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The Effect of Polyphenol Supplementation on Recovery Following High-Intensity Interval Training

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ABSTRACT

Polyphenols are a diverse group of plant-derived compounds. As a part of the human diet, they have a broad spectrum of biological activity, including antioxidant, anti-inflammatory, and immunomodulatory properties through multiple metabolic pathways. High-intensity interval training (HIIT) exercise, although widely used to improve physical performance, is associated with

causing substantial metabolic and oxidative stress, inflammation, hormonal imbalance, and muscle damage. Some studies indicate that polyphenol intake may inhibit reactive oxygen species formation, modulate cytokine release, lower lipid peroxidation and damage of deoxyribonucleic acid, and improve markers of muscle recovery, potentially accelerating recovery after HIIT. However, other research contradicts the positive impact of polyphenol supplementation on recovery or performance.

Current data indicate that nutrition is a key factor in the recovery process, and multiple studies suggest that polyphenol-rich diets — emphasizing fruits, vegetables, whole grains, legumes, nuts, cocoa, tea, and berries — may be an effective strategy to combat exercise-induced stress and fatigue.

Keywords: polyphenols, high-intensity interval training, regeneration, antioxidants, oxidative stress, inflammation, muscle damage

Content

1. Introduction

High-intensity interval training (HIIT) is one of the most effective and time-efficient strategies for improving cardiorespiratory fitness and athletic performance across diverse populations, from athletes to clinical rehabilitation patients. Characterized by short bursts of near-maximal effort interspersed with recovery periods, HIIT delivers superior physiological adaptations in less time than traditional endurance training (1,2).

However, HIIT's intensity imposes significant demands on regenerative capacity. Excessive or prolonged disturbances can lead to accumulated fatigue, increased injury risk, and reduced training quality in subsequent sessions (3,4).

Intense contractions drive rapid increases in oxygen consumption and reactive oxygen species (ROS) production, inducing oxidative stress alongside mechanical microtrauma manifesting as exercise-induced muscle damage (EIMD) (5,6). These stressors trigger inflammatory cascades—neutrophil infiltration, cytokine release, and macrophage activation—that persist 24-48 hours post-exercise, during which satellite cells activate for tissue repair via myogenic regulatory factors (7,8).

These findings underscore strategies to limit excessive oxidative damage while supporting homeostasis restoration, particularly in frequent HIIT protocols. Recovery encompasses coordinated processes: redox balance restoration, inflammation resolution, EIMD repair, and stress response normalization. Nutrition serves as a key modulator alongside rest and individual characteristics (9,10).

Polyphenols — abundant in berries, tea, coffee, cocoa—offer promising antioxidant and anti-inflammatory effects, scavenging ROS, modulating inflammation, supporting satellite cell function and mitochondrial biogenesis without fully blunting adaptive signaling (11–14).

This review synthesizes evidence on polyphenol-rich diets and supplementation for post-HIIT recovery, examining: (1) HIIT-induced oxidative/inflammatory/EIMD responses; (2) polyphenol classification, bioavailability, mechanisms; (3) clinical/preclinical recovery, biomarker/performance outcomes; (4) research gaps and recommendations. Integrating pathophysiology with nutritional pharmacology, it guides strategies balancing stress signaling with recovery optimization.

2. Polyphenols - promising natural antioxidants

2.1. Polyphenols: scientific overview

Polyphenols are a structurally diverse class of plant-derived secondary metabolites. They are characterized by multiple phenolic rings and hydroxyl groups. These compounds are widespread in the plant kingdom. Polyphenols play essential roles in plant defense, pigmentation, and growth regulation. In human nutrition, polyphenols have attracted much attention due to their broad spectrum of biological activities. These include antioxidant, anti-inflammatory, immunomodulatory, and metabolic effects. Such activities support the prevention and management of chronic diseases (11–21).

2.2. Definition and chemical structure

Polyphenols are defined by their chemical structure, which includes one or more aromatic rings with two or more hydroxyl groups. This basic structure allows them to interact with biomolecules and influence physiological pathways. Polyphenol diversity results from differences in ring structure, degree of polymerization, and substitution patterns. This variety produces many compounds with distinct biological properties (14–16,18).

2.3. Classification

Polyphenols are grouped into four major types based on chemical structure (15,18).The most common group is flavonoids, divided into flavonols, flavones, flavanones, isoflavones, anthocyanidins, and catechins. Flavonoids are found widely in fruits, vegetables, tea, and wine (15,18,21). Phenolic acids are another group and are split into hydroxybenzoic acids (such as gallic acid) and hydroxycinnamic acids (like caffeic acid). These are found in coffee, grains, and berries (14,15). Stilbenes, including resveratrol, are less common and present in grapes and wine (14,15). Lignans occur in seeds (especially flaxseed), whole grains, and some vegetables (15,18).

