Παχυσαρκία

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Contents lists available at ScienceDirect

Metabolism Clinical and Experimental

journal homepage: www.metabolismjournal.com

The epidemiology of obesity☆

Yu Chung Chooi [a], Cherlyn Ding [a], Faidon Magkos [a,b,c,*]

- Ο παγκόσμιος επιπολασμός της παχυσαρκίας έχει διπλασιαστεί από το 1980.
- Σχεδόν το ένα τρίτο του παγκόσμιου πληθυσμού ταξινομούνται πλέον ως υπέρβαροι ή παχύσαρκοι
- Αυξάνει τον κίνδυνο ανάπτυξης πολλαπλών ασθενειών και καταστάσεων, όπως ο σακχαρώδης διαβήτης, οι καρδιαγγειακές παθήσεις, αρκετοί τύποι καρκίνου, μια σειρά από μυοσκελετικές διαταραχές, και σχετίζεται με κακή ψυχική υγεία

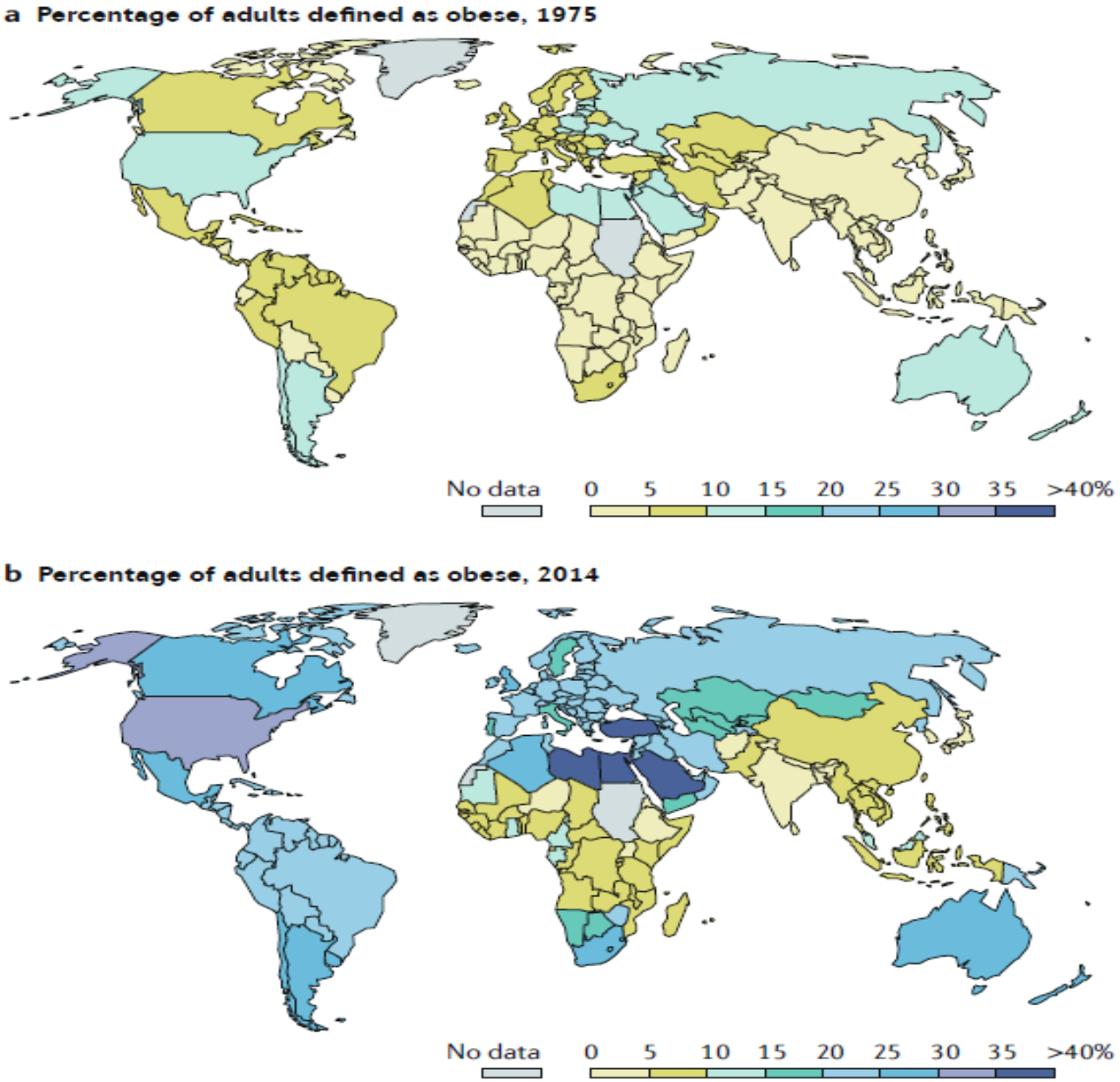
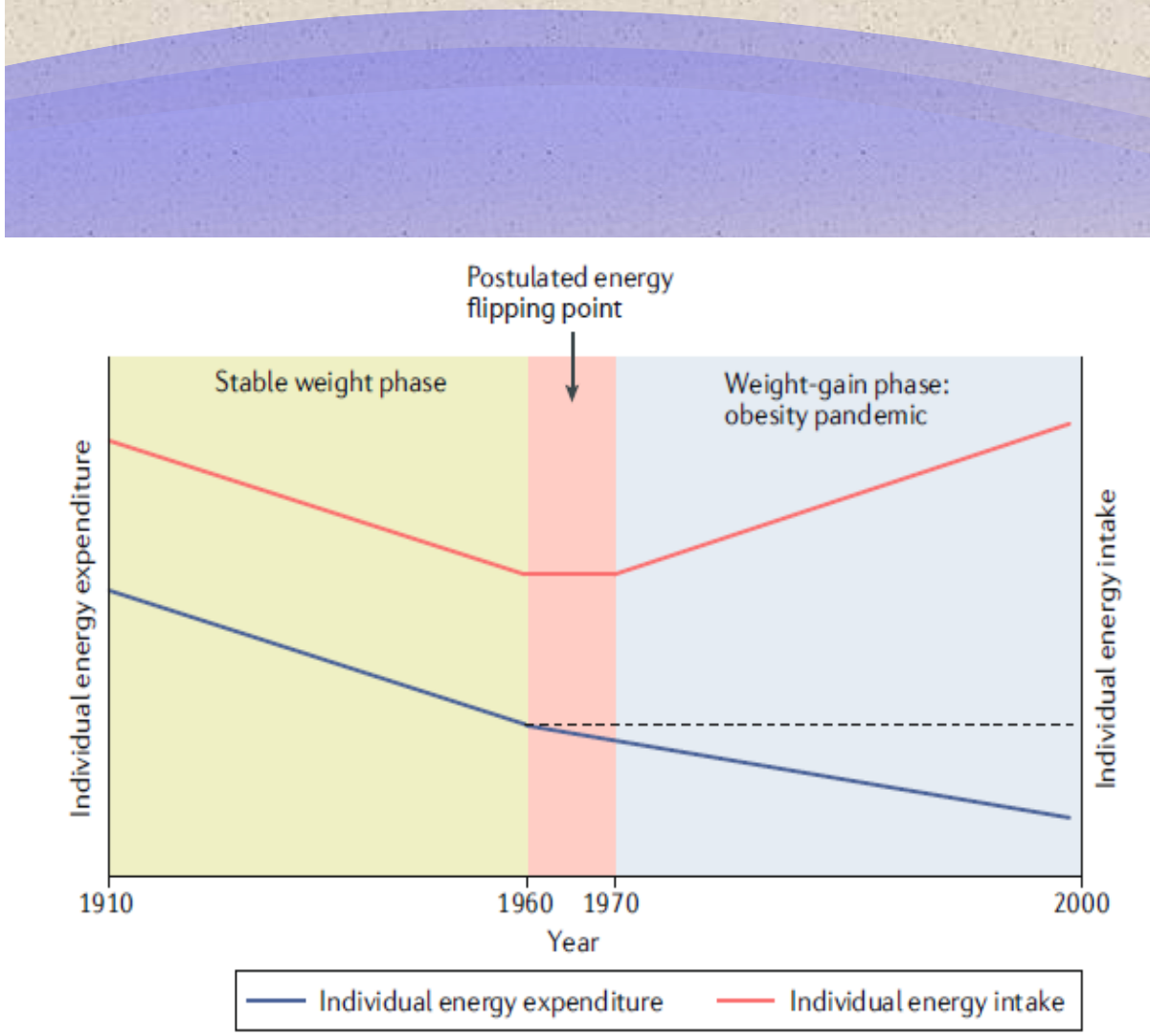


Fig. 4 | **Increase in prevalence of obesity over time.** Percentage of adults defined as obese by country in 1975 (part **a**) and 2014 (part **b**). The number of adults with obesity increased substantially between 1975 and 2014. Data from the WHO, Global Health Observatory.



Εισαγωγή

Contents lists available at ScienceDirect

Maturitas

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

Review

Economic costs of adult obesity: A review of recent European studies with a focus on subgroup-specific costs

Thomas von Lengerke[a,*], Christian Krauth[b]

[a] Hannover Medical School, Centre for Public Health and Healthcare, Medical Psychology Unit, OE 5430, Carl-Neuberg-Str. 1, 30625 Hannover, Germany
[b] Hannover Medical School, Centre for Public Health and Healthcare, Institute for Epidemiology, Social Medicine and Health Systems Research, OE 5410, Carl-Neuberg-Str. 1, 30625 Hannover, Germany

- Ο ΠΟΥ ορίζει το υπερβολικό βάρος και την παχυσαρκία ως μη φυσιολογική ή υπερβολική συσσώρευση λίπους που παρουσιάζει κίνδυνο για την υγεία
- Στις ΗΠΑ υπολογίζεται ότι το κόστος υγείας ανέρχεται σε 149,4 δισεκατομμύρια δολάρια
- Στην Ευρώπη, το συνολικό άμεσο και έμμεσο κόστος που αποδίδεται στην παχυσαρκία ήταν ισοδύναμο με 0,47–0,61%του ΑΕΠ

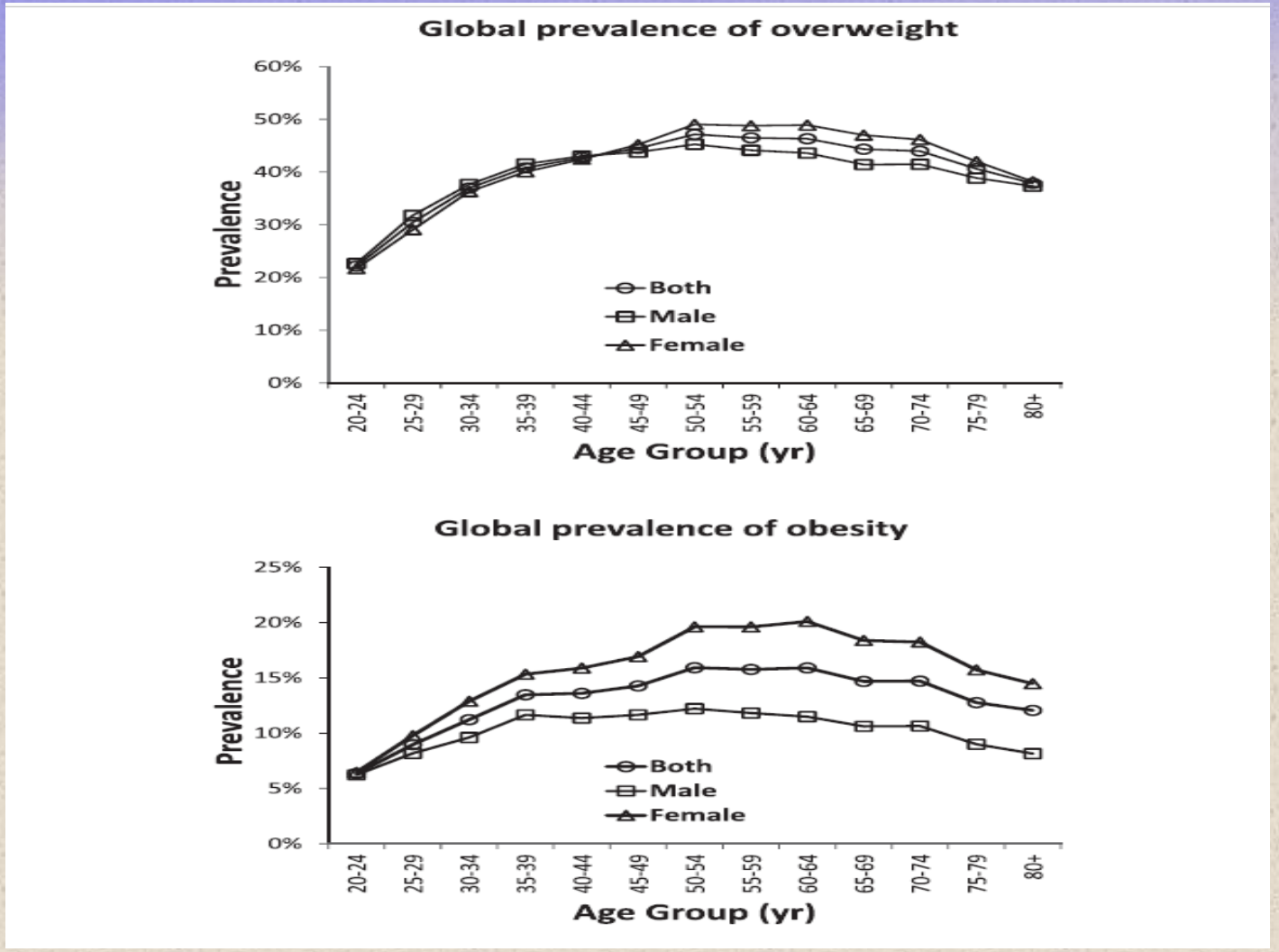
Δείκτης μάζας σώματος

- Για ενήλικες, οι παρούσες κατευθυντήριες οδηγίες από τα Κέντρα Ελέγχου και Πρόληψης Νοσημάτων των ΗΠΑ (CDC) και ο ΠΟΥ ορίζουν:
  - ΔΜΣ από 18,5 έως 24,9 kg/m2 ως φυσιολογικός
  - ΔΜΣ ≥ 25 kg/m2 ως υπέρβαρος
  - ΔΜΣ ≥ 30 kg/m2 ως παχύσαρκος,
  - ΔΜΣ ≥ 40 kg/m2 ως σοβαρή παχυσαρκία

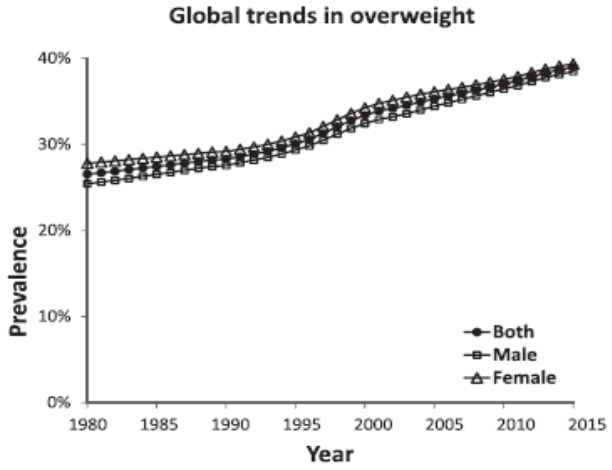
Δείκτης Μάζας Σώματος

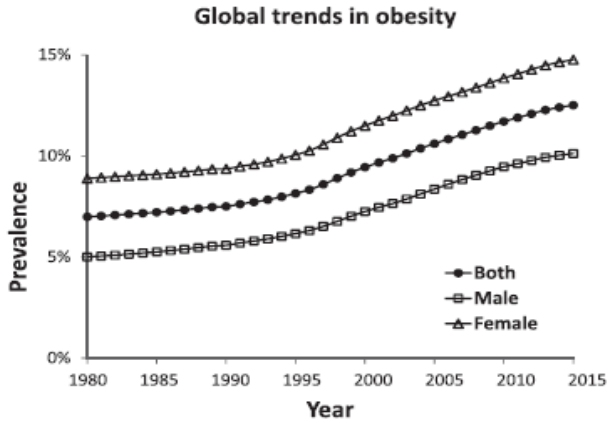
$$B.M.I = \frac{\text{βάρος}}{\text{ύψος}^2}$$

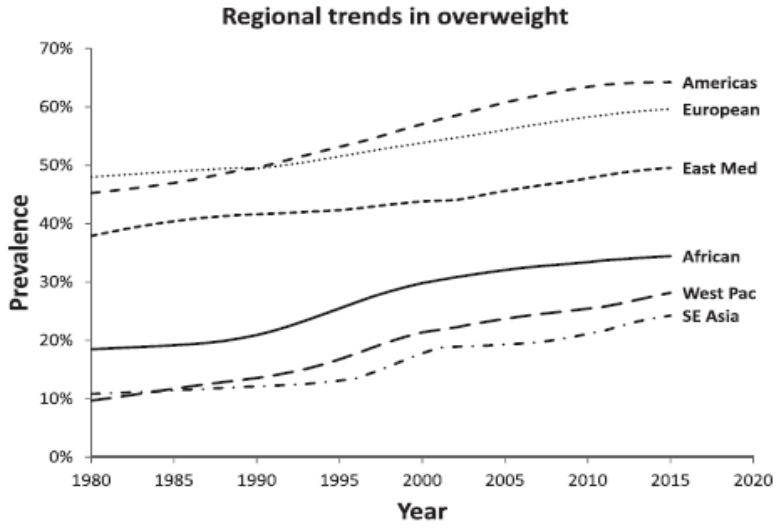
Επιπολασμός: Παγκόσμια



Επιπολασμός: Τάση στο χρόνο







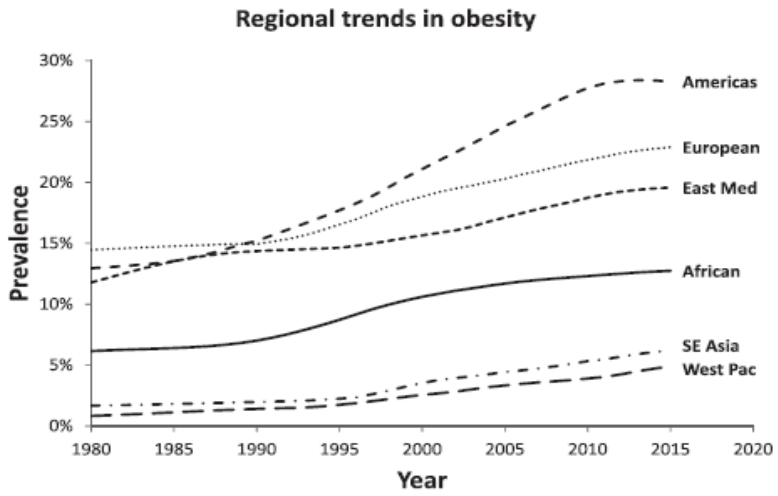


Fig. 2. Age-standardised global prevalence of overweight (top) and obesity (bottom) in men and women > 20 years old by year (ca. 1980–2015).

Παθ/Φυσ.

