EU4Nutrition LIVE

CONTINUITY OF NUTRITION CARE

optimal nutritional care for all

THE POWER OF CONCERTED EFFORTS AGAINST MALNUTRITION

The European Nutrition for Health Alliance

NESPEN clinical nutrition and metabolism

kenniscentrum ondervoeding

Nederlandse Vereniging van Diëtisten

Nutrition & Healthcare Alliance

The collision of two epidemics: sarcopenic obesity

Lorenzo M Donini

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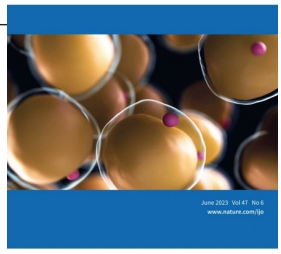
Department of Experimental Medicine
Pathophysiology, Food Science and Endocrinology section

Prevalence of obesity, sarcopenia and sarcopenic obesity

International Journal of Obesity (2008) 32, 1431–1437

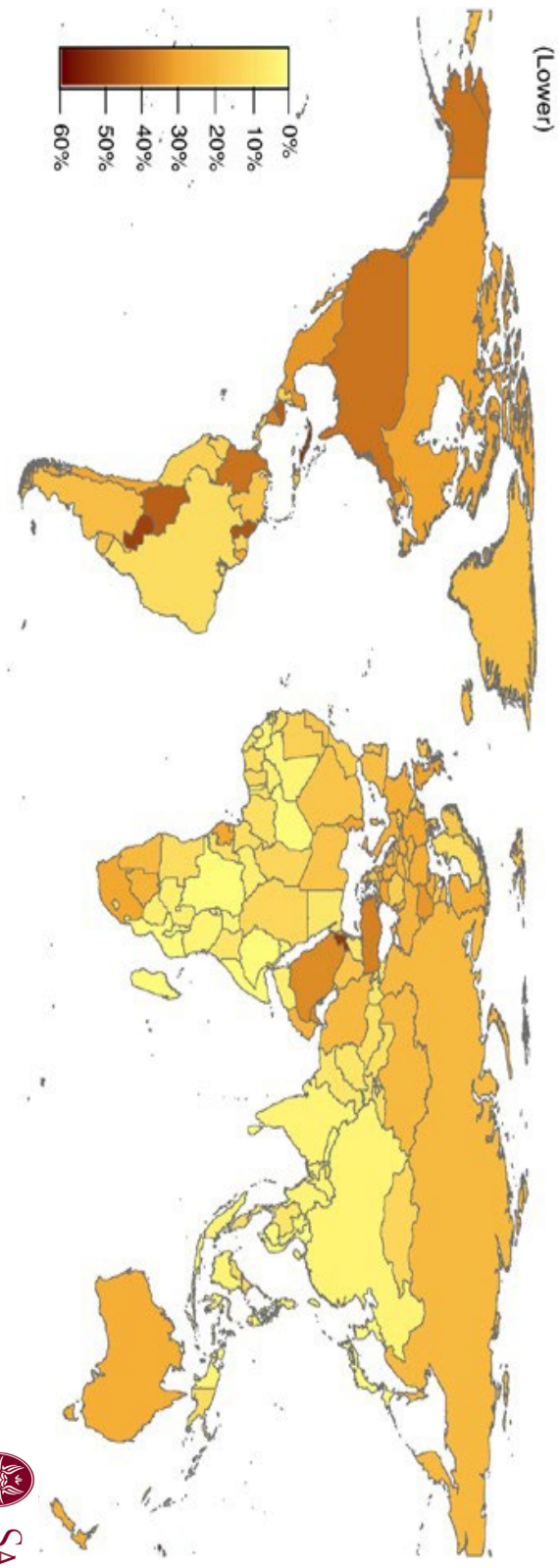
0307-0565/08 $32.00
www.nature.com/ijo



ORIGINAL ARTICLE

Global burden of obesity in 2005 and projections to 2030

T Kelly[1], W Yang[1], C-S Chen[1], K Reynolds[1] and J He[1,2]



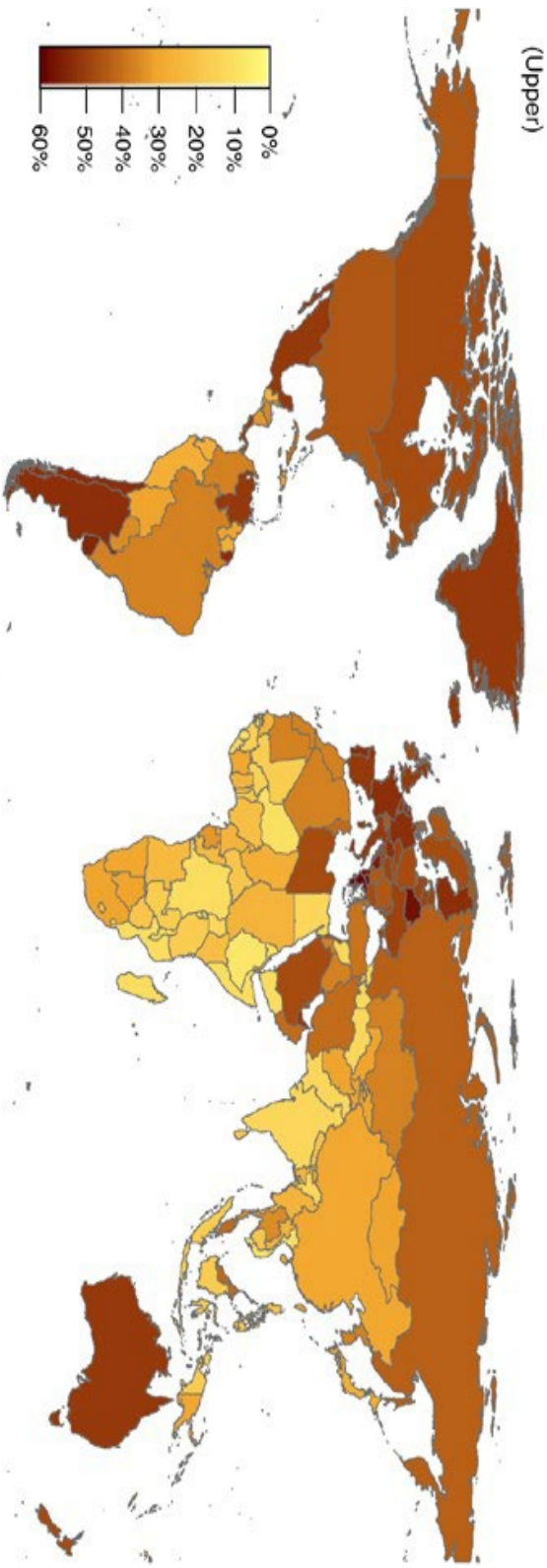
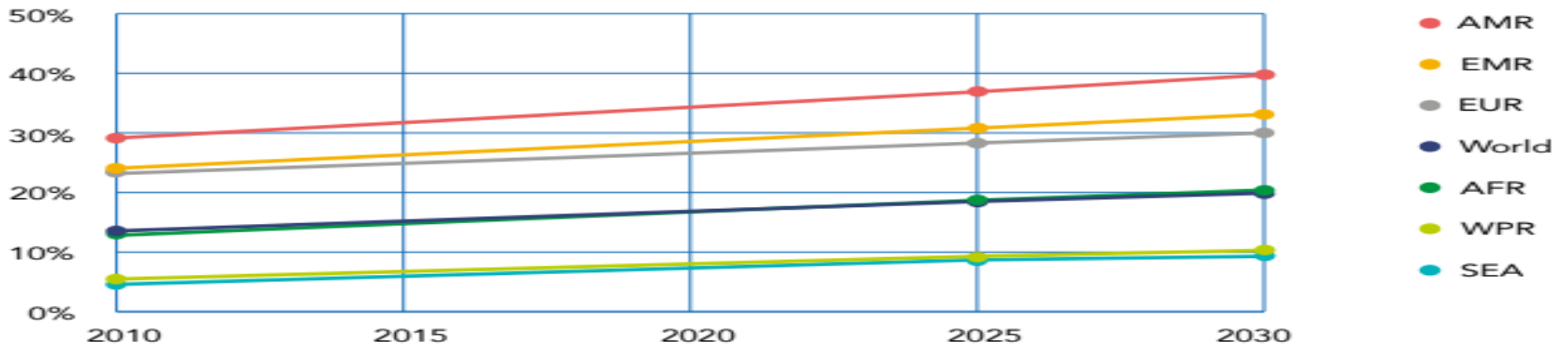
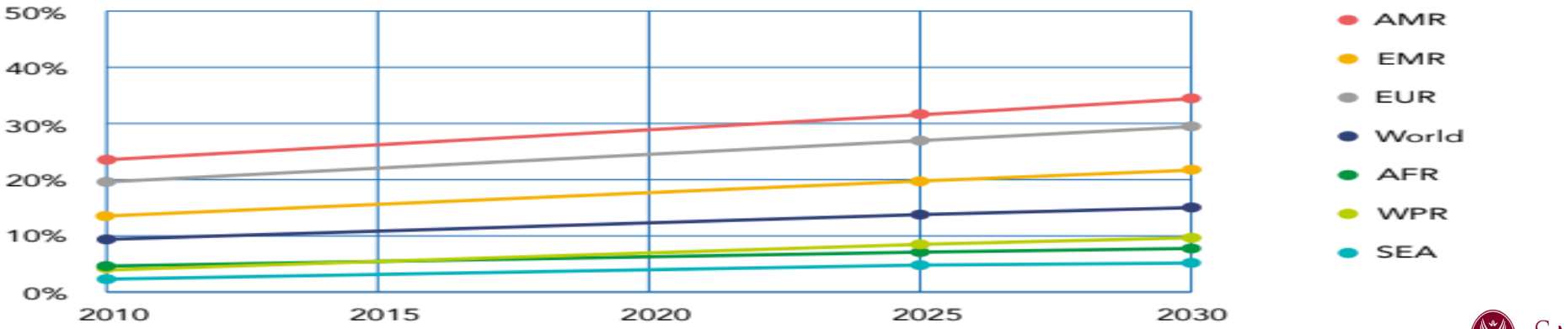


