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Systematic Review & Meta-Analysis

Training and Tapering in High-Level Judo Athletes: A Biochemical and Autonomic Perspective

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Abstract

International Journal of Exercise Science 19(2): 2014, 2026. This study aimed to investigate the physiological, metabolic, and inflammatory responses to a four-week systematic training program followed by a 15-day tapering period in high-level judo athletes. Eighteen elite judokas (20.2 ± 2.4 years) were monitored throughout the training cycle. Internal training load was quantified using session rating of perceived exertion (sRPE). Autonomic regulation was assessed via heart rate variability coefficient of variation (HRVcv), while blood samples were analyzed for creatine kinase (CK), creatinine, albumin, total proteins, cortisol, and cytokines (IL-6, IL-10, TNF-α). Training load decreased progressively across the weeks ($p < 0.05$), confirming a tapering phase. HRVcv remained stable throughout. Post-training analyses revealed significant increases in CK ($p = 0.0002$), IL-10 ($p = 0.01$), and creatinine ($p = 0.05$), while total protein levels decreased ($p = 0.01$). HRVcv correlated positively with internal load and negatively with albumin and total proteins. Additional correlations were observed between cortisol and total proteins, CK and albumin, and TNF-α with both creatinine and uric acid. Despite initial training-induced stress, the tapering phase facilitated physiological adaptation, as evidenced by autonomic balance and an enhanced anti-inflammatory response. Integrating internal load metrics, HRV, and biochemical markers may improve training monitoring and recovery strategies in high-performance judo athletes.

Keywords: Heart rate variability, biochemical markers, inflammation, muscle damage, training load

Introduction

Judo is an intermittent, high-intensity combat sport that requires a combination of strength, power, endurance, and technical-tactical skills to achieve success in competition.[1] The sport involves repeated explosive movements, such as throws, takedowns, and groundwork techniques, interspersed with short recovery periods. Given its high physiological demands,

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judokas experience significant physical and metabolic stress, necessitating well-structured training programs to enhance performance and recovery.[2]

Systematic training programs are designed to optimize athletic performance while mitigating excessive fatigue and overtraining risks.[3] In elite sports, monitoring training load is crucial for ensuring that athletes experience positive physiological adaptations rather than maladaptive responses.[4] Subjective tools, such as the session rating of perceived exertion (sRPE),[5] and objective physiological markers, including autonomic, inflammatory, and metabolic responses, are essential for assessing the effects of training load.[6]

Heart rate variability (HRV) has emerged as a key marker of autonomic nervous system regulation and is widely used to assess training responses in athletes.[7] As a non-invasive tool, HRV provides insights into autonomic balance and recovery capacity, with indices such as the natural logarithm of the root mean square of successive differences (LnRMSSD) and its coefficient of variation are helpful for monitoring athletes' physiological states.[8] In high-intensity sports like judo, training can induce to HRV reductions, indicating autonomic imbalance and fatigue accumulation.[9] Understanding these responses is crucial in optimizing recovery strategies and adjusting training loads.

Beyond autonomic regulation, high-intensity training can induce transient increases in inflammatory markers, such as creatine kinase (CK), tumor necrosis factor-alpha (TNF-α), interleukins (IL) 6 and IL-10.[10,11] In fact, while CK is commonly used as a marker of muscle damage, elevations in the circulating levels of TNF-α and IL-6 could indicate a pro-inflammatory systemic status induced by the exercise training session.[12] Moreover, the balance between pro- and anti-inflammatory cytokines, such as IL-6 and IL-10, respectively, plays a corollary role in determining recovery and adaptation to exercise training.[13] Additionally, hormonal and metabolic markers, including cortisol, albumin, total proteins, and creatinine provide valuable insights into training-induced stress and physiological adaptations. Elevated cortisol levels reflect increased catabolic activity, which may negatively impact recovery and performance.[14] Variations in albumin, total protein, and creatinine levels can indicate changes in hydration status, protein metabolism, and renal function due to training intensity.[6]

