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Monitoring and Managing Inflammation in Professional Tennis: A Biomarker-based Review of Recovery Interventions

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ABSTRACT

Inflammation is a necessary biological response to physical exertion, initiating muscle repair at the cellular level. However, in professional tennis, the compressed tournament calendar and back-to-back match schedules repeatedly trigger the inflammatory cycle before it has fully resolved, shifting the response from adaptive to potentially degenerative. This review examines exercise-induced inflammation in elite tennis players by reviewing 11 blood-based biomarkers across four major phases: pro-inflammatory, transitional, anti-inflammatory, and stress markers. Biomarker fluctuation patterns are analyzed across tennis and adjacent sports literature and compared against aerobic and team sports contexts to identify shared and sport-specific inflammatory trends. Findings indicate that while the general sequence of the inflammatory response is consistent across sports, tennis players exhibit specific unique trends. Elite tennis athletes exhibit a cortisol response driven by psychological stress unique to individual-sport competition, chronically elevated IL-6 levels throughout full competitive seasons, and progressive CK accumulation across matches and tournaments. Among the recovery interventions reviewed, whole-body cryostimulation was the only method that produced meaningful changes in cytokines, whereas mixed-methods recovery protocols provided no additional benefit compared to passive rest. These findings suggest that the timing of intervention relative to the inflammatory phase, rather than the type of intervention alone, is a critical determinant of effective recovery for professional tennis players.

INTRODUCTION

Inflammation is a functionally beneficial immune response that is vital in signaling muscle repair and tissue remodeling following high-intensity exertion. Historically, in sports medicine, inflammation following physical exertion has been considered detrimental to muscular health, as it is associated with pain, tissue damage, and delayed recovery (Peake et al., 2017). Inflammation simultaneously serves as one of the body's most essential recovery processes and one of the greatest threats to both athletes' careers and health, creating a paradox that complicates its role in professional sports.

Inflammation

Inflammation is a key process in muscular repair and regeneration. At the cellular level, the acute inflammatory response is a protective response triggered by muscular stress or tissue damage. At the site of the injury, immune cells such as neutrophils and macrophages accumulate, break down damaged muscle tissue through phagocytosis, and release proteolytic enzymes and reactive oxygen and nitrogen species, signalling molecules that activate satellite cells (Peake et al., 2017). Satellite cells are responsible for muscle fiber repair and regeneration. Without the inflammatory signal, the muscular repair process can not begin, so suppressing inflammation following exercise would be counterproductive to muscular regrowth.

The same inflammatory response can have negative consequences when not properly resolved. Pro-inflammatory signalling can persist beyond its window of local perturbations, such as with repetitive muscle trauma, or systemic disorders such as COPD (Duchesne et al., 2017). When this occurs, the signals impair the skeletal muscle healing process by affecting the regenerative capacity of satellite cells, leading to dysfunctional repair mechanisms, such as fat buildup and muscle fibrosis. Therefore, failure to resolve inflammation, rather than the inflammation itself, represents the real threat to athletes and their ultimate performance.

Components of Inflammatory Response

To effectively manage this inflammatory cycle, it is necessary to monitor its progression. This is possible by analyzing a range of biomarkers that are detectable in blood and serum, which accurately represent the stage or phase of the inflammatory response at any given point. The key markers relevant to exercise-induced inflammation in athletes are outlined in Table 1.

The markers in Table 1 are most effective when assessed from a collective point of view, in which they serve as a comprehensive panel rather than independent variables. Together, these markers demonstrate the full inflammatory response. They can be grouped by their roles in the process, starting with pro-inflammatory markers. Pro-inflammatory markers include Interleukin- 1β (IL-1β), tumor necrosis factor-alpha (TNF-α), hydrogen peroxide (H_2O_2), and Myoglobin, which signal tissue damage and make up the acute inflammatory process. Next, transitional markers, Interleukin-6 (IL-6) and Cortisol, represent the transfer phase from pro-inflammation to anti-inflammation, signaling the shift from damage response to repair. Anti-inflammatory markers, Interleukin-10 (IL-10), Creatine Kinase (CK) and Heat Shock Protein 27 (HSP27), are indicators that the regenerative process has commenced, and stress markers include Heat Shock Protein 70 (HSP70) and C-reactive Protein (CRP), which indicate that the inflammation has exceeded the body's capacity to resolve the issue. To analyze the inflammatory cycle at any given point, tracking these markers across all groups is necessary to understand the true state. When considering intervention, it is important to analyze current biomarkers and the stage of the inflammatory cycle to select an intervention that best supports recovery.

