Exercise-induced low-grade inflammation in bodybuilding/fitness: adaptation vs. chronic risk

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Abstract. Exercise in bodybuilding and fitness induces a complex, transient inflammatory and redox response that is essential for skeletal muscle adaptation. Acute resistance training elevates pro-inflammatory cytokines such as IL-6, IL-8, and TNF-α, along with reactive oxygen and nitrogen species (ROS/RNS), which act as key signaling mediators for hypertrophy, mitochondrial biogenesis, and metabolic remodeling. In parallel, repeated training promotes an anti-inflammatory milieu characterized by increased IL-10 and IL-1 receptor antagonist, contributing to improved systemic immune and metabolic profiles. However, when recovery is insufficient or in the presence of metabolic dysregulation such as obesity, these same pathways may shift toward chronic low-grade inflammation (LGCI), impairing muscle anabolism and metabolic health. A central controversy in sports physiology is the hormetic role of ROS: while physiological ROS signaling is necessary for adaptation, excessive scavenging via high-dose antioxidant supplementation (e.g., vitamins C and E) may blunt training-induced adaptations. Evidence from randomized controlled trials and meta-analyses suggests that such supplementation does not enhance performance and may attenuate hypertrophy. Moreover, adiposity-driven inflammation exacerbates insulin resistance and muscle catabolism, but regular exercise and dietary interventions can partially reverse this inflammatory state. Overall, optimal adaptation in bodybuilding depends on maintaining a balance between transient exercise-induced inflammatory signaling and the avoidance of chronic systemic inflammation through appropriate training load, recovery, and nutritional strategies.

Key Words: low-grade inflammation, exercise-induced inflammation, cytokines, IL-6, TNF-α, reactive oxygen species, oxidative stress, antioxidant supplementation, vitamin C, vitamin E, muscle hypertrophy, metabolic adaptation, hormesis, obesity, skeletal muscle remodeling.

Introduction. Exercise in bodybuilding/fitness triggers transient inflammatory and oxidative responses that are central to muscle adaptation (Petrescu-Mag 2023, Petrescu-Mag et al 2025). In contrast, obesity-related metabolic syndrome is characterized by persistent low grade inflammation that impairs muscle and metabolic health. The key controversy is how to exploit acute, "physiological" inflammation and reactive oxygen species (ROS) while avoiding chronic, disease-like inflammation, and how supplements, adiposity, and diet shift this balance.

Purpose of this mini-review. The purpose of this mini-review is to critically examine the dual role of exercise-induced inflammation and oxidative stress in bodybuilding and fitness, distinguishing between their beneficial acute signaling functions and their potential contribution to chronic low-grade inflammatory pathology. Specifically, it aims to (i) clarify the mechanistic basis of cytokine and ROS signaling during resistance training, (ii) evaluate how these processes contribute to muscle adaptation versus maladaptation, (iii) analyze the impact of exogenous antioxidant supplementation on training outcomes, and (iv) integrate the modulatory roles of adiposity and diet in shaping inflammatory responses. By synthesizing current evidence, the review seeks to provide a physiologically grounded framework for optimizing training adaptation while minimizing chronic inflammatory risk.

Acute vs. chronic inflammation in training adaptation. Acute resistance or muscle strengthening exercise increases intramuscular IL 6, IL 8, TNF α and other cytokines, along with ROS/RNS production (Della Gatta et al 2014; Pillon et al 2022; Sellami et al 2021; Bouvière et al 2021; Powers et al 2009; Zhou et al 2024). These responses act as signals for muscle remodeling, hypertrophy, mitochondrial biogenesis, angiogenesis, and metabolic adaptation rather than simply damage (Merry & Ristow 2016; Powers et al 2024; Powers et al 2009; Bouvière et al 2021; Zhou et al 2024). Acute bouts also induce anti inflammatory mediators such as IL 10 and IL 1 receptor antagonist, especially when repeated as chronic training, shifting the resting profile toward lower TNF α, IL 6 and higher IL 10 in various populations (Sellami et al 2021; Burini et al 2020; Della Guardia & Codella 2021). IL 6 exemplifies this duality: muscle derived IL 6 acutely supports energy homeostasis and anti inflammatory effects via "classical" signaling during exercise, whereas chronically elevated IL 6 via "trans signaling" is linked to inflammatory disease and obesity (Nash et al 2022; Burini et al 2020; Della Guardia & Codella 2021). Thus, in well recovered training, inflammation is pulsatile and resolving, supporting adaptation; with inadequate recovery or dysmetabolism, the same mediators can contribute to low grade chronic inflammation (Table 1, Figure 1).

