Risposte fisiologiche all’allenamento nell’atleta master. Cosa cambia con l’età

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Fisiopatologia dell'invecchiamento

-L'invecchiamento è un fenomeno complesso derivante dall'interazione tra il decadimento biologico di cellule e organi e i fenomeni patologici che questo comporta. Si accompagna a un decremento di indici funzionali quali la filtrazione glomerulare, il flusso plasmatico renale, la capacità polmonare e la velocità di conduzione nervosa, l'indice cardiaco, il metabolismo basale, il flusso massimo respiratorio, e a una lenta ma significativa riduzione della massa muscolare (sarcopenia) e ossea

Adattamenti cardio/respiratori

Adattamenti metabolici

Adattamenti muscolari

Cambiamenti strutturali

Patologie concomitanti

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L'invecchiamento (aging) comporta un inevitabile declino della funzionalità muscolare, associato ad un fenomeno di **sarcopenia**.

In mancanza di uno stimolo allenante adeguato i livelli di forza e potenza muscolare declinano a partire dalla terza decade di vita, con un calo particolarmente significativo a ridosso dei 40 anni di età.

In assenza di stimoli allenanti adeguati, la massa muscolare tende a diminuire in maniera fisiologica di un tasso del 3-8% per decade di vita dopo i 30 anni.

La capacità aerobica (VO2Max) tende ugualmente a diminuire, fino al 10% per decade dopo i 30 anni di vita. Nel complesso dunque il potenziale motorio di un atleta va in declino dopo una fase di picco che normalmente si manifesta tra i 26 ed i 31 anni per atleti maschi, 23 e 29 anni per atlete femmine.

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GIORNALE ITALIANO DI CARDIOLOGIA

Ottobre 2010, Vol. 11, *Suppl. 1 al N. 10*

G Ital Cardiol 2010;11(10 Suppl. 1):114S-117S

Variazioni cardiache anatomo funzionali

-Progressivo lieve incremento dello spessore parietale assoluto e relativo ed una tendenza alla riduzione volumetrica del ventricolo sinistro ("rimodellamento concentrico"),

- aumentata rigidità arteriosa (riduzione tessuto elastico e degenerazione collagene

- adattamento cardiaco all'età differente nei due sessi, con tendenza a una lieve ipertrofia concentrica nelle donne e a un rimodellamento concentrico negli uomini

GIORNALE ITALIANO DI CARDIOLOGIA

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Variazioni cardiache anatomo funzionali

Steno/insufficienza valvolare

Modificazioni biochimico-strutturali

Involuzione del tessuto di conduzione

Variazioni muscolari con l'età

Calo nel tasso di sintesi proteica facilitato da un declino a livello ormonale.

Aumento nella resistenza insulina, con conseguenze a livello di turnover proteico e catabolismo muscolare.
Inefficiente estrazione di ossigeno a livello tissutale, con conseguente rallentamento metabolico.

Questi fattori, parzialmente riconducibili ad un cambiamento nel profilo ormonale di un atleta, sembrano affliggere molto più l'uomo che la donna la quale ne risente molto meno del fisiologico calo di estrogeni post-menopausa di quanto non risenta un uomo del fisiologico calo di testosterone (andropausa).

Tanto l'uomo quanto la donna soffrono in egual misura di un calo di attività a livello dell'asse ormone della crescita-IGF1, il che compromette la capacità di rigenerare tessuto muscolare ad un tasso adeguato ai livelli di degradazione tissutale associati all'attività fisica.

Possibile identificare un cambiamento livello funzionale per quanto riguarda il sistema neuromuscolare: alterazione del profilo funzionale di muscoli fasici che vede una progressiva degenerazione delle fibre a scossa rapida (IIa-IIx) ed un incremento di fibre di tipo I.

Questo fenomeno viene spesso indicato in letteratura come aging motor unit pool o gruppo di unità motorie denervate per via del processo di invecchiamento.

L'esercizio fisico, tuttavia, ha la possibilità di rallentare il fisiologico processo di declino a livello della funzionalità muscolare e metabolico, preservando un livello di prestazione fisica adeguato anche nella fascia di età superiore i 40 anni.
Un atleta master è quindi fisiologicamente diverso da un soggetto della stessa età che non abbia intrapreso un programmi di allenamento adeguato

Table 1

Comparison of 1896 Olympic winning times in running events and current Masters records that surpass those winning times (from ESPN and World Masters Records)

Running events	1896 Olympic winning time (from the first Olympic games in Athens)	Current age-group records that surpass 1896 Olympic times and age at which these records were achieved
100 m (s)	12.0	11.7 (61 years)
200 m (s)	22.2	22.1 (46 years)
400 m (s)	54.2	53.9 (63 years)
800 m (min:s)	2:11.0	2:10.4 (60 years)
1500 m (min:s)	4:33:2	4:27:7 (60 years)
Marathon (h:min:s)	2:58:50	2:54:5 (73 years)

Nutrients. 2021 May; 13(5): 1409.
Published online 2021 Apr 22. doi: 10.3390/nu13051409

Nutrition for Older Athletes: Focus on Sex-Differences

Barbara Strasser,[1,*†] Dominik Pesta,[2,3,4,5,6,7] Jörn Rittweger,[2] Johannes Burtscher,[8] and Martin Burtscher[7]

According to population-based studies, the prevalence of sarcopenia in healthy adults aged 60 years and older is about 10% for men and 10% for women, respectively

Intriguingly, in a cross-sectional study including 156 female and male masters athletes aged between 40–79 years, no individual was categorized as sarcopenic, i.e., below normal levels of muscle mass and muscle strength or performance, according to the definition of the European Working Group for Sarcopenia in Older People

Data from cross-sectional studies indicate that lean muscle mass and muscle strength did not decline with age in individuals aged 40–81 years who trained 4 to 5 times per week