Other notable subclasses include tannins and proanthocyanidins, which contribute to the astringency and health benefits of certain foods (11,14,15).

2.4. Metabolism and bioavailability

The bioavailability of polyphenols is a critical determinant of their biological activity. After ingestion, polyphenols undergo extensive metabolism in the gastrointestinal tract and liver, including phase I reactions (oxidation, reduction, hydrolysis) and phase II reactions (conjugation with glucuronic acid, sulfate, or methyl groups). The gut microbiota plays a pivotal role in transforming polyphenols into bioactive metabolites, which can exert systemic effects. Despite their promising activities, the clinical utility of polyphenols is limited by poor oral bioavailability, rapid metabolism, and variable absorption (14,15,19). Strategies such as nano- and liposomal-based delivery systems are being developed to enhance their systemic exposure and therapeutic efficacy (15,19).

2.5. Dietary Sources

Polyphenols are found in many plant foods as shown in Table 1. Their concentration depends on species, ripening, and environment. Polyphenol content varies widely, so regularly eating a range of plants is recommended for maximum benefits (11,14,15,17,19,21). Studies show diets high in polyphenols reduce the risk of chronic diseases, including cardiovascular disease, cancer, diabetes, obesity, and neurodegenerative disorders. These effects involve antioxidant, anti-inflammatory, metabolic, and immunomodulatory actions (11,14,15,17–20).

Rich sources of flavonoids and phenolic acids are fruits, such as berries, apples, grapes, citrus fruits, cherries, and plums. Significant amounts of flavonoids and other polyphenols are also present in vegetables - onions, spinach, broccoli, artichokes, and tomatoes. Whole grains, legumes, and seeds (especially flaxseed) provide lignans and phenolic acids. Major dietary sources of polyphenols include walnuts, almonds, flaxseed, cloves, oregano, thyme, and rosemary. Beverages as tea (green and black), coffee, red wine, and cocoa are also great sources of polyphenols (11,14,15,17–19,21).

2.6. Antioxidant and anti-inflammatory effects of polyphenols

Antioxidants are substances that neutralise or reduce free radicals by donating electrons (22). In the human body, enzymes protect against free radical damage, especially reactive oxygen species. These include superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) (23). However, there is also a group of dietary antioxidants such as polyphenols, vitamin C and E. Both enzymatic systems and non-enzymatic components work together to maintain redox balance (24).

Polyphenols include many compounds that can undergo electron-transfer reactions (22). They directly scavenge reactive oxygen species and free radicals, chelate transition metals to reduce

oxidative reactions, inhibit pro-oxidant enzymes, and increase endogenous antioxidants (11,12,14–17,20,21).

Flavonoids and phenolic acids have antioxidant properties due to the presence of resorcinol and catechol groups. The catechol group transfers two electrons, with the first used in neutralizing free radicals. When the resorcinol group is oxidized, its products can neutralize more molecules than one catechol group (22).

Carotenoids act as antioxidants in two ways. They directly neutralize ROS and lipid radicals. They also stimulate the antioxidant system through redox signaling pathways. During carotenoid metabolism, electrophilic derivatives, such as apocarotenoids, are formed. These activate the nuclear factor erythoid 2 (Nrf2) pathway and cellular antioxidant mechanisms. This helps the cell fight oxidative stress (25).

In addition to their antioxidant properties, polyphenols also modulate key inflammatory pathways and immune responses (12,13,17). They participate in inhibiting transcription factors, such as nuclear factor kappa-light-chain-enhancer of activated B cells and activator protein 1, as well as signaling cascades (such as mitogen-activated protein kinase, mechanistic target of rapamycin complex 1, janus kinase/signal transducer and activator of transcription). They are also responsible for suppression of pro-inflammatory cytokine synthesis and downregulation of toll-like receptor expression, inhibition of enzymes involved in eicosanoid synthesis, such as cyclooxygenase, lipooxygenase, and phospholipase A2, reduction of prostaglandin and leukotriene production, and modulation of immune cell function and gene expression. Finally, they attenuate chronic inflammation and immune dysregulation (12,13,16–19).

Studies confirm the antioxidant and anti-inflammatory properties of polyphenols. A study by Grabež et al. examined the antioxidant capacity of pomegranate peel extract (PoPEx) and quantified levels of tannins, phenolic acids, and flavonoids. PoPEx was given in a capsule to patients with type 2 diabetes mellitus (T2DM). As a result, reduction was observed in the oxidative stress biomarkers such as thiobarbituric acid reactive substances (TBARS), nitric oxide (NO), superoxide anion radical, hydrogen peroxide, but also in the plasma concentration of inflammatory factors, such as interleukin-6 (IL-6), tumor necrosis factor (TNF), and high sensitivity C-reactive protein (hs-CRP), while total antioxidant capacity (TAC) increased (26). Wang et al. also investigated the effects of polyphenols in mice. Administration of polyphenols from green tea increased antioxidant levels by activating uperoxidase dismutase, catalase, and glutathione S-transferase, and by downregulating malondialdehyde (MDH) and NO. What is more, the activity parameters of oxidative stress were

also lowered, inhibiting TNF, transforming growth factor-β, and interleukin 1 beta (IL-1β), IL-6, and the Nrf2 signaling pathway was regulated (27).