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
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https://doi.org/10.1016/j.jacc.2017.11.011

THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Obesity

Pathophysiology and Management

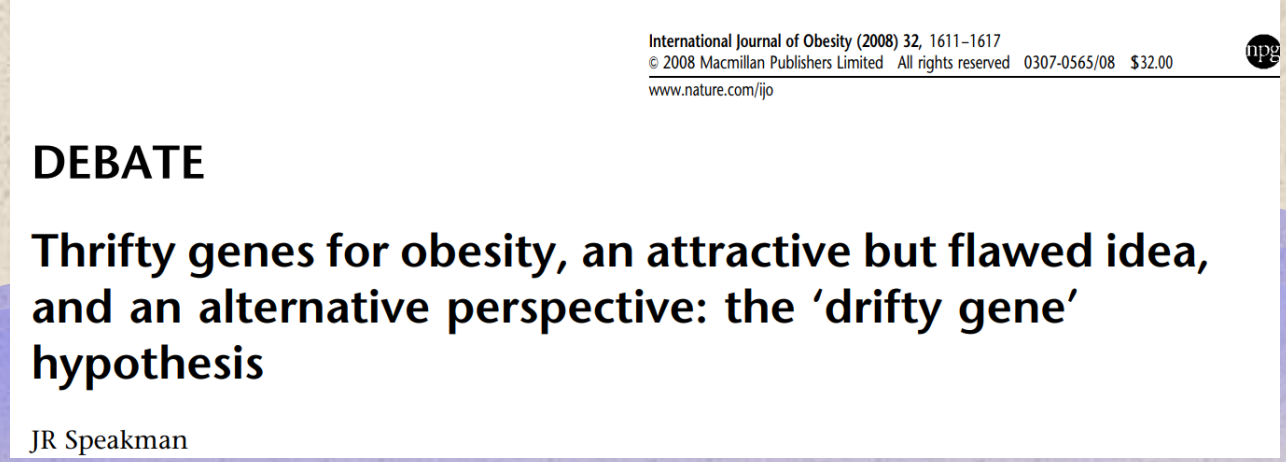
Kishore M. Gadde, MD, Corby K. Martin, PhD, Hans-Rudolf Berthoud, PhD, Steven B. Heymsfield, MD

- Ενέργεια: Θερμίδες
- Διατροφή: Είναι όλες οι θερμίδες το ίδιο;
- Το σωματικό βάρος τείνει να διατηρείται ή να ρυθμίζεται ενεργά
- Άρα πρόκειται για νόσο
- Γενετική προδιάθεση με 140 Χρωμοσωματικές περιοχές
- Σημαντική γονιδιακή έκφραση στο ΚΝΣ

Obesity Pathogenesis: An Endocrine Society Scientific Statement

Michael W. Schwartz,[1] Randy J. Seeley,[2] Lori M. Zeltser,[3] Adam Drewnowski,[4] Eric Ravussin,[5] Leanne M. Redman,[5] and Rudolph L. Leibel[3,6]

Οικονομικά vs Παρασυρόμενα γονίδια



International Journal of Obesity (2008) 32, 1611–1617

0307-0565/08 $32.00
www.nature.com/ijo

DEBATE

Thrifty genes for obesity, an attractive but flawed idea, and an alternative perspective: the 'drifty gene' hypothesis

JR Speakman

- Η παχυσαρκία προκύπτει από μια αλληλεπίδραση περιβάλλοντος/τρόπου ζωής και γενετικής προδιάθεσης
- Η υπόθεση του «οικονομικού» γονιδίου υποθέτει ότι τα γονίδια που προάγουν την πρόσληψη ενέργειας και την υψηλή ενεργειακή αποδοτικότητα είναι αποτέλεσμα της εξέλιξης
- Η υπόθεση του «παρασυρόμενου» γονιδίου υποστηρίζει ότι η εξελικτική πίεση επιλογής για γονίδια που διατηρούν το σωματικό βάρος στο ελάχιστο μειώθηκε όταν οι άνθρωποιπριν από περίπου 2 εκατομμύρια χρόνια

Η αλήθεια μάλλον βρίσκεται ενδιάμεσα

REVIEW

CURRENT OPINION

Epigenetics of obesity: beyond the genome sequence

Paul Cordero[a], Jiawei Li[a], and Jude A. Oben[a,b]

- Δεδομένης της δυσανάλογα υψηλής έκφρασης των γονίδιων που σχετίζονται με την παχυσαρκία και τις επιγενετικές τροποποιήσεις στο κεντρικό νευρικό σύστημα, είναι πολύ πιθανό ότι τα γονίδια της παχυσαρκίας δρουν, όχι μόνο εντός του υποθαλάμου ως ομοιοστατικός ρυθμιστής του ενεργειακού ισοζυγίου
- Επιπλέον δρουν στα νευρωνικά κυκλώματα που εμπλέκονται σε αλληλεπιδράσεις με περιβάλλον, συμπεριλαμβανομένων των κυκλωμάτων στα οποία βασίζεται η απόφαση που βασίζεται σε ανταμοιβή

Γονιδιακά

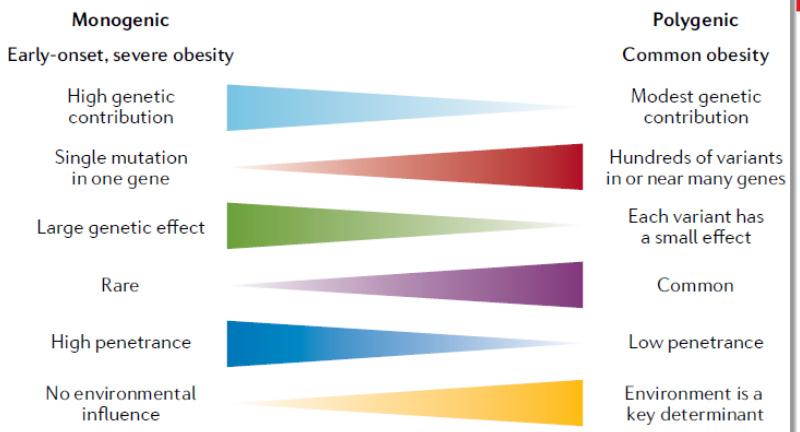


Fig. 2 | **Key features of monogenic and polygenic forms of obesity.**

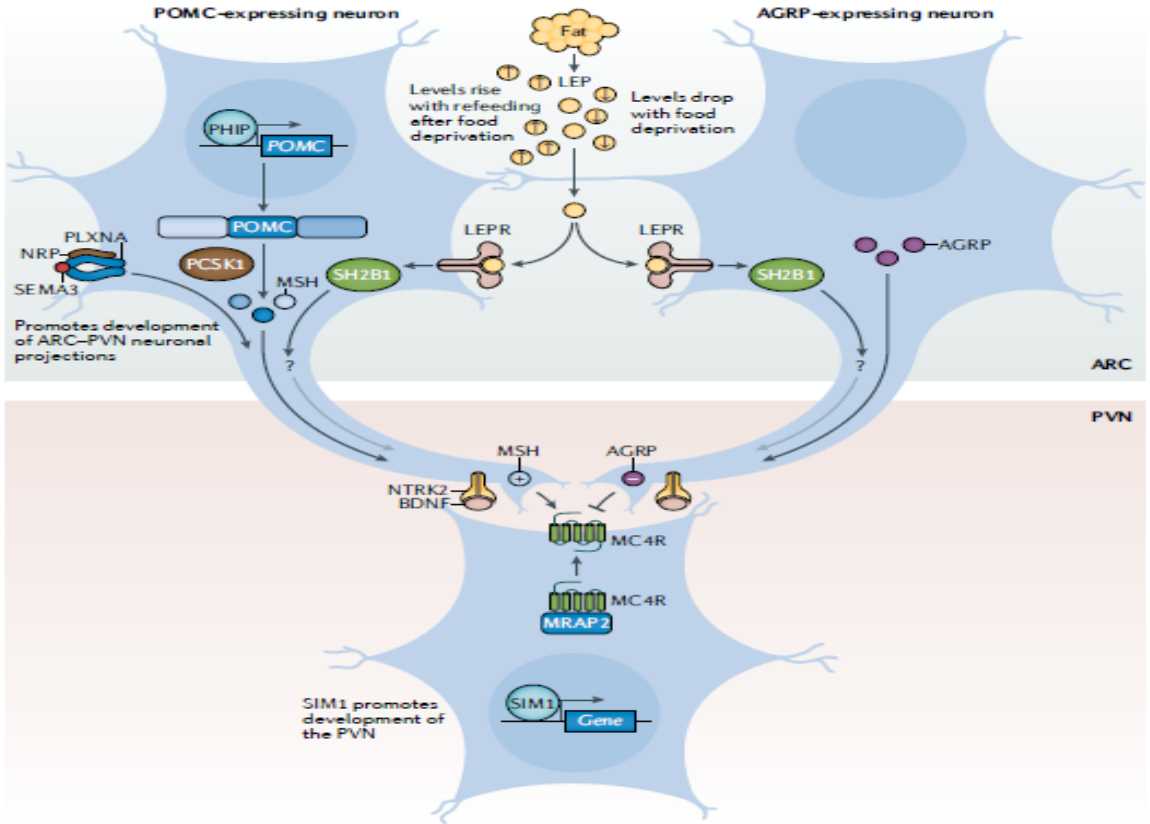


Fig. 4 | **The leptin–melanocortin pathway.** Pro-opiomelanocortin (POMC)-expressing neurons and agouti-related protein (AGRP)-expressing neurons within the arcuate nucleus of the hypothalamus (ARC) act to sense circulating leptin (LEP) levels, which reflect fat mass. These neurons signal to melanocortin 4 receptor (MC4R)-expressing neurons in the paraventricular nucleus of the hypothalamus (PVN), which controls appetite, thus linking long-term energy stores to feeding behaviour. Binding of class 3 semaphorins (SEMA3) to their receptors NRP and PLXNA influences the projection of POMC neurons to the PVN. Binding of brain-derived neurotrophic factor (BDNF) to its receptor neurotrophic receptor tyrosine kinase 2 (NTRK2) is thought to be an effector of leptin-mediated synaptic plasticity of neurons, including those in the ARC and PVN. The transcription factor SIM1 is crucial for the proper development of the PVN. +, agonist; –, antagonist; LEPR, leptin receptor; MRAP2, melanocortin receptor accessory protein 2; MSH, melanocyte-stimulating hormone; SH2B1, SH2B adaptor protein 1.

Physiology & Behavior 104 (2011) 29–39

Contents lists available at ScienceDirect

Physiology & Behavior

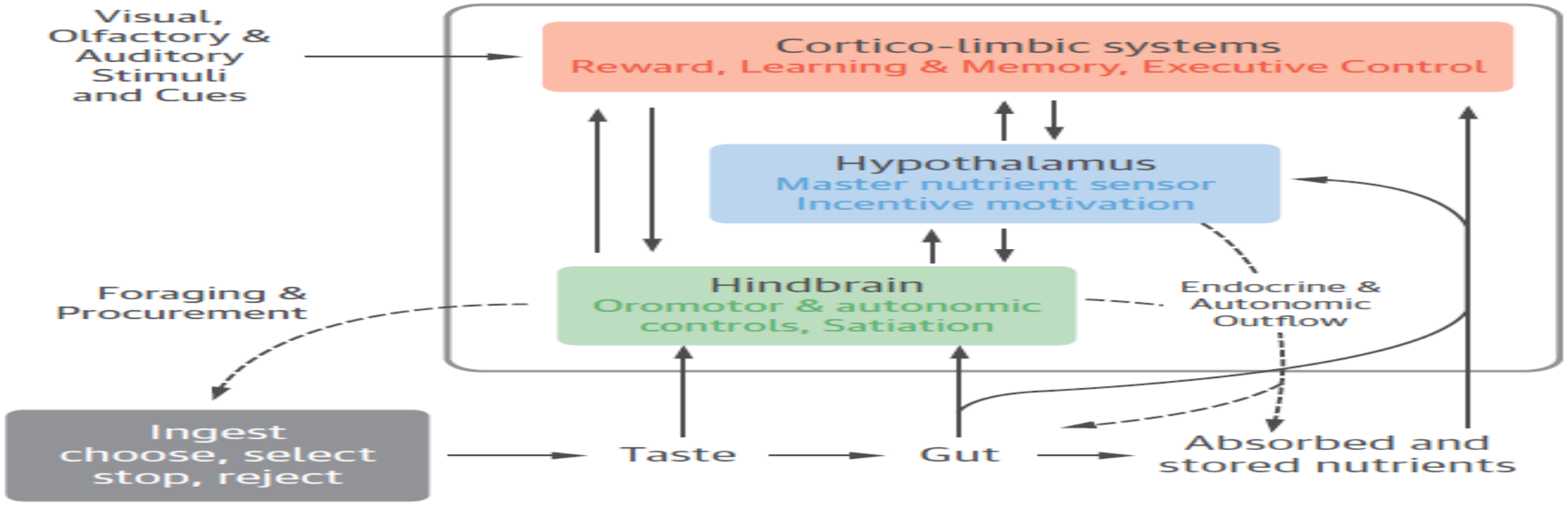
journal homepage: www.elsevier.com/locate/phb

The lateral hypothalamus as integrator of metabolic and environmental needs: From electrical self-stimulation to opto-genetics

Hans-Rudi Berthoud *, Heike Münzberg

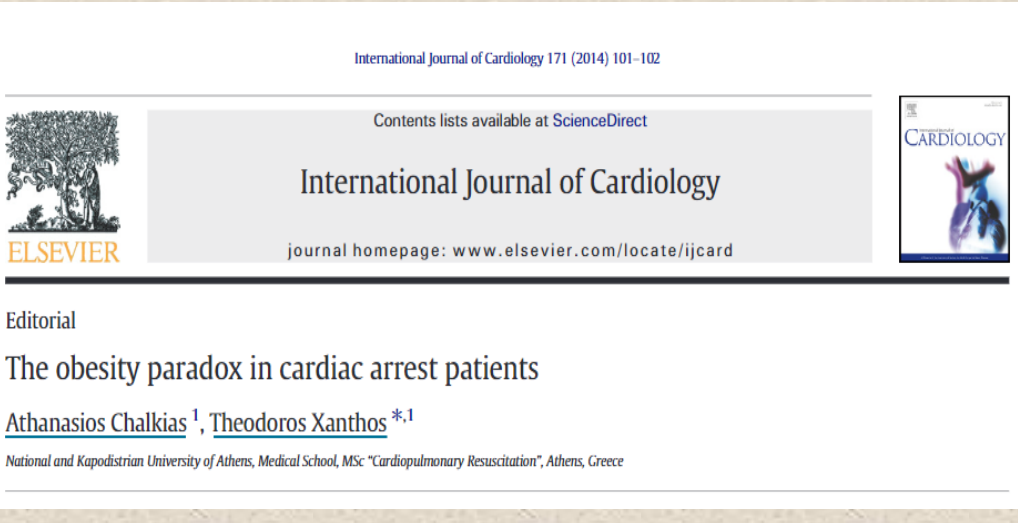
Neurobiology of Nutrition Laboratory, Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, Louisiana, USA

FIGURE 1 Neural Pathways and Systems Controlling Ingestive Behavior and Energy Balance



ΔΜΣ, Σωματότυπος και κίνδυνος νόσου

- Το λίπος αυξάνεται καμπυλόγραμμα με το ΔΜΣ
- Η χωρίς λίπος μάζα αυξάνεται επίσης με την αύξηση του ΔΜΣ
- Το ΔΜΣ ως παράμετρός παχυσαρκίας και υγείας συχνά αμφισβητείται σε ατομικό επίπεδο γιατί το ποσοστό λίπους για οποιοδήποτε ΔΜΣ ποικίλει
- «Το παράδοξο της παχυσαρκίας»


International Journal of Cardiology 171 (2014) 101–102

Contents lists available at ScienceDirect

International Journal of Cardiology

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

Editorial

The obesity paradox in cardiac arrest patients

Athanasios Chalkias [1], Theodoros Xanthos *,[1]

National and Kapodistrian University of Athens, Medical School, MSc "Cardiopulmonary Resuscitation", Athens, Greece

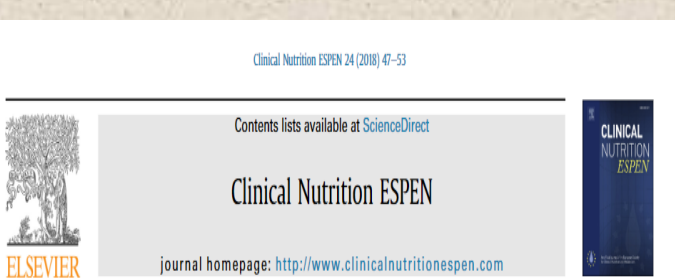

Mayo Clinic Proceedings

Volume 92, Issue 2, February 2017, Pages 266-279

Review

Obesity and Heart Failure: Focus on the Obesity Paradox

Salvatore Carbone MS [a b], Carl J. Lavie MD [c], Ross Arena PhD, PT [d]


Clinical Nutrition ESPEN 24 (2018) 47–53

Contents lists available at ScienceDirect

Clinical Nutrition ESPEN

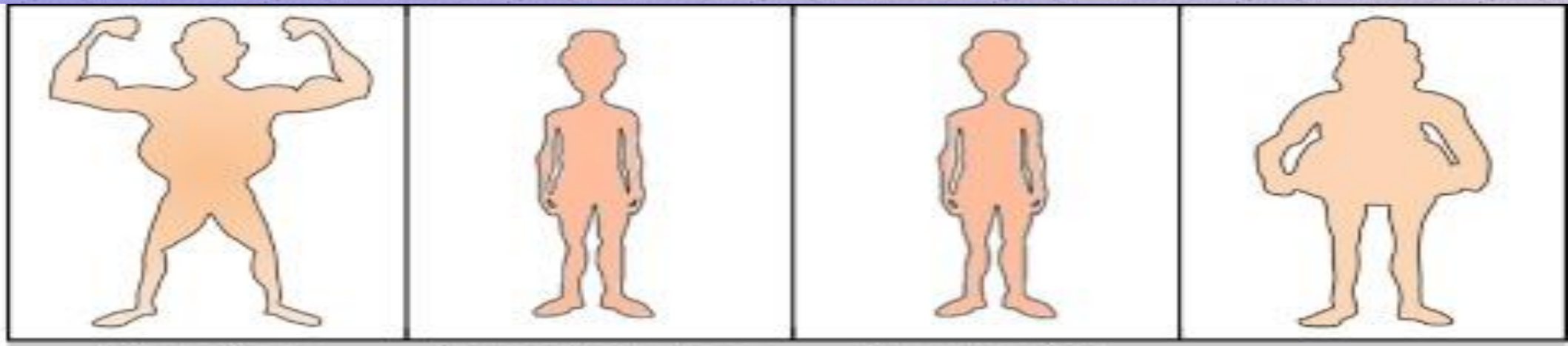
journal homepage: http://www.clinicalnutritionespen.com

Original article

The impact of body mass index on post resuscitation survival after cardiac arrest: A meta-analysis

Sotirios Kakavas [a,b,*], Georgios Georgiopoulos [c], Dimitrios Oikonomou [d], Dimitrios Karayiannis [e], Stefano Masi [f], Georgios Karlis [a,b], Theodoros Xanthos [b,g]

Οι φαινότυποι της παχυσαρκίας



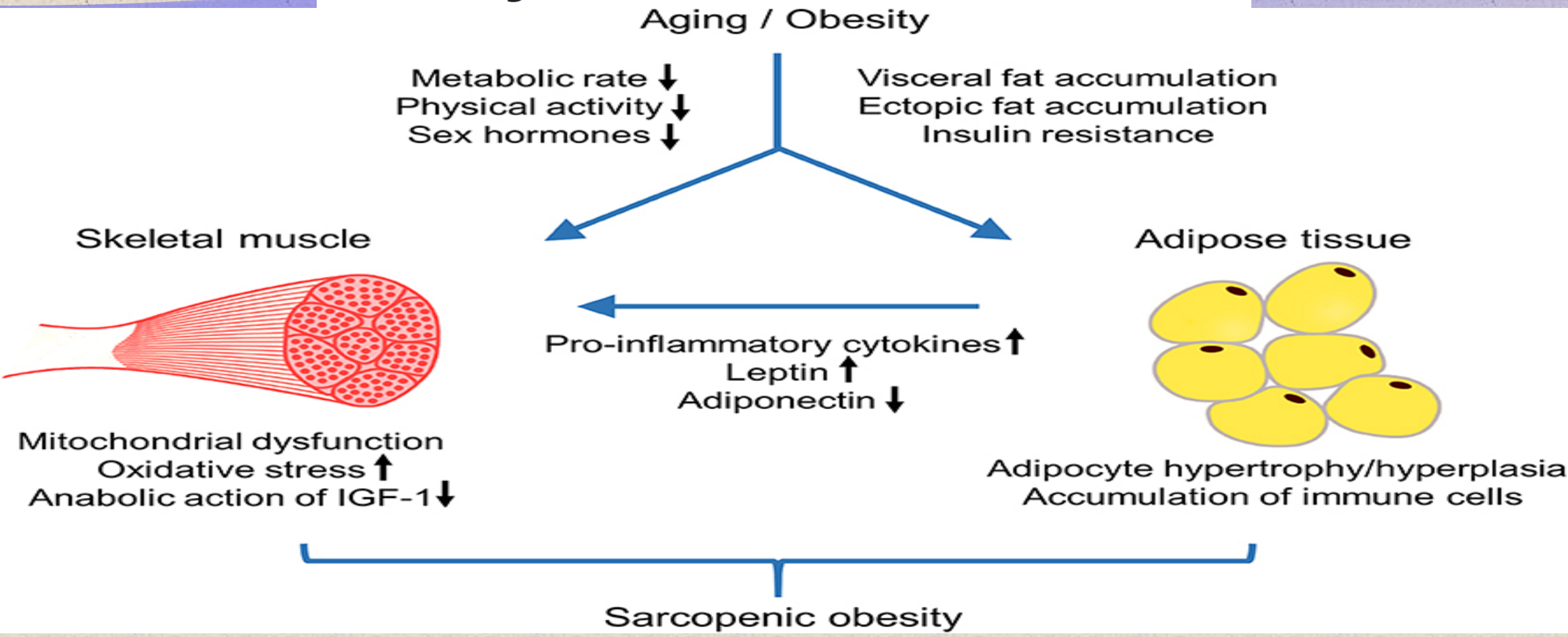
	Metabolically healthy obese	Metabolically obese normal weight	Normal weight obese	Sarcopenic obese
BMI (kg/m²)	> 30	18,5 - 25	18,5 - 25	> 30
VAT/FM	Low VAT	High VAT	Fat mass > 30%	High VAT
Lean mass	High	Normal	Normal	Low
CRF	High	Low	-	Low
Metabolic abnormalities	Absent	Present	Absent	Present

Obesity Facts
The European Journal of Obesity

Obes Facts 2018;11:294–305
DOI: 10.1159/000490361
Received: April 26, 2018
Accepted: May 17, 2018
Published online: July 18, 2018

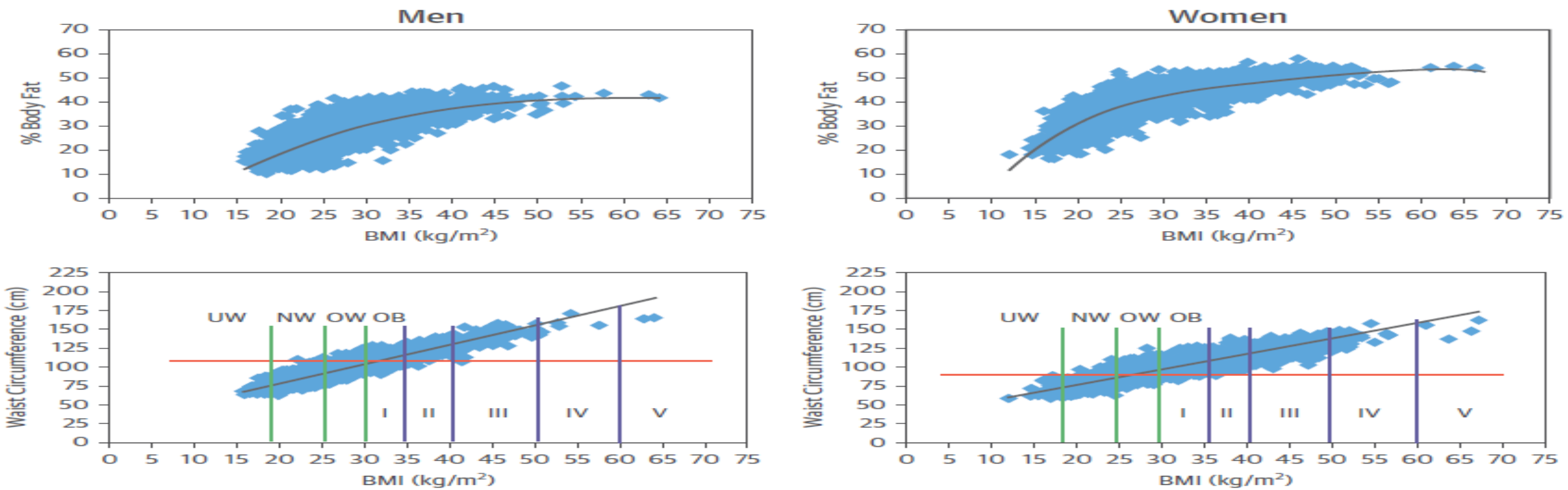
Review Article

Sarcopenic Obesity: Time to Meet the Challenge



Άλλοι δείκτες

FIGURE 2 Interrelations Among Adiposity Biomarkers



(Upper panels) Percent (%) body fat measured by dual-energy x-ray absorptiometry versus BMI in participants of the National Health and Nutrition Survey (1999 to 2006). The data were fit with polynomial regression lines (R^2 men, 0.61; women, 0.68; both $p < 0.001$). Note the curvilinear relation between body mass index (BMI) and % fat and the wide range of % body fat at any given level of BMI. **(Lower panels)** Waist circumference versus BMI in the same group of subjects as in the **upper panels** (R^2 men 0.84; women 0.80; both $p < 0.001$). **Horizontal lines** are shown at waist circumference cutpoints (>102 cm men, >88 cm women), above which health risks increase within the BMI range 24.9 to 34.9. No additional waist circumference predictive value is present with BMI levels ≥35. **Vertical lines** identify BMI ranges for underweight (UW), normal weight (NW), overweight (OW), and obesity (OB) classes I to V. Definitions of severe obesity are variable in the medical literature.