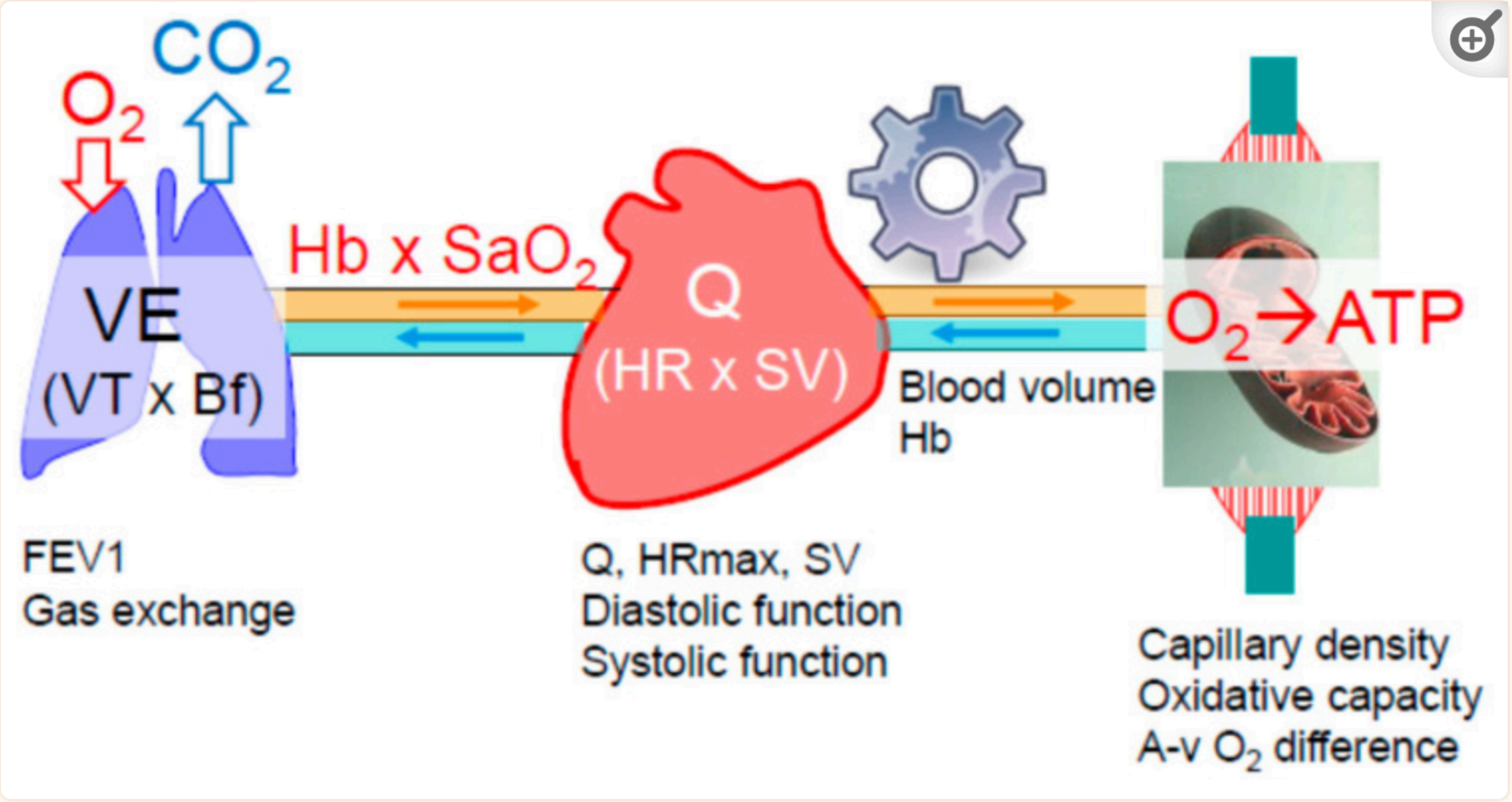

Figure 2

The lung–heart–muscle axis involved in oxygen delivery and utilization. Main cardiorespiratory parameters specifying organ function at rest and during exercise: minute ventilation (VE), cardiac output (Q), and oxygen extraction in the skeletal muscle. Parameters listed below the organs represent those which are primarily affected by the aging process. Oxygen, O_2; carbon dioxide, CO_2; tidal volume, VT; breathing frequency, Bf; hemoglobin concentration, Hb; arterial oxygen saturation, SaO_2; heart rate, HR; adenosine triphosphate, ATP.

VO2max restriction by the pulmonary system depends on the level of fitness (the higher the more likely) and the age-related degree of decrease in respiratory function along with structural changes, i.e., declining respiratory muscle strength and endurance, enhanced rigidity of the chest wall, loss of elastic recoil, reduction of the alveolar surface area and the number of capillaries perfusing the lung [11,15,16]. A low BR, reduced forced expiratory volume in one second (FEV1) and exercise-induced hypoxemia are potential markers for pulmonary limitations of VO2max in the older athlete

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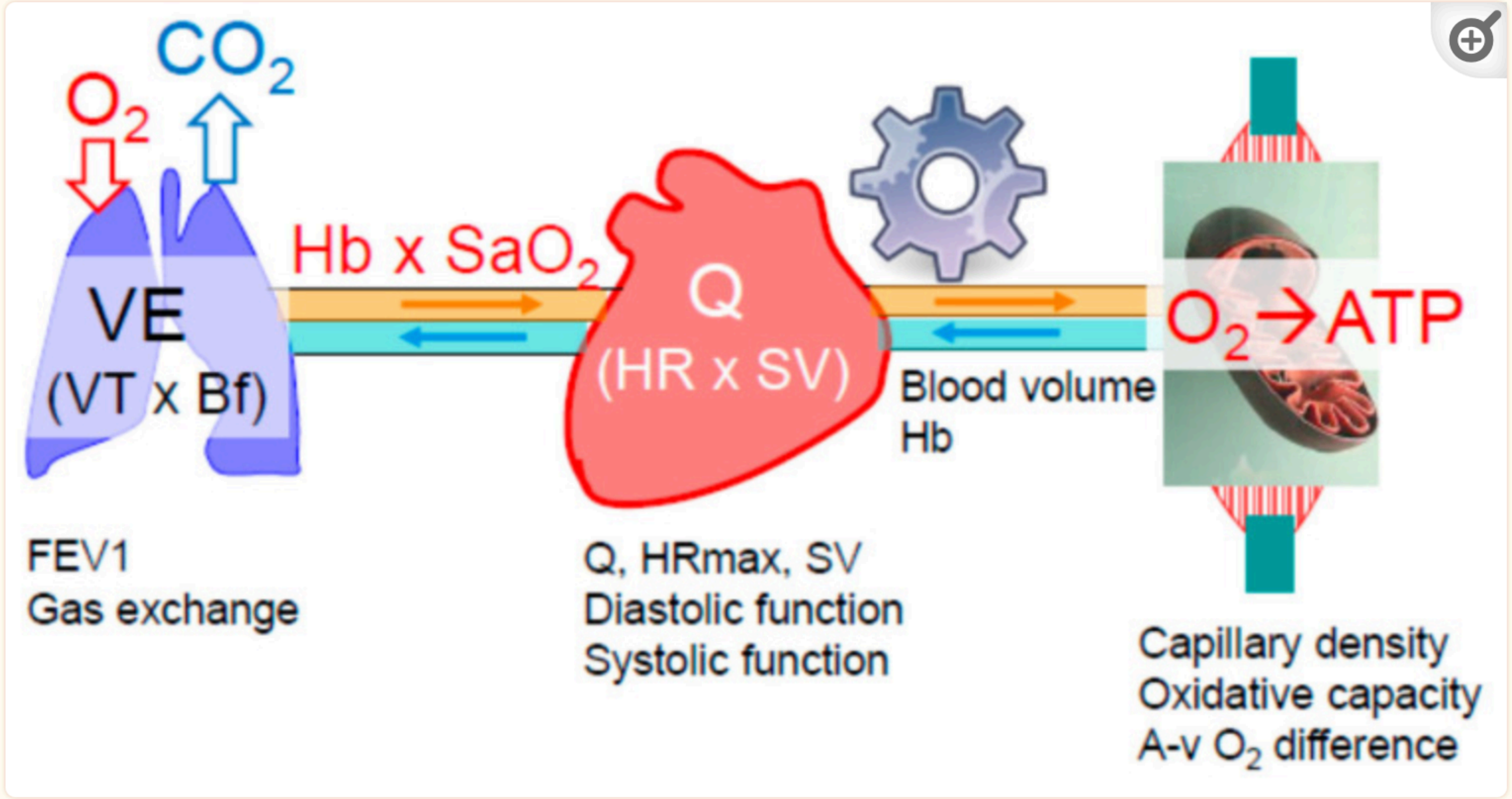

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The age-related decrease in cardiovascular function (particularly decline of Q which is HR x SV) will considerably affect VO2max. HRmax decreases according to the formula (208 (beats per min)—0.7 × age) in healthy sedentary and trained subjects, probably due to the decrease in intrinsic heart rate [20]. In addition, lower SV associated with reduced left ventricular (LV) compliance (diminished diastolic function) was shown in healthy sedentary people but seems to be preserved in masters athletes [21]. Impaired ability to modulate sympathetic vasoconstrictor activity (functional sympatholysis) and a reduced exercise hyperemia are also characteristics of aging. Again, regular PA was shown to offset these impairments

A recent review confirmed, based on current data from masters athletes, that most of the physiological mechanisms determining VO2max (i.e., pulmonary and cardiovascular function, blood oxygen transport capacity, skeletal muscle capillary density and oxidative capacity) are profoundly modulated by regular PA during the entire life-span [9].

