UNIVERSITÀ GIUSTINO FORTUNATO
D.M. 13 aprile 2006 - G.U. n° 104 del 6/05/2006 - TELEMATICA
UniforJob ACADEMY
Accademia Eraclitea
ENTE DI RICERCA E DI ALTA FORMAZIONE ACCREDITATO

Master Universitario di primo livello in "Deglutologia geriatrica" A.A. 2023/24

L'anziano

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Medico Geriatra
Professore associato in Medicina Interna
Università di Bologna

Obiettivi Formativi

- Le basi Biologiche dell'invecchiamento
- **Come si manifesta clinicamente l'invecchiamento**
- **Sarcopenia e Fragilità**

Tabella 2 – Classificazione eziopatogenetica della sarcopenia

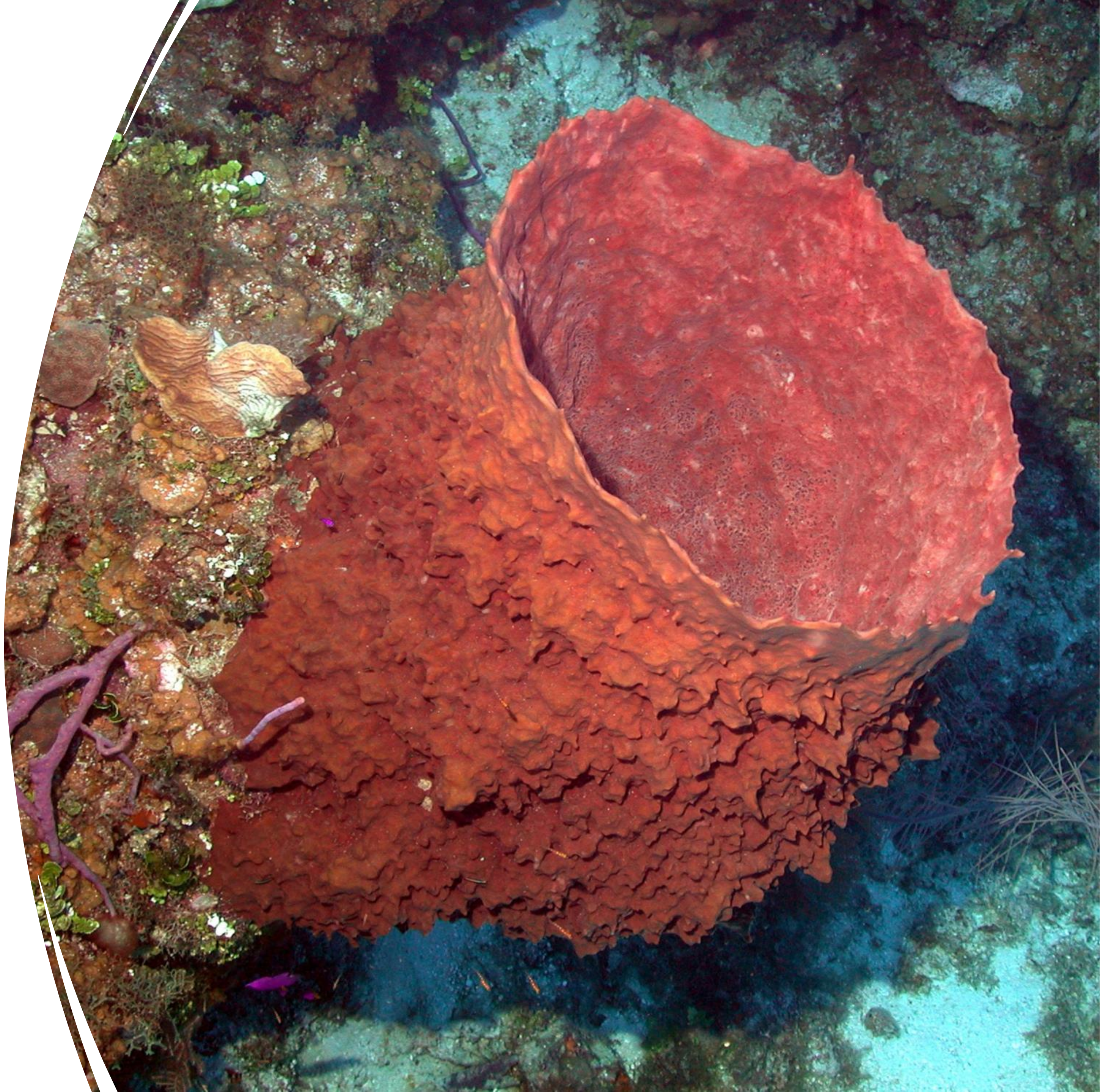
Sarcopenia primaria	
Età correlata	Assenza di altre cause eccetto l'invecchiamento
Sarcopenia secondaria	
Attività fisica correlata	Sedentarietà, allettamento, decondizionamento, assenza di gravità
Malattia-correlata	Scompenso d'organo (cuore, polmoni, reni, fegato, cervello), malattie infiammatorie, endocrine, neoplasie
Nutrizione-correlata	Inadeguato introito calorico/proteico, malassorbimento, farmaci anoressizanti

Fonte: Volpato S., Bianchi L. (2016), Sarcopenia, *in Zuliani G., Volpato S., a cura di,* Lezioni di Geriatria e Gerontologia, *Universitas Studiorum*

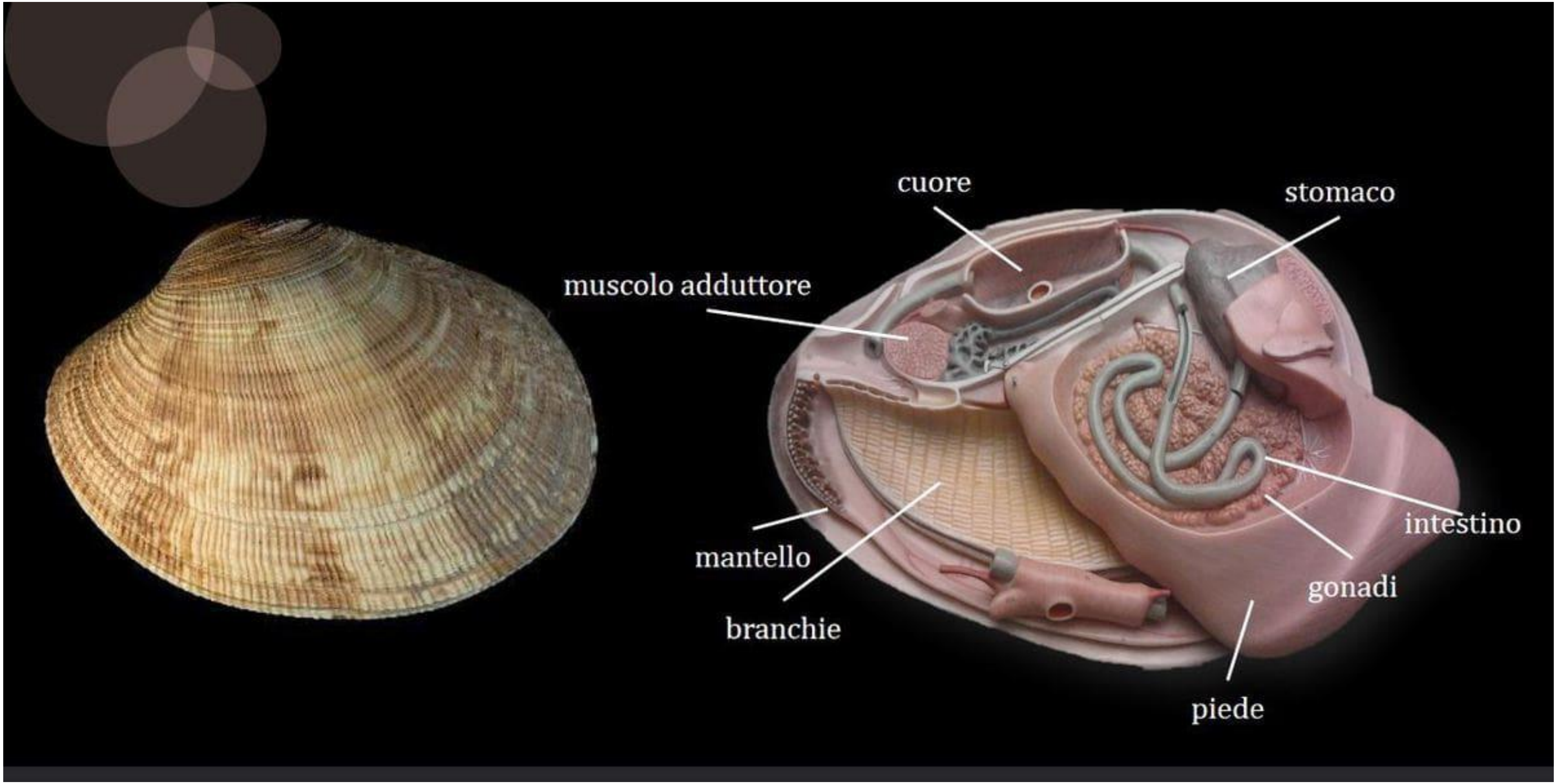
Il muscolo, dal punto di vista evolutivo, porta una certa «sfortuna»

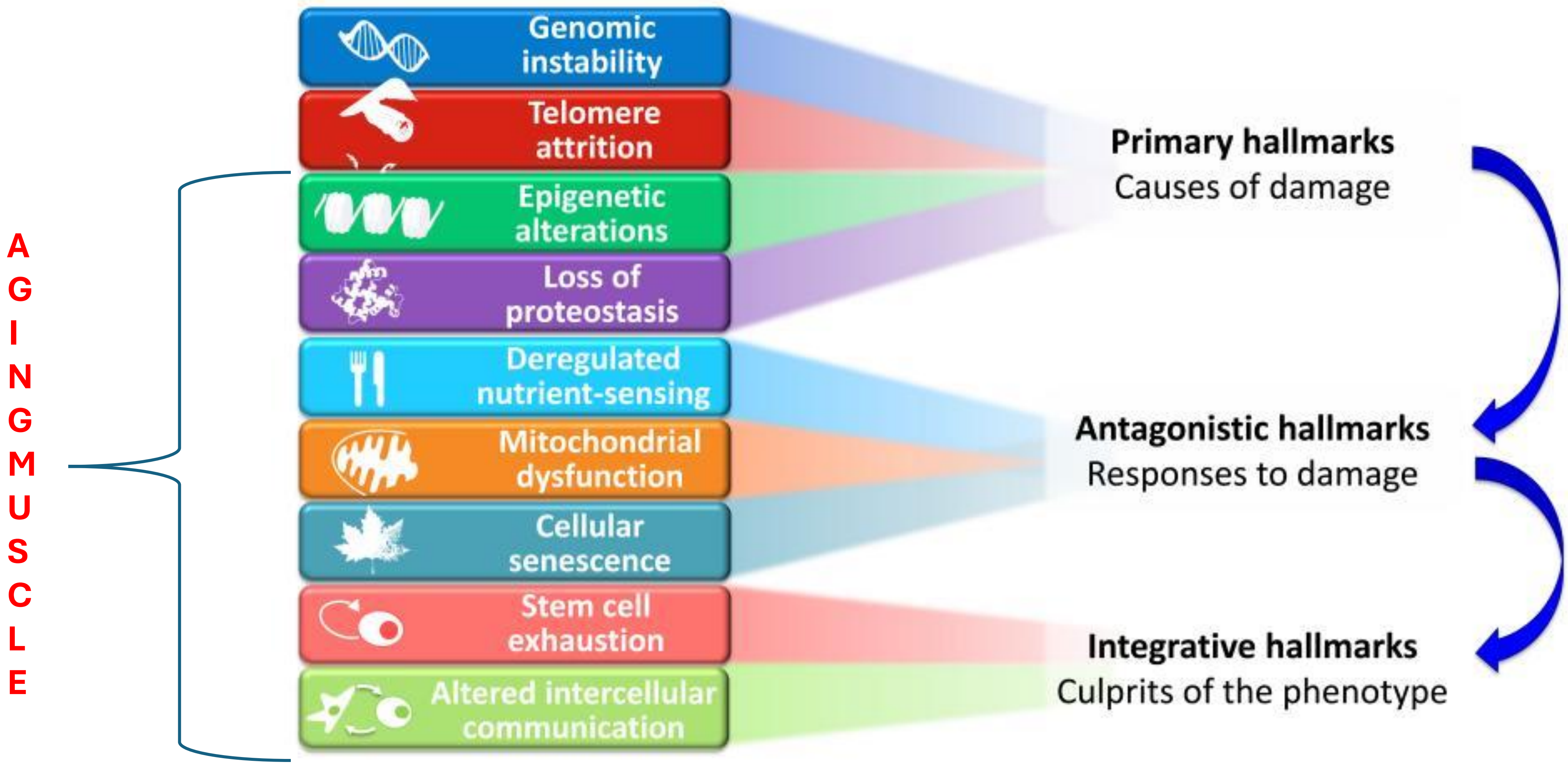
xestospongia muta

- Spugna gigante arriva a vivere 2200 anni



Questa cozza vive 450-500 anni!!


cuore
stomaco
muscolo adduttore
intestino
mantello
gonadi
branchie
piede


A
G
I
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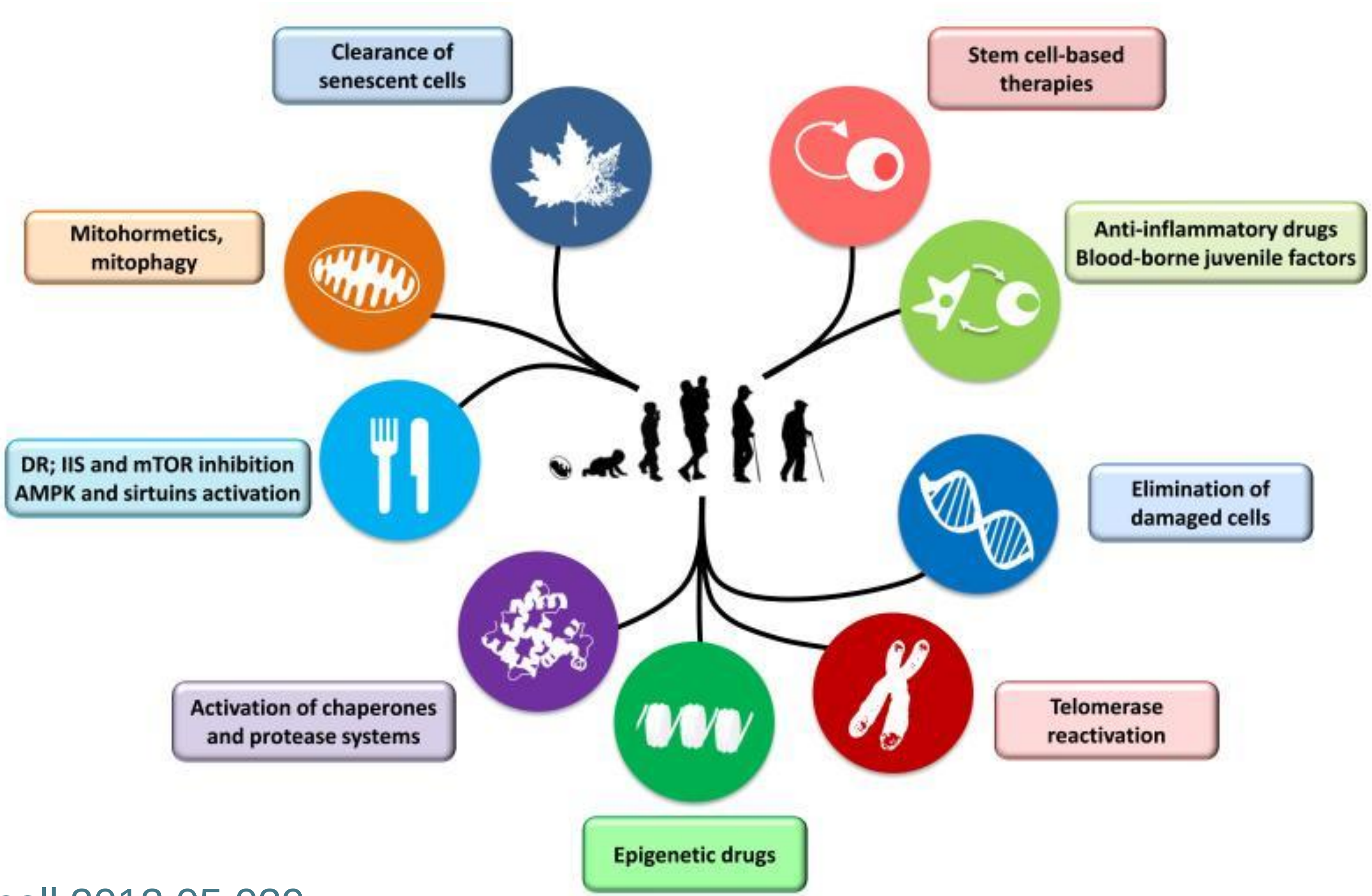
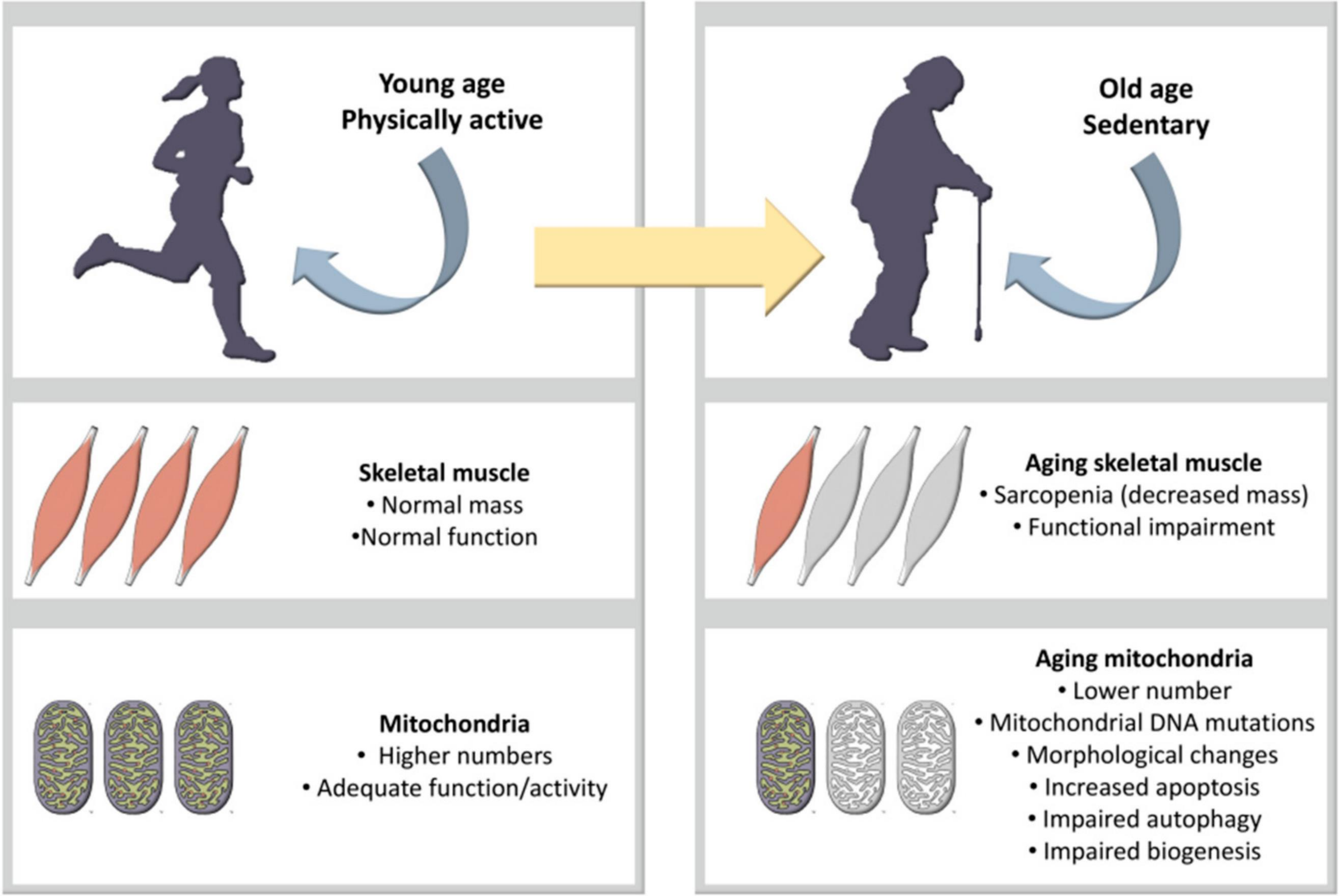
Genomic instability
Telomere attrition
Epigenetic alterations
Loss of proteostasis
Deregulated nutrient-sensing
Mitochondrial dysfunction
Cellular senescence
Stem cell exhaustion
Altered intercellular communication
Primary hallmarks
Causes of damage
Antagonistic hallmarks
Responses to damage
Integrative hallmarks
Culprits of the phenotype

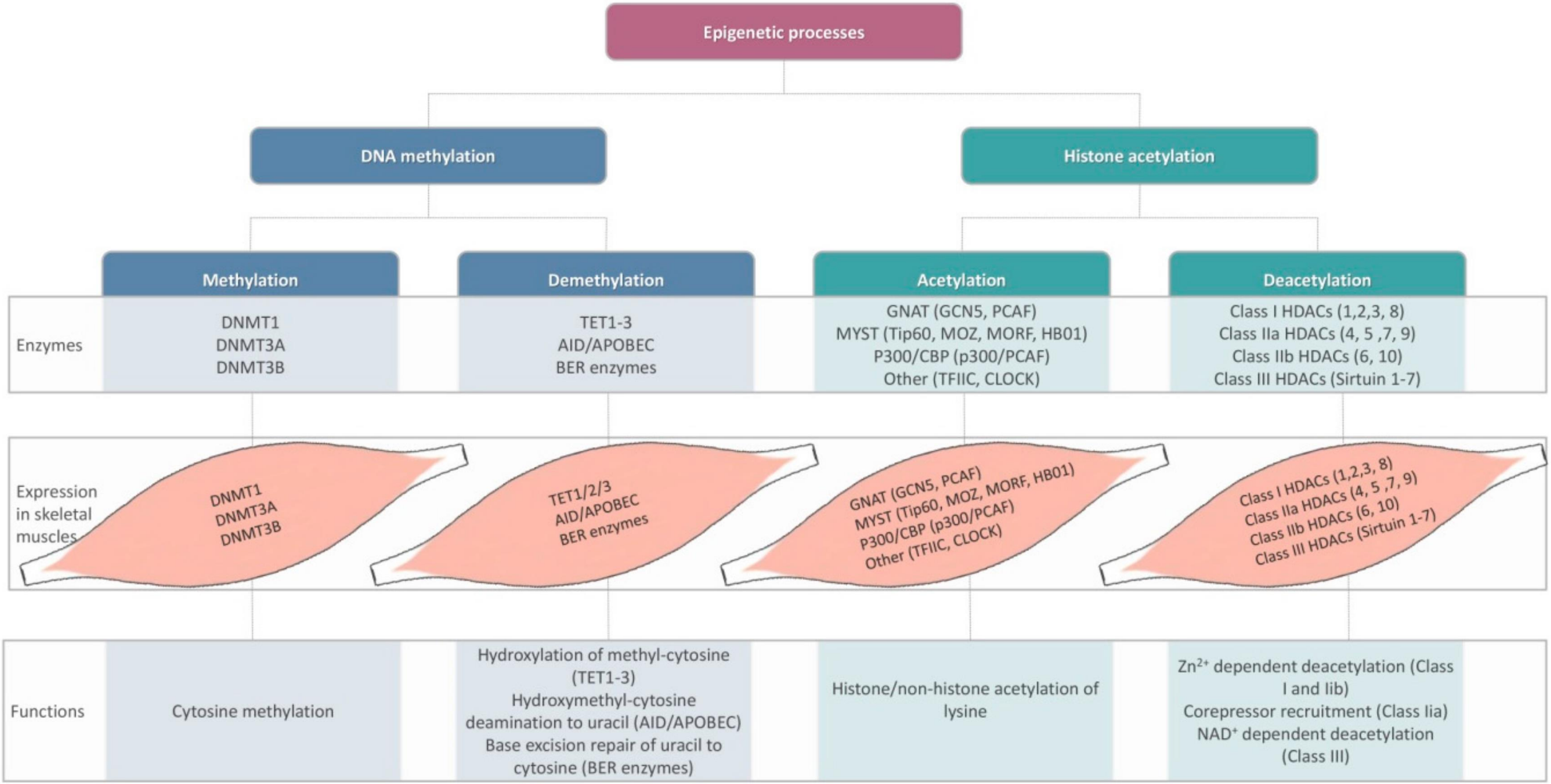
Future og Anti-aging Medicine



DOI: 10.1016/j.cell.2013.05.039



https://doi.org/10.3390/ijms21020592



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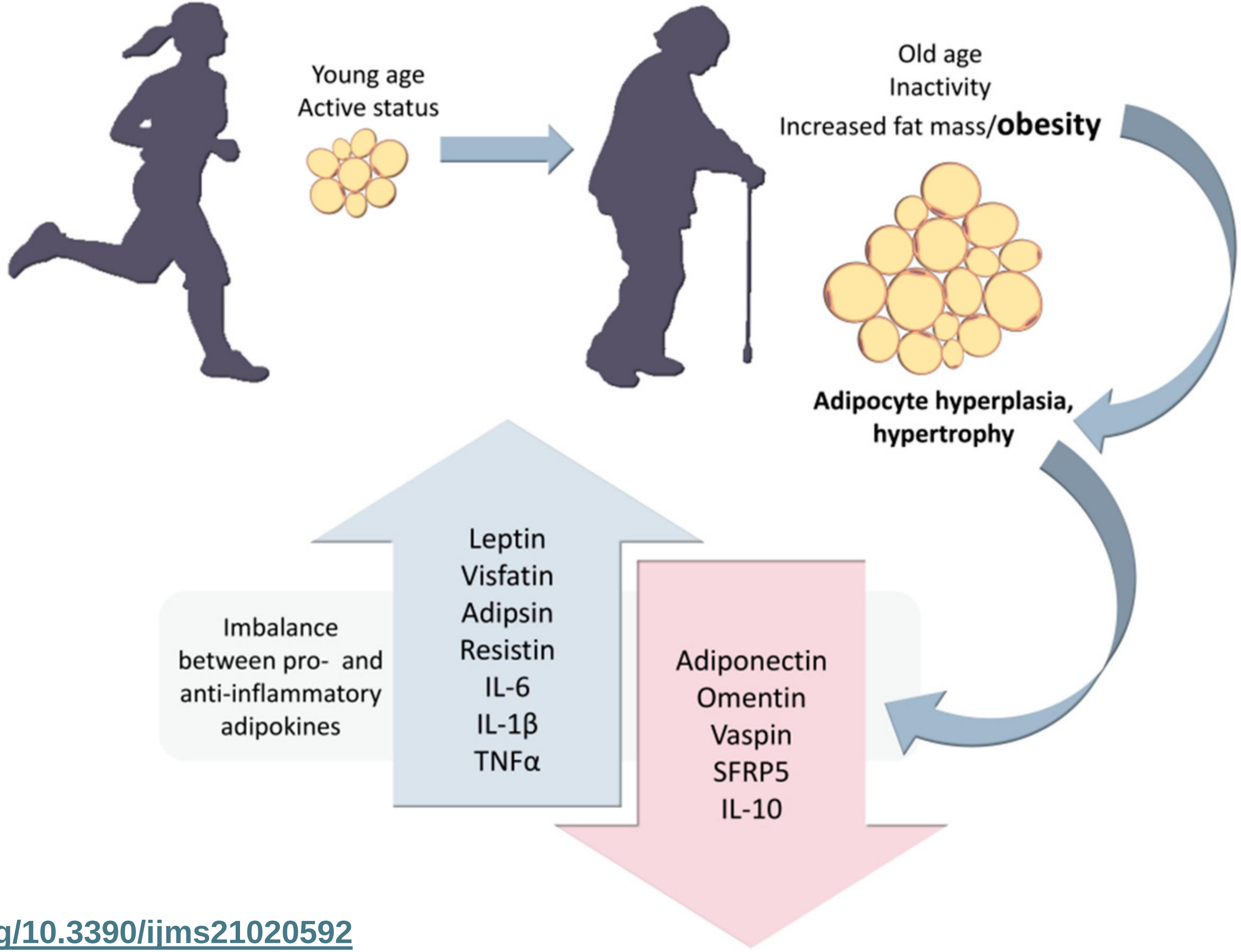

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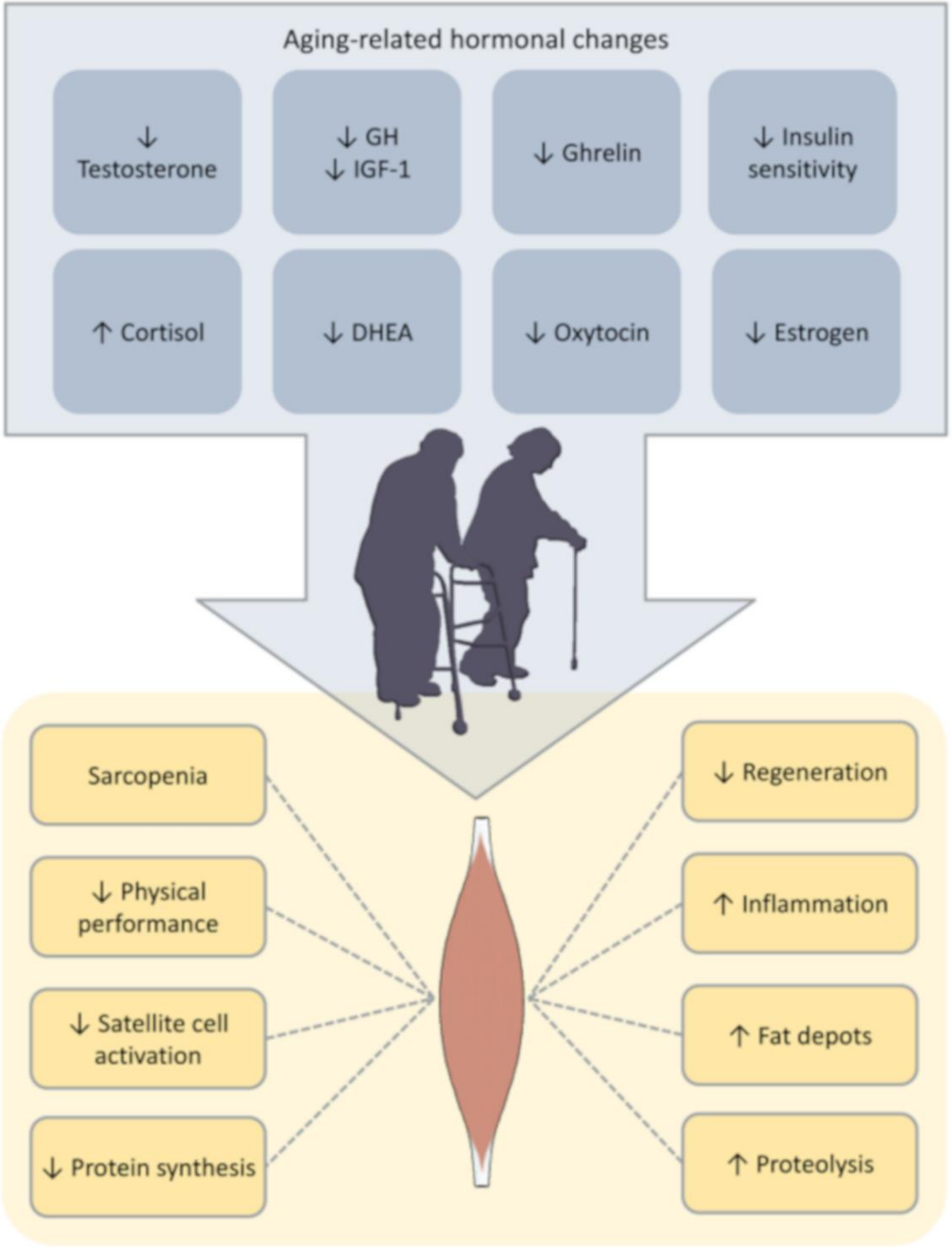
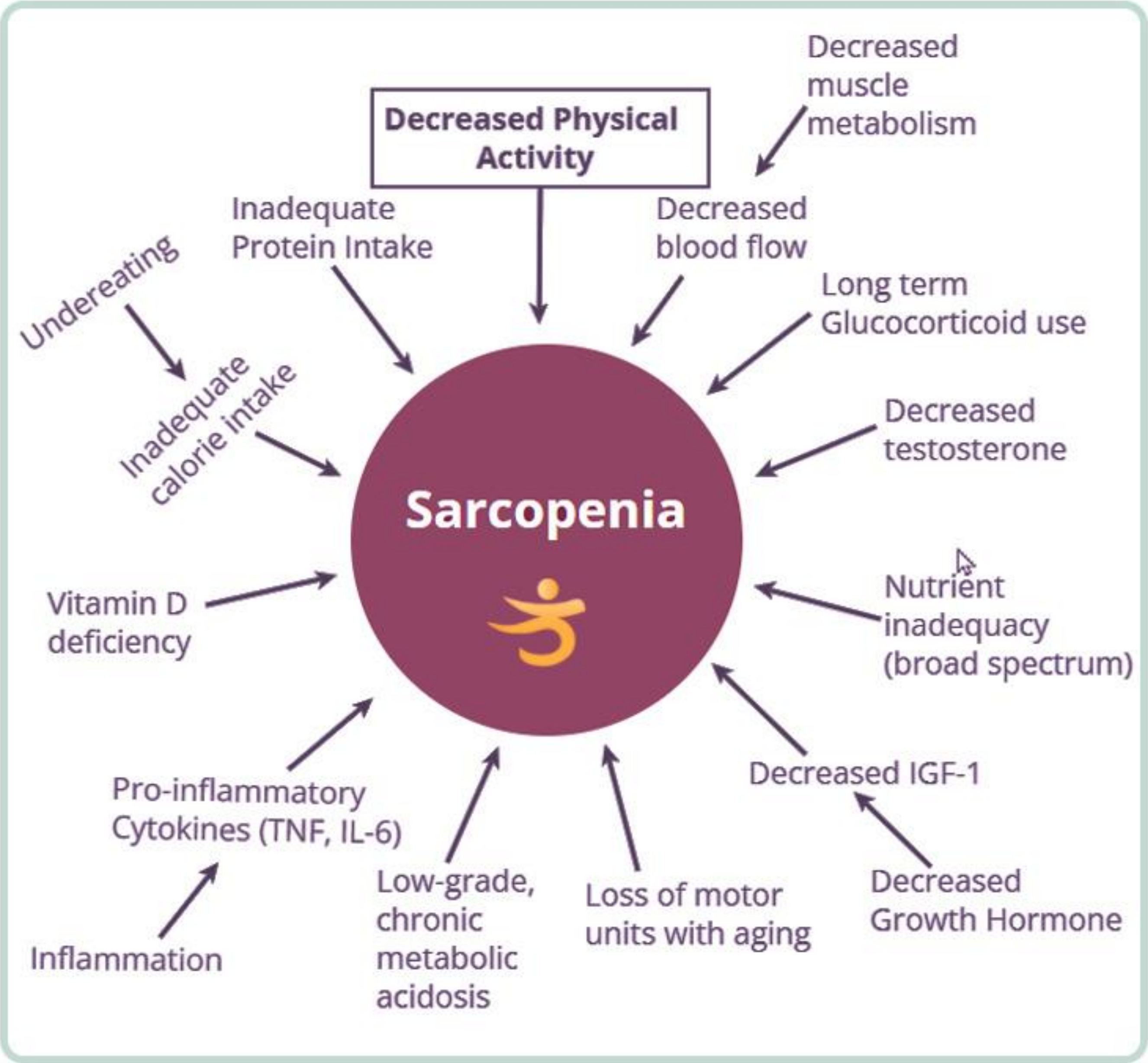


https://doi.org/10.3390/ijms21020592

The muscle loss cascade of sarcopenia



https://www.womenshealthnetwork.com/bone-health/are-you-at-risk-for-sarcopenia-muscle-loss-aging/