Δείκτες σωματότυπου

- Δείκτης σχήματος σώματος

- Δείκτης στρογγυλότητας σώματος

OPEN ACCESS Freely available online

PLoS one

A New Body Shape Index Predicts Mortality Hazard Independently of Body Mass Index

Nir Y. Krakauer[1]*, Jesse C. Krakauer[2]

1 Department of Civil Engineering, The City College of New York, New York, New York, United States of America, **2** Middletown Medical, Middletown, New York, United States of America

Abstract

Background: Obesity, typically quantified in terms of Body Mass Index (BMI) exceeding threshold values, is considered a leading cause of premature death worldwide. For given body size (BMI), it is recognized that risk is also affected by body shape, particularly as a marker of abdominal fat deposits. Waist circumference (WC) is used as a risk indicator supplementary to BMI, but the high correlation of WC with BMI makes it hard to isolate the added value of WC.

Methods and Findings: We considered a USA population sample of 14,105 non-pregnant adults (age ≥ 18) from the National Health and Nutrition Examination Survey (NHANES) 1999–2004 with follow-up for mortality averaging 5 yr (828 deaths). We developed A Body Shape Index (ABSI) based on WC adjusted for height and weight:

$$\mathrm{ABSI} \equiv \frac{\mathrm{WC}}{\mathrm{BMI}^{2/3}\mathrm{height}^{1/2}}.$$

Original Article
CLINICAL TRIALS: BEHAVIOR, PHARMACOTHERAPY, DEVICES, SURGERY

Obesity

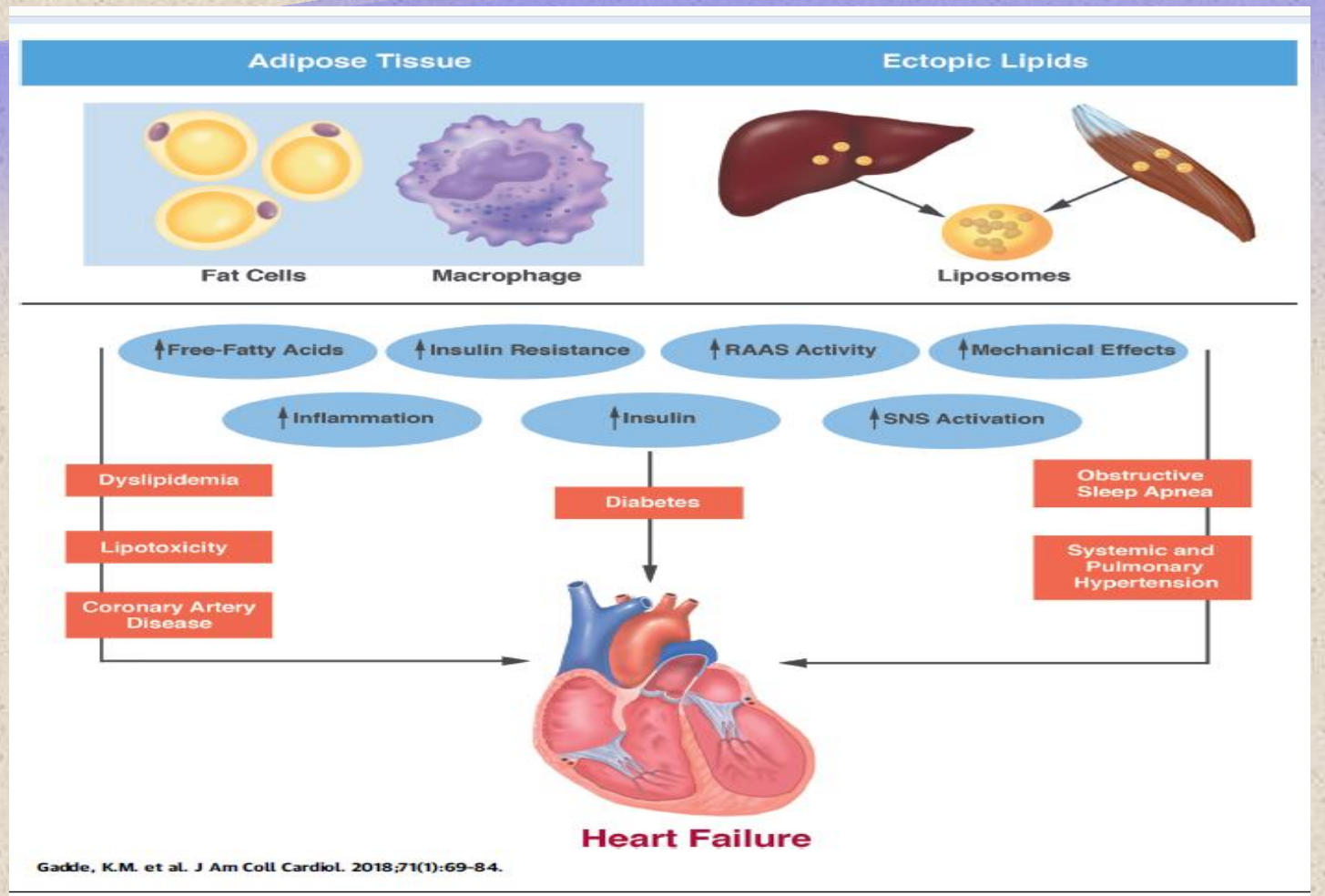
Relationships Between Body Roundness with Body Fat and Visceral Adipose Tissue Emerging from a New Geometrical Model

Diana M. Thomas[1], Carl Bredlau[1], Anja Bosy-Westphal[2], Manfred Mueller[2], Wei Shen[3], Dympna Gallagher[3], Yuna Maeda[1], Andrew McDougall[1], Courtney M. Peterson[4], Eric Ravussin[4] and Steven B. Heymsfield[4]

Objective: To develop a new geometrical index that combines height, waist circumference (WC), and hip circumference (HC) and relate this index to total and visceral body fat.
Design and Methods: Subject data were pooled from three databases that contained demographic, anthropometric, dual energy X-ray absorptiometry (DXA) measured fat mass, and magnetic resonance imaging measured visceral adipose tissue (VAT) volume. Two elliptical models of the human body were developed. Body roundness was calculated from the model using a well-established constant arising from the theory. Regression models based on eccentricity and other variables were used to predict %body fat and %VAT.
Results: A body roundness index (BRI) was derived to quantify the individual body shape in a height-independent manner. Body roundness slightly improved predictions of %body fat and %VAT compared to the traditional metrics of body mass index (BMI), WC, or HC. On this basis, healthy body roundness ranges were established. An automated graphical program simulating study results was placed at http://www.pbrc.edu/bodyroundness.
Conclusion: BRI, a new shape measure, is a predictor of %body fat and %VAT and can be applied as a visual tool for health status evaluations.

Obesity (2013) **21**, 2264–2271. doi:10.1002/oby.20408

Η περιφέρεια της μέσης και η καρδιά



Gadde, K.M. et al. J Am Coll Cardiol. 2018;71(1):69-84.

Increased plasma levels of free-fatty acids and cytokines, intracellular non-adipose tissue lipids (e.g., liposomes), and ectopic adipose tissue depots (e.g., within the visceral compartment) can contribute to systemic inflammation, insulin resistance, and overactivity of the sympathetic nervous system. The metabolic and anatomic effects of excess adiposity can lead to the development of type 2 diabetes, nonalcoholic fatty liver disease, obesity-related dyslipidemias, high blood pressure, and osteoarthritis. The cascade of these pathophysiological mechanisms and associated diseases are the main contributors to obesity-related heart failure. HF = heart failure; RAAS = renin-angiotensin aldosterone system; SNS = sympathetic nervous system.

Obesity: global epidemiology and pathogenesis

Matthias Blüher

Abstract | The prevalence of obesity has increased worldwide in the past ~50 years, reaching pandemic levels. Obesity represents a major health challenge because it substantially increases the risk of diseases such as type 2 diabetes mellitus, fatty liver disease, hypertension, myocardial infarction, stroke, dementia, osteoarthritis, obstructive sleep apnoea and several cancers, thereby contributing to a decline in both quality of life and life expectancy. Obesity is also associated with unemployment, social disadvantages and reduced socio-economic productivity, thus increasingly creating an economic burden. Thus far, obesity prevention and treatment strategies — both at the individual and population level — have not been successful in the long term. Lifestyle and behavioural interventions aimed at reducing calorie intake and increasing energy expenditure have limited effectiveness because complex and persistent hormonal, metabolic and neurochemical adaptations defend against weight loss and promote weight regain. Reducing the obesity burden requires approaches that combine individual interventions with changes in the environment and society. Therefore, a better understanding of the remarkable regional differences in obesity prevalence and trends might help to identify societal causes of obesity and provide guidance on which are the most promising intervention strategies.

Παθογένεση

obesity reviews

doi: 10.1111/j.1467-789X.2009.00689.x

Diagnostic in Obesity and Complications

Obesity is a sign – over-eating is a symptom: an aetiological framework for the assessment and management of obesity

A. M. Sharma and R. Padwal

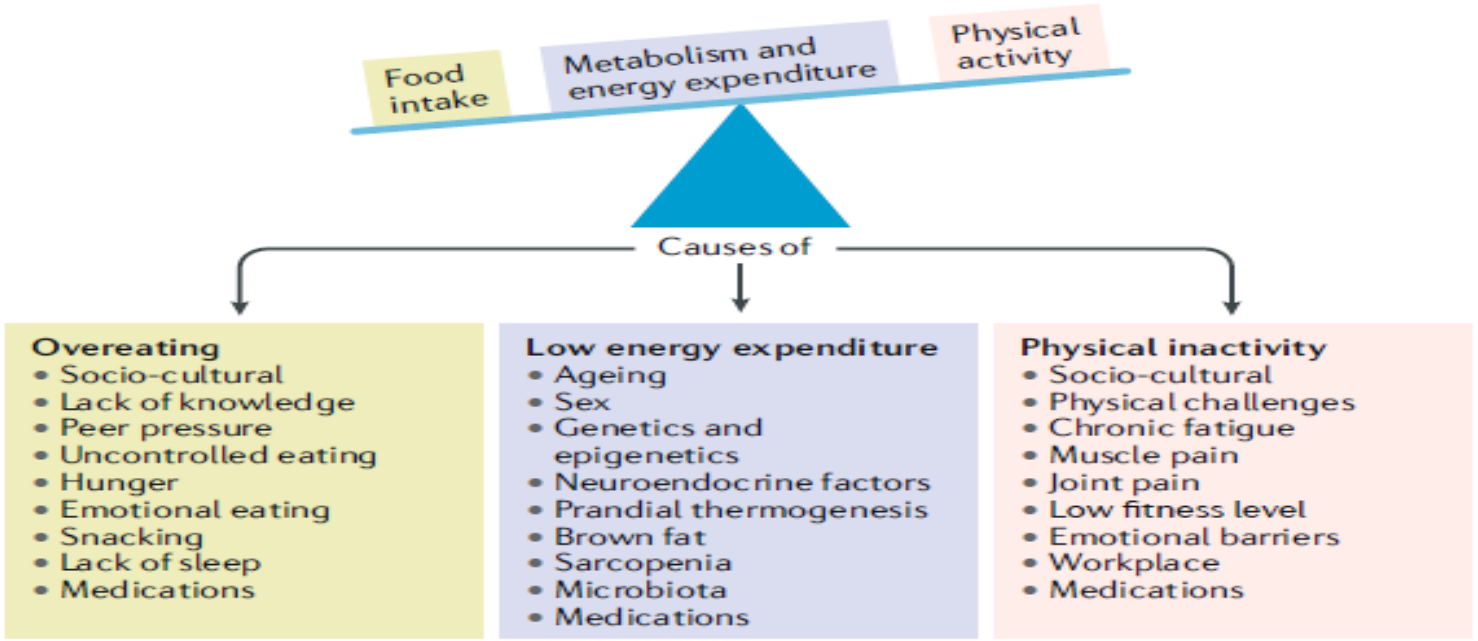


Fig. 1 | **Factors that can influence the chronic positive energy balance, thus subsequently causing obesity.** Weight gain can result from a combination of increased energy intake, low physical activity and reduced energy expenditure. Adapted with permission from REF.[28], Wiley-VCH.

Παθογένεση

Published in final edited form as:
Gastroenterology. 2017 May ; 152(7): 1728–1738. doi:10.1053/j.gastro.2016.12.050.

Blaming the brain for obesity: Integration of hedonic and homeostatic mechanisms

Hans-Rudolf Berthoud, **Heike Münzberg**, and **Christopher D. Morrison**
Neurobiology of Nutrition & Metabolism Department, Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, LA, USA

REVIEWS

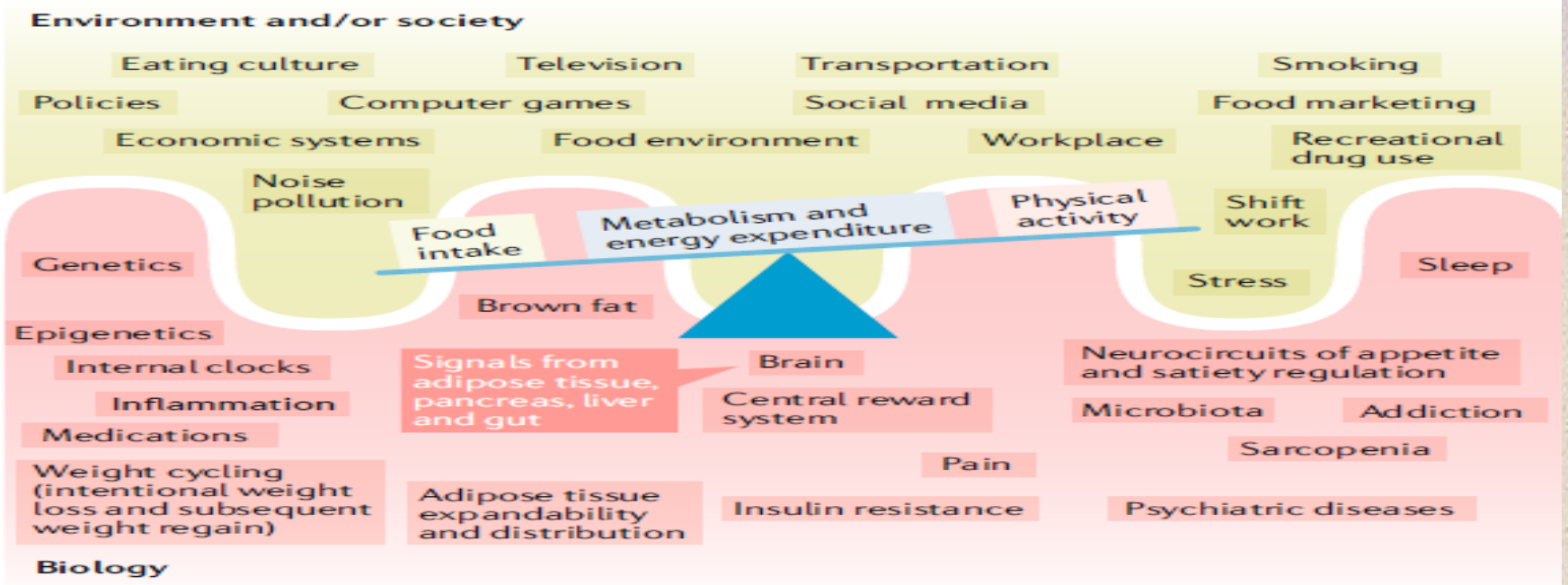
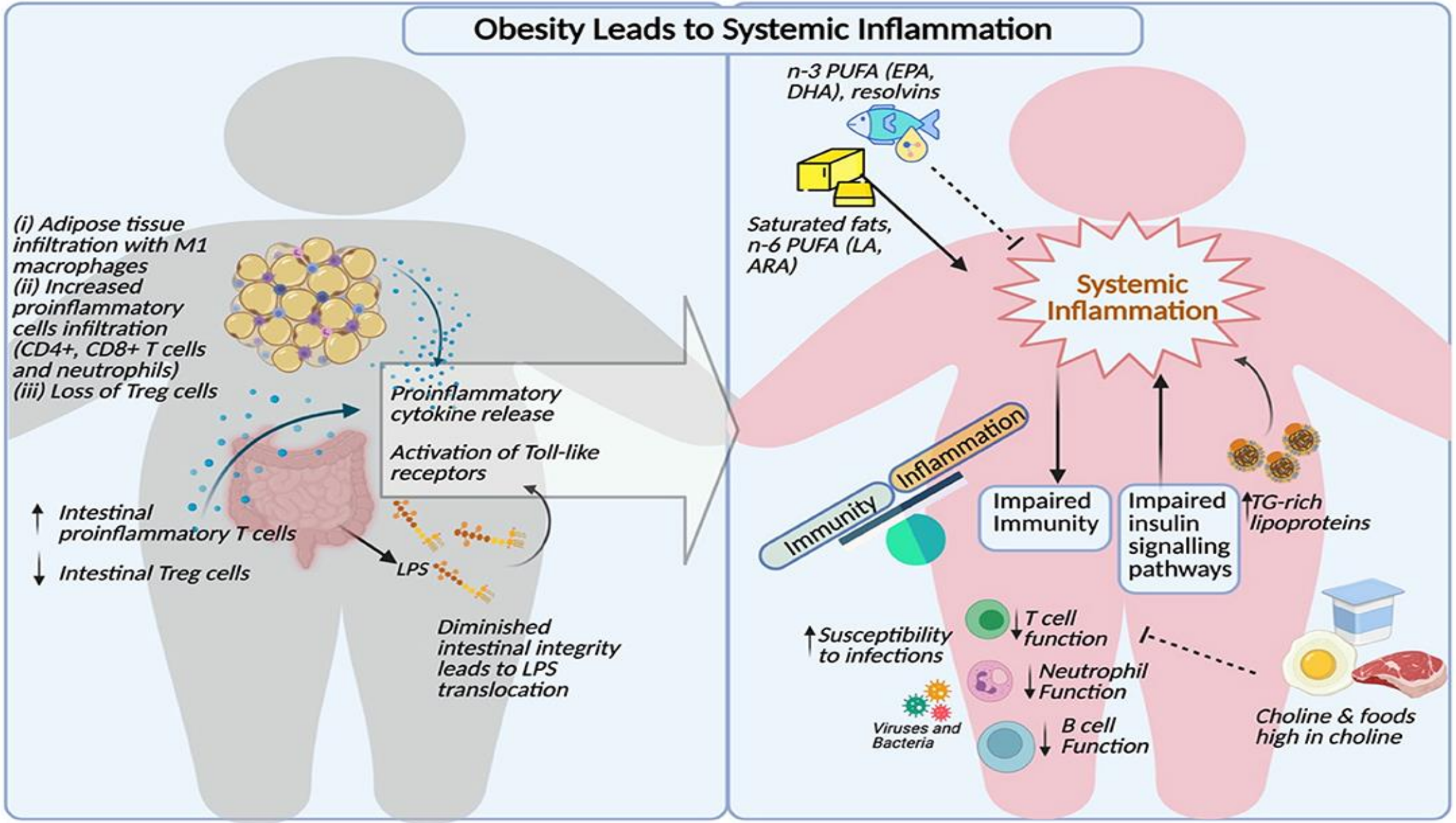


Fig. 2 | **Complex biological, environmental and societal factors contributing to obesity.** Individual factors (such as genetic background or the gut–brain–hormone axis) influence susceptibility to obesity, which may develop in an obesogenic environment (for example, influenced by eating culture, transportation and computerization).