In practice, training monitoring in judo has traditionally relied on a combination of subjective questionnaires, performance-based tests, and selected hormonal or biochemical measures, each capturing specific aspects of the training process.[9,15] Psychological tools and session rating of perceived exertion provide valuable information on internal load and perceived stress,[16,17] while performance tests are commonly used to track physical readiness across training phases.[18] However, when applied in isolation, these approaches may not fully capture the complex interaction between training load, recovery status, and physiological adaptation. Likewise, hormonal and biochemical markers offer important insights into training stress[15,19] but are constrained by sampling logistics and limited temporal resolution for day-to-day decision-making. In this context, the combined interpretation of internal load metrics with non-invasive autonomic indicators and complementary biochemical measures has been proposed as a more informative strategy to better understand training responses in elite judo athletes.

Despite the well-established importance of monitoring physiological and perceptual responses in elite athletes,[20] limited studies have examined how autonomic, inflammatory, and metabolic markers jointly respond to systematic training and tapering in elite judo.[9,21,22] Tapering is commonly defined as a planned and progressive reduction in training volume and/or intensity aimed at reducing accumulated fatigue, enhancing recovery processes, and facilitating performance expression prior to competition, rather than maximizing recovery per se.[21,22] Most available evidence has focused on isolated outcomes or endurance-based sports,[23] providing limited insight into the specific physiological demands of judo training. Consequently, the interaction between training load, recovery status, and multi-system physiological adaptation during planned training cycles remains insufficiently explored in this population.

Although the relevance of autonomic,[24] inflammatory,[25] and metabolic markers[11] for monitoring training responses is well established, their application in judo has largely remained fragmented, with most studies examining these variables in isolation or outside the context of systematic training and tapering. This approach limits the understanding of how autonomic regulation, muscle damage, inflammatory balance, and metabolic stress coexist and interact in response to planned variations in training load. Given the intermittent and high-impact nature of judo, mismatches between training stress and recovery may lead to persistent muscle damage, altered inflammatory profiles, and autonomic instability, even in highly trained athletes. Therefore, integrating internal load measures with heart rate variability and biochemical markers may provide a more comprehensive framework to capture training-induced stress and recovery dynamics, particularly during tapering phases, when physiological adaptations are expected to consolidate.

Therefore, the present study investigated the effects of four weeks of systematic training with 15-day tapering period on HRV and circulating levels of CK, TNF-α, IL-6, IL-10, cortisol, albumin, total proteins, and creatinine in high-level judokas. Additionally, were explored the associations between training load and RPE perception with physiological/metabolic/inflammatory markers to provide insights into optimizing training strategies for high-performance athletes.

Methods

This longitudinal quasi-experimental study monitored a group of judo athletes over four weeks, to assess the effects of a 15-day tapering period on physiological and biochemical parameters. Internal load was monitored to confirm the presence of tapering. Heart rate variability, a physiological indicator of recovery, was measured daily throughout the period.
Blood analyses included biomarkers of muscle damage (CK), inflammation (IL-6, IL-10, and TNF-alpha), hormones (cortisol) and metabolism (albumin, total proteins, creatinine, uric acid, glucose, triglycerides, cholesterol). Serum samples were obtained from blood samples collected before and after four weeks of training to investigate their associations with recovery during tapering.

The study was conducted in a high-performance team preparing for the annual main national competition. All athletes followed the same training model before and during the study.

Participants

The sample size was calculated using G*Power software, based on an ANOVA – repeated measures within factors model with one group and two measurements. A priori parameters were established, including $\alpha = 0.05$, power $(1-\beta) = 0.8$, correlation among repeated measures = 0.5, and an effect size of 0.35, derived from Campos et al,[9] which analyzed the HRV of judo athletes longitudinally (4 training weeks). The final sample included 18 high-level judo athletes (4 women and 16 men), black belts from different competitive levels, including 6 state-level, 5 national-level, and 7 international-level athletes (characteristics of subjects are in Figure 1). Used as an eligibility criterion to participate in the study, all participants had been competing at a high-performance level for a minimum of two years, were registered with the sport's national governing body, and were not receiving physical therapy that could hinder them from completing the full training load prescribed by the technical staff.