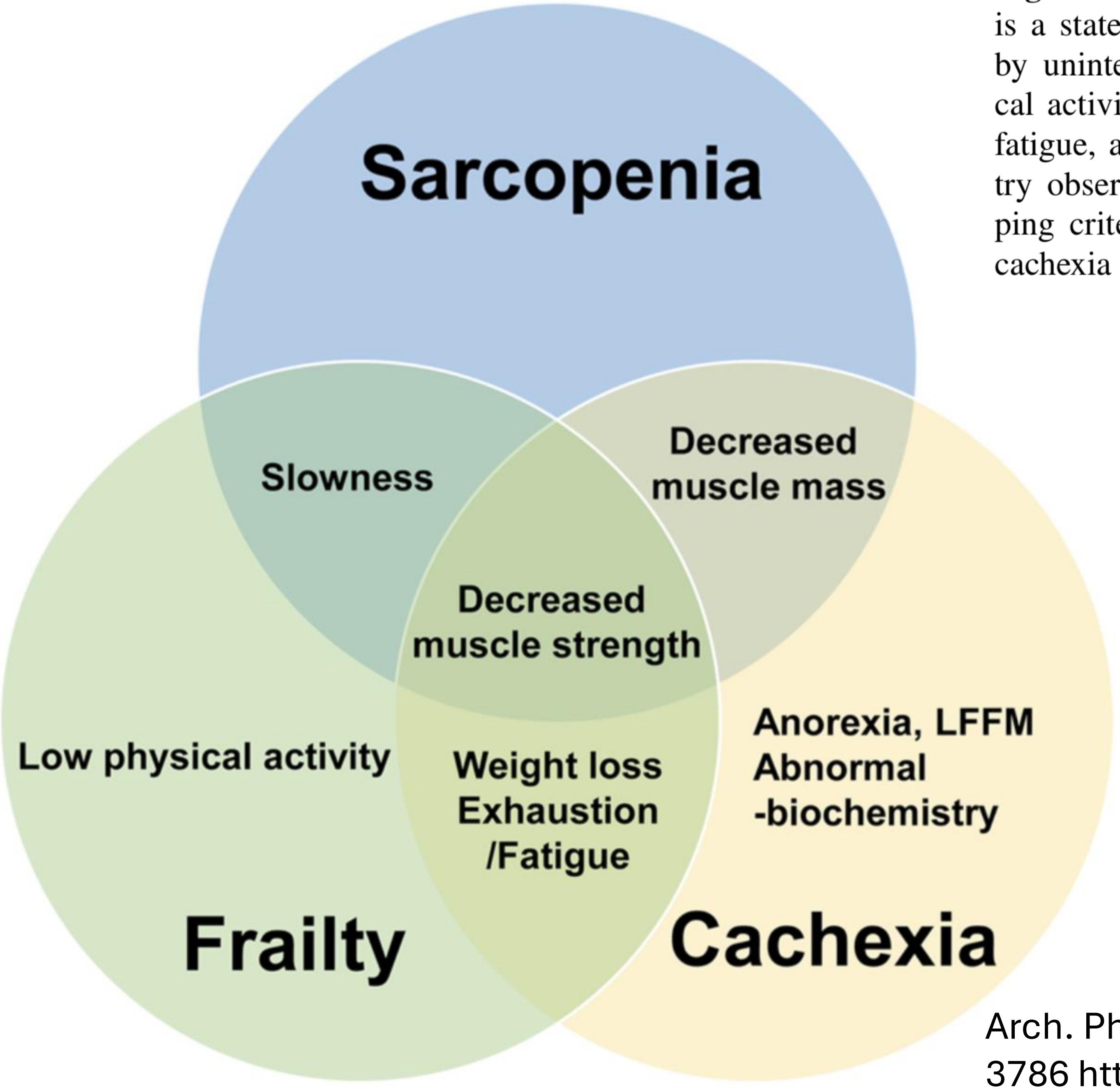


Fig. 1 Diagram of defining sarcopenia, frailty and cachexia. Frailty is a state of declined functions of multiple organ systems followed by unintentional weight loss, exhaustion, slowness and low physical activity. Cachexia is associated with decreased muscle strength, fatigue, anorexia, low fat-free mass index and abnormal biochemistry observed in cancer, AIDS, or end-stage organ failure. Overlapping criteria have been noticed for defining sarcopenia, frailty and cachexia

Arch. Pharm. Res. (2021) 44:876–889 Online ISSN 1976-3786 https://doi.org/10.1007/s12272-021-01349-z

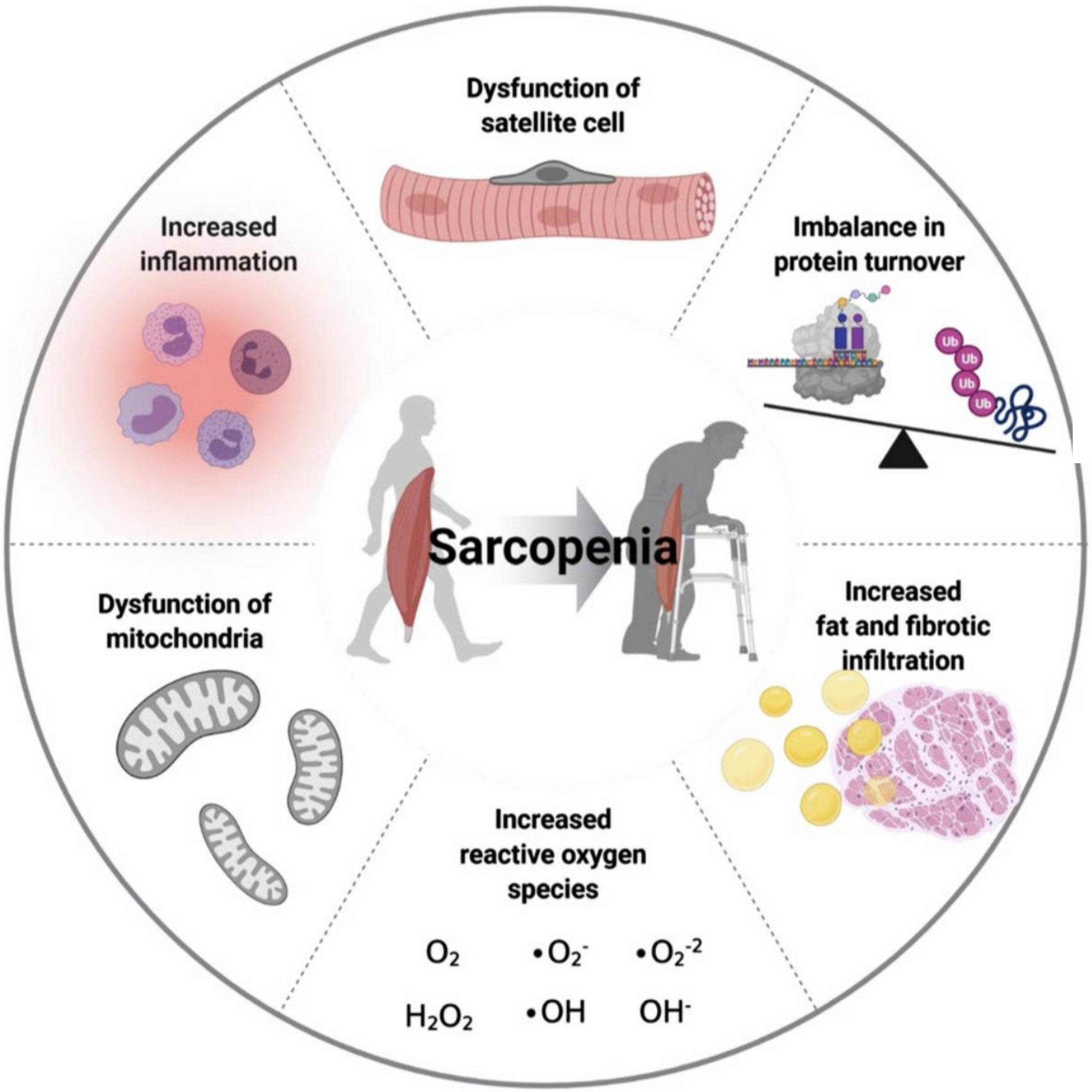


Fig. 2 Age-related factors causing sarcopenia. Decreased self-renewal and differentiating capacity of satellite cells cause impaired muscle regeneration. Increased protein degradation and decreased protein synthesis lead reduced muscle mass. Fatty and fibrotic accumulation cause poor muscle quality. Increased ROS induces oxidative stress leading muscle loss and strength. Associated with excessive ROS, dysfunction of mitochondria causes reduced ATP production. Lastly, elevated inflammation also induces oxidative stress and anabolic resistance leading loss of muscle. Created with BioRender.com

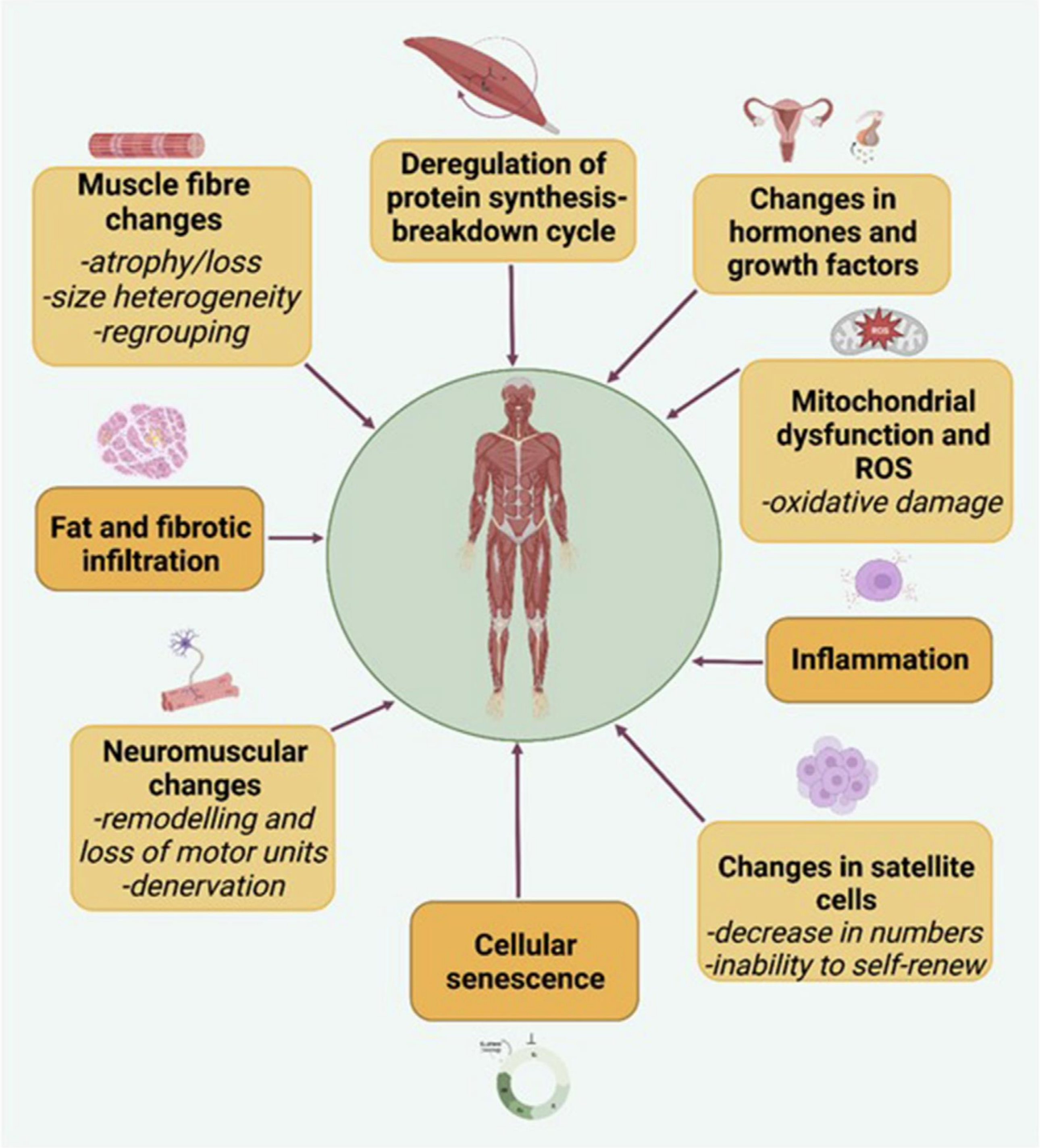


FIGURE 1. Mechanisms implicated in the pathogenesis of sarcopenia.Mechanisms of sarcopenia are complex and include skeletal muscle fiber atrophy, imbalance of muscle protein synthesis and breakdown, mitochondrial dysfunction and accumulation of ROS, and neuromuscular changes. (Created with BioRender.com). ROS, reactive oxygen species.

https://doi.org/10.1016/j.ajcnut.2023.08.015

C. Nelke, R. Dziewas and J. Minnerup et al. / EBioMedicine 49 (2019) 381–388

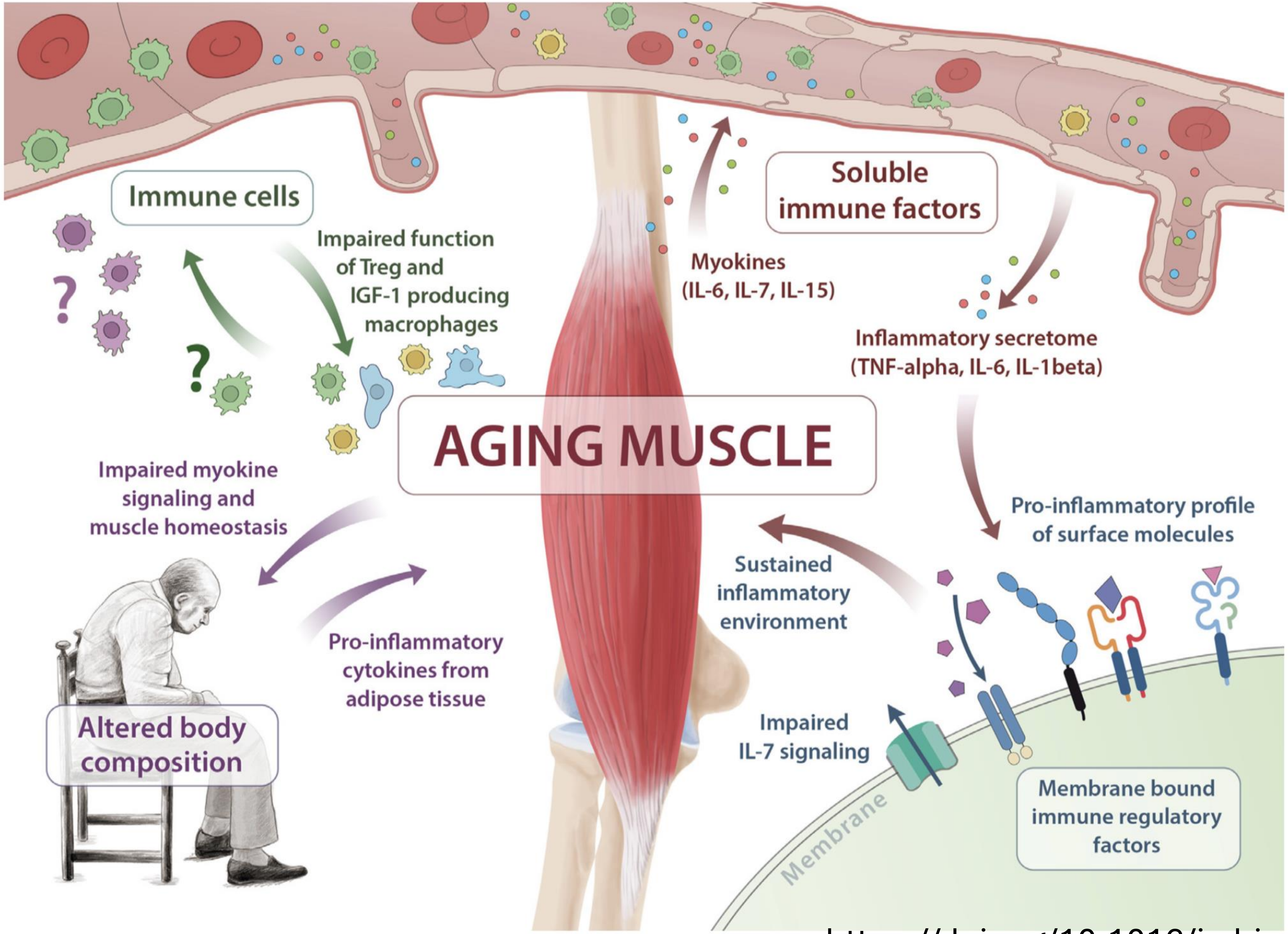


Fig. 1. Aging of skeletal muscle is central in the pathogenesis of immune senescence and sarcopenia. Multiple pathways are affected, including insufficient myokine signalling (IL-6, IL-7, IL-15), shifting of membrane bound immune regulatory factors towards a pro-inflammatory profile, impaired immune cell function and altered body composition.

https://doi.org/10.1016/j.ebiom.2019.10.034

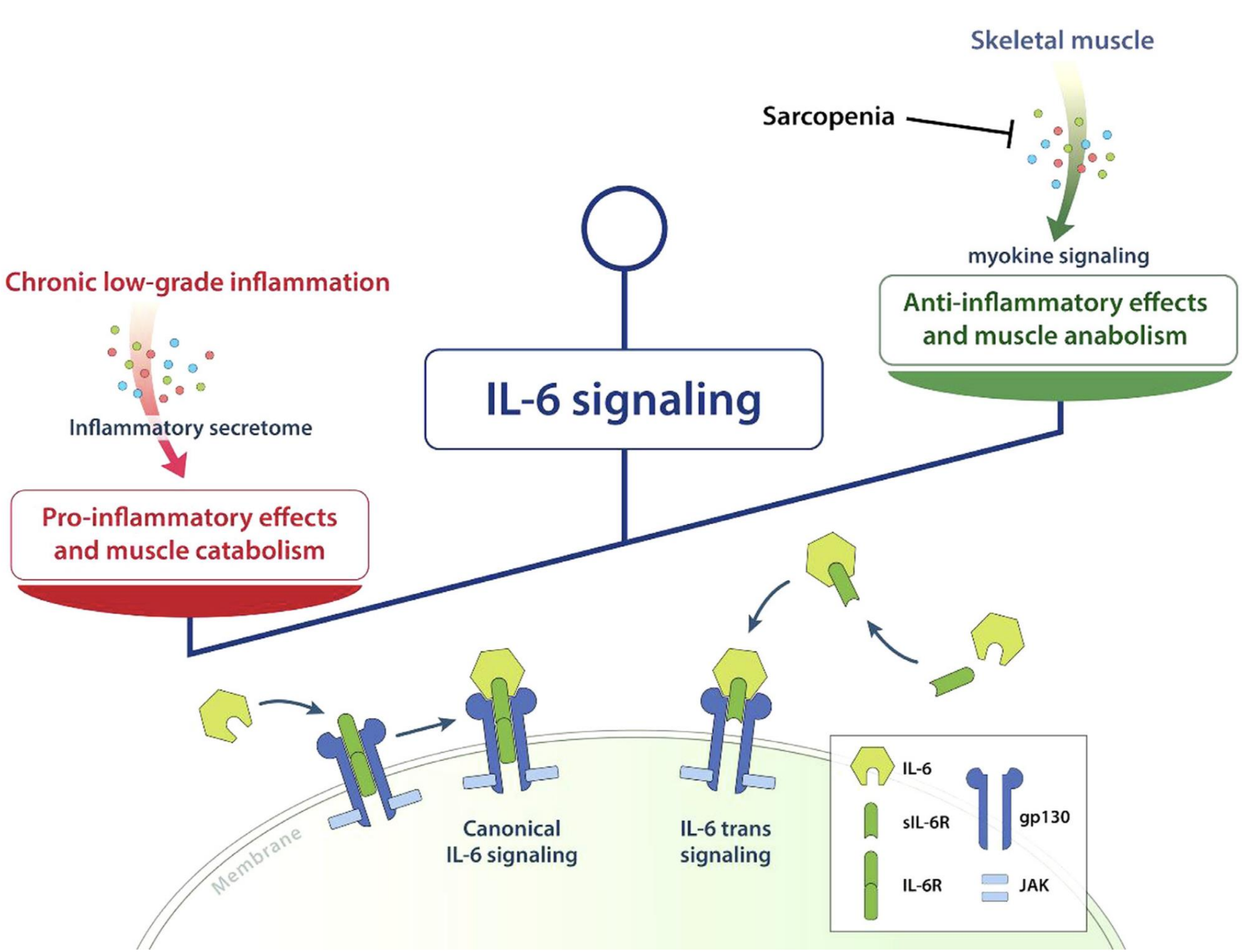
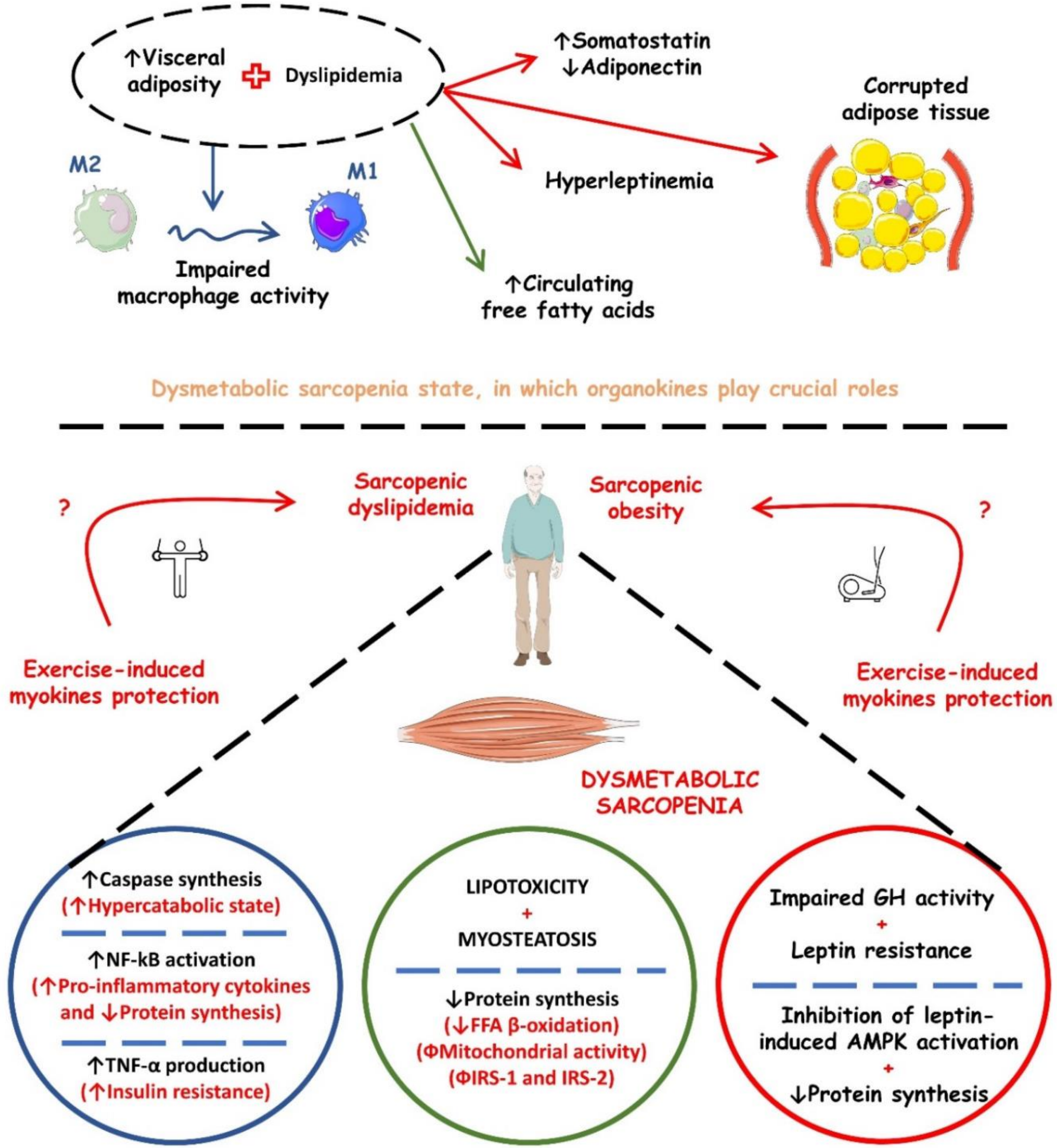
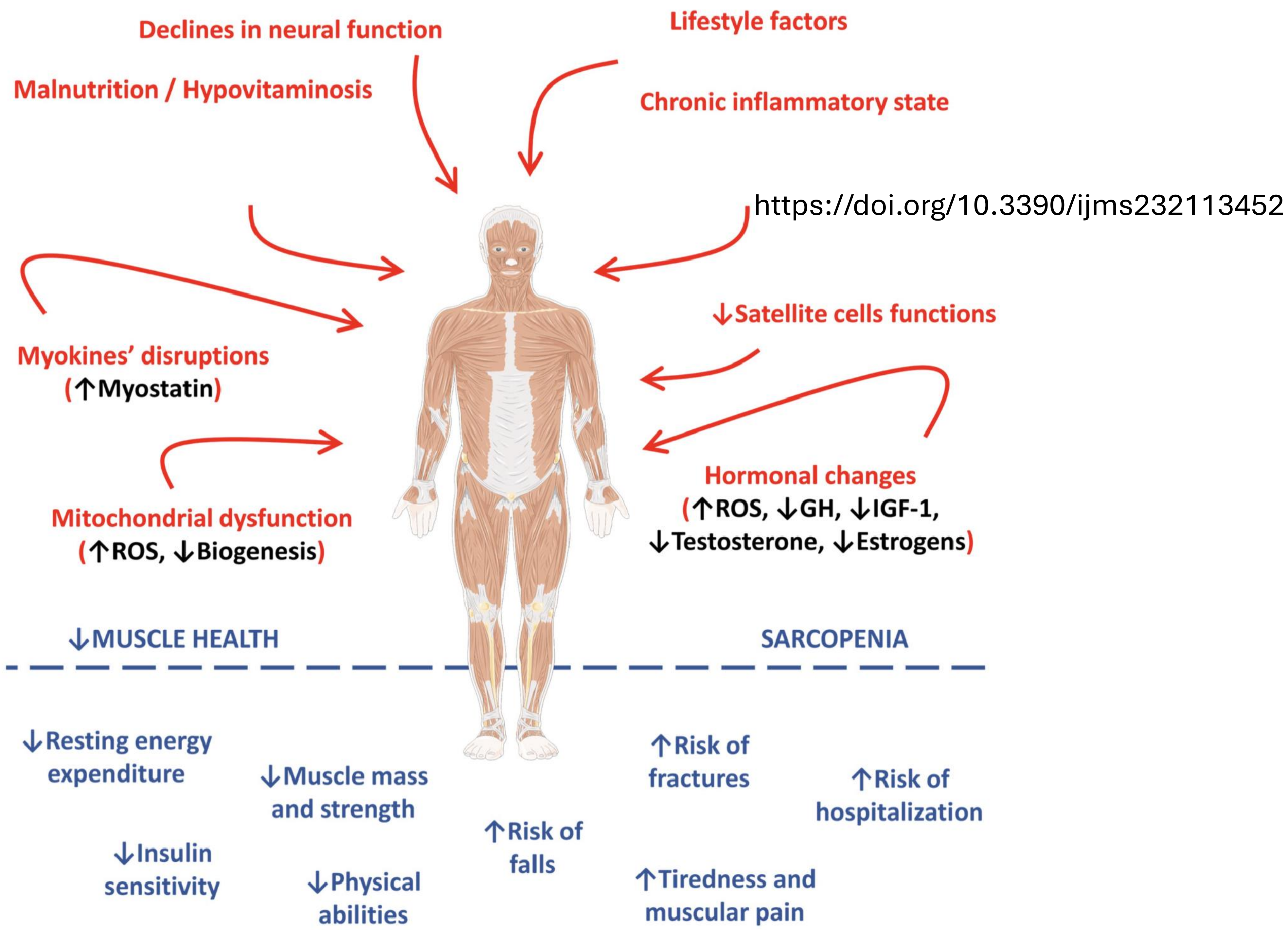
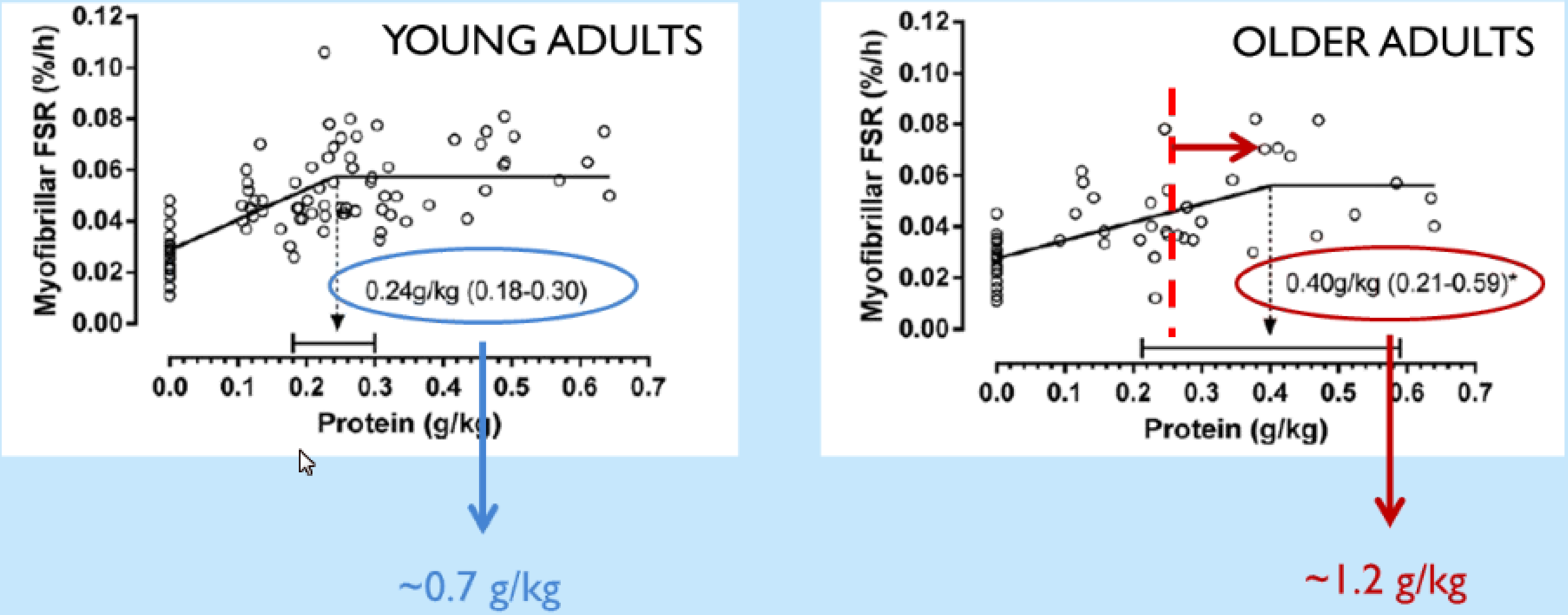


Fig. 2. Aging tips the scales of IL-6 signalling. Chronic exposure to IL-6 and the concomitant release of pro-inflammatory cytokines promote pro-inflammatory effects and muscle catabolism due to IL-6 signalling. The pulsatile release of IL-6 in response to exercise is impaired in sarcopenia resulting in reduced anti-inflammatory effects and impaired muscle anabolism mediated by IL-6. The biological effect of IL-6 is mediated both by canonical and by trans-signalling.


↑Visceral adiposity
Dyslipidemia
↑Somatostatin
↓Adiponectin
Corrupted adipose tissue
M2
M1
Hyperleptinemia
Impaired macrophage activity
↑Circulating free fatty acids
Dysmetabolic sarcopenia state, in which organokines play crucial roles
?
Sarcopenic dyslipidemia
Sarcopenic obesity
?
Exercise-induced myokines protection
Exercise-induced myokines protection
DYSMETABOLIC SARCOPENIA
↑Caspase synthesis
(↑Hypercatabolic state)
↑NF-kB activation
(↑Pro-inflammatory cytokines and ↓Protein synthesis)
↑TNF-α production
(↑Insulin resistance)
LIPOTOXICITY
+
MYOSTEATOSIS
↓Protein synthesis
(↓FFA β-oxidation)
(ΦMitochondrial activity)
(ΦIRS-1 and IRS-2)
Impaired GH activity
+
Leptin resistance
Inhibition of leptin-induced AMPK activation
+
↓Protein synthesis


Declines in neural function
Lifestyle factors
Malnutrition / Hypovitaminosis
Chronic inflammatory state
https://doi.org/10.3390/ijms232113452
↓Satellite cells functions
Myokines' disruptions
(↑Myostatin)
Hormonal changes
(↑ROS, ↓GH, ↓IGF-1,
↓Testosterone, ↓Estrogens)
Mitochondrial dysfunction
(↑ROS, ↓Biogenesis)
↓MUSCLE HEALTH
SARCOPENIA
↓Resting energy expenditure
↓Muscle mass and strength
↑Risk of fractures
↑Risk of hospitalization
↑Risk of falls
↓Insulin sensitivity
↓Physical abilities
↑Tiredness and muscular pain

ANABOLIC RESISTANCE WITH AGING



Moore DR *et al. J Gerontol A Biol Sci Med Sci.* 2015;**70**:57-62.

Resistenza anaerobica

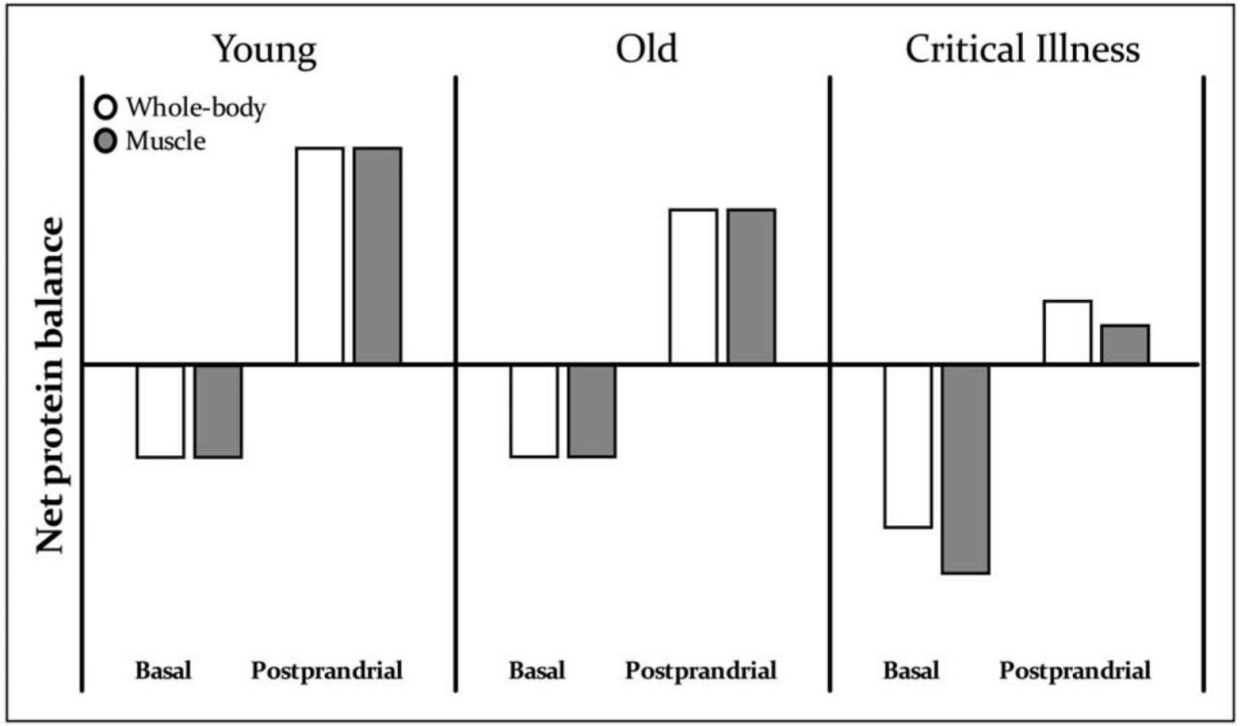


FIGURE 1. Net whole-body and muscle protein balance in young, old, and critically ill persons. There is no change in basal protein turnover between young and older individuals but there is a dramatic reduction in net whole-body protein balance with critical illness, primarily driven by skeletal muscle. Older individuals are anabolically resistant to hyperaminoacidemia, which is exaggerated during critical illness because of substantial disuse.

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Resistenza anaerobica

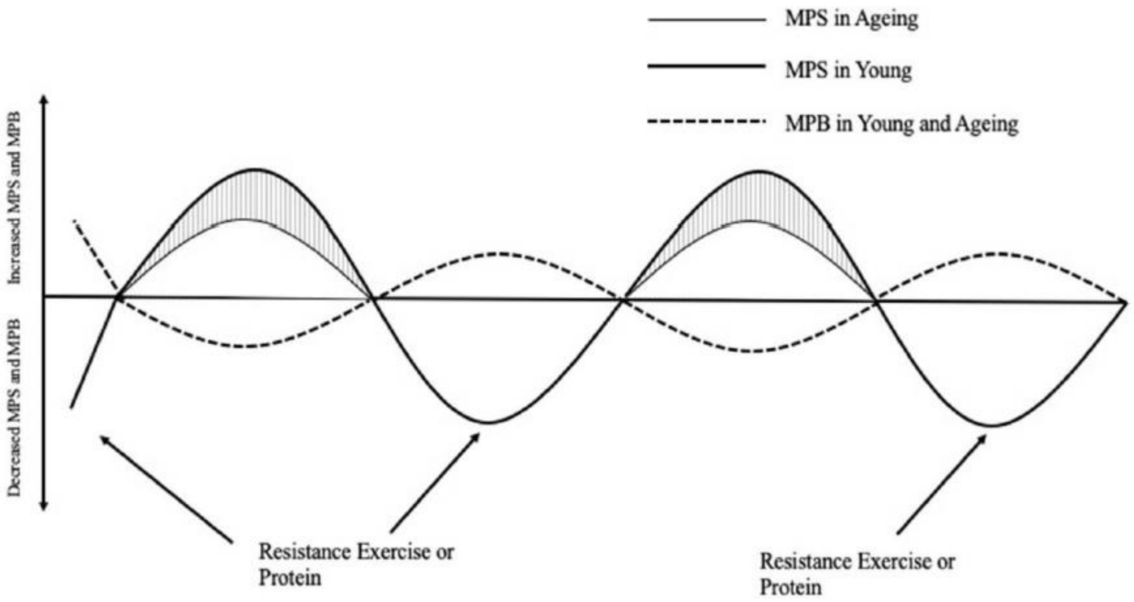


FIGURE 1 | The response of muscle protein synthesis (MPS) and breakdown (MPB) on net protein balance after acute resistance exercise or protein ingestion in young and aging populations [Adapted from Breen and Phillips (12)]. In the morning after an overnight fast, muscle protein breakdown exceeds muscle protein synthesis such that net protein balance is negative. After a bout of resistance exercise or the ingestion of protein, young people respond greater in their myofibrillar protein synthesis response compared to aging people, which is appears to be the major attenuating factor to decreased NPB leading to skeletal muscle protein loss over time. MPS, Muscle protein synthesis; MPB, Muscle protein breakdown.