In contrast to cardiovascular and skeletal muscle adaptations to PA and exercise training, pulmonary adaptations seem to play a rather minor role for the maintenance of performance in the aging athletes [9,10].

In summary, all these findings indicate that endurance performance (i.e., VO2max) inevitably declines when getting older but can be favorably modified by regular PA and especially by exercise training at all ages in both sexes

Composizione corporea e struttura muscolare

The question arises, which factors may trigger the age-related adipose tissue accumulation. Lifelong training increased the proportion of type I muscle fibres with a concomitant decrease of carbohydrate oxidation independent of intensity level in older athletes compared to younger man [42]. While fat oxidation capacity was similar in both groups, older athletes compensated with a higher exercise metabolic efficiency

In summary, the decline in metabolic rate, along with simultaneous declines and inclines, respectively, in lean mass and fat mass in the elderly can only in part be ascribed to the aging process per se.

Rather, these effects seem confounded by declining levels of PA and inadequate energy intake in this population.

Although body composition of older athletes is considerably better than that of less physically active age-matched individuals, the age-related decline and alterations in body composition and metabolism also takes place in this group and can in part be compensated for by higher exercise metabolic efficiency.

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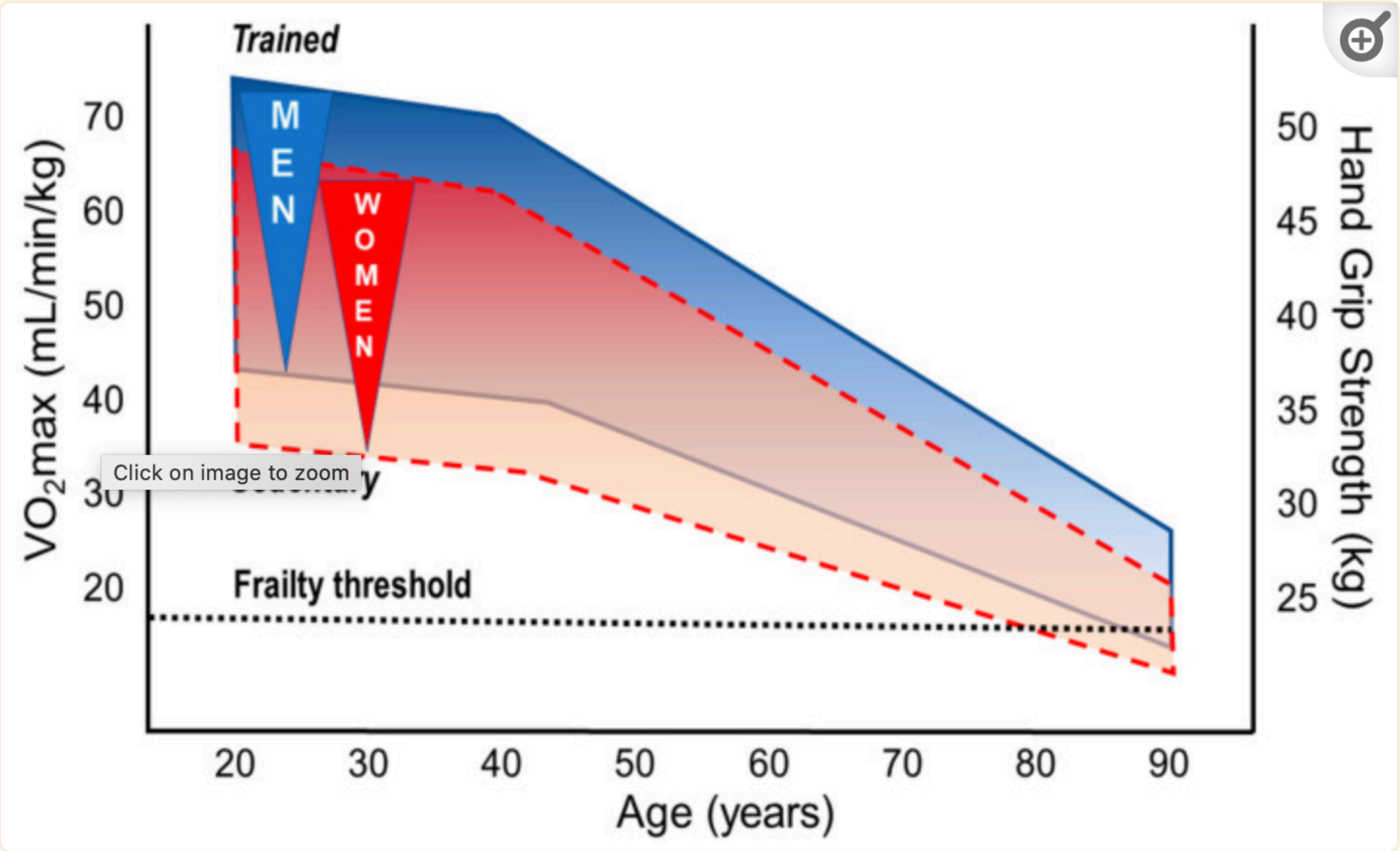

Figure 3

Schematic presentation of the age-related performance decline, i.e., in aerobic capacity (VO_2max) and maximal grip strength, for both sexes, depending on physical training status, ranging from well-trained to sedentary. The figure is based on the meta-analysis from Fitzgerald et al. (1997) [130], with contributions from Burtscher et al. (2008) [28], Booth et al. (2010) [131], Landi et al. (2017) [132], and (defining frailty threshold as VO_2max of 18 mL/min/kg) Carr et al. (2006) [133].

Extraordinarily high aerobic capacity has been reported in lifelong physically active very old individuals of both sexes. For instance, VO2max values of 38 (±1) vs. 21 (±1) mL/min/kg were shown for 81-year-old male endurance athletes (*n* = 9) vs. age-matched healthy untrained persons [136]. Even more impressive, a VO2max of 42.3 mL/min/kg has been recently reported in a 83-year old female masters runner [137]. On top, a 101-yr old athlete improved his VO2max following 2 years of specific training (to 103 years of age) from 31 to 35 mL/min/kg

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The age of males/females when performance had declined to 50% of the maximum performance at 30–35 years were 90/84 years for walking, 87/84 years for swimming, 78/72 years for jumping, and 74/60 years for weight lifting

Regular exercise training of masters athletes can prevent the shrinking of type I muscle fibers [144], and minimize loss of type II fibers, and it may also prevent fiber type grouping [145] following denervation-reinnervation cycles with aging in both sexes.

Notably, females seem to develop a slower muscle fiber phenotype due to progressive slowing of discharge rates [146], likely explaining the relatively large sex difference in the decline of sprint cycling performance

The aging process results in decline of muscle mass and strength by about 1% per year starting in the fourth decade [148].