Inflammation markers	Description	Baseline measure	When it increases	Citations (APA)
Creatine Kinase (CK)	Enzyme released into the blood flow from damaged skeletal muscle; indirect marker of muscle damage	35–175 U/L with ranges from 20 to 16,000 U/L in general healthy population	Progressively rises during repeated exercise, peaks around 24 hours after damaging activity and remains elevated up to 7 days	(Baird, 2012)
C-reactive Protein (CRP)	Acute phase protein produced in response to pro-inflammatory cytokines; marker of systemic inflammation	0.2 mg/L to 13.9 mg/L in young adults/athletes	Slow elevation, peaking 24-72 hours after exercise, remaining above baseline for 2-3 days before gradually returning to normal	(Kasapis & Thompson, 2005)
Interleukin-6 (IL-6)	Cytokine with pro and anti-inflammatory roles; produced by contracting skeletal muscles; marker of transition from inflammation to repair	< ~1.6 pg/ml in young adults/athletes	Spikes immediately post-exercise, then rapidly declines to baseline within 24-48 hours	(Ziemann et al., 2013; Pedersen & Febbraio, 2008)
Interleukin- 1β (IL-1ß)	Pro-inflammatory cytokine; part of immune cell response to exertion; initiating and amplifying the acute phase and stress responses; marker of inflammation	< 1.0 pg/mL at rest in general healthy population	Peaks at the end of exercise and remains elevated up to 24 hours post-exercise	(Moldoveanu et al., 2000)

Tumor Necrosis Factor-α (TNF-α)	Pro-inflammatory cytokine; promotes inflammation; significant short- and long-term effects on protein synthesis; part of stress response; marker of inflammation	<0.04 pg/ml at rest in general healthy population	Peaks at the end of exercise and remains elevated up to 24 hours post-exercise	(Moldoveanu et al., 2000)
Interleukin-10 (IL-10)	Anti-inflammatory cytokine; blocks pro-inflammatory signaling and promotes tissue repair; induced by IL-6	1.51 ± 0.86 pg/mL	Highest peak in phase immediately after exercise, returning to baseline within 60 minutes in moderate exercise, varies with longer bouts of activity	(Cabral-Santos et al., 2016)
Heat Shock Protein 70 (HSP70)	Heat shock protein; pro-inflammatory signal; marks acute protein damage and homeostatic disruption	15 ± 3 ng/15 µg in the general healthy population	Elevated after cellular stress, peaking around 48 hours post-exercise, declining to baseline as body recovers	(Paulsen et al., 2012; Thompson et al., 2002)
Heat Shock Protein 27 (HSP27)	Heat shock protein; anti-inflammatory signalling recovery and structural repair; inversely tracking HSP70 in recovery phase	300 ± 63 ng/15 µg in general healthy population	Rises during recovery phase as HSP70 falls; inverse relationship with HSP27; protective response to cellular stress	(Paulsen et al., 2012; Thompson et al., 2002)

Hydrogen Peroxide (H202)	Reactive oxygen species; produced by skeletal muscle contraction; oxidative stress marker increased during bouts of aerobic exercise	2.5 - 3.0 µM in general healthy population	Increases immediately after exercise then returns to near baseline levels at 3 hours post-exercise; chronically elevated with repeated exercise and inadequate recovery	(Thomas et al., 2023)
Cortisol	Stress biomarker; biomarker for catabolic and anabolic state of body; increases availability of all fuel substrates	~250 nmol/L	Increase during exercise, peaking at point of maximal effort; gradual decline as recovery period progresses	(Popovic et al., 2019)
Myoglobin	Indicator of muscle injury, stored in muscle fibers, released when muscle cell membranes are disrupted, marker of muscular stress	24.6 ± 12.4 ng/ml	Increases significantly during large bouts of exercise, peaking 1-3 hours after exertion; may leak into circulation during muscle damage	(Baird, 2012; Sabriá et al., 1983)