Obesity Leads to Systemic Inflammation
n-3 PUFA (EPA, DHA), resolvins
Saturated fats, n-6 PUFA (LA, ARA)
(i) Adipose tissue infiltration with M1 macrophages
(ii) Increased proinflammatory cells infiltration (CD4+, CD8+ T cells and neutrophils)
(iii) Loss of Treg cells
Proinflammatory cytokine release
Activation of Toll-like receptors
Intestinal proinflammatory T cells
Intestinal Treg cells
LPS
Diminished intestinal integrity leads to LPS translocation
Systemic Inflammation
Immunity
Inflammation
Impaired Immunity
Impaired insulin signalling pathways
TG-rich lipoproteins
Susceptibility to infections
T cell function
Neutrophil Function
B cell Function
Viruses and Bacteria
Choline & foods high in choline

Obesity as a Disease

Jagriti Upadhyay, MD[a,b,c,*], Olivia Farr, PhD[c],
Nikolaos Perakakis, MD, PhD[c], Wael Ghaly, MD[c], Christos Mantzoros, MD, PhD[a,c]

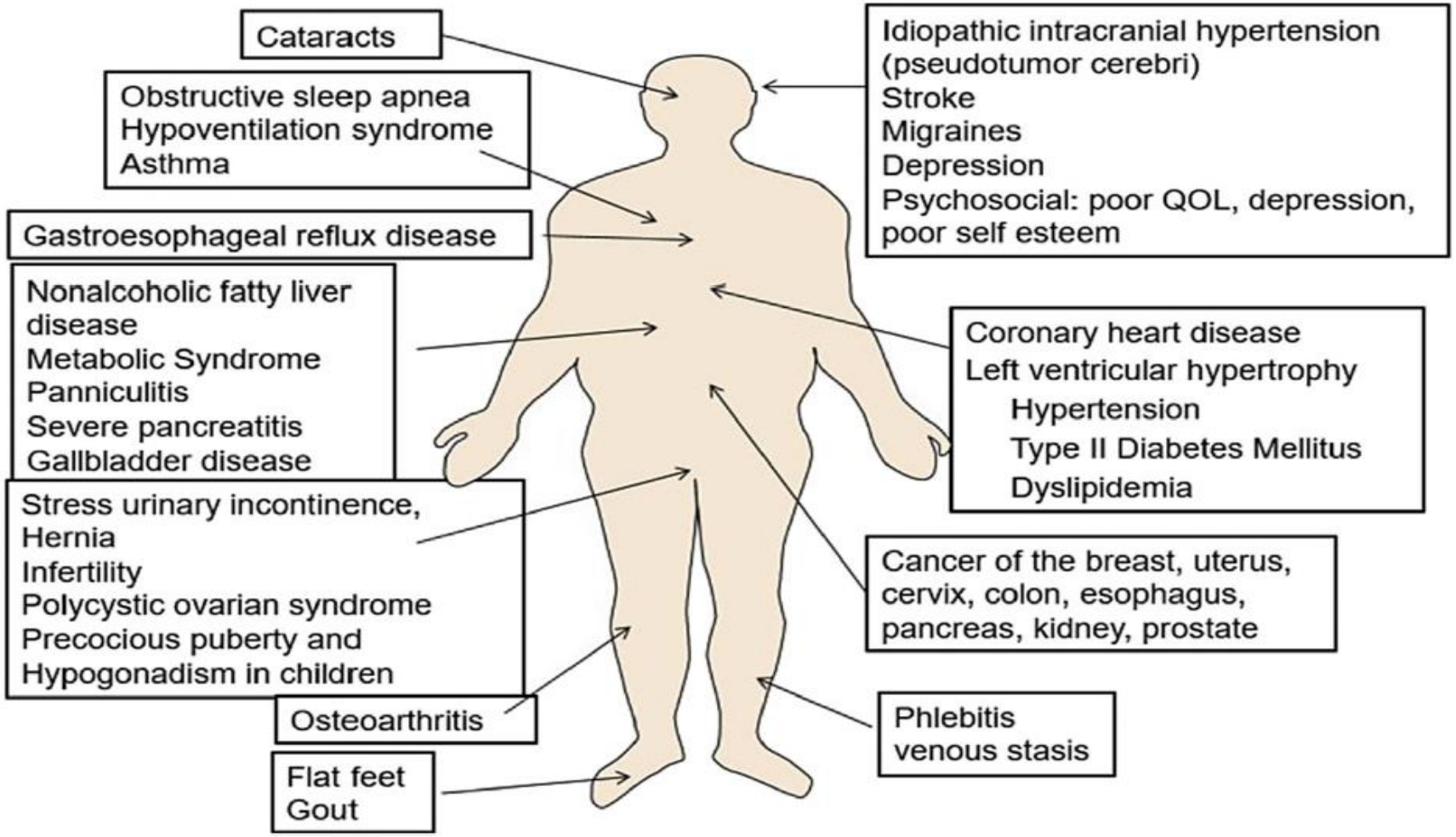


Fig. 1. Comorbidities associated with obesity. QOL, quality of life.

Παχυσαρκία και Αιμοδυναμικά

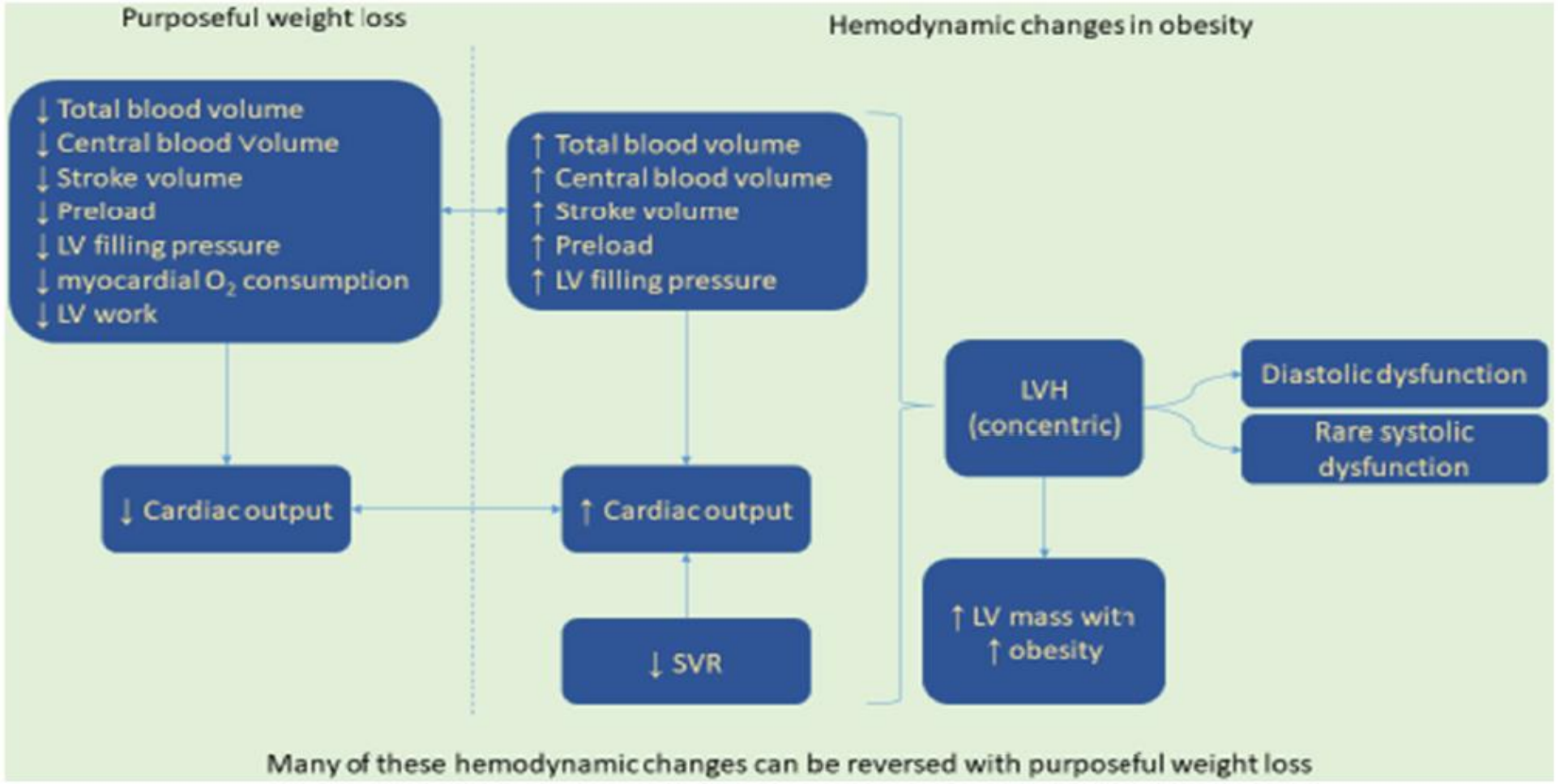
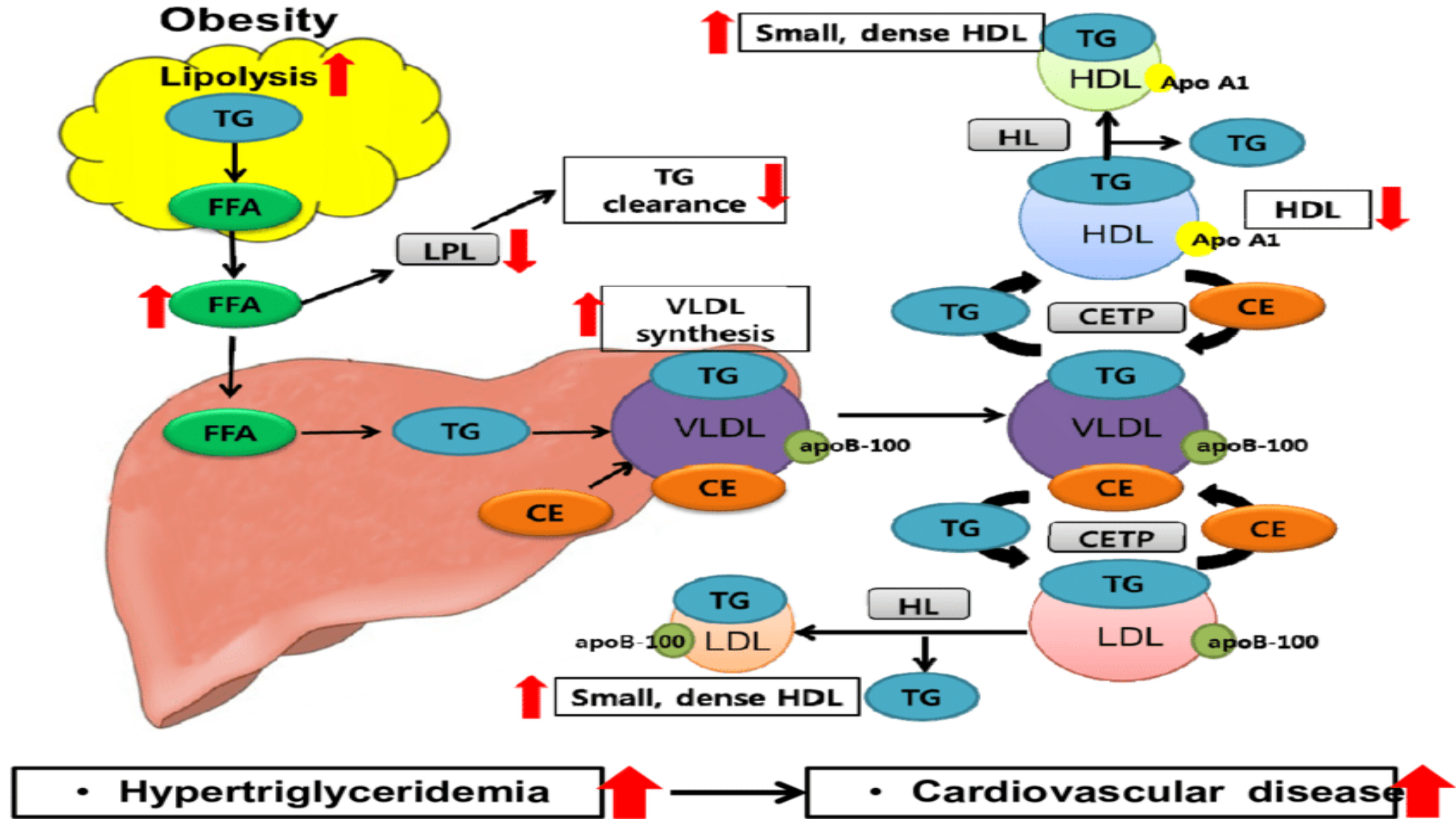


Fig. 2. Hemodynamic changes in obesity. LV = Left ventricular; LHV = Left ventricular hypertrophy; SVR = Systemic vascular resistance.

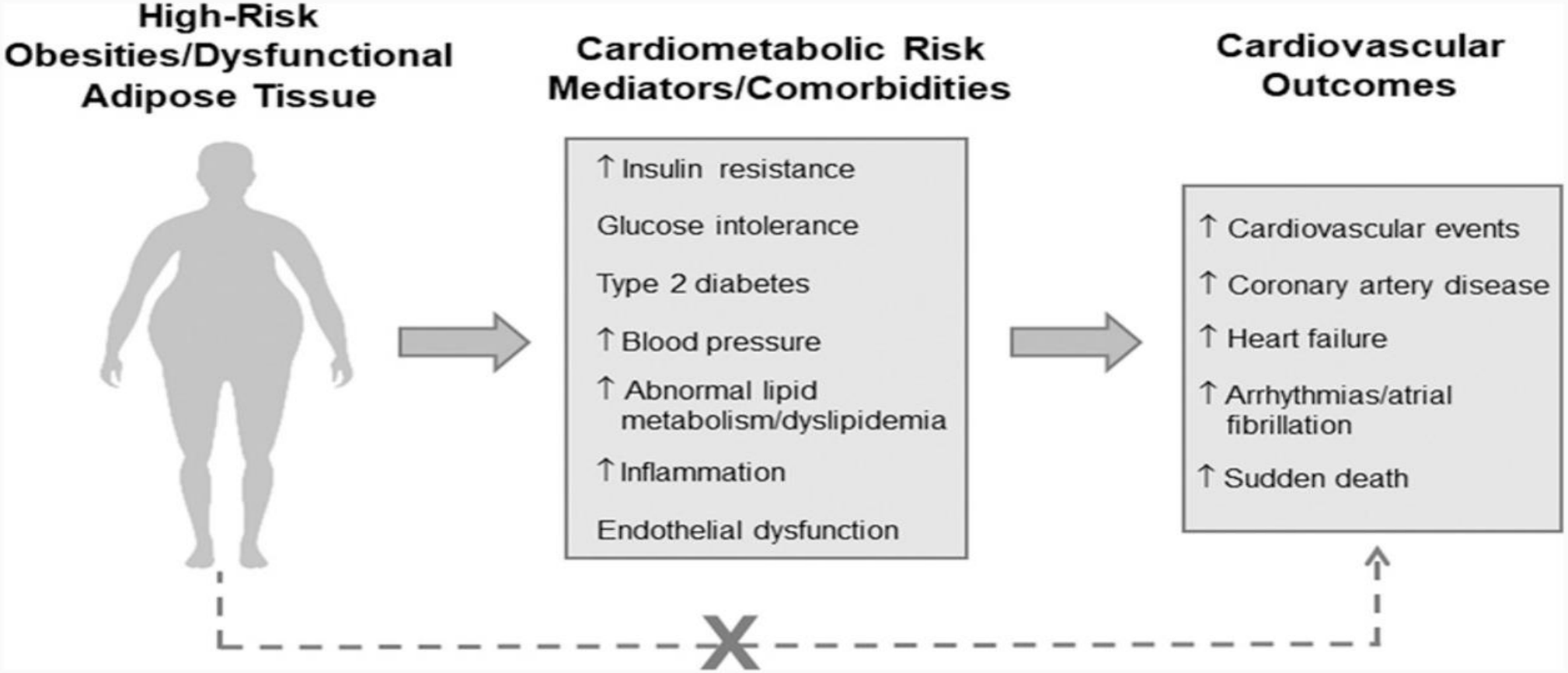

Obesity
Lipolysis
TG
FFA
LPL
TG clearance
FFA
FFA
TG
VLDL synthesis
TG
VLDL
apoB-100
CE
CE
Small, dense HDL
TG
HDL
Apo A1
HL
TG
TG
HDL
Apo A1
HDL
TG
CETP
CE
TG
VLDL
apoB-100
CE
TG
CETP
CE
TG
LDL
apoB-100
HL
TG
apoB-100
LDL
Small, dense HDL
TG
• Hypertriglyceridemia
• Cardiovascular disease

Circulation Research
Volume 126, Issue 11, 2020; Pages 1477-1500
https://doi.org/10.1161/CIRCRESAHA.120.316101

OBESITY, METABOLIC SYNDROME, AND CVD COMPENDIUM

Obesity Phenotypes, Diabetes, and Cardiovascular Diseases

Marie-Eve Piché, André Tchernof, and Jean-Pierre Després



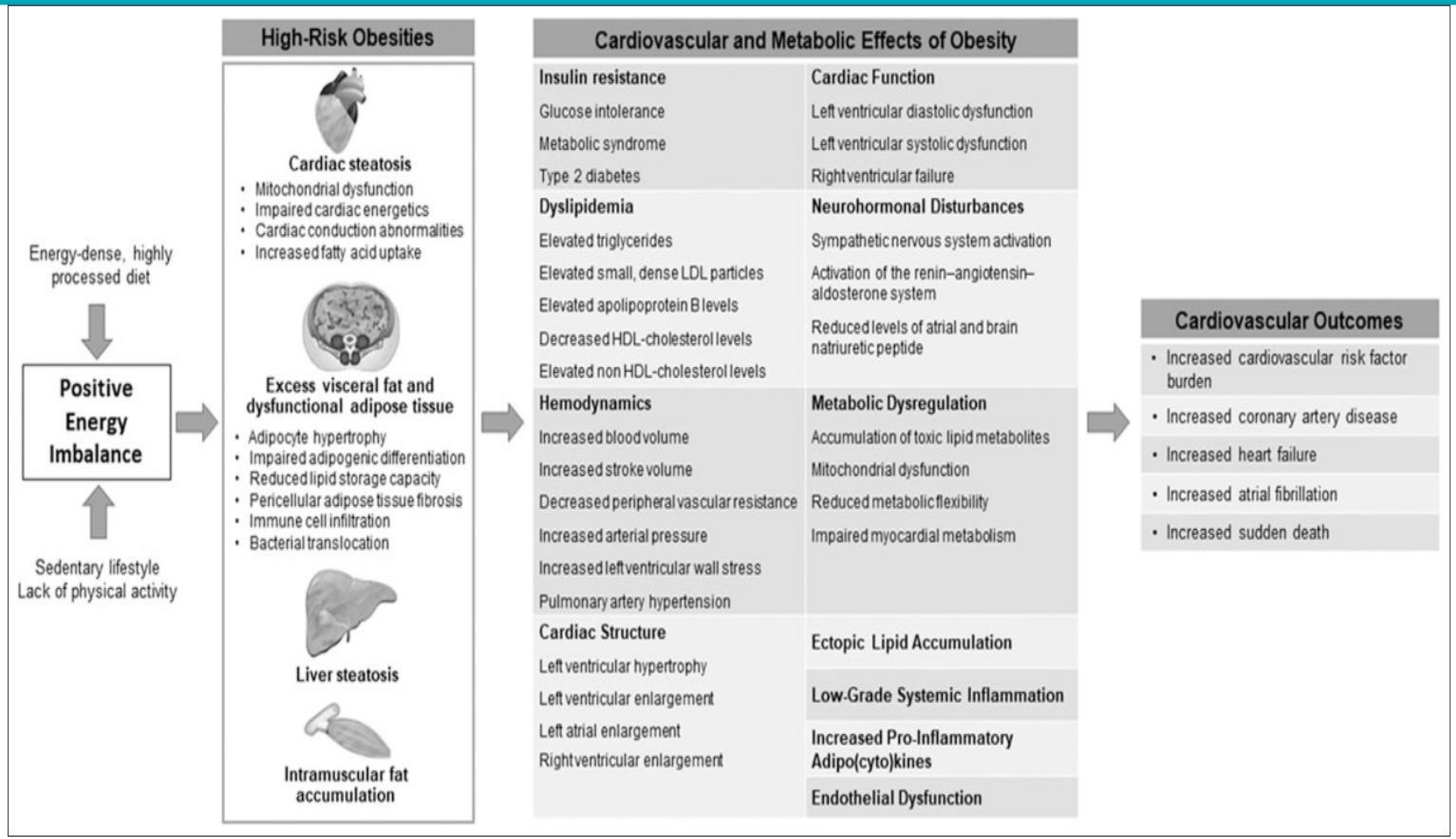


Figure 5. Cardiovascular and metabolic consequences of high-risk obesities. The inherited inability of some low-risk adipose depots to store excess triglycerides is an important driver of excessive adipose tissue accumulation in visceral and ectopic sites. The related metabolic alterations impact negatively the cardiovascular system, increasing the risk of coronary artery disease, heart failure, atrial arrhythmias, and sudden death. HDL indicates high-density lipoprotein; and LDL, low-density lipoprotein.

Παχυσαρκία & Θρόμβωση

Eur J Vasc Endovasc Surg 33, 223–233 (2007)
doi:10.1016/j.ejvs.2006.10.006, available online at http://www.sciencedirect.com on ScienceDirect

REVIEW

Obesity and Thrombosis

K.A.L. Darvall,[1*] R.C. Sam,[2] S.H. Silverman,[1] A.W. Bradbury[2] and D.J. Adam[2]

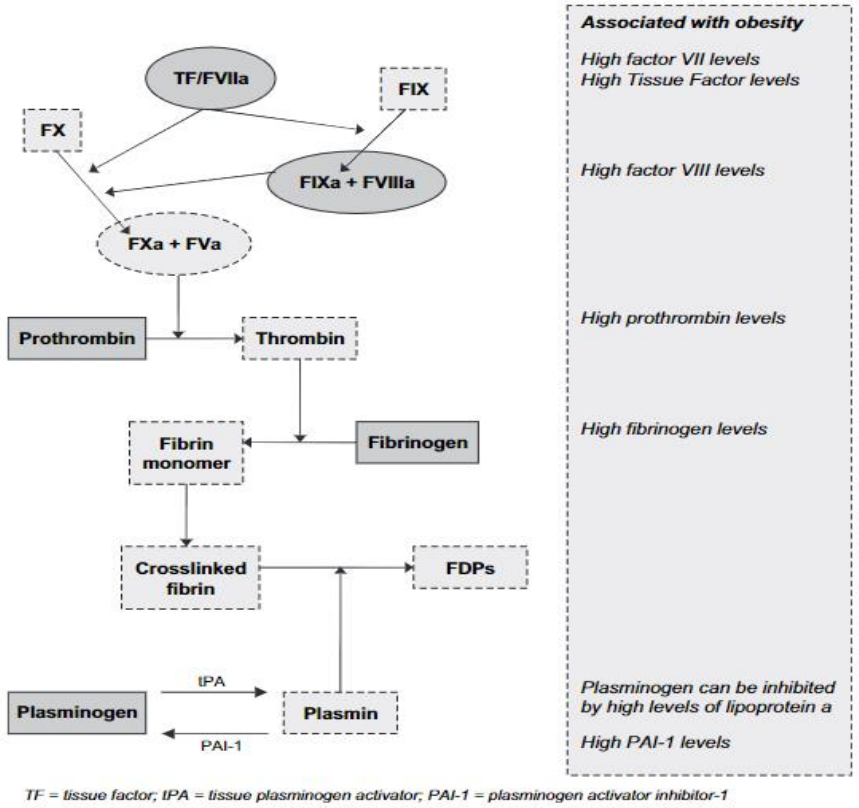


Fig. 1. Obesity and the coagulation cascade: the different pathways in the coagulation and fibrinolytic cascades that may be affected by obesity.