Although polyphenols have many health benefits, translating them into clinical practice is challenged by issues of bioavailability, dietary intake variability, and the need for standardized dosing and delivery systems (15,19). Emerging research highlights the potential for targeted nutritional interventions and improved delivery technologies to enhance therapeutic outcomes. Integrating polyphenol-rich foods into daily dietary practices may offer promising avenues for disease prevention and health promotion across the lifespan, especially for conditions associated with oxidative stress and inflammation.

Table 1. Types of polyphenols and representative dietary sources relevant to polyphenol-rich diets. Based on: (11,15,21,28–31)**.**

Main Class	Subclass	Representative Compounds	Major Dietary Sources
Flavonoids	Flavonols	Quercetin, Kaempferol, Myricetin, Rutin, Isorhamnetin	Onions, apples, berries, tea, red wine, leafy vegetables, broccoli, capers
	Flavones	Luteolin, Apigenin, Chrysin, Tangeretin	Parsley, celery, chamomile tea, citrus fruits, olive oil
	Flavanones	Hesperidin, Naringenin, Eriodictyol	Citrus fruits (oranges, grapefruits, lemons, limes), tomatoes
	Flavanols (Catechins)	Catechin, Epicatechin, EGCG, Proanthocyanidins	Green tea, black tea, cocoa, dark chocolate, red wine, apples, grapes
	Isoflavones	Genistein, Daidzein, Glycitein, Formononetin	Soybeans, soy products (tofu, tempeh, miso), legumes
	Anthocyanidins	Cyanidin, Pelargonidin, Delphinidin, Malvidin, Peonidin	Berries (blueberries, blackberries, strawberries), red grapes, cherries, red cabbage, eggplant
	Flavanonols	Taxifolin, Astilbin	Citrus fruits, milk thistle
Phenolic Acids	Hydroxybenzoic acids	Gallic acid, Protocatechuic acid, Vanillic acid, Syringic acid, Ellagic acid	Tea, red wine, berries (raspberries, strawberries), pomegranates, walnuts, chestnuts

Main Class	Subclass	Representative Compounds	Major Dietary Sources
	Hydroxycinnamic acids	Caffeic acid, Chlorogenic acid, Ferulic acid, p-Coumaric acid, Sinapic acid	Coffee, cereals, whole grains, potatoes, apples, pears, berries, artichokes
Stilbenes	–	Resveratrol, Pterostilbene, Piceatannol, Pinosylvin	Red grapes, red wine, peanuts, blueberries, cranberries, mulberries
Lignans	–	Secoisolariciresinol, Matairesinol, Pinoresinol, Lariciresinol, Sesamin	Flaxseeds, sesame seeds, whole grains, legumes, vegetables (broccoli, cabbage)
Tannins	Hydrolyzable tannins	Gallotannins, Ellagitannins (Punicalagin, Castalagin)	Pomegranates, walnuts, raspberries, strawberries, oak-aged wines
	Condensed tannins	Proanthocyanidins (Procyanidin B1, B2)	Cocoa, grapes, apples, cranberries, persimmons, cinnamon
Other Polyphenols	Curcuminoids, Coumarins	Curcumin, Demethoxycurcumin, Coumarin, Scopoletin, Rosmarinic acid	Turmeric, citrus fruits, rosemary, oregano, herbs and spices

3. Interval exercise and fatigue with recovery processes – mechanisms of fatigue, oxidative stress and inflammation, stress markers

3.1. High-intensity interval training

HIIT involves repeated bouts of near-maximal exercise with short recovery periods. This pattern produces strong metabolic, mechanical, and redox disturbances that contribute to the development of acute fatigue and drive post-exercise recovery. Original research indicates that HIIT induces metabolic, oxidative, and inflammatory stress (4).

3.2. Pathophysiology of HIIT - oxidative stress, inflammation, muscle damage

Oxidative stress is defined as “an imbalance between oxidants and antioxidants in favor of the oxidants, leading to a disruption of redox signaling and control, and/or molecular damage” (5).