Table 1: Inflammation Marker Physiology

The Sport of Tennis

Given the unique nature of tennis and its demands on the human body, the regulation and management of inflammation is critical to tennis players. The movements in tennis vary significantly and place stress on multiple muscles throughout the body. Players are required to perform multiple repetitive overhead loads during serves and overhead smashes, explosive start-stop sprinting patterns, and repeated lateral changes in direction, placing stress on lower-body muscles. This combination of upper- and lower-body demands is maintained throughout multiple sets and consecutive match days.

The training status of athletes is a controlling variable in the inflammatory response, with more conditioned athletes demonstrating reduced inflammatory responses and faster recovery times (Gomes,

2014). This shows that the severity of inflammation is not uniform across all players. The question of whether inflammation is beneficial or detrimental in tennis has no direct answer. Acute inflammation is necessary for adaptation and repair, the tennis calendar creates conditions where the inflammatory response is repeatedly activated before full muscle regeneration has occurred, causing it to become potentially degenerative.

The professional tennis agenda is a unique stressor for these athletes, with compressed schedules from back-to-back tournaments and limited recovery time between matches and events (Kozłowska, 2021). Unlike in team sports, tennis players compete daily or every other day in tournaments, creating conditions in which performance markers degrade. Maximal voluntary contraction (MVC), the highest amount of force a muscle or muscle group can generate through a voluntary effort, and rate of force development (RFD) of professional players decline significantly throughout tournaments, which causes meaningful decrements in performance (Ojala & Häkkinen, 2013). CK progressively rises from match to match, and delayed-onset muscle soreness (DOMS) worsens over time. 48 hours after the tournament's end, multiple markers remain unrecovered, representing residual inflammation and signaling the body's cumulative response to the net stress of the tournament rather than to single bouts of exertion (Ojala & Häkkinen, 2013).

Interventions for Inflammation in Tennis

The current state of research on inflammation suppression in tennis is still in its early stages. While a broad range of recovery interventions specific to inflammation has been analyzed in tennis, these methods have largely been evaluated in isolation rather than in a combined approach (Wiewelhove et al., 2022). The timing of intervention (and suppression of the inflammatory response) has not been accounted for in prior research. While biomarker data suggest that interventions may affect the inflammatory response, no clear answer has emerged (Kovacs & Baker, 2014). Despite evidence supporting multiple recovery methods, limited information is available on the role of intervention timing relative to inflammatory marker levels in professional tennis players.

CYTOKINE FLUCTUATION

The immunological response to professional sports and exertion is not uniform across biomarkers. Various biomarkers and cytokines exhibit individual patterns depending on their physiologic roles, an athlete's conditions, and training status. Understanding how these patterns work and how they differ across multiple sports is vital in developing a clear understanding of the inflammatory response.

Pro-inflammatory Markers

Amongst the pro-inflammatory markers, IL-1β has been shown to rise slowly yet significantly during prolonged periods of exertion, such as marathons, with levels continuing to increase during the recovery period (Nieman, 2001; Pedersen, 2000). TNF-α, another pro-inflammatory cytokine, represents a similar pattern following exertion. TNF-α levels increase during fatigue, triggering inflammation and muscle fiber breakdown (Pelana, 2025). H_2O_2, a reactive oxygen species, increases significantly following

exercise, which reflects increased oxidative stress post-exertion (González-Haro, 2020). Myoglobin levels increase acutely after intense physical activity or exertion (Pelana, 2025). Myoglobin is released in the body's circulation following muscle fiber damage and signals muscular stress and inflammation.

Transitioning Markers

Within the transitioning biomarkers group, IL-6 levels rise in association with low muscle glycogen stores and serve as a marker for exercise-induced inflammation, with significant increases during the recovery period (Pelana, 2025; Duarte, 2026). Cortisol, a primary stress hormone, increases in response to more psychological and physiological stress during intense physical activity. However, its mismanagement and excessive elevation can contribute to fatigue and impair recovery.