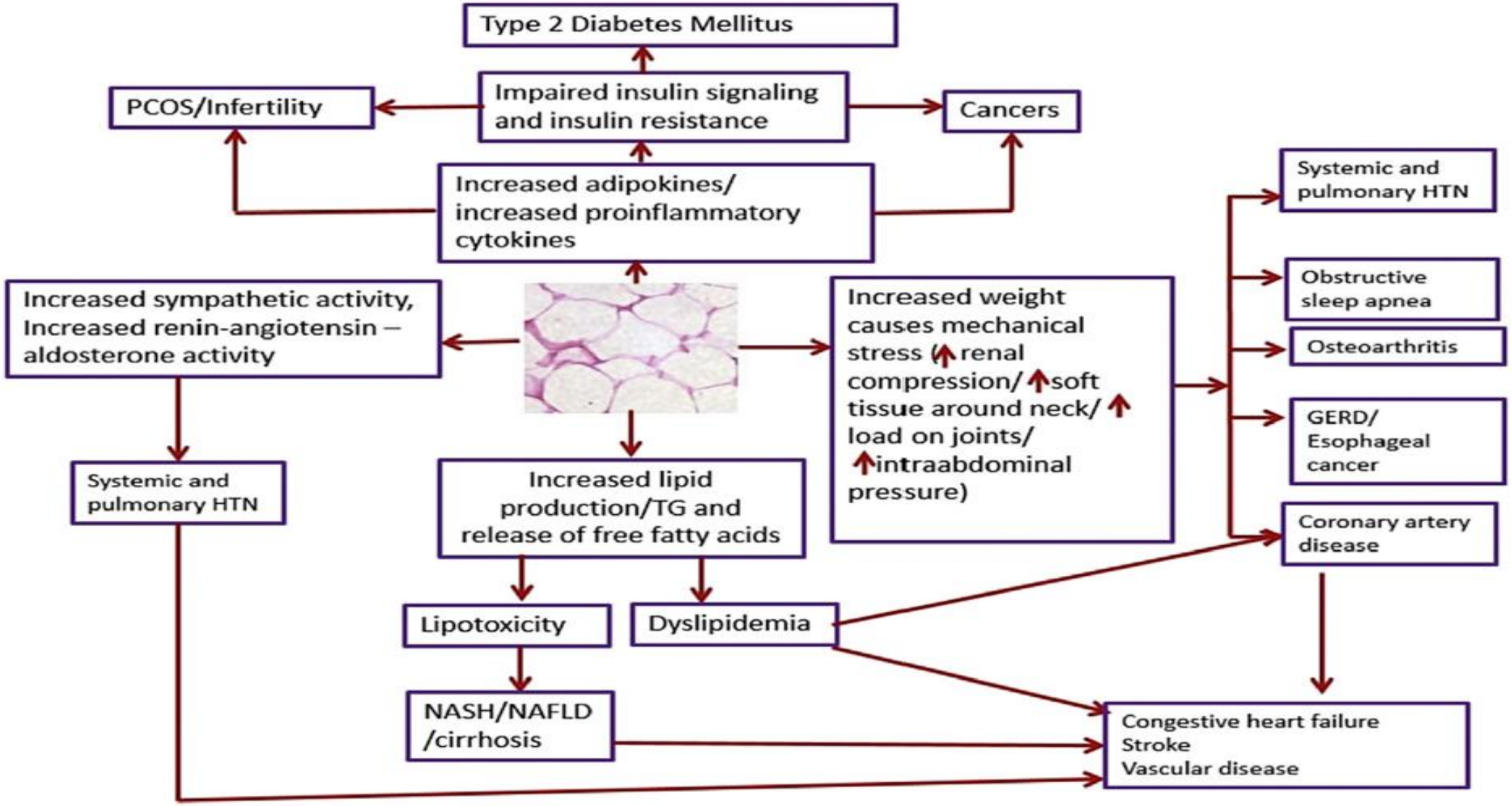
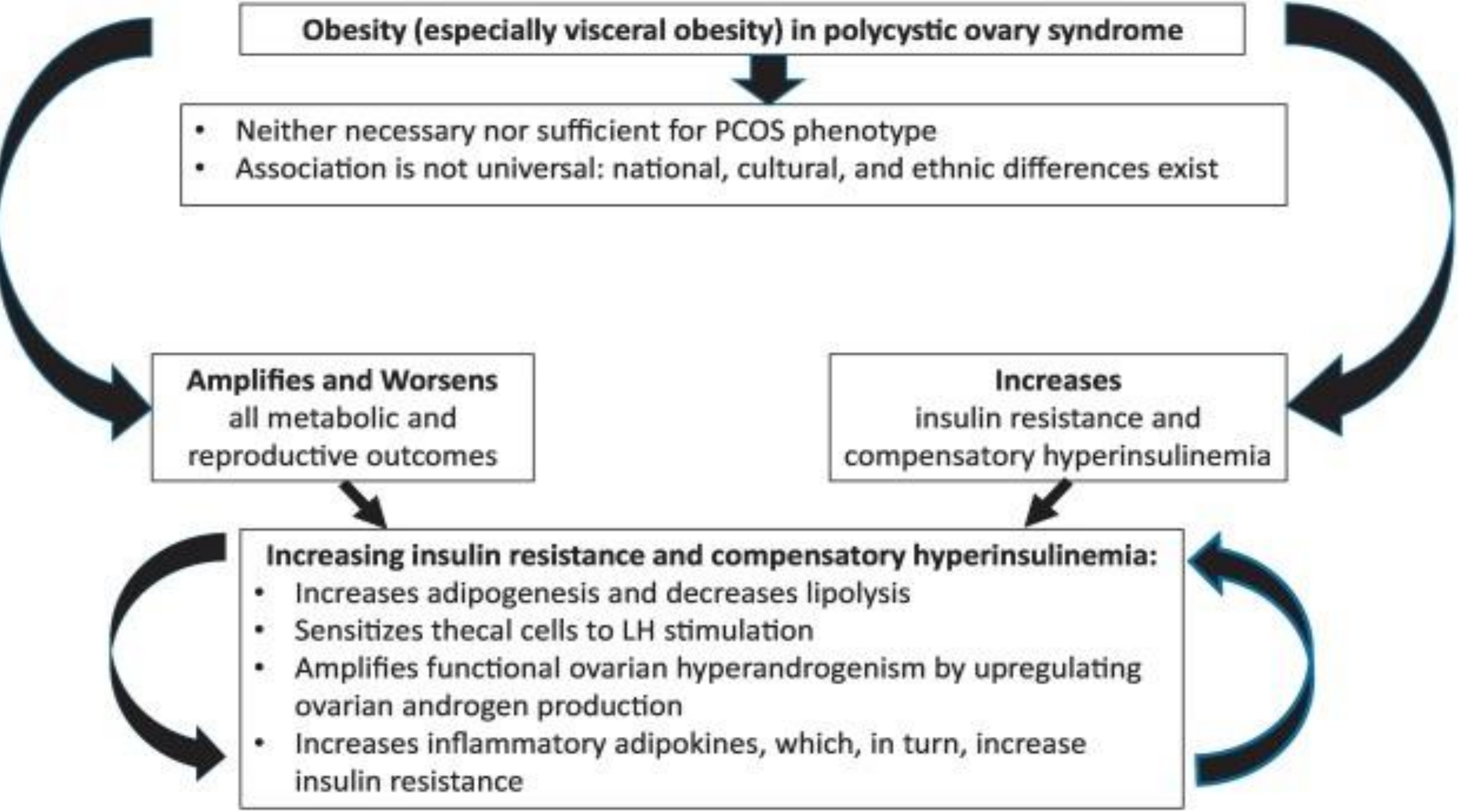


Fig. 2. Pathways through which obesity leads to comorbidities. GERD, Gastroesophageal reflux disease; HTN, Hypertension; NAFLD, Nonalcoholic fatty liver disease; NASH, Nonalcoholic steatohepatitis.


Obesity (especially visceral obesity) in polycystic ovary syndrome
• Neither necessary nor sufficient for PCOS phenotype
• Association is not universal: national, cultural, and ethnic differences exist
Amplifies and Worsens
all metabolic and
reproductive outcomes
Increases
insulin resistance and
compensatory hyperinsulinemia
Increasing insulin resistance and compensatory hyperinsulinemia:
• Increases adipogenesis and decreases lipolysis
• Sensitizes thecal cells to LH stimulation
• Amplifies functional ovarian hyperandrogenism by upregulating ovarian androgen production
• Increases inflammatory adipokines, which, in turn, increase insulin resistance

CHEST Postgraduate Education Corner
CONTEMPORARY REVIEWS IN SLEEP MEDICINE

Interactions Between Obesity and Obstructive Sleep Apnea

Implications for Treatment

Abel Romero-Corral, MD, MSc; Sean M. Caples, DO; Francisco Lopez-Jimenez, MD, MSc; and Virend K. Somers, MD, PhD, FCCP

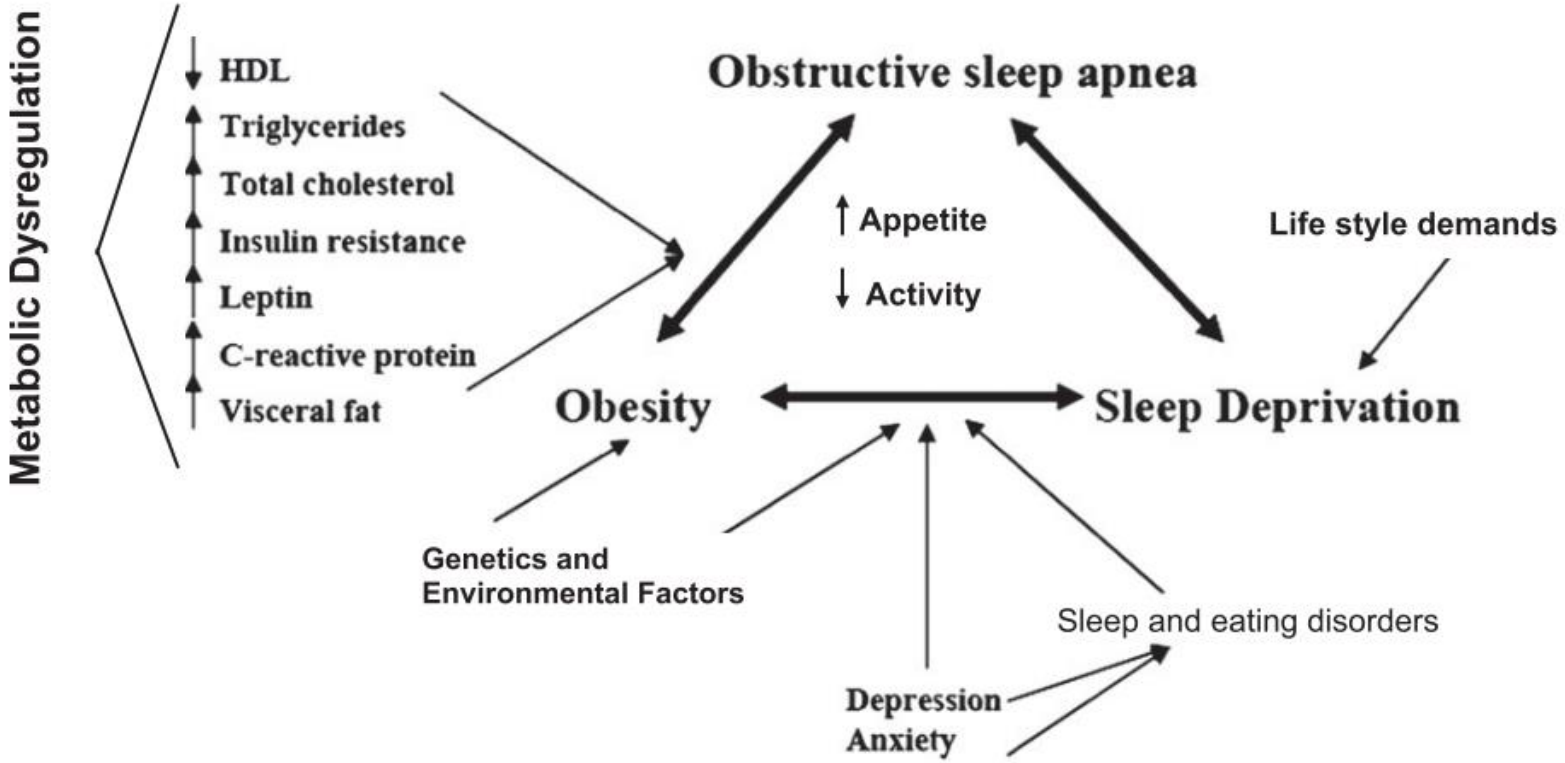


FIGURE 1. Interaction between obstructive sleep apnea, obesity, sleep deprivation, and metabolic abnormalities. HDL = high-density lipoprotein.

Contents lists available at ScienceDirect

Metabolism Clinical and Experimental

journal homepage: www.metabolismjournal.com

Obesity and cancer risk: Emerging biological mechanisms and perspectives

Konstantinos I. Avgerinos [a,1], Nikolaos Spyrou [a,1], Christos S. Mantzoros [b], Maria Dalamaga [c,*]

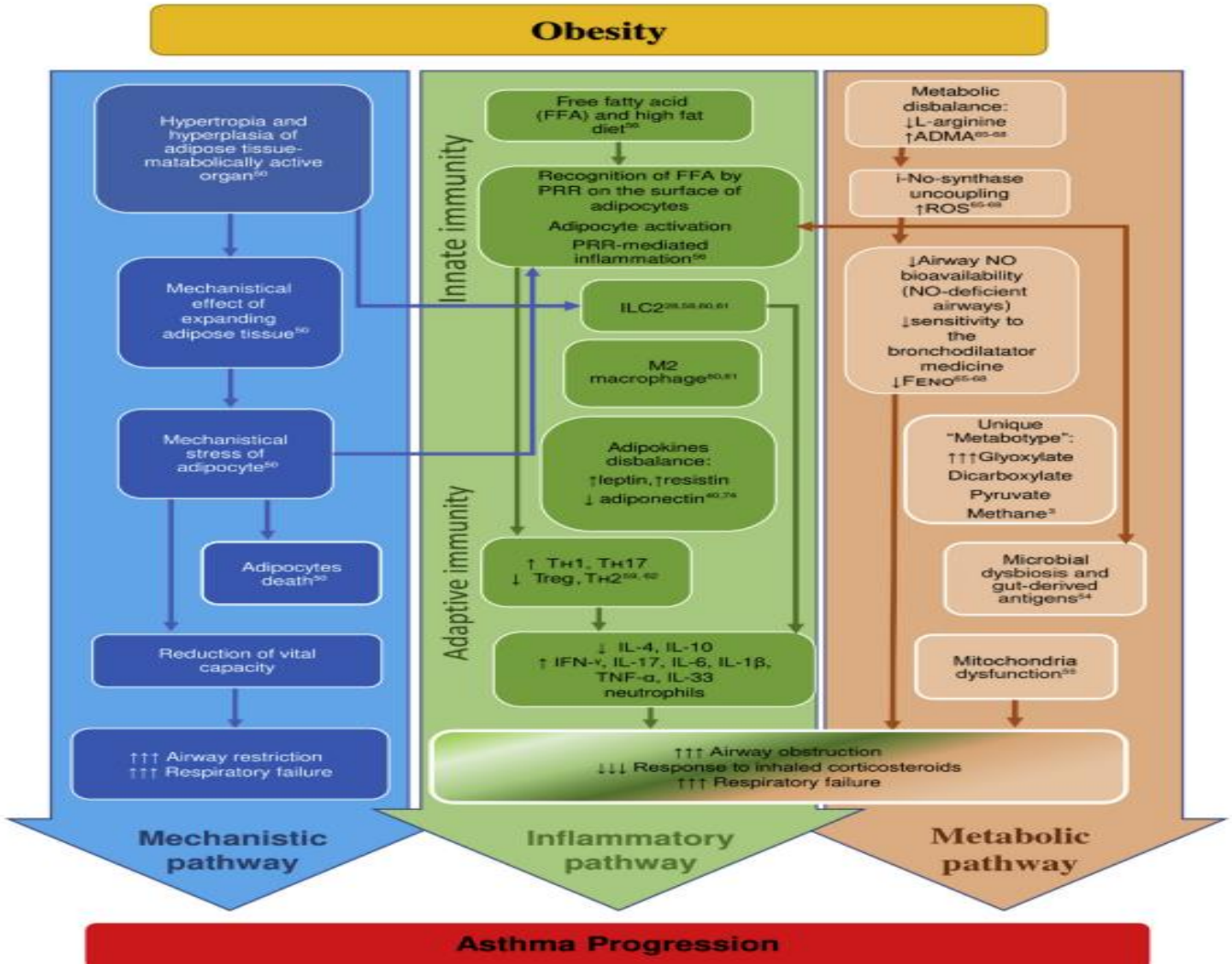
Highlights

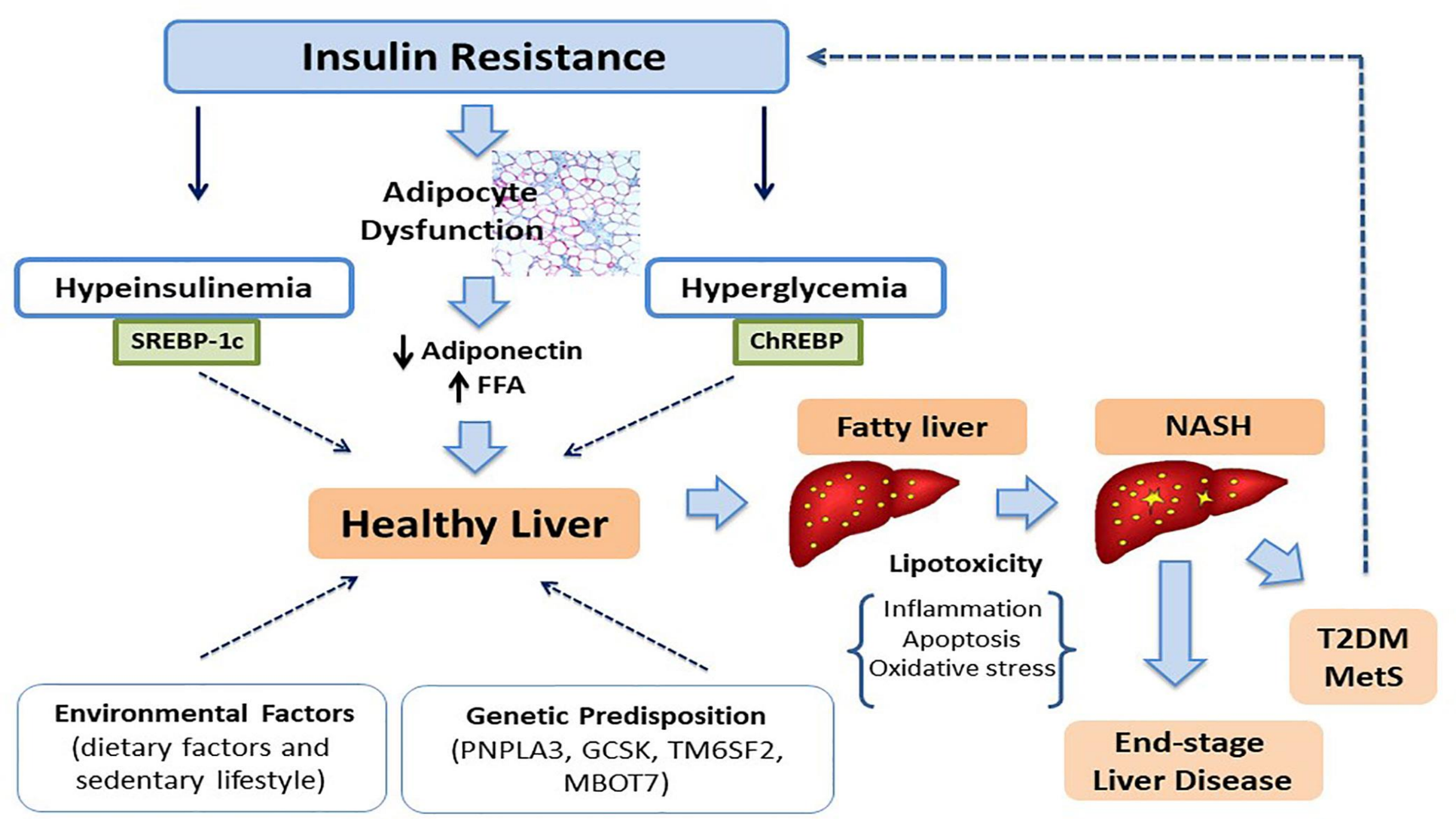
- Excess body weight is associated with an increased risk for a range of malignancies.
- The triad of obesity, insulin resistance and adipokine aberrations is linked to cancer.
- Subclinical chronic low-grade inflammation and oxidative stress is another mechanism.
- Altered gut microbiome contributes to inflammation and release of carcinogenic products.
- Microenvironmental perturbations and circadian rhythm disruption are additional mechanisms.

