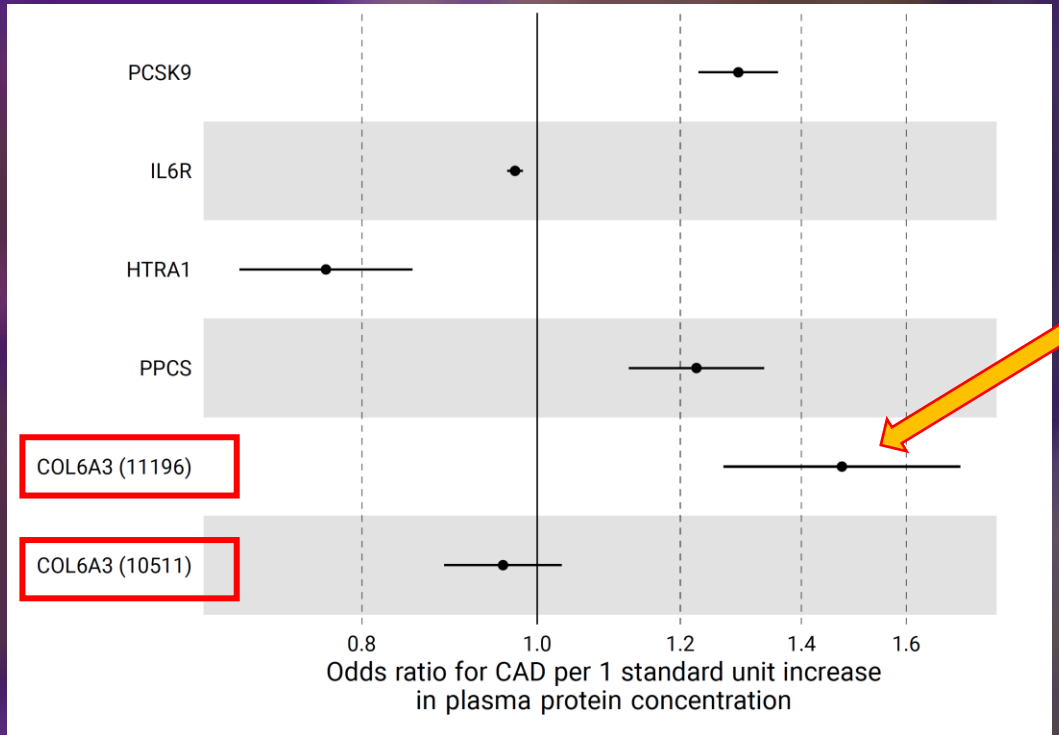
Instrumental variable and colocalization analyses identify endotrophin and HTRA1 as potential therapeutic targets for coronary artery disease

Paul C. Lee,[1] In-Hyuk Jung,[1] Shreeya Thussu,[1] Ved Patel,[1] Ryan Wagoner,[1] Kendall H. Burks,[1] Junedh Amrute,[1] Jared S. Elenbaas,[1] Chul Joo Kang,[2] Erica P. Young,[1,2] Philipp E. Scherer,[3] and Nathan O. Stitziel[1,2,4,5,*]

Lee et al., iScience *27*, 110104
July 19, 2024

https://doi.org/10.1016/j.isci.2024.110104





Endotrophin

with CAD risk by IV analyses and demonstrated evidence of genetic colocalization. Notably, our analyses validated well-established targets such as PCSK9 and ANGPTL4 while also identifying HTRA1 and endotrophin (a cleavage product of COL6A3) as proteins whose levels are causally associated with CAD risk. Further experimental studies are needed to confirm the causal role of

Lee et al. 2024 iScience.; 27(7):110104

EBioMedicine 68 (2021) 103391

Contents lists available at ScienceDirect

EBioMedicine

journal homepage: www.elsevier.com/locate/ebiom

Research paper

Endotrophin is associated with chronic multimorbidity and all-cause mortality in a cohort of elderly women

Line Mærsk Staunstrup[a,b,*], Cecilie
Søren Brunak[d], Claus Christiansen[b]

EBioMedicine 69 (2021) 103447

Contents lists available at ScienceDirect

EBioMedicine

journal homepage: www.elsevier.com/locate/ebiom

Endotrophin: Nominated for best supporting actor in the fibro-inflammatory saga

Philipp E. Scherer[a,b], Olga T. Gupta[a,c,*]

[a] Touchstone Diabetes Center, Department of Internal Medicine, the University of Texas Southwestern Medical Center, Dallas, TX, USA
[b] Department of Cell Biology, University of Texas Southwestern Medical Center, Dallas, TX, USA
[c] Department of Pediatrics, University of Texas Southwestern Medical Center, Dallas, TX, USA

Matrix Biology 132 (2024) 1–9

Contents lists available at ScienceDirect

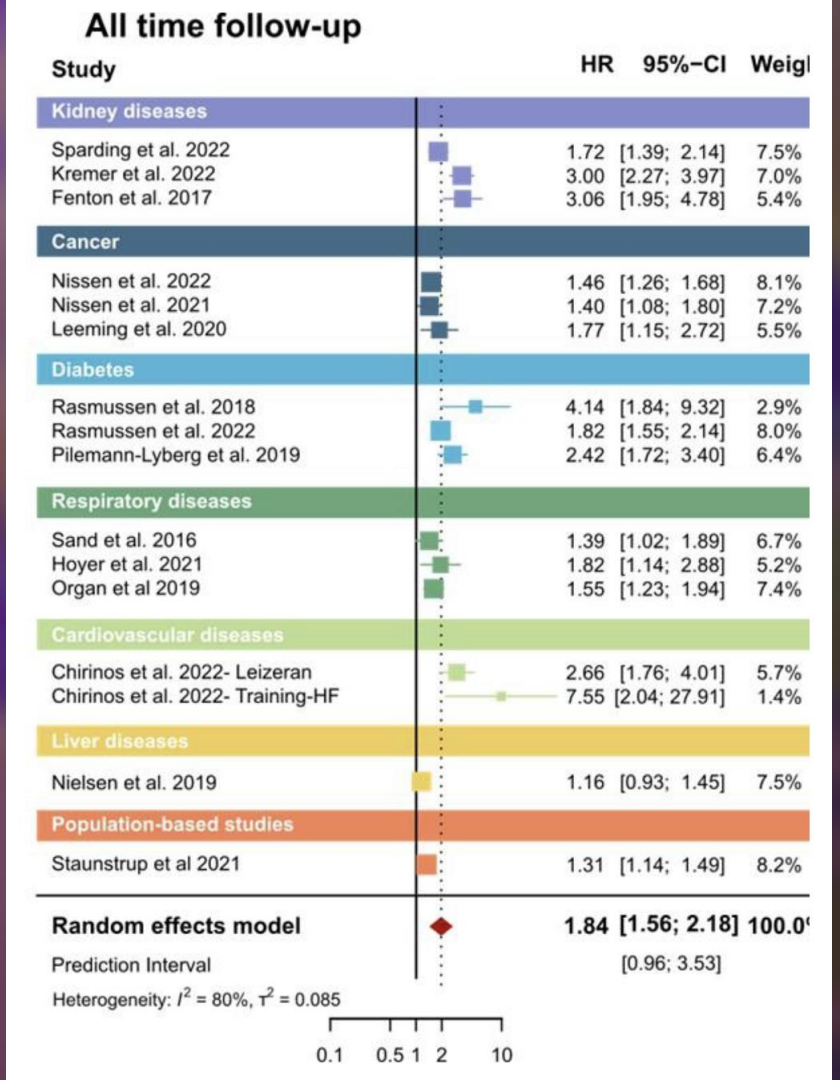
Matrix Biology

journal homepage: www.elsevier.com/locate/matbio

Check for updates

The fibroblast hormone Endotrophin is a biomarker of mortality in chronic diseases

Federica Genovese [a,1,*], Cecilie Bager [a,1], Peder Frederiksen [a], Dario Vazquez [a], Jannie Marie Bülow Sand [a], R Gisli Jenkins [b], Toby M. Maher [c], Iain D. Stewart [b], Philip L. Molyneaux [b], William A Fahy [d], Louise V. Wain [e,f], Jørgen Vestbo [g], Carmel Nanthakumar [h], Saher Burhan Shaker [i], Nils Hoyer [i], Diana Julie Leeming [a], Jacob George [j], Jonel Trebicka [k], Daniel Guldager Kring Rasmussen [a], Michael K. Hansen [l], Paul Cockwell [m], Daan Kremer [n], Stephan JL Bakker [n], Nicholas M Selby [o], Alexander Lynge Reese-Petersen [a], Arantxa González [p,q], Julio Núñez [r], Peter Rossing [s,t], Neel I. Nissen [a], Mogens Karsbøl Boisen [u], Inna M. Chen [u], Lei Zhao [v], Morten A. Karsdal [a,1], Detlef Schuppan [w,1]