Figure 2.0: Prevalence of obesity (BMI ≥30kg/m²) amongst women by regions in 2010–2030

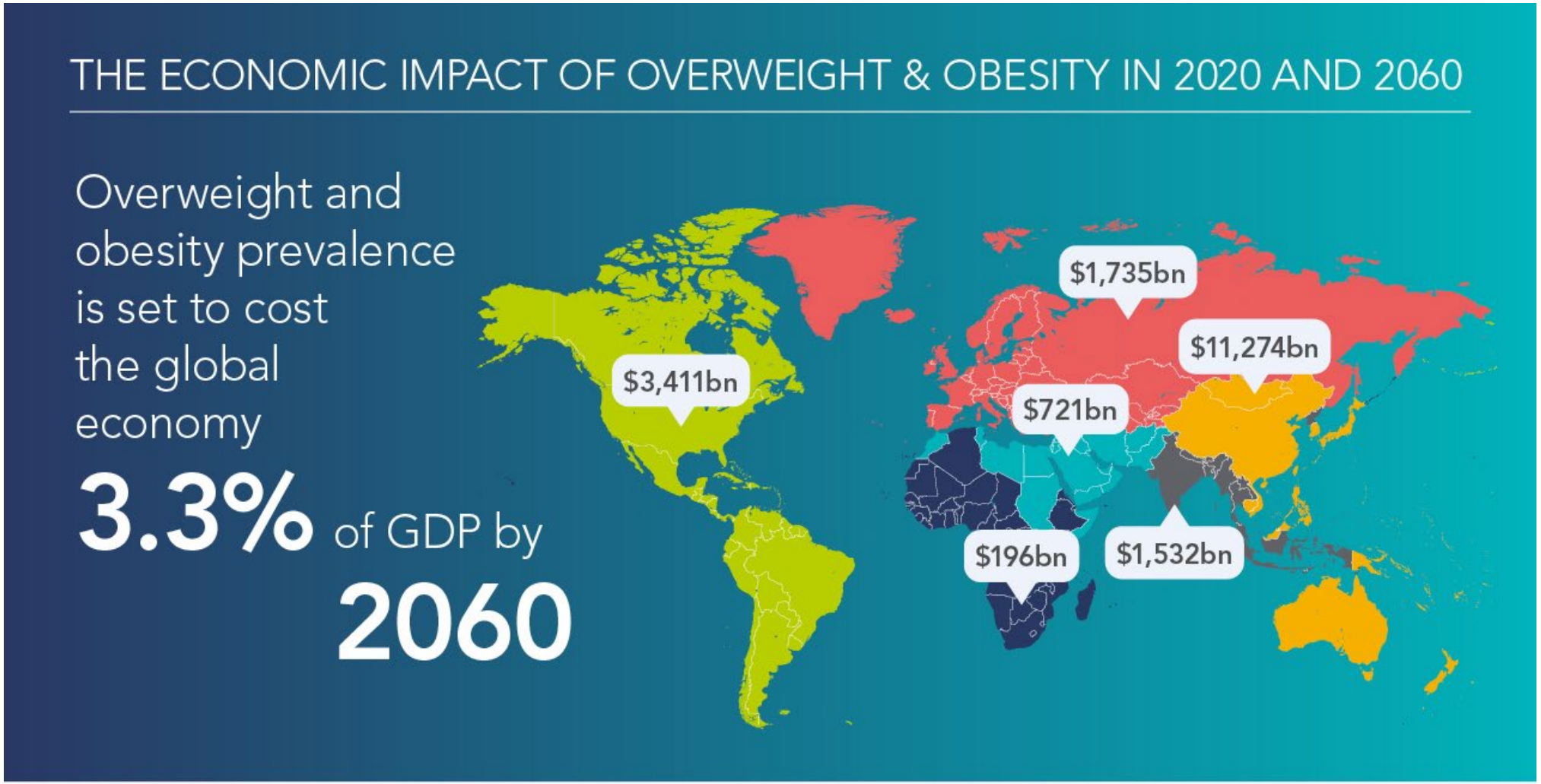


Source: NCD Risk Factor Collaboration (2017) and World Obesity Federation projections

Figure 2.1: Prevalence of obesity (BMI ≥30kg/m²) amongst men by regions in 2010–2030



Source: NCD Risk Factor Collaboration (2017) and World Obesity Federation projections



WORLD OBESITY

RTI INTERNATION

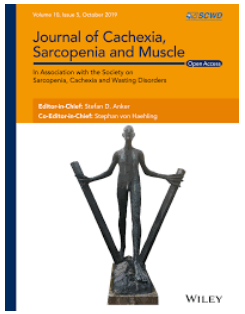
SAPIENZA UNIVERSITÀ DI ROMA

REVIEW

Journal of Cachexia, Sarcopenia and Muscle 2022; **13**: 86–99
Published online 23 November 2021 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12783