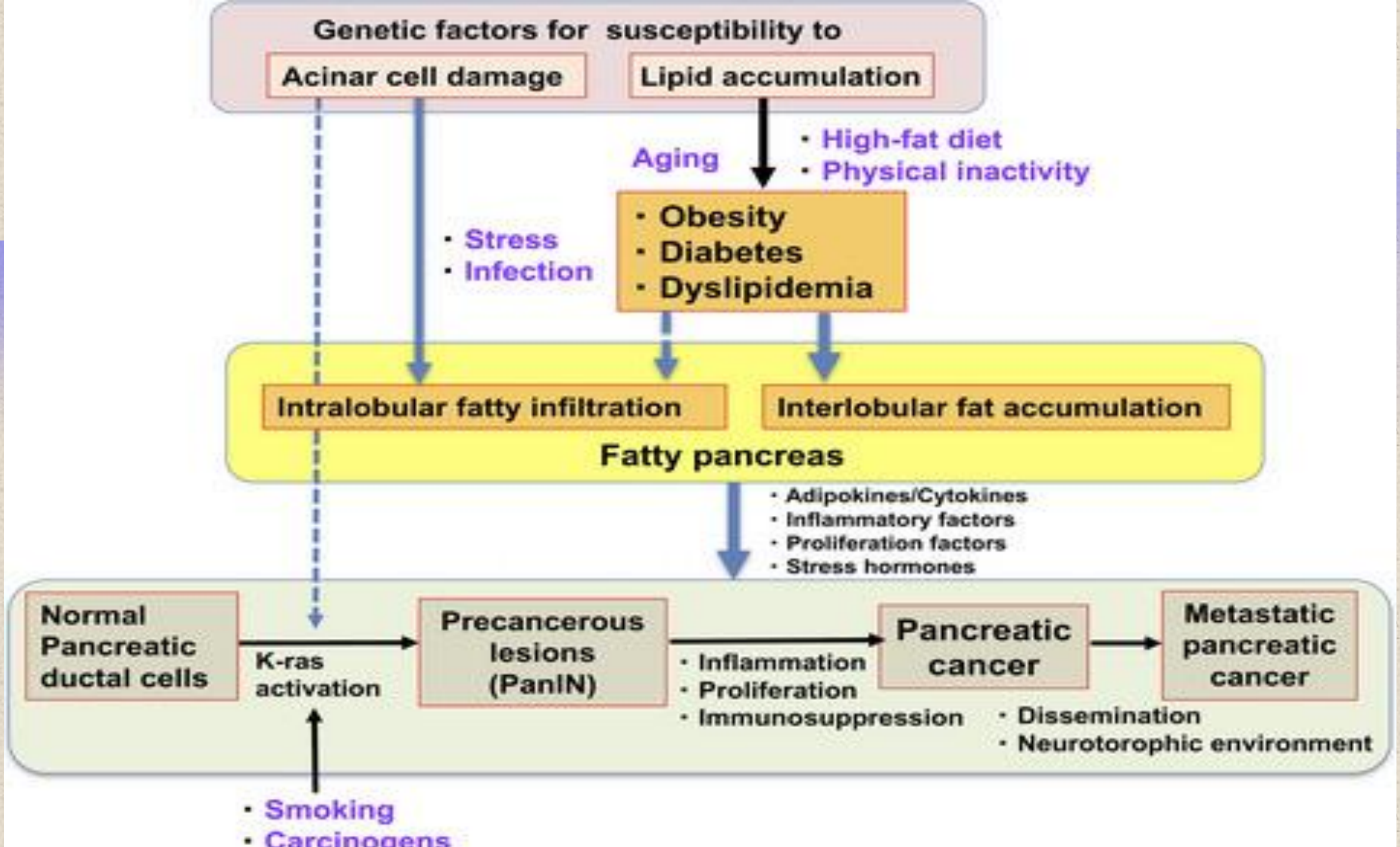
Clinical reviews in allergy and immunology

Obesity and asthma

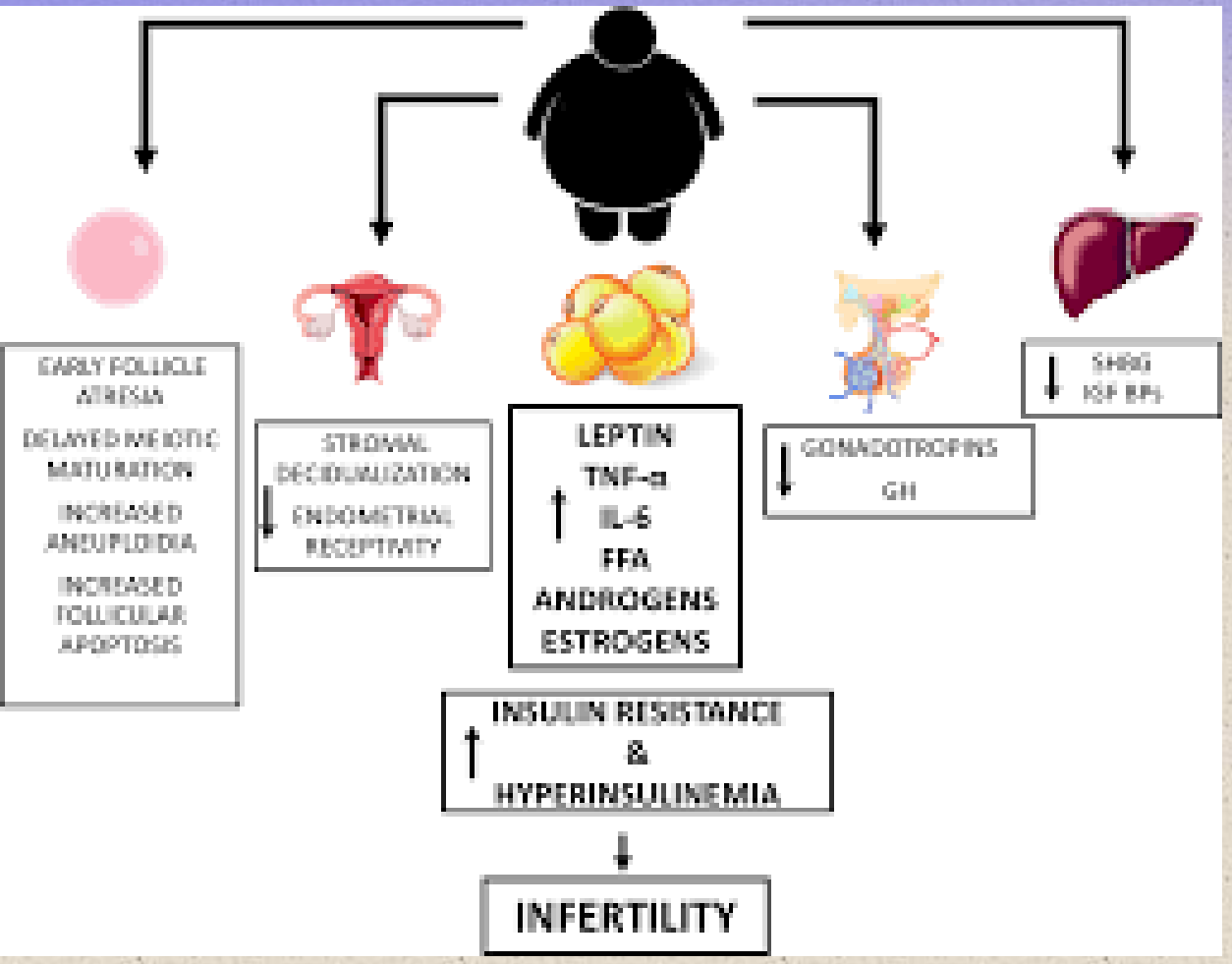
Sarah Miethe, MSc,[a]* Antonina Karsonova, MD, PhD,[b]* Alexander Karaulov, MD, PhD, DMedSc,[b]‡ and Harald Renz, MD[a,c]‡ *Marburg, Germany, and Moscow, Russia*

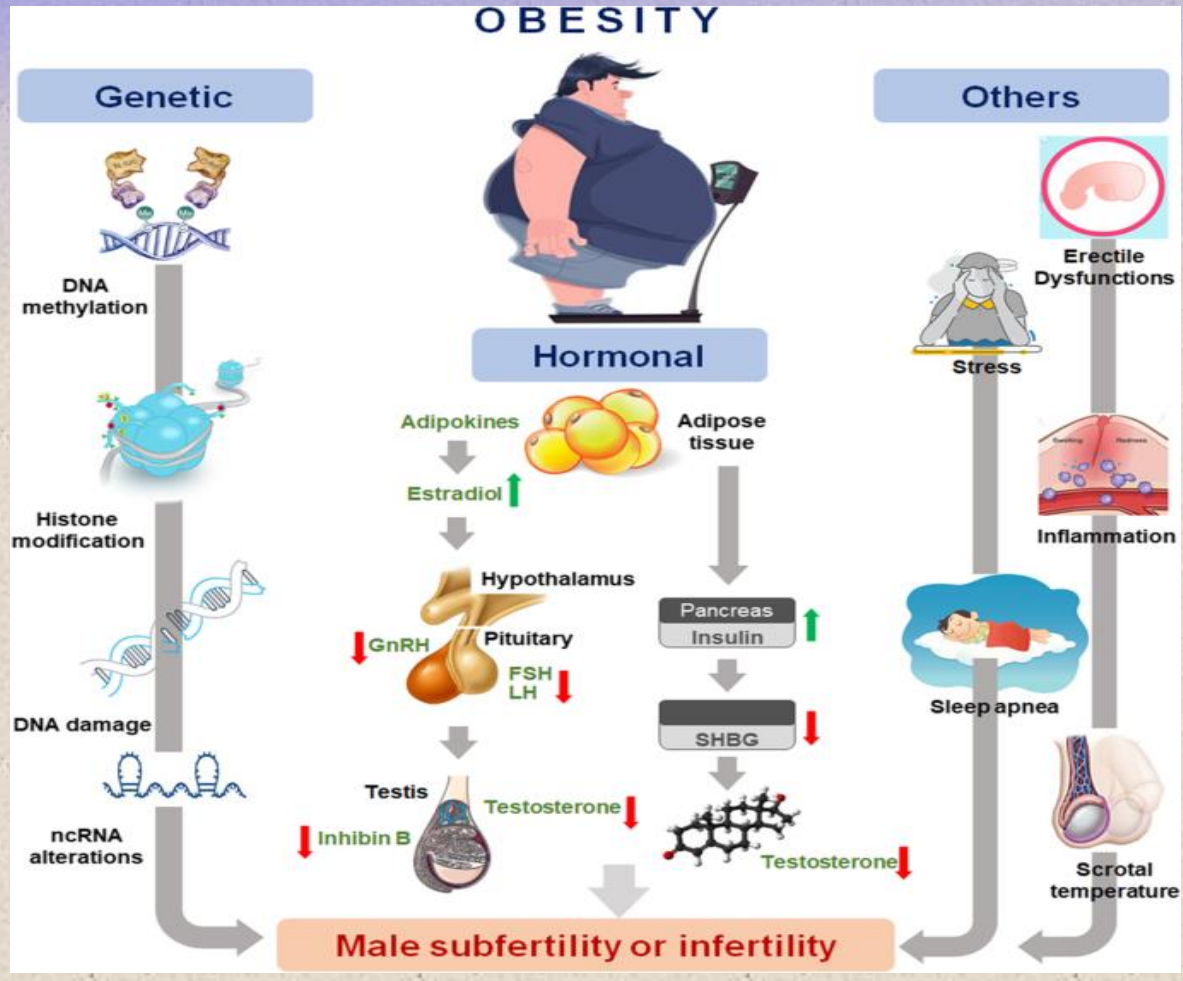



Insulin Resistance
Adipocyte Dysfunction
Hypeinsulinemia
SREBP-1c
Hyperglycemia
ChREBP
↓ Adiponectin
↑ FFA
Healthy Liver
Fatty liver
NASH
Lipotoxicity
Inflammation
Apoptosis
Oxidative stress
T2DM
MetS
End-stage
Liver Disease
Environmental Factors
(dietary factors and
sedentary lifestyle)
Genetic Predisposition
(PNPLA3, GCSK, TM6SF2,
MBOT7)


Genetic factors for susceptibility to
Acinar cell damage
Lipid accumulation
Aging
• High-fat diet
• Physical inactivity
• Stress
• Infection
• Obesity
• Diabetes
• Dyslipidemia
Intralobular fatty infiltration
Interlobular fat accumulation
Fatty pancreas
• Adipokines/Cytokines
• Inflammatory factors
• Proliferation factors
• Stress hormones
Normal Pancreatic ductal cells
K-ras activation
Precancerous lesions (PanIN)
• Inflammation
• Proliferation
• Immunosuppression
Pancreatic cancer
Metastatic pancreatic cancer
• Dissemination
• Neurotorophic environment
• Smoking
• Carcinogens

Παχυσαρκία & Υπογονιμότητα





Παχυσαρκία & Μυοσκελετικά

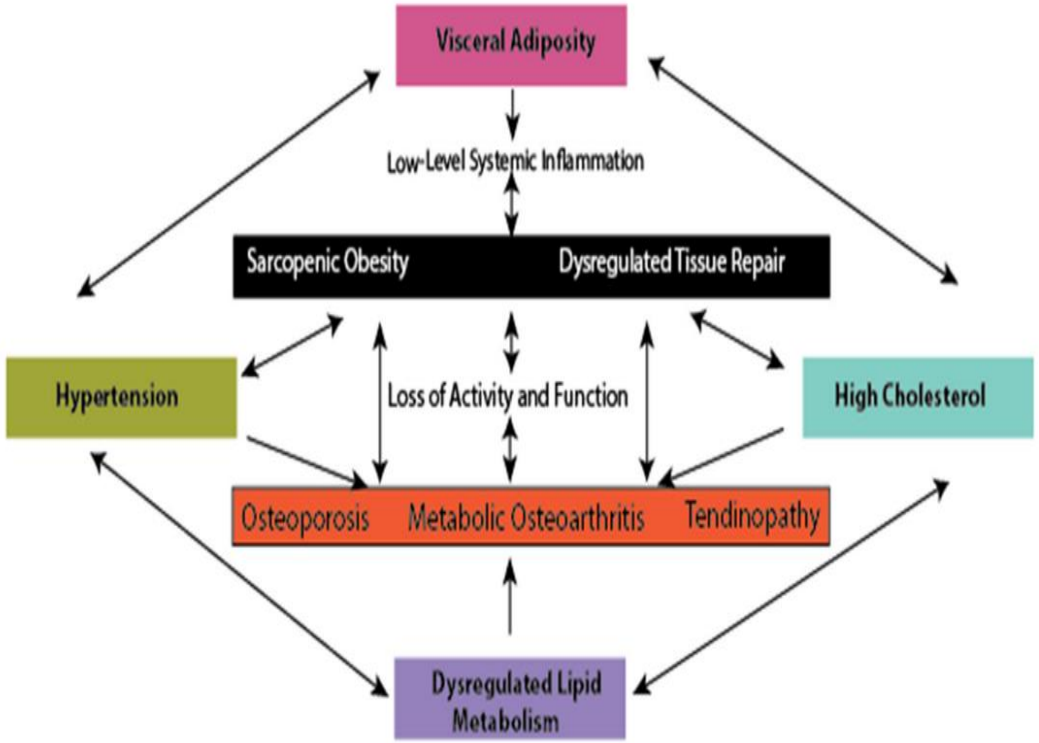


FIGURE 1 | The interface between metabolic complications and musculoskeletal compromise.

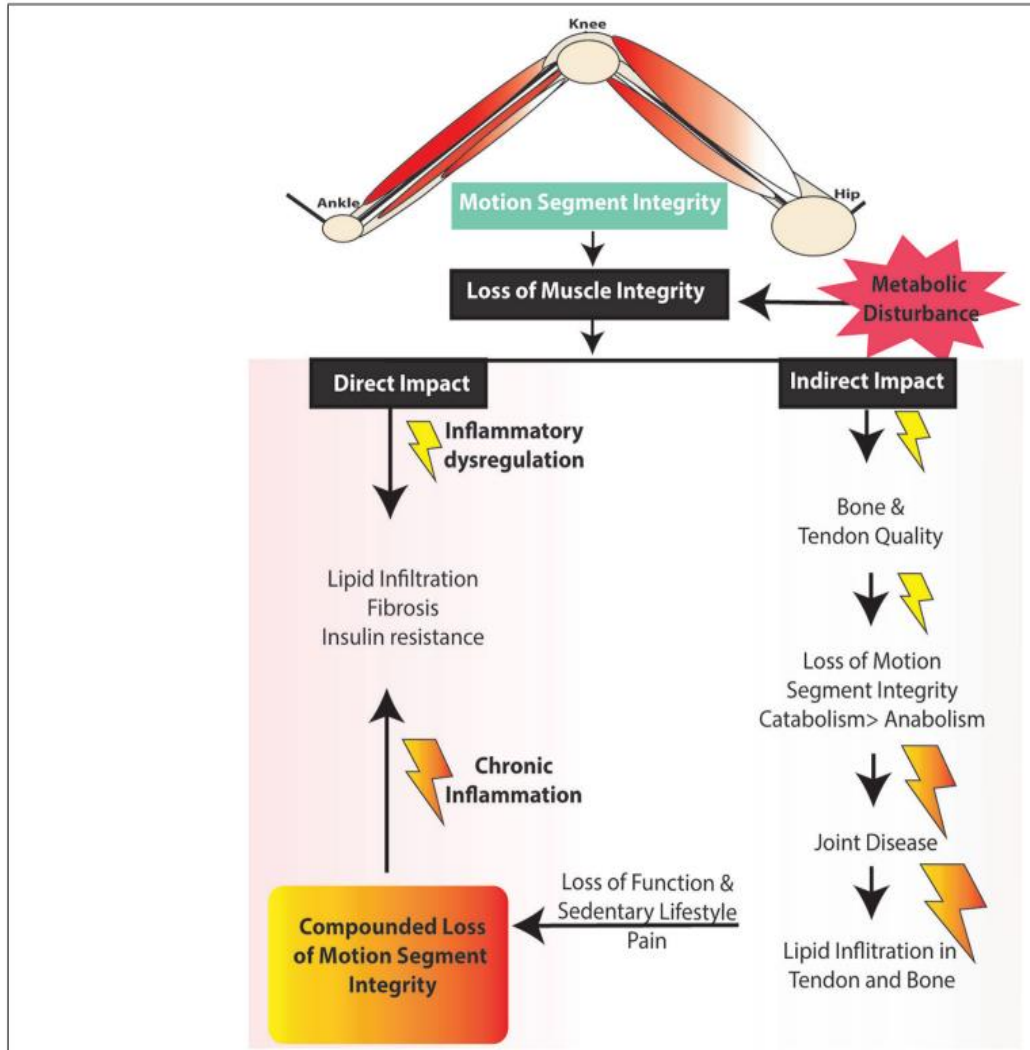
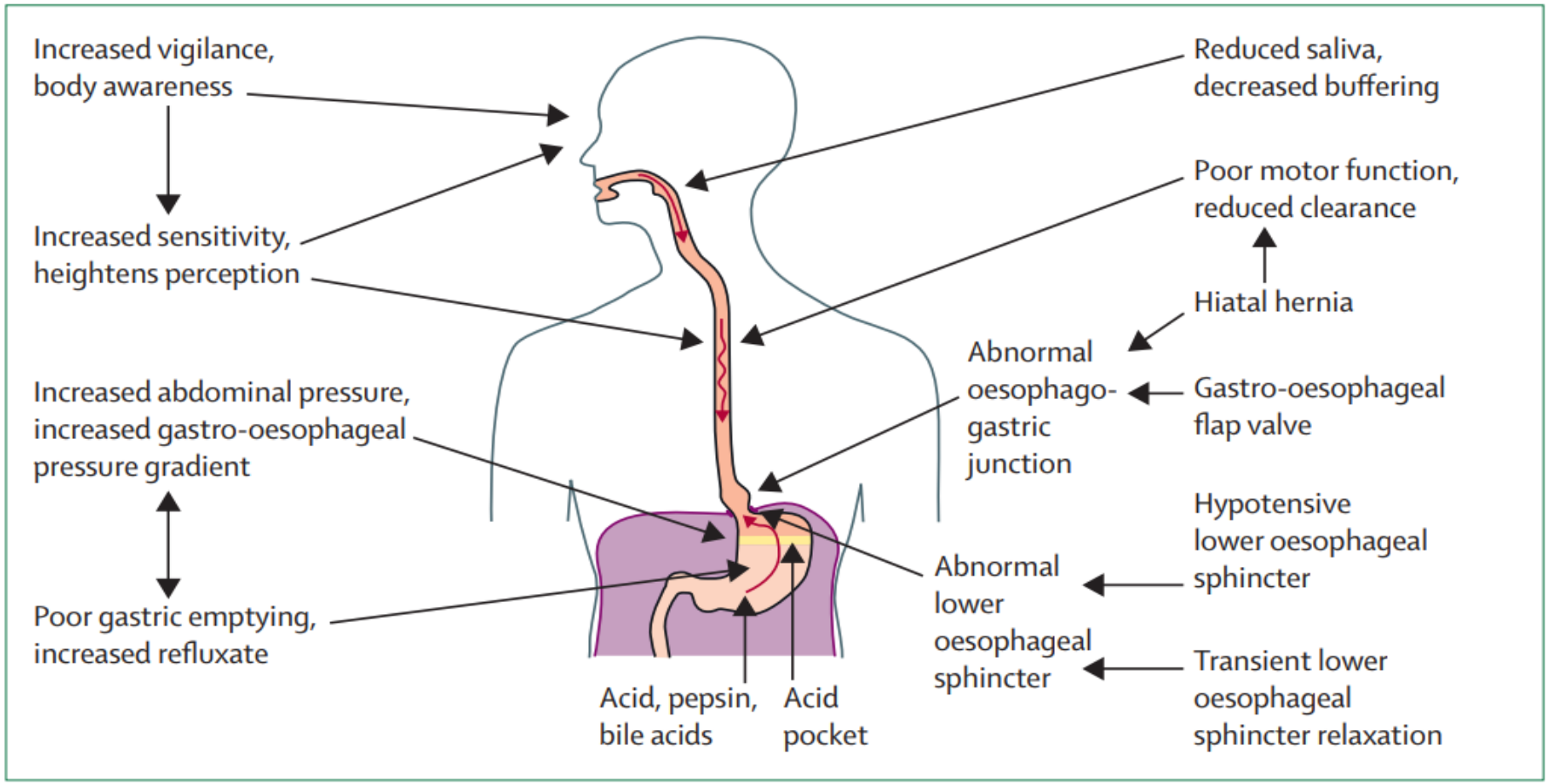


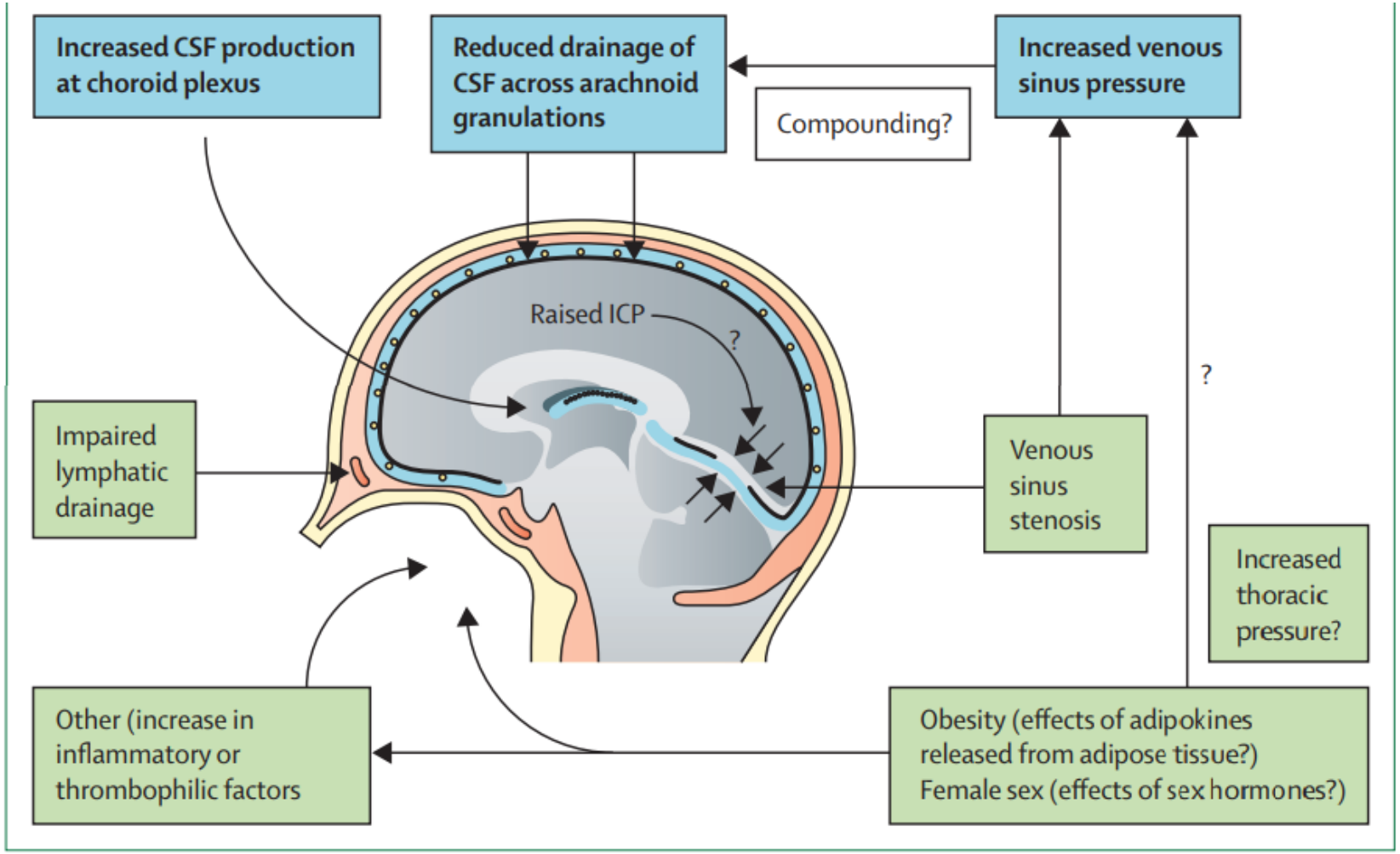
FIGURE 2 | Potential impact of changes in muscle damage on lower limb motion segment integrity.