Resistenza anaerobica

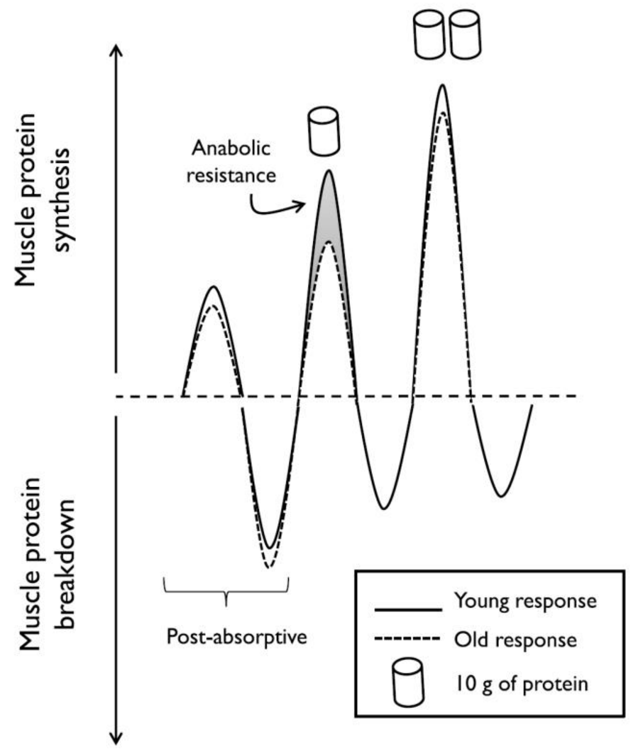
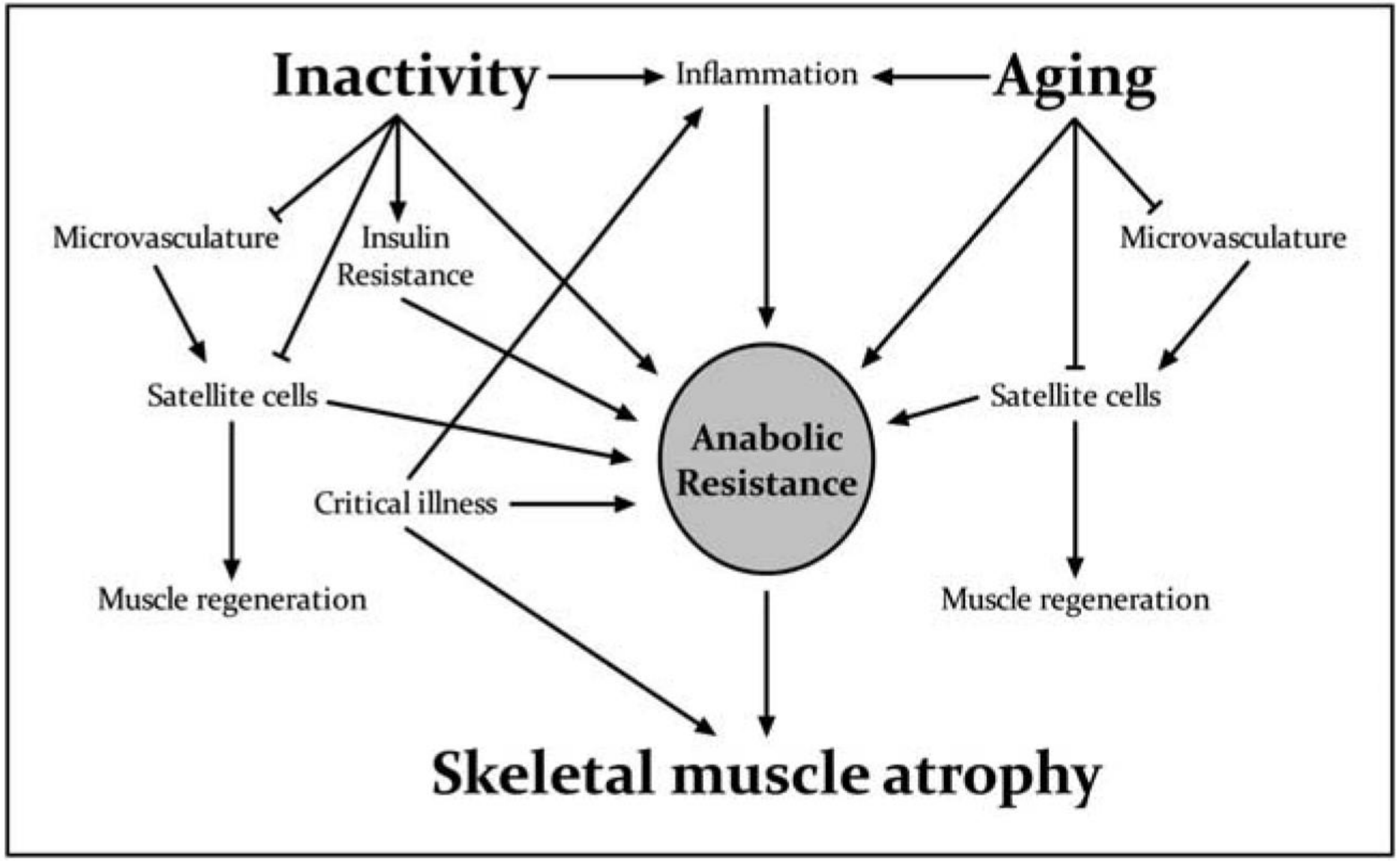


Figure 3. Post–absorptive muscle protein synthesis and muscle protein breakdown rates do not differ between the healthy young and old. Anabolic resistance of muscle protein synthesis rates may arise after consumption of smaller amounts of dietary protein. These postprandial differences between the young and old are no longer evident after consumption of ample amounts of dietary protein. Note that protein synthesis and breakdown simultaneously occur in a physiological system.

Burd NA Exerc Sport Sci Rev 2013

Resistenza anaerobica: meccanismi



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Resistenza anaerobica: meccanismi

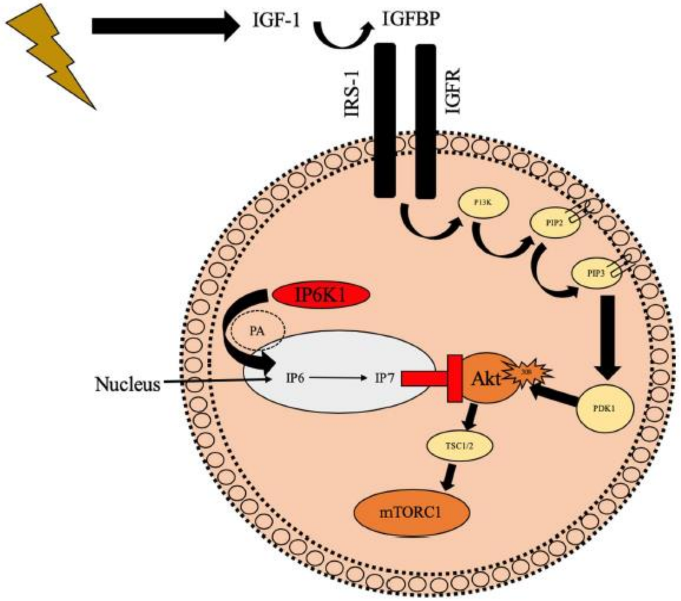

FIGURE 3 | Schematic diagram illustrating the potential negative role of IP6K1 on Akt translocation to the cell membrane preventing phosphorylation of Akt^{308} which may reduce mTORC1. IP6K1 enters the nucleus via PA and it then synthesizes IP7 from IP6 which prevents Akt from translocating to the cell membrane and ultimately preventing Akt^{308} phosphorylation. IGFBP, Insulin like growth factor binding proteins; IGF-1, Insulin like growth factor-1; IP6K1, inositol hexakisphosphate kinase 1; IGFR, Insulin like growth factor receptor; IRS-1, Insulin receptor substrate 1; P13K, phosphoinositide 3-kinase; PIP2, hosphatidylinositol (4, 5)-bisphosphate; PIP3, hosphatidylinositol 3,4,5-trisphosphate; PDK1, phosphoinositide-dependent kinase-1; Akt, Protein kinase B; mTORC2, Mechanistic target of rapamycin; PA, Phosphotadic acid; IP6, inositol hexaphosphate; IP7, Inositol pyrophosphate; Illustrates contraction of skeletal muscle; Illustrates binding/translocationto the cell membrane; Illustrates activation; Illustrates phosphorylation; Illustrates binding to PH domain and downregulating Akt; Illustrates preventing translocation to cell membrane.

Barclay RD Frontiers in Nutrition 2019

Resistenza anaerobica: possibili interventi

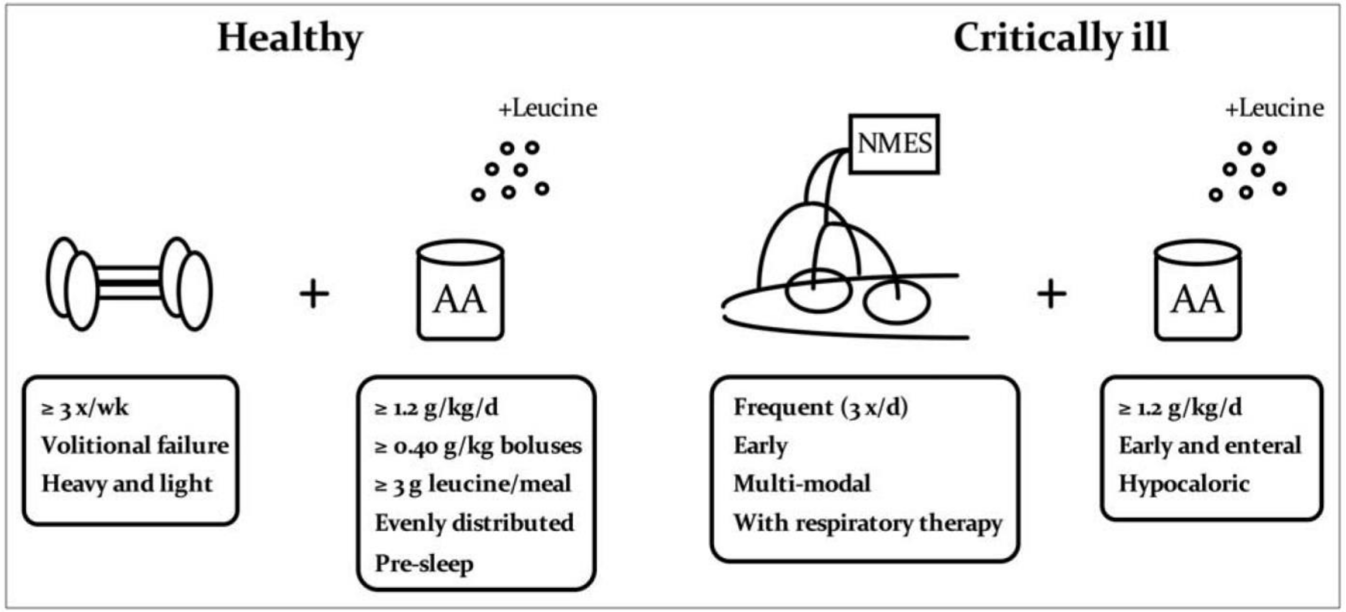

FIGURE 3. Recommendations for maintaining skeletal muscle mass in healthy and critically ill patients. AA, amino acids and NMES, neuromuscular electrical stimulation (as an example of a physical therapy to be combined with conventional therapies).

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Resistenza anabolica: le cause

- Sequestro splenico di amminoacidi
- Minore disponibilità post prandiale di amminoacidi
- Ridotta perfusione del muscolo
- Alterazione vie di signaling intracellulare
- Problemi digestivi

Resistenza anabolica: le cause

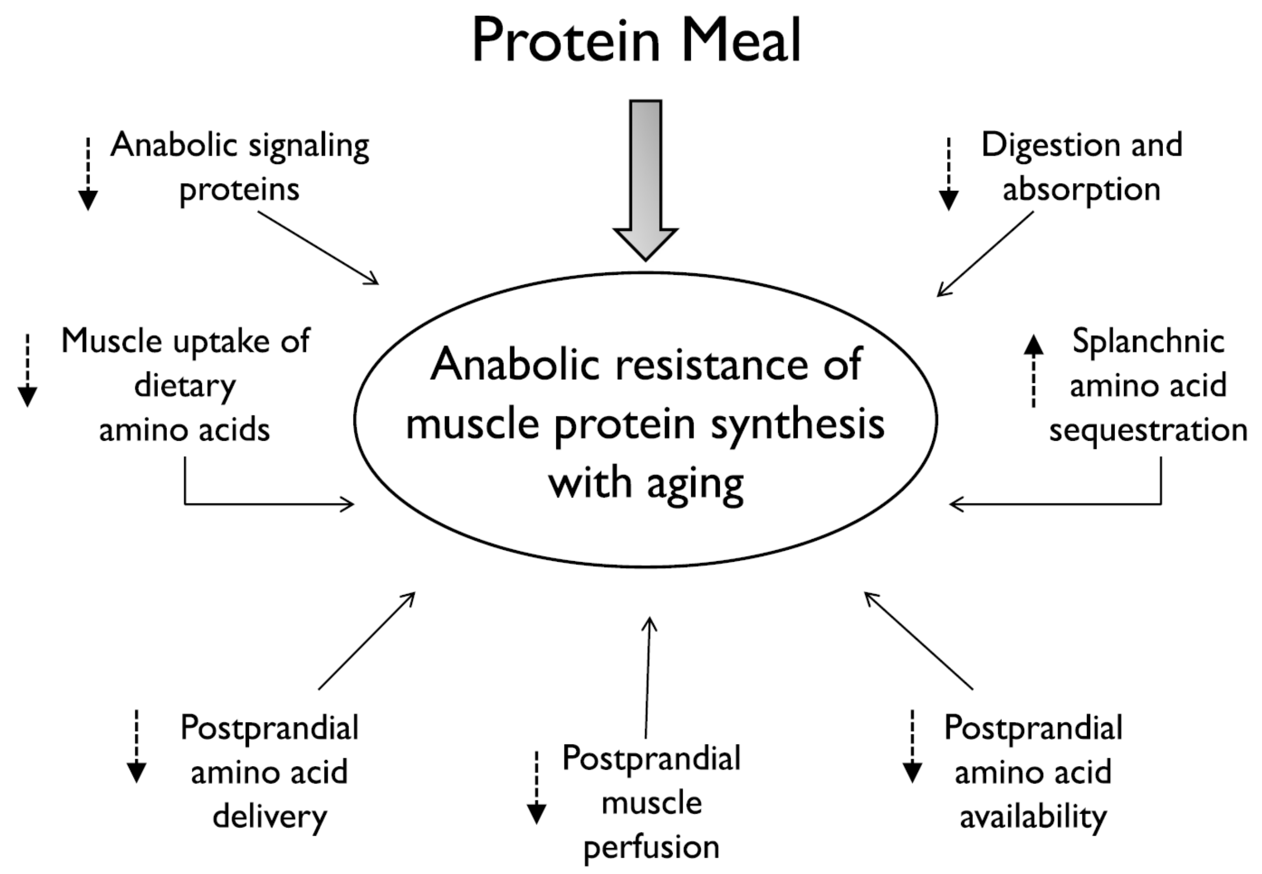


Figure 2. Protein intake stimulates muscle protein synthesis. However, a multitude of secondary factors may occur between the protein meal and the stimulation of muscle protein synthesis that may lead to anabolic resistance with aging.

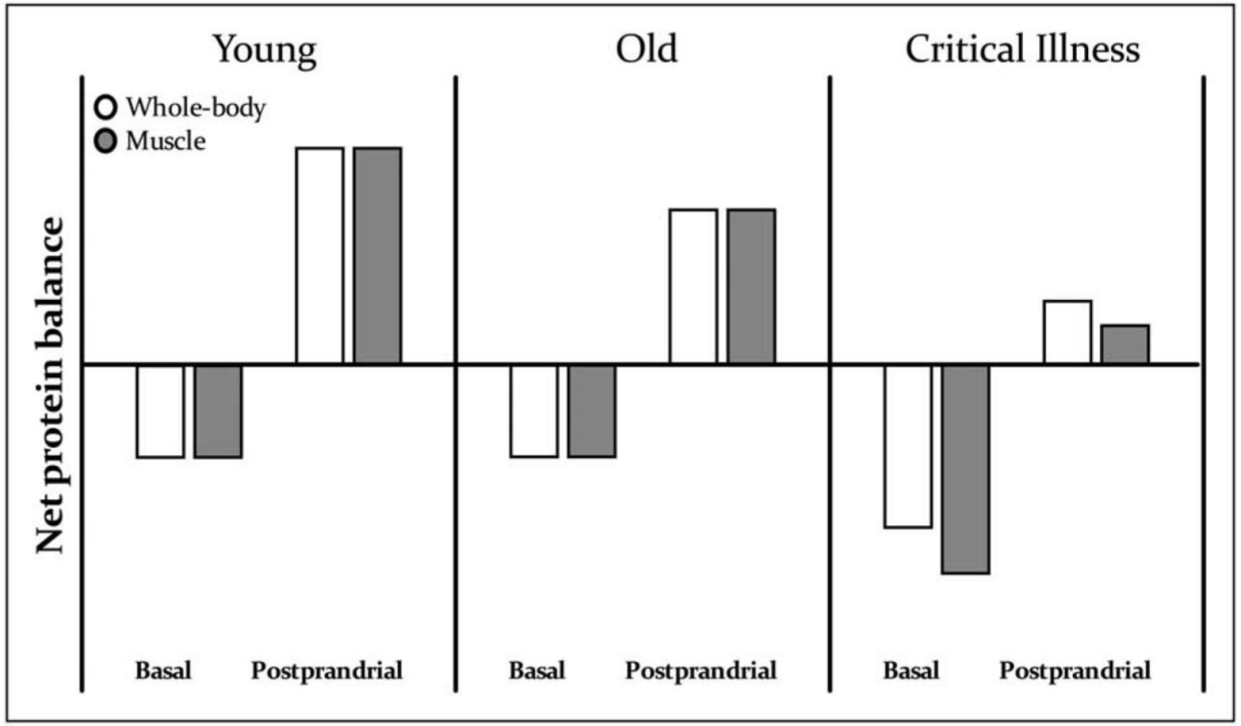
Burd NA Exerc Sport Sci Rev 2013

Amount of protein

Debate continues about whether a per-meal threshold amount of protein intake is needed to stimulate protein synthesis in older adults(41) or whether protein synthesis is linearly related to protein intake.(15) Either way, evidence suggests that older adults who consume more protein are able to maintain muscle mass and strength.(8, 10, 42, 43) Older adults who consumed 1.1 g protein/kg body weight/day lost less lean body mass (muscle) than did those who consumed only 0.7 to 0.9 g protein/kg body weight/day.(10) Among hospitalized older patients, at least 1.1 g protein/kg body weight/day was needed to achieve nitrogen balance, and safe intake was up to 1.6 g protein/kg body weight/day.(43)

Recent dietary recommendations for older adults are now including higher protein intake than for younger adults.(7, 44) The international PROT-AGE study group recommended 1.0–1.5 g protein/kg body weight/day for individuals older than 65 years(7) with or without disease, and the new Nordic Nutrition Recommendations suggest targeting 1.2–1.4 g protein/kg body weight/day with protein as 15–20% of total energy intake for healthy older adults.(44, 45)

RESISTENZA ANABOLICA



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RESISTENZA ANABOLICA: LE CAUSE

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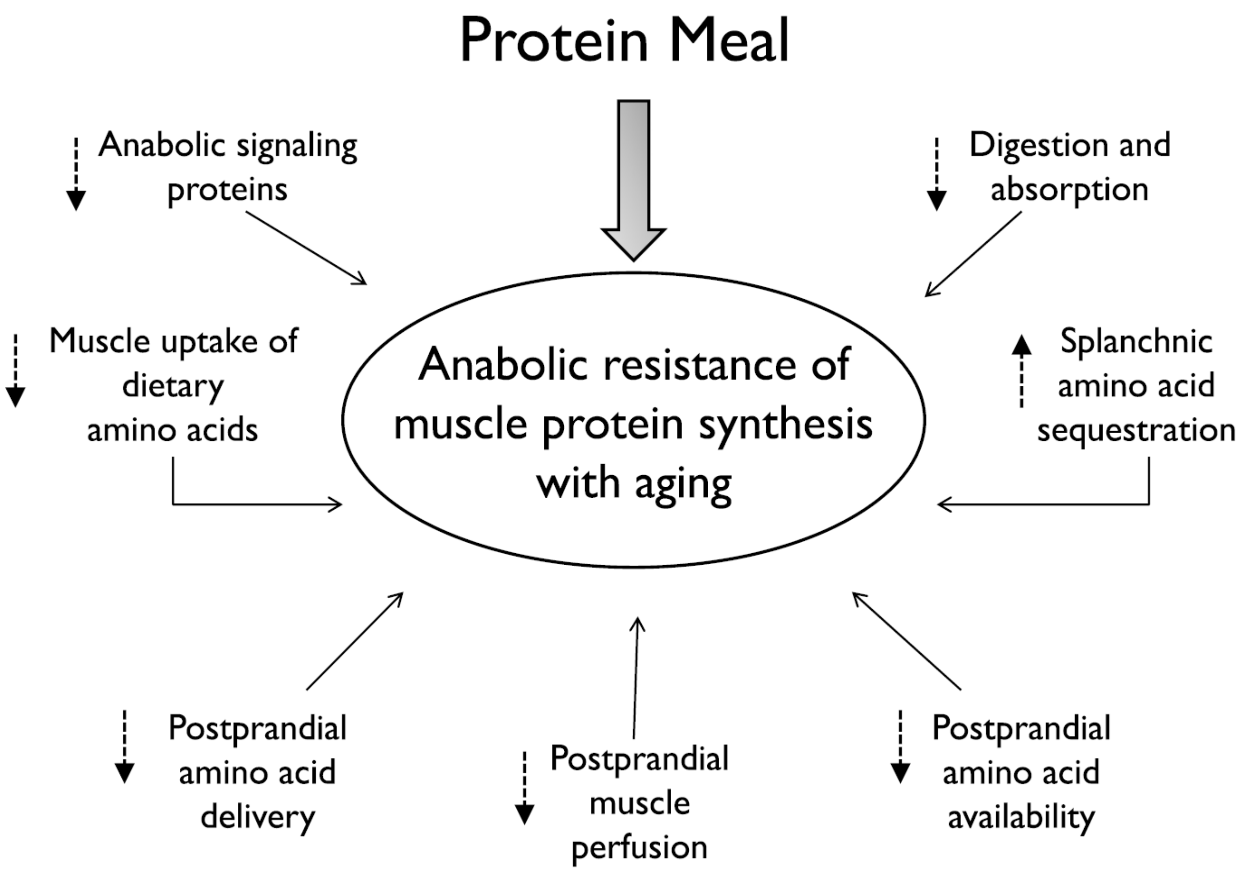
RESISTENZA ANABOLICA: LE CAUSE



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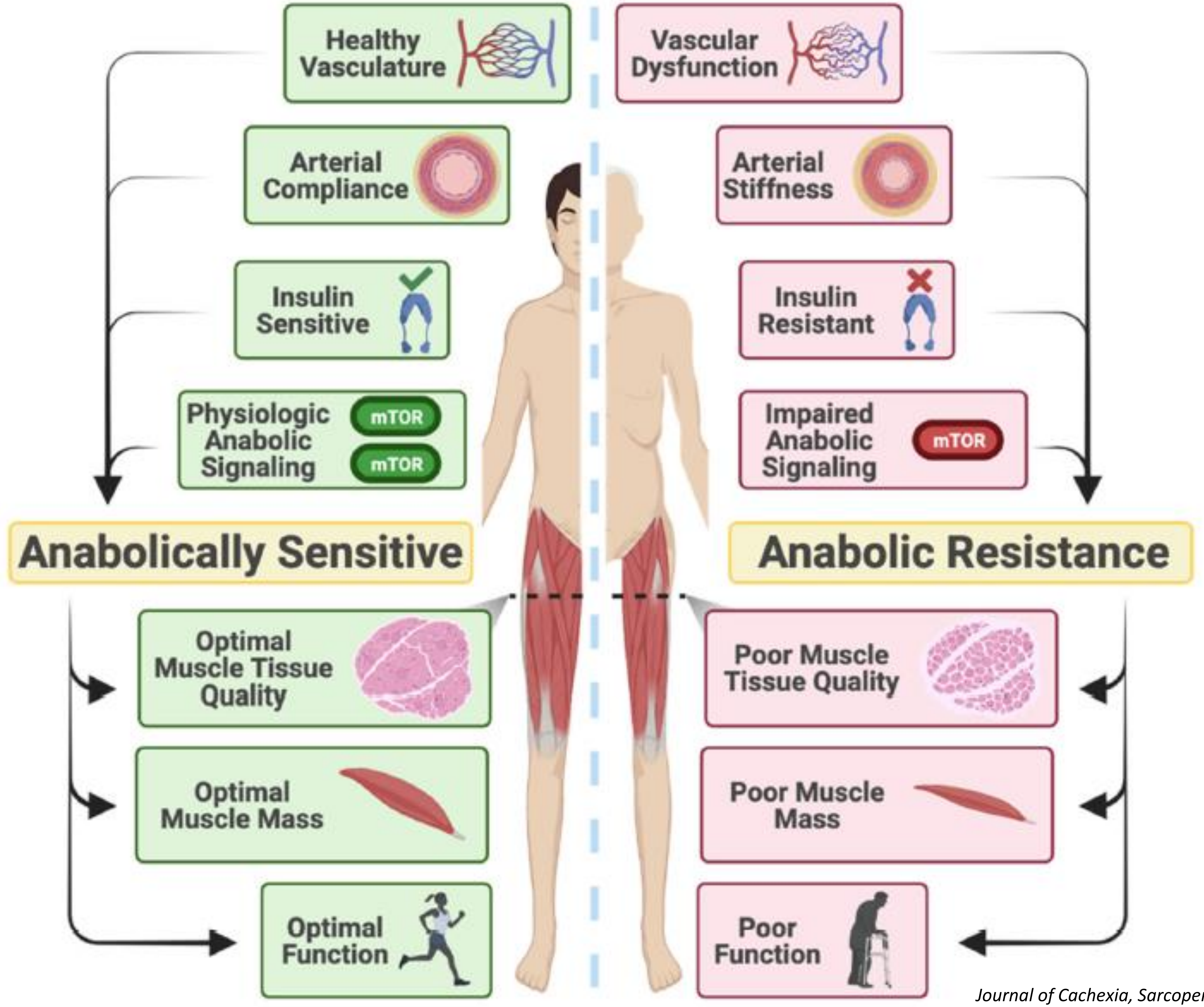
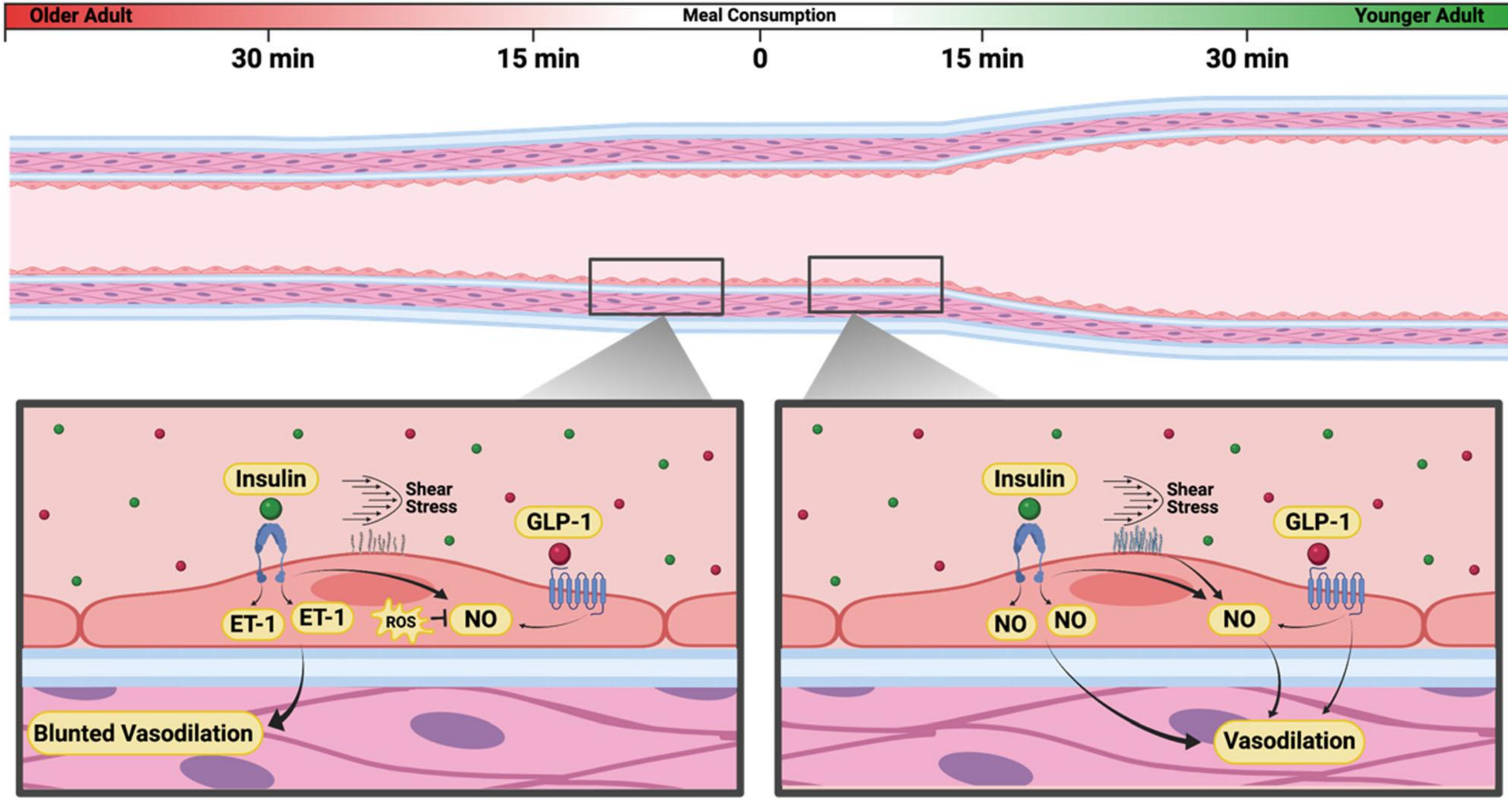


During the ageing process, there are gradual impairments in vascular function, anabolic signalling, arterial compliance, and insulin sensitivity.

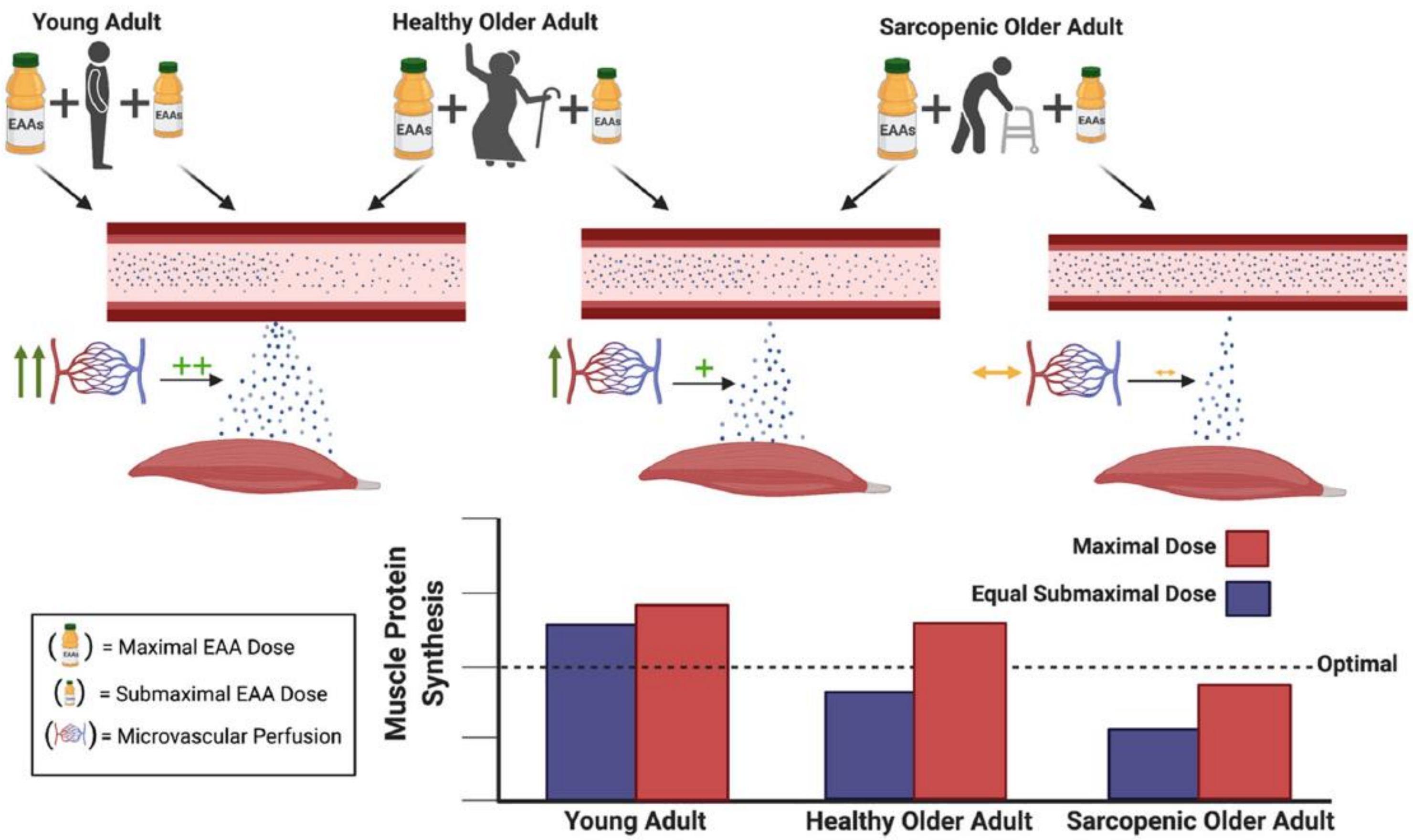
These impairments may ultimately lead and/or contribute to anabolic resistance, or a reduced ability to mount a muscle protein synthetic response to anabolic stimuli. Over time, anabolic resistance promotes sarcopenia, or the age-related loss in muscle mass and function, resulting in a loss of functional ability and independence in older adults

Journal of Cachexia, Sarcopenia and Muscle 2022; **13**: 114–127 DOI: 10.1002/jcsm.12898



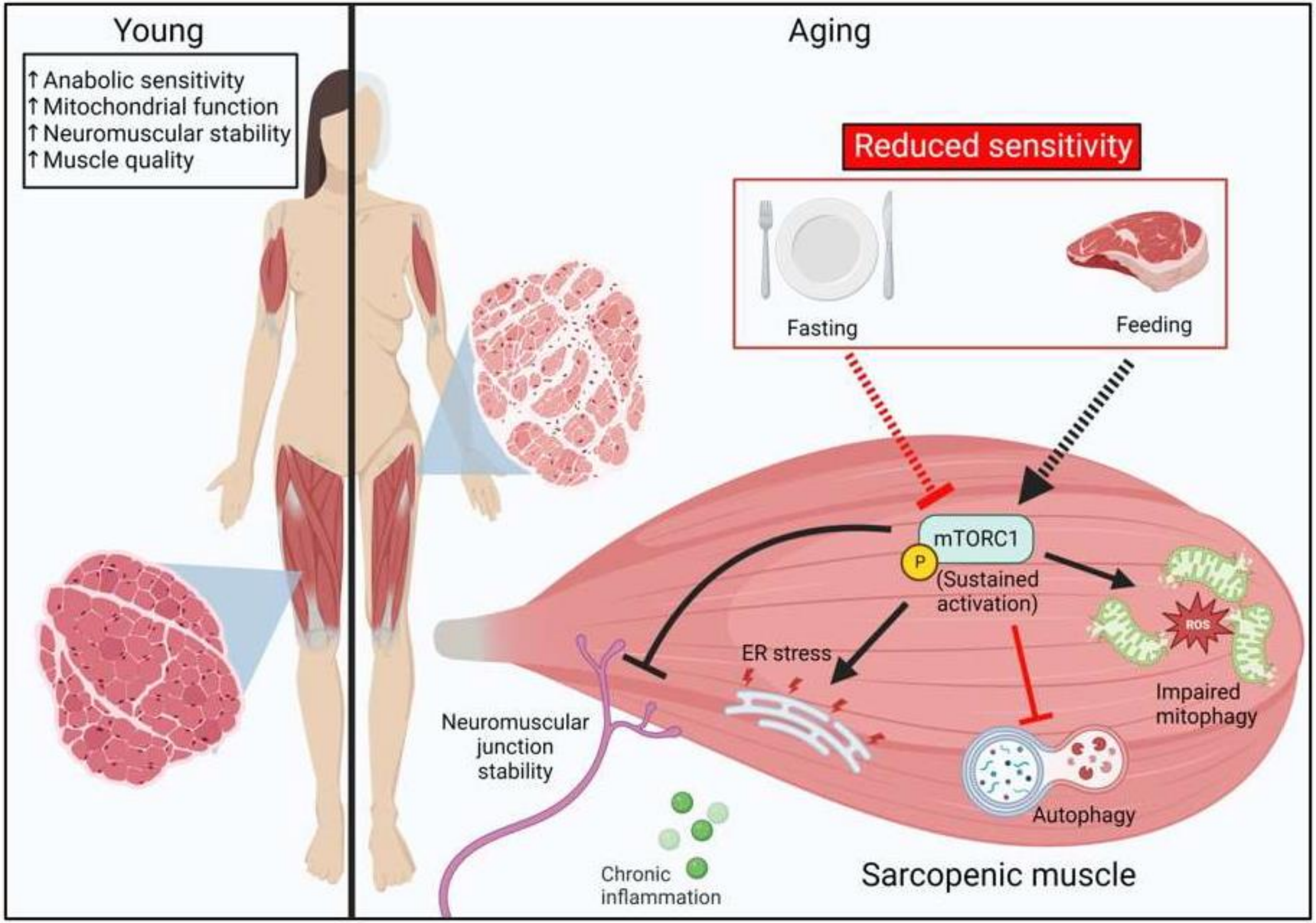
The impact of meal consumption in an older (left) and younger adult (right). In the older adult, there is an increased tendency for insulin to stimulate endothelin-1 (ET-1) release from the vascular endothelium, rather than nitric oxide (NO) as typically observed in healthy younger adults. Additionally, shear stress subsequent to an increase in blood flow stimulates the glycocalyx to release NO from the vascular endothelium in younger adults, whereas this effect is significantly reduced with ageing. Glucagon-like peptide-1 (GLP-1) also stimulates vasodilation through NO-dependent and NO-independent mechanisms in a postprandial state. Finally, the NO that is produced in older adults is more likely to be scavenged by overproduced and/or unregulated reactive oxygen species (ROS) (i.e. oxidative stress). Consequently, meal consumption in younger adults is ultimately more likely to cause a robust vasodilatory response, thus enhancing the anabolic potential of meal consumption via greater nutrient delivery to skeletal muscle, when compared with older adults.