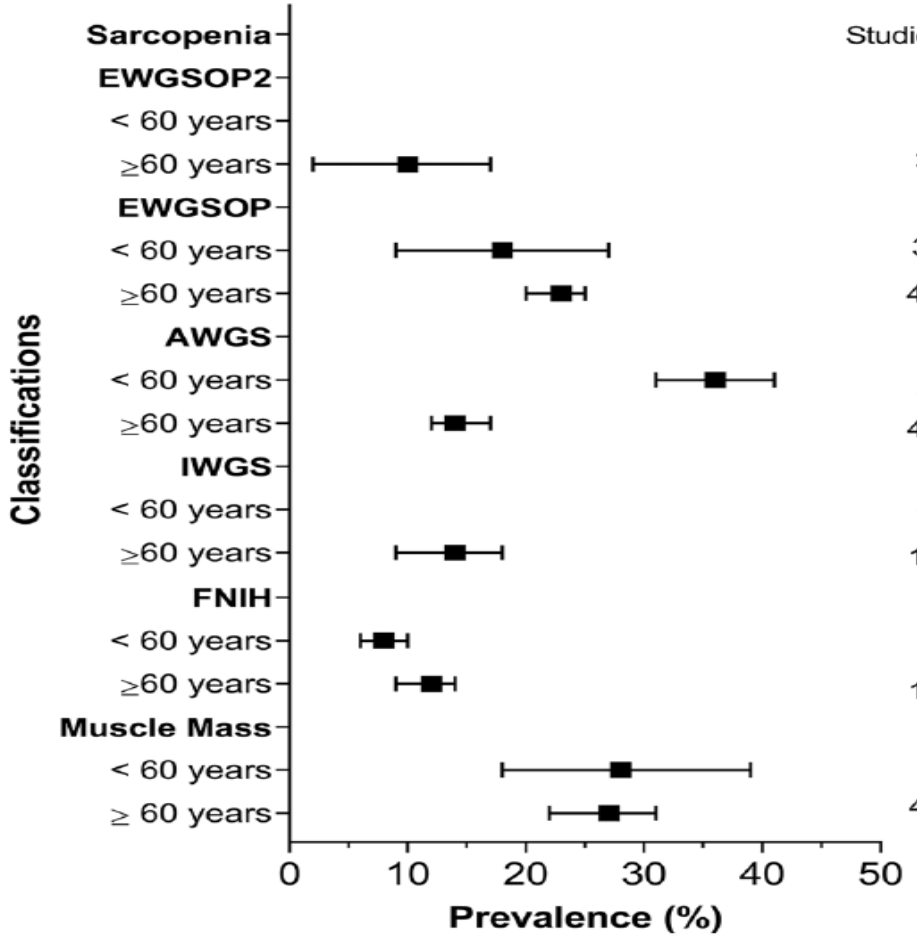
Global prevalence of sarcopenia and severe sarcopenia: a systematic review and meta-analysis

Fanny Petermann-Rocha[1,2,3†], Viktoria Balntzi[2†], Stuart R. Gray[2], Jose Lara[4], Frederick K. Ho[1‡], Jill P. Pell[1‡] & Carlos Celis-Morales[2,5,6*‡]



Prevalence in
< 60 years: 8-36%
(14 articles)

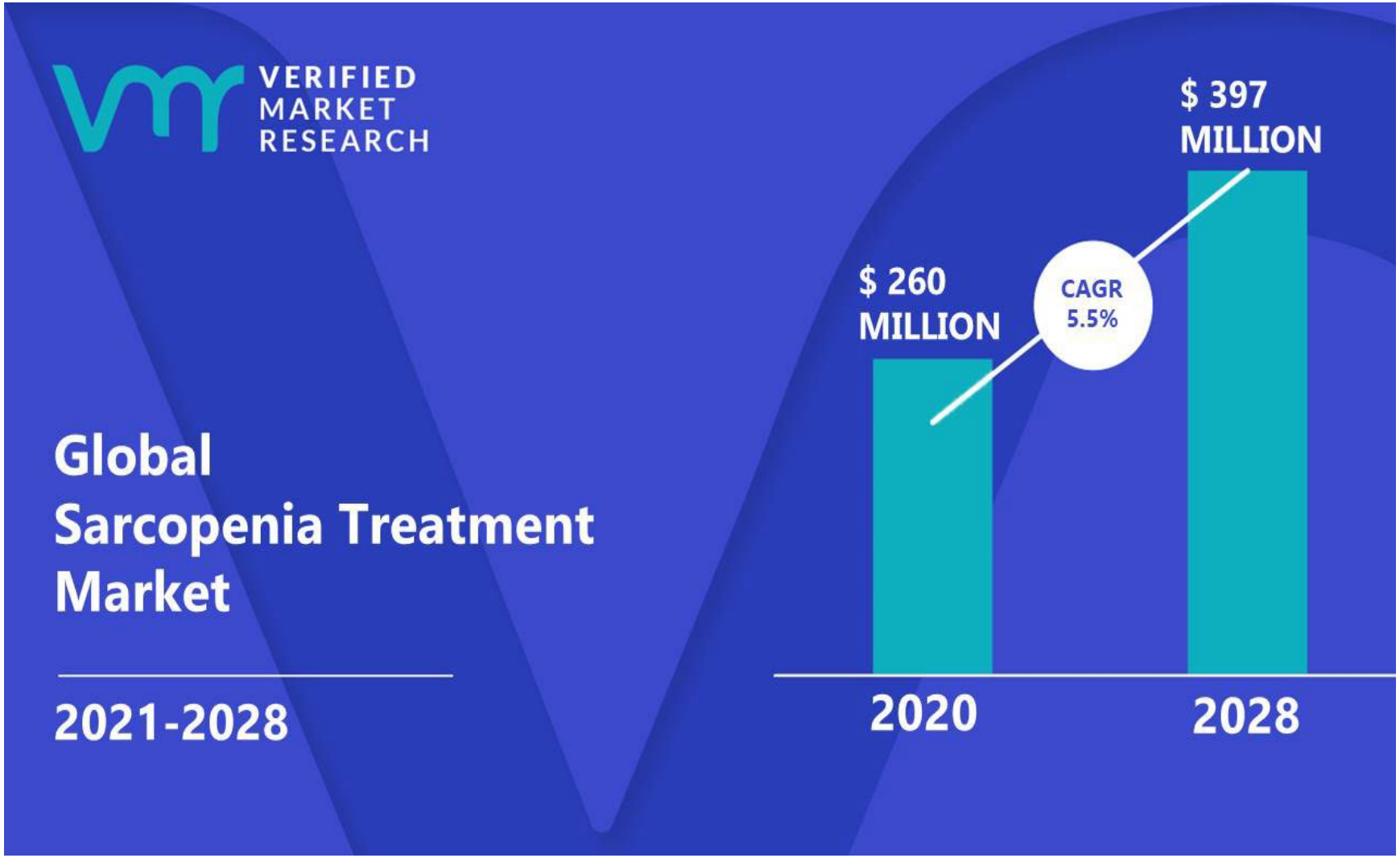
≥ 60 years: 10-27%
(167 articles)



	Studies (n)	Participants (n)	% (95% CI)	Heterogeneity
Sarcopenia				
EWGSOP2				
< 60 years	-	-	-	-
≥60 years	3	5,720	10.0 (2.00; 17.0)	99.09% (p<0.001)
EWGSOP				
< 60 years	3	351	18.0 (9.00; 27.0)	-
≥60 years	45	200,239	23.0 (20.0; 25.0)	98.63% (p<0.001)
AWGS				
< 60 years	2	344	36.0 (31.0; 41.0)	-
≥60 years	44	27,596	14.0 (12.0; 17.0)	97.90% (p<0.001)
IWGS				
< 60 years	-	-	-	-
≥60 years	12	11,890	14.0 (9.00; 18.0)	98.36% (p<0.001)
FNIH				
< 60 years	1	609	8.00 (6.00; 10.0)	-
≥60 years	19	27,255	12.0 (9.00; 14.0)	98.11% (p<0.001)
Muscle Mass				
< 60 years	8	5554	28.0 (18.0; 39.0)	98.70% (p<0.001)
≥ 60 years	44	33,246	27.0 (22.0; 31.0)	99.02% (p<0.001)

Global Sarcopenia Treatment Market Size By Treatment Type

(Protein Supplement, Vitamin B12 Supplement), By Distribution Channel (Hospital Pharmacies, Retail Pharmacies, Online Pharmacies), By Geographic Scope And Forecast