Peak instantaneous power declines by approximately 7% per decade when assessed via vertical jump test in endurance runners, in sprinters as well as in the general population, and very similar are reported for ergometric lower extremity power testing

Most important characteristics associated with aging are the muscle architecture and fiber type composition, tendon properties and vascular control of the contracting muscle

Accordingly, higher levels of muscle strength and power in the aging athletes are not surprising but seem predominantly due to hypertrophy of remaining fibers as the loss of fiber numbers seems not to be preventable by lifelong PA

While eccentric actions in a hypertrophy-targeted resistance training seem to be slightly more effective than concentric actions, both types of training should be included [157]. Skeletal muscles of males compared to females are generally stronger and more powerful, but muscles of males might be more easily fatigable. While those sex differences are primarily caused by differences in contractile mechanisms, other mechanisms, e.g., muscle perfusion, voluntary activation, etc., also represent contributing factors

Mitochondrial functions, such as for example ATP production, are compromised in advanced age [167].

Accordingly, aging is also associated with oxidative stress and with inflammation [168], as well as with impaired mitochondrial biogenesis.

The latter is at least in part mediated by reduced AMPK activity with increasing age [169], an effect that can be prominently attenuated in the skeletal muscle by exercise

Review Article | Published: 29 January 2021

Can High-Intensity Interval Training Promote Skeletal Muscle Anabolism?

Marcus J. Callahan, Evelyn B. Parr, John A. Hawley & Donny M. Camera

High-intensity interval training (HIIT) comprises short bouts of exercise at or above the power output/speed that elicits individual maximal aerobic capacity, placing high tensile stress on skeletal muscle, and somewhat resembling the demands of resistance exercise.

While HIIT induces rapid increases in skeletal muscle oxidative capacity, the anabolic potential of HIIT for promoting concurrent gains in muscle mass and cardiorespiratory fitness has received less scientific inquiry.

In this review, we discuss studies that have determined muscle growth responses after HIIT, with a focus on molecular responses, that provide a rationale for HIIT to be implemented among populations who are susceptible to muscle loss (e.g. middle-aged or older adults) and/or in clinical settings (e.g. pre- or post-surgery)

Randomized Controlled Trial > Med Sci Sports Exerc. 2021 Oct 1;53(10):2023-2036.
doi: 10.1249/MSS.0000000000002684.

Skeletal Muscle Adaptive Responses to Different Types of Short-Term Exercise Training and Detraining in Middle-Age Men

Marcus J Callahan [1], Evelyn B Parr [1], Tim Snijders [2], Miguel S Conceição [3], Bridget E Radford [1]
Ryan G Timmins, Brooke L Devlin [4], John A Hawley [1], Donny M Camera [5]

Conclusions:
Six weeks of HIIT induced widespread adaptations before detraining in middle-age men. Exercise training-induced increases in aerobic capacity declined during 2.5 wk of detraining, but gains in lean mass and muscle strength were maintained

Randomized Controlled Trial > Exp Gerontol. 2021 Jul 1;149:111321.
doi: 10.1016/j.exger.2021.111321. Epub 2021 Mar 20.

Effects of high-intensity interval training combined with traditional strength or power training on functionality and physical fitness in healthy older men: A randomized controlled trial

Diana Carolina Müller [1], Francesco Pinto Boeno [1], Mikel Izquierdo [2], Per Aagaard [3], Juliana Lopes Teodoro [1], Rafael Grazioli [1], Giovani Cunha [1], Rodrigo Ferrari [4], Mikel L Saez de Asteasu [5], Ronei Silveira Pinto [1], Eduardo Lusa Cadore [6]

Concurrent training (CT) is an efficient strategy to improve neuromuscular function and cardiorespiratory fitness in older adults, which are factors of pivotal importance for the maintenance of functional capacity with aging. However, there is a lack of evidence about the effectiveness of power training (PT) as an alternative to traditional strength training (TST) during CT. Thus, the aim of the present study was to examine the effect of 16 weeks (twice weekly) TST combined with high intensity interval training (TST + HIIT) vs. PT combined with HIIT (PT + HIIT) on functional performance, cardiorespiratory fitness and body composition in older men.

The groups improved similarly ($P < 0.05$) with training in all functional capacity outcomes, W_{max}, cycling economy, VO_{2peak} and body composition ($P < 0.05$). These findings suggest that HIIT based CT programs involving TST vs. PT are equally effective in improving functionality, cardiorespiratory fitness and body composition in healthy older men.

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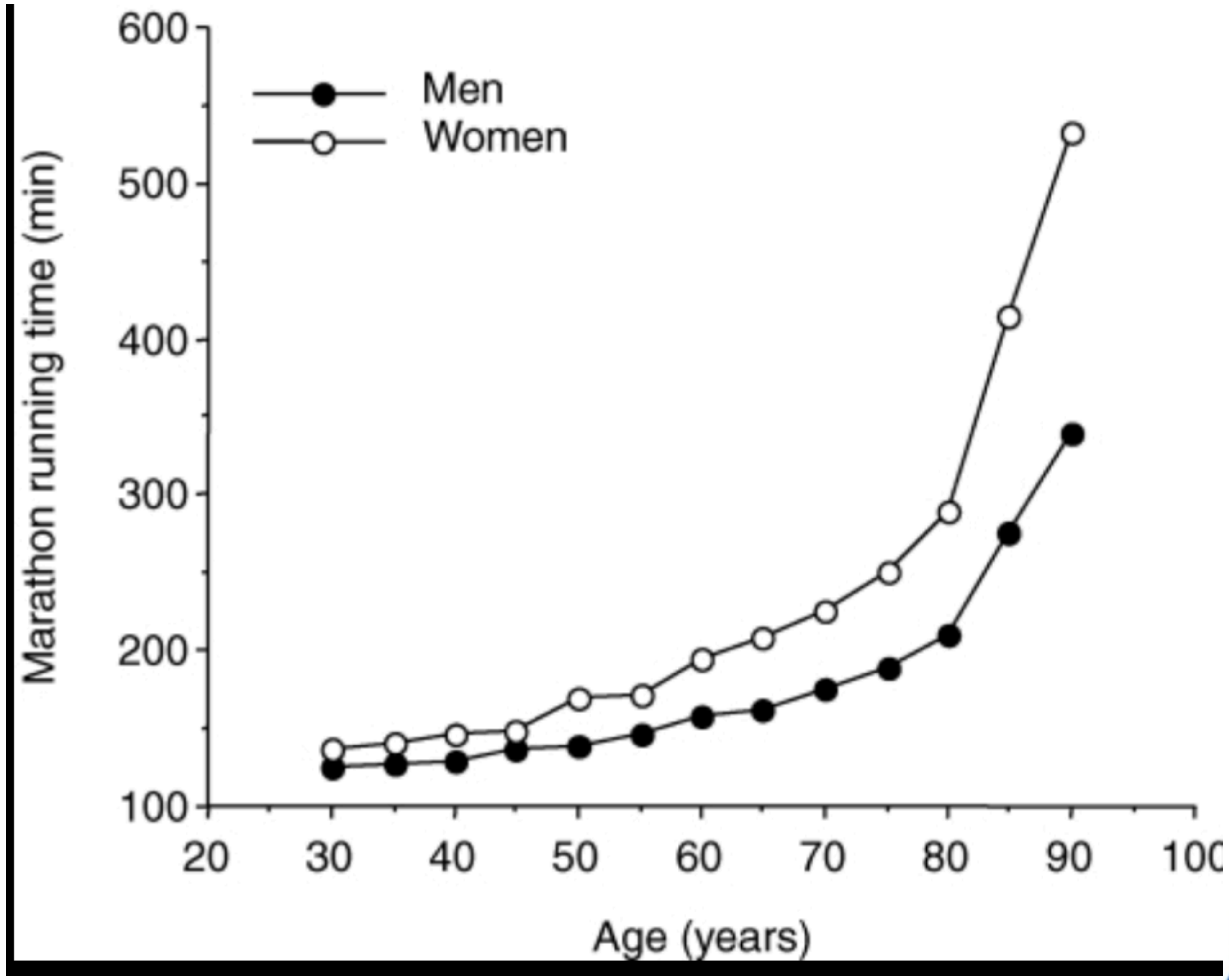
High-intensity interval training (HIIT) can effectively increase peak oxygen consumption, body composition, physical fitness, and health-related characteristics of adults; however, its impact in the older population remains highly debated. This review and meta-analysis aimed to evaluate the effects of high-intensity interval training on cardiorespiratory fitness, body composition, physical fitness, and health-related outcomes in older adults.