During physical activity, particularly HIIT, a sharp increase in oxygen consumption occurs due to intense muscle contractions, leading to an imbalance between oxidants and antioxidants (5,6). In particular, ROS can be synthesized in contracting muscle by mitochondria, phospholipase A2 (PLA2), and nicotinamide adenine dinucleotide phosphate (NADPH) oxidases. Available sources suggest that during exercise, mitochondrial oxidative phosphorylation increases. PLA2 is an enzyme that, except for the release of arachidonic acid, activates NADPH oxidases, and its calcium-dependent form causes ROS production. NADPH exists in two forms in muscle: NOX2 (isoform 2 of NADPH oxidase) and NOX4 (isoform 4 of NADPH oxidase). However, NOX2 generates ROS in contracting muscles.

In the muscles, the NO synthase (NOS) is also expressed. NO is a reactive nitrogen species, and has an ability to react with other compounds, i.e., metals or superoxide anion, and then it is transformed into the reactive form - peroxynitrite (5).

The high mechanical and metabolic load of HIIT can also cause microdamage to myofibers and membranes, releasing intracellular contents that act as pro-inflammatory danger signals and initiate local immune activation (7,8). In the early recovery phase, circulating leukocytes rise and neutrophils are recruited into stressed tissue, where they generate reactive species and chemotactic signals that promote subsequent macrophage accumulation and inflammatory amplification (8,32). Macrophages initially adopt a more pro-inflammatory (M1-like) profile that supports debris clearance and remodeling (including via cytokines such as TNF-α), then progressively transition toward M2-like phenotypes under anti-inflammatory cytokine influence to facilitate satellite-cell activation, repair, and resolution-highlighting that tightly regulated inflammation is integral to regeneration rather than purely detrimental (8,32). Contracting muscle also releases myokines (notably IL-6 in an exercise context) that contribute to a systemic anti-inflammatory milieu (e.g., via induction of IL-1 receptor antagonist and IL-10), helping to terminate the inflammatory response and support recovery (8).

EIMD is caused by mechanical stress, primarily during eccentric contractions, where muscles lengthen under load, leading to ultrastructural changes in muscle fibers and disruption of the sarcomere architecture (32). High mechanical tension overstretches the weakest sarcomeres and disrupts the T-tubule and sarcoplasmic reticulum membranes, which in turn alters calcium

homeostasis and excitation–contraction coupling, impairing force transmission and muscle strength. The resulting increase in intracellular calcium activates proteolytic enzymes, further impairing muscle integrity and increasing sarcolemmal permeability, which allows muscle proteins like creatine kinase (CK), lactate dehydrogenase (LDH), and myoglobin (Mb) to leak into the bloodstream (7). Additionally, metabolic stress from intense or unaccustomed exercise contributes to muscle fiber disruption by generating reactive oxygen species and perturbing energy balance within the fibers. These structural and biochemical changes are clinically observed as transient strength loss, swelling and delayed onset muscle soreness (DOMS) following exercise (8).

All the mentioned effects of HIIT are shown on Figure 1.

3.3. Interval exercise - oxidative stress and hormonal imbalance in studies

The study by Bogdanis et al. investigated changes in oxidative stress biomarkers and antioxidant status indices following a 3-week HIIT regimen. It indicates that a single HIIT session leads to a large increase in oxidative stress markers in healthy, active men. 24 hours post key markers, which include protein carbonyls (PC), TBARS, GPX, CAT, TAC, increased. These indicate high ROS production, which leads to inflammatory activation, as leukocyte recruitment contributes to ROS after muscle stress. CK level was also found to be elevated, which indicates mild muscle damage. Those changes are consistent with neutrophil-related secondary oxidative activity following intense muscular stress. Those findings suggest that a single bout of HIIT produces high metabolic fatigue. According to the study, a 48-hour window is needed for normalization of most markers. The study then compared the results after 3 weeks of regular HIIT training. It was found that the oxidative stress response was reduced while antioxidant defense was enhanced. Those findings indicate that regular HIIT sessions cause adaptation and improve resilience to interval-induced fatigue. Crucially, the study confirms that interval training attenuates the oxidative and inflammatory components of fatigue while enhancing recovery efficiency (1).

A study by Ting-Ting Lee et al. investigated the effects of acute HIIT on immune function and oxidative stress in male canoe/kayak athletes who were well-trained. The key findings of the acute response align with other papers indicating that the HIIT protocol induced a marked oxidative stress response, reflected in significant alterations in endogenous antioxidant systems. Participants demonstrated changes in key antioxidant enzymes, including SOD and catalase, indicating increased ROS production during the interval session. However, the athletes in this study did not demonstrate pathological levels of oxidative or immune disturbance. Instead, their responses reflect a well-regulated, transient stress response, characteristic of trained individuals whose antioxidant systems

and immune networks are primed for rapid restoration of homeostasis, further confirming that regular training promotes adaptation to acute oxidative and inflammatory stress.