Anti-Inflammatory Markers

The anti-inflammatory response is characterized by the rise of suppressive mediators, such as IL-10, HSP27, and CK. IL-10 rises significantly following intense exertion, such as marathons or races, and remains elevated for several hours during the recovery period, indicating a sustained immune response (Nieman, 2001). HSP27 follows a more incremental pathway, not rising immediately after exertion but increasing and peaking during the recovery phase, indicating its role in muscle repair rather than acute response. CK elevation is dependent on the context of the situation, mainly increasing when muscle damage is serious, and muscle fatigue and adaptation are both indicated by the elevation of CK.

Stress Markers

Certain cytokines and biomarkers are simply indicators of stress, signalling recovery status after exertion. HSP70 has been shown to rise significantly after exercise and return to baseline around 24 hours later (Gavrilov, 2020). A faster muscle recovery is exemplified by an accelerated return of HSP70 to baseline levels. CRP reflects the body's recovery status and indicates systemic inflammation when elevated (Pelana, 2025). In non-athletes and the general population, CRP exhibits an upward trend immediately after exertion and a slow, downward trend over time. (Herawati, 2025) However, in pro-athletes, CRP decreases immediately post-exercise, indicating a reduced need for an anti-inflammatory response and greater adaptation to recovery.

BIOMARKER FLUCTUATIONS IN TENNIS

Although the general fluctuations of inflammatory biomarkers have been reviewed above in various athletic contexts, the unique demands of elite tennis players, characterized by repetitive high-intensity movements, unique biomechanical demands such as overhead loading and lateral force production, and compressed tournament schedules, suggest that these athletes may exhibit distinct immunological patterns. The continuous cycle of exertion and inadequate recovery periods may lead to prolonged systemic stress and residual inflammation. Given the need to effectively manage this response, the specific biomarker fluctuations reported in Table 2 reveal unique trends that reflect the pattern of inflammation observed in tennis athletes.

Citation	Paper Context	Participant Characteristics	Biomarker	Timeframe	
				5 months	**9 months**
(Kozłowska, 2021)	Observational study; 38 young male tennis players'(14-23 y/o) immunological responses were tracked across a season (Jan.-Sep.)	**Cadets:** "<16 years (n = 12, age 14 ± 0.5, height 176 ± 8 cm, and body mass 63 ± 7 kg)"	*TNF-α*	/	"+"
			IL-6	"+++"	"+++"
			IL-10	"++"	"+++"
			HSP70	"+"	"+"
			HSP27	"+"	"+"
		Juniors: "16–18 years (n = 11, age 17 ± 1, height 181 ± 6 cm, and body mass 72 ± 9 kg)"	*TNF-α*	"+"	/
			IL-6	"++++"	"++++"
			IL-10	"++"	/
			HSP70	"+"	"+"
			HSP27	"+"	"+"
		Senior: ">18 years (n = 13, age 22 ± 3, height 187 ± 6 cm, and body mass 78 ± 5 kg)"	*TNF-α*	"+"	"+"
			IL-6	"++++"	"++++"
			IL-10	"+++"	"++"
			HSP70	"+"	"+"
			HSP27	"++"	"++"
(Ziemann et al., 2013)	Observational study; a group of young tennis players' (16 y/o) inflammatory mediators and heat shock proteins were recorded during a training camp following a season.	Juniors: 15 tennis players (16 y/o)		**4 days**	**2 weeks**
			H2O2	"-"	"-"
			IL-ß	"--"	"-"
			TNF-α	"-"	"+"
			IL-6	"+"	"+"
			IL-10	"-"	"+"
			HSP27	"++++"	"+"
			HSP70	"-"	"-"
			CK	"-"	"++++"
(Ziemann et al., 2012)	Controlled study; 12 professional male tennis players'	Cryostimulation: 6 national-level tennis players		**10 days**	
			TNF-α	"--"	