HIIT significantly improved the maximum rate of oxygen consumption (VO2peak) as compared to a moderate intensity continuous training (MICT) protocol (HIIT vs. MICT: weighted mean difference = 1.74, 95% confidenc interval: 0.80-2.69, $p < 0.001$). Additional subgroup analyses determined that training periods >12 weeks, training frequencies of 2 sessions/week, session lengths of 40 min, 6 sets and repetitions, training times per repetition of >60 s and rest times of <90 s were more effective for VO2peak. This systematic review and meta-analysis showed that HIIT induces favorable adaptions in cardiorespiratory fitness, physical fitness, muscle power, cardiac contractile function mitochondrial citrate synthase activity, and reduced blood triglyceride and glucose levels in older individuals, which may help to maintain aerobic fitness and slow down the process of sarcopenia.

Review > J Physiol. 2008 Jan 1;586(1):55-63. doi: 10.1113/jphysiol.2007.141879.
Epub 2007 Aug 23.

Endurance exercise performance in Masters athletes: age-associated changes and underlying physiological mechanisms

In summary, Masters endurance athletes are capable of remarkable athletic and physiological functional performance, thereby representing a uniquely positive example of 'exceptional ageing'. Nevertheless, endurance exercise performance decreases during middle-age and declines at an even more rapid rate in older age.



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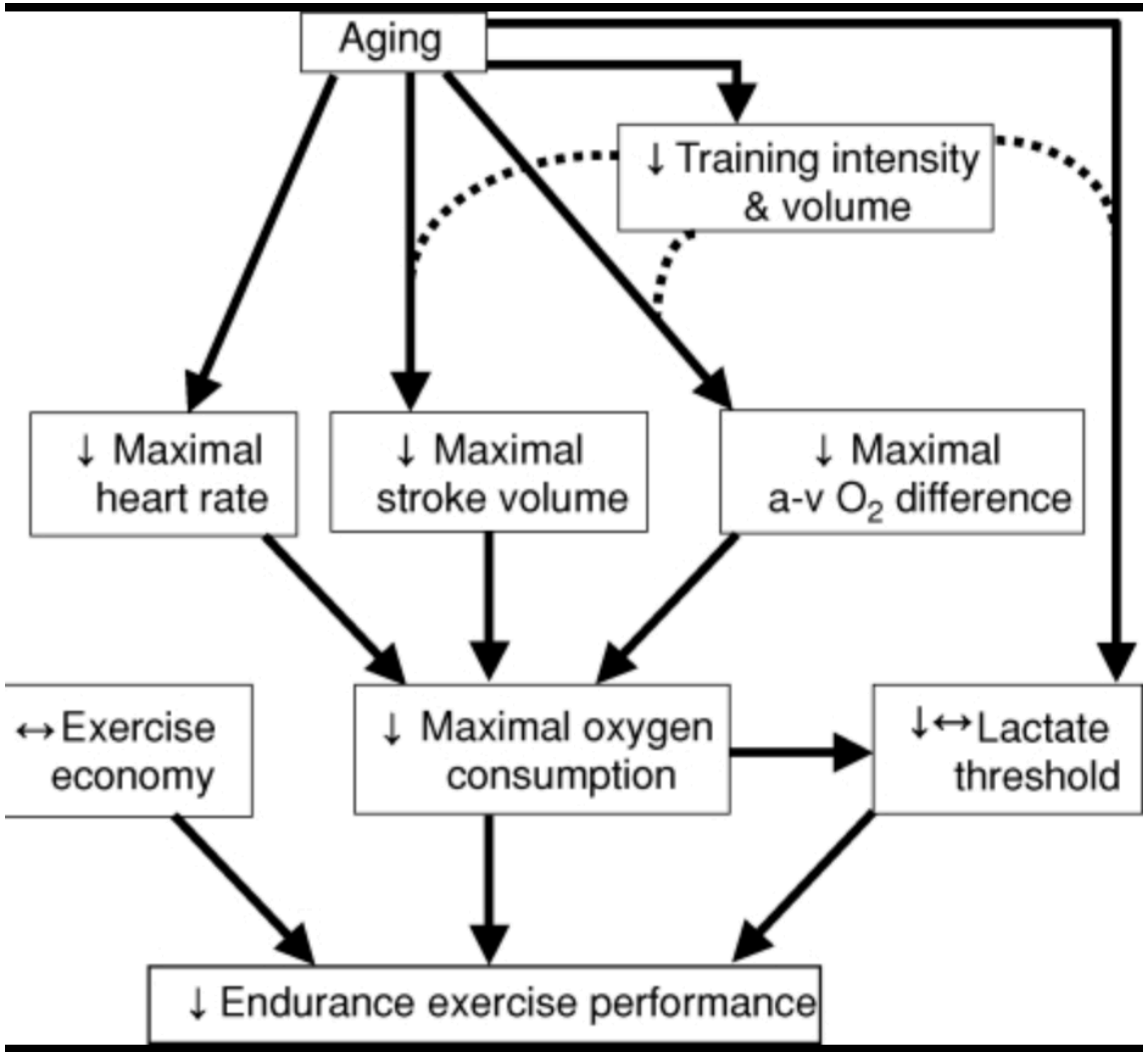
The available data indicate that decreases in VO2max are the most clear and consistent contributor to these declines in performance. Reductions in the lactate threshold also may contribute, whereas submaximal exercise economy is preserved with ageing in endurance athletes.

The age-associated decreases in VO2max in endurance exercise-trained adults are mediated by reductions in maximal cardiac output and maximal arterio-venous O2 difference, with reductions in both maximal stroke volume and heart rate contributing to the former.

The decreases in endurance exercise performance and VO2max with ageing in endurance exercise-trained athletes are associated most closely with reductions in exercise training intensity and volume, probably as a consequence of changes in a number of physical and behavioural factors (e.g. increased prevalence of injuries, and reductions in energy, time and motivation to train)


Aging
↓ Training intensity & volume
↓ Maximal heart rate
↓ Maximal stroke volume
↓ Maximal a-v O_2 difference
↔ Exercise economy
↓ Maximal oxygen consumption
↓↔ Lactate threshold
↓ Endurance exercise performance