In the study, participants' serum cortisol levels were measured; concentrations were significantly higher immediately after a HIIT session, remained elevated at 3h and 24h post-exercise, and returned to normal levels by the 48-hour mark. This pattern reflects yet another pathway of significant systemic stress linked to inflammation and immune function and might further indicate the time needed to recover (2).

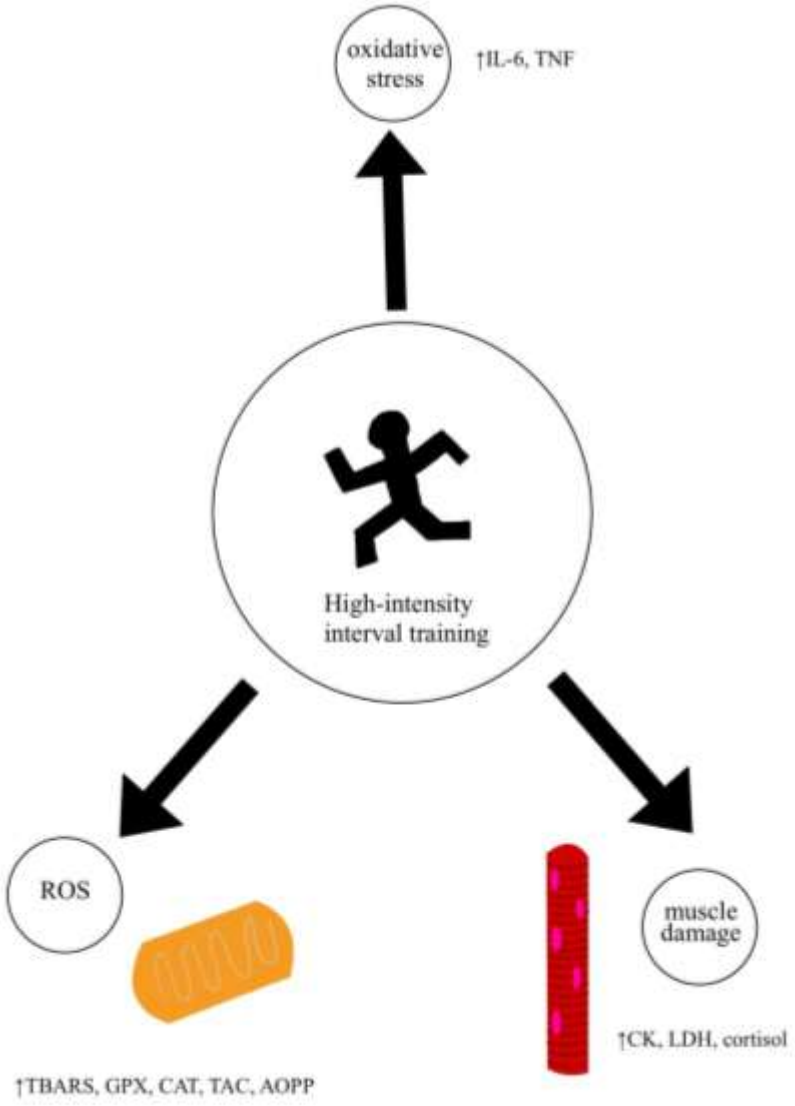

Figure 1. The impact of high-intensity interval training.

Abbreviations: ROS - reactive oxygen species, TBARS - thiobarbituric acid reactive substances, GPX - glutathione peroxidase, CAT - catalase, TAC - total antioxidant capacity, AOPP - advanced oxidation protein products, CK - creatine kinase, LDH - lactate dehydrogenase, IL-6 - interleukin-6, TNF - tumor necrosis factor.

3.4. HIIT-induced stress and regeneration in studies

A study by Souza-Silva et al. further reinforces the claim that HIIT induces oxidative, metabolic, and inflammatory stress. However, this study highlights that external conditions, such as heat, are important in shaping how athletes experience fatigue and their ability to recover. The findings from this study suggest that HIIT performed in hot conditions produced significantly higher TBARS than in thermoneutral conditions, while no increase in protein carbonyls was observed. This indicates that environmental heat amplifies oxidative stress, primarily affecting lipid structures rather than proteins (3).

A 2017 study by Cipryan further supports the claim that HIIT triggers a coordinated stress response involving inflammation, oxidative balance, and muscle-damage signaling, all of which contribute to fatigue and shape the subsequent recovery process. It also indicates that the body rapidly activates protective antioxidant mechanisms, suggesting that HIIT-induced oxidative and inflammatory stress is essential for adaptation. The study confirmed that HIIT elevates IL-6 levels, an important marker of inflammation and muscle damage (10).