	cytokine, hormonal, and hematologic responses were tracked across a 5-day whole-body cryostimulation and training program during a post-season camp.	("ATP singles ranking ¼ 150–900, ATP doubles ranking ¼ 11–15")	*IL-6*	"+"	
			Cortisol	"+"	
			CK	"-"	
		Control: 6 national level tennis players ("ATP singles ranking ¼ 150–900, ATP doubles ranking ¼ 11–15")	*TNF-α*	"-"	
			IL-6	"-"	
			Cortisol	"-"	
			CK	"+"	
(Ojala & Häkkinen, 2013)	Observational study; 8 national-level male tennis players' physiological and performance levels were analyzed during a 3-day tennis tournament and the following 2-day recovery period.			**During exercise**	**24**
			CK	"++"	"+"
			Cortisol	"++"	"+"
"+" = small increase (<0.59 %); "++" = medium increase (0.60% – 1.19%) "+++" = large increase (1.2% – 1.99%) "++++" = very large increase (2.0% – 3.99%) "-" = small decrease (<0.59 %) "--" = medium decrease (0.60% – 1.19%) "---" = large decrease (1.2% – 1.99%) "----" = very large decrease (2.0% – 3.99%) / = unclear					

Table 2: Overview of Inflammation Markers in Tennis Athletes

Comparison of tennis-associated inflammation with other sports

Pro-inflammatory Markers

The pro-inflammatory response following exertion exhibits similar trends between aerobic sports and tennis. IL-1β follows a consistent pattern of elevation after exertion, showing a steady but significant rise in general sports and a large increase immediately after exertion in tennis (Nieman, 2001; Pedersen, 2000; Ziemann, 2013). IL-1β is also a pro-inflammatory marker of acute stress across all sports. TNF-α is linked to muscle fatigue and is generally associated with increases in physiological stress. Across studies of tennis and other sports, TNF-α is elevated after exertion (Ziemann, 2013; Kozłowska, 2021). Both categories demonstrate significant post-exercise increases of H2O2, signaling oxidative stress as a

universal consequence of physical activity (González-Haro, 2020; Ziemann, 2013). Myoglobin, driven by muscular stress, exhibits acute increases following intense exercise in both tennis and aerobic sports, indicating a shared mechanism in myoglobin regulation. These shared characteristics between tennis and all sports represent a consistent pattern of post-exertion pro-inflammatory response, a positive sign that tennis athletes are initiating recovery in ways similar to others.

Transition Markers

Some biomarkers are categorized as both pro- and anti-inflammatory, signaling transitions between the two phases of the inflammatory response. IL-6 is widely recognized across sports as a dual-role cytokine, with both tennis and aerobic sports literature describing it as a transitional biomarker which elevates during the recovery period and can support or contribute to inflammation.(Pelana, 2025; Kozłowska, 2021). Cortisol is a stress-responsive hormone that rises with exercise intensity. Both tennis and aerobic sports literature describe cortisol as a biomarker that shows moderate increases during and after exertion (Ojala & Häkkinen, 2013; Pelana, 2025). The parallels between transition markers in tennis athletes and those in other athletes show that the relative timing and progression of phases of the inflammatory response are similar.

Anti-Inflammatory Markers

The secondary stage of the inflammatory response comprises anti-inflammatory cytokines and biomarkers that suppress muscle inflammation. IL-10 is widely recognized in tennis and aerobic sports as an anti-inflammatory mediator, rising alongside IL-6 following exercise (Nieman, 2001; Kozłowska, 2021; Ziemann, 2013). CK is a marker of muscle damage and fatigue, showing increases following long-term exertion in studies of aerobic sports and tennis (Pelana, 2025; Ojala & Häkkinen, 2013). HSP27 shows very similar trends across both categories, showing increased levels during the repair period rather than immediately post-exertion (Ziemann, 2013; Kozłowska, 2021). For athletes, the presence of these markers collectively indicates the initiation of the recovery process, making their constant elevation a positive sign that the inflammatory cycle is resolving rather than accumulating.

Stress Markers

Specific biomarkers exhibit trends that signal inflammatory patterns during and after exertion, making the phases of the recovery process more quantifiable. In general, across aerobic sports and tennis, HSP70 is the only known biomarker that shows consistent patterns. HSP70 is identified in both categories as a post-exercise stress marker that rises following cellular disruption and returns to baseline, signaling recovery progression (Gavrilov, 2020; Kozłowska, 2021). For athletes, the rate at which HSP70 declines and returns to baseline is therefore a practical indicator of recovery quality, with accelerated declines suggesting greater physiological resilience and ability to compete.