Journal of Cachexia, Sarcopenia and Muscle 2022; **13**: 114–127 DOI: 10.1002/jcsm.12898



The postprandial skeletal muscle protein synthetic (MPS) response is dependent on amino acid delivery, which is the product of amino acid availability (e.g. concentrations) and blood flow (e.g. perfusion). In healthy younger adults, submaximal doses of essential amino acids (EAAs) are able to optimally stimulate MPS, whereby increasing to a maximal dose of EAAs does not result in further increases in MPS. In older healthy adults, submaximal doses of EAAs are often not able to optimally stimulate MPS, but when maximal doses are given, these individuals are often able to saturate the MPS response. In older sarcopenic adults, neither submaximal nor maximal doses of EAAs are able to optimally stimulate MPS. We propose that a rate-limiting factor for older adults consuming submaximal and older sarcopenic adults consuming maximal EAA doses to be an inability of the meal consumption to promote adequate skeletal muscle perfusion, resulting in high circulating amino acid concentrations in these populations, but poor delivery and consequently impaired increases in MPS.

Journal of Cachexia, Sarcopenia and Muscle 2022; **13**: 114–127 DOI: 10.1002/jcsm.12898



Proposed model of mTORC1-mediated muscle atrophy in sarcopenia. Aged skeletal muscle exhibits anabolic resistance to hyper-aminoacidemia characterized by an impaired protein synthetic response. Basal mTORC1 exhibits sustained activation in aged skeletal muscle, that can contribute to neuromuscular junction instability, endoplasmic reticulum (ER) stress, and impaired mitophagy. Poor cellular quality control mechanisms result in a compromised mitochondrial pool, leading to impaired bioenergetics and enhanced reactive oxygen species production.

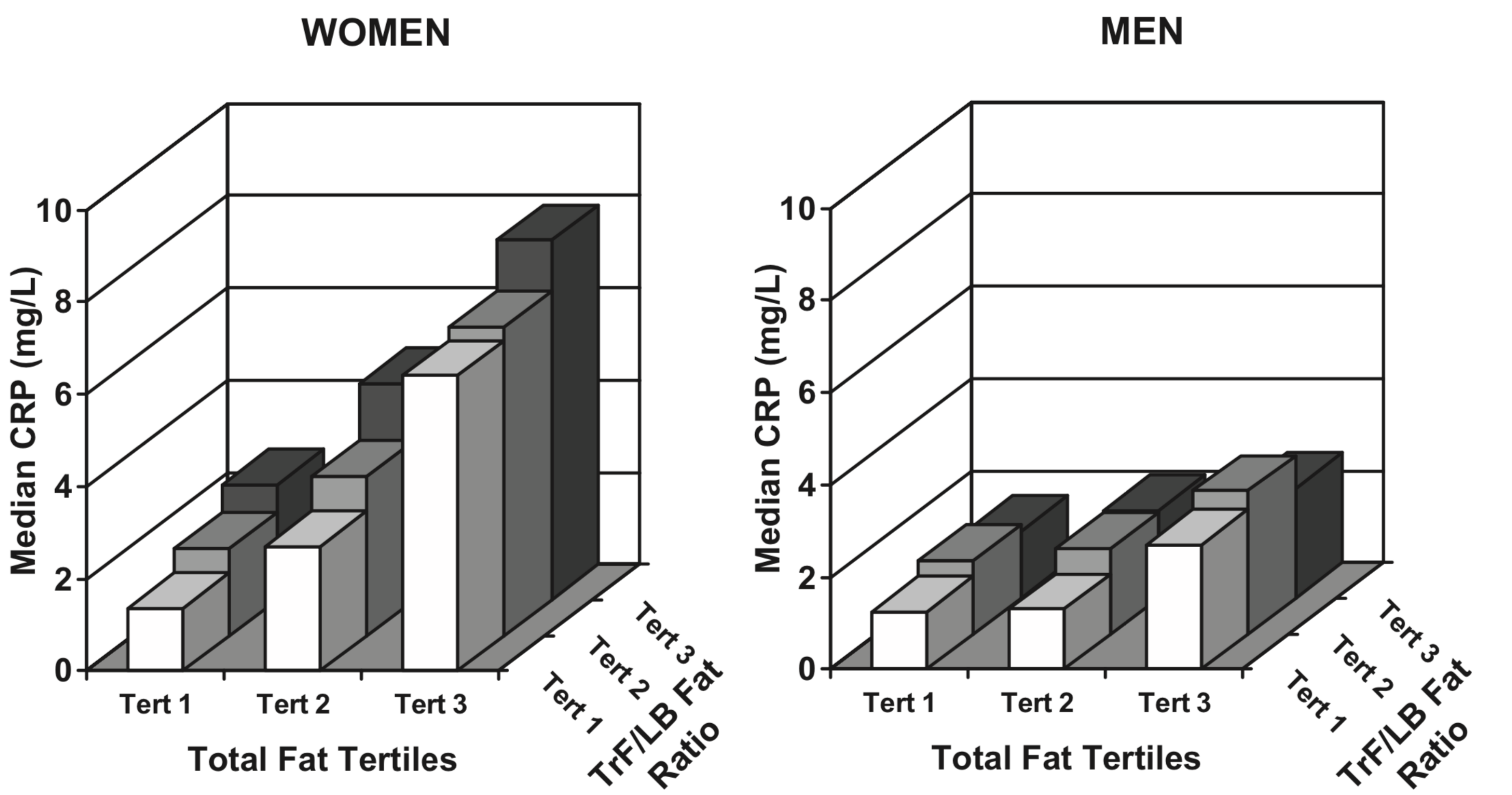
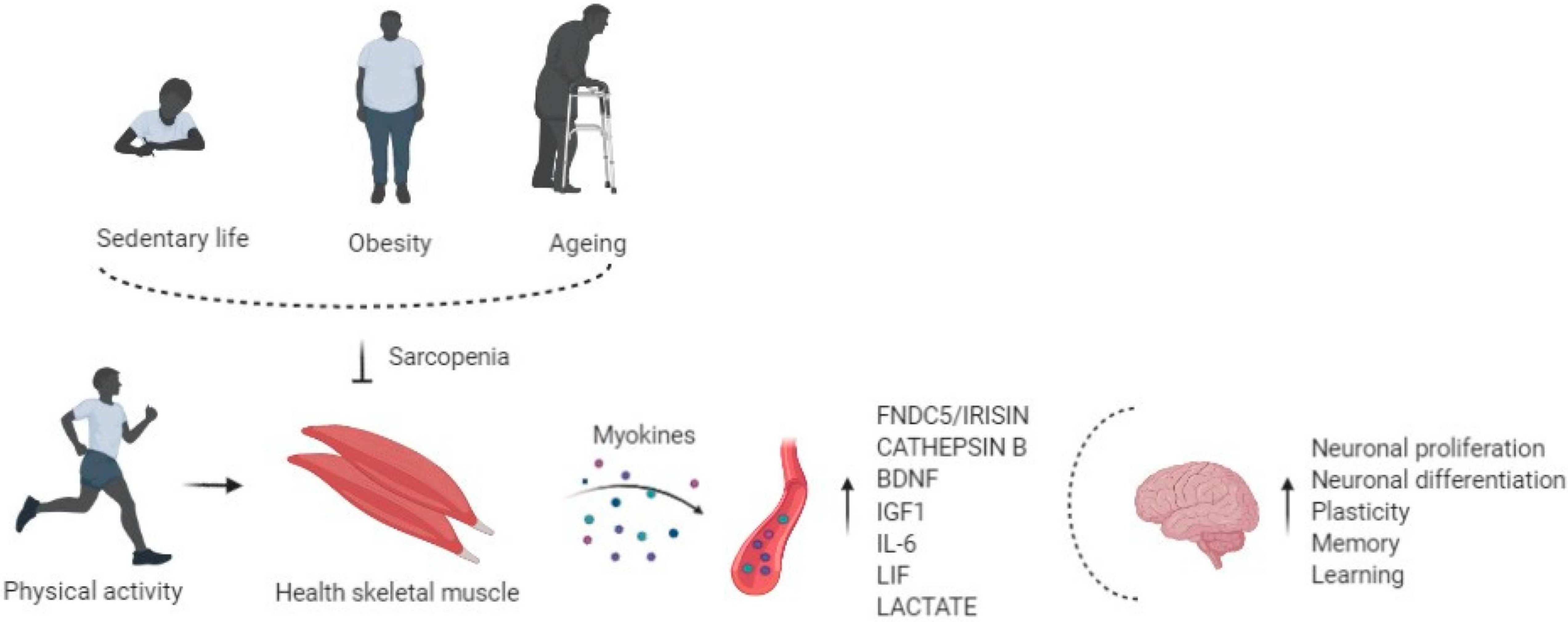


FIG. 2. Impact of TrF/LBF ratio on CRP levels. Median CRP levels by truncal to lower body fat ratio, stratified by gender-specific total fat values. Tert, Tertile.

https://doi.org/10.1210/jc.2008-2406



https://doi.org/10.3390/life11020173

IL MUSCOLO NON È SOLO FORZA/MOVIMENTO

MANTENERE L’EQUILIBRIO

RESPIRARE

ANDARE DI CORPO

PARLARE

MASTICARE

DEGLUTIRE

IL MUSCOLO NON È SOLO FORZA/MOVIMENTO

MANTENERE L'EQUILIBRIO

ANDARE DI CORPO

MASTICARE

RESPIRARE

PARLARE

DEGLUTIRE

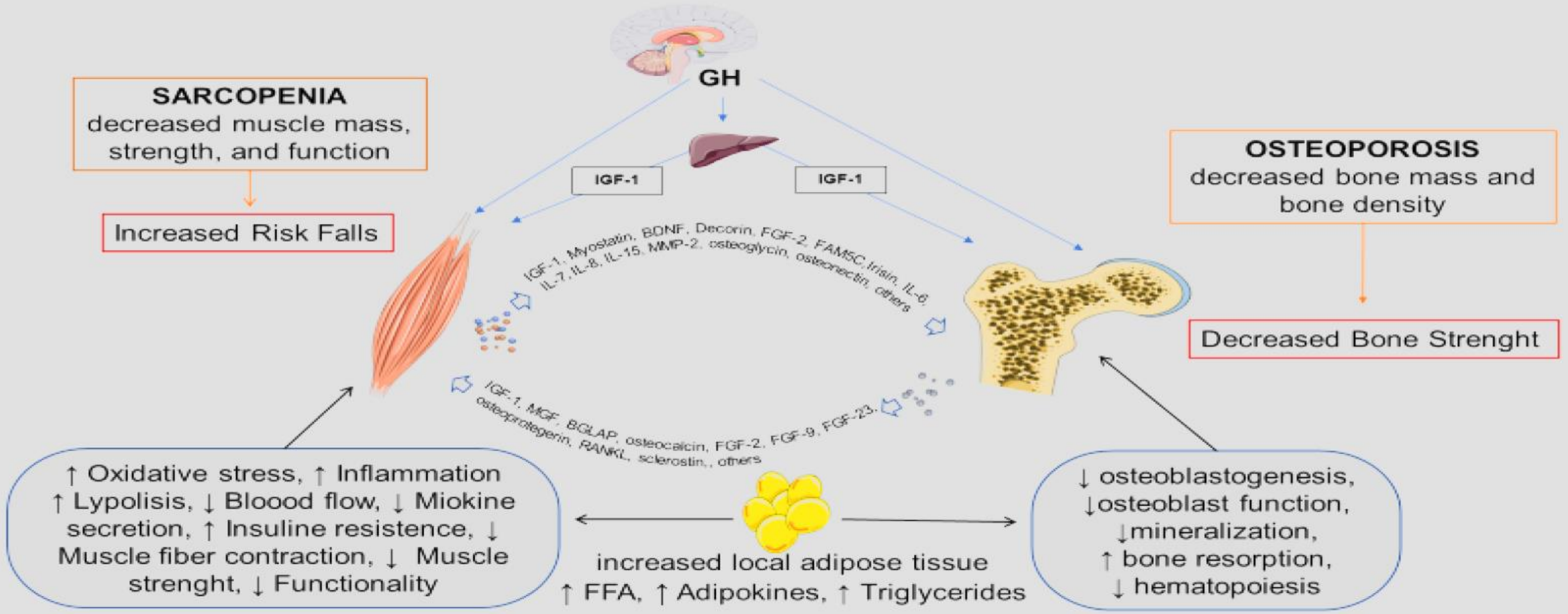
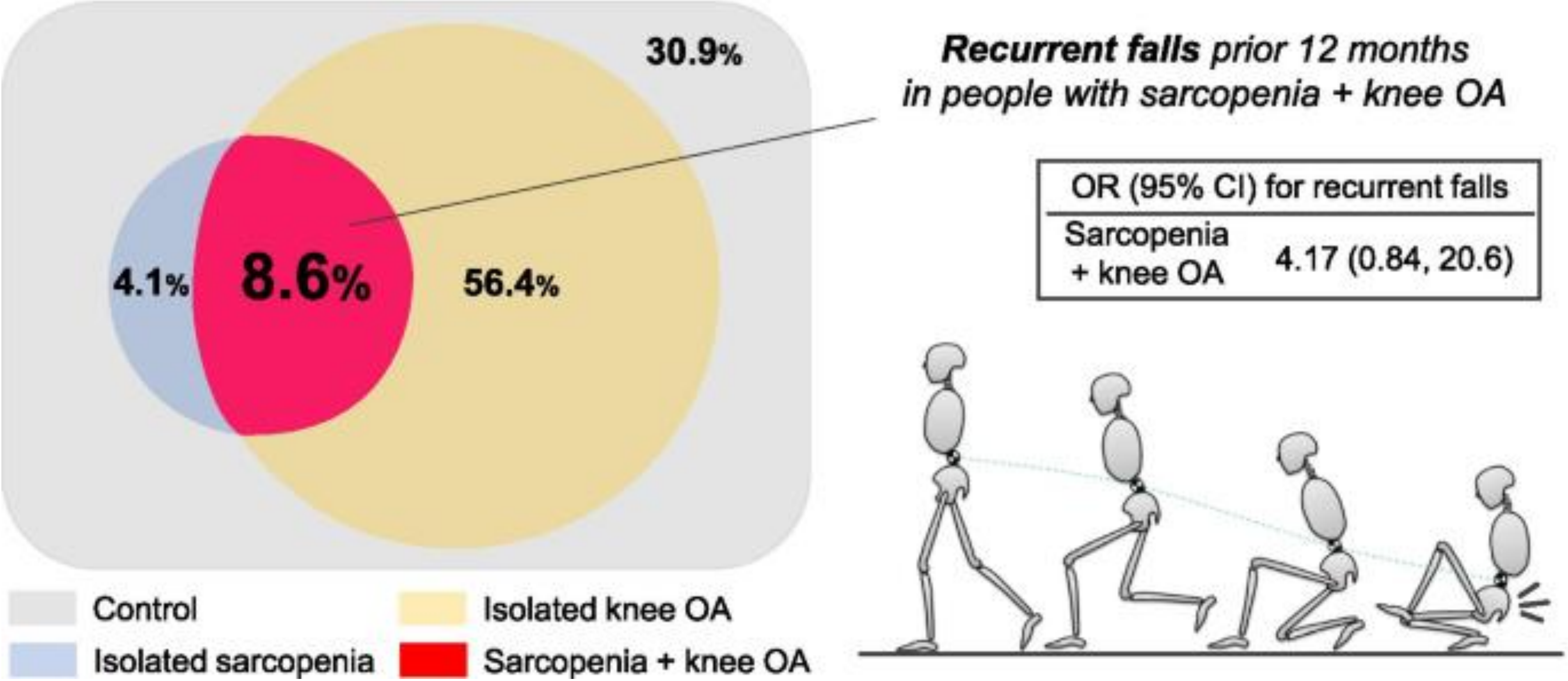


Figure 1. Pathogenesis and etiology of osteosarcopenia.

https://doi.org/10.3390/ijms23105591



https://doi.org/10.1186/s12877-021-02654-4

IL MUSCOLO NON È SOLO FORZA/MOVIMENTO

MANTENERE L'EQUILIBRIO

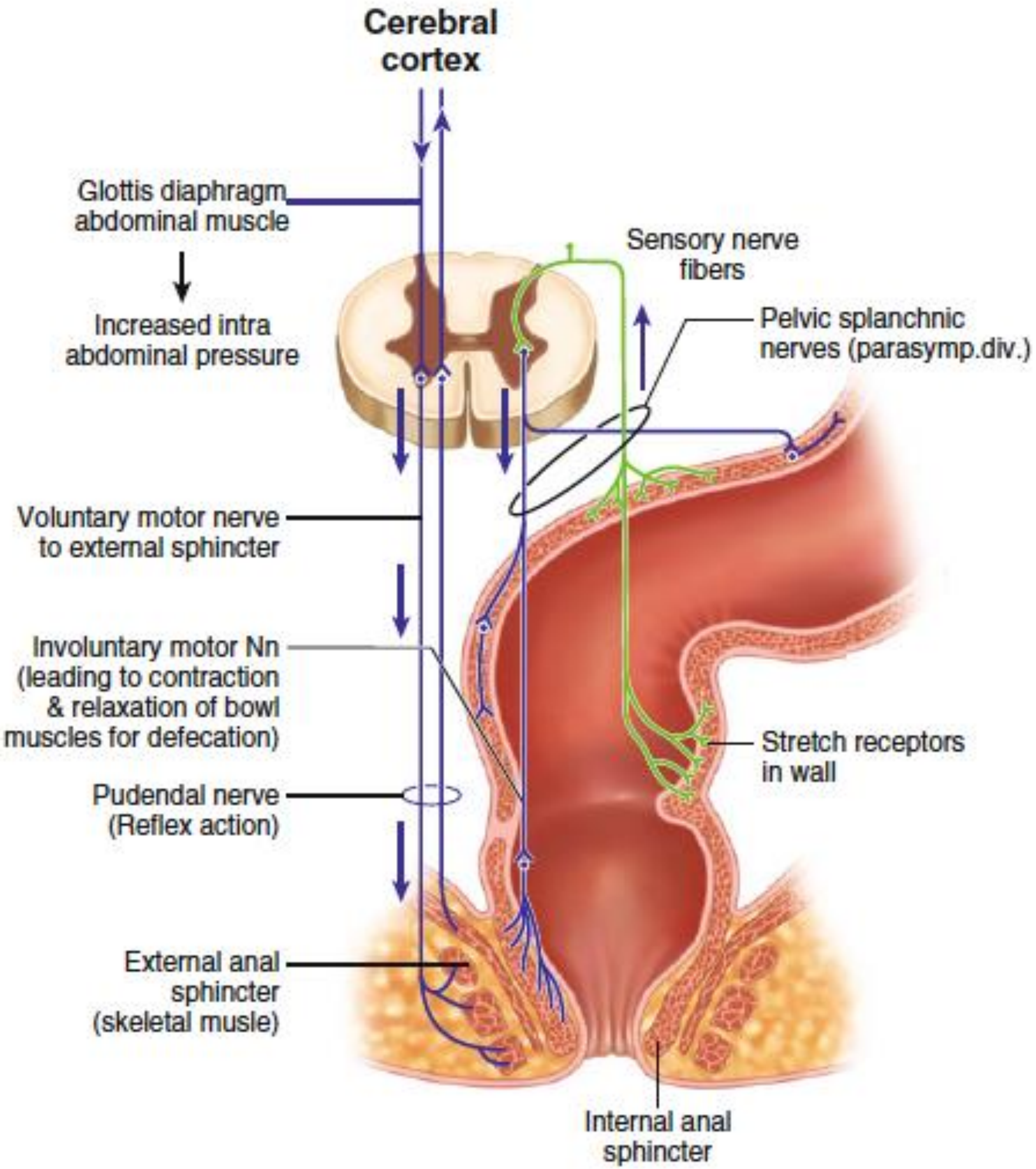
ANDARE DI CORPO

MASTICARE

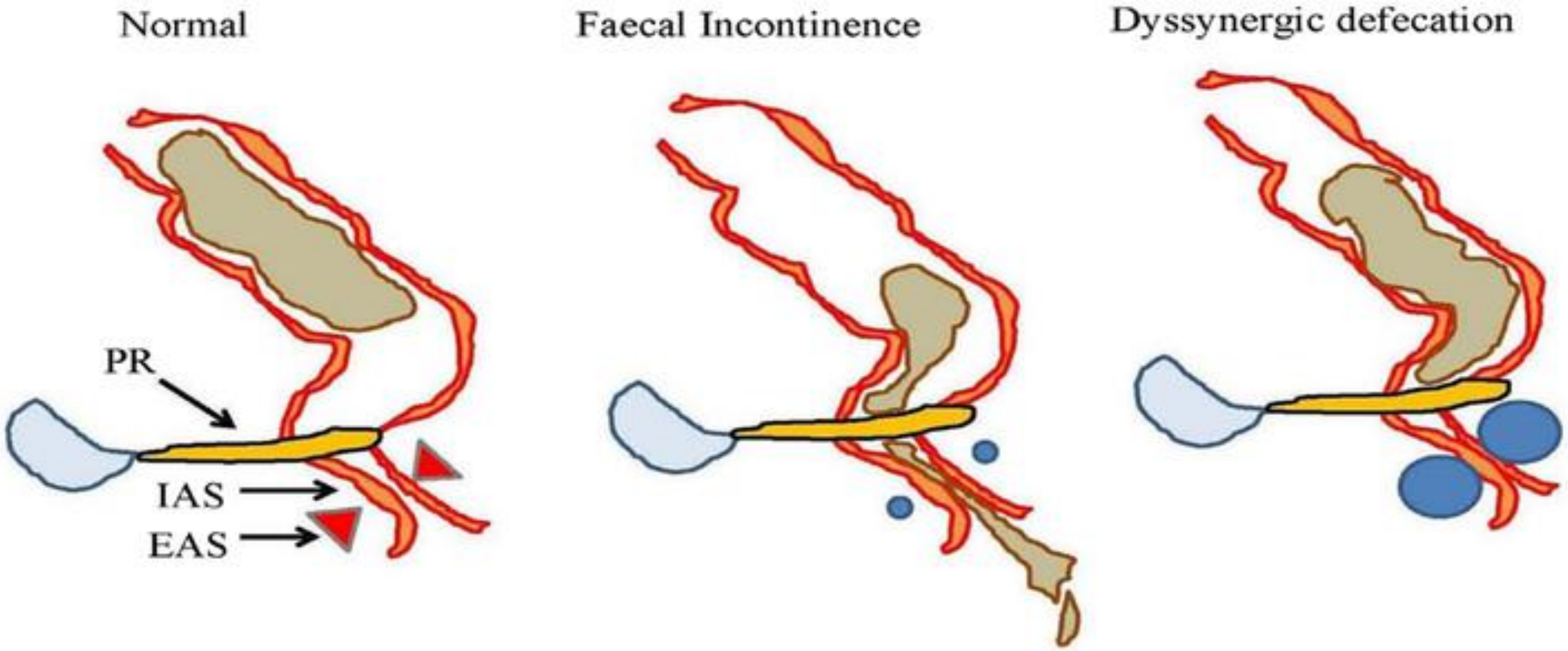
RESPIRARE

PARLARE

DEGLUTIRE



Cerebral cortex
Glottis diaphragm abdominal muscle
Increased intra abdominal pressure
Sensory nerve fibers
Pelvic splanchnic nerves (parasymp.div.)
Voluntary motor nerve to external sphincter
Involuntary motor Nn (leading to contraction & relaxation of bowl muscles for defecation)
Stretch receptors in wall
Pudendal nerve (Reflex action)
External anal sphincter (skeletal musle)
Internal anal sphincter



Normal defecation	Faecal incontinence	Dyssynergic defecation
Normal stool perception Normal rectal compliance Relaxation of EAS and PR	Altered stool perception Reduced rectal compliance Low EAS and IAS pressure Weak PR Neuropathy	Rectal hyposensitivity Abnormal rectal compliance Paradoxical anal sphincter contraction Poor abdominal-rectal propulsive force

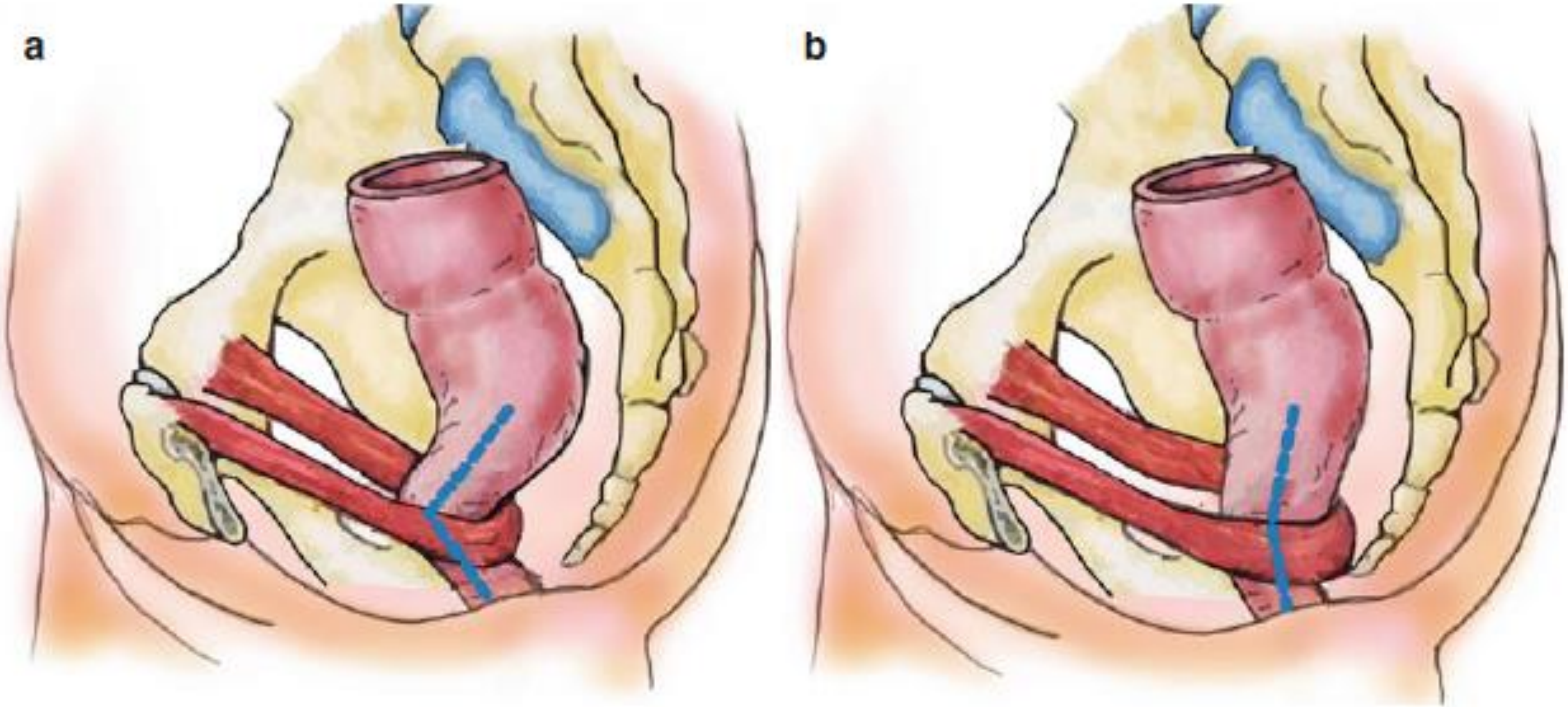


Fig. 2.2 Puborectalis action and anorectal angle. (**a**) Puborectalis sling forming an angle. (**b**) Puborectalis relaxed for defecation

IL MUSCOLO NON È SOLO FORZA/MOVIMENTO

MANTENERE L’EQUILIBRIO

RESPIRARE

ANDARE DI CORPO

PARLARE

MASTICARE

DEGLUTIRE

SPECIAL ARTICLE

Sarcopenia and dysphagia: Position paper by four professional organizations

Ichiro Fujishima,[1] Masako Fujiu-Kurachi,[2] Hidenori Arai,[3] Masamitsu Hyodo,[4] Hitoshi Kagaya,[5] Keisuke Maeda,[6] Takashi Mori,[7] Shinta Nishioka,[8] Fumiko Oshima,[9] Sumito Ogawa,[10] Koichiro Ueda,[11] Toshiro Umezaki,[12] Hidetaka Wakabayashi,[13] Masanaga Yamawaki[14] and Yoshihiro Yoshimura[15]

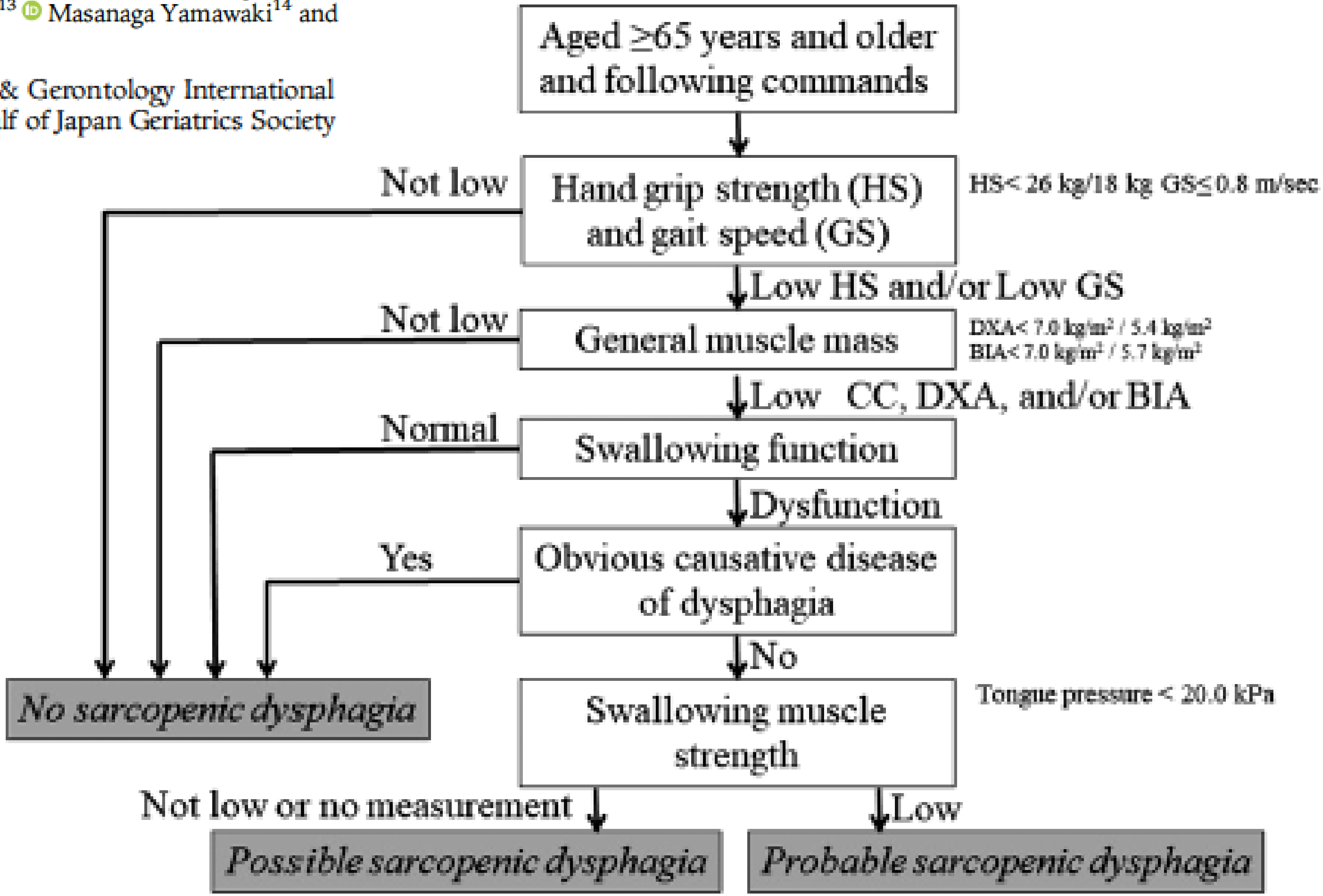
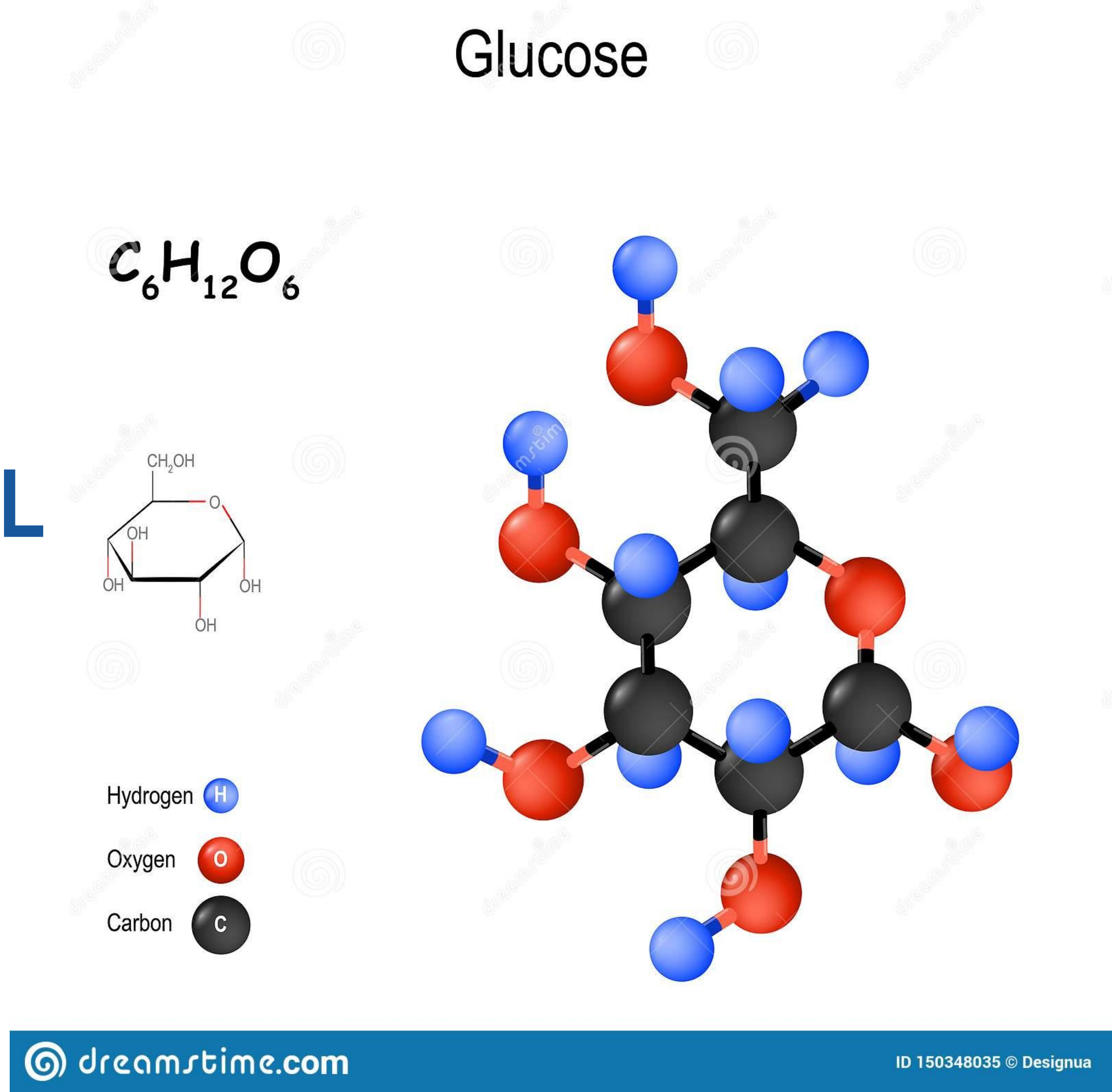


Figure 1 Diagnostic algorithm for sarcopenic dysphagia. CC, calf circumference; DXA, dual-energy X-ray absorptiometry; BIA, bioimpedance analysis.