A 2021 Study by Sarkar et al. evaluated the impact of 8 weeks of repeated HIIT on muscle damage indices, inflammatory markers, oxidative stress variables, and physical fitness parameters in trained athletes. The participants in the study exhibited progressive increases in oxidative stress markers (advanced oxidation protein products, TBARS) and cortisol, while immunoglobulin A levels were suppressed. The metabolic changes were not matched by improvements in athletic performance during the training block, indicating that the endocrine and oxidative burdens accumulated faster than adaptive processes could compensate for them. Sarkar's results therefore suggest that, even in well-trained endurance athletes, intensive HIIT protocols may exceed the capacity for optimal recovery, potentially predisposing them to chronic overload and immunological vulnerability, thereby reducing training quality (4).

The study by Al-Rawaf et al. (2023) determined the effects of repeated HIIT on sedentary patients with T2DM. It was discovered that this regimen imposes substantial metabolic and oxidative stress on skeletal muscle, reflected in elevated baseline levels of p53, cytochrome c (Cycs), and 8-hydroxy-2'-deoxyguanosine - markers associated with mitochondrial dysfunction, oxidative stress, and apoptosis. These metabolic changes contribute to fatigue, including impaired mitochondrial adenosine triphosphate (ATP) production, disrupted redox balance, and activation of pro-apoptotic pathways. Interestingly, 12 weeks of HIIT reversed many of these fatigue-related defects: p53 and Cycs levels decreased, TAC increased, and mitochondrial (mtDNA) content rose in both muscle

and serum, indicating improved mitochondrial biogenesis and enhanced resistance to oxidative stress. These findings highlight the beneficial effects of HIIT on mitochondrial biogenesis and on diabetes regulation via anti-apoptotic and antioxidant pathways (9).

In conclusion, across the literature, a consistent model emerges: acute HIIT increases oxidative stress markers (MDA, GPx, SOD, reduced glutathione (GSH)), inflammatory markers (IL-6, TNF-α), muscle damage markers (LDH, CK, cortisol), and disrupts antioxidant balance. Recovery depends on recovery intervals, the duration of HIIT protocols, and the environmental context. Adaptations vary - the balance between stress and recovery might determine whether HIIT acts as a beneficial adaptive stressor or a source of chronic overload.

4. Polyphenols as a potential remedy for oxidative stress and enhancer of regeneration

4.1. Supplementation of polyphenols in physical activity

As intensive, prolonged high-intensity training can induce fatigue, impair muscle function, and increase oxidative stress, professional sports teams and amateur athletes have begun to recognize the benefits of natural plant extracts and phytochemicals for accelerating post-exercise recovery and maintaining overall physical health (33). In the context of supplementation during HIIT, phytochemicals appear to play a vital role, as both short- and long-term HIIT induce intense ROS production and inflammation, which may lead to muscle damage and, in turn, muscle soreness. That may place a burden on an athlete's health (4). Analysis of 11 trials found that polyphenol supplementation may enhance endurance in regularly physically active individuals, especially with grape seed extract, green tea extract, and New Zealand blackcurrant extract (33). That's why it is important to investigate supplementary strategies that may enhance recovery, lower oxidative stress, and support overall muscle function in physically active individuals. The searched effects of polyphenols on oxidative stress, muscle damage, and recovery following physical activity are shown in Table 2.

Berry fruits are a great source of polyphenols, especially anthocyanins. The highest amounts of anthocyanins are found in chokeberries, black elderberries, blueberries, and blackcurrants (34). Bell et al. demonstrated in their study that supplementation with Montmorency Tart Cherry Concentrate in semi-professional soccer players for 8 days was associated with a decrease in some inflammatory markers, such as IL-6. In this study, polyphenol supplementation reduced inflammatory responses, potentially accelerating regeneration and muscular endurance. Moreover, maximal voluntary isometric contraction, countermovement jump, 20 m sprint, and 5-0-5 agility performances were

superior at 72 hours post-exercise with supplementation vs placebo, indicating that supplementation protected muscles against reduced endurance after intensive physical activity (35). Paton et al. conducted a study with 12 endurance-trained cyclists who received supplementation with blackcurrant and caffeine extracts. During maximal-intensity cycling in this study, there were no significant changes in either physiological or cognitive variables with any supplement treatment relative to placebo (36). Mendes et al. reported different outcomes in oxidative stress recovery following running. The study was conducted with 15 physically active, non-athlete runners, divided into groups receiving Juçara fruit juice, a known Brazilian source of anthocyanins (JFJ), or a placebo before HIIT. Supplementation with JFJ extract significantly reduced markers of oxidative stress, such as IL-6, TNF, and IL-1β, and accelerated recovery of muscle power after a sprint in the study group (37).