THE POWER OF CONCERTED EFFORTS AGAINST MALNUTRITION

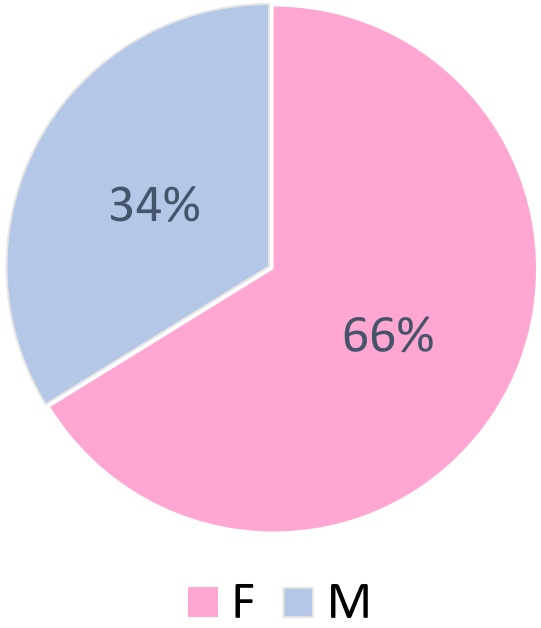
CONTINUITY OF NUTRITION CARE

International Conference Amsterdam 15 & 16 June

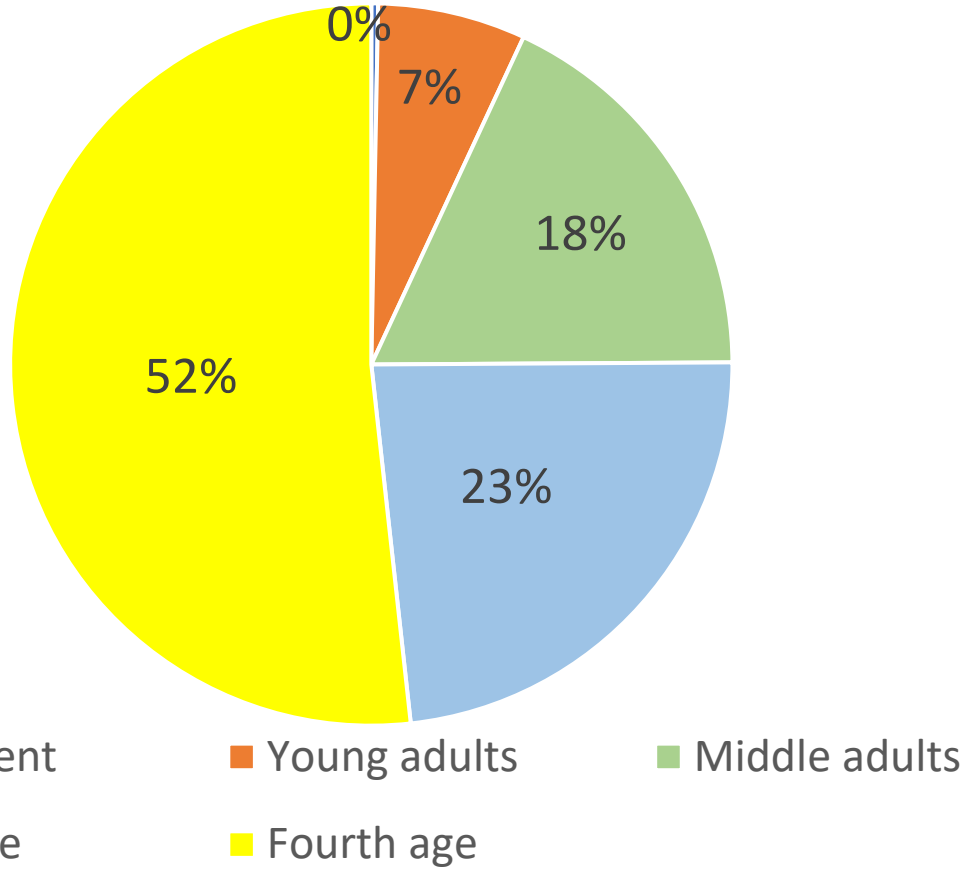
optimal nutritional care for all

F = 5703

M = 2912



Adolescent (<20 yrs)	40
Young adults (20-44 yrs)	872
Middle adults (45-61 yrs)	2366
Third age (62-74 yrs)	3073
Fourth age (>75 yrs)	6810



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THE POWER OF CONCERTED EFFORTS AGAINST MALNUTRITION

CONTINUITY OF NUTRITION CARE

Sbj over 60 years old	F	M
Rome (57 F/ 16 M)	3,5%	6,3%
Cagliari (85 F/ 78 M)	2,4%	5,1%
Piancavallo (1114 F/ 609 M)	17,1%	15,3%
Amsterdam (300 F/ 212 M)	1%	0%
Czech Republic (99 F/ 27 M)	2%	7,4%
North Carolina (64 F/ 22 M)	0%	0%
Total (3877 F/ 1789)	5,1%	5,6%

Sbj 40-59 years old	F	M
Roma (124 F/27 M)	5,6%	3,7%
Piancavallo (1178 F/ 792 M)	8,4%	6,4%
Total (1445 F/ 887 M)	7,3%	5,9%

Sbj < 40 years old	F	M
Rome (46 F/8 M)	13%	0%
Piancavallo (287 F/ 199 M)	3,5%	3%
Total (364 F/ 218 M)	4,4%	2,8%

optimal nutritional care for all

Why the two epidemics converge on sarcopenic obesity ?

THE POWER OF CONCERTED EFFORTS AGAINST MALNUTRITION

CONTINUITY OF NUTRITION CARE

CURRENT OBESITY REPORTS

Current Obesity Reports (2019) 8:458–471
https://doi.org/10.1007/s13679-019-00359-9

METABOLISM (M DALAMAGA, SECTION EDITOR)

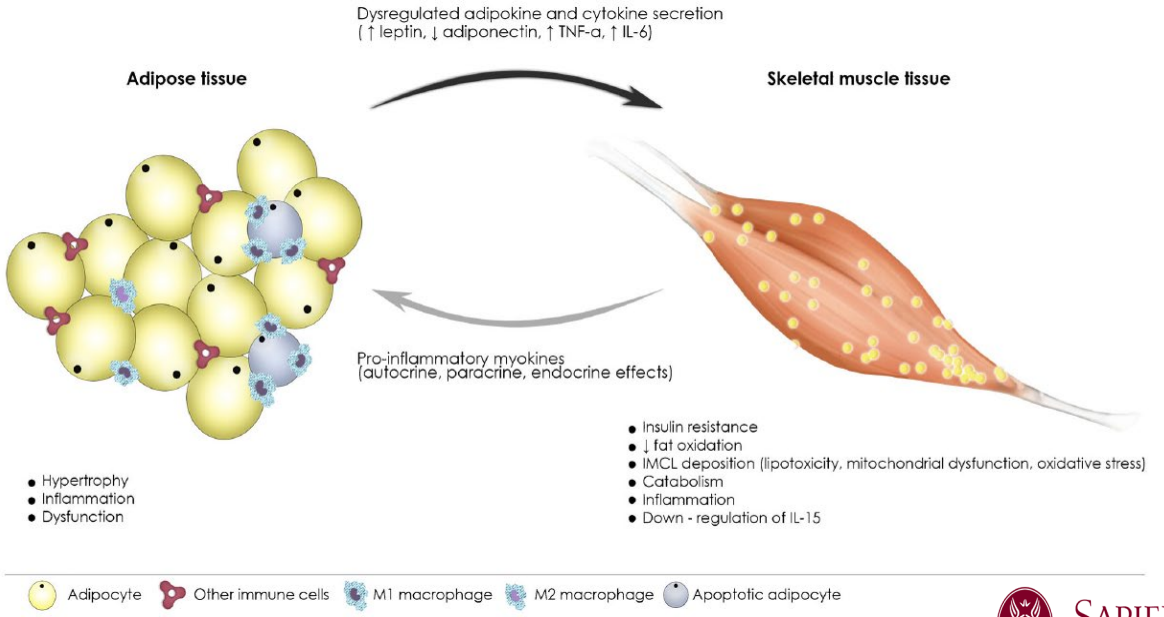
Sarcopenic Obesity: Epidemiologic Evidence, Pathophysiology, and Therapeutic Perspectives

Chrysi Koliaki[1] · Stavros Liatis[1] · Maria Dalamaga[2] · Alexander Kokkinos[1]

Bidirectional association between sarcopenia and obesity in the pathogenesis of SO:

1. low SMM can lead to reduced RMR and TEE, promoting fat gain
2. obesity may favor the development and progression of sarcopenia through a multifactorial network of clustered alterations

Pathogenetic cascade of SO mainly originates from adipose tissue dysfunction and inflammation (**obese sarcopenia** ??)