All time follow-up
Study
HR
95%−CI
Weigl
Kidney diseases
Sparding et al. 2022 1.72 [1.39; 2.14] 7.5%
Kremer et al. 2022 3.00 [2.27; 3.97] 7.0%
Fenton et al. 2017 3.06 [1.95; 4.78] 5.4%
Cancer
Nissen et al. 2022 1.46 [1.26; 1.68] 8.1%
Nissen et al. 2021 1.40 [1.08; 1.80] 7.2%
Leeming et al. 2020 1.77 [1.15; 2.72] 5.5%
Diabetes
Rasmussen et al. 2018 4.14 [1.84; 9.32] 2.9%
Rasmussen et al. 2022 1.82 [1.55; 2.14] 8.0%
Pilemann-Lyberg et al. 2019 2.42 [1.72; 3.40] 6.4%
Respiratory diseases
Sand et al. 2016 1.39 [1.02; 1.89] 6.7%
Hoyer et al. 2021 1.82 [1.14; 2.88] 5.2%
Organ et al 2019 1.55 [1.23; 1.94] 7.4%
Cardiovascular diseases
Chirinos et al. 2022- Leizeran 2.66 [1.76; 4.01] 5.7%
Chirinos et al. 2022- Training-HF 7.55 [2.04; 27.91] 1.4%
Liver diseases
Nielsen et al. 2019 1.16 [0.93; 1.45] 7.5%
Population-based studies
Staunstrup et al 2021 1.31 [1.14; 1.49] 8.2%
Random effects model 1.84 [1.56; 2.18] 100.0
Prediction Interval [0.96; 3.53]
Heterogeneity: I^2 = 80%, τ^2 = 0.085
0.1
0.5
1
2
10

Endocrine Reviews, 2023, **00**, 1–18
https://doi.org/10.1210/endrev/bnad036
Advance access publication 13 December 2023
Review

Endotrophin, a Key Marker and Driver for Fibroinflammatory Disease

Kim Henriksen,[1] Federica Genovese,[1] Alexander Reese-Petersen,[1] Laurent P. Audoly,[2] Kai Sun,[3] Morten A. Karsdal,[1] and Philipp E. Scherer[4]

[1]Department of Cardiovascular Disease, Nordic Bioscience A/S, DK-2730 Herlev, Denmark
[2]Privebio Inc., Boston, MA 02445, USA
[3]Center for Metabolic and Degenerative Diseases, Institute of Molecular Medicine, University of Texas Health Science Center at Houston, Houston, TX 77030, USA
[4]Touchstone Diabetes Center, The University of Texas Southwestern Medical Center, Dallas, TX 75390, USA

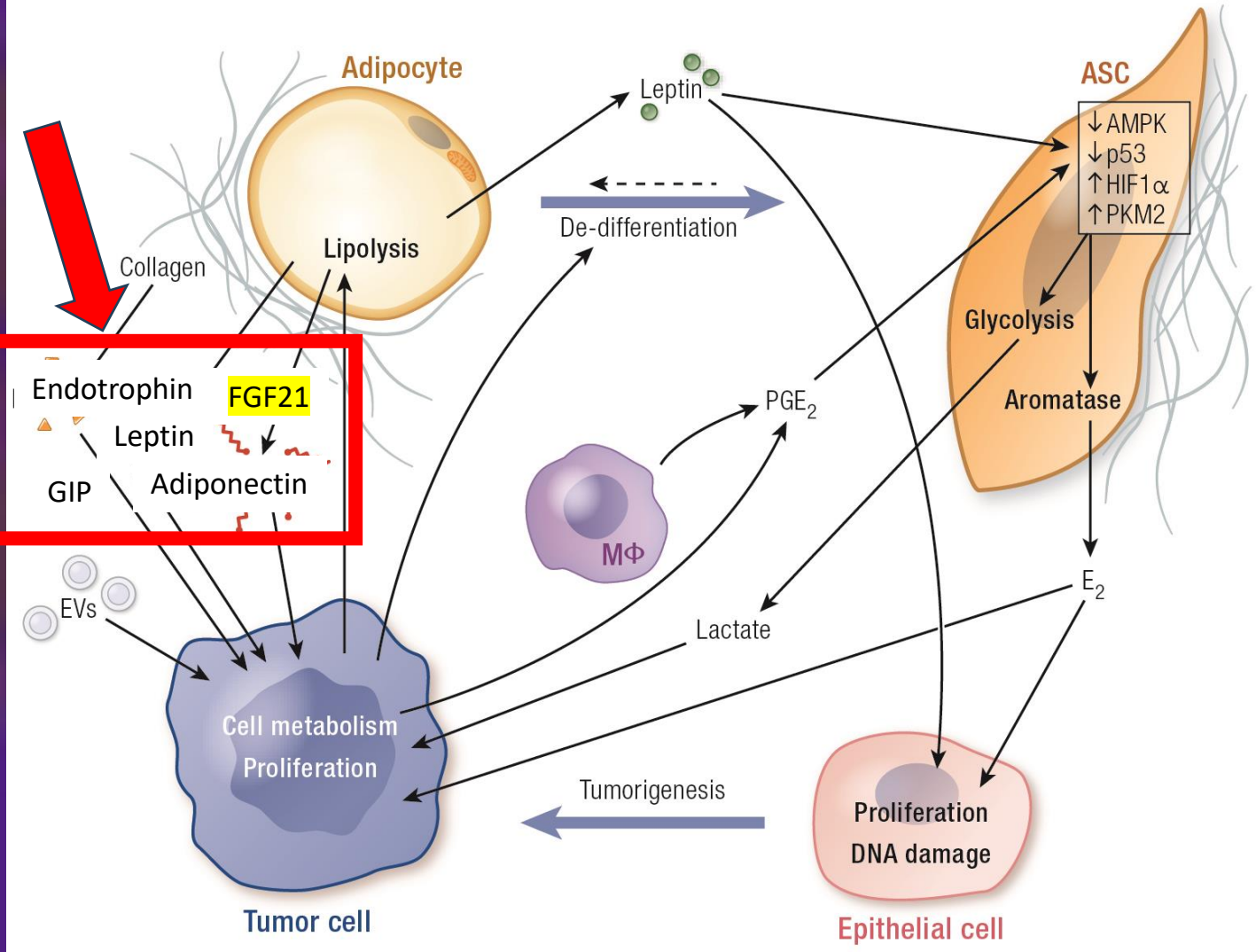
Summary Endotrophin

- **Biomarker:** Endotrophin is an excellent fibro-inflammatory marker in plasma

- **Cancer:** Pro-mitogenic and inducing chemoresistance
  Breast cancer; HCC, colon cancer, ovarian cancer

- **Fibrosis:** Endotrophin is a direct driver of fibrosis in the liver, heart, kidney and adipose tissue

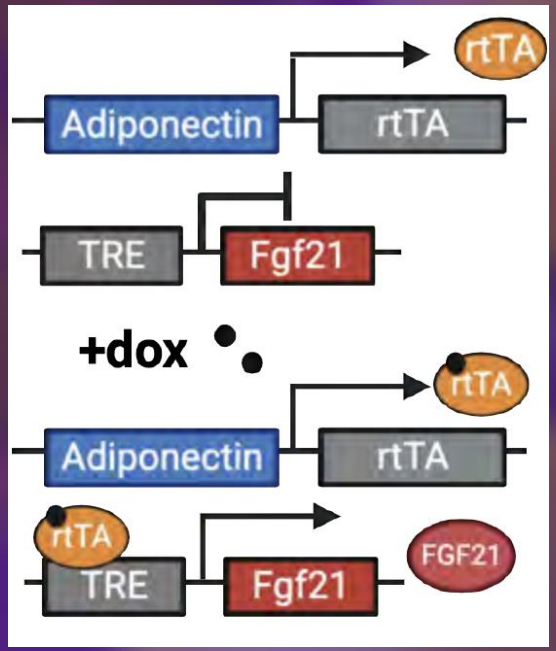
- **Endotrophin neutralization** as a direct intervention in cancer and fibrosis