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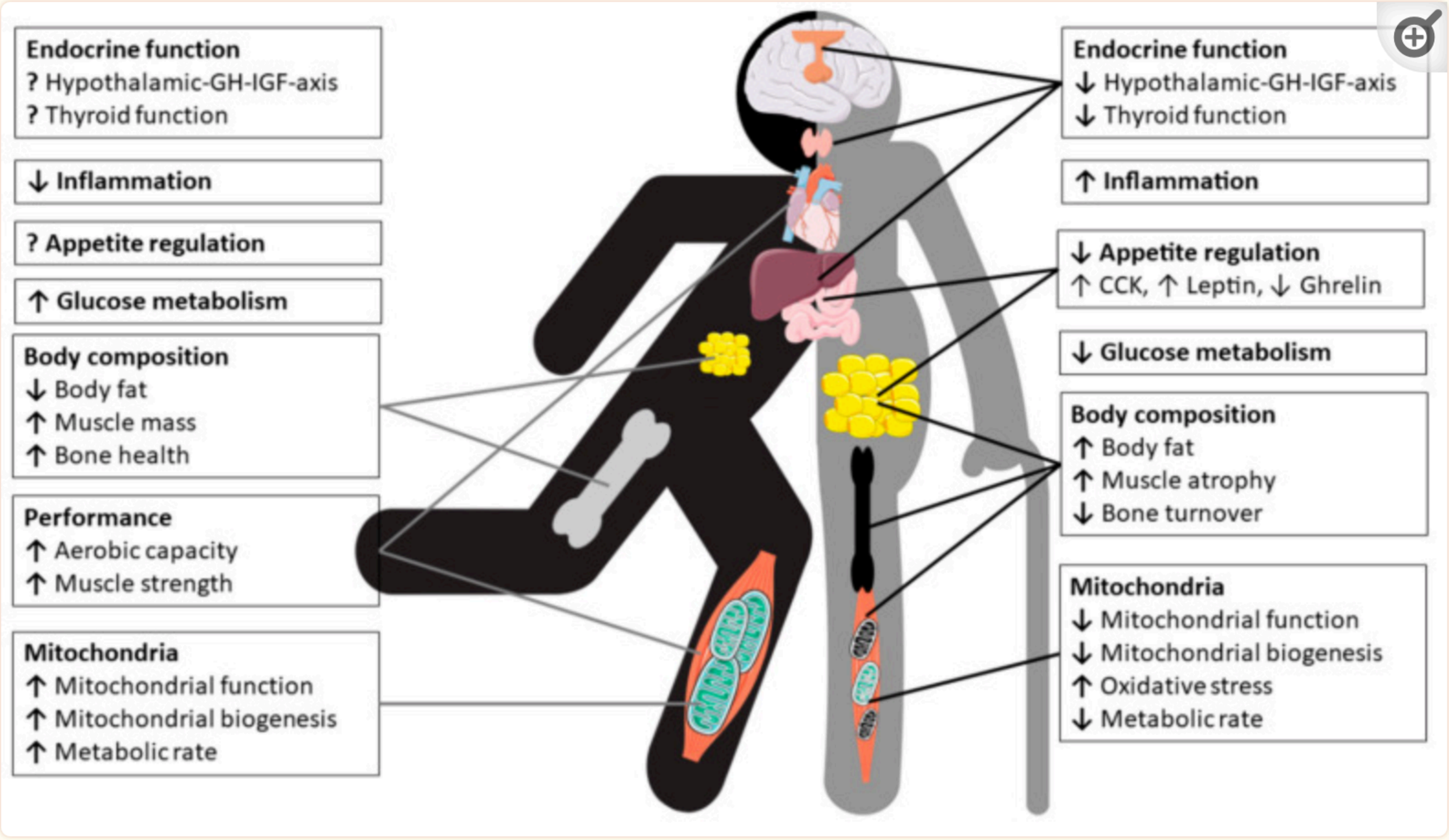

Figure 5

Pleiotropic effects of physical exercise and nutrition on healthy aging. The right side depicts effects of aging on endocrine function, appetite regulation, glucose metabolism, body composition and mitochondria. The left side illustrates how physical exercise can potentially mitigate effects of aging on the human body of masters athletes. ↑, positive change; ↓, negative change; ?, unknown effect. Cholecystokinine, CCK; growth hormone, GH; insulin-like growth factor, IGF.

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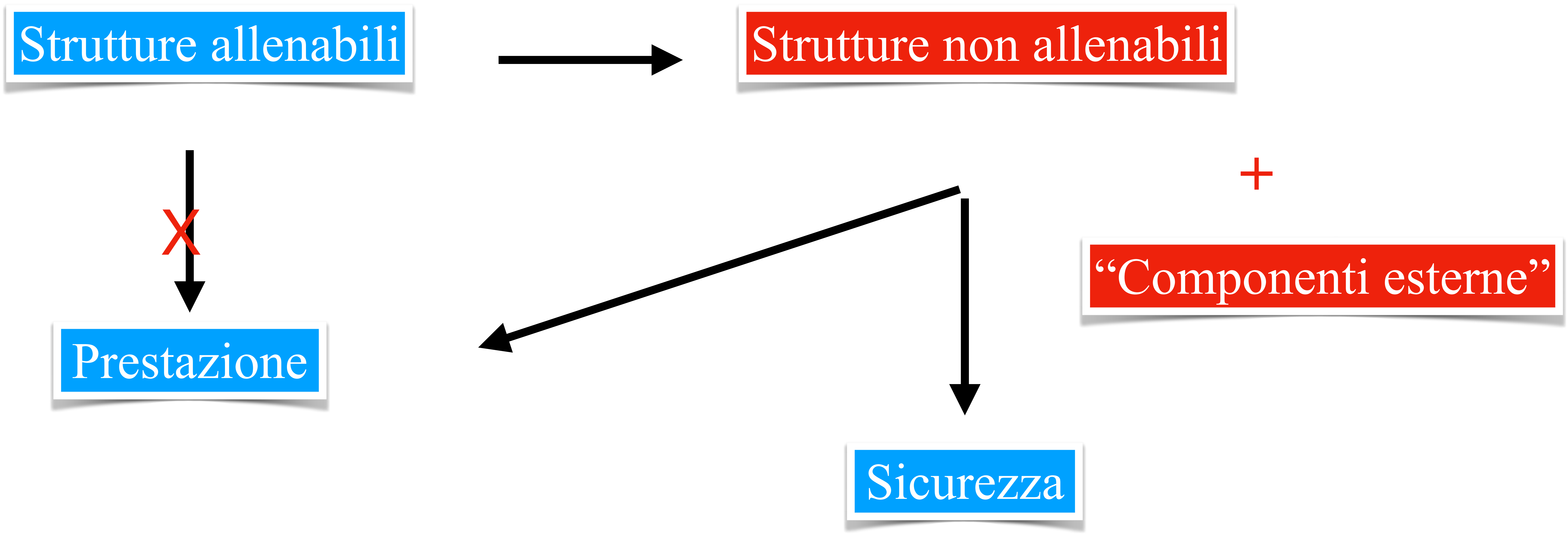