International Journal of Molecular Sciences

MDPI

Editorial

Molecular Mechanism and Pathogenesis of Sarcopenia: An Overview

Anna Picca [1,2,*] and Riccardo Calvani [1,2]

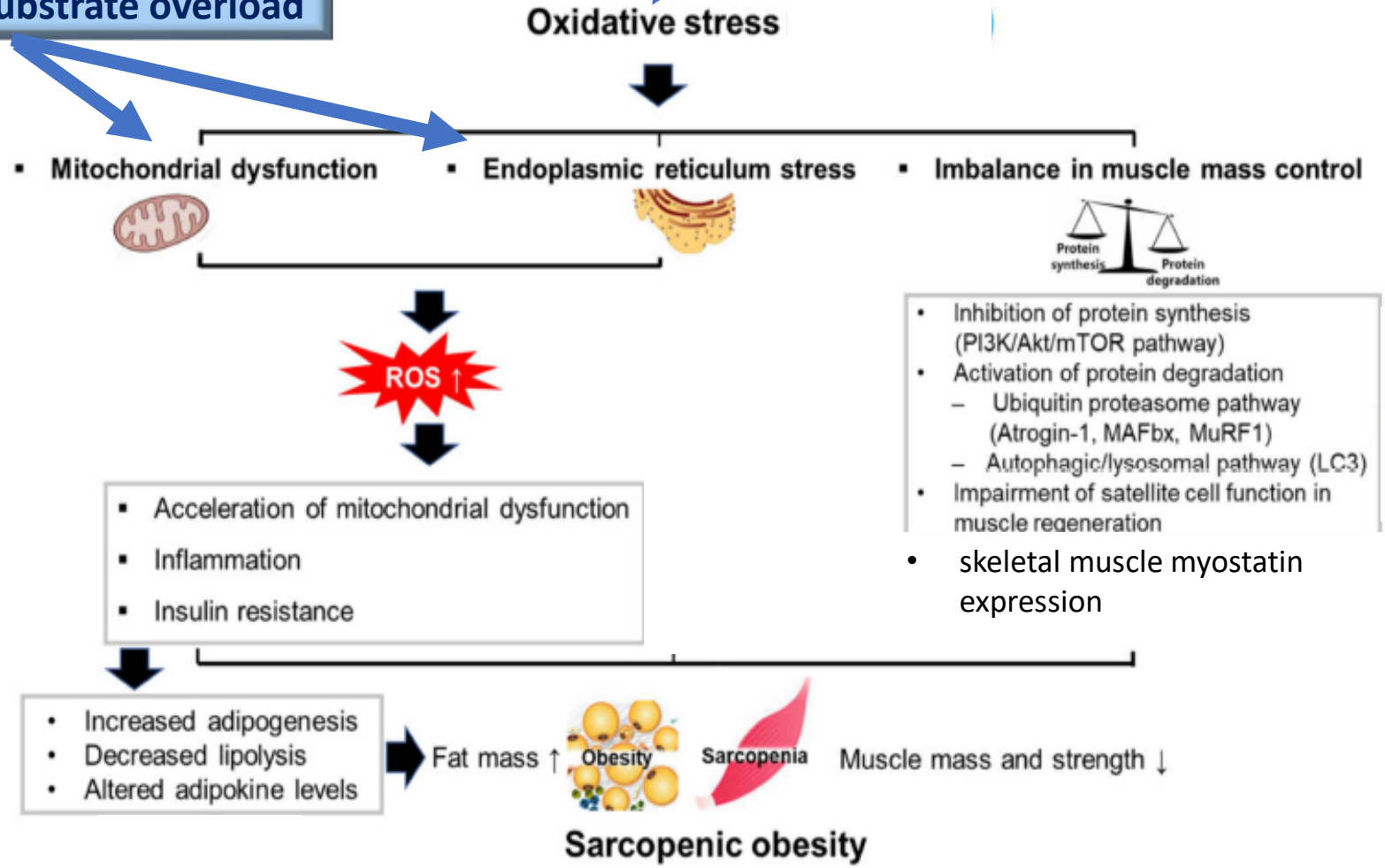
- Strong reliance of skeletal muscle cells on oxidative metabolism that makes them highly **susceptible to the detrimental effects of overproduction of ROS** as a bioproduct of their metabolism.

- In sarcopenic muscles dysmorphic, **ROS-producing mitochondria are inefficiently cleared and accumulate within cells**.

- The presence of great amounts of mitochondrial ROS in skeletal muscle cells with accumulation of single-strand breaks in telomere regions may **accelerate telomere erosion and trigger cellular senescence.**

hypertrophic adipocytes ⇒ mitochondrial dysfunction.

mitochondrial substrate overload



- skeletal muscle myostatin expression

MDPI
antioxidants
Review
Sarcopenic Obesity: Involvement of Oxidative Stress and Beneficial Role of Antioxidant Flavonoids
Un Ju Jung

- A **pro-inflammatory milieu** mainly involving interleukin IL1, IL6, and TNF-α may contribute in the pathogenesis of sarcopenia.

⇐ excessive activation of **proteolysis** driven by a catabolic response (more than decreased myogenesis) with a higher expression of p38 Mitogen-Activated Protein Kinase (p38 MAPK), and nuclear factor kappalight-chain-enhancer of activated B cells (NF-B)

- NF-B and p38-MAPK are known activators of the **ubiquitin proteasome system** triggered by IL1 signaling

CURRENT OBESITY REPORTS

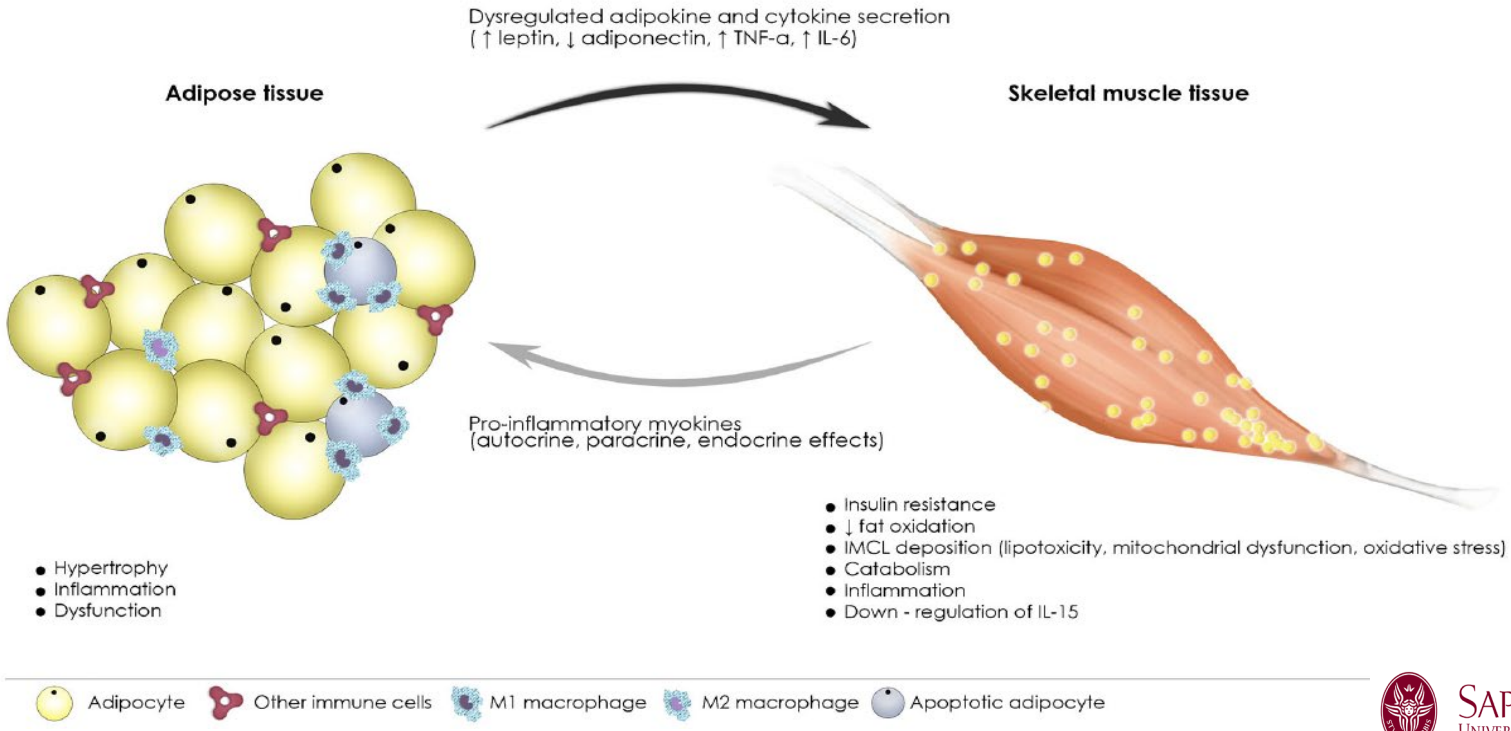
METABOLISM (M DALAMAGA, SECTION EDITOR)