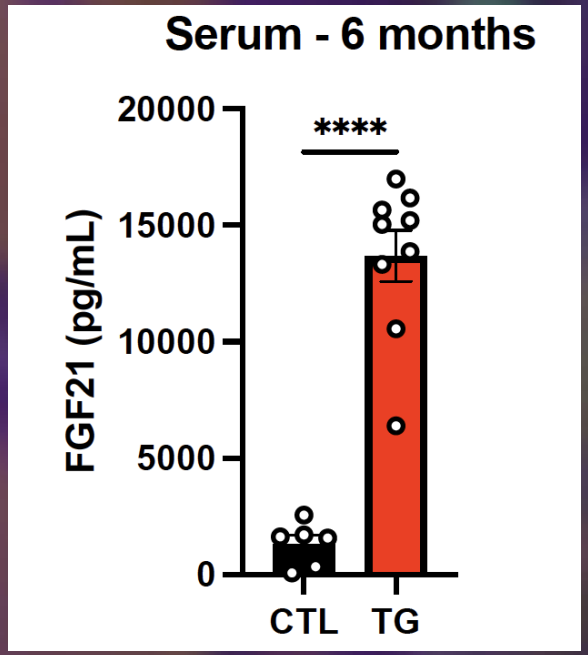


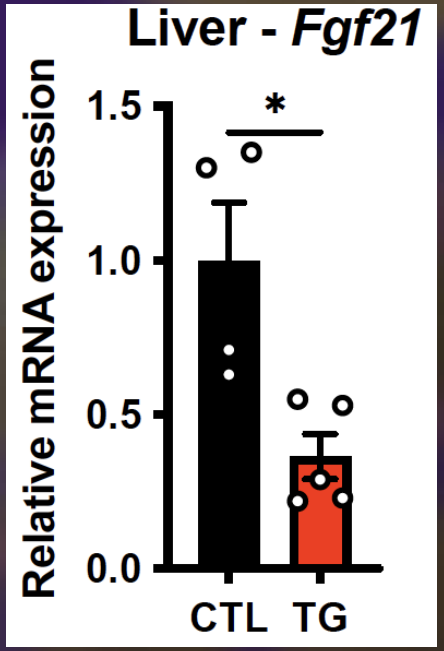
Adapted from:

Kristy A. Brown
Philipp E. Scherer
Endocrine Reviews, 2023
44(6):961-974

Adipocyte-specific FGF21 overexpression in the adult stage

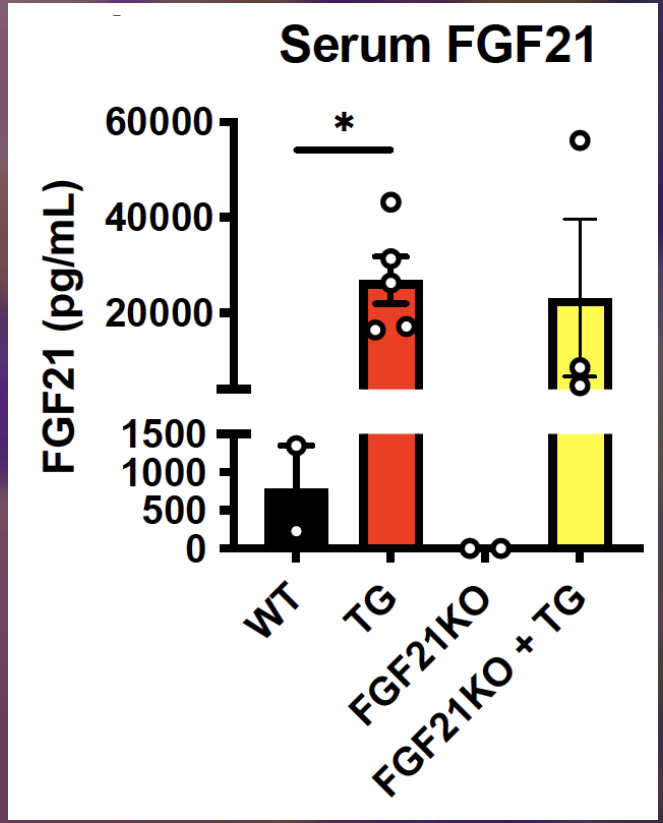






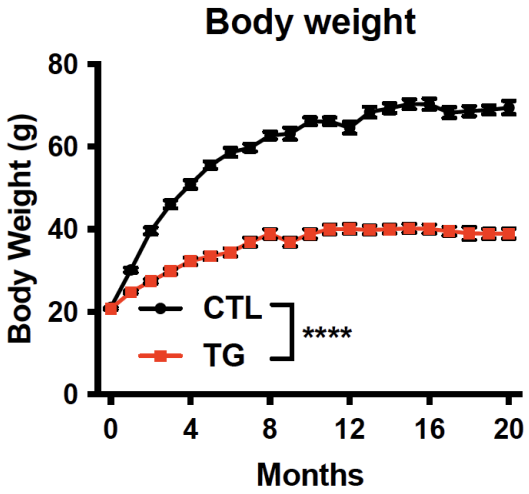
Christy Gliniak, unpublished

Adipocyte-derived FGF21 reaches the circulation



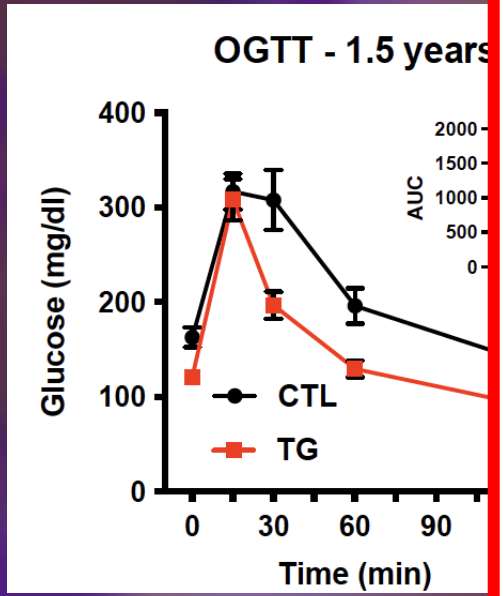
Christy Gliniak, unpublished

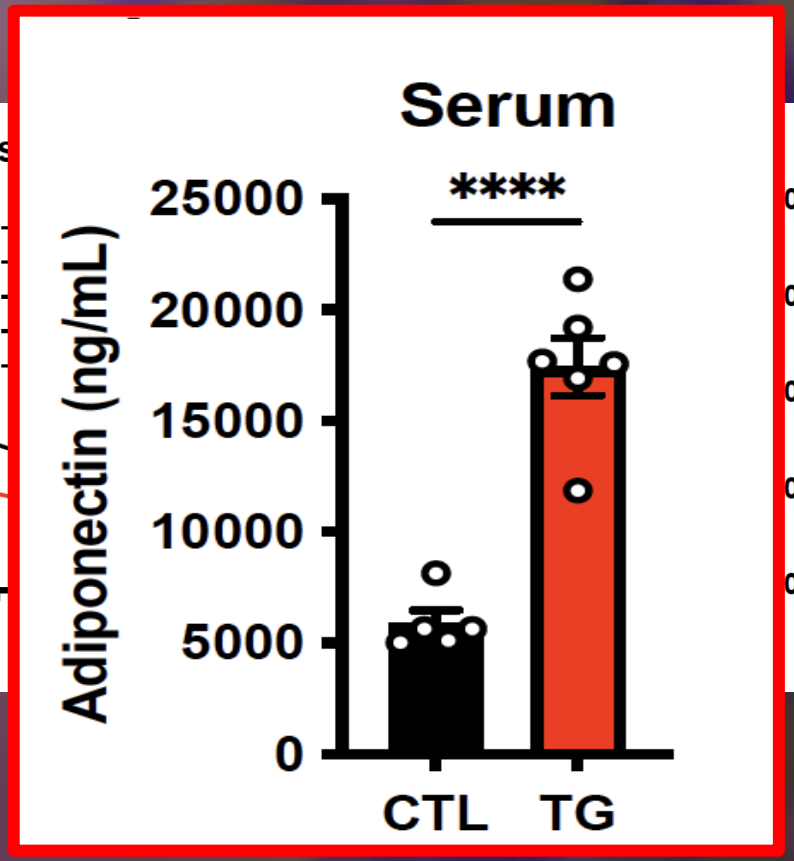
Adipocyte-derived FGF21 prompts reduced weight gain