INOLTRE.....

IL MUSCOLO E' FONDAMENTALE PER IL METABOLISMO DEL GLUCOSIO



frontiers in Endocrinology
REVIEW
published: 25 August 2020
doi: 10.3389/fendo.2020.00568

Diabetes and Sarcopenic Obesity: Pathogenesis, Diagnosis, and Treatments

Mina Wang[1,2†], Yan Tan[1†], Yifan Shi[1], Xu Wang[1], Zehuan Liao[3,4*] and Peng Wei[1*]

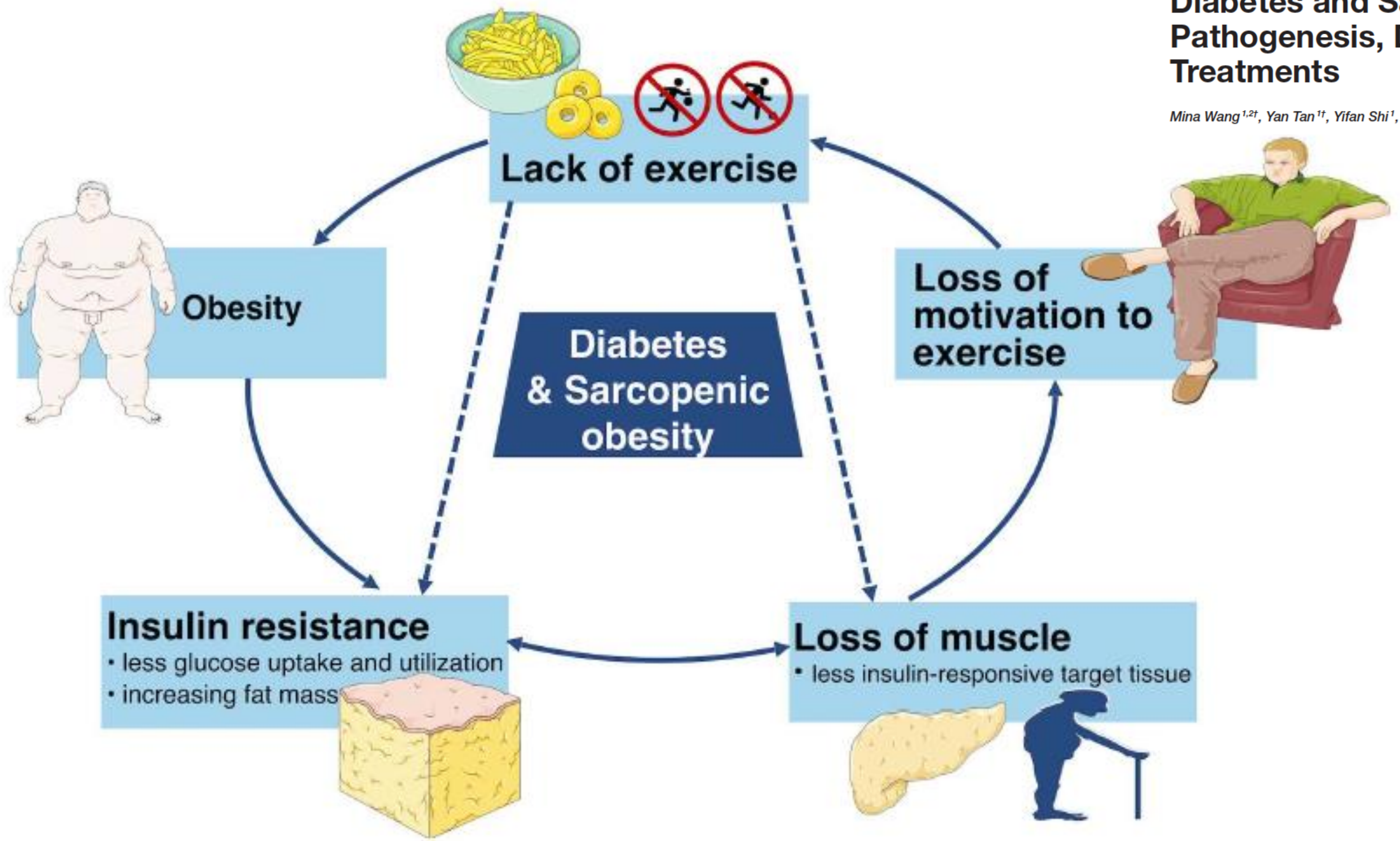


FIGURE 1 | This simplified schematic diagram depicts the vicious cycle of unhealthy lifestyle which can eventually lead to diabetes and sarcopenic obesity as well as other adverse metabolic conditions.

frontiers in Endocrinology
REVIEW
published: 25 August 2020
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Diabetes and Sarcopenic Obesity: Pathogenesis, Diagnosis, and Treatments

Mina Wang[1,2†], Yan Tan[1†], Yifan Shi[1], Xu Wang[1], Zehuan Liao[3,4*] and Peng Wei[1*]

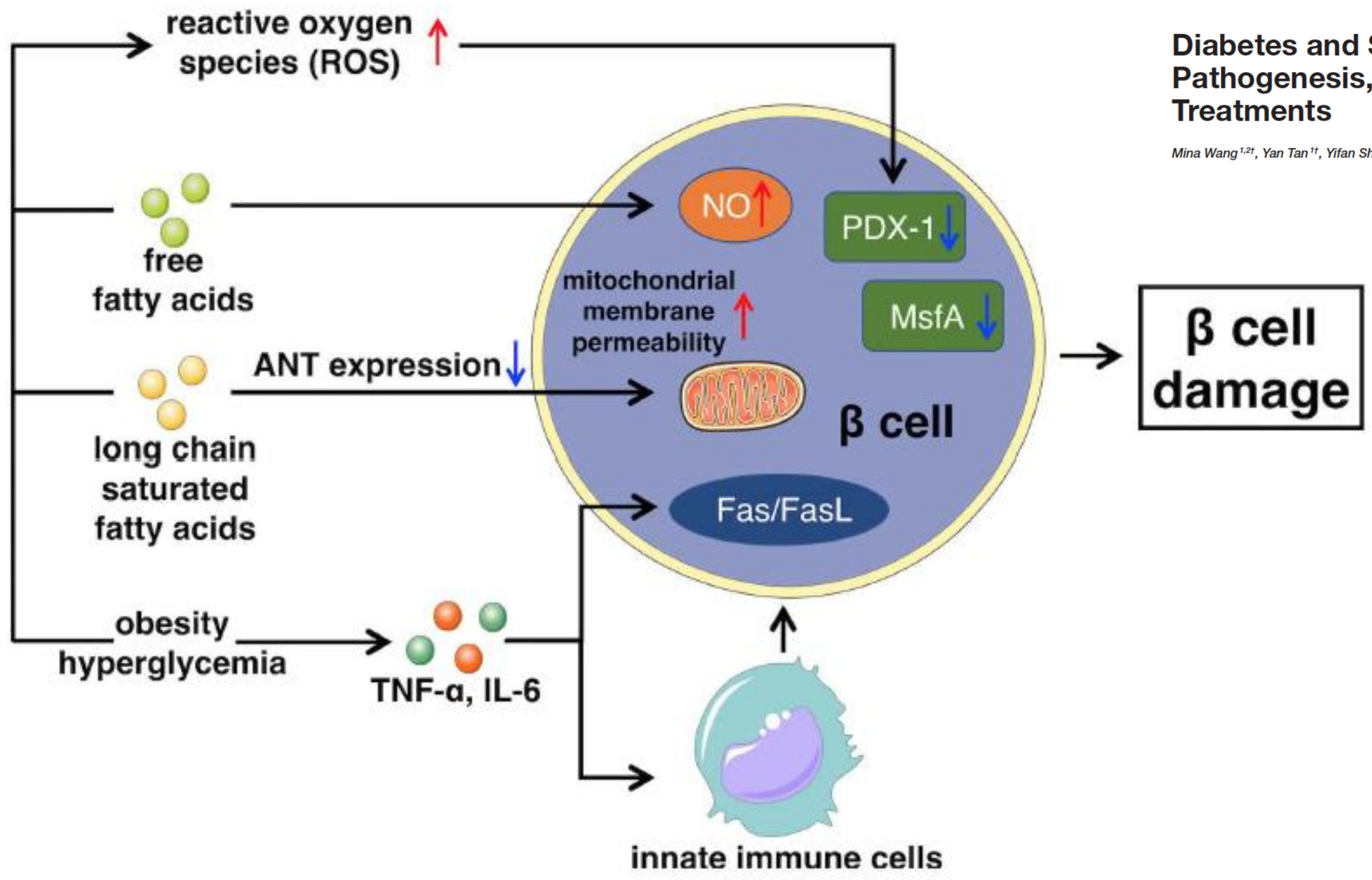
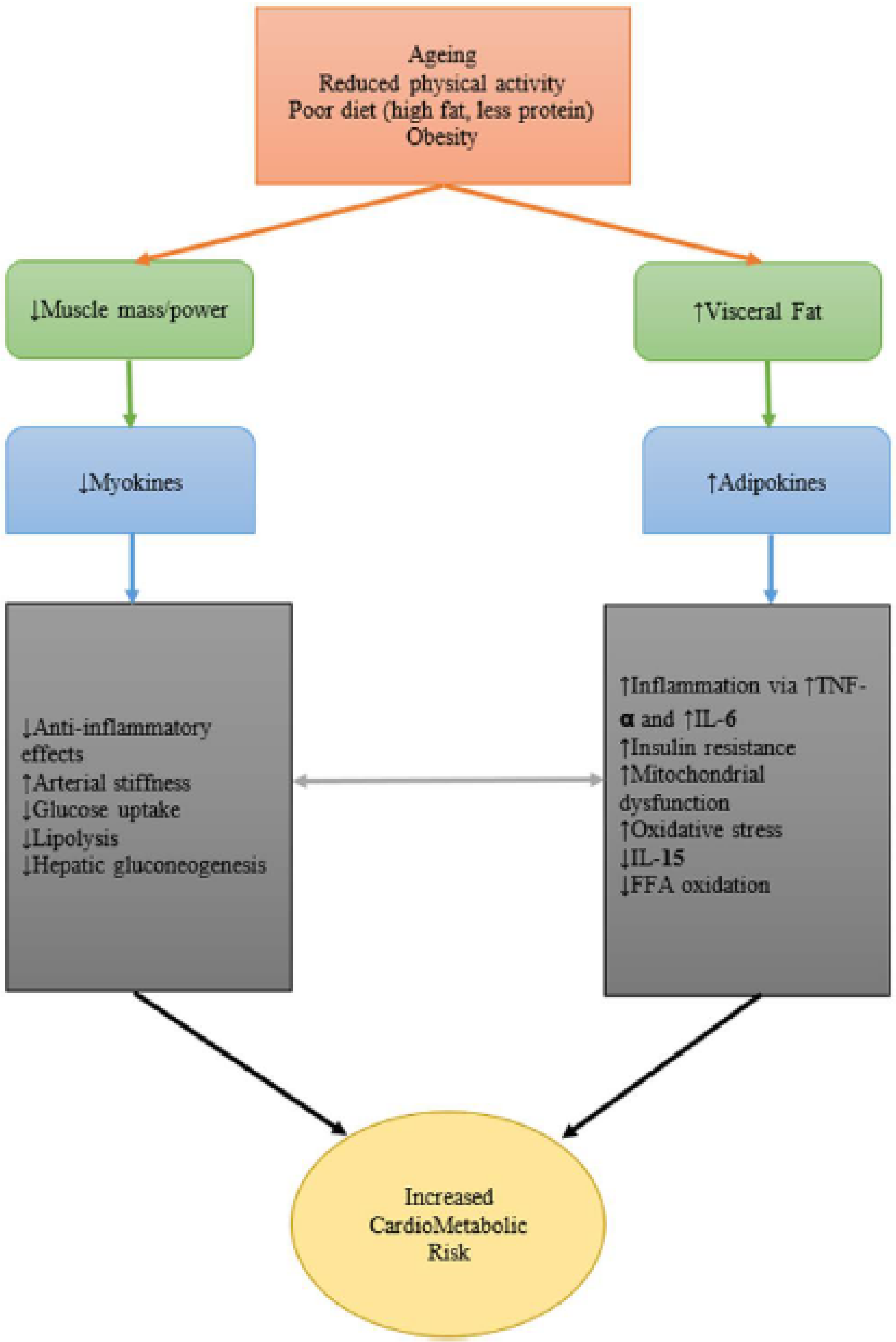
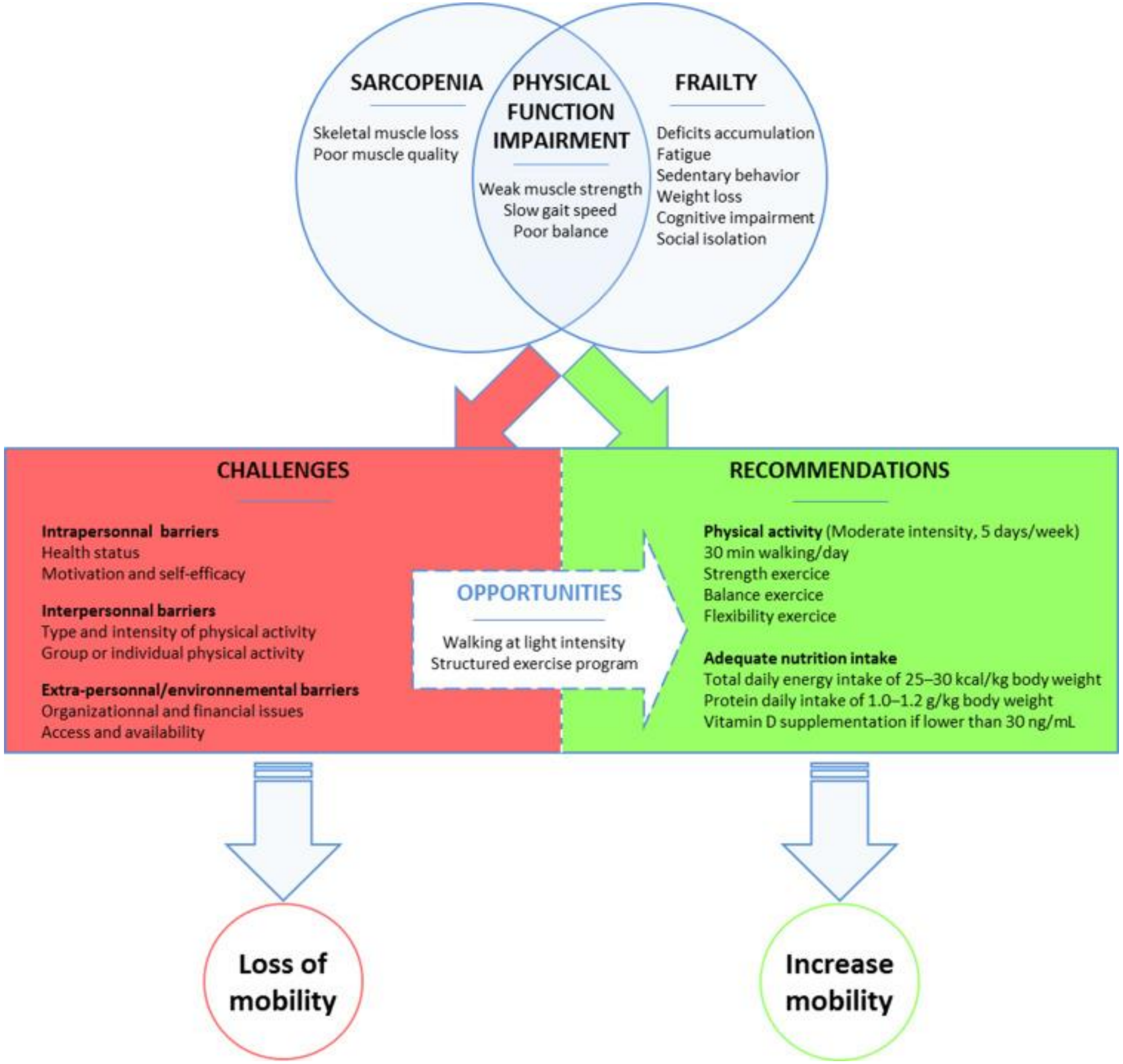


FIGURE 2 | This figure illustrates the main mechanism of impaired insulin secretion that glucose toxicity, lipid toxicity, immunoinflammatory response, and oxidative stress lead to *β* cell damage. ANT, Adenine nucleotide translocator.

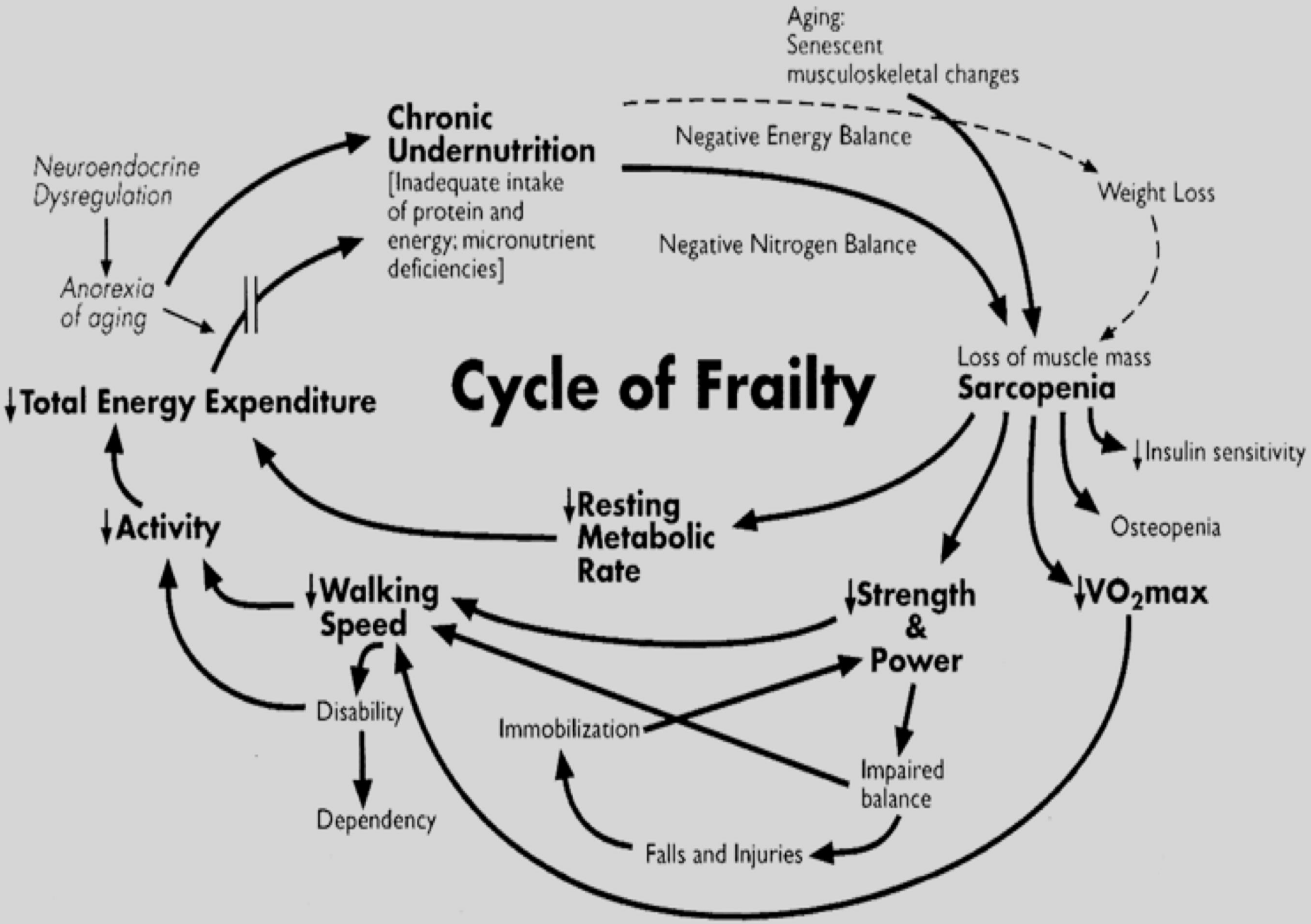


AJ Sinclair et al. Cardiovasc Endocrinol Metab. 2020;22;9:90-95

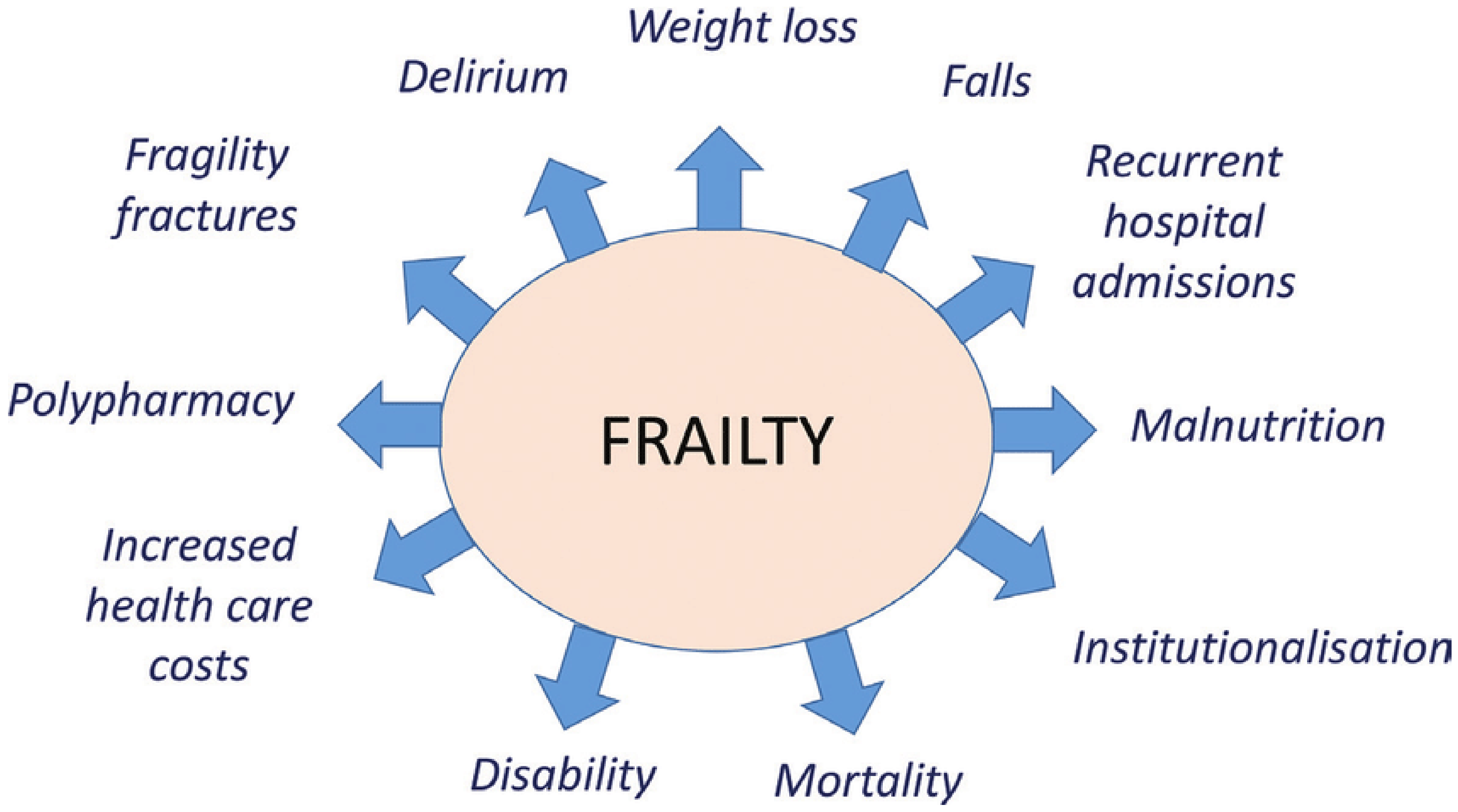


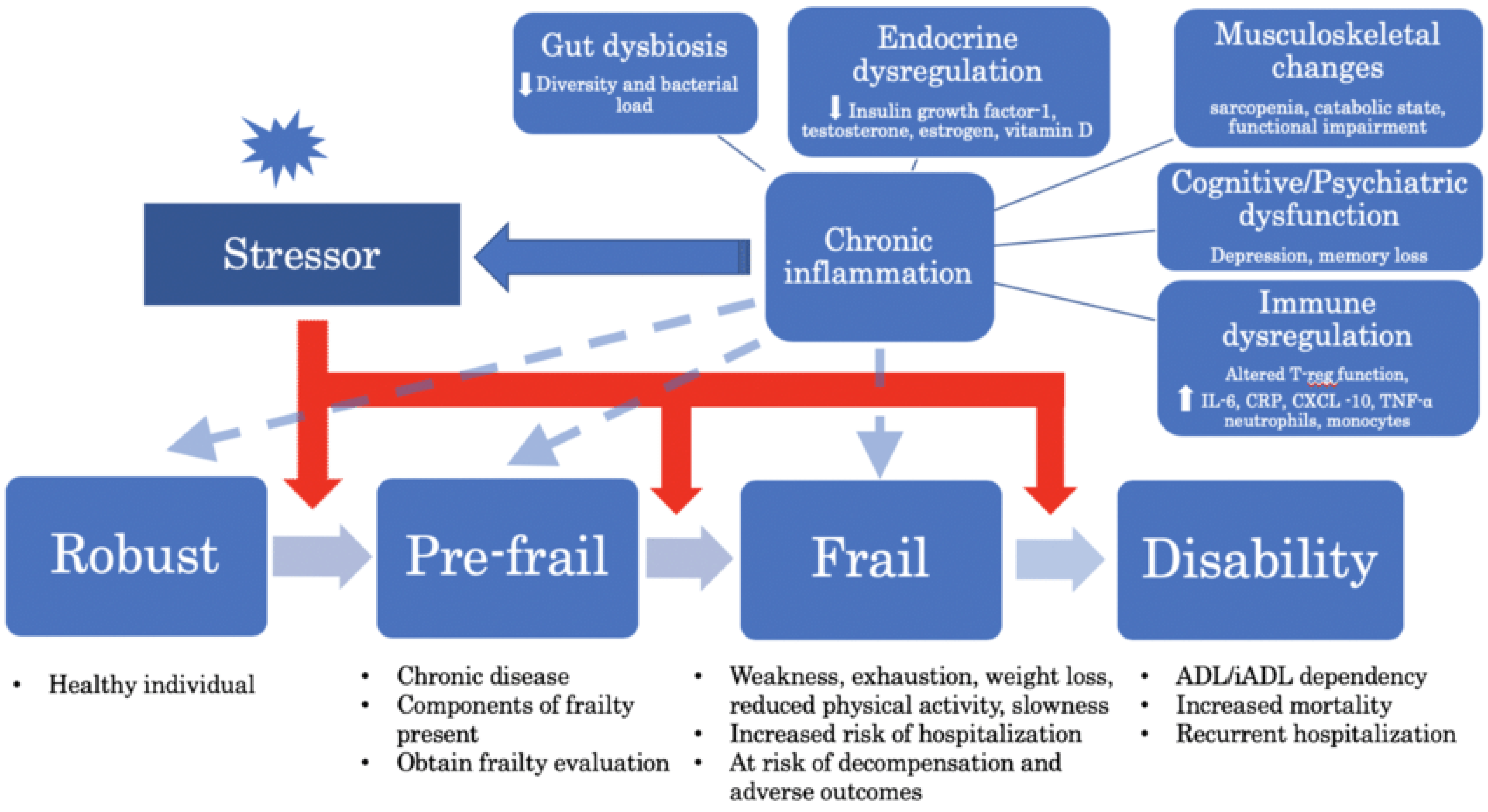
Billot M et al Clin Interv Aging. 2020 Sep 16;15:1675-1690

frailty is not a synonym of sarcopenia

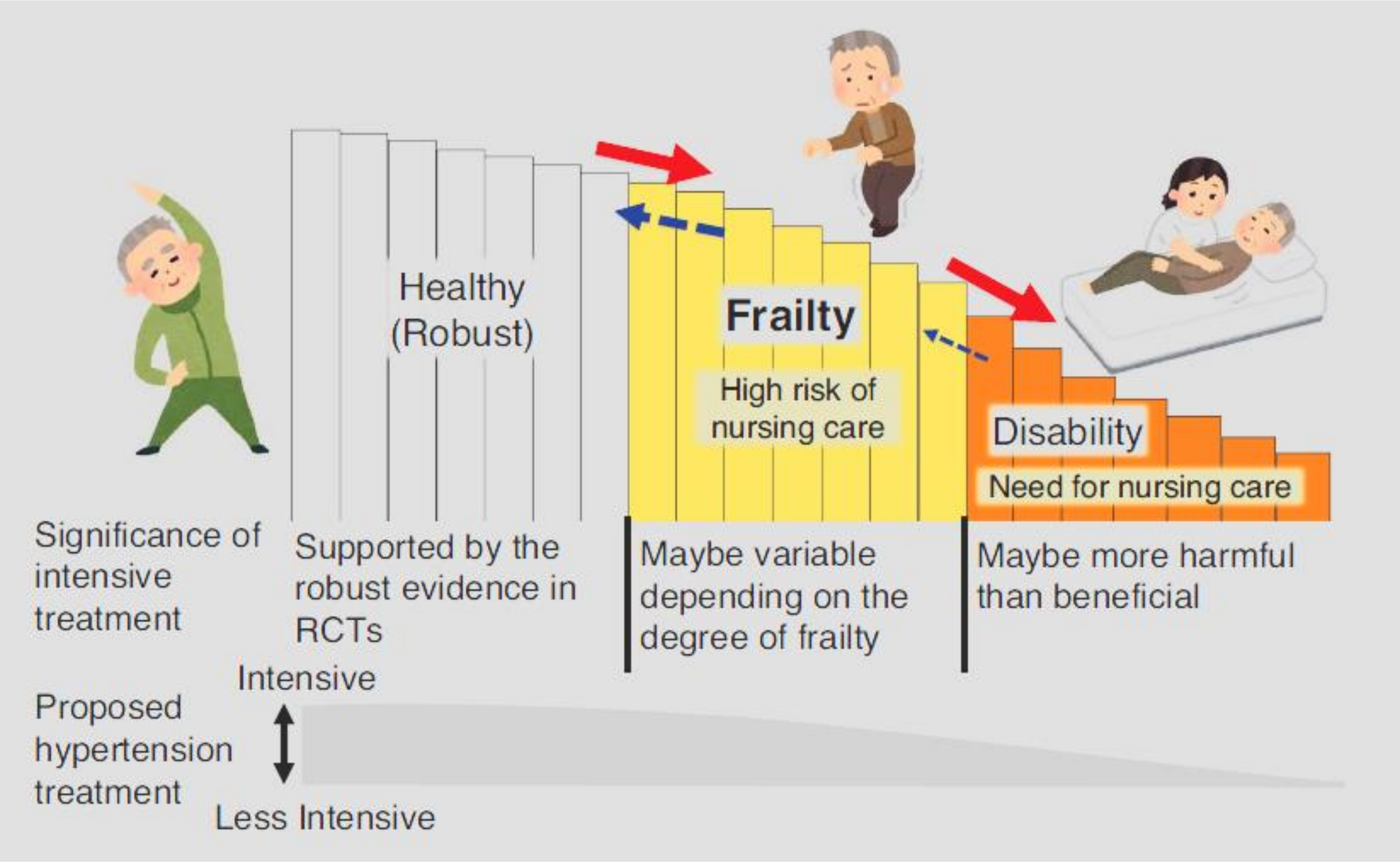


Aging:
Senescent
musculoskeletal changes
Chronic
Undernutrition
[Inadequate intake
of protein and
energy; micronutrient
deficiencies]
Negative Energy Balance
Weight Loss
Neuroendocrine
Dysregulation
Negative Nitrogen Balance
Anorexia
of aging
Loss of muscle mass
Sarcopenia
Cycle of Frailty
↓Total Energy Expenditure
↓Insulin sensitivity
↓Resting
Metabolic
Rate
↓Activity
Osteopenia
↓Walking
Speed
↓Strength
&
Power
$\downarrow VO_2max$
Disability
Immobilization
Impaired
balance
Dependency
Falls and Injuries


Weight loss
Delirium
Falls
Fragility fractures
Recurrent hospital admissions
Polypharmacy
FRAILTY
Malnutrition
Increased health care costs
Institutionalisation
Disability
Mortality


Gut dysbiosis
Diversity and bacterial load
Endocrine dysregulation
Insulin growth factor-1, testosterone, estrogen, vitamin D
Musculoskeletal changes
sarcopenia, catabolic state, functional impairment
Cognitive/Psychiatric dysfunction
Depression, memory loss
Immune dysregulation
Altered T-reg function,
IL-6, CRP, CXCL -10, TNF-α neutrophils, monocytes
Stressor
Chronic inflammation
Robust
Pre-frail
Frail
Disability
• Healthy individual
• Chronic disease
• Components of frailty present
• Obtain frailty evaluation
• Weakness, exhaustion, weight loss, reduced physical activity, slowness
• Increased risk of hospitalization
• At risk of decompensation and adverse outcomes
• ADL/iADL dependency
• Increased mortality
• Recurrent hospitalization