Sarcopenic Obesity: Epidemiologic Evidence, Pathophysiology, and Therapeutic Perspectives

Chrysi Koliaki[1] · Stavros Liatis[1] · Maria Dalamaga[2] · Alexander Kokkinos[1]

Current Obesity Reports (2019) 8:458–471
https://doi.org/10.1007/s13679-019-00359-9

- Dysregulated adipokine and cytokine secretion as a result of an expanded, **inflamed, and dysfunctional adipose tissue** (increased leptin, TNF-α and IL-6, decreased adiponectin) ⇒ adverse effects upon skeletal muscle (impaired insulin sensitivity, reduced fat oxidation, IMCL deposition, induction of catabolism and inflammation, and downregulation of muscle IL-15)

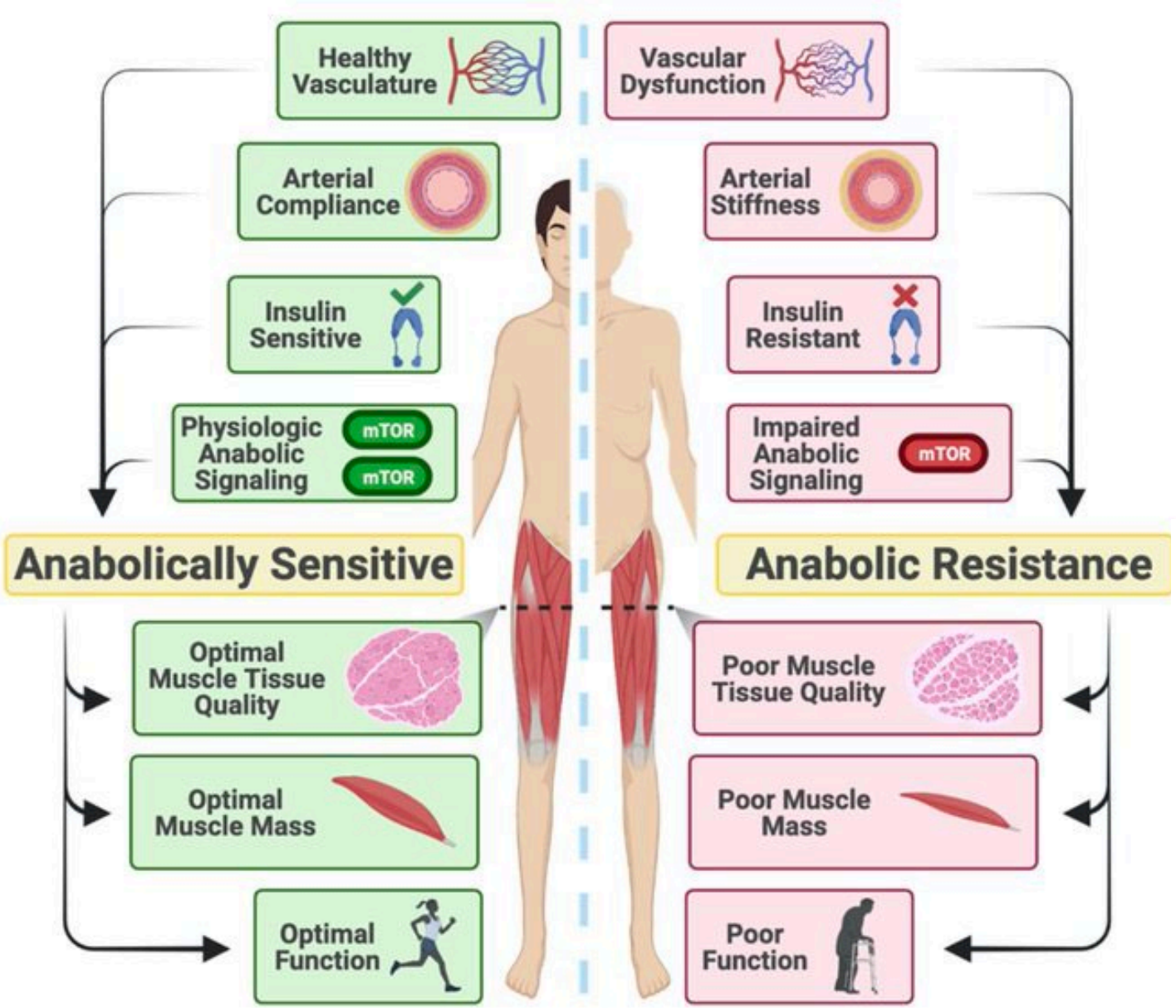


REVIEW

Journal of Cachexia, Sarcopenia and Muscle 2022; **13**: 114–127
Published online 23 December 2021 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12898

The contributory role of vascular health in age-related anabolic resistance

Nile F. Banks[1], Emily M. Rogers[1], David D. Church[2], Arny A. Ferrando[2] & Nathaniel D.M. Jenkins[1,3]*



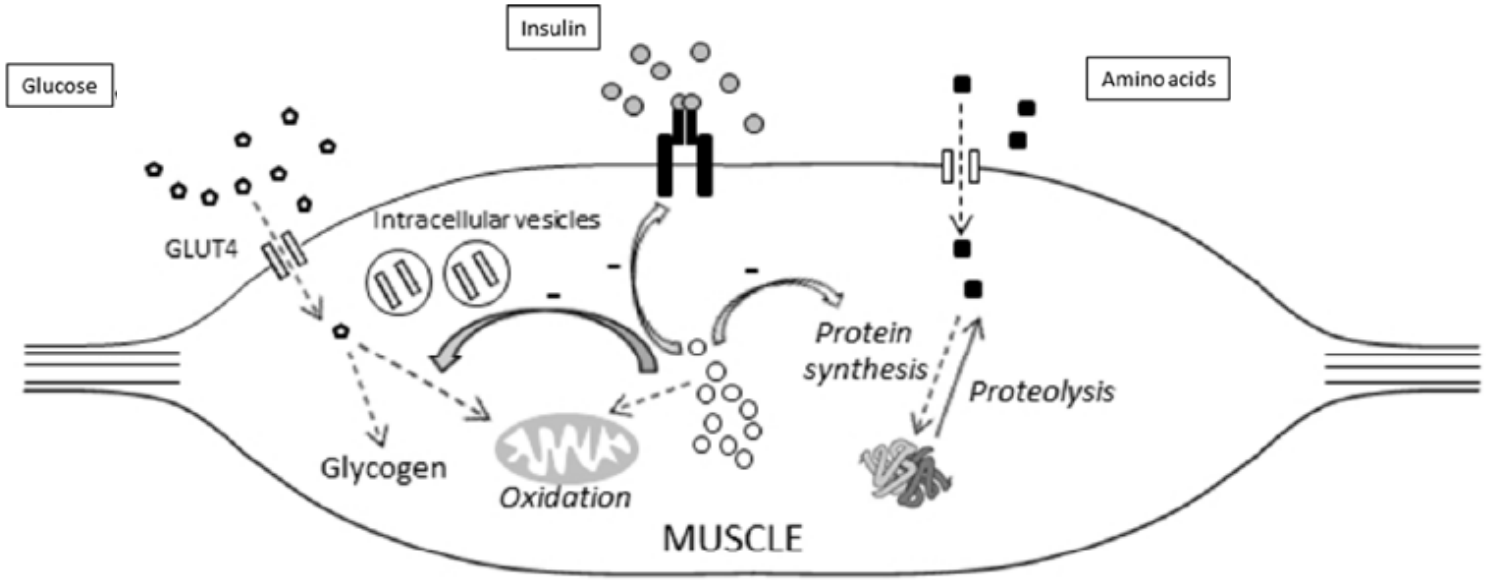
⇐ attenuated effect of dietary EAA in stimulating muscle protein synthesis
⇐ diminished expression of Akt/mTOR signaling