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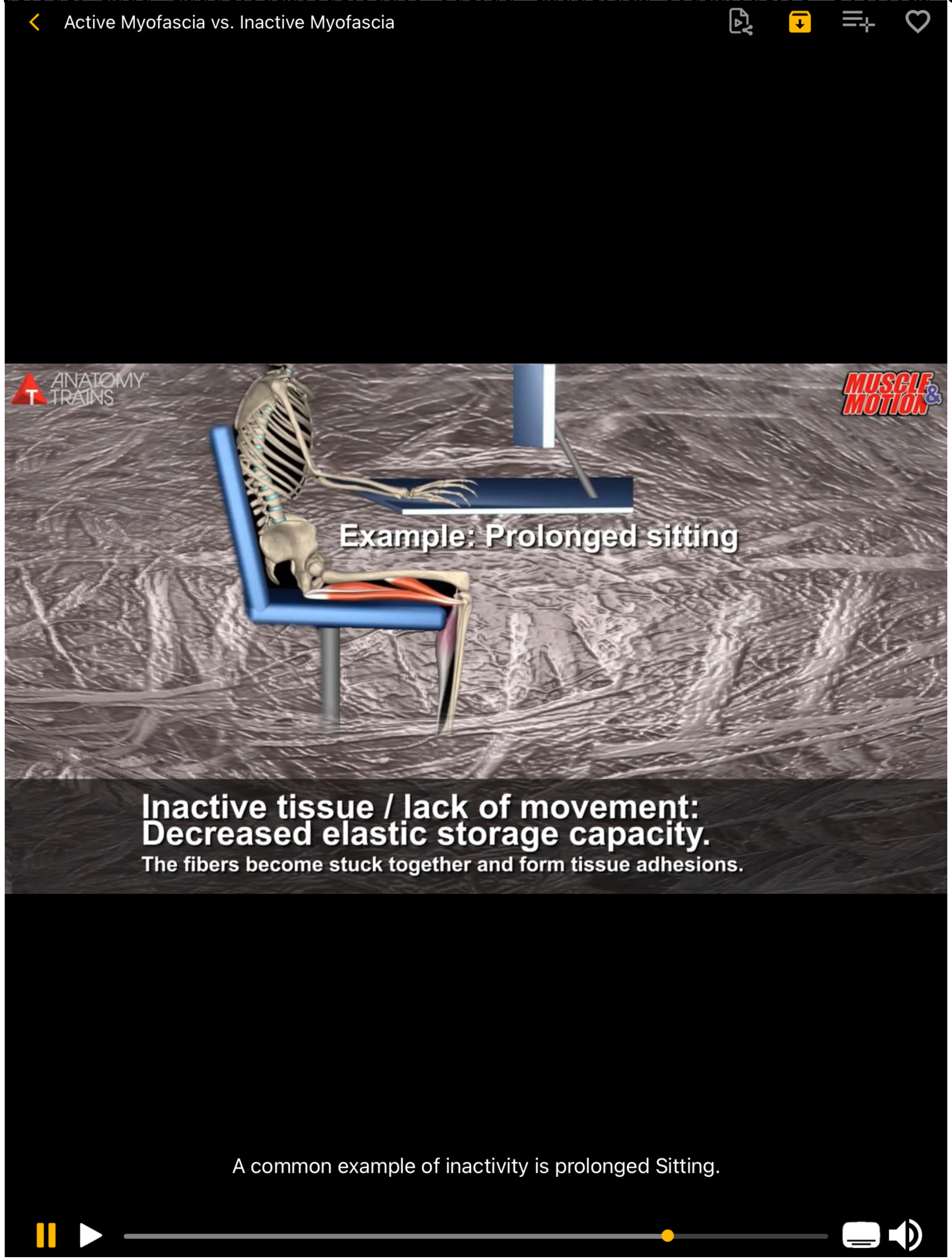

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Nutrizione ed adattamenti all'allenamento

↓

Apporto proteico | *Infiammazione* | *Assorbimento…..*

↓

Catabolismo muscolare

Infiammazione

Supplementazione ω3 vs ω6 (umani)

- Aumenta il livelli di EPA/DHA
- Può ridurre lo stato infiammatorio a riposo e post-ex
- Stimola la sintesi proteica indotta da un pasto aminoacidico
- Favorisce l'aumento della forza e capacità muscolare in anziani
- Efficace nel trattamento di tendiniti croniche

Omega 3/Omega 6 Ratio

La supplementazione con Omega 3 può essere utile

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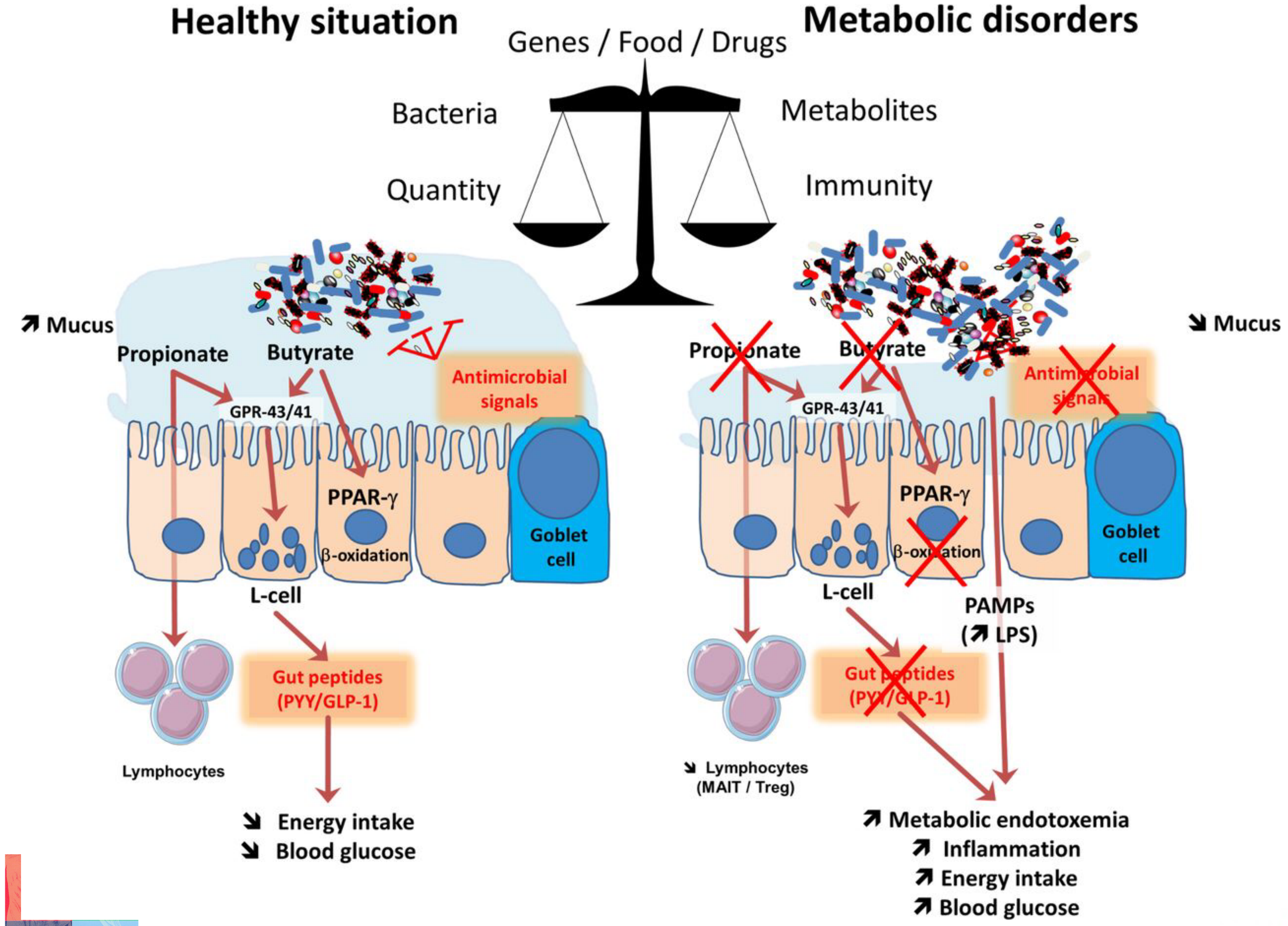
The GI microbiota is also crucial to the *de novo* synthesis of essential vitamins which the host is incapable of producing [149]. Lactic acid bacteria are key organisms in the production of vitamin B12, which cannot be synthesised by either animals, plants or fungi [149,150]. *Bifidobacteria* are main producers of folate



REVIEW Open Access

International Society of Sports Nutrition position stand: safety and efficacy of creatine supplementation in exercise, sport, and medicine

Richard B. Kreider[1*], Douglas S. Kalman[2], Jose Antonio[3], Tim N. Ziegenfuss[4], Robert Wildman[5], Rick Collins[6], Darren G. Candow[7], Susan M. Kleiner[8], Anthony L. Almada[9] and Hector L. Lopez[4,10]

Collectively, these findings suggest that creatine supplementation lessened the amount of muscle atrophy and detrimental effects on muscle associated with immobilization while promoting greater gains in strength during rehabilitation.