Frailty and Hypertension treatment



https://doi.org/10.1038/s41440-023-01310-1

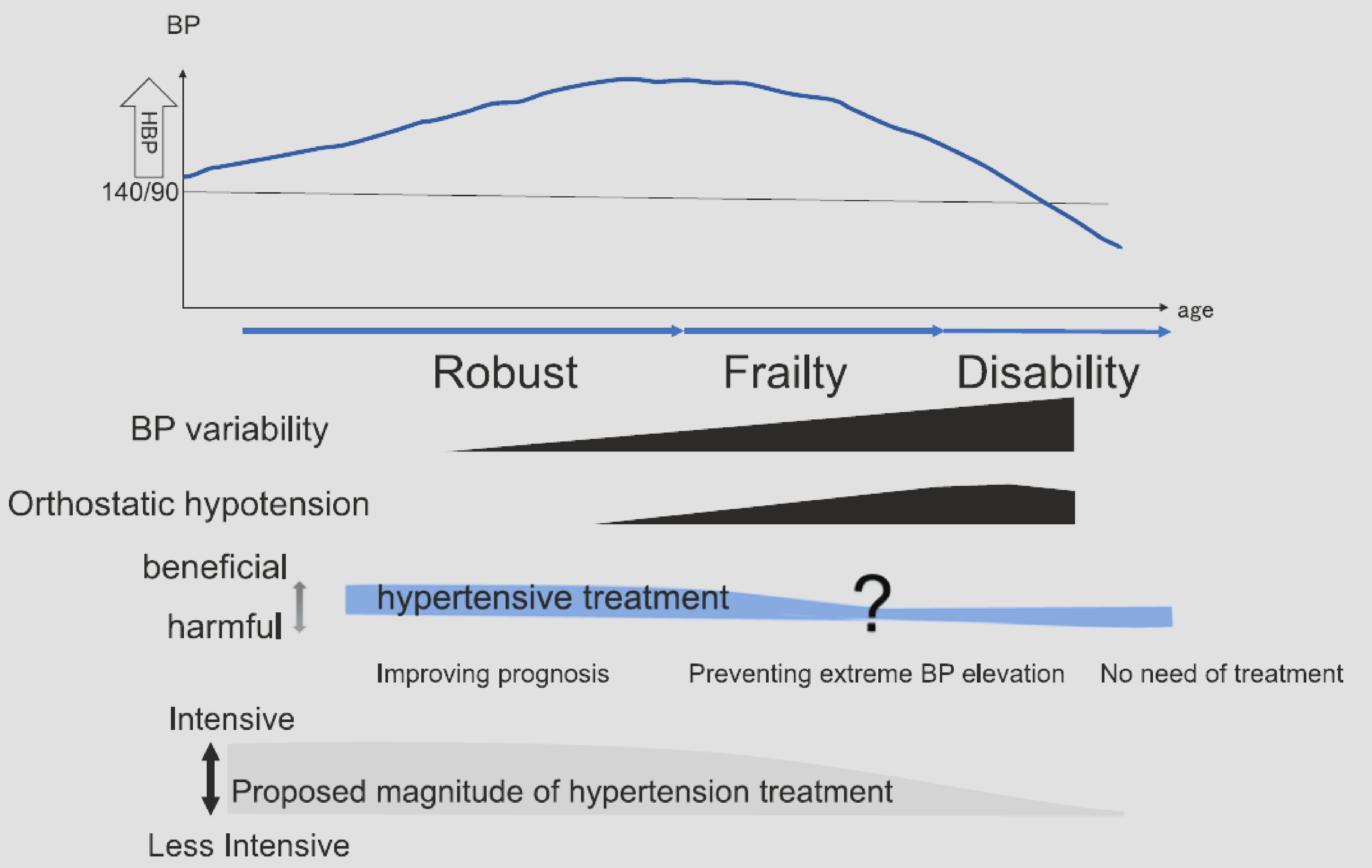


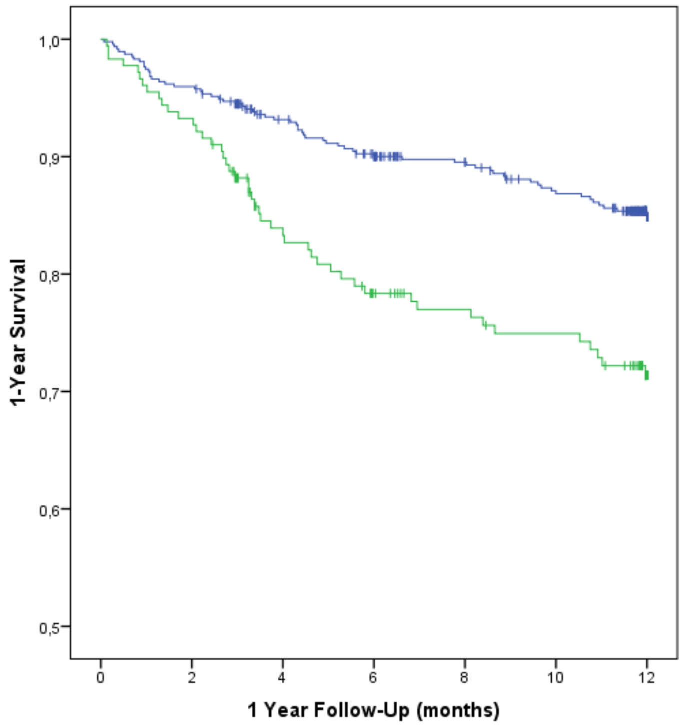
Fig. 2 BP trajectory, functional status, and temporal changes in the significance of hypertension treatment in old age

https://doi.org/10.1038/s41440-023-01310-1

IL MUSCOLO HA UN RUOLO FONDAMENTALE NELL'INVECCHIAMENTO

SOGGETTI SARCOPENICI PRESENTANO UNA RIDOTTA SOPRAVVIVENZA DOPO EVENTO ACUTO

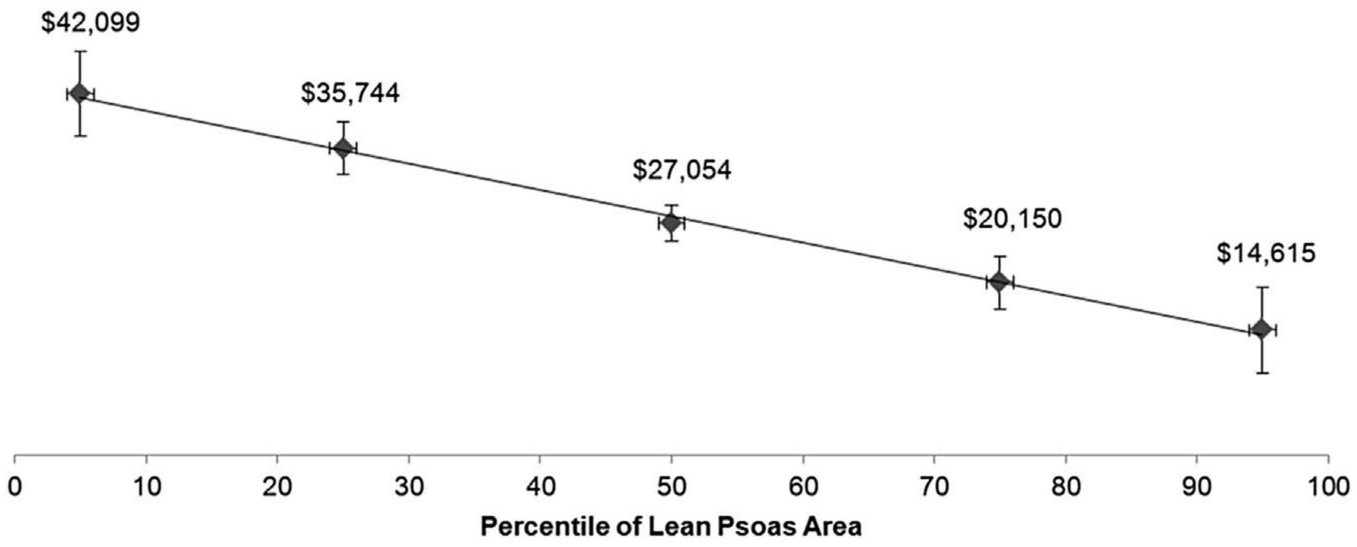
Curve di sopravvivenza a un anno dopo ricovero ospedaliero in base alla presenza di sarcopenia (Studio CRIME)



Vetrano D et al JGMS,2014;69:1154-61

LA SARCOPENIA AUMENTA I COSTI DELL'ASSISTENZA SANITARIA

I pazienti con sarcopenia severa quando operati generano costi che sono 3 volte superiori ai coetanei con massa muscolare superiore all'età



Sheetz KH et al. J Am CollSurg2013;217:813-818

Acute Sarcopenia Secondary to Hospitalisation - An Emerging Condition Affecting Older Adults

Welch Carly 1, 2 ;K Hassan-Smith Zaki 2, 3, 4 ;A Greig Carolyn 5, 6 ;M Lord Janet 1, 6 ;A Jackson Thomas 1, 2 ;

1 Institute of Inflammation and Ageing, College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK ; 2 Queen Elizabeth Hospital Birmingham, Edgbaston, Birmingham: B15 2WB, UK ; 3 Institute of Metabolism and Systems Research, College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK ; 4 Centre for Endocrinology, Diabetes and Metabolism, Birmingham Health Partners, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK ; 5 School of Sport, Exercise #cod#x00026; Rehabilitation Sciences, College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK ; 6 MRC Arthritis Research UK Centre for Musculoskeletal Ageing Research, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK ;

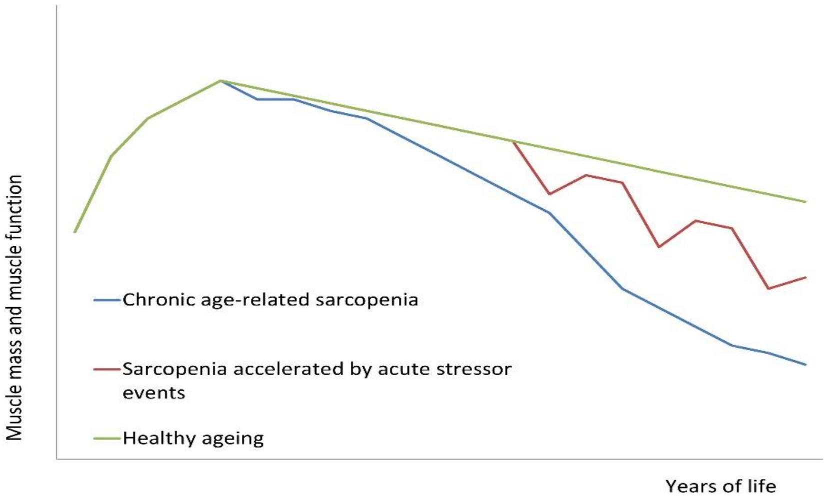
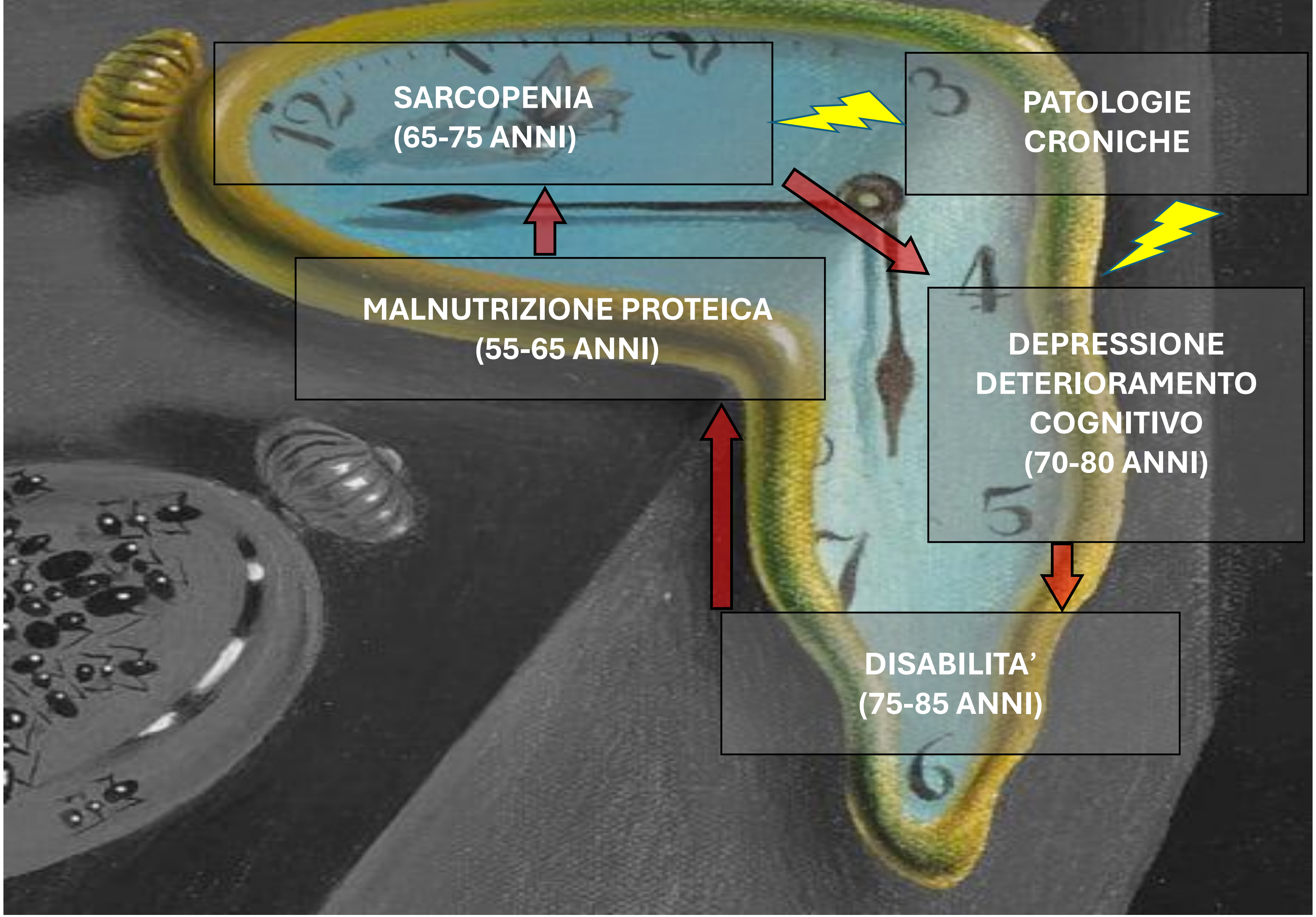


Figure 2. Proposed disease trajectories associated with sarcopenia. This diagram demonstrates proposed trajectories associated with the development of sarcopenia over time. The green line demonstrates expected changes of muscle mass and function associated with healthy ageing; there may be some inevitable loss of muscle mass and function but not to such an extent as to cause detriment. The blue line demonstrates the development of chronic sarcopenia over time. The red line demonstrates our proposed model of how episodes of acute sarcopenia can potentially lead to the development of chronic sarcopenia over time.

null,null,0(0),null-null. Doi:10.14336/AD.2017.0315

CIRCOLO VIZIOSO DELLA DISABILITA' LEGATA ALL'INVECCHIAME



COME MISURIAMO LA SARCOPENIA

SISTEMI USATI PER MISURARE LA MASSA MUSCOLARE

TAC (Tomografia assiale computerizzata)

Radiazioni Ionizzanti
Costi
Impossibile rivalutazione periodica

RMN (Risonanza Magnetica Nucleare)

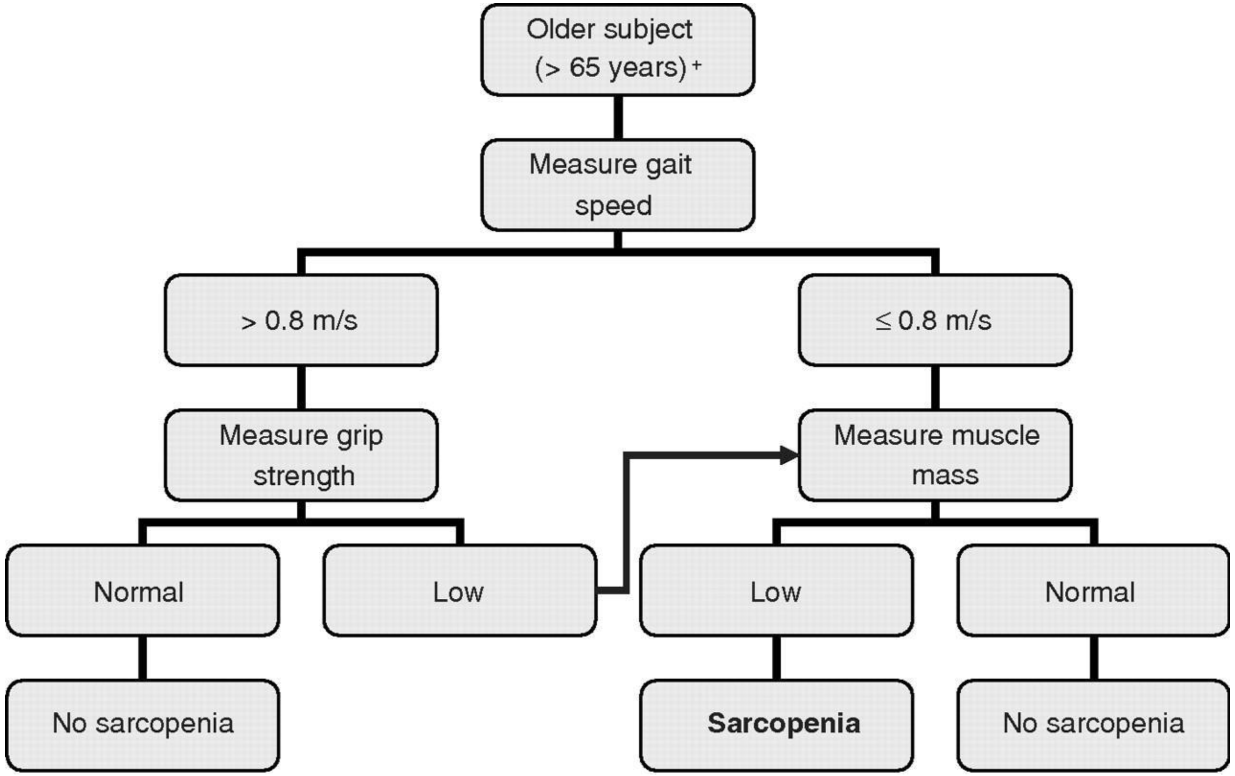
Tempi lunghi di esecuzione
Costi
Impossibile rivalutazione periodica

DEXA (Densitometria ossea NON a ULTRASUONI)

Attrezzatura pesante ed ingombrante
Algoritmi proprietari
Impossibile rivalutazione periodica

COME SEMPLIFICHIAMO LA SITUAZIONE?

COME MISURIAMO LA SARCOPENIA



Cruz-Jentoft *et al. Age Ageing 2010*

COME MISURIAMO LA SARCOPENIA

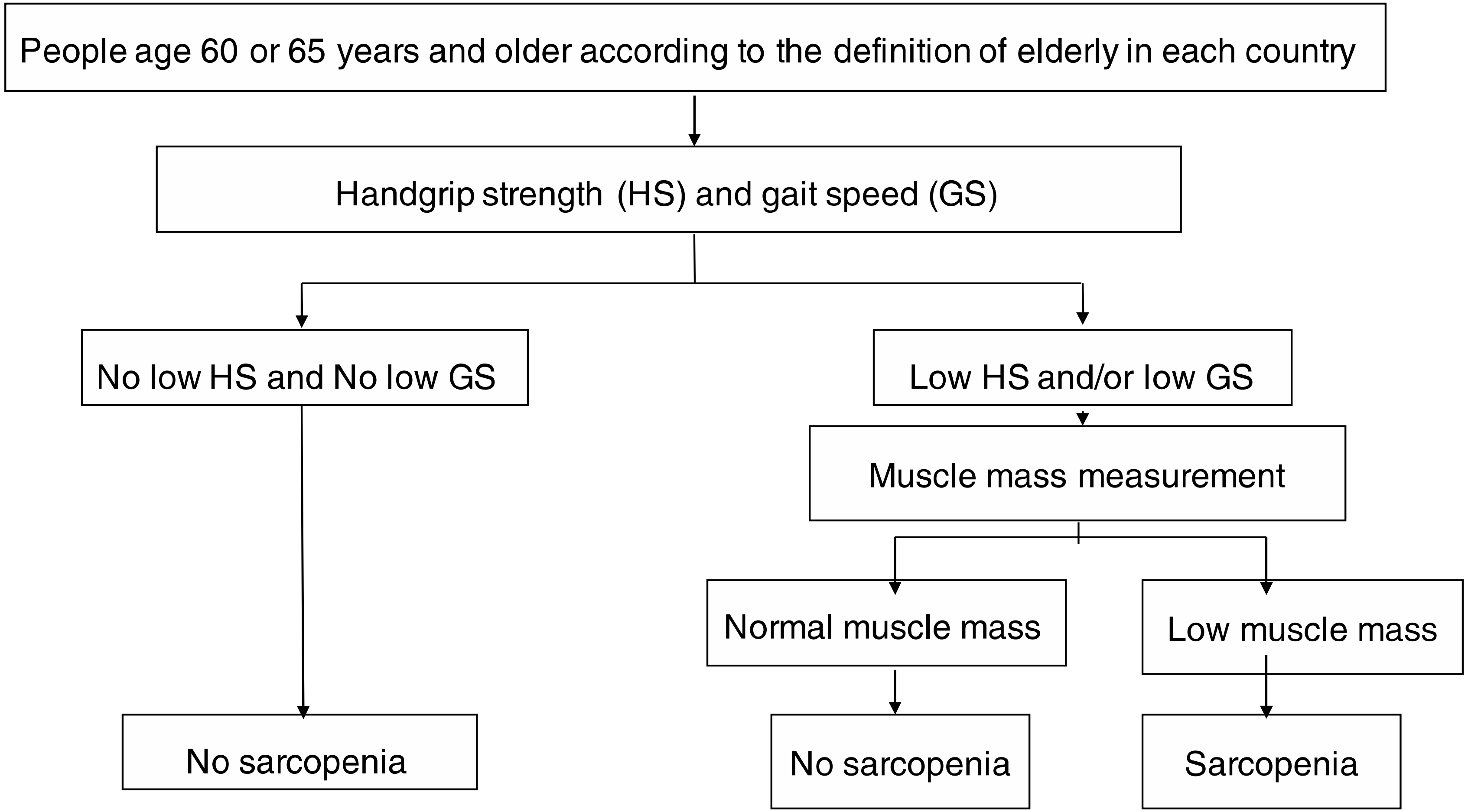


Figure 1 Diagnostic criteria by the Asian Working Group for Sarcopenia.[1]

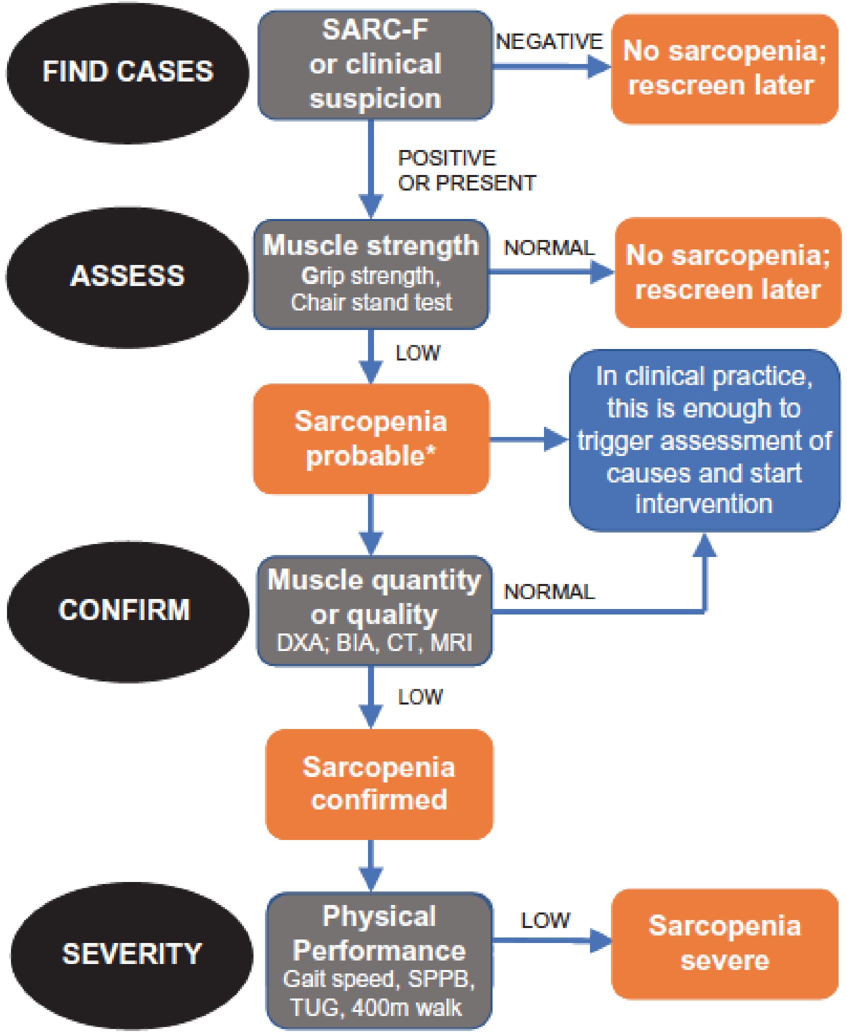


Figure 1. Sarcopenia: EWGSOP2 algorithm for case-finding, making a diagnosis and quantifying severity in practice. The steps of the pathway are represented as Find-Assess-Confirm-Severity or F-A-C-S. *Consider other reasons for low muscle strength (e.g. depression, sroke, balance disorders, peripheral vascular disorders).

Age and Ageing 2019; **48:** 16–31
doi: 10.1093/ageing/afy169
Published electronically 24 September 2018

GUIDELINES

Sarcopenia: revised European consensus on definition and diagnosis

Alfonso J. Cruz-Jentoft[1], Gülistan Bahat[2], Jürgen Bauer[3], Yves Boirie[4], Olivier Bruyère[5], Tommy Cederholm[6], Cyrus Cooper[7], Francesco Landi[8], Yves Rolland[9], Avan Aihie Sayer[10], Stéphane M. Schneider[11], Cornel C. Sieber[12], Eva Topinkova[13], Maurits Vandewoude[14], Marjolein Visser[15], Mauro Zamboni[16], Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2

GLI STRUMENTI

Callosità della pianta dei piedi e sudorazione alterano la misurazione

GLI STRUMENTI

Misura solo massa muscolare
degli arti superiori

MYSURABLE
HI-TECH SOLUTIONS FOR SUCCESSFUL AGING

GLI STRUMENTI

Spessore della cute sul dorso delle mani e dei piedi più costante nel tempo e zone a sudorazione ridotta

COME MISURIAMO LA SARCOPENIA

Table 2. Choosing tools for sarcopenia case finding and for measurement of muscle strength, muscle mass and physical performance in clinical practice and in research

Variable	Clinical practice	Research studies	Video for practical instruction, reference
Case finding	SARC-F questionnaire Ishii screening tool	SARC-F	Malmstrom *et al.* (2016) [12] Ishii *et al.* (2014) [40]
Skeletal muscle strength	Grip strength Chair stand test (chair rise test)	Grip strength Chair stand test (5-times sit-to-stand)	Roberts *et al.* (2011) [41] American Academy of Orthotists & Prosthetists https://www.youtube.com/watch?v=_jPl-IuRJ5A
Skeletal muscle mass orSkeletal muscle quality	Appendicular skeletal muscle mass (ASMM) by Dual-energy X-ray absorptiometry (DXA)*	ASMM by DXA	Schweitzer (2015) [42] Mitsiopoulos (1998) [43]
	Whole-body skeletal muscle mass (SMM) or ASMM predicted by Bioelectrical impedance analysis (BIA)*	Whole-body SMM or ASMM by Magnetic Resonance Imaging (MRI, total body protocol)	Shen (2004) [44] Sergi (2017) [45] Maden-Wilkinson (2013) [46] Heymsfield (1990) [47] Kim (2002) [48] Yamada (2017) [49]
		Mid-thigh muscle cross-sectional area by Computed Tomography (CT) or MRI	Lee (2004) [50]
	Lumbar muscle cross-sectional area by CT or MRI	Lumbar muscle cross-sectional area by CT or MRI	Van der Werf (2018) [51] Derstine (2018) [52]
		Muscle quality by mid-thigh or total body muscle quality by muscle biopsy, CT, MRI or Magnetic resonance Spectroscopy (MRS)	Goodpaster (2000) [53] Reinders (2016) [54] Grimm (2018) [55] Distefano (2018) [56] Ruan (2007) [57]
Physical performance	Gait speed	Gait speed	NIH Toolbox 4 Meter Walk Gait Speed Test https://www.nia.nih.gov/research/labs/leps/short-physical-performance-battery-sppb https://www.youtube.com/watch?v=xIScK_NXUN0
	Short physical performance battery (SPPB)	SPPB	Short Physical Performance Battery Protocol https://research.ndorms.ox.ac.uk/prove/documents/assessors/outcomeMeasures/SPPB_Protocol.pdf NIH Toolbox https://www.nia.nih.gov/research/labs/leps/short-physical-performance-battery-sppb
	Timed-up-and-go test (TUG) 400-meter walk or long-distance corridor walk (400-m walk)	TUG 400-m walk	Mathias (1986) [40] Newman (2006) [41]

*Sometimes divided by height^2 or BMI to adjust for body size.

COME MISURIAMO LA SARCOPENIA

Table 1 SARC-F screen for sarcopenia

Component	Question	Scoring
Strength	How much difficulty do you have in lifting and carrying 10 pounds?	None = 0 Some = 1 A lot or unable = 2
Assistance in walking	How much difficulty do you have walking across a room?	None = 0 Some = 1 A lot, use aids, or unable = 2
Rise from a chair	How much difficulty do you have transferring from a chair or bed?	None = 0 Some = 1 A lot or unable without help = 2
Climb stairs	How much difficulty do you have climbing a flight of ten stairs?	None = 0 Some = 1 A lot or unable = 2
Falls	How many times have you fallen in the last year?	None = 0 1–3 falls = 1 4 or more falls = 2

COME MISURIAMO LA SARCOPENIA

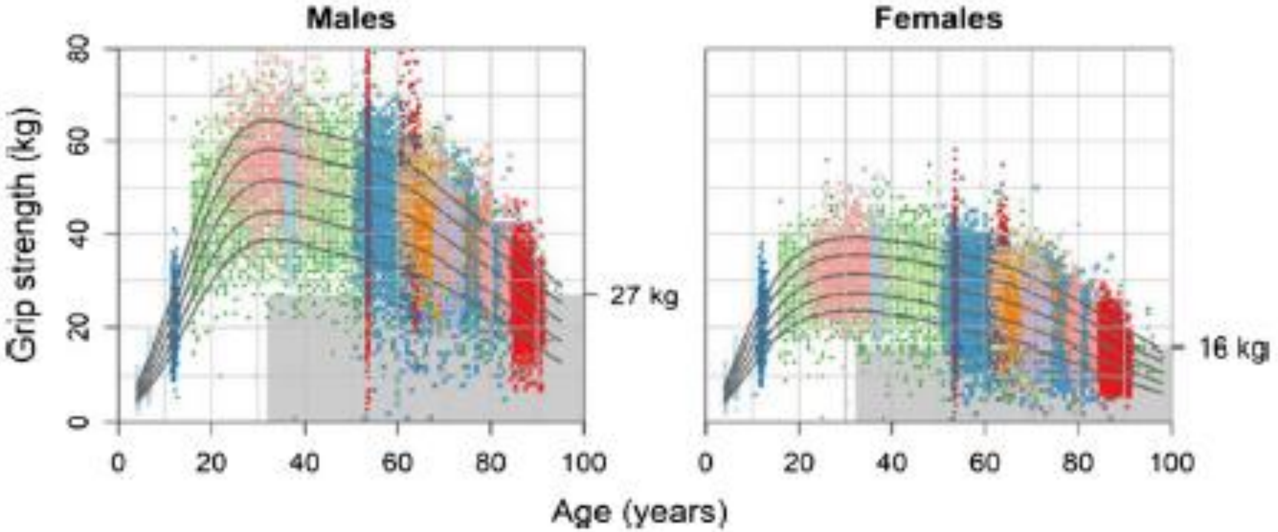


Figure 2. Normative data for grip strength across the life course in men and women in the UK (Dodds RM, *et al.* PLoS One. 2014;9:e113637). Centiles shown are 10th, 25th, 50th, 75th and 90th. Cut-off points based on *T*-score of ≤ -2.5 are shown for males and females (≤27 kg and 16 kg, respectively). Color-coding represents different birth cohorts used for the study (Figure adapted with permission from R Dodds and PLOS One).

COME MISURIAMO LA SARCOPENIA

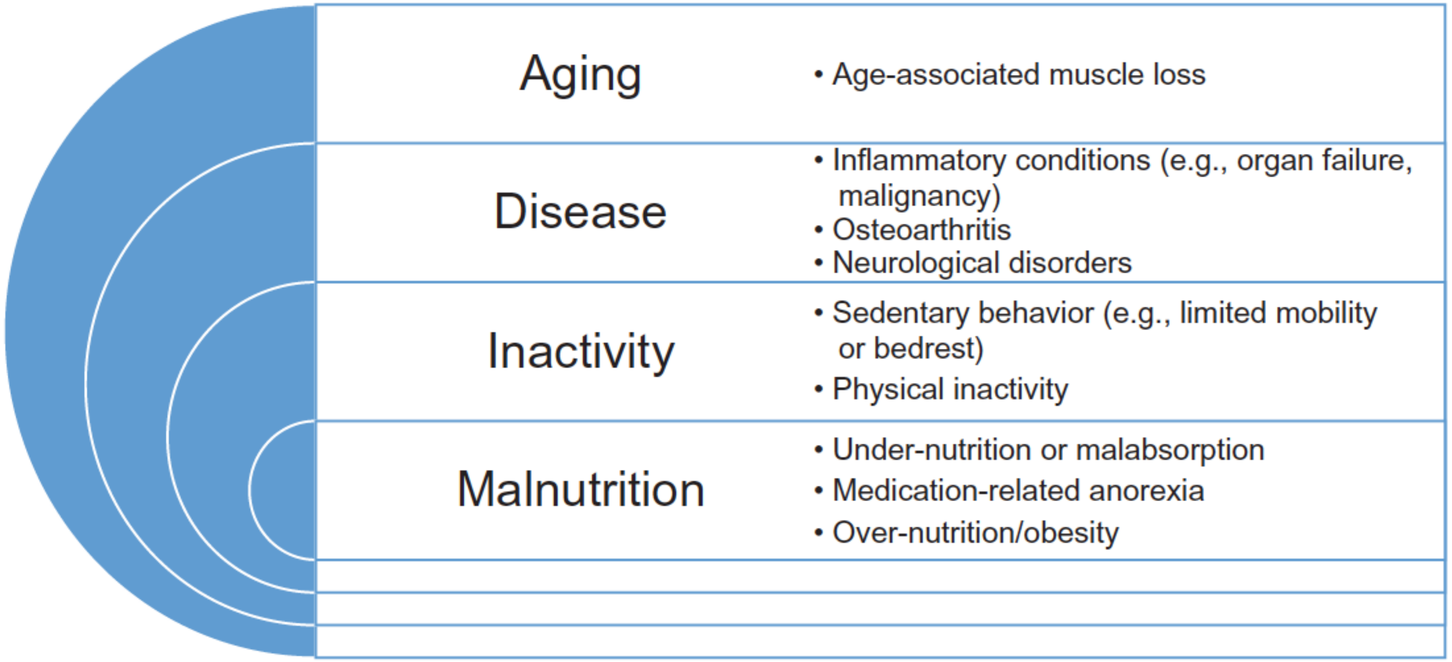


Figure 4. Factors that cause and worsen muscle quantity and quality, sarcopenia, are categorised as primary (ageing) and secondary (disease, inactivity, and poor nutrition). Because a wide range of factors contribute to sarcopenia development, numerous muscle changes seem possible when these multiple factors interact.

COME MISURIAMO LA SARCOPENIA

EDITORIAL

Journal of Cachexia, Sarcopenia and Muscle 2015; **6**: 312–314
Published online in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12079

Rapid screening for sarcopenia

John E. Morley[1*] & Li Cao[2]

[1]*Divisions of Geriatric Medicine and Endocrinology, Saint Louis University School of Medicine, St. Louis, MO, USA;* [2]*The Center of Gerontology and Geriatrics, West China Hospital, Sichuan University, Sichuan, China*

Come si previene la sarcopenia

- Prevenzione dieta ipoproteica frequente nell’anziano
    - Cibi ricchi di carboidrati sono
        - Economici
        - Facili da cucinare
        - Facili da masticare
- Prevenzione ipomobilità
    - L’anziano si muove poco perchè
        - Dolore artrosico (attenzione alla cura del piede)
        - Non gestisce l’incontinenza
        - Ha paura di cadere

E il dolore?

Table 1. Demographics and basal clinical features of the participants

	Total (n = 210)	SARC-F ≥ 4 (n = 126)	SARC-F < 4 (n = 84)	p-value
Age (y)	72.4 ± 7.0	73.8 ± 7.6	70.3 ± 5.7	< 0.001*
BMI (kg/m^2)	28.7 ± 6.1	29.7 ± 6.9	27.0 ± 3.9	0.001*
Sex, female	109 (51.9)	80 (63.5)	29 (34.5)	< 0.001*
Education (y)				
No	72 (34.2)	54 (42.9)	18 (21.4)	0.002*
0–5	107 (51)	59 (46.8)	48 (57.1)	
≥ 6	31 (14.8)	13 (10.3)	18 (21.5)	
Number of comorbidity	2 (0–6)	2 (0–6)	1 (0–4)	< 0.001*
Number of medications	2 (0–11)	2 (0–11)	1 (0–9)	< 0.001*
Falls	1 (0–2)	1 (0–2)	0 (0–2)	< 0.001*
Chronic pain, yes	195 (92.9)	125 (99.2)	70 (83.3)	< 0.001*
SARC-F (0–10)	4 (0–10)	6 (4–10)	2 (0–3)	< 0.001*
Geriatric pain measure	62 ± 22.7	73.9 ± 16.0	43.3 ± 18.6	< 0.001*
Pain intensity today (0–10)	6 (0–10)	7 (0–10)	4 (0–10)	< 0.001*
Pain intensity last 7 days (0–10)	6 (0–10)	6 (2–10)	4 (0–10)	< 0.001*
Number of pain sites				< 0.001*
0	15 (7.1)	1 (0.8)	14 (16.7)	
1	46 (21.9)	16 (12.7)	30 (35.7)	
2	51 (24.3)	29 (23)	22 (26.2)	
3	35 (16.7)	25 (19.8)	10 (11.9)	
4$^+$	63 (30)	55 (43.7)	8 (9.6)	

Values are presented as mean±standard deviation or number (%) or median (min–max).
SARC-F, strength, assistance with walking, rising from a chair, ascending stairs, and falls.
*p<0.05.

https://doi.org/10.4235/agmr.23.0081

Table 2. SARC-F: subgroup prevalence and item-response of indicators

SARC-F	Response (%)			p-value
	Mild pain (0–29)	Moderate pain (30–69)	Severe pain (70–100)	
Subgroup prevalence (%)	11.4	48.1	40.5	
Item-response				
Strength-difficulty lifting and carrying 10 lb				< 0.001*
0 (None)	58.3	35.6	9.4	
1 (Some)	41.7	51.5	40.0	
2 (A lot or unable)	0	12.9	50.6	
Climb stairs-difficulty climbing a flight of 10 stairs				< 0.001*
0 (None)	41.7	16.8	2.4	
1 (Some)	58.3	64.4	24.7	
2 (A lot or unable)	0	18.8	72.9	
Assistance in walking-difficulty walking across a room				< 0.001*
0 (None)	83.3	66.3	20.0	
1 (Some)	16.7	32.7	51.8	
2 (A lot, use aids, or unable)	0	1	28.2	
Rise from a chair-difficulty transferring from a chair or bed				< 0.001*
0 (None)	70.8	41.6	3.5	
1 (Some)	29.2	54.5	57.6	
2 (A lot or unable without help)	0	4	38.8	
Falls-times fallen in the past year				0.005*
0 (None)	70.8	52.5	35.3	
1 (1–3 falls)	20.8	36.6	38.8	
2 (≥ 4 falls)	8.3	10.9	25.9	
SARC-F (total) ≥ 4				< 0.001*
No	91.7	55.4	7.1	
Yes	8.3	44.6	92.9	

SARC-F, strength, assistance with walking, rising from a chair, ascending stairs, and falls.
*$p<0.05$.