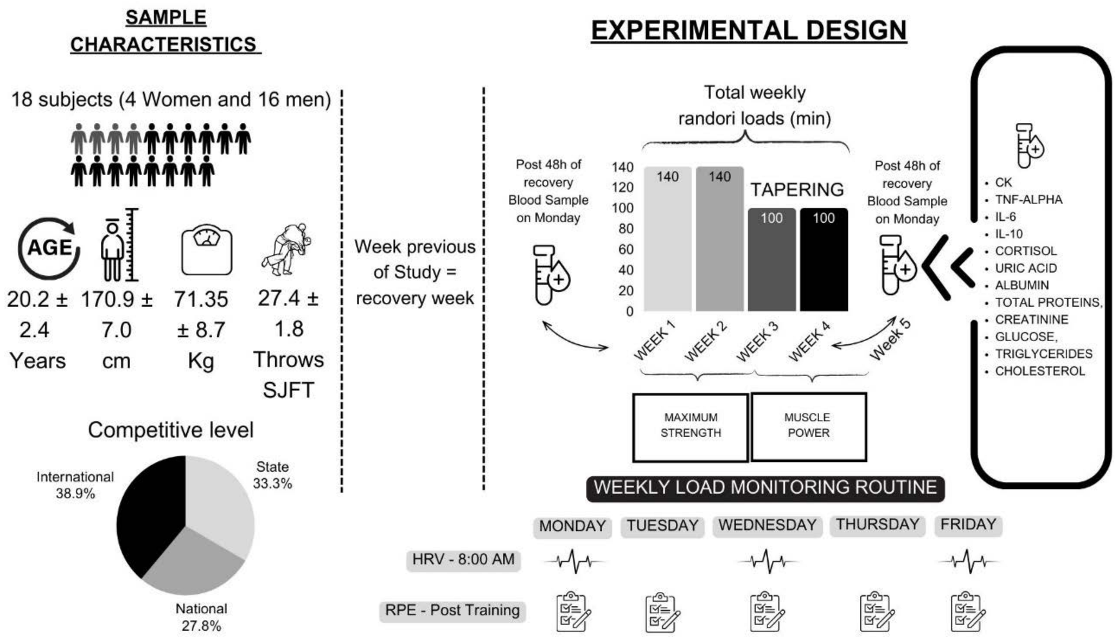

Figure 1. Experimental design. HRV – Heart Rate Variability. SJFT – Special Judo Fitness Test.

All procedures were approved by the Research Ethics Committee of Serviço Social da Indústria SESI-SP (protocol 51970721.7.0000.5435) and were conducted according to the regulations established in the Declaration of Helsinki. After receiving written instructions describing all the procedures, risks, and benefits related to the study, the subjects signed an informed consent form to participate in this study. This research was carried out fully in accordance with the ethical standards of the *International Journal of Exercise Science*.[26]

Protocol

Training Program.

Athletes completed two daily training sessions, with morning strength and conditioning on Monday, Wednesday, and Friday, and afternoon technical-tactical judo training from Monday to Saturday.

The four-week strength and conditioning program was divided into two phases. The first two weeks focused on maximum strength, following Franchini et al.[27] Athletes performed fundamental exercises—Bench Press, Bent-Over Row, Shoulder Press, Back Squat, Hip Thrust, and Barbell Lunges—in 4 sets of 3–5 repetitions at 80%–95% of 1RM. The final two weeks served as a tapering phase, emphasizing muscular power development.[28,29] Sessions included Olympic weightlifting exercises (Clean & Jerk, Snatch), ballistic movements (Medicine Ball Throw, Pull-Ups), and plyometric drills (Drop Jump, Jump Squat, Countermovement Jump), performed in 4 sets of 6 repetitions at 30%–70% of 1RM.