Insulin resistance and sarcopenia: mechanistic links between common co-morbidities

Journal of Endocrinology (2016) **229**, R67–R81

Mark E Cleasby[1], Pauline M Jamieson[2] and Philip J Atherton[3]

- **insulin** = anabolic action (promoting skeletal muscle protein uptake) + redistribution of blood flow from nonnutritive to nutritive capillaries + activation of endothelial NO in precapillary muscle arterioles (increasing the capillary surface area for nutrient exchange)
- **intramyocellular lipid deposition** ⇒ impaired insulin signalling, protein synthesis due to reduced anabolic response to exercise & AA



Broken straight arrows: reduced metabolite flux.

Open curved arrows: inhibitory effect.

MDPI
cells

Review

The Role of Crosstalk between Adipose Cells and Myocytes in the Pathogenesis of Sarcopenic Obesity in the Elderly

Mauro Zamboni [1,*], Gloria Mazzali [2], Anna Brunelli [1], Tanaz Saatchi [1], Silvia Urbani [1], Anna Giani [1], Andrea P. Rossi [3], Elena Zoico [2] and Francesco Fantin [2]

Adipomyokine	Effects–Skeletal Muscle	Effects—Adipose Tissue	Aging
IL-6	+muscle hypertrophy +glucose uptake +glycogenolysis, lipolysis	+lipolysis +free fatty acid (FFA) oxidation browning of WAT	⇑
Irisin	+glucose uptake +muscle trophism	+lipolysis browning of WAT —lipid accumulation	⇓
IL-15	+glucose uptake +mitochondrial activity	+adiponectin secretion	⇓
BAIBA	+mitochondrial FFA oxidation +insulin sensitivity	+mitochondrial FFA oxidation	⇓
Meteorin-like	+energy expenditure +glucose tolerance	browning of WAT	?
LIF	+muscle hypertrophy +satellite cell proliferation regeneration after muscle damage	+adipocyte differentiation	⇓

MDPI

cells

Review

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Mauro Zamboni [1,*], Gloria Mazzali [2], Anna Brunelli [1], Tanaz Saatchi [1], Silvia Urbani [1], Anna Giani [1], Andrea P. Rossi [3], Elena Zoico [2] and Francesco Fantin [2]

Adipomyokine	Effects–Skeletal Muscle	Effects—Adipose Tissue	Aging
Myostatin	- muscle hypertrophy	+adipogenesis	⇑
Apelin	improves muscle metabolism	glucose uptake −lipid storage	⇓
ANGPTL4	+FFA oxidation	+lipolysis	?
FGF-21	+thermogenesis	+glucose uptake	⇓
Follistatin-like 1	+endothelial cells function and survival		?
IL-8	+insulin resistance	+insulin resistance	⇓
MCP-1	−glucose uptake		?
PEDF	+insulin resistance +ectopic lipid deposition	+insulin resistance +pro-inflammatory pathway	?

Current Obesity Reports (2019) 8:458–471
https://doi.org/10.1007/s13679-019-00359-9

METABOLISM (M DALAMAGA, SECTION EDITOR)

Sarcopenic Obesity: Epidemiologic Evidence, Pathophysiology, and Therapeutic Perspectives

Chrysi Koliaki[1] · Stavros Liatis[1] · Maria Dalamaga[2] · Alexander Kokkinos[1]

Other mechanisms may also explain how obesity can elicit **muscle catabolic pathways**, together with **muscle quality and metabolism impairment** (⇐ mitochondrial dysfunction and oxidative stress, inflammation, atherosclerotic altered muscle tissue perfusion) **:**

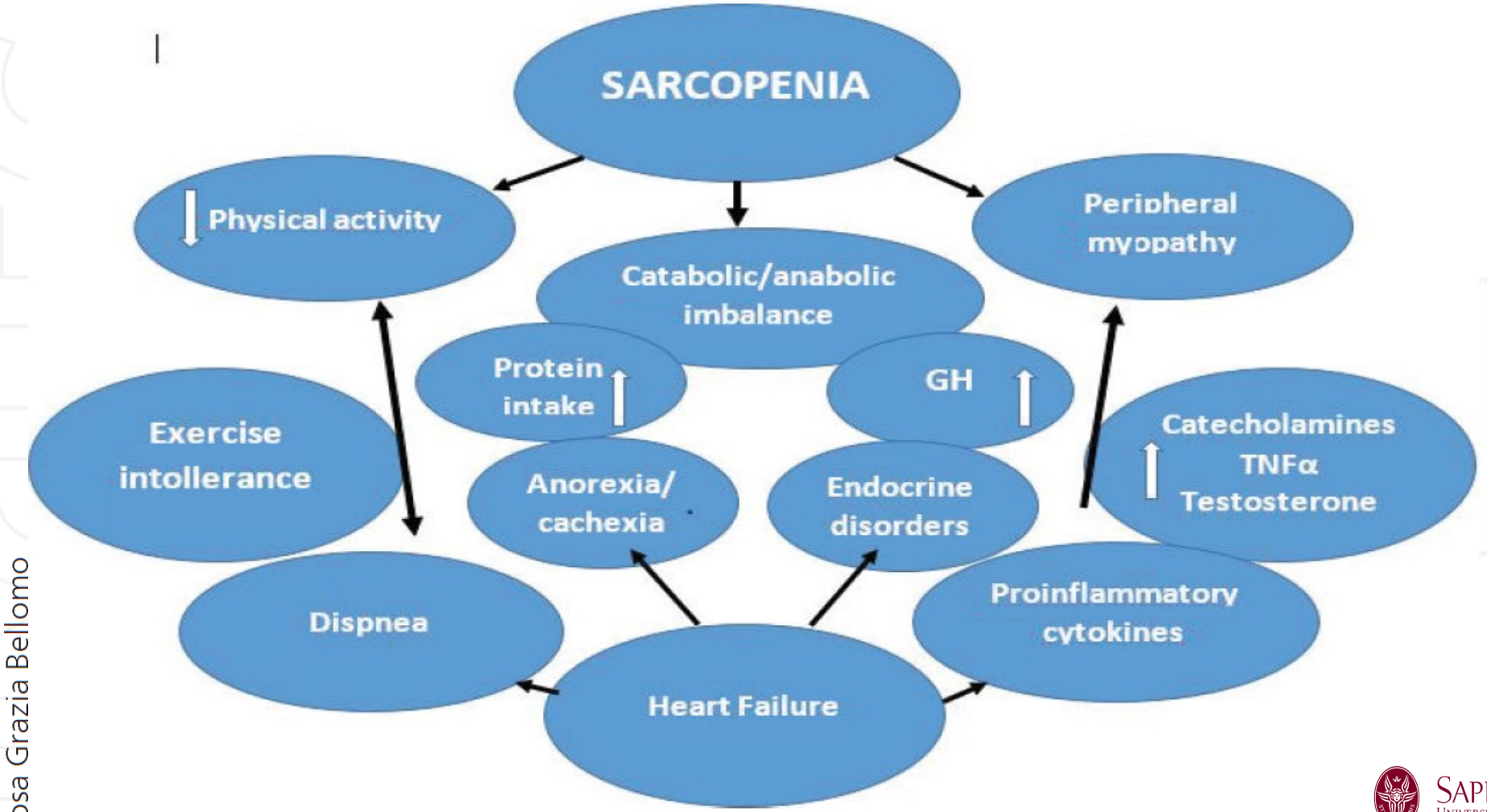
- **physical inactivity** as a result of obesity-associated musculo-skeletal complications
- **selective undernutrition** (⇐ energy dense nutrient-poor diets)
- axonal degeneration, neuronal hypoexitability, loss of α-motoneurons ⇒ **dysregulation in the denervation–reinnervation cycle of motor neurons** "⇒ impairments in contractile velocity, muscle synergy, muscle weakness
- **obesity-related chronic conditions** (T2DM, HF, COPD, kidney disease, and cancer)