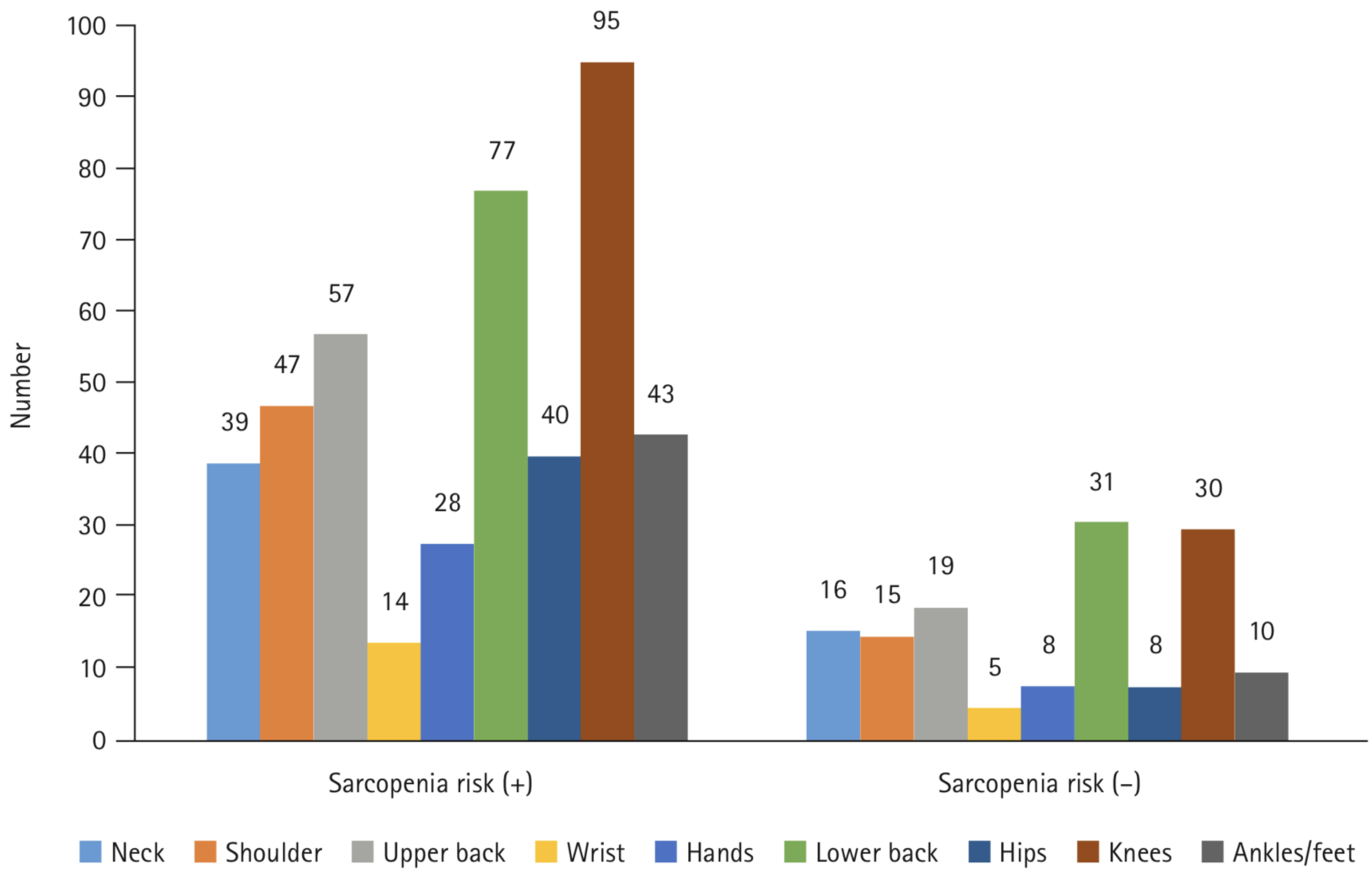


Fig. 2. Distributions of pain site in older adults with and without sarcopenia risk.

Table 3. Correlations among sarcopenia risk and chronic pain intensity, multisite pain, and total score of GPM

	Multisite pain	Pain intensity today	Pain intensity last 7 days	GPM	SARC-F
Multisite pain	-	0.436**	0.493**	0.547**	0.442**
Pain intensity today	0.436**	-	0.727**	0.847**	0.506**
Pain intensity last 7 days	0.493**	0.727**	-	0.833**	0.584**
GPM	0.547**	0.847**	0.833**	-	0.730**
SARC-F	0.442**	0.506**	0.584**	0.730**	-

GPM, Geriatric Pain Measure; SARC-F, strength, assistance with walking, rising from a chair, ascending stairs, and falls.
**$p<0.001$.

Table 4. Logistic regression analysis between multisite pain, GPM score, and sarcopenia risk status

	B	SE	Wald	df	Sig.	Exp(B)	
						OR	95% CI
Age	0.111	0.036	9.365	1	0.002*	1.117	1.041–1.199
BMI	0.118	0.046	6.526	1	0.011*	1.126	1.028–1.233
GPM	0.101	0.014	49.206	1	< 0.001*	1.106	1.075–1.138
Constant	-16.732	3.428	23.817	1	< 0.001	0.000	-

GPM, Geriatric Pain Measure; BMI, body mass index; SE, standard error; OR, odds ratio; CI, confidence interval.
Omnibus test (χ^2=128.534, df=3, $p<0.001$), Hosmer–Lemeshow test ($p>0.05$), Nagelkerke R^2=0.621.
*$p<0.05$.

Study or Subgroup	Mean Difference	SE	Weight	Mean Difference IV, Random, 95% CI
Fransen 2014	0.0417335	0.008012	10.3%	0.04 [0.03, 0.06]
Imai 2022	0.22	0.0585833	4.7%	0.22 [0.11, 0.33]
Lin 2022	0.1024096	0.0166395	9.6%	0.10 [0.07, 0.14]
Maruya 2019	0.0441176	0.011137	10.1%	0.04 [0.02, 0.07]
Sakai 2017	0.4	0.0489898	5.7%	0.40 [0.30, 0.50]
Scott 2021	0.0569948	0.0118	10.0%	0.06 [0.03, 0.08]
Smith 2022	0.1379175	0.0034127	10.5%	0.14 [0.13, 0.14]
Sun 2017	0.0751979	0.0095784	10.2%	0.08 [0.06, 0.09]
Tanishima 2017	0.0714286	0.021766	9.0%	0.07 [0.03, 0.11]
Tsuji 2020	0.0767386	0.0130347	9.9%	0.08 [0.05, 0.10]
Veronese 2023	0.1618056	0.0097048	10.2%	0.16 [0.14, 0.18]
Total (95% CI)			**100.0%**	**0.11 [0.08, 0.14]**

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 283.53$, df = 10 ($P < 0.00001$); $I^2 = 96\%$

Test for overall effect: $Z = 6.39$ ($P < 0.00001$)

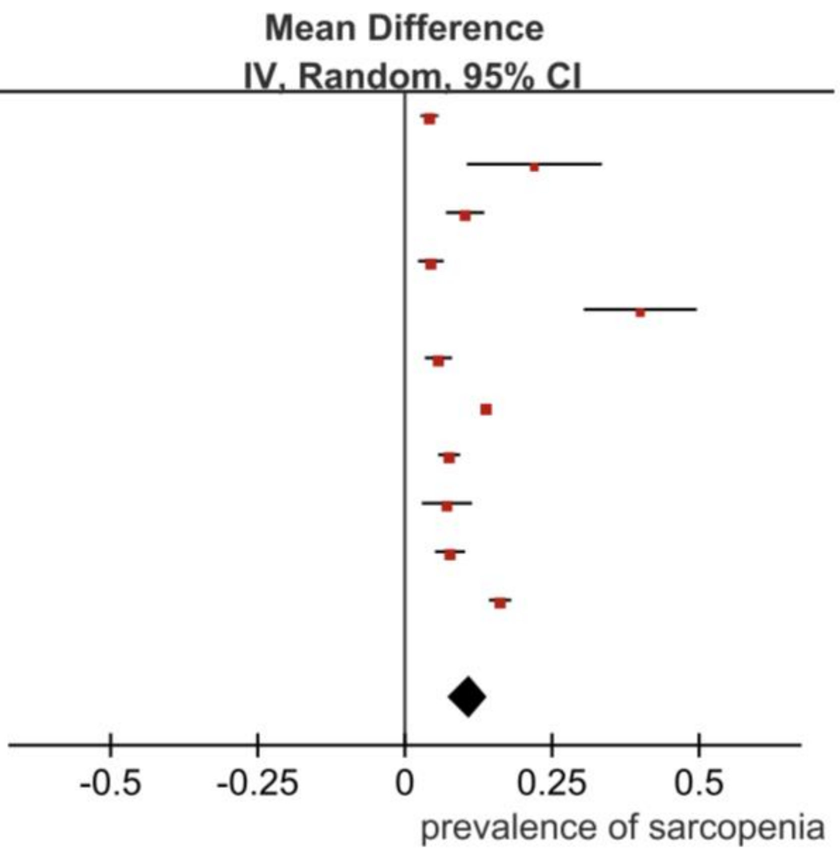


Figure 2 Forest plot of the prevalence of sarcopenia among older adults with chronic pain.

DOI https://doi.org/10.2147/JPR.S435866

https://doi.org/10.3389/fmed.2021.630009

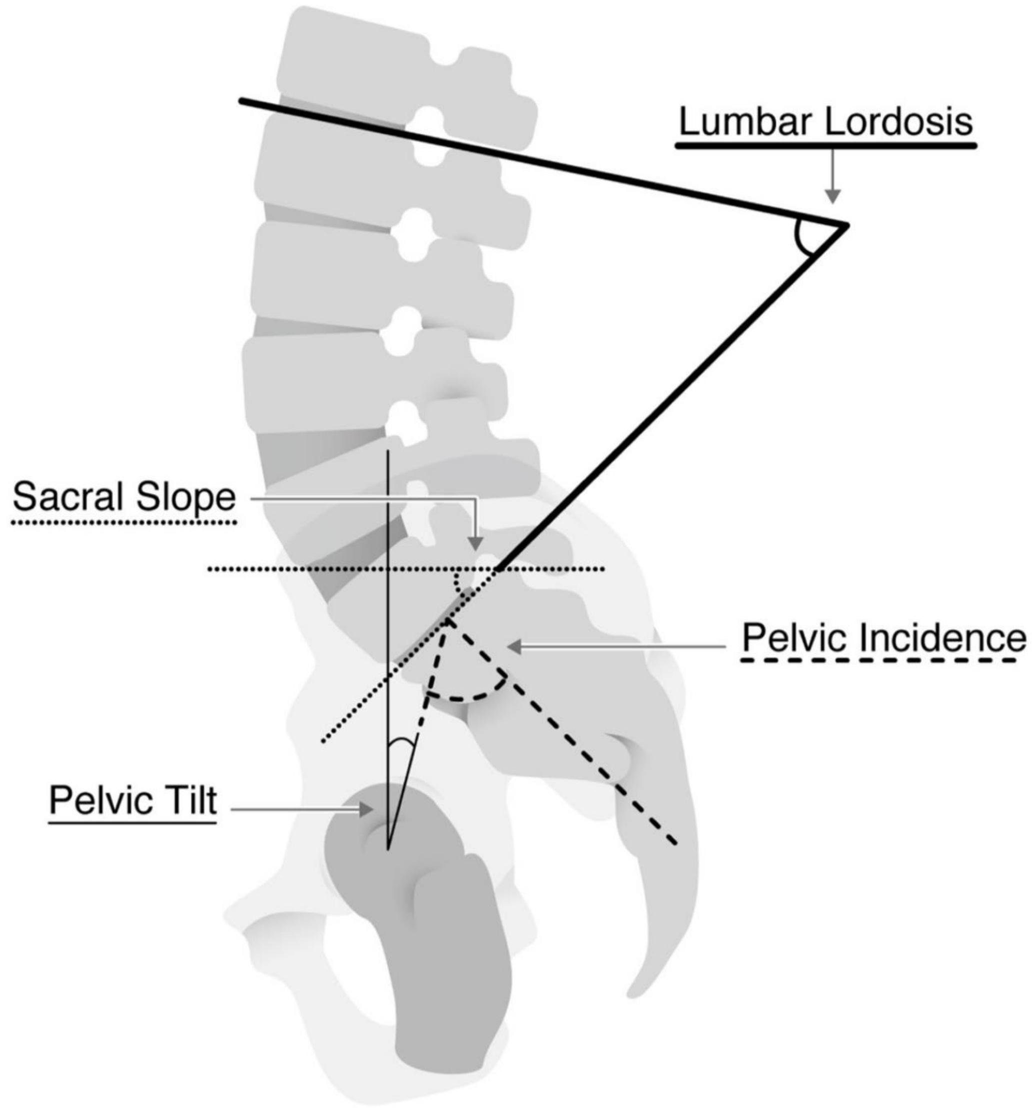


Fig. 1 Radiographic parameter of spinal alignment. LL is measured from the inferior endplate of T12 to the superior endplate of S1. SS is measured as the angle between the sacral plate and the horizontal line.PT is measured by the angle between the vertical and the line through the midpoint of the sacral plate to the femoral heads axis. PI is measured as the angle between the line perpendicular to the sacral plate at its midpoint and the line connecting this point to the femoral head's axis. "PI = PT -SS. Abbreviations:LL, lumbar lordosis; PI, pelvic incidence; SS, sacral slope; PT, pelvic tilt

https://doi.org/10.1186/s12891-022-05086-2

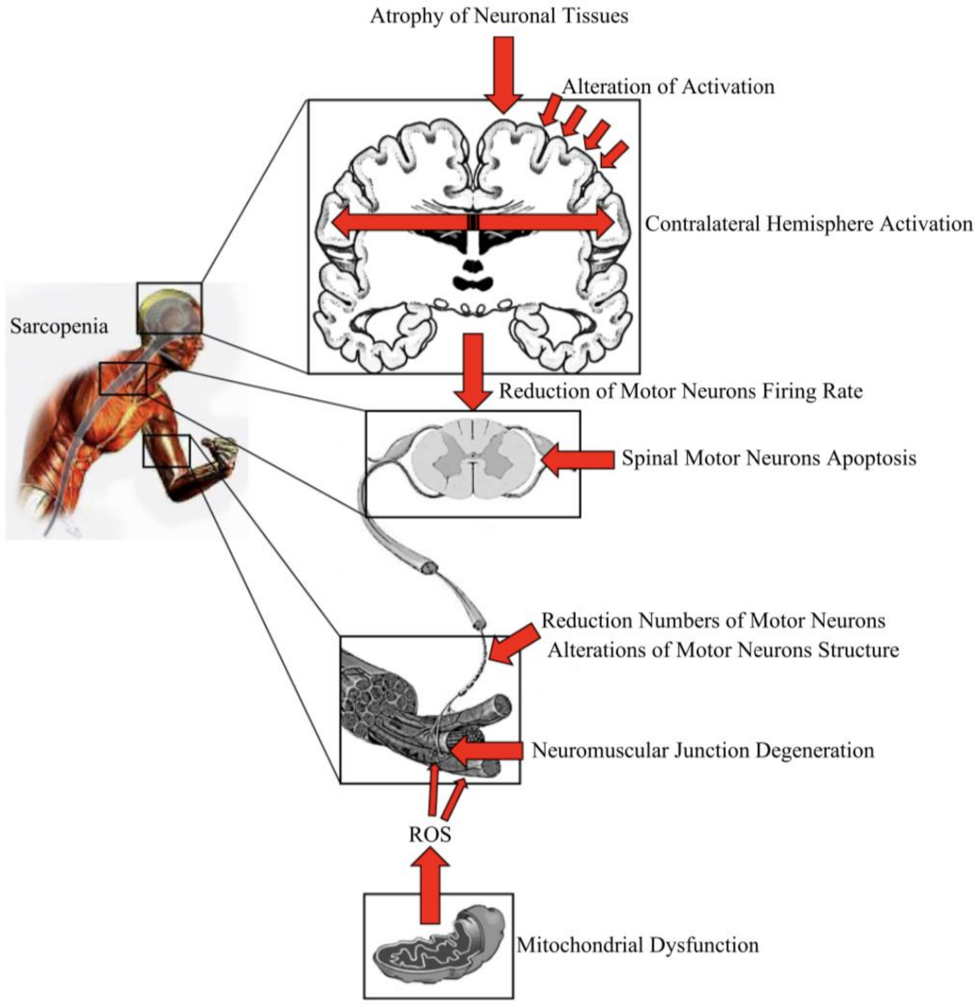
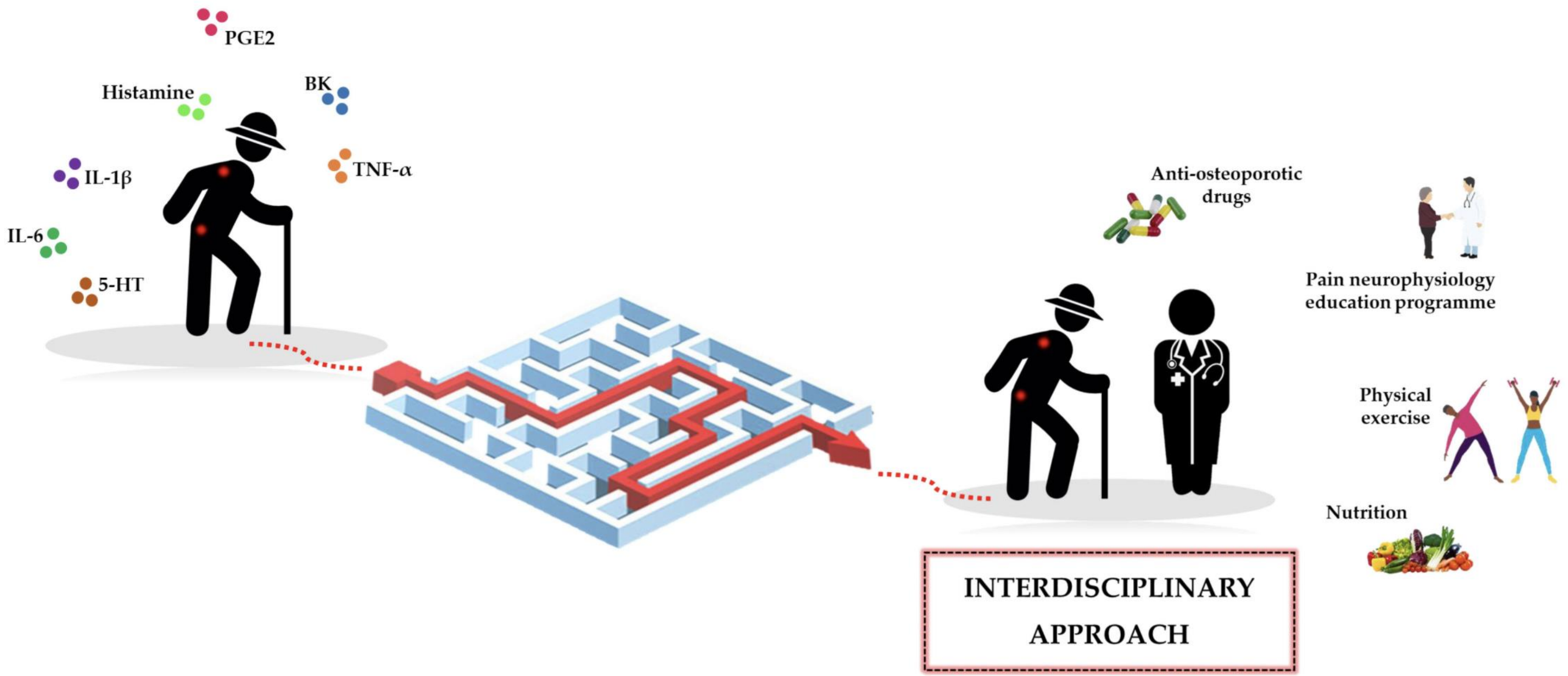


Fig. 1 Conceptual schematic of the neuromuscular factors responsible for age-related sarcopenia. The reduction in cortical inputs, increased cerebral cortex atrophy, increased motor-neurons apoptosis and neuromuscular junction impairment associated with increased levels of reactive oxygen species (ROS) derived from mitochondrial dysfunction are key factors underpinning the loss of skeletal muscle mass and function

https://doi.org/10.1007/s40520-022-02082-3



A possible way out of the labyrinth of pain in osteosarcopenia. Pain is a condition found in many osteosarcopenic patients. Several molecular mediators and complex biological mechanisms are involved in the development and persistence of pain in this geriatric syndrome. An interdisciplinary approach should necessarily be taken to improve musculoskeletal health and reduce the algic condition. The combination of pharmacological and non-pharmacological strategies appears to be crucial to get out of the pain labyrinth.

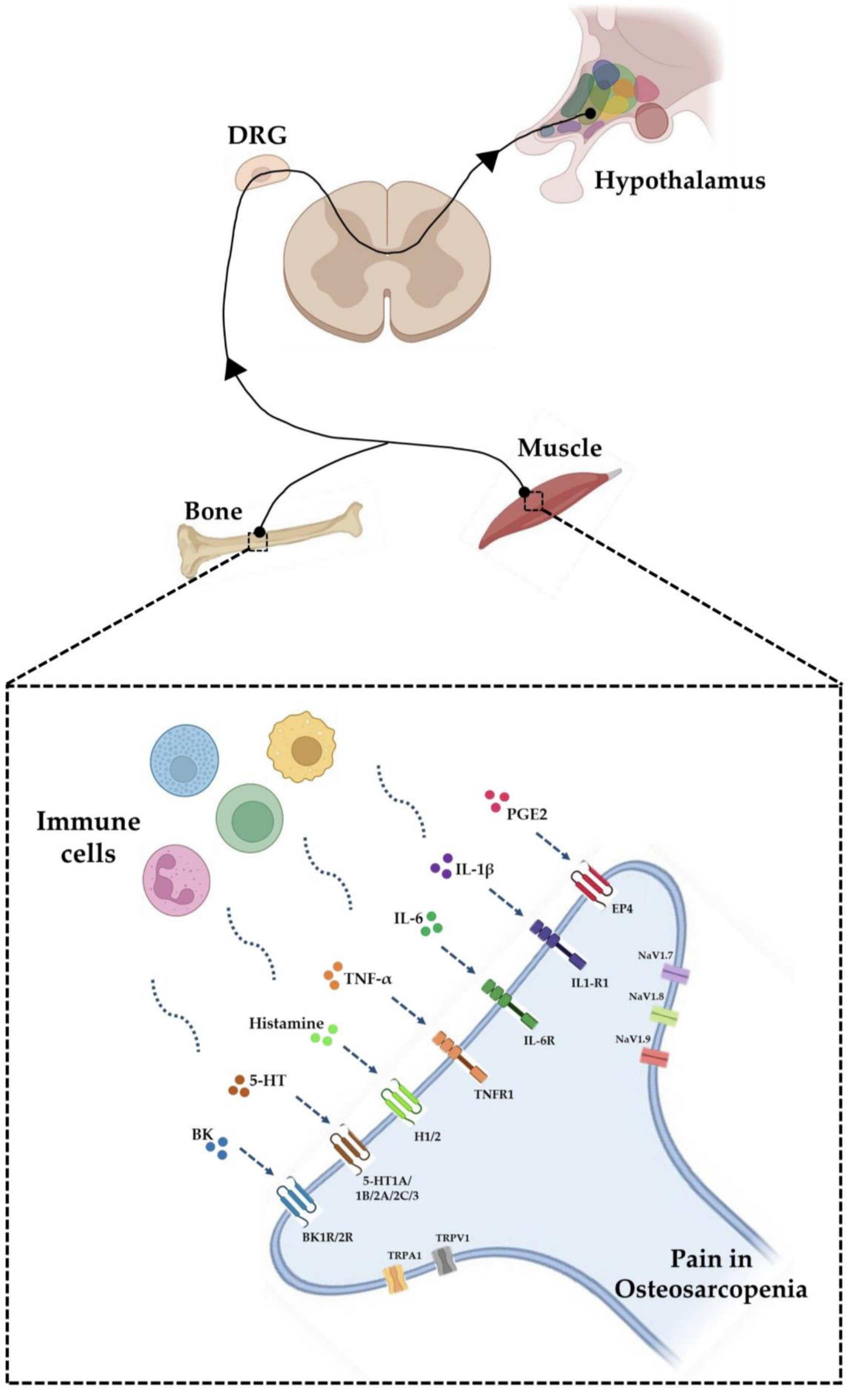


Figure 1. Development and transmission of the nociceptive signal in the presence of bone and/or muscle damage. The neurons responsible for encoding and transducing harmful musculoskeletal stimuli are in the dorsal root ganglia (DRG). In the presence of bone and/or muscle tissue damage, immune cells release numerous mediators, including lipid mediators such as prostaglandin E2 (PGE2), cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor α (TNF-α), as well as neurotransmitters such as histamine, serotonin (5-HT) and bradykinin (BK). All these binds to their receptors on the membrane of the nociceptive axon terminal, inducing the opening of ion channels, including transient receptor potential cation channel subfamily V member 1 (TRPV1), transient receptor ankyrin 1 (TRPA1) and voltage-dependent sodium channels (NaV1.7, NaV1.8 and NaV1.9). The resulting flux of ions promotes depolarization of the axon terminal of the nociceptive fibre, promoting the generation and transmission of the nociceptive signal.

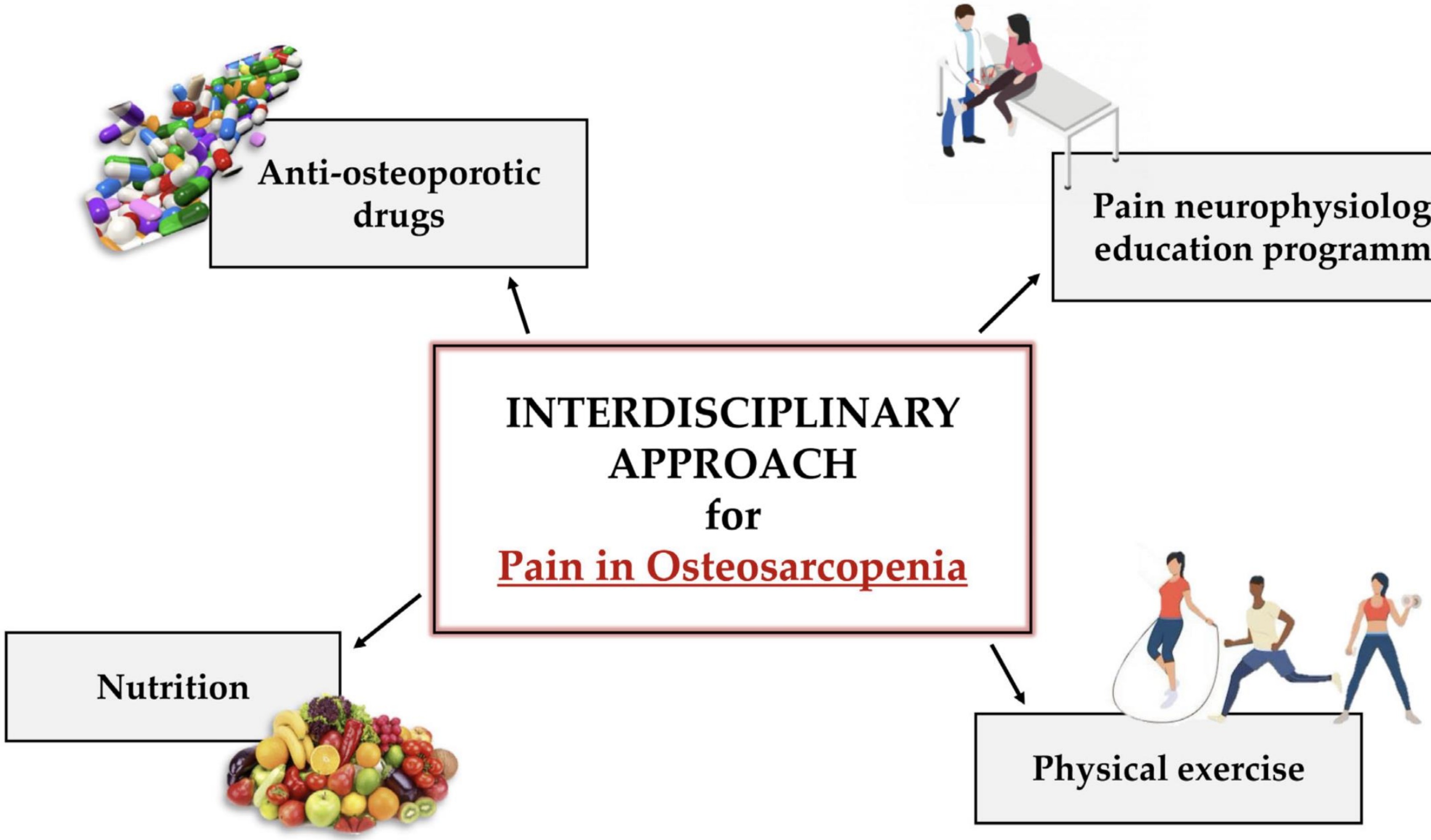


Figure 2. Interdisciplinary approach for the management of osteosarcopenic (OSP) patients. The pain management in OSP patients should involve an interdisciplinary approach, in which several professionals collaborate in the development of a therapy aimed at minimizing the pain perception. This strategy should include both multimodal pharmacological therapies, based on the use of anti-osteoporotic drugs, and non-pharmacological therapies including pain neurophysiology education programme (PNE), physical exercise and nutrition. Such an integrated approach will be essential to act simultaneously on the algic and musculoskeletal components.

Biomedicines 2023, 11, 1285. https://doi.org/10.3390/biomedicines11051285

Table 2 Patient characteristics at the time of visit

	Sarcopenia ($N=32$)		Non-sarcopenia ($N=68$)		p
	Median (IQR)	Range (min-max)	Median (IQR)	Range (min-max)	
Age	81.5 (74.5-87.8)	63-97	71.5 (68.3-80.0)	60-91	0.0001
Sex (female/male)	28/4		60/8		0.9159
BMI (kg/m^2)	20.7 (18.8-23.1)	14.3-26.3	22.5 (20.7-24.7)	17.5-32.2	0.0072
SMI (kg/m^2)	5.1 (4.7-5.5)	4.2-6.8	6.1 (5.7-6.7)	4.9-7.9	<.0001
BMD (femoral neck)	0.50 (0.42-0.56)	0.38-0.83	0.56 (0.50-0.64)	038-0.90	0.0079
Knee extension torque (kgf/kg)	11.7 (6.4-15.8)	3.4-20.3	17.1 (13.1-21.5)	3.9-46.3	<.0001
History of vertebral fracture (N)	19(55.88%)		14(20.89%)		0.0004
Adult spinal deformity (N)	14(43.75%)		12(17.91%)		0.0063
Pelvic Tilt (°)	29.0 (21.2-37.8)	8.7-57.0	22.0 (16.0-28.0)	4.0-39.7	0.005
Lumbar Lordosis (°)	36.0 (26.3-49.8)	12.0-68.0	43.0 (36.0-52.9)	3.0-79.0	0.0445
Pelvic Incidence (°)	54.0 (49.0-68.5)	38.0-91.0	55.0 (50.0-61.0)	34.0-85.0	0.6614
Sacral Slope (°)	28.0 (23.3-36.8)	13.0-56.2	33.2 (27.0-40.0)	2.0-54.0	0.0441

Abbreviations: BMI body mass index, *BMD* bone mineral density, *SMI* skeletal muscle mass, *VAS* Visual Analogue Scale

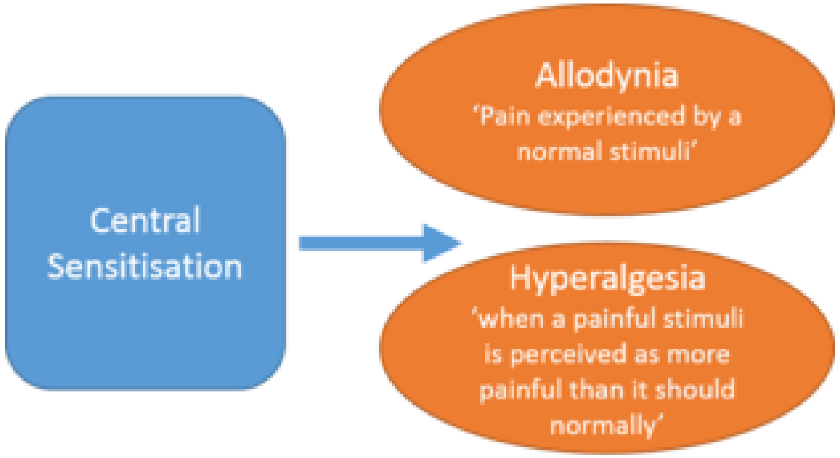
Physiopedia

Pain Neuroscience Education (PNE)

With respect to PNE, chronic pain is not viewed as a result of unhealthy or dysfunctional tissues. Rather, it is due to brain plasticity leading to hyper-excitability of the central nervous system, known as central sensitization.[3] The ultimate goal for Pain Neuroscience Education (PNE) is to increase pain tolerance with movement (e.g., be able to perform exercise with mild discomfort), reduce any fear associated with movement, and reduce central nervous system hypersensitivity. In practice, this often includes the use of educational pain analogies, re-education of patient misconceptions regarding disease pathogenesis, and guidance about lifestyle and movements modifications that can be introduced.