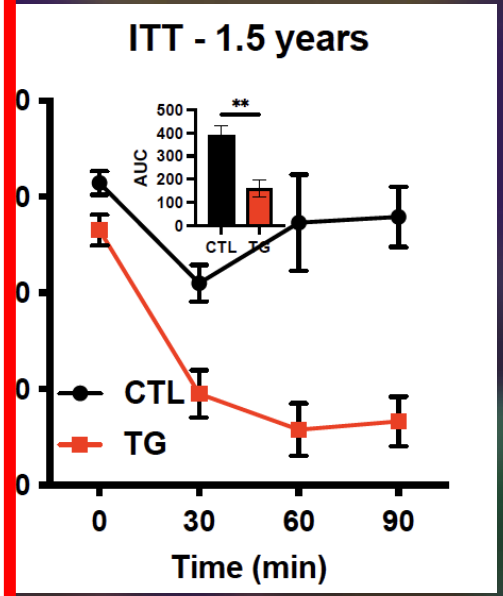


Christy Gliniak, unpublished

Adipocyte-derived FGF21 prompts improved insulin sensitivity

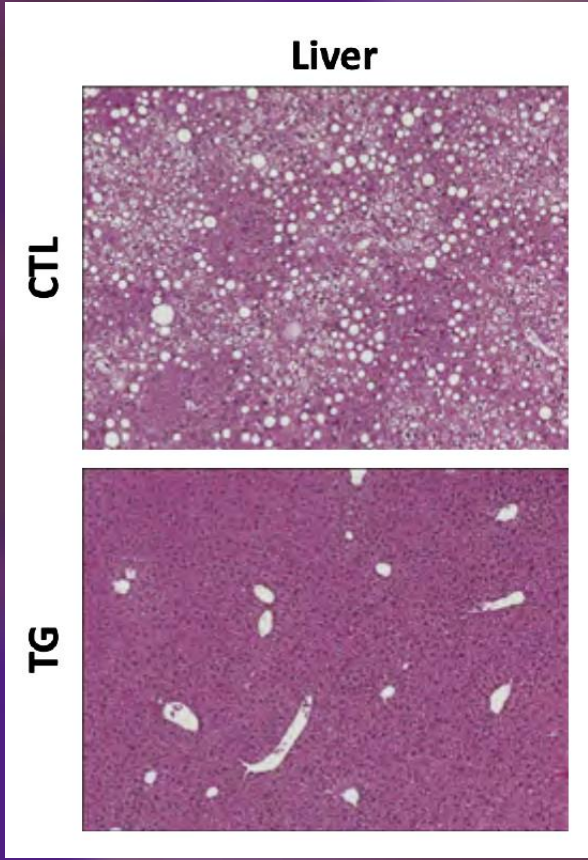


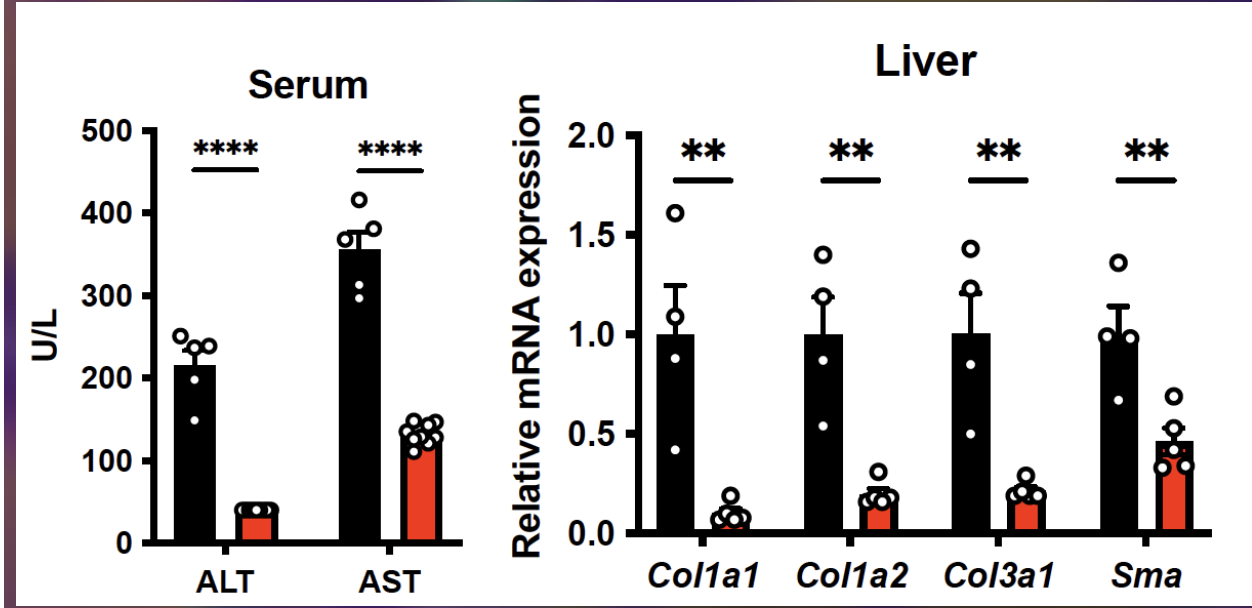




Christy Gliniak, unpublished

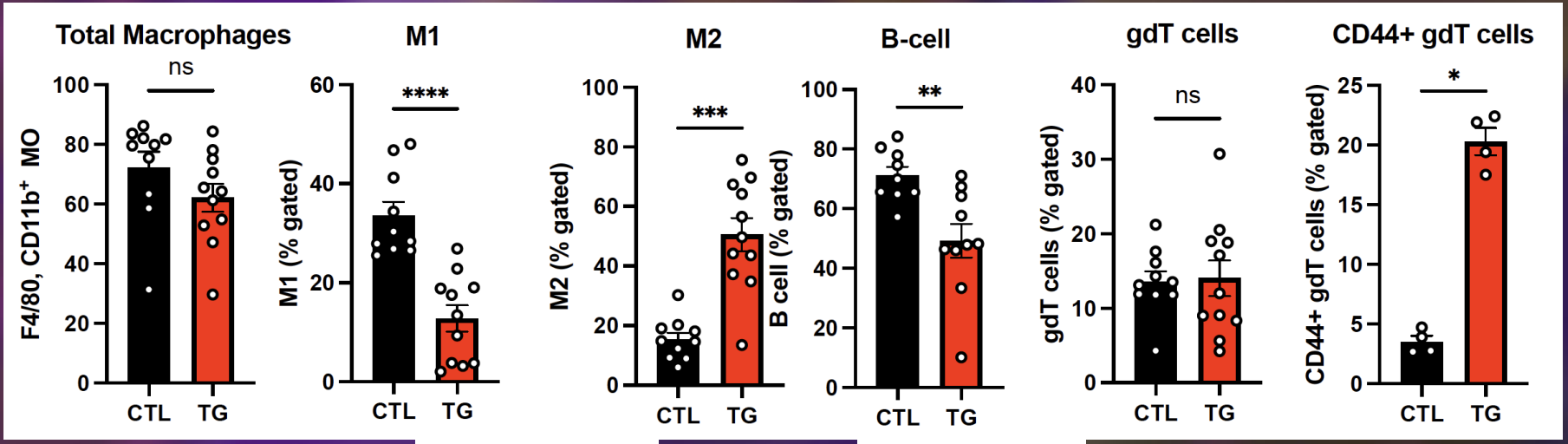
Adipocyte-derived FGF21 improves liver function