Although some experiments showed beneficial effects of berry-derived polyphenol supplements, some discrepancies remain. Due to differences in methodologies, dosing, frequency, and forms of supplementation, official recommendations for the use of polyphenol supplements are not justified at this time. However, future research in this area is required, as the high polyphenol content of berry-derived supplements offers a promising approach for preventing exercise-induced oxidative stress (34).

Resveratrol is widely present in red wine, grapes, berries, and nuts (38). Amirazodi et al. conducted a study in mice after swimming, with resveratrol (RES) supplementation. In the group of mice treated with HIIT and RES, antioxidant protection and mitochondrial function were higher than in the group without intervention or in the group treated with a single HIIT. What is more, a group of mice treated with HIIT and RES improved their swimming speed and distance covered (39). Another study in swimming rats supplemented with resveratrol confirmed that these interventions enhance antioxidant protection in the brain and support the function of energy systems (40). Different responses were observed in a study of 16 well-trained men: the intervention consisted of consuming 150 mg of grape juice per day for 4 days. After that HIIT, markers of oxidative protection were lower in the interventional group than in the placebo group, as resveratrol can modulate the naturally occurring antioxidant activity of enzymes induced by HIIT. That means chronic supplementation with RES during HITT can reduce post-exercise adaptation in skeletal muscles (41).

Although resveratrol appears to have a favorable effect on recovery after physical activity, the literature also indicates harmful effects of resveratrol intake (42). That means there are still no recommendations regarding nutritional prescription (43).

Green tea is made from the leaves of *Camellia sinensis*. It contains catechins (such as epigallocatechin-3-gallate) as well as quercetin, thearubigins, theaflavins, theanine, caffeine, chlorogenic acid, and gallic acid (44). Ghaemsi et al. observed a favorable effect of 1500 mg/day of green tea extract on HIIT in a group of obese endurance-trained women. Plasma levels of CAT and SIRT1 were higher in the group receiving green tea extract and performing HIIT than in the other groups. Moreover, the greatest increase in VO2max was observed in this group. It suggests that green tea extract not only enhances oxidant adaptation but also aerobic endurance (45). The similar effects of green tea extract on endurance were investigated in a group of 30 overweight women again by Ghaesmi et al. Sessions of HIIT with supplementation of 1500 mg of green tea extract again induced aerobic endurance by raising VO2max (46). What is more, in both studies, the protective effect of HIIT and supplementation on the cardiovascular system was found (45,46).

Curcumin and quercetin were also investigated for their effects on endurance and muscle regeneration. Kisiol et al. conducted a study on 16 male and 20 female cyclists who received curcumin during a 14-day HIIT protocol. Results suggest that HIIT assisted with supplementation does not reduce endurance or lactic acid responses, supporting regeneration (47). Effects of quercetin were observed in twelve physically active students receiving 1,000 mg of the substance per day for 7 days. After HIIT and supplementation, their plasma levels of TAC and SOD activities were higher, while MDA were lower. Also, V̇O2max was significantly improved by 75%. Also, inflammatory markers, such as CK and IL-6, were lower (48).

Studies on supplementation for physical activity also focus on cocoa, a great source of flavanols that support the body's antioxidant defense mechanisms. The results of this study were confirmed by Cavaretta et al. 24 elite football players were assigned to either a dark chocolate (>85% cocoa) intake group or a control group for 30 days. After 30 days of intervention, elite athletes who consumed dark chocolate showed increased antioxidant capacity and a significant reduction in markers of muscle damage (49). However, there are still not enough studies to verify if these effects also happen following HIIT. The studies focus in detail on various compounds, taking into account their sources, analyzing various parameters, and are conducted with people of varying degrees of training, which significantly hinders a detailed analysis of the effects of polyphenols (50,51).

Table 2. Effects of polyphenols on oxidative stress, muscle damage and recovery after physical activity.

Polyphenol	**Discipline of sport**	**Effects on oxidative stress**	**Effects on muscle damage and recovery**	**Source**
Anthocyanins	Soccer	↓ IL-6	↓ Time of 20 m sprint ↓ 5-0-5 agility ↑ maximal voluntary isometric contraction ↑ countermovement jump	Bell et al.
	Cycling	No significant changes	No significant changes	Paton et al.
	Running	↓ IL-6 ↓ TNF ↓ IL-1β	↑rPPO	Mendes et al.
Resveratrol	Swimming	↑ SOD ↓ SIRT3, SIRT4	↑ swimming speed ↑ distance covered	Amirazodi et al
	Swimming	↑ SOD1, SOD2 ↓ SIRT4	No significant changes	Mehrabi et al.
	HIIT training	↓ SIRT1 ↓ SOD2	No significant changes	Scribbans et al.
Catechins	Running	Parameters weren't tested	↑ VO2max	Ghaemsi et al.
	Running	Parameters weren't tested	↑ VO2max	Ghaemsi et al.