Differences between inflammation in tennis and other sports

These shared patterns highlight the universal nature of the body's response to physical stress, though certain biomarkers function differently in the context of tennis. While a variety of cytokines have been

mentioned above, those discussed in the sections below exhibit distinct trends that indicate inflammatory regulatory mechanisms for tennis athletes.

Pro-inflammatory Markers

Among the pro-inflammatory markers, IL-1β exhibits a unique suppression mechanism in tennis players. In aerobic sports, IL-1β follows a slowly rising, persistent pattern through the recovery period (Nieman, 2001). In tennis, small-to-medium decreases in IL-1β were observed after exercise, coinciding with large increases in HSP27 (Ziemann, 2013). This suggests an inverse relationship between HSP27 and IL-1β, a trend not widely articulated in aerobic sports data. The association between HSP27 and IL-1β indicates a physiological shift from a pro-inflammatory to an anti-inflammatory response, as suppressive cytokines increase and pro-inflammatory cytokines decrease, signaling progression of the recovery phase.

Transition Markers

IL-6 levels in tennis players over full seasons are markedly elevated (Table 2). While on average, aerobic sports demonstrate significant IL-6 increases post-exertion, tennis players exhibit markedly larger increases in IL-6 among junior and senior players at both 5 and 9 months into the competitive season, with levels sustained throughout the full training year. (Pelana, 2025; Duarte, 2026; Kozłowska, 2021). This suggests that repetitive, high-intensity competition with inadequate recovery periods within a compressed window may drive chronically elevated IL-6 levels that exceed those typically reported in aerobic sports contexts. In tennis, cortisol is stimulated from pre-match preparation to post-match recovery, rising with exertion and stress (Pelana, 2025; Ojala & Häkkinen, 2013). Specifically, in tennis, cortisol rises are driven by psychological stress before play has even begun (Filaire, 2009). Cortisol also varies with match outcomes, a pattern not often seen in aerobic sports, reflecting the psychological pressure of individual sport competition, where there are no teammates, thereby reducing accountability (Lautenbach, 2015).

Anti-Inflammatory Markers

Aerobic sports often observe CK levels peaking around 24 hours after exertion, remaining elevated for up to 7 days during recovery, then slowly declining (Baird, 2012). In tennis tournaments, however, CK exhibits a far more complex trajectory, progressively accumulating from match to match (Ojala & Häkkinen, 2013). Professional tennis players often exhibit these unique, cumulative CK patterns during the recovery phase, suggesting that the response pattern is specific to their cumulative stress profile. Recognizing these unique cytokine trajectories is essential for practitioners to move beyond generalized recovery protocols and develop precision-timed interventions that align with the specific physiological window of the tennis athlete.

MANAGING INFLAMMATION AND RECOVERY

Best Practice

The following section reviews the most commonly applied recovery interventions in professional tennis and evaluates their effects on the inflammatory biomarkers outlined in Table 1. Although various methods of inflammation management have been studied across various sports contexts, the cytokine-specific effects of various recovery methods in tennis athletes remain inconsistently reported. The interventions discussed below are based on available evidence from tennis-specific and adjacent literature, with particular attention to their influence on pro-inflammatory, transitional, anti-inflammatory, and stress markers that characterize the inflammatory response discussed previously. The effects on biomarker fluctuations are not uniform across modalities, and the extent to which each effectively regulates the inflammatory response varies considerably.

Massage involves the physical, therapeutic manipulation of soft tissues through techniques such as effleurage (stroking), petrissage (kneading), and friction (rubbing), targeting the major muscles of the back, shoulders, arms, and legs (Wiewelhove et al., 2022). The physical procedure stimulates circulation to reduce muscular stiffness and improve tissue recovery, and massage likely enhances blood perfusion, supporting the redistribution and removal of metabolic waste products (Ismail, 2026). However, when measured directly, massage applied as part of a mixed-methods protocol showed little to no significant effect on the inflammatory cytokines discussed (Wiewelhove et al., 2022). The limited effect of this intervention may be further explained by the heat generated by friction during massage, which may counteract the vasoconstriction induced by the preceding CWI, potentially confounding the findings of the study.