Pathohysiology of secondary sarcopenia

Sarcopenia in Chronic Illness and Rehabilitative Approaches

http://dx.doi.org/10.5772/intechopen.70223

Raoul Saggini, Simona Maria Carmignano, Lucia Cosenza, Tommaso Palermo and Rosa Grazia Bellomo



HYPERTROPHY/HYPERPLASIA OF ADIPOCYTES with disturbed capacity to store lipids

ECTOPIC ACCUMULATION IN SKELETAL MUSCLE

INFLAMMATION (local pro-inflammatory status, chronic low grade systemic inflammation inflammaging)

MITOCHONDRIAL DYSFUNCTION (impaired oxidation capacity and increased ROS formation)

accumulation of PRO-INFLAMMATORY MACROPHAGES and other immune cells + dysregulated production of various ADIPOKINES

DYSREGULATION OF MYOKINES SYNTHESIS

LIPOTOXIC ENVIRONMENT, INSULIN RESISTANCE, enhanced secretion of pro-inflammatory cytokines

ANABOLIC RESISTANCE

SENESCENT CELLS and the immune cell-released cytokines and chemokines

MUSCLE DYSFUNCTION

Targeting sarcopenic obesity

ESPEN Guideline

Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement☆

Lorenzo M. Donini [a, *], Luca Busetto [b], Stephan C. Bischoff [c], Tommy Cederholm [d], Maria D. Ballesteros-Pomar [e], John A. Batsis [f], Juergen M. Bauer [g], Yves Boirie [h], Alfonso J. Cruz-Jentoft [i], Dror Dicker [j], Stefano Frara [k], Gema Frühbeck [l], Laurence Genton [m], Yftach Gepner [n], Andrea Giustina [k], Maria Cristina Gonzalez [o], Ho-Seong Han [p], Steven B. Heymsfield [q], Takashi Higashiguchi [r], Alessandro Laviano [a], Andrea Lenzi [a], Ibolya Nyulasi [s], Edda Parrinello [a], Eleonora Poggiogalle [a], Carla M. Prado [t], Javier Salvador [u], Yves Rolland [v], Ferruccio Santini [w], Mireille J. Serlie [x], Hanping Shi [y], Cornel C. Sieber [z], Mario Siervo [aa], Roberto Vettor [b], Dennis T. Villareal [ab], Dorothee Volkert [z], Jianchun Yu [ac], Mauro Zamboni [ad], Rocco Barazzoni [ae, **]

Obesity Facts

Consensus Statement

Obes Facts
DOI: 10.1159/000521241

Received: November 21, 2021
Accepted: November 26, 2021
Published online: February 23, 2022

Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement

Lorenzo M. Donini[a] Luca Busetto[b] Stephan C. Bischoff[c] Tommy Cederholm[d] Maria D. Ballesteros-Pomar[e] John A. Batsis[f] Juergen M. Bauer[g] Yves Boirie[h] Alfonso J. Cruz-Jentoft[i] Dror Dicker[j] Stefano Frara[k] Gema Frühbeck[l] Laurence Genton[m] Yftach Gepner[n] Andrea Giustina[k] Maria Cristina Gonzalez[o] Ho-Seong Han[p] Steven B. Heymsfield[q] Takashi Higashiguchi[r] Alessandro Laviano[a] Andrea Lenzi[a] Ibolya Nyulasi[s] Edda Parrinello[a] Eleonora Poggiogalle[a] Carla M. Prado[t] Javier Salvador[u] Yves Rolland[v] Ferruccio Santini[w] Mireille J. Serlie[x] Hanping Shi[y] Cornel C. Sieber[z] Mario Siervo[A] Roberto Vettor[b] Dennis T. Villareal[B] Dorothee Volkert[z] Jianchun Yu[C] Mauro Zamboni[D] Rocco Barazzoni[E]

ESPEN The European Society for Clinical Nutrition and Metabolism

EASO European Association for the Study of Obesity

Sapienza Università di Roma

ESPEN The European Society for Clinical Nutrition and Metabolism

EASO European Association for the Study of Obesity

THE POWER OF CONCERTED EFFORTS AGAINST MALNUTRITION

CONTINUITY OF NUTRITION CARE

- High **BMI or WC** (based on ethnic cut-points)
- **Surrogate parameters for sarcopenia** [clinical symptoms, clinical suspicion or questionnaires (e.g. SARC-F in older subjects)]

Both conditions *must be present to proceed with the diagnostic process.*

It will be performed in ***two steps:***

1. ALTERED SKELETAL MUSCLE FUNCTIONAL PARAMETERS considering **strength** (HGS, chair stand test)

If muscle functional parameters suggest the presence of SO, the diagnostic process will continue considering body composition.

2. ALTERED BODY COMPOSITION: increased FM (FM%) and **reduced muscle mass** assessed as ALM/W by DXA or as SMM/W by BIA

Both ***altered body composition*** *and* ***altered skeletal muscle functional parameters*** *should be present to assess the presence of SO.*

A two-level STAGING should be performed, based on the presence of complications **resulting from high FM and low ASMM,** to better mirror the progression/severity of SO:

- **STAGE I: NO complications**
- **STAGE II: presence of at least one complication attributable to SO** (e.g. metabolic diseases, functional disabilities, cardiovascular and respiratory diseases).

SO algorithm in the literature

79 citations for Obes Facts + 49 citations for Clin Nutr version
(Scopus, 10th June 2023)

- The ESPEN/EASO SO consensus identified a high and variable prevalence of SO in post-BS patients depending on the **BC technique** used; SO prevalence was higher when assessed by DXA (Texeira Vieira, Clin Nutr 2022).
- SO was associated, with:
  - in **older adults**,
    - **decreased higher-level functional capacity** (Ida S, End J 2022; Yoshimura Y, Nutrients 2022)
    - poor **nutritional status** at MNA (Murawiak M, Nutrients 2022)
    - higher level of **cognitive decline** (Unsal P, Nutr Clin Pract 2023)
  - higher **mortality** in non-small cell lung cancer (639 participants – 229 F; mean age 58.6 years) (Zhou J, Clin Nutr 2023).

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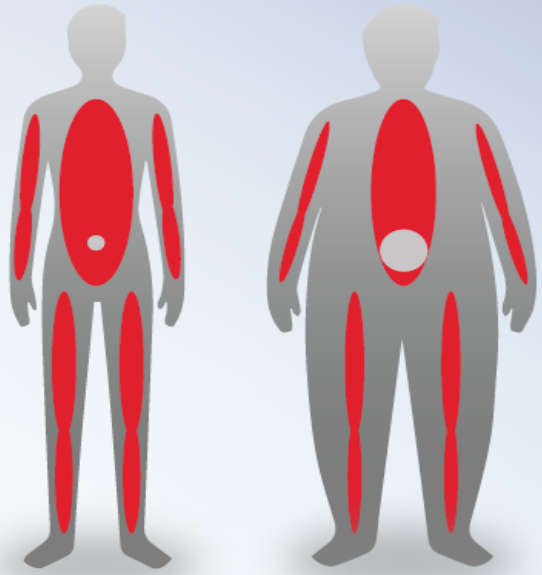
UNIVERSITÀ DI ROMA

SAPIENZA UNIVERSITY – ROME (Italy)
Piazzale Aldo Moro, 5 – Rome (Italy)
Organi Collegiali Meeting Room of the Rectorate

FOOD SCIENCE CONFERENCE /XII EDITION

SARCOPENIC OBESITY GLOBAL LEADERSHIP INITIATIVE (SOGLI): "THE JOURNEY CONTINUES"

Rome, Thursday 9th – Friday 10th November 2023



An initiative from

Sapienza University (Rome, Italy)

with the cooperation of ESPEN and EASO

Ara Pacis Augustae
consecrated on 9 BC