Technical and tactical training occurred five days per week. In the first two weeks, randori volume was higher (140 minutes/week) as it is the primary method for simulating competition. Sessions involved longer bouts (>5 min), more repetitions (>10 per session), and short rest intervals (1–2 min). In the final two weeks, randori volume was reduced (100 minutes/week), with shorter bouts (>2 min), fewer repetitions (6–10 per session), and longer rest intervals (3–4 min), required to characterize the tapering phase.

The organization of two daily training sessions reflects the habitual training routine of elite judo athletes, in which complete recovery between sessions is rarely attainable. Therefore, recovery management in the present study was primarily achieved through a planned tapering strategy rather than extended inter-session recovery periods. The substantial reduction in training volume and internal load during the final two weeks was designed to promote recovery compensation and physiological consolidation. This approach allowed the investigation of autonomic, biochemical, and inflammatory responses under ecologically valid training conditions, particularly during the tapering phase, when recovery processes are expected to be enhanced.

No standardized guidelines currently define minimum recovery requirements for elite judo training involving multiple daily sessions. Consequently, the recovery rationale adopted in this study was based on evidence from prior investigations in judo athletes. Studies have shown that creatine kinase concentrations typically peak 24–48 h after intense training sessions,[11,15,30] indicating that short inter-session recovery periods may promote cumulative muscle damage during dense training phases. In contrast, previous research has demonstrated that a 10–15-day tapering period is sufficient to elicit improvements in hormonal status and performance in judokas.[21] Therefore, the present experimental design intentionally combined a phase of high training density with a subsequent tapering period to investigate the behavior of autonomic, biochemical, and inflammatory markers during recovery consolidation.

Blood sampling.

Blood sample collection was performed exclusively on Mondays from 08:00 - 09:00 a.m., without fasting. All volunteers had their usual breakfast between 7:00 and 7:30 AM, were not following a weight loss regimen, and were instructed to maintain a consistent eating routine throughout the data collection period. The athletes should refrain physical activity by the last 48 hours. Samples were collected in tubes, that were centrifuged (10 min; 3500rpm; at 4°C), and the serum obtained was stored at -80°C until their use in the laboratory assessment.

Determination of biochemical, hormone, and inflammatory markers.

Circulating levels of creatine kinase (CK, a muscle damage marker), IL-6, IL-10, and TNF-α, (inflammatory cytokines), and cortisol (stress hormone) were determined using commercial kits for ELISA test (R&D Systems, Minneapolis, MN, USA), following the manufacturer´s instruction. Additionally, levels of creatinine, uric acid, albumin, total proteins as well as triglycerides, total cholesterol, and glucose were determined using colorimetric commercial kits (BioClin, Minas Gerais, Brazil), following the manufacturer´s instruction.

Heart Rate Variability (HRV).

The volunteers remained seated for five minutes and were instructed to stay calm, with normal breathing, in silence, and with minimal body movement. A heart rate monitor (Polar® H9, Kempele, Finland) was used to data acquisition with an acquisition frequency of 1000 Hz for 5 minutes, disregarding the first 2 minutes of analysis. This acquisition frequency is suggested for capturing the R-R interval.[31] The heart rate monitor was connected to a smartphone via Bluetooth and the HRV data was analyzed by the previously validated Elite HRV application.[31] HRV data were extracted from this application and recorded in a spreadsheet for further analysis. The present study only considered time domain analyses, registering and analyzing the root mean square of successive squared differences between adjacent RR (RMSSD). The RMSSD is assumed as an indicator of parasympathetic nervous system activity and is considered one of the best indexes for monitoring athletes.[32] This decision was based on evidence indicating that time-domain measures, particularly RMSSD, exhibit lower day-to-day variability and a superior signal-to-noise ratio compared with frequency-domain indices during short resting recordings in field-based settings.[33] Previous studies have shown that the coefficient of variation of RMSSD is substantially lower than that observed for spectral indices, such as the LF/HF ratio, which may present excessive variability and reduced sensitivity to training-induced adaptations.[33] Consequently, resting HR and time-domain HRV indices have been recommended as priority measures for athlete monitoring outside laboratory conditions. The RMSSD was transformed into its natural logarithm before analysis [LnRMSSD; following standards already adopted in the literature].[34] The weekly coefficient of variation LnRMSSD (LnRMSSDcv) for each athlete was also calculated from the ratio between the individual weekly standard deviation of LnRMSSD and the individual weekly average of LnRMSSD.[34] This parameter was used as a representative of HRV (HRV_{cv}).