While not all studies show benefit, there is evidence that creatine supplementation may help lessen muscle atrophy following immobilization and promote recovery during exercise-related rehabilitation in some populations. Thus, creatine supplementation may help athletes and individuals with clinical conditions recover from injuries.

Individuals with a low initial muscle creatine content, like vegetarians, respond better to creatine supplementation than others with a high natural muscle creatine content.

Review > Nutrients. 2020 Feb 23;12(2):579. doi: 10.3390/nu12020579.

Role of Vitamin D in Athletes and Their Performance: Current Concepts and New Trends

Mirian de la Puente Yagüe [1], Luis Collado Yurrita [2], Maria J Ciudad Cabañas [2], Marioa A Cuadrado Cenzual [2]

Vitamin D has been shown to be a powerful modulator of skeletal muscle physiology [57,58,59,60]. Vitamin D influences it by activating the expression of genes that influence muscle growth and differentiation, particularly in fast-twitch fibers (type II) [60,61,62]. In addition, enlarged interfibrillar spaces and infiltration of fat, fibrosis and glycogen in muscular dystrophies are shown in muscle biopsies of individuals with VITD deficiency.

The VDR exerts its effects in two pathways:

- The first, the genomic pathway (slow or nuclear), through which the transcription and translation of the target genes are modified. This finding suggests that vitamin D promotes muscle cell proliferation and differentiation
- The second mechanism is the non-transcriptional signaling pathway associated with the membrane (rapid, non-genomic or membrane), in which the receptor for 1.25-OHVITD is located. It has been shown that this mechanism enhances the interaction between myosin and actin in the sarcomere, making the force of muscle contraction stronger

Risposte fisiologiche all'allenamento nell'atleta master. Cosa cambia con l'età

Bone Health

- Loss of bone mass
- Increased risk of fatigue fracture
- Osteoarthritis

EA >30 to ideal 45 kcal/kg LBM/d

Calcium + Vitamin D supplementation (25(OH)D >50nmol/L)

Fermented dairy products

Skeletal Muscle

- Loss of muscle mass, strength and power
- Anabolic resistance
- More fatigable

Protein: ≥30g/meal (≥1.2g/kg BM/d)

Leucine: ~3g/meal

Omega-3 PUFA: 3g/d

Whole protein foods (food-first approach)

Immune System

- Immune aging
- Inflammation
- Increased risk of infections (URI)

CHO: ≥8g/kg BM/d 30-70g/h exercise

Vitamin D: 1,000 IU (winter months)

Probiotics: 10^{10} CFU (3 weeks prior travel and competion)

Gut Microbiota

- GI disturbances
- Gut dysbiosis
- Mood disorders (gut-brain axis)

CHO + dietary fibers

Protein (plant-based) and supplementation (whey) combined with exercise

Mediterranean-style diet (↑microbiota diversity + stability)

"La realtà è fatta di circonferenze, ma noi vediamo soltanto linee rette"

Leonardo da Vinci

"Quando il saggio indica la luna lo stolto guarda il dito"

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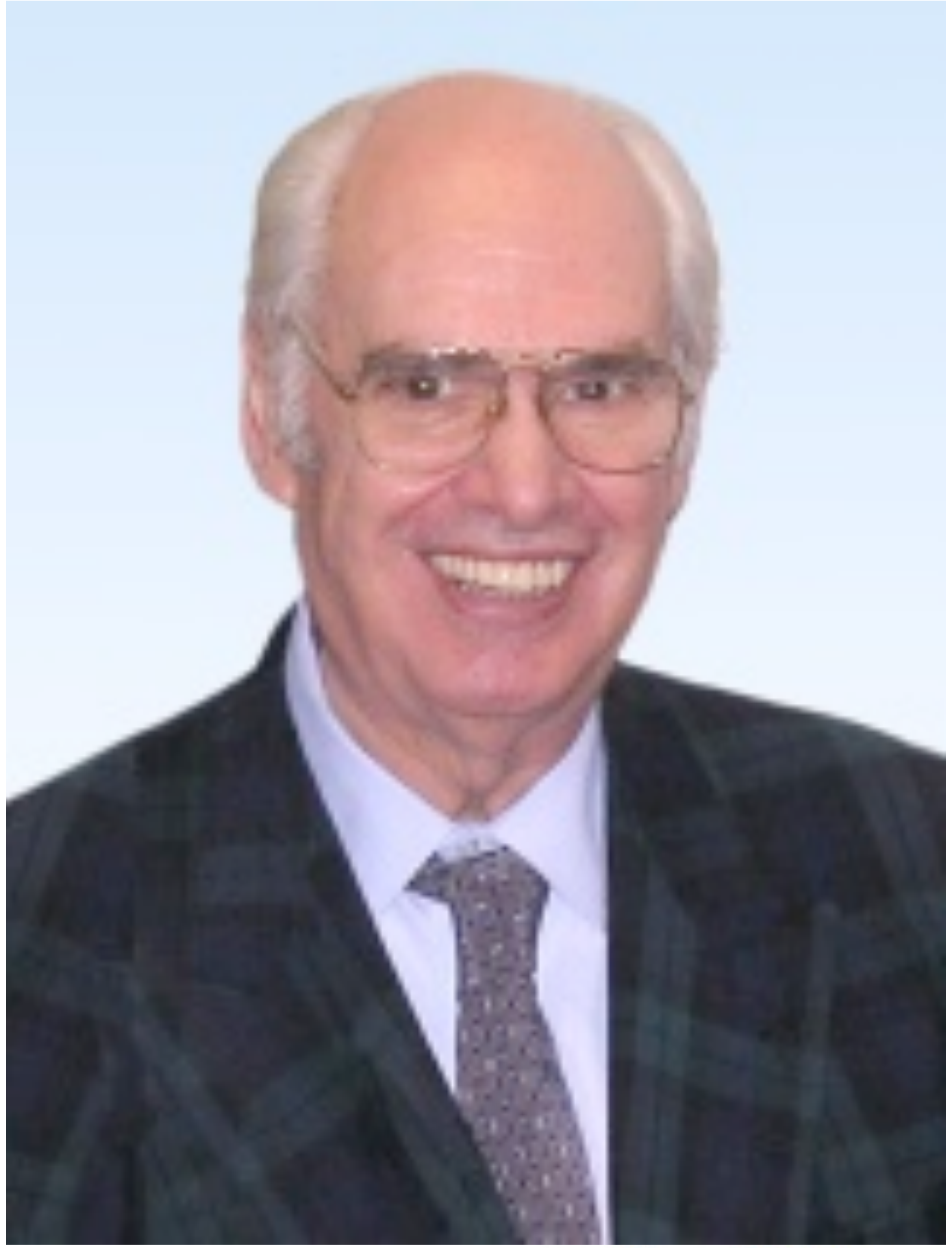

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