Curcumin	Cycling	No significant changes	No significant changes	Kisiolek et al.
Quercetin	Cycling	↑ TAC ↑ SOD ↓MDA ↓ IL-6	↑ VO2max ↓CK	Tsao et al.

Abbreviations: IL-6 - interleukin-6, IL-1β - interleukin 1 beta, TNF - tumor necrosis factor, SIRT - sirtuins, MDA - malondialdehyde, TAC - total antioxidant capacity, SOD - superoxide dismutase, CK - creatine kinase, LDH - lactate dehydrogenase, VO2max - maximal oxygen uptake, VT1 - ventilatory threshold 1, VT2 - ventilatory threshold 2, rPPO - relative peak power output, ↓ - decrease, ↑ - increase.

4.2. Polyphenols vs adaptive response to training

Antioxidant supplementation is commonly used to enhance athletic performance (52). Even though polyphenols have proven effects on reducing ROS after exercise, some evidence suggests that antioxidant supplementation may also impair these adaptations under overload stress which cause remodeling of skeletal muscle following resistance and high-intensity exercise and are more dependent on ROS signalling.

ROS, through their signalling capacity, can act as inducers of adaptive responses in muscles. As a result, they increase the activity of antioxidant enzymes, promote mitochondrial biogenesis, and enhance insulin sensitivity. When antioxidants are supplemented, they interact with ROS, especially when used regularly, and can interrupt these processes (53).

A systematic review on this topic by Pastor et al. suggests that the antioxidant response to exercise depends on two factors: frequency and timing of supplementation. Frequent supplementation can impair adaptation processes and is rather beneficial for those with a low basal antioxidant level. In those who take supplements shortly before or during physical activity, antioxidants delay fatigue and shorten the recovery period (54). According to that, it is recommended that special caution should be taken regarding supplementation of antioxidants. The recommended intake of micronutrients should be met for those who undertake regular physical activity (52).

5. Conclusions

Collectively, the evidence summarised in this study indicates that polyphenols—through their antioxidant, anti-inflammatory, immunomodulatory, and metabolic actions—represent promising nutritional tools to modulate the redox and inflammatory burden imposed by HIIT, while also supporting broader cardiometabolic health. Acute HIIT repeatedly emerges as a potent systemic stressor that increases markers of oxidative damage, inflammatory cytokines, and indices of muscle disruption, with recovery trajectories strongly influenced by training status, environmental conditions, and the density of interval sessions. At the same time, both human and experimental data confirm that this stress is not purely detrimental: repeated HIIT can upregulate endogenous antioxidant defences, improve mitochondrial function, and enhance resilience to metabolic fatigue when the balance between load and recovery is preserved. Against this background, polyphenols—including berry anthocyanins, pomegranate, grape-derived products, green tea catechins, resveratrol, curcumin, quercetin and cocoa flavanols—consistently demonstrate the ability to lower lipid peroxidation and DNA damage, enhance activities of key antioxidant enzymes (SOD, CAT, GPx), and attenuate circulating inflammatory mediators in both clinical and preclinical models of oxidative stress and muscle injury. Some controlled trials and meta-analytic data suggest that such effects may translate into improved endurance, reduced inflammation, or better maintenance of muscle function and performance after strenuous exercise, particularly with selected berry extracts, tart cherry, grape products, and mixed polyphenol formulations. However, other studies report null effects of polyphenol supplementation on recovery or performance, and long-term cocoa or green tea interventions have in some cases failed to enhance, or even appeared to dampen, markers of mitochondrial adaptation despite favourable changes in oxidative stress biomarkers. Importantly, mechanistic work on antioxidant supplementation indicates that chronic, high-dose attenuation of exercise-induced reactive oxygen species can interfere with redox-sensitive signalling pathways that drive mitochondrial biogenesis, endogenous antioxidant upregulation and other beneficial training adaptations, making dose, formulation and timing critical variables. Overall, the most coherent interpretation of the current literature is that polyphenol-rich diets should be prioritized as a foundational strategy to support redox homeostasis, immune function and long-term health in physically active individuals, while targeted, short-term supplementation may be considered around periods of exceptional load or insufficient recovery, particularly in those with low baseline antioxidant status or metabolic comorbidities. At the same time, current data do not justify blanket recommendations for high-dose polyphenol supplements in athletes or highly active populations; instead, future work should focus on well-controlled, sport-specific trials that systematically vary

polyphenol type, dose and timing, account for bioavailability and gut microbiota interactions, and integrate both performance and molecular endpoints to clarify when polyphenols function as beneficial hormetic modulators of HIIT-induced stress and when they risk attenuating desirable training adaptations

Disclosure

Supplementary Materials

There are no supplementary data connected with this article.

Author Contributions

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Conflicts of Interest

The authors declare no conflicts of interest.

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