There are two clinical indications for initiating Pain Neuroscience Education (PNE)[4]:

- the clinical picture is dominated by central sensitization
- illness coping mechanisms or poor illness perception is present



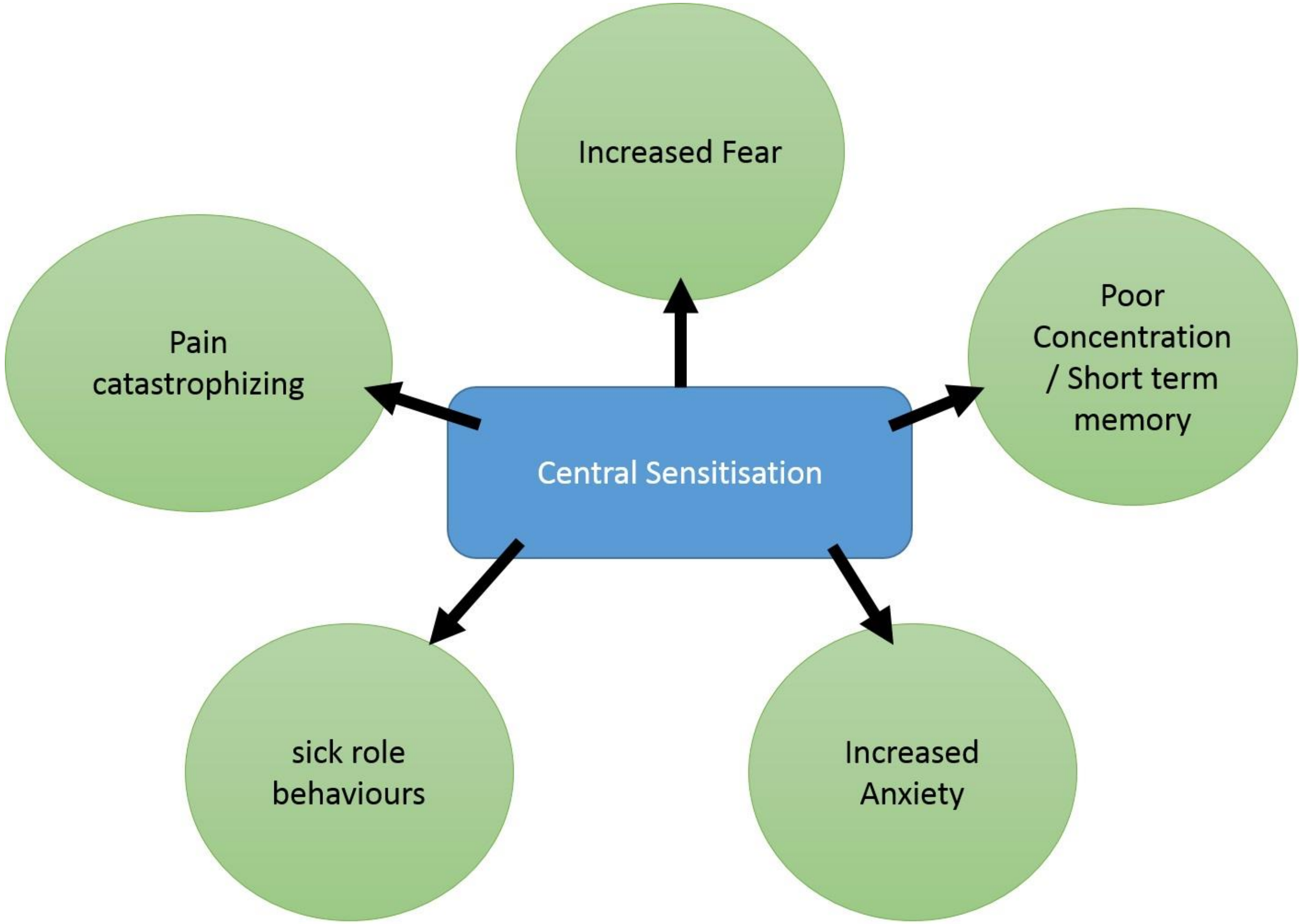
Effects of central sensitization

Central sensitization is when there is amplification of pain in the central nervous system. It can result in hypersensitivity to stimuli, responsiveness to non-noxious stimuli, and increased pain response evoked by stimuli outside the area of injury, an expanded receptive field. [5]This can be assessed during the subjective and objective portion of a patient's evaluation. A physical therapist can determine what a patient's perception of their own pain is and how they cope with their pain.

https://www.physio-pedia.com/Pain_Neuroscience_Education_(PNE)

PNE aims to reconceptualize pain to patients with these four main points:

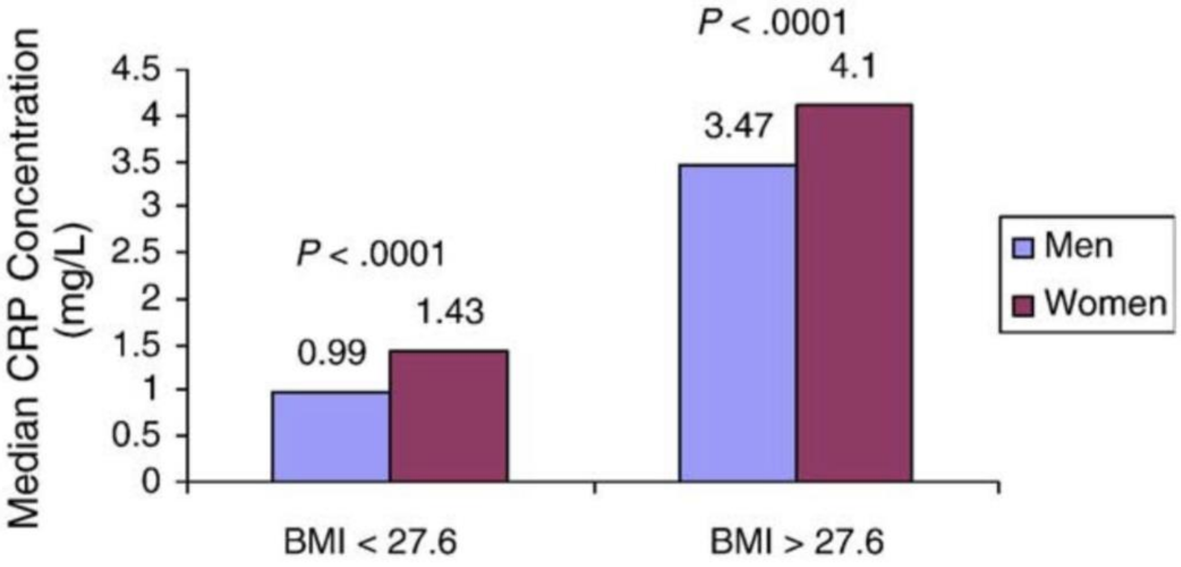
- Pain does not provide a measure of the state of the tissues
- Pain is modulated by many factors from somatic, psychological, and social domains
- The relationship between pain and the state of tissues becomes less predictable as pain persists
- Pain can be conceptualized as the conscious correlate of the implicit perception that tissue is in danger[6]



Application of PNE

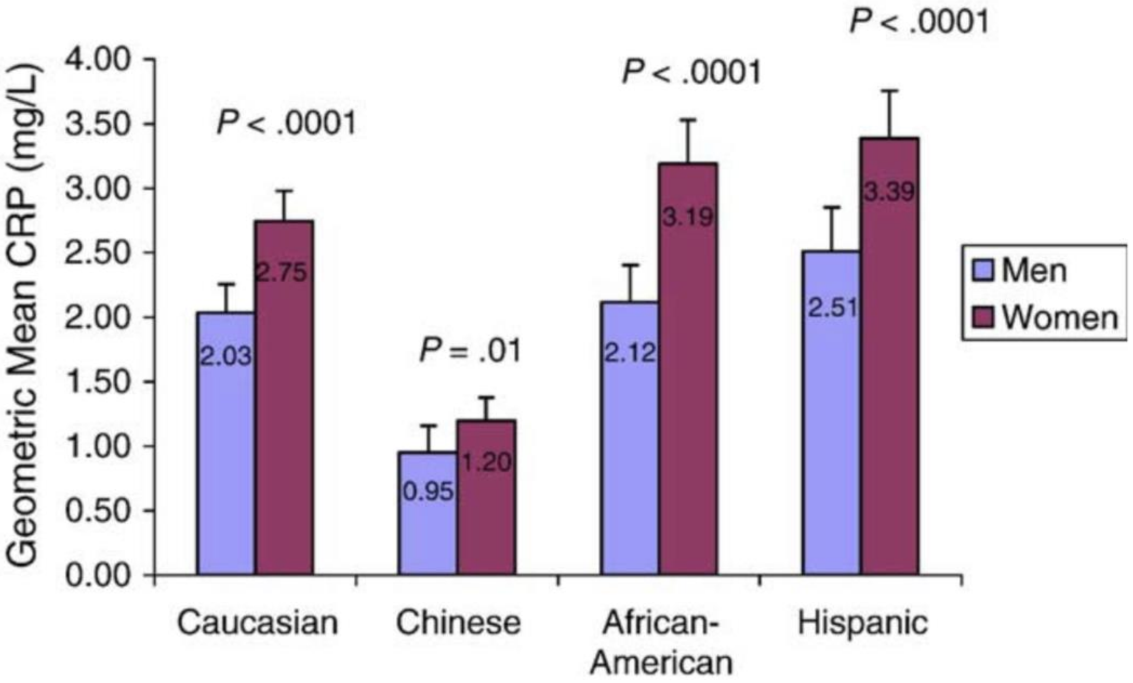
The application of PNE is most useful as part of a combination therapy for chronic pain that includes physiotherapy intervention (including exercise therapy) and may or may not include pharmacological treatment. Its application is best applied by trained and skilled clinicians with experience in managing patients with chronic pain conditions. Overall, PNE serves as a method of reconceptualizing a patient's perception of their pain experience, providing an avenue for reducing pain, disability and improving quality of life[6]. PNE puts the complex process of describing the nerves and brain into a format that is easy to understand for everyone regardless of age, educational level, or ethnic group.[7]

Figure 3



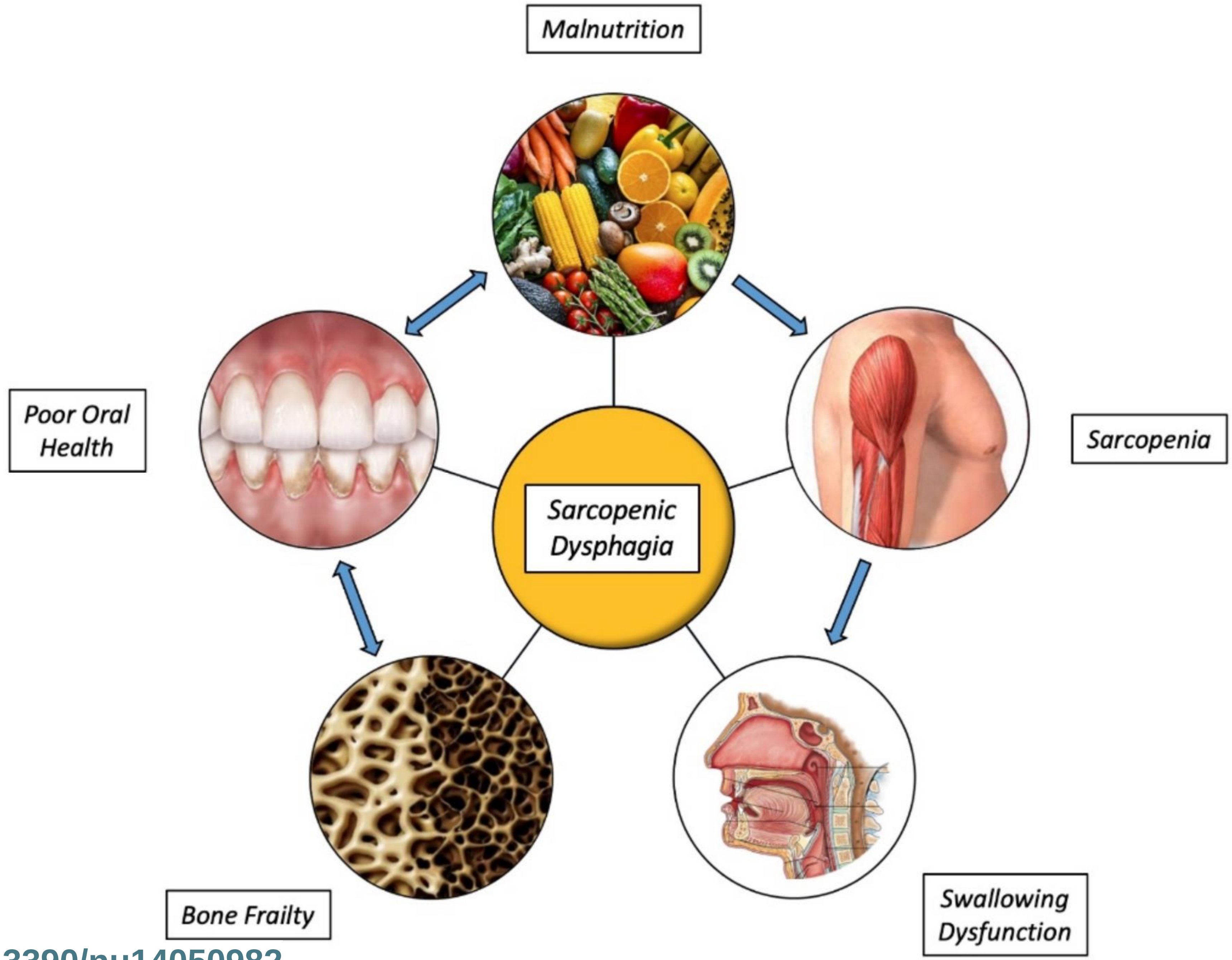
Median CRP concentration in men and women stratifying by BMI.

Figure 4



Adjusted geometric mean CRP concentration in men and women by ethnic group, adjusted for age, BMI, diabetes, hypertension, smoking, alcohol use, HMG-CoA reductase inhibitors, aspirin use, estrogen medications, physical activity, LDL, and HDL.

https://doi.org/10.4235/agmr.23.0081



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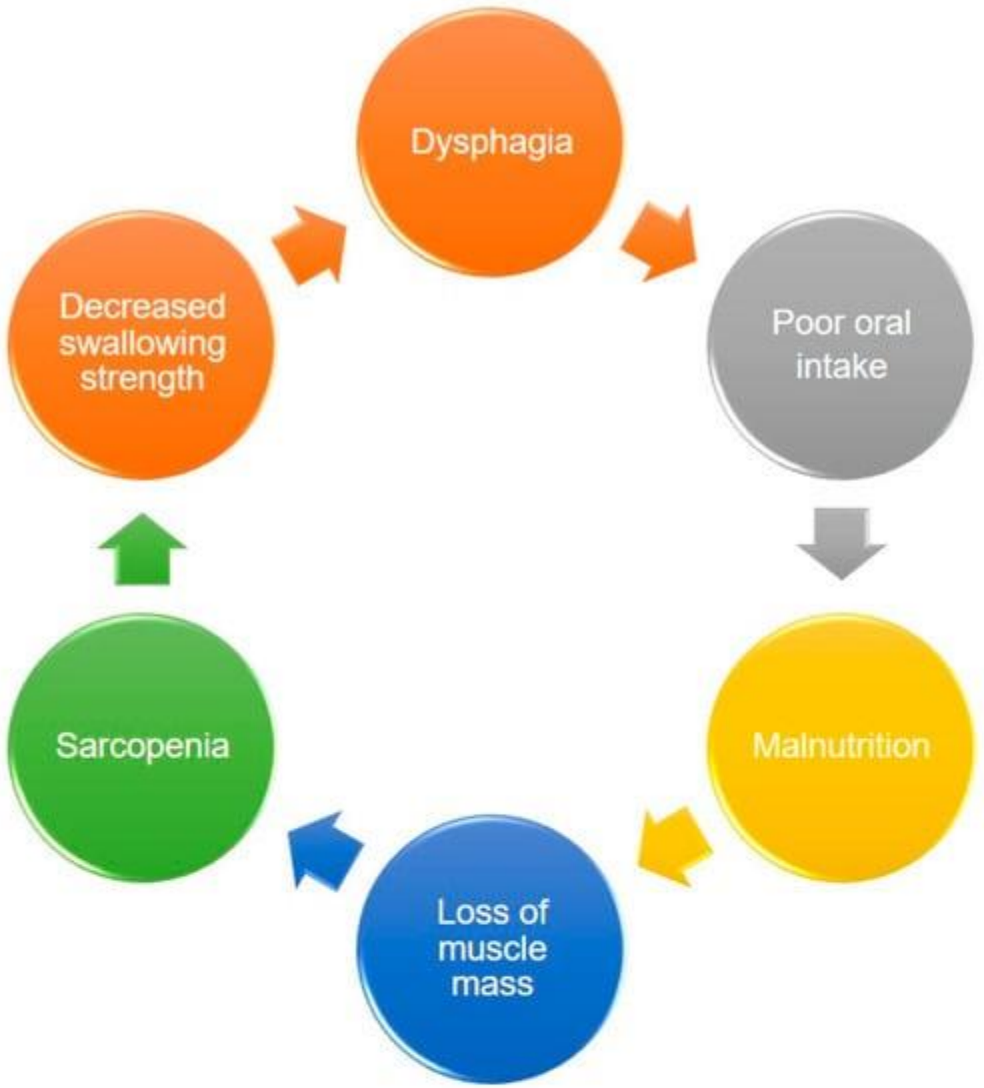

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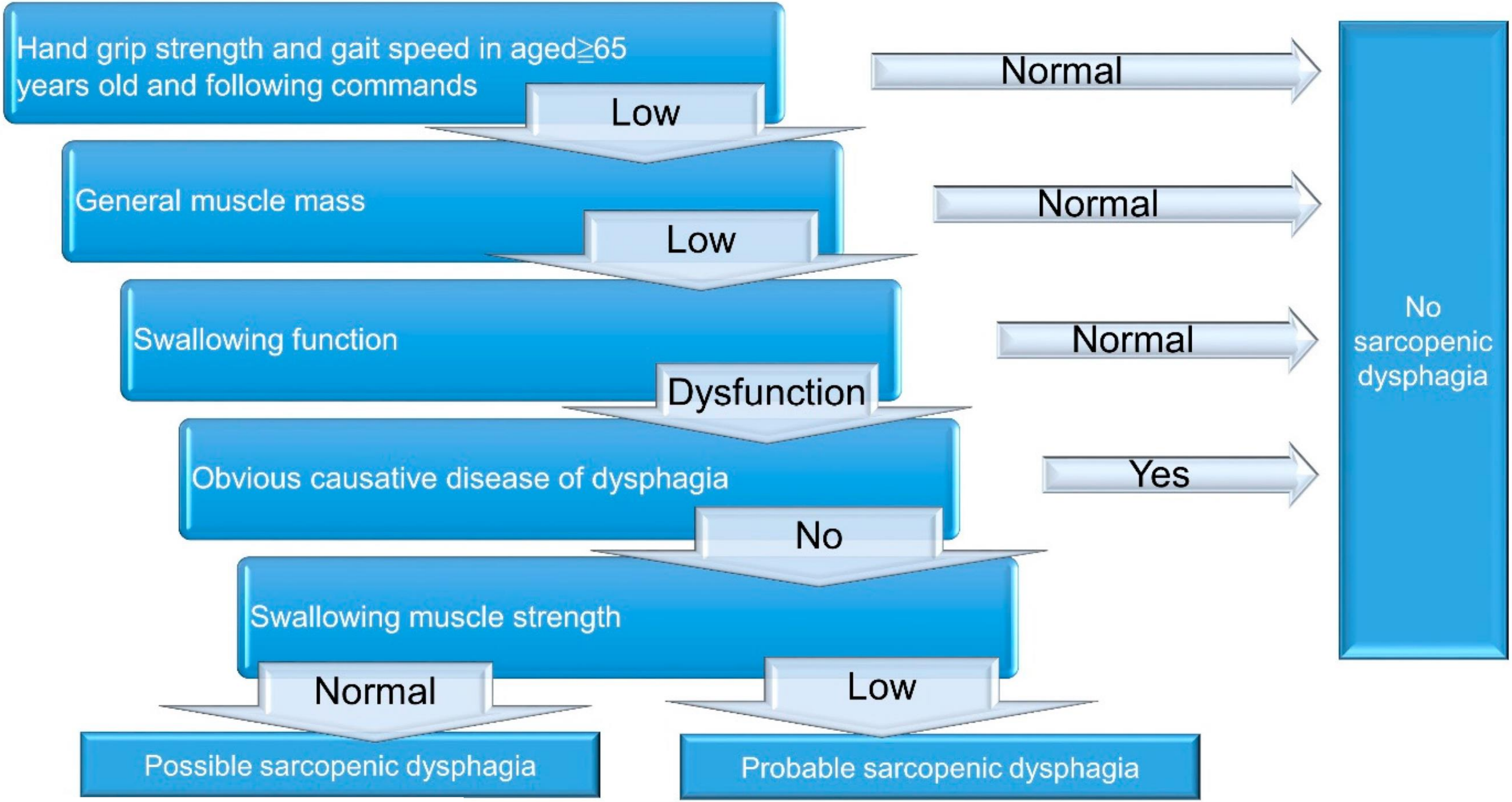


https://doi.org/10.3390/nu13114043

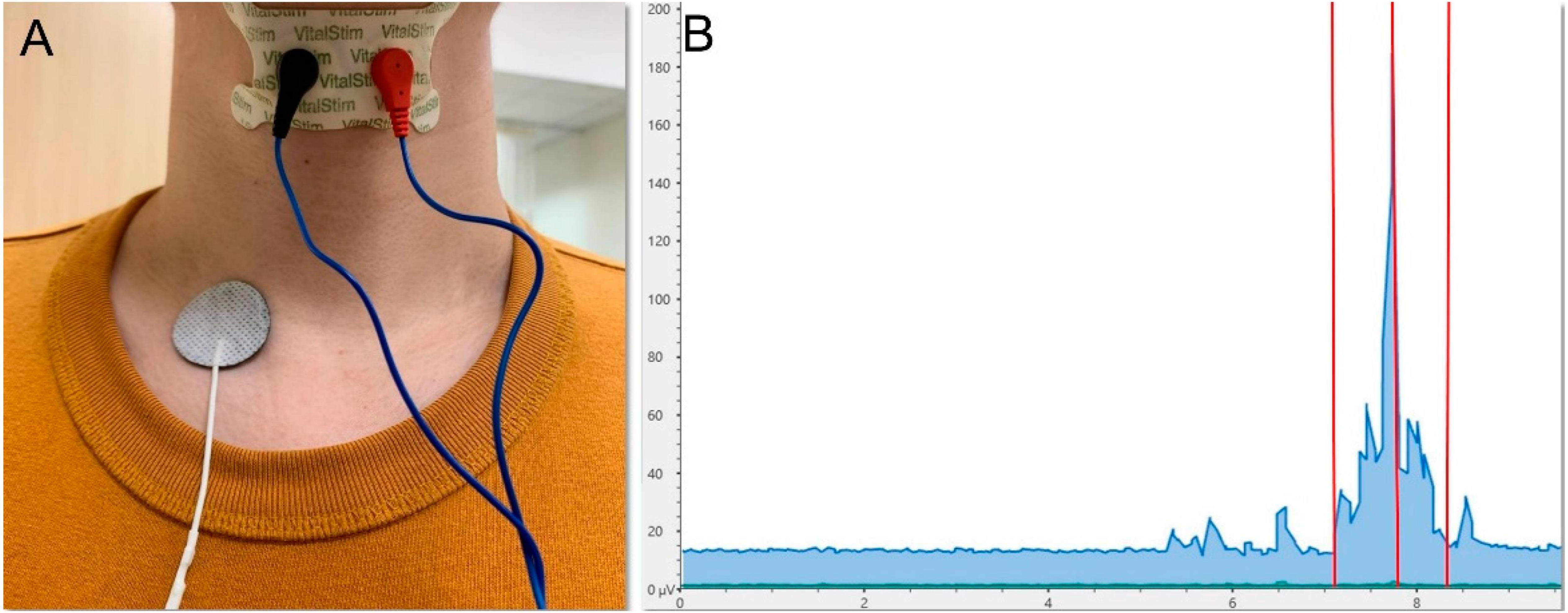
Table 2. The tools for assessing sarcopenic dysphagia.

Evaluating Target	Tools
Muscle mass	Dual-energy X-ray absorptiometry (DXA), bioimpedance analysis (BIA)
Muscle strength	Dynamometer
Physical performance	Six-minute walk test (6MWT), Short Physical Performance Battery score (SPPB), five-time chair stand test (5TSTS), timed up-and-go test (TUG), 400 m walk test (400MWT)
Swallowing function	Eating Assessment Tool (EAT-10), dysphagia severity scale (DSS), repetitive saliva swallowing Test (RSST), Functional Oral Intake Scale (FOIS), Food Intake Level Scale (FILS), modified water swallowing test (MWST), videofluoroscopy swallowing study (VFSS)
Swallowing muscle strength	JMS tongue pressure measuring instrument (JMS, Hiroshima, Japan), Iowa Oral Performance Instrument (IOPI), jaw-opening force trainer KT2016 (Livet Inc., Tokyo, Japan), Lip de Cum (Cosmo Instruments Co., Ltd., Tokyo, Japan), surface electromyography(sEMG), high-resolution manometry (HRM)
Swallowing muscle mass	Ultrasonography, magnetic resonance imaging (MRI)

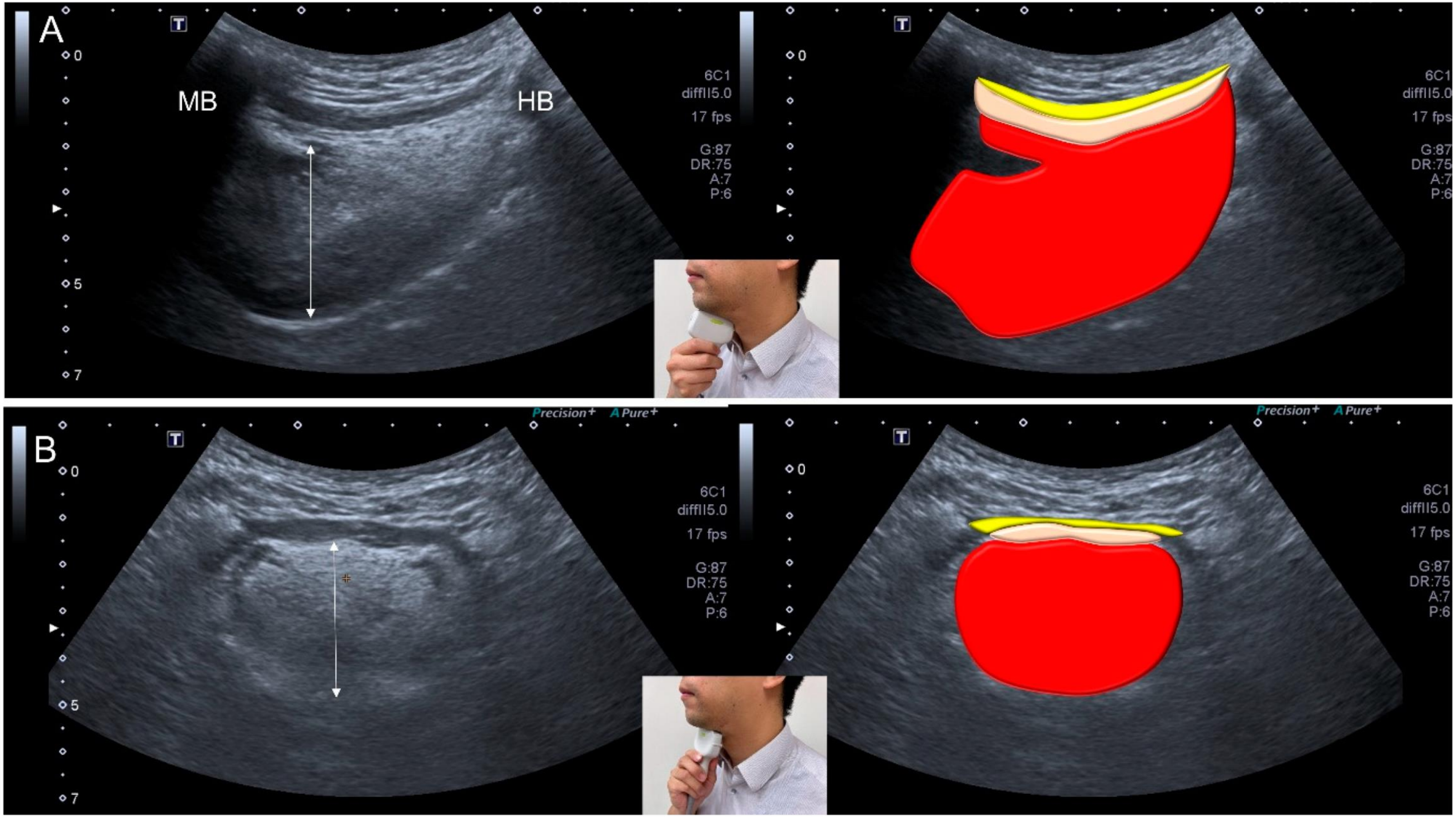
Table 3. The cut-off point of the tools for the diagnosis of sarcopenic dysphagia from the available literature.

Evaluating Tool	Cut-Off Point
Muscle mass	
Dual-energy X-ray absorptiometry(DXA)	<7.0 kg/m^2 in men and <5.5 kg/m^2 in women [a] <7.0 kg/m^2 in men and <5.4 kg/m^2 in women [b]
Bioimpedance analysis (BIA)	<7.0 kg/m^2 in men and <5.5 kg/m^2 in women [a] <7.0 kg/m^2 in men and <5.7 kg/m^2 in women [b]
Muscle strength	
Dynamometer	<27 kg for men and <16 kg for women [a] <28 kg for men and <18 kg for women [b]
Physical Performance	
6 min walk	<0.8 m/s [a] <1.0 m/s [b]
Short Physical Performance Battery	≤8 [a] ≤9 [b]
5-time chair stand test	≥15 s [a] ≥12 s [b]
Timed up-and-go test	≥20 s [a]
400 m walk	≥6 min [a]
Swallowing function	
Eating Assessment Tool (EAT-10)	≥3
Dysphagia severity scale (DSS)	≤4
Repetitive saliva swallowing Test (RSST)	≤2
Functional Oral Intake Scale (FOIS)	≤5
Food Intake Level Scale (FILS)	Not available
Modified water swallowing test (MWST)	Not available
Videofluoroscopy swallowing study (VFSS)	Not available
Swallowing muscle strength	
Maximal isometric tongue pressure	<20 kPa
Jaw-opening force	Not available
Lip force	<10.4 Newton for men and <8.5 Newton for women
Surface electromyography (sEMG)	<387.09% of jaw open contraction for maximal amplitude <1.96 s for total duration
High-resolution manometry (HRM)	Not available
Swallowing muscle mass	
Ultrasonography	<1536 mm^2 for the cross-sectional area of the tongue muscle <75.1 mm^2 for the cross-sectional area of the digastric muscle
Magnetic resonance imaging (MRI)	Not available

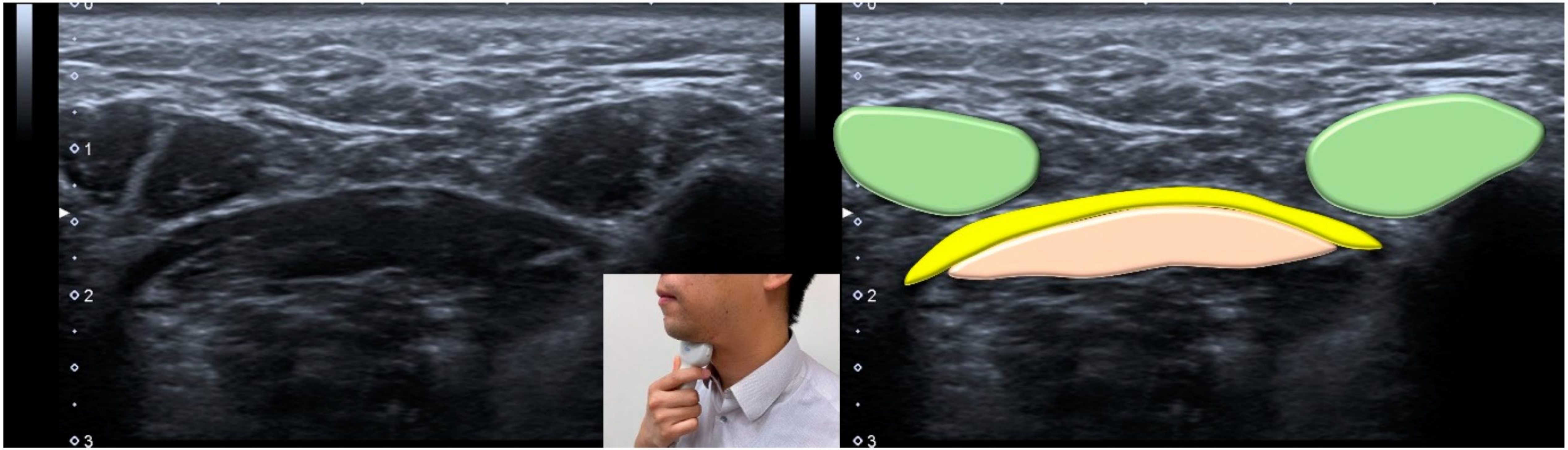
he Iowa oral performance instrument (**A**) and how it is used for obtaining maximal tongue pressure (**B**).



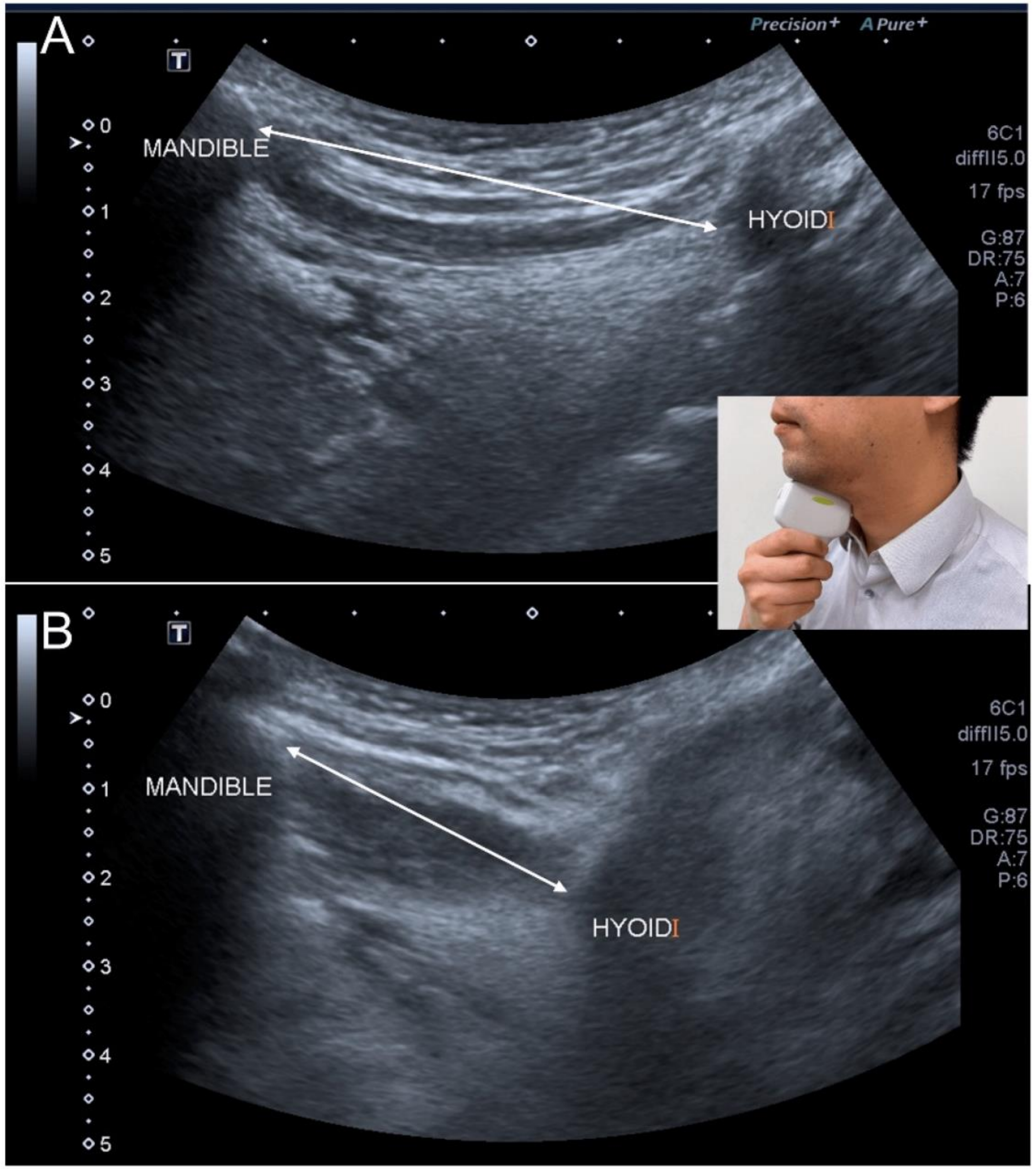
Electrode placement (**A**) and signal display (**B**) for the electromyographic assessment of swallowing muscle activity. Left red vertical line, onset of swallowing; middle red vertical line, peak amplitude; right red vertical line, end of swallowing.



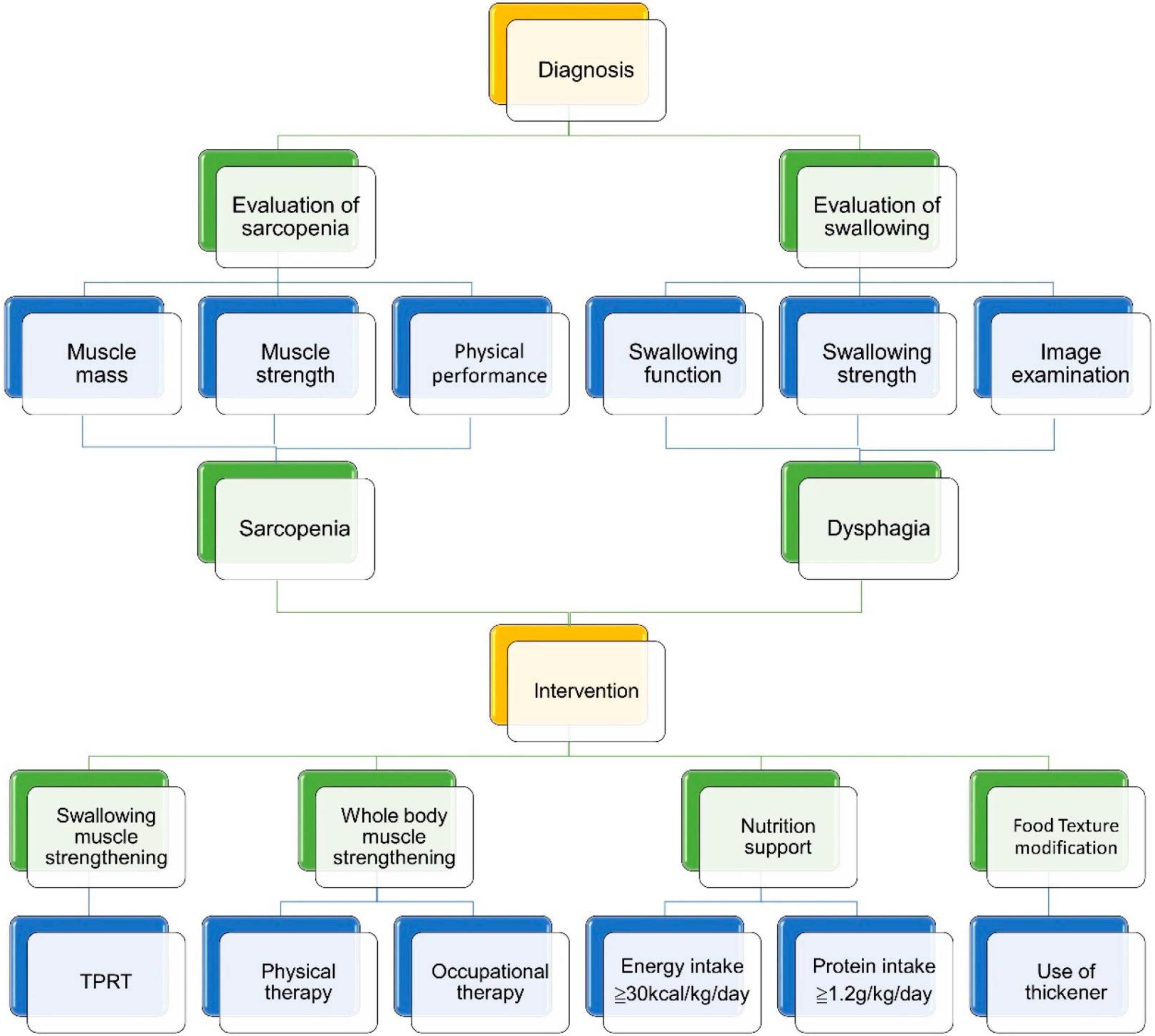
Ultrasonographic images and schematic drawings of tongue and adjacent muscles in the sagittal (A) and coronal (B) planes. Tongue muscle: red color block; geniohyoid muscle, pink color block; mylohyoid muscle, yellow color block; MB: mandible; HB: hyoid bone; double arrowed line, thickness of the tongue muscle.



Ultrasonographic images and schematic drawing of the anterior belly of the digastric muscle in the coronal plane. Digastric muscle, green color block; mylohyoid muscle, yellow color block; geniohyoid muscle, pink color block



Ultrasonographic images show the hyoid movement at the onset of swallowing (A) and the moment of maximal displacement (B). Double-headed line, the distance between the mandible and hyoid bone.



Diagnosis
Evaluation of sarcopenia
Evaluation of swallowing
Muscle mass
Muscle strength
Physical performance
Swallowing function
Swallowing strength
Image examination
Sarcopenia
Dysphagia
Intervention
Swallowing muscle strengthening
Whole body muscle strengthening
Nutrition support
Food Texture modification
TPRT
Physical therapy
Occupational therapy
Energy intake ≧30kcal/kg/day
Protein intake ≧1.2g/kg/day
Use of thickener