Table 1

Comparison of acute vs chronic inflammatory patterns with exercise and obesity

Context	*Cytokine/ROS pattern*	*Functional consequence*	*References*
Single intense session	Sharp rise in IL-6, IL-8, TNF-α; increased ROS/RNS; later rise in IL-10/IL-1ra	Signals hypertrophy, mitochondrial and metabolic adaptation; transient tissue stress	Pillon et al 2022; Sellami et al 2021; Bouvière et al 2021; Della Gatta et al 2014; Merry & Ristow 2016; Powers et al 2009; Zhou et al 2024
Regular training, healthy	Lower resting TNF-α, IL-6; higher IL-10; improved immune profile	Anti-inflammatory, cardiometabolic protection, better regeneration	Burini et al 2020; Sellami et al 2021; Della Guardia & Codella 2021; Sumi et al 2019
Obesity/MetS, inactivity	Chronically elevated TNF-α, IL-6, IL-1β, CRP; ROS excess	Insulin resistance, muscle atrophy risk, metabolic syndrome	Burini et al 2020; Gkrinia & Belančić 2025; Sánchez et al 2025; Della Guardia & Codella 2021

ROS/Redox signaling and antioxidant supplementation (vitamin C, E). Contracting muscle generates ROS/RNS from mitochondria, NADPH oxidases and other sources; these are now recognized as essential signaling molecules for skeletal muscle adaptation (Merry & Ristow 2016; Powers et al 2024; Powers et al 2009; Bouvière et al 2021; Zhou et al 2024). Redox sensitive pathways (e.g., AMPK, MAPKs, NF κB, NRF2, PGC 1α) regulate glucose uptake, antioxidant defense, mitochondrial biogenesis, and hypertrophy (Powers et al 2024; Powers et al 2009; Bouvière et al 2021; Zhou et al 2024). Many adaptations to endurance and resistance training are blunted when exercise induced ROS is excessively scavenged by exogenous antioxidants (Merry & Ristow 2016; Powers et al 2024; Bouvière et al 2021; Zhou et al 2024). Human and animal work suggests that high dose vitamin C and/or E can attenuate overload induced hypertrophy and endurance adaptations, even though they may reduce markers of oxidative stress (Merry & Ristow 2016; Kim 2023; Powers et al 2009; Wadley 2013; Bouvière et al 2021; Zhou et al 2024). In a 10 week resistance training RCT in trained men with caloric surplus, combined vitamin C/E supplementation did not improve performance, and

tended to blunt upper body fat free mass gains compared with placebo; strength gains were similar or slightly smaller with vitamins, while placebo showed clearer hypertrophy (Martínez-Ferrán et al 2022). A systematic review/meta analysis of strength training plus vitamins C/E found no benefit for strength and suggested possible attenuation of muscle growth over time (Dutra et al 2020). Narrative and mini reviews converge that long term high dose C/E may impair physiological phenomena required for adaptation (strength, hypertrophy, endurance), and that diets rich in fruits and vegetables are preferable to supplementation for antioxidant support (Merry & Ristow 2016; Kim 2023; Higgins et al 2020; Bouvière et al 2021; Zhou et al 2024). At the same time, excessive ROS (e.g., in chronic inflammation or extreme overload) can be cytotoxic, so an optimal "hormetic" zone of ROS exposure is proposed: physiological exercise induced ROS is beneficial, while chronic high levels (such as in obesity related low grade inflammation) are detrimental (Merry & Ristow 2016; Powers et al 2024; Powers et al 2009; Bouvière et al 2021; Zhou et al 2024).