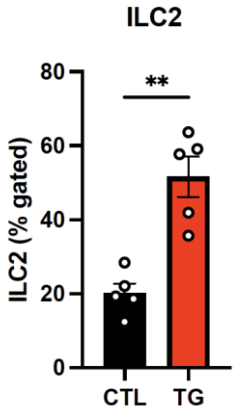


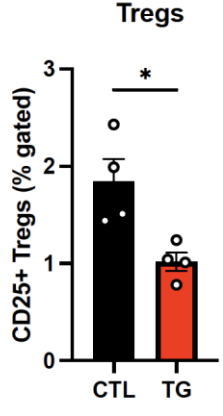


Christy Gliniak, unpublished

Adipocyte-derived FGF21 improves immunometabolism

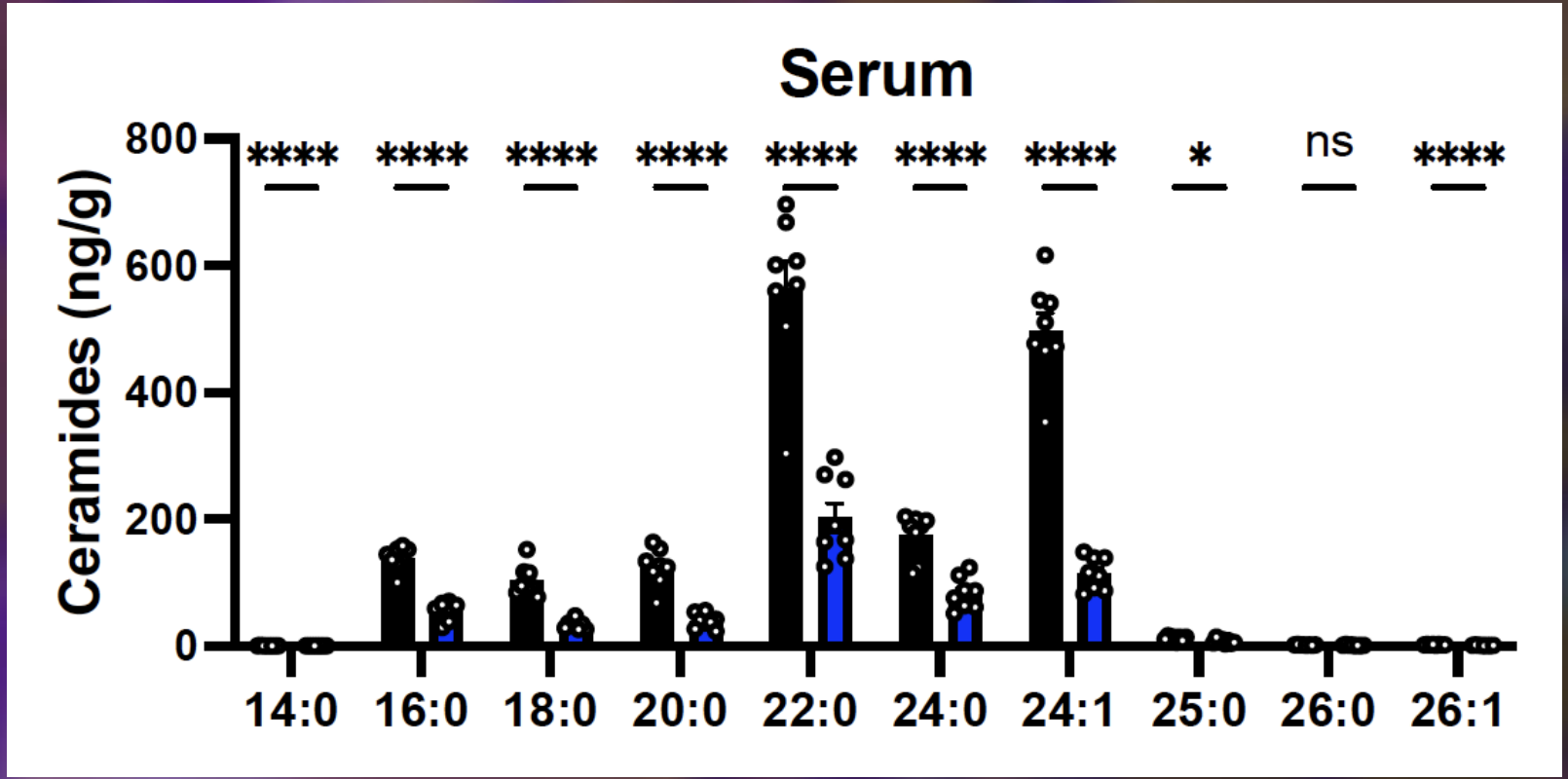




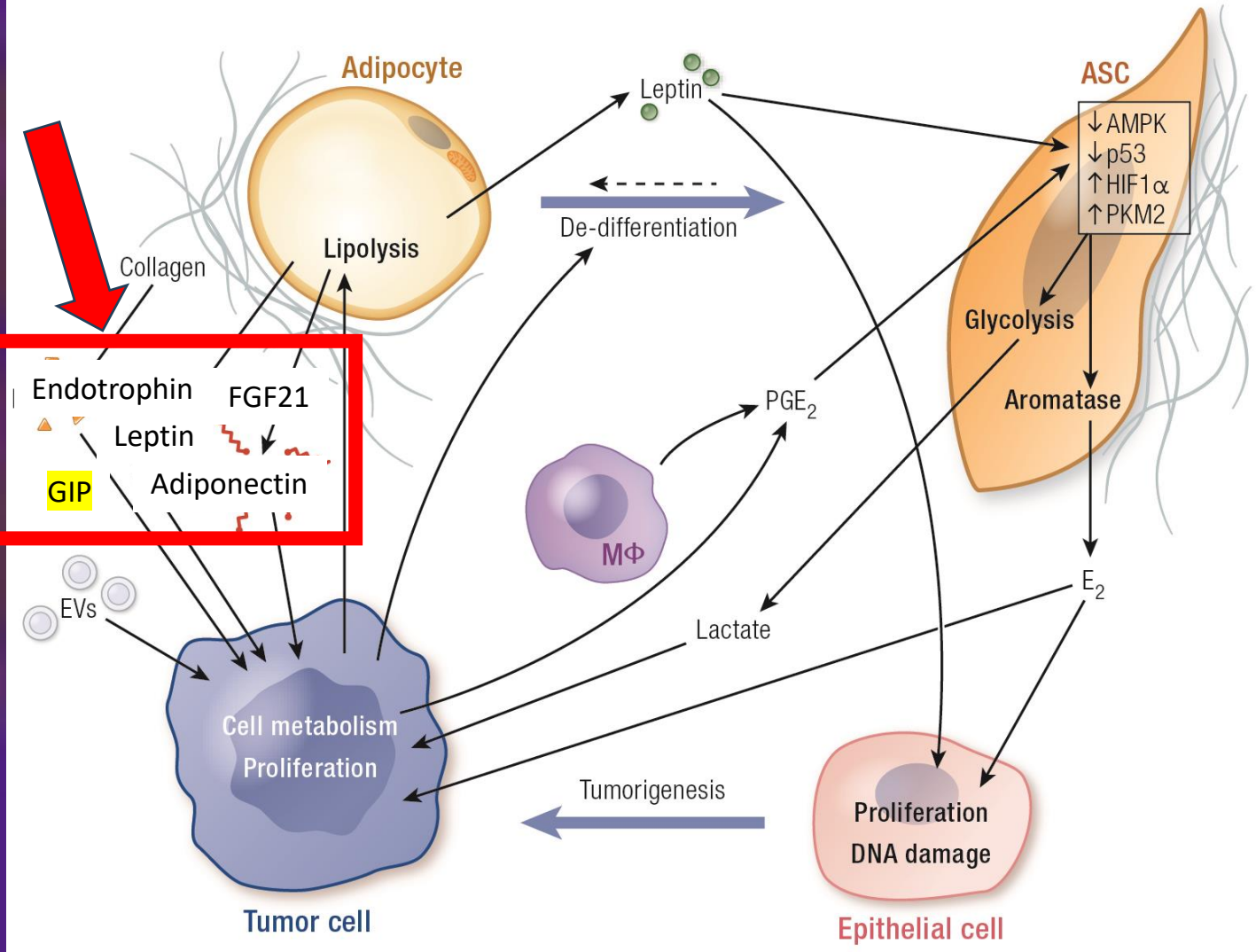


Christy Gliniak, unpublished

Adipocyte-derived FGF21 lowers ceramides



Christy Gliniak, unpublished



Adapted from:

Kristy A. Brown
Philipp E. Scherer
Endocrine Reviews, 2023
44(6):961-974

Cell Metabolism

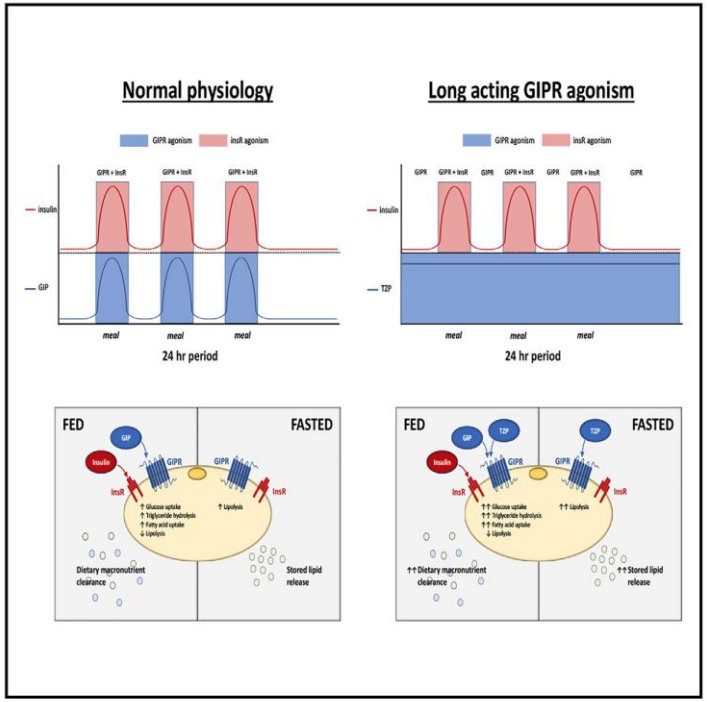
Article

Tirzepatide modulates the regulation of adipocyte nutrient metabolism through long-acting activation of the GIP receptor