Cold water immersion (CWI) involves submersion of the body in water at 10-15 °C and is one of the most common methods of post-exercise recovery used by tennis (Duffield et al., 2014; Kovacs & Baker, 2014). CWI is associated with reduced post-exercise muscle temperatures, hydrostatic pressure-mediated clearance of metabolic waste products, and similar symptoms, which demonstrate minimized tissue damage and reduction of post-exercise inflammation
(Kovacs & Baker, 2014). Contrast therapy, a similar alternative to CWI, involves alternating between cool and warm water immersion sessions or the alternating application of cold and hot compresses directly to major working muscles. The goal of contrast therapy is to stimulate vascular responses and enhance tissue recovery within inflamed muscles (Ismail, 2026). Contrast therapy demonstrates faster clearance of blood lactate and creatine kinase, with accelerated muscle recovery post-inflammation (Kovacs & Baker, 2014).

Whole-body cryostimulation (WBC) differs from other low-temperature-based methods in both the evidence for cytokines and the underlying physical mechanisms. Rather than water immersion, WBC involves brief exposure of athletes to extremely cold air in a cryogenic chamber at temperatures between -110 C and -120 C immediately post-exercise or in the days following competition (Kovacs & Baker, 2014). WBC is the only intervention with direct cytokine-measurement data showing a clear anti-inflammatory benefit for tennis athletes. The application of WBC twice daily, combined with

moderate-intensity training, produced a 60% reduction in TNF-α, compared with 35% in the training-only control group; a 23% increase in IL-6, compared with a slight decline in the control group; and a significant decrease in CK (Ziemann et al., 2012). Following the administration of WBC, the cytokine patterns exhibit an accelerated transition from the pro-inflammatory phase, with a reduction in TNF-α, and towards the repair phase, with an increase in IL-6 as a transitional marker, while simultaneously reducing CK as a marker of residual muscle damage.

Mixed-method recovery (MMR) comprises active recovery, stretching, CWI, and massage, which are applied sequentially following match play. The rationale of MMR is that combining multiple methods of suppression may produce greater cumulative recovery benefits than any single intervention alone. However, the available evidence does not support this assumption. When applied over a five-day tournament, MMR had no significant effect on any measured biomarker, with CK and CRP following the same trajectory as passive recovery across all time periods (Wiewelhove et al. 2022). Collectively, these findings suggest that the sequencing, timing, and specific parameters of each component within a combined protocol may be as important as the choice of method itself.

Active recovery consists of a light, self-paced movement routine immediately following match play to promote gradual physiological normalization by enhancing blood circulation and facilitating the removal of metabolic byproducts (Ismail, 2026; Wiewelhove et al., 2022). While active recovery is widely utilized in practice, the evidence suggests it is limited in its ability to meaningfully limit exercise-induced fatigue, accelerate performance restoration, or prevent injuries when applied alone (Wiewelhove et al., 2022). Passive recovery involves unstructured rest without active physiological stimulation, composed of basic, self-selected techniques such as stretching. Despite the absence of structured intervention, passive recovery showed a biomarker response almost indistinguishable from MMR over a five-day tournament, with an identical inflammatory marker trajectory. CK and CRP showed the same significant time-based changes, suggesting that the body, stimulated under repeated daily match play, follows an inflammatory trajectory that is largely resistant to the suppression methods most commonly utilized in professional tennis, with the notable exception of WBC, which remains the only intervention in this review which exhibited the ability to alter the cytokine environment in a directionally meaningful way.

CONCLUSION

The inflammatory response in professional tennis represents a fundamental physiological paradox. Inflammation is not inherently harmful, as it is one of the body's most beneficial recovery processes, which initiates muscle repair and tissue remodeling following intense exertion. The real threat to an athlete's health and performance is not the presence of inflammation but the failure to resolve it. When the inflammatory cycle is re-initiated before full resolution, as the structure of professional tennis constantly demands, the response shifts from adaptive to potentially degenerative.