Adiposity, chronic low-grade inflammation and interaction with training. Obesity and central adiposity are characterized by low grade chronic inflammation (LGCI), with hypertrophic adipocytes and infiltrating immune cells secreting TNF α, IL 6, IL 1β and other adipokines, activating JNK and NF κB pathways and driving insulin resistance and metabolic syndrome (Burini et al 2020; Gkrinia & Belančić 2025; Sánchez et al 2025; Della Guardia & Codella 2021). This chronic inflammatory milieu also affects skeletal muscle: CI promotes muscle atrophy and impairs protein metabolism, partly via inflammatory signaling and Akt/FOXO1 pathways (Sumi et al 2019). In an animal CI model, resistance exercise still activated anabolic signaling (ERK, p70S6K, 4E BP1) and mitigated muscle atrophy, but the anabolic effects were partially attenuated by inflammatory molecules; combining resistance exercise with anti inflammatory nutrients (EPA/DHA and α lactalbumin) better preserved muscle mass (Sumi et al 2019). In dysmetabolic humans, regular exercise reduces circulating TNF α, IL 1β, free fatty acids, MCP 1, resting IL 6, and advanced glycation end products, while muscle derived IL 6 during acute exercise promotes fat loss and increases IL 10 and IL 1ra (Della Guardia & Codella 2021; Burini et al 2020; Sánchez et al 2025). Chronic training improves adipose tissue function (including "browning" of white adipocytes) and gut microbiota composition, reducing endotoxemia and systemic sub inflammation (Burini et al 2020; Sánchez et al 2025; Della Guardia & Codella 2021). Thus, in obese or metabolically compromised individuals, resistance and combined training can gradually shift inflammation from chronic, adipose driven LGCI toward a more adaptive, muscle driven acute profile, although high CI levels can dampen the full anabolic response (Burini et al 2020; Gkrinia & Belančić 2025; Sánchez et al 2025; Sumi et al 2019; Della Guardia & Codella 2021).

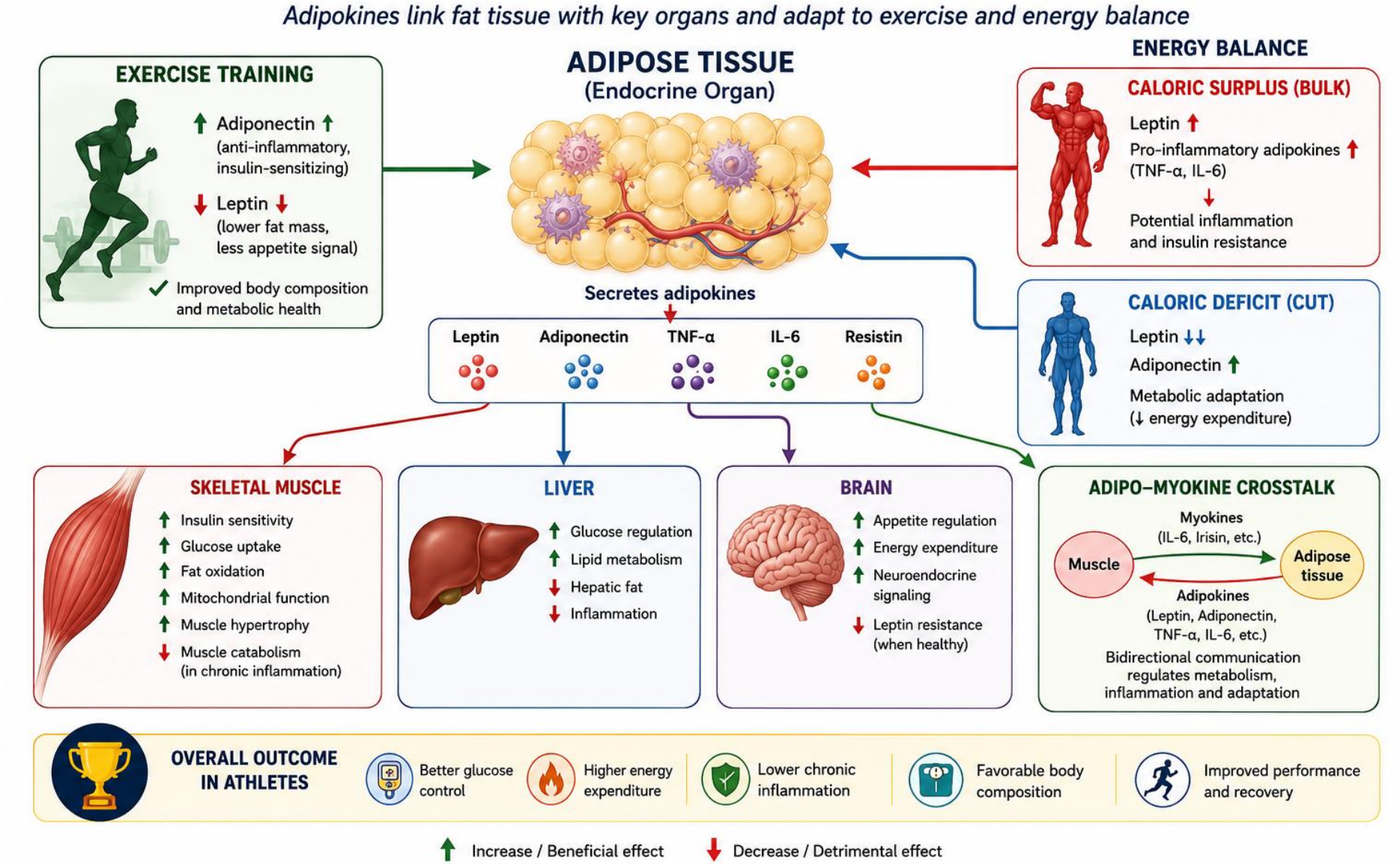

Figure 1. Adipokines link fat tissue with key organs and adapt to exercise and energy balance.