Authors

Ajit Regmi, Eitaro Aihara, Michael E. Christe, ..., Tamer Coskun, Melissa K. Thomas, William Roell

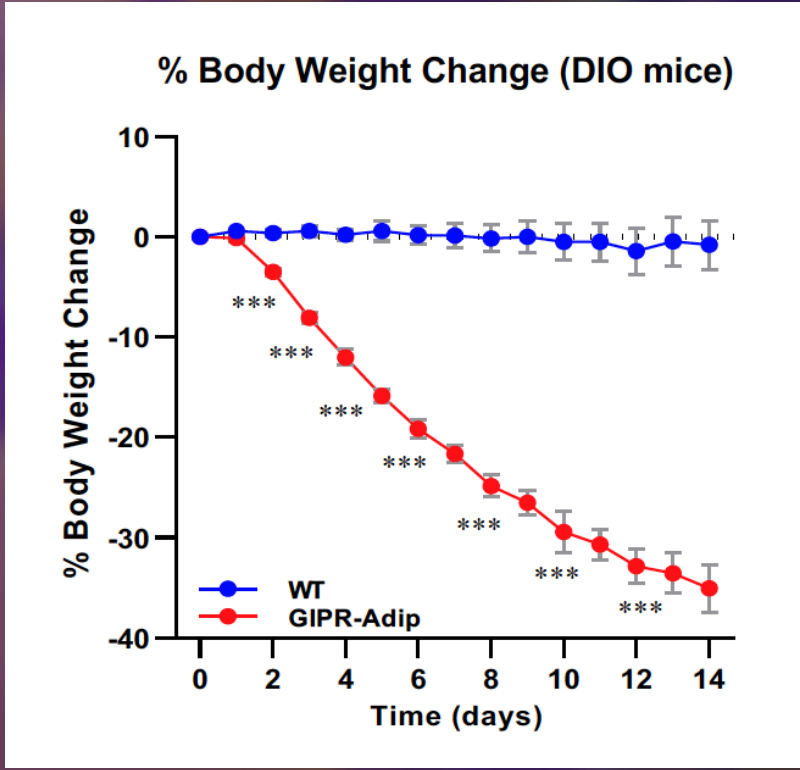
Regmi et al., 2024, Cell Metabolism *36*, 1–16
July 2, 2024



Highlights

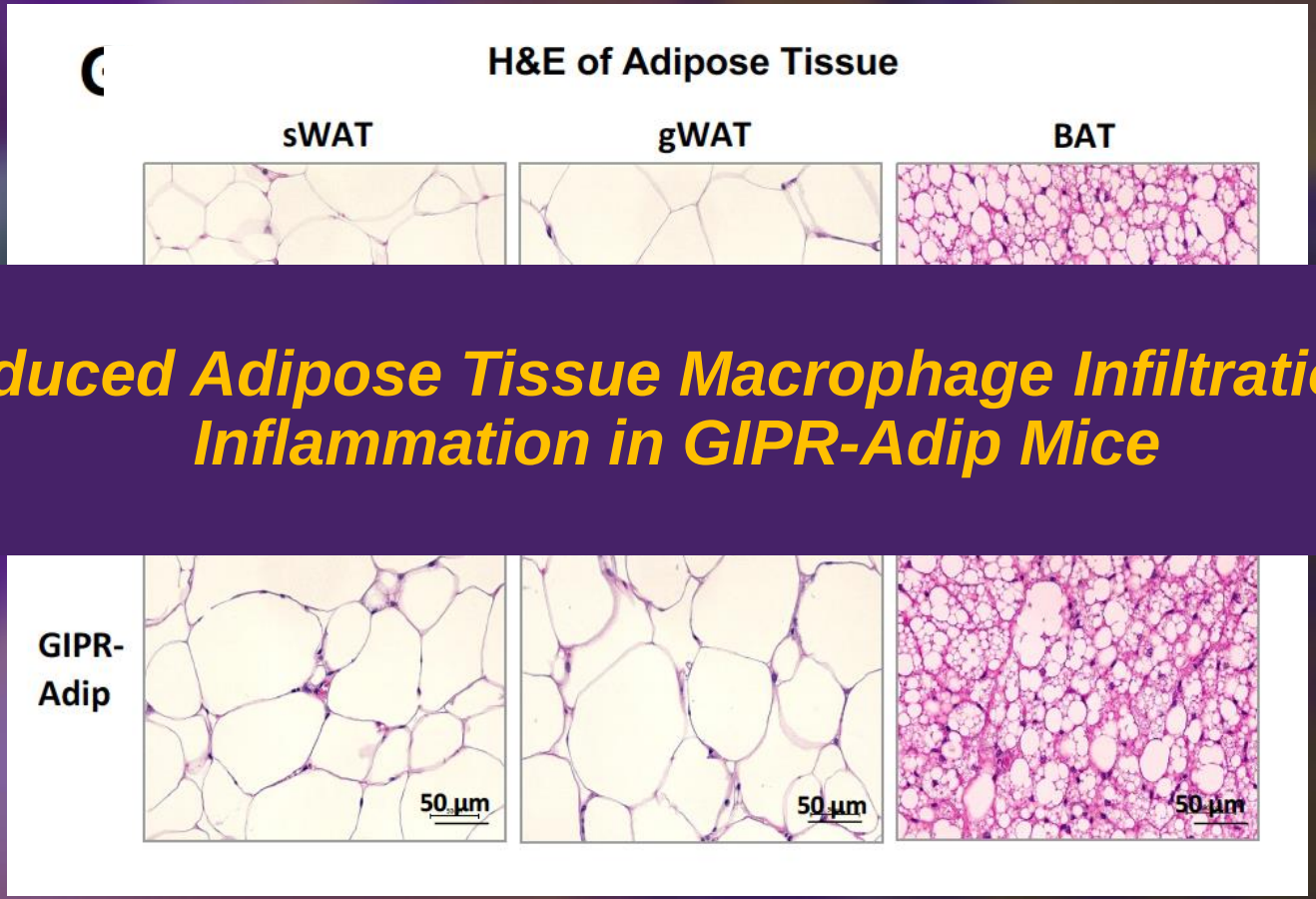
- GIPR is expressed and functionally signals in both human and mouse adipocytes
- Tirzepatide signals to adipocytes via long-acting GIPR agonism
- Tirzepatide regulates adipocyte lipid and glucose storage as well as lipid efflux
- Tirzepatide regulation of adipocyte function likely contributes to overall efficacy