Effectively monitoring this inflammatory cycle requires assessing multiple markers and cytokines. The panel of eleven biomarkers outlined in Table 1 maps the full inflammatory response across four phases. Pro-inflammatory markers, IL-1β, TNF-α, H_2O_2, and myoglobin signal tissue damage and comprise the

acute inflammatory process. Transitional markers, such as IL-6 and Cortisol, represent the transition from pro-inflammatory to anti-inflammatory states, signaling the shift from the damage response to repair. Anti-inflammatory markers, IL-10, CK, and HSP27, are indicators of the initiation of the regenerative process, and stress markers, HSP70 and CRP, indicate that inflammation has exceeded the body's capacity to resolve it.

The biomarker data reviewed throughout the paper reveal two sets of findings: those consistent across tennis and other sports, and those specific to tennis athletes. The acute inflammatory response in tennis follows the same universal biological sequence observed in both aerobic sports and team sports, with each biomarker category progressing through similar trajectories regardless of sport type. However, several markers exhibit tennis-specific behaviors that cannot be explained in general sports contexts. IL-6 is chronically elevated in tennis players throughout full competitive seasons, far exceeding levels reported in studies of aerobic sports, a trend likely driven by the compressed tournament schedules and inadequate recovery windows in professional tennis. Instead of peaking and declining slowly following exertion as observed in other sports, CK accumulates progressively from match to match, confirming that tennis players do not reach full muscular recovery between exertion periods. Cortisol in tennis involves a psychological element, rising before match play even begins and varying with match outcomes, reflecting the unique stresses and pressures associated with individual-sport competition. Finally, IL-β shows an inverse relationship with HSP27 in tennis, suggesting an accelerated exit from the pro-inflammatory phase, a pattern not widely observed in the aerobic sports literature.

Regarding interventions, WBC is the only method in this review that directly influences cytokine levels and has definitive scientific evidence of a meaningful effect, producing a 60% reduction in TNF-α, a significant increase in IL-6 as a transition marker, and a significant decrease in CK. Additionally, the mixed-methods protocol consisting of active recovery, stretching, CWI, and massage had no effect on any measured biomarker, indicating it was as effective as passive rest over a five-day tournament. Given that MMR is one of the world's most commonly used recovery methods in professional tennis, this suggests that the timing and phase of intervention may have a greater effect on the inflammatory response than the choice of method itself. Applying an intervention without knowing which stage of the inflammatory cycle the athlete is in risks applying it at the wrong moment and producing little to no meaningful effect.

Several limitations constrain the conclusions drawn from this review. The scarcity of direct cytokine measurement data in tennis-specific recovery research, combined with inconsistencies across studies in participant age, timeframe, and methodology, restricts the ability to make definitive clinical recommendations. Many of the biomarker changes reported in Table 2 are also small in absolute magnitude, with several falling below 0.59% change. The cited studies enrolled between 8 and 15 participants, and these limited sample sizes likely reduced statistical power to reliably detect changes of this scale — readers should therefore interpret the smaller reported values with caution. Beyond sample size, the cited studies do not consistently report inclusion or exclusion criteria regarding athletes' baseline inflammatory health, leaving it unclear whether researchers screened for or excluded athletes with chronic inflammatory conditions. This matters because pre-existing chronic inflammation could elevate baseline biomarker levels and confound the interpretation of exercise-induced changes. Future researchers should

explicitly document participants' inflammatory health status, employ larger and more adequately powered samples, and adopt more methodologically consistent designs to confirm whether the trends identified here reflect true physiological signals or statistical noise.

Ultimately, managing inflammation in professional tennis is a matter of resolution. Suppressing inflammation too early disrupts the signaling necessary for proper, effective muscle repair, while allowing it to accumulate to extreme levels significantly increases the athlete's recovery burden over the course of a tournament. Resolution requires knowing exactly where in the inflammatory cycle the athlete is at any given moment, which is only possible through continuous blood-panel-based biomarker monitoring. Until recovery methods are designed around the inflammatory cycle rather than the clock, professional tennis players will continue to compete under a residual inflammatory load, which will hinder their performance, and current standard methods have proven largely unable to resolve. Future researchers should take these suggestions into consideration to directly maximize athletes' physiological health and recovery methodologies.

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