Diet quality, antioxidant intake and inflammatory milieu. Dietary patterns strongly modulate both oxidative stress and LGCI. Obesogenic, high saturated fat, high sugar diets promote adipose expansion, gut dysbiosis, and chronic cytokine production (TNF α, IL 6, IL 1β), maintaining metabolic syndrome and insulin resistance (Burini et al 2020; Gkrinia & Belančić 2025; Sánchez et al 2025; Della Guardia & Codella 2021). In contrast, dietary approaches such as Mediterranean and DASH patterns, rich in unsaturated fats, fiber, and bioactive compounds, reduce oxidative stress markers, improve antioxidant enzyme activity, and decrease inflammatory mediators in MetS (Onu et al 2025; Gkrinia & Belančić 2025; Sánchez et al 2025). Weight loss through caloric control and improved diet quality is a core strategy to reduce obesity related LGCI and restore insulin signaling (Onu et al 2025; Burini et al 2020; Gkrinia & Belančić 2025; Sánchez et al 2025). Reviews on antioxidant supplementation emphasize that, for athletes and active individuals, obtaining antioxidants from fruits and vegetables is more appropriate than high-dose vitamin C/E pills, supporting endogenous redox balance without abolishing ROS mediated signaling necessary for adaptation (Merry & Ristow 2016; Kim 2023; Higgins et al 2020; Bouvière et al 2021; Zhou et al 2024). In obesity management contexts, diets rich in polyunsaturated fatty acids and other anti inflammatory nutrients further modulate adipokine and myokine profiles, complementing exercise in lowering LGCI (Gkrinia & Belančić 2025; Sánchez et al 2025; Sumi et al 2019; Della Guardia & Codella 2021).

Conclusions. Exercise-induced inflammation in bodybuilding and fitness should be understood as a fundamentally adaptive and time-dependent biological process rather than a pathological response. Acute elevations in cytokines and ROS function as essential signals for muscle remodeling, metabolic regulation, and mitochondrial adaptation. When properly regulated through adequate recovery and progressive training, these transient inflammatory events resolve into a net anti-inflammatory systemic profile, contributing to improved health and performance.

In contrast, chronic low-grade inflammation—commonly associated with obesity, poor dietary patterns, and insufficient recovery—represents a distinct pathological state that impairs anabolic signaling, promotes insulin resistance, and accelerates muscle catabolism. Importantly, the boundary between adaptive and maladaptive inflammation is influenced by energy balance, adiposity, and lifestyle factors.

Evidence also indicates that indiscriminate suppression of exercise-induced ROS signaling via high-dose antioxidant supplementation may be counterproductive, as it can blunt hypertrophic and endurance adaptations by interfering with redox-sensitive signaling pathways. Consequently, antioxidant intake should preferentially derive from whole-food sources rather than pharmacological dosing.

Overall, optimal bodybuilding adaptation depends on maintaining a controlled hormetic environment in which transient inflammatory and oxidative signals are preserved for signaling purposes, while chronic systemic inflammation is minimized through structured training, adequate recovery, and anti-inflammatory dietary patterns.

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References

Bouvière J., Fortunato R., Dupuy C., Werneck-De-Castro J., Carvalho D., Louzada R., 2021 Exercise-stimulated ROS sensitive signaling pathways in skeletal muscle. Antioxidants 10(4):537. https://doi.org/10.3390/antiox10040537

Burini R., Anderson E., Durstine J., Carson J., 2020 Inflammation, physical activity, and chronic disease: An evolutionary perspective. Sports Medicine and Health Science 2(1):1-6.

Della Gatta P., Garnham A., Peake J., Cameron-Smith D., 2014 Effect of exercise training on skeletal muscle cytokine expression in the elderly. Brain, behavior, and immunity 39:80-86.

Della Guardia L., Codella R., 2021 Exercise tolls the bell for key mediators of low-grade inflammation in dysmetabolic conditions. Cytokine & Growth Factor Reviews 62:83-93.