Internal training load.

After complete each training session the athlete reported a value on the RPE-session scale that covers the range of 0-10, with the number 0 representing rest and the number 10 representing maximum effort. From this result, the Internal Training Load produced by the session was calculated and determined by the product between the number answered by the athlete on the scale and the training duration in minutes. The resulting value should be expressed in arbitrary units (A.U.).[35] Thus, the arbitrary units of each training session were recorded, and at the end of the week, the individual loads were added to follow the total internal load for the week.

Statistical Analysis

Before the inferential analysis, data normality and homoscedasticity were tested and confirmed with the Shapiro-Wilk and Levene tests. To identify potential outliers, the ROUT method was applied, detecting 1% of outliers in the sample.[36] Outliers were detected and removed from the sample in the variables (IL10 = 1, Creatinine = 2). Then, a one-way repeated-measures ANOVA was performed to analyze the internal load and HRV across the weeks. Bonferroni's *post-hoc* was used when necessary. A paired t-test was performed to compare pre- and post-blood measurements. Cohen's *d* effect size (*d*) was calculated to assess the magnitude of the difference between competitive levels. The *d* values were considered as trivial ($d < 0.2$), small ($0.2 < d < 0.6$), moderate ($0.6 < d < 1.2$), and large ($d \geq 1.2$) (ref).

Percentage changes in biochemical and physiological variables (defined as Δ = (post-training – Pre-training) / Pre-training) were calculated for the correlation analysis. Pearson's correlation coefficient analysis was used to assess the associations between biomarkers and internal load. The correlation magnitude was classified from the value of r, with values <0.3 considered small, 0.3 - 0.5 moderate, 0.5 - 0.7 high,> 0.7 very high.[37,38]

To analyze the associations between the selected physiological and biochemical variables, a chord diagram was generated based on the Pearson´s correlation coefficients analysis (R) and their respective significance values (p-values). The correlation matrix was computed to quantify the strength and direction of the associations between variables. A threshold of $p < 0.05$ was used to determine statistical significance, allowing for the inclusion of trends that approached significance. The chord diagram was constructed using network analysis, where each node represents a variable, and the edges (connections) represent the correlations between them. These exclusively statistical analyses (Chord Diagram) and visualizations were conducted using the statistical R program.

All other data were analyzed and plotted using the statistical package Graph Pad Prism™ (version 5.0, GraphPad Software, San Diego, CA, USA). A significance level set at $p \leq 0.05$ was used in all inferential analyzes.

Results

Internal load (A.U.) was different over time ($F = 17.75$; $p < 0.0001$; $\eta^2 = 0.52$, Figure 2). Internal load (A.U.) in week 1 was greater than in week 2 (Week 1: 8250 ± 1031 A.U; Week 2: 7216 ± 1416 A.U.; $p = 0.02$), week 3 (5989 ± 1434 A.U.; $p < 0.0001$), and week 4 (5876 ± 2159 A.U.; $p = 0.0002$). Week 2 was greater than week 3 ($p = 0.01$). Week 3 and Week 4 were not different between them ($p = 0.99$). Thus, there was a significant reduction in the internal load, characterizing a tapering period. The HRV_{CV} did not differ between weeks ($F = 0.697$; $p = 0.514$; $\eta^2 = 0.04$; Week 1: 6.21 ± 4.17 %; Week 2: 7.50 ± 4.76 %; Week 3: 9.43 ± 5.75; Week 4: 9.53 ± 6.47).