Παχυσαρκία & Γαστροοισοφαγική παλινδρόμηση



Understanding idiopathic intracranial hypertension: mechanisms, management, and future directions

Keira A Markey, Susan P Mollan, Rigmor H Jensen, Alexandra J Sinclair

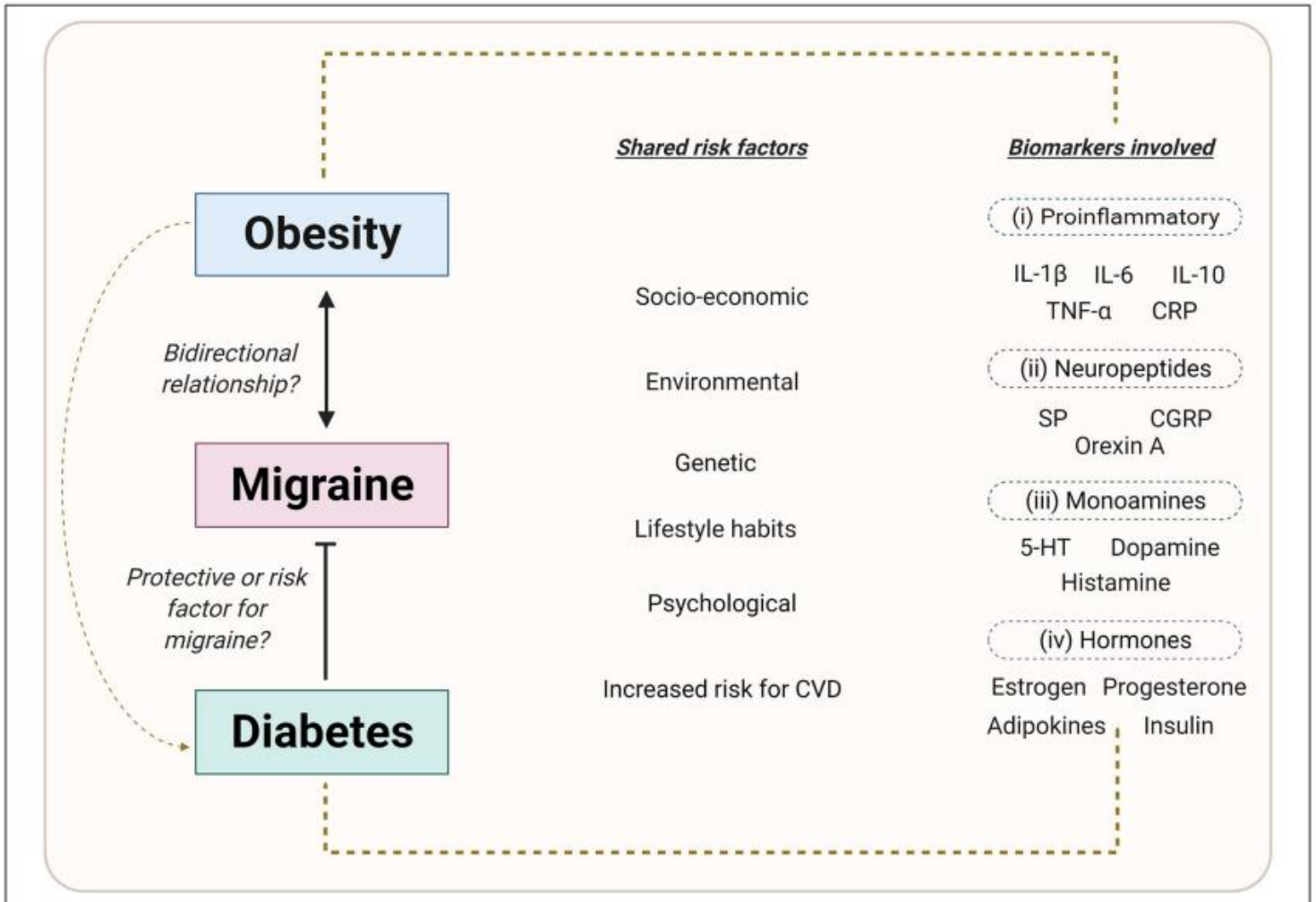


frontiers in Neurology

REVIEW
published: 09 June 2021
doi: 10.3389/fneur.2021.686398

Metabolic Aspects of Migraine: Association With Obesity and Diabetes Mellitus

*Eduardo Rivera-Mancilla[1], Linda Al-Hassany[1], Carlos M. Villalón[2] and Antoinette MaassenVanDenBrink[1]**

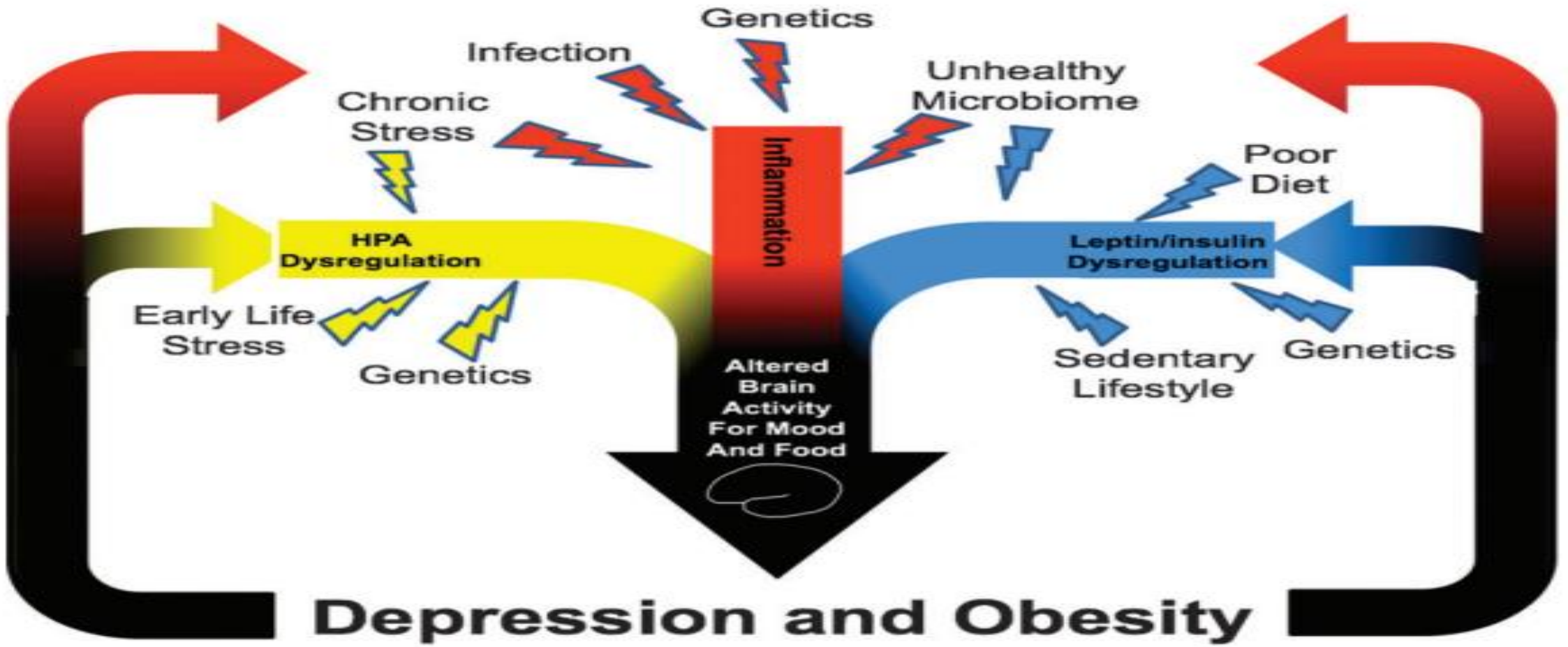


Molecular Psychiatry (2019) 24:18–33
https://doi.org/10.1038/s41380-018-0017-5

EXPERT REVIEW

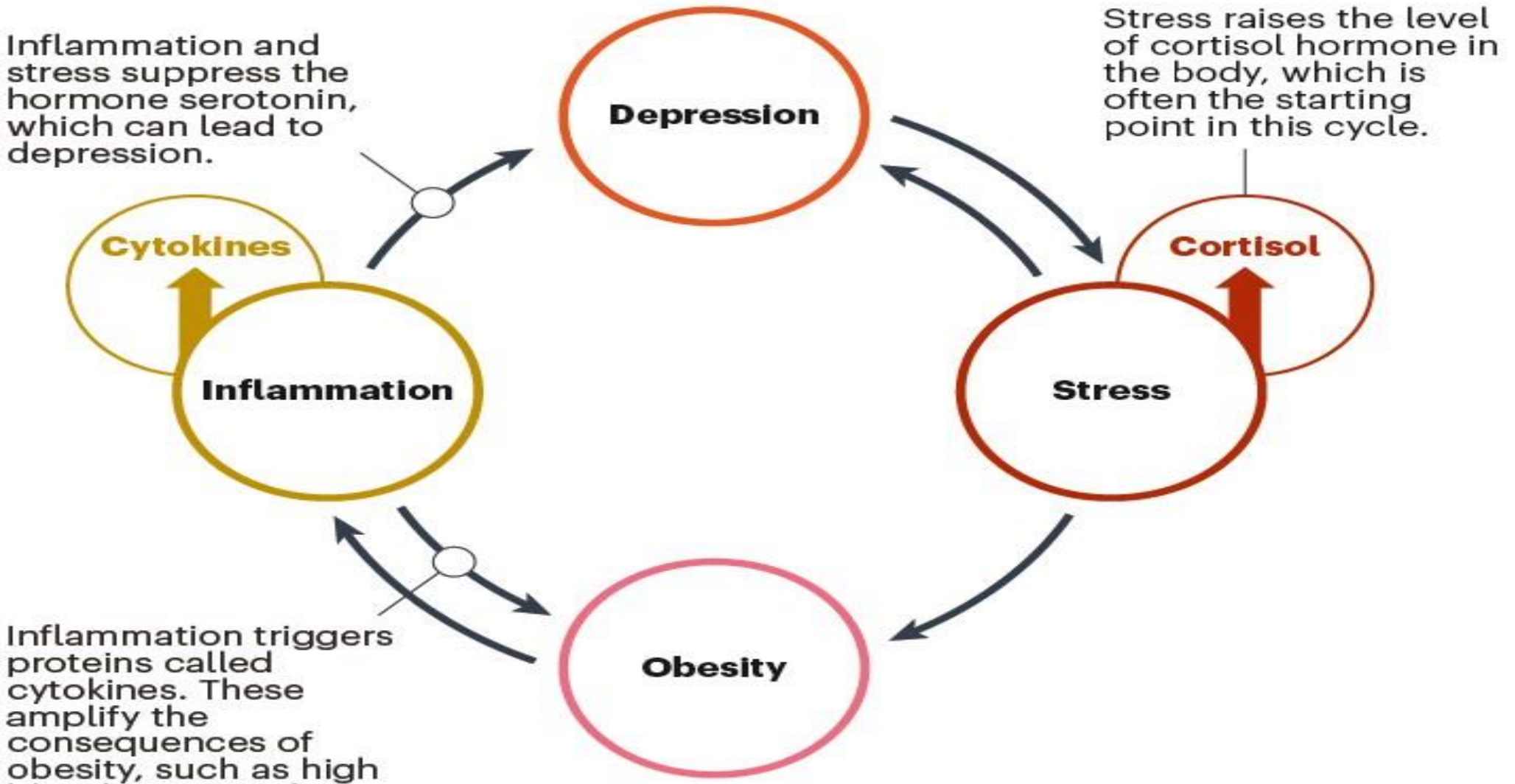
Depression and obesity: evidence of shared biological mechanisms

Yuri Milaneschi[1] · W. Kyle Simmons[2,3] · Elisabeth F. C. van Rossum[4] · Brenda WJH Penninx[1]



VICIOUS CYCLES

Depression, obesity and stress are biochemically linked, with each tending to increase the risk and severity of the others. Inflammation has a role in both depression and obesity.



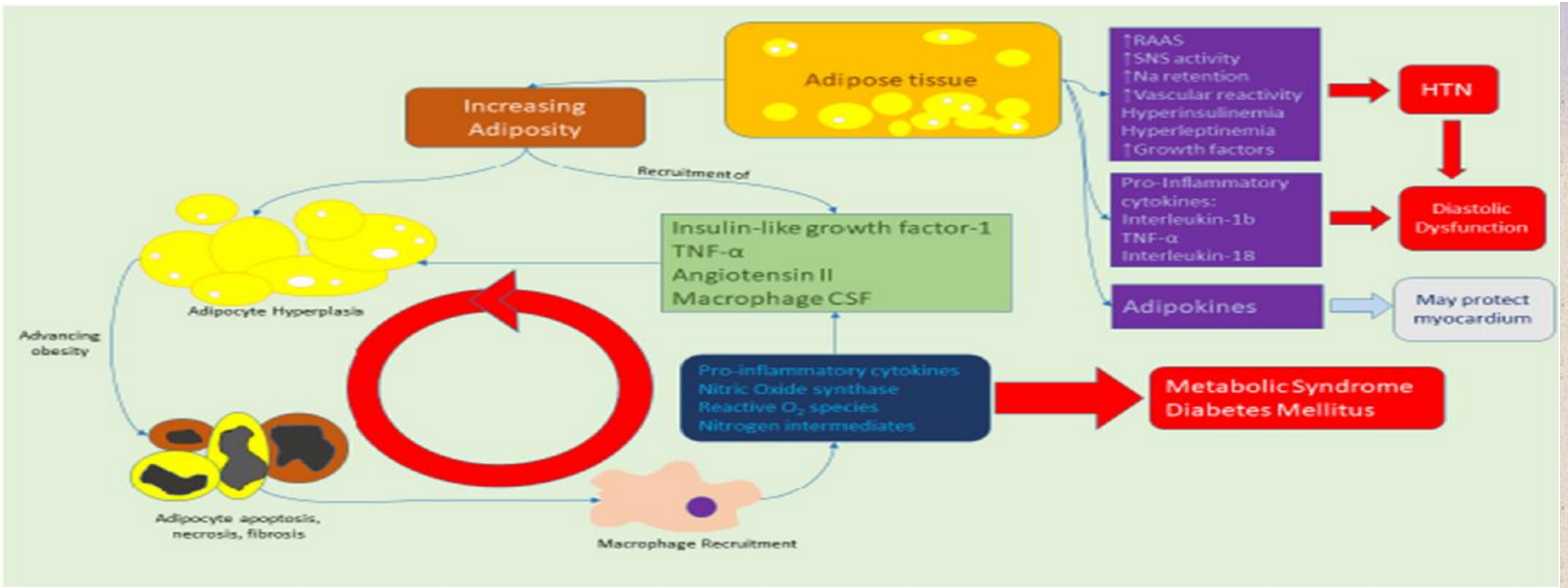
Contents lists available at ScienceDirect

Progress in Cardiovascular Diseases

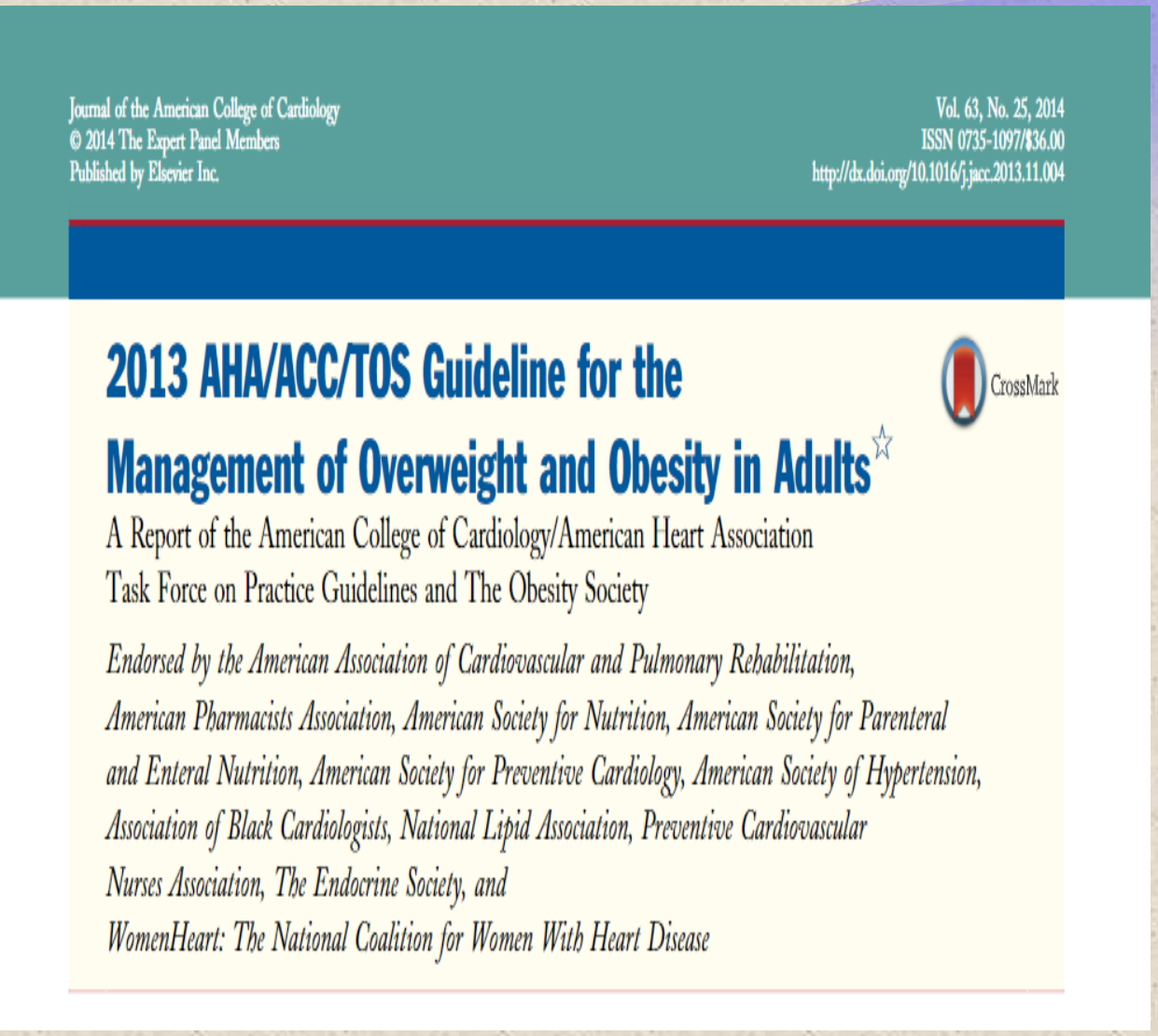
journal homepage: www.onlinepcd.com

An Overview and Update on Obesity and the Obesity Paradox in Cardiovascular Diseases ☆

Andrew Elagizi [a], Sergey Kachur [a], Carl J. Lavie [a,*], Salvatore Carbone [b], Ambarish Pandey [c], Francisco B. Ortega [d], Richard V. Milani [a]



Διαχείριση Ασθενών με παχυσαρκία



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Vol. 63, No. 25, 2014
ISSN 0735-1097/$36.00
http://dx.doi.org/10.1016/j.jacc.2013.11.004

2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults☆

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Pharmacists Association, American Society for Nutrition, American Society for Parenteral and Enteral Nutrition, American Society for Preventive Cardiology, American Society of Hypertension, Association of Black Cardiologists, National Lipid Association, Preventive Cardiovascular Nurses Association, The Endocrine Society, and WomenHeart: The National Coalition for Women With Heart Disease

Η διαχείριση των ασθενών με παχυσαρκία είναι η έμφαση σε μια παρέμβαση τρόπου ζωής που περιλαμβάνει αλλαγές στη διατροφή, αύξηση της σωματικής δραστηριότητας και τροποποίηση συμπεριφοράς. Φαρμακοθεραπεία, ιατρικές συσκευές και η βαριατρική χειρουργική είναι επιλογές για ασθενείς που χρειάζονται πρόσθετες παρεμβάσεις.

nutrients

MDPI

Review

Nutrition Concepts for the Treatment of Obesity in Adults

Meike Wiechert and Christina Holzapfel *

Figure 2. Overview of weight management goals.