Dutra M. T., Martins W. R., Ribeiro A. L. A., Bottaro M., 2020 The effects of strength training combined with vitamin C and E supplementation on skeletal muscle mass and strength: A systematic review and meta-analysis. Journal of Sports Medicine 2020. https://doi.org/10.1155/2020/3505209

Gkrinia E., Belančić A., 2025 The mechanisms of chronic inflammation in obesity and potential therapeutic strategies: A narrative review. Current Issues in Molecular Biology 47(5):357. https://doi.org/10.3390/cimb47050357

Higgins M., Izadi A., Kaviani M., 2020 Antioxidants and exercise performance: With a focus on vitamin E and C supplementation. International Journal of Environmental Research and Public Health 17(22):8452. https://doi.org/10.3390/ijerph17228452

Kim J., 2023 Effect of high-dose vitamin C and E supplementation on muscle recovery and training adaptation: A mini review. Physical Activity and Nutrition 27(2):8-12.

Martínez-Ferrán M., Berlanga L., Barcelo-Guido O., Matos-Duarte M., Vicente-Campos D., Jorge S., Romero-Morales C., Munguía-Izquierdo D., Pareja-Galeano H., 2022 Antioxidant vitamin supplementation on muscle adaptations to resistance training: A double-blind, randomized controlled trial. Nutrition 105:111848. https://doi.org/10.1016/j.nut.2022.111848

Merry T. L., Ristow M., 2016 Do antioxidant supplements interfere with skeletal muscle adaptation to exercise training? The Journal of Physiology 594(18):5135-5147.

Nash D., Hughes M., Butcher L., Aicheler R., Smith P., Cullen T., Webb R., 2022 IL-6 signaling in acute exercise and chronic training: Potential consequences for health and athletic performance. Scandinavian Journal of Medicine & Science in Sports 33(1):4-19.

Onu A., Trofin D., Tutu A., Onu I., Galaction A., Sardaru D., Trofin D., Onita C., Iordan D., Matei D., 2025 Integrative strategies for preventing and managing metabolic syndrome: The impact of exercise and diet on oxidative stress reduction—A review. Life 15(5):757. https://doi.org/10.3390/life15050757

Petrescu-Mag I. V., 2023 Muscle fiber development in humans and other mammals. ABAH Bioflux 15(1):21-23.

Petrescu-Mag I. V., Gavriloaie C., 2025 Reconsidering oxidative stress: from marker of senescence to driver of adaptation. HVM Bioflux 17(1):26-27.

Pillon N. J., Smith J. A. B., Alm P., Chibalin A., Alhusen J., Arner E., Carninci P., Fritz T., Otten J., Olsson T., van Doorslaer de ten Ryen S., Deldicque L., Caidahl K., Wallberg-Henriksson H., Krook A., Zierath J., 2022 Distinctive exercise-induced inflammatory response and exerkine induction in skeletal muscle of people with type 2 diabetes. Science Advances 8:eabo3192. https://doi.org/10.1126/sciadv.abo3192

Powers S. K., Radák Z., Ji L. L., Jackson M., 2024 Reactive oxygen species promote endurance exercise-induced adaptations in skeletal muscles. Journal of Sport and Health Science 13(6):780-792.

Powers S. K., Duarte J., Kavazis A. N., Talbert E. E., 2009 Reactive oxygen species are signalling molecules for skeletal muscle adaptation. Experimental Physiology 95(1):1-9.

Sánchez S. S., Quevedo R. G., Fernandes T., da Fonseca G. W. P., 2025 The role of exercise and nutrition in modulating inflammatory cytokines activity for obesity management. Frontiers in Endocrinology 16. https://doi.org/10.3389/fendo.2025.1618360
Sellami M., Bragazzi N. L., Aboghaba B., Elrayess M. A., 2021 The impact of acute and chronic exercise on immunoglobulins and cytokines in elderly: Insights from a critical review of the literature. Frontiers in Immunology 12:631873. https://doi.org/10.3389/fimmu.2021.631873
Sumi K., Ashida K., Nakazato K., 2019 Resistance exercise with anti-inflammatory foods attenuate skeletal muscle atrophy induced by chronic inflammation. Journal of Applied Physiology 128(1). https://doi.org/10.1152/japplphysiol.00585.2019
Wadley G. D., 2013 A role for reactive oxygen species in the regulation of skeletal muscle hypertrophy. Acta Physiologica 208(1):9-10.
Zhou Y., Zhang X., Baker J. S., Davison G. W., Yan X., 2024 Redox signaling and skeletal muscle adaptation during aerobic exercise. iScience 27. https://doi.org/10.1016/j.isci.2024.109643

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