Fat-specific GIPR mice *Reverse* Diet-induced Obesity: Massive Weight Loss



Kusminski Lab
Cell Metabolism
in press

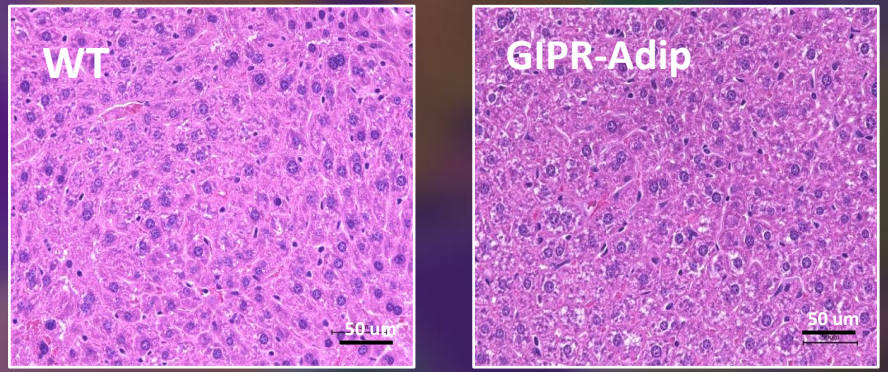
Normal Adipose Tissue Morphology



Reduced Adipose Tissue Macrophage Infiltration & Inflammation in GIPR-Adip Mice

Kusminski Lab
Cell Metabolism
in press

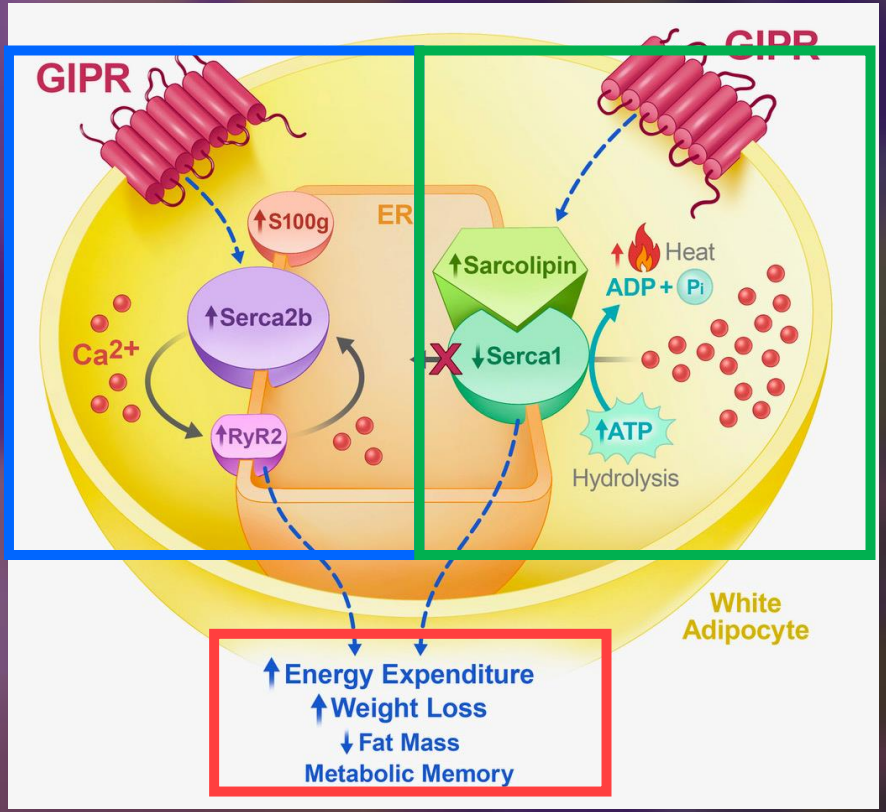
Fat-specific GIPR mice do Not Display Hepatic Steatosis



Kusminski Lab
Cell Metabolism
in press

A Mechanism of GIPR Action in Adipose Tissue:

The GIPR Activates Calcium Cycling in the Adipocyte to Enhance Thermogenesis, Increase Energy Expenditure and Drive Weight Loss



Kusminski Lab
Cell Metabolism
in press

Leptin Endotrophin

FGF21 Adiponectin

Adipocyte GIP-R Action

Want to fix the liver ?

fix the Adipose tissue first!

Is it always the adipocyte?
Liver autonomous effects!

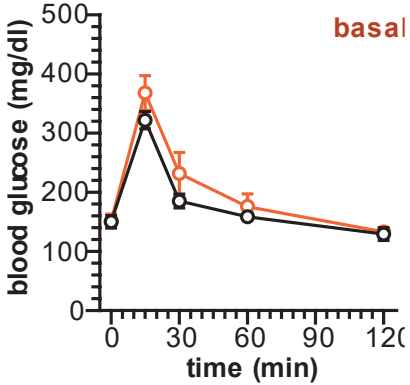
A novel liver dysfunction model:
The Hepato-Mitokiller

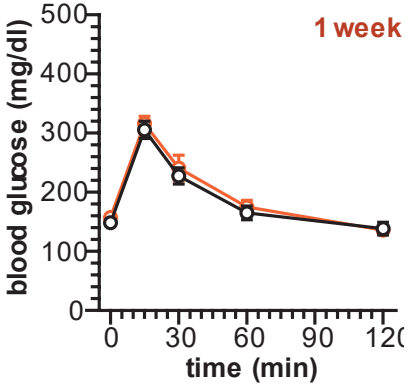
Jan Funcke, unpublished

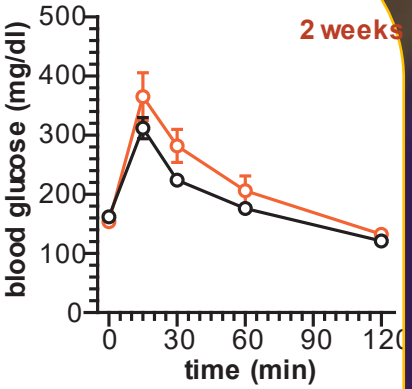
Elimination of mitochondrial DNA:

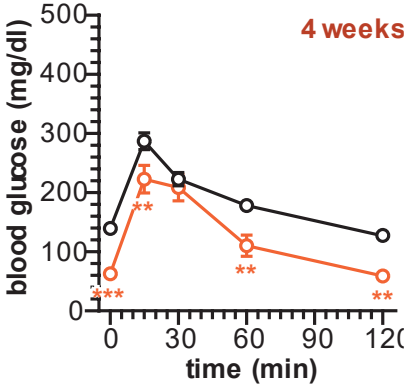
The rho^{-} hepatocyte

Jan Funcke, unpublished

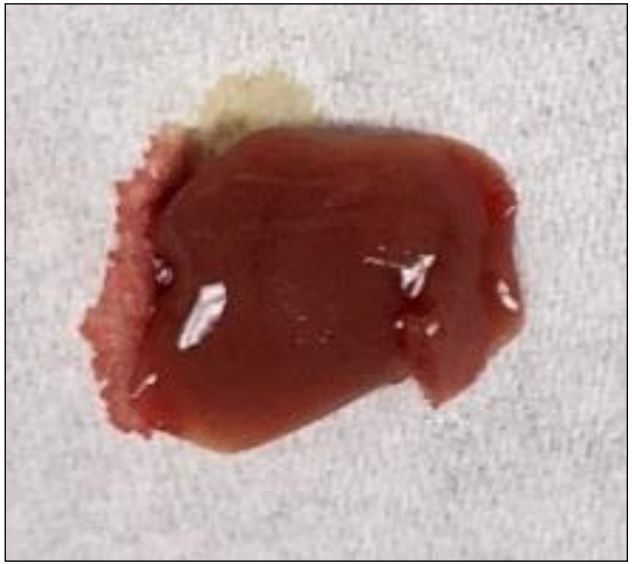




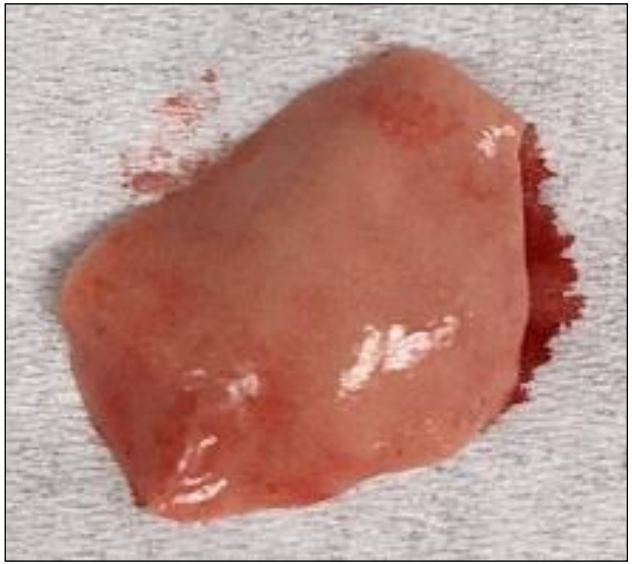




Jan Funcke, unpublished



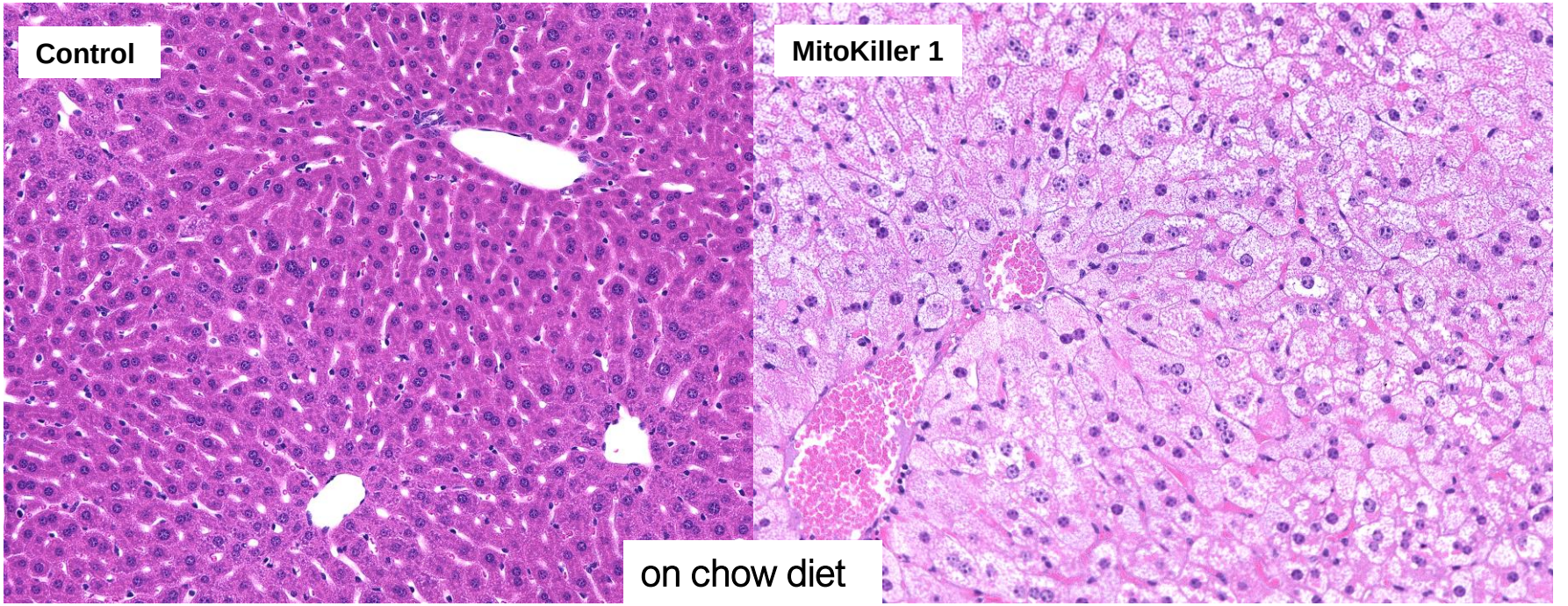
ctrl



hepato-mitoKiller

on chow diet

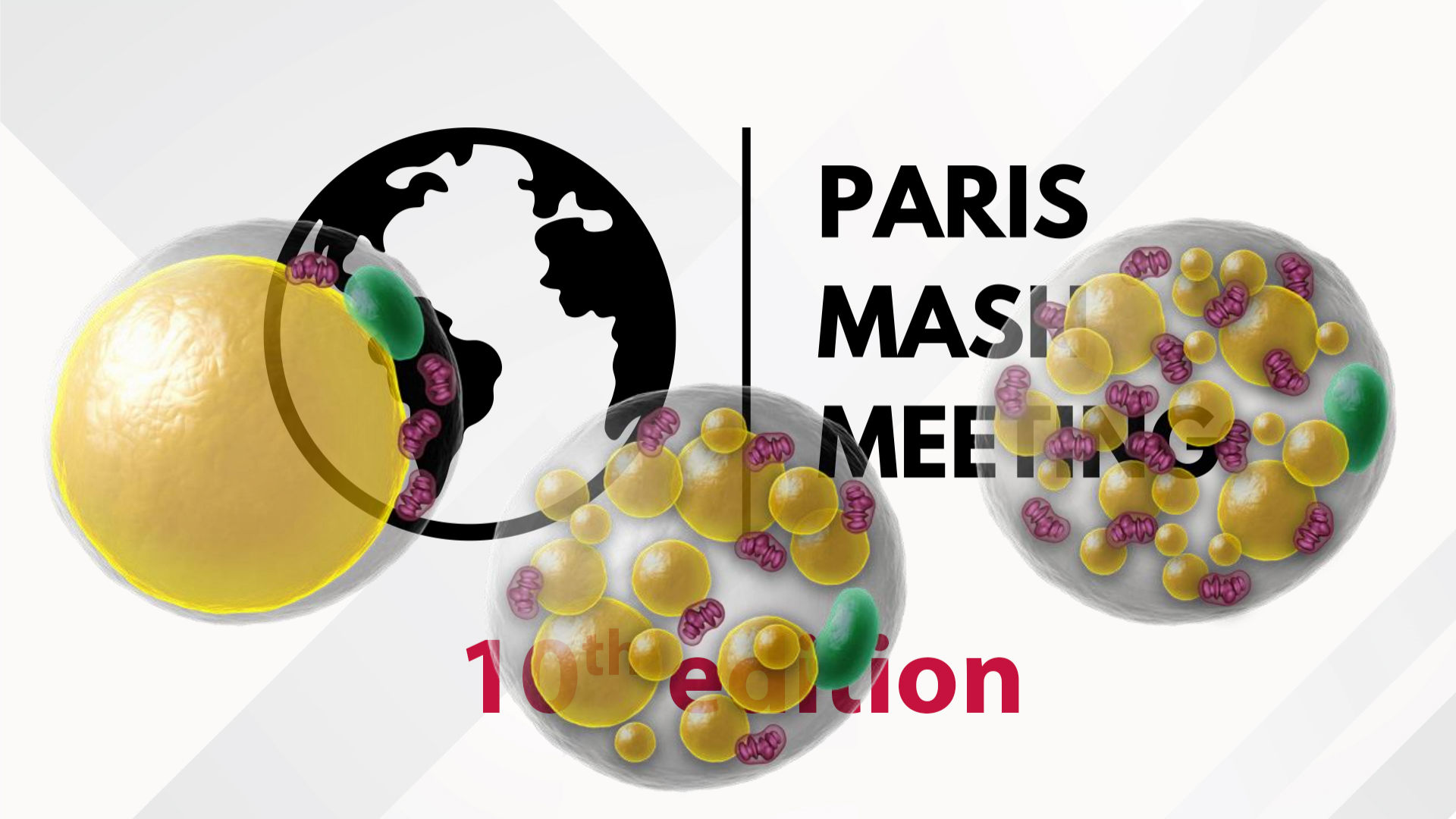
Jan Funcke, unpublished



Jan Funcke, unpublished

Hepato - mitoKillers


Touchstone Diabetes Center
UT SOUTHWESTERN MEDICAL CENTER
60 years NIDDK NATIONAL INSTITUTE OF DIABETES AND DIGESTIVE AND KIDNEY DISEASES
60 Years of Advancing Research to Improve Health
Research supported by NIDDK
American Diabetes Association.
JDRF IMPROVING LIVES. CURING TYPE 1 DIABETES.
Christine Kusminski
Qingzhang Zhu
Line Pedersen, Daeseok Kim
Shangang Zhao
Jan-Bernd Funcke
Toshiharu Onodera
Bianca Field, Dawei Bu
David Chien, Megan Paredes
Qian Lin, Christy Gliniak
Heidi Schumacher
Chao Li, Nancy Sun
Chanmin Joung, Ivan Valdez
Miguel Talamo, Joselin Velasco
Katarzyna Walendzik
Kyounghee Min, David Chen
Lisandro Maya-Ramos
Yan Li, Giovanna Degasperi
Annabel Wang
Kai Sun
Joseph Rutkowski
Zhao Wang
Anders Berg, Na Li
Jiyoung Park, Min Kim
Jayoung Kim
Puneeth Iyengar
William Holland
Ingrid Wernstedt
Jennifer Stern
Nils Halberg
Utpal Pajvani
Yi Zhu
Yingfeng Deng
Zhuzhen Zhang
Clair Crewe
Aaron An
Nolwenn Joffin
Leon Straub
Samir Parikh
Amanda Clark
Sam Klein
Ruth Gordillo
Orson Moe
Javier Neyra
Joseph Hill
Thomas Gillette
Prasanna Alluri
Emina Huang
Ricardo Samms
Matthew Coghlan
Zhiqiang An
Ningyan Zhang
Christy Glini
Deep Dixit
Yun-Hee Hou
Tamas Horva
Morten Karso
SHIMADZU
Lilly


PARIS
MASH
MEETING
10th edition


5323
Southwestern Medical
AT DALLAS