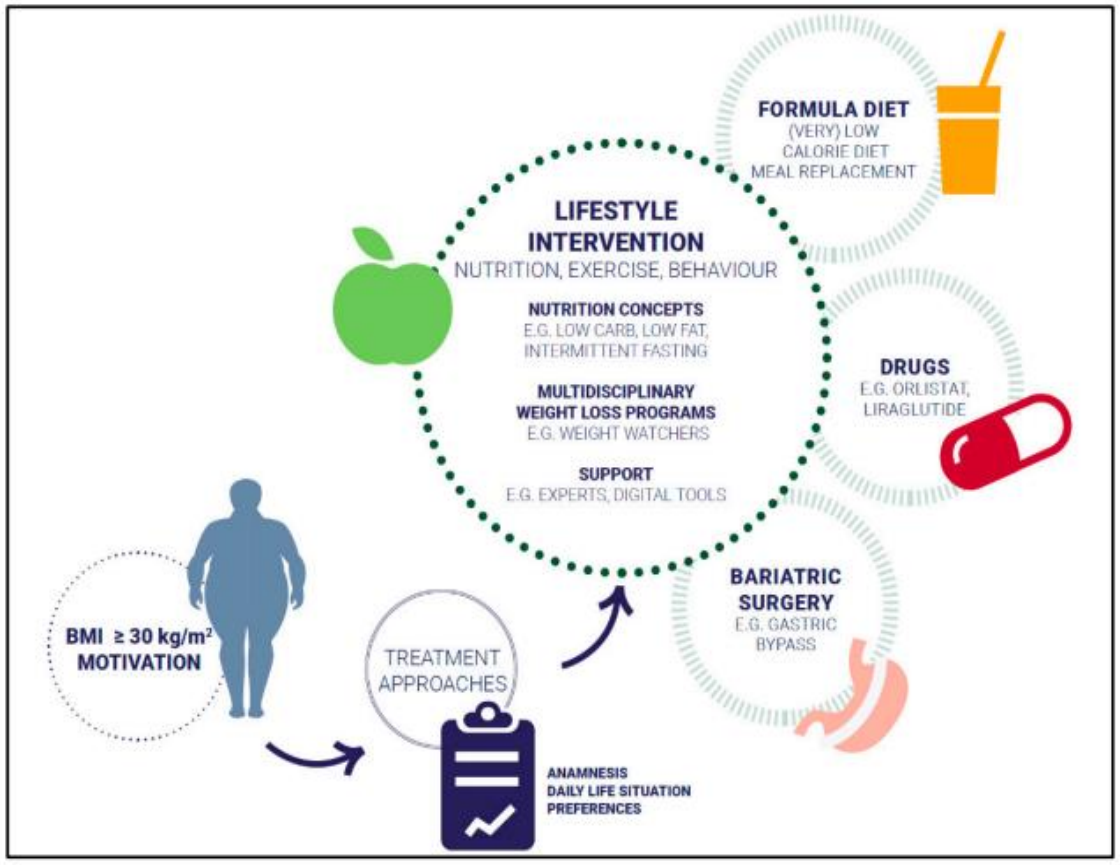


Figure 1. Treatment approaches.

Φάρμακα

TABLE 1 Drugs for Long-Term Weight Management

Drug	1-Year Weight Loss, Placebo-Subtracted	Adverse Events	Precautions	Contraindications and Limitations
Orlistat	~3%	Fecal urgency, fecal incontinence, flatus with discharge, oily spotting	Daily multivitamin to make up for malabsorption of fat-soluble vitamins	Chronic malabsorption syndrome; cholestasis
Lorcaserin	3.0% to 3.6%	Headache, dizziness, fatigue, nausea, dry mouth, constipation, cough, hypoglycemia in patients with diabetes	Monitor for symptoms of serotonin toxicity. Monitor for signs and symptoms of valvular heart disease.	Safety of use in patients taking antidepressants is unknown
Liraglutide	4.0% to 5.4%	Nausea, vomiting, diarrhea, constipation, dyspepsia, abdominal pain, headache, fatigue, hypoglycemia, increased lipase	Causes thyroid C-cell tumors in rats and mice. Discontinue if pancreatitis is suspected.	Personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia syndrome type 2. Do not use with insulin or other GLP-1 agonists.
Phentermine/topiramate	8.6% to 9.3%	Paresthesia, dizziness, insomnia, dysguesia, constipation, dry mouth	Small increase in heart rate. Monitor electrolytes to detect metabolic acidosis and elevated creatinine. Monitor closely for depression, anxiety, and memory problems.	Glaucoma; hyperthyroidism; within 2 weeks of taking MAOIs. REMS requires negative pregnancy test before treatment and monthly thereafter to reduce the risk of teratogenicity
Naltrexone/bupropion	3.3% to 4.8%	Nausea, vomiting, headache, dizziness, insomnia, dry mouth, diarrhea	Monitor for suicidal ideation and behavior. Monitor for increases in heart rate and blood pressure. Rare cases of hepatotoxicity	Uncontrolled hypertension; seizure disorders; chronic opioid use; anorexia nervosa or bulimia; during withdrawal from alcohol, barbiturates, benzodiazepines, and antiepileptic drugs; within 2 weeks of taking MAOIs; coadministration with other bupropion-containing products

Pregnancy is a contraindication for all. Lorcaserin and phentermine/topiramate are Schedule IV controlled substances. For orlistat, weight loss is based on various meta-analyses. For all others, weight losses shown are from phase 3 trials. When multiple doses were tested, the weight loss shown is for the most effective dose.

MAOIs = monoamine oxidase inhibitors; REMS = risk evaluation and mitigation strategy.

Annu. Rev. Med. 2023. 74:125–39

The *Annual Review of Medicine* is online at med.annualreviews.org

https://doi.org/10.1146/annurev-med-043021-014919

Annual Review of Medicine

New Frontiers in Obesity Treatment: GLP-1 and Nascent Nutrient-Stimulated Hormone-Based Therapeutics

Ania M. Jastreboff[1] and Robert F. Kushner[2]

[1] Departments of Medicine (Endocrinology & Metabolism) and Pediatrics (Pediatric Endocrinology), Yale University School of Medicine, New Haven, Connecticut, USA; email: ania.jastreboff@yale.edu

[2] Departments of Medicine (Endocrinology) and Medical Education, Northwestern University Feinberg School of Medicine, Chicago, Illinois, USA; email: rkushner@northwestern.edu

Keywords

GLP-1 receptor agonists, antiobesity medications, dual receptor agonists, triple receptor agonists, nutrient-stimulated hormone-based therapies, NuSH-based therapies

Abstract

Nearly half of Americans are projected to have obesity by 2030, underscoring the pressing need for effective treatments. Glucagon-like peptide 1 receptor agonists (GLP-1 RAs) represent the first agents in a rapidly evolving, highly promising landscape of nascent hormone-based obesity therapeutics. With the understanding of the neurobiology of obesity rapidly expanding, these emerging entero-endocrine and endo-pancreatic agents combined or coformulated with GLP-1 RAs herald a new era of targeted, mechanism-based treatment of obesity. This article reviews GLP-1 RAs in the treatment of obesity and previews the imminent future of nutrient-stimulated hormone-based anti-obesity therapeutics.

Νέος παίκτης

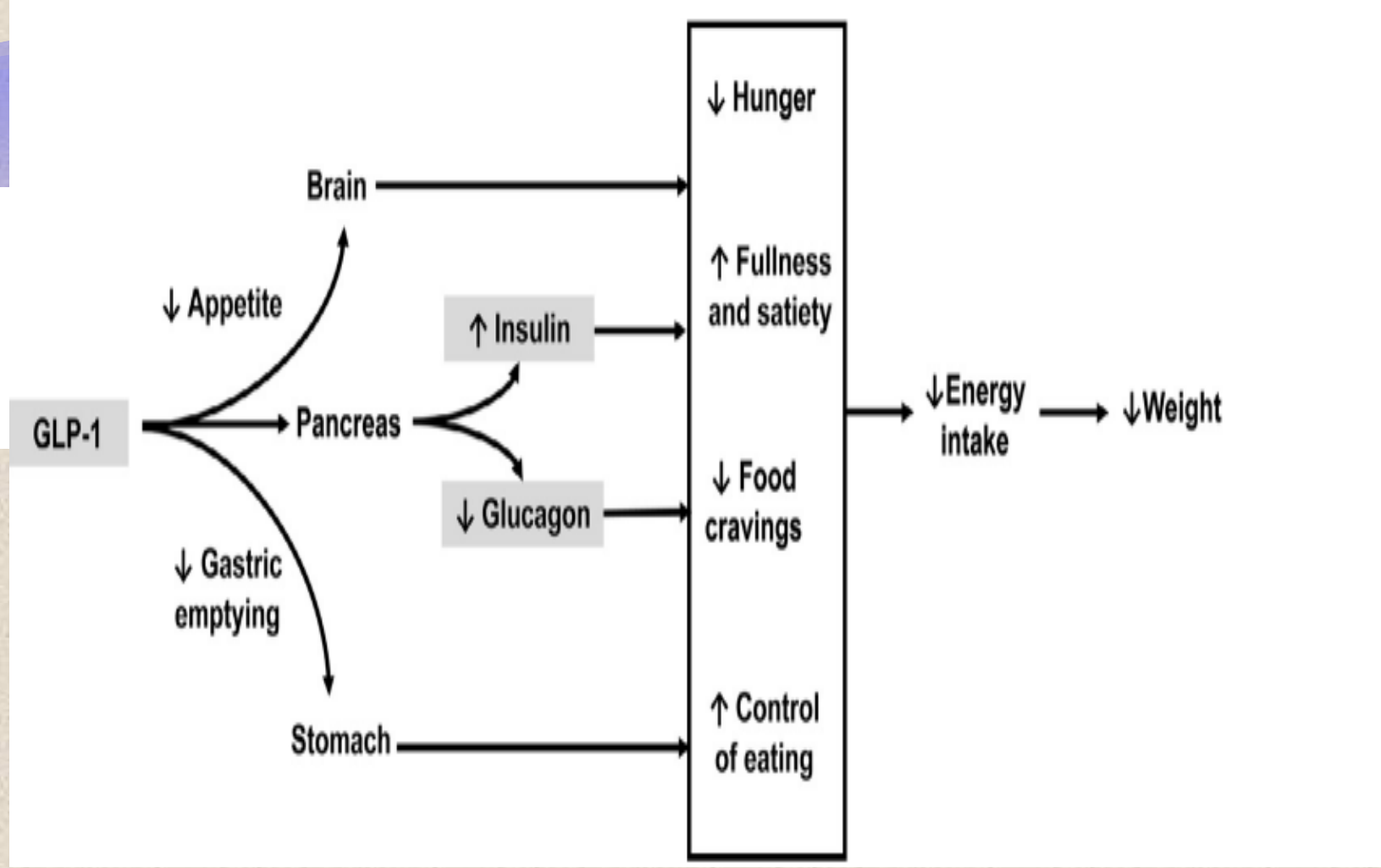
Contents lists available at ScienceDirect

Trends in Cardiovascular Medicine

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

Semaglutide for the treatment of obesity

Ariana M. Chao[a,b,*], Jena S. Tronieri[b], Anastassia Amaro[c], Thomas A. Wadden[b]



Semaglutide **T**reatment **E**ffect in **P**eople with obesity (**STEP**)

OBESITY SOCIETY

Phase 3 trial program
Semaglutide 2.4 mg for the treatment of obesity

Primary endpoint
for all STEP trials is **weight loss**

Trial design

STEP 1 — Weight management — 68-week treatment

STEP 2 — Weight management in T2D — 68-week treatment

STEP 3 — Weight management with IBT — 68-week treatment

Change to: IBT, intensive behavioral therapy; T2D, type 2 diabetes.

STEP 4 — Sustained weight management — 68-week treatment

STEP 5 — Long-term weight management — 104-week treatment

16-week dose escalation

7 weeks off treatment follow-up for safety assessments

Eligibility criteria

Unsuccessful diet history

Age ≥18 years

BMI
≥30 kg/m² or ≥27 kg/m² + weight-related complications

STEP 2 ≥27 kg/m² + T2D

No >5 kg weight change, <90 days before screening

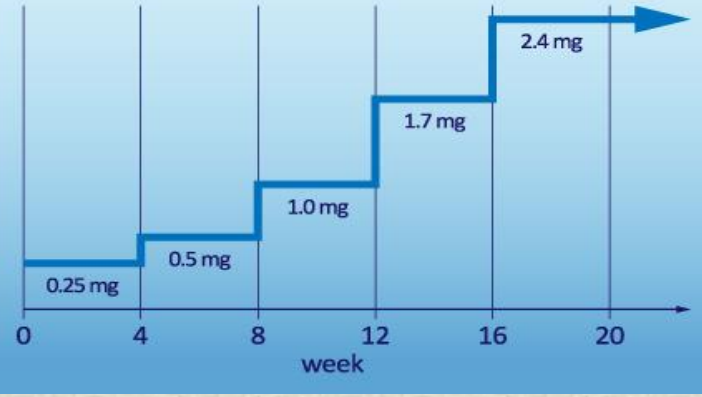
Treatment

WEEKLY — Semaglutide

VS

WEEKLY — Placebo

Dose escalation



STEP 1, 2, 4, and 5: Lifestyle intervention

150 min/week physical activity

–500 kcal/day diet

STEP 3: Intensive behavioral therapy

Dietitian counseling

Increased physical activity

Initial 8-week low-calorie diet

60-week hypocaloric diet

Πολλά πρωτόκολλα για μασάζ

Study Protocol Systematic Review

Medicine

OPEN

Massage for simple obesity

A protocol for systematic review

Yazheng Pang, MM[a], Kai Wang, MM[b], Shucheng Chen, MM[c], Tian Huang, MB[a], Mengsen Zhang, MB[a], Bin Zhang, MB[a], Juan Yu, MD[d,*]

Abstract

Background: Obesity has become the most serious public health problem in developed and developing countries, and simple obesity accounts for approximately 95% of the total cases. This study aims to assess the effects and safety of massage therapy for the treatment of simple obesity.

Methods: We will search foreign and Chinese databases, including PubMed, EMBASE, MEDLINE, CENTRAL, CNKI, WanFang Data, CBM, and VIP from the inception of the coverage of these databases to July 2020. Cochrane's collaboration tool will be used to assess the quality of the studies. RevMan 5.3 software will be used for the data analysis.

Results: This study will evaluate whether massage therapy is an effective intervention for simple obesity.

Conclusion: This study will provide evidence regarding whether massage therapy is beneficial for treating simple obesity in humans.

PROSPERO registration number: NO.CRD42020197635.

Abbreviations: BMI = body mass index, BW = body weight, CBM = Chinese Biological Medicine Database, CENTRAL = Cochrane central register of controlled trials, CIs = confidence intervals, CNKI = China National Knowledge Infrastructure, F% = body fat distribution rate, HC = hip circumference, MD = mean difference, RR = relative risks, SMD = standardized mean difference, VIP = Chinese Scientific Journal Database, WC = waist circumference.

Keywords: massage, simple obesity, systematic review, Traditional Chinese Medicine, tuina

Study Protocol Systematic Review

Medicine

OPEN

Effectiveness and safety of massage in the treatment of obesity

A protocol for systematic review and meta-analysis

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Abstract

Background: Obesity has become one of the largest chronic diseases in the world. It is a chronic metabolic disease caused by various factors. In recent years, massage has been used more and more widely in the treatment of obesity diseases. However, the effectiveness and safety of massage in the treatment of adult obesity are still unclear. The purpose of this study is to evaluate the effectiveness and safety of massage in the treatment of adult obesity.

Methods: We will conduct a comprehensive review in Medline, PubMed, Cochrane System Evaluation Database, embbase, Chinese Biomedical Literature Database, China National Knowledge Infrastructure, Wang Fang Database, Chinese Science Journal Database. There is no language restriction for the literature search from its establishment to February 2021. In addition, we will manually search for references to unpublished studies and originally included articles. Reviewers will identify the research, extract the data, and independently assess the quality. Results of interest include: total effective rate; total nasal symptom score; rhinitis quality-of-life questionnaire; visual analog scale; laboratory test indicators: IgE, IL6, IL10, or TNF-α levels; recurrence rate; adverse events. Randomized clinical trials will be collected, the Cochrane bias risk assessment tool will be used to assess methodological quality, and recommendations, evaluation, development, and evaluation methods will be used to assess the level of evidence. The meta-analysis will be performed using RevMan 5.4.0 software. A heterogeneity test will be conducted between studies, and $P < .1$ and $I2 > 50\%$ are the thresholds for testing. According to the degree of heterogeneity, we will use a fixed effects model or a random effects model.

Results: The results of this study will provide sufficient evidence to judge whether massage is an effective and safe treatment for adult obesity.

Conclusions: This study will provide evidence to determine whether massage is an effective intervention for Adult obesity. The research results will also be published in a peer-reviewed journal.

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Abbreviations: 95% CI = 95% confidence interval, BMI = body mass index.

Keywords: massage, obesity, protocol, systematic review

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Original Article

Clinical observation on acupuncture combined with massage therapy in simple obesity

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Abstract: Objective: To explore the clinical effect of acupuncture combined with massage therapy in patients with simple obesity. Methods: In total, 113 simple obesity patients admitted to our hospital were enrolled for retrospectively analysis of clinical data and divided into two groups according to the therapies. The 56 patients in group A were treated with acupuncture only, while the 57 in group B were treated with massage in addition to acupuncture; so as to determine the overall response rate, body weight, body mass index (BMI), body circumference, blood lipid changes, severity of illness, and quality of life. Results: The overall response rate (ORR) in group B was 92.98%, which was higher than 73.21% in group A ($P < 0.05$). The body weight and BMI, hip circumference and waist circumference, as well as TG and TC levels of patients in group B after treatment were smaller than those of patients in group A ($P < 0.05$ for all). The proportion of cases in normal condition and cases with grade I obesity was larger in group B compared with group A; besides, the constituents of cases with grade II and III in group B were smaller than that in group A ($P < 0.05$). After treatment, patients in group B showed superior scores in evaluation of social function, physical function, role function, cognitive function and emotional function, as compared with patients in group A ($P < 0.05$). Conclusion: Acupuncture combined with massage therapy in the treatment of obesity can reduce the body weight, BMI, hip circumference and waist circumference, and improve blood lipids as well as quality of life to a certain extent.

Keywords: Acupuncture, massage, simple obesity, combination therapy, body circumference, blood lipids, severity of illness

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Randomised Controlled Trial

The effects of extracorporeal shock wave therapy vs hand massage on serum lipids in overweight and obese women

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ABSTRACT

Background: The purpose of our study was to investigate the effects of extracorporeal shock wave therapy (ESWT) and hand massage therapy (HMT) on serum lipids and body composition in Korean women.

Materials and methods: We randomly classified 60 participants into overweight and obese groups. Subjects received ESWT and HMT twice a week for six weeks (a total of 12 sessions).

Results: Body weight and body mass index decreased significantly in obese women from both groups. Waist circumference significantly declined in obese women and overweight women in both treatment groups ($p < 0.001$). Body fat significantly decreased in the ESWT group of obese women ($p < 0.01$), while a significant reduction in abdominal obesity was noted only in the HMT group of overweight women ($p < 0.01$) and the ESWT group of obese women ($p < 0.01$). There was a significant decrease in triglycerides in the ESWT group of obese women ($p < 0.01$).

Conclusions: These results suggest that ESWT and HMT could be helpful for the management of people with excess abdominal fat and obesity. Moreover, ESWT is more effective than HMT for improving abdominal obesity and triglyceride levels in obese women as compared to overweight women.

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ORIGINAL PAPER

Abdominal Massage Alleviates Skeletal Muscle Insulin Resistance by Regulating the AMPK/SIRT1/PGC-1α Signaling Pathway

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Abstract

Abdominal massage (AM), a traditional Chinese medicine-based treatment method, has received considerable attention in the recent years. The aim of the present study was to investigate the effect of AM on high-fat diet (HFD)-induced insulin resistance (IR) in comparison with resveratrol (RSV) treatment. Forty-eight male Sprague-Dawley rats were randomly divided into the following four groups: standard chow diet (control group), high-fat diet (model group), HFD + abdominal massage (AM group), and HFD + resveratrol (RSV group). A rat model of IR was established by feeding HFD to rats for 8 weeks followed by treatment with AM or RSV for 4 weeks. The underlying HFD-induced IR molecular mechanisms were studied in rat serum and skeletal muscles. RSV and AM significantly improved glucose intolerance, hyperglycemia, obesity, and significantly reduced lipid accumulation [triglyceride (TC), total cholesterol (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C)], adipocytokine [free fatty acids (FFA), adiponectin (ADPN)] and serum pro-inflammatory cytokines (IL-6 and TNF-α) secretion. In addition, AM activated the AMPK/SIRT1 signaling pathway in rat skeletal muscle. In conclusion, our results showed that AM could improve IR by regulating the secretion of adipocytokines, pro-inflammatory cytokines as well as related signaling pathways in the skeletal muscle of rats, which might provide insights into development of new treatment methods for the clinical treatment of IR.

Keywords Abdominal massage · Insulin resistance · Inflammatory cytokines · Adipocytokine · AMPK/SIRT1/PGC-1α

Humans against Obesity: Who Will Win?

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ABSTRACT

The global obesity epidemic continues its relentless advance, currently affecting >2 billion people. This paper explores alternative ways to assess the potential disease impact of the epidemic, which is currently based almost exclusively on body mass index (BMI) data. It also argues in favor of concerted efforts to modify the built ecosystem that is driving the obesity epidemic. Most of the epidemiologic data on obesity are based on BMI (in kg/m^2) and use the ranges of 18.5–24.9 for normality, 25–29.9 for overweight, and ≥30 for obesity. But the gap between the median of the "normal" BMI distribution (~22) and the current population BMI of, for example, the United States (27.7) has become so wide that it is unlikely that we will be able to close that gap in the near future. Furthermore, the correlation between BMI and disease risk is not linear. Over 60% of the global disease burden of obesity affects individuals with a BMI ≥30, who comprise only ~10% of the global population of overweight/obese persons. Furthermore, BMI accounts for only ~17% of the risk of insulin resistance and subsequent type 2 diabetes in the BMI ≥25 population. Epigenetics, specifically DNA methylation, appears to play a far more important role than BMI in determining the risk of obesity's comorbidities, such as diabetes. Similarly, socioeconomic status carries a higher risk than BMI level for the development of obesity-related noncommunicable diseases. Finally, the built environment that sustains our species' lifestyle is a major driver of the obesity epidemic. Modifying that ecosystem will require no less than a social movement, one able to promote and sustain the necessary coordinated action of virtually all sectors of society. *Adv Nutr* 2019;10:S4–S9.


It's mine, you understand?!