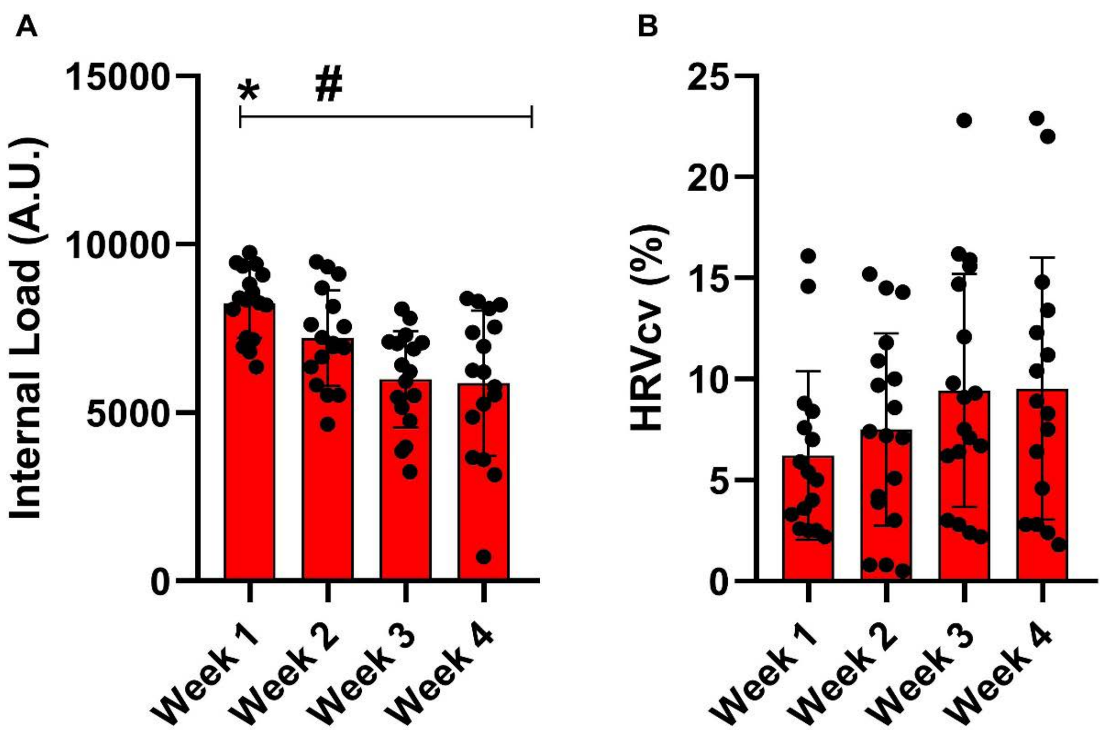

Figure 2. Internal Load and HRVcv. **Panel A**: Internal Load - * Week 1 was greater than in week 2 (p = 0.02), week 3 (p < 0.0001), and week 4 (p = 0.0002). #Week 2 was greater than week 3 (p = 0.01).

Figure 3 shows the data obtained in the blood biomarkers analysis. Higher circulating CK concentrations (p = 0.0002, d = 1.09), and IL-10 (p = 0.01, d = 0.75) were evidenced post-training than pre. Otherwise, while levels of TNF-α (p = 0.06, d = 0.46) and IL-6 (p = 0.13, d = 0.37), as well as the ratio between systemic levels of the pro- (IL-6) and anti-inflammatory (IL-10) cytokines did not show statistically significant difference between the time points here (p = 0.08, d = 1.89). Other metabolic variables not showed significant effects, cortisol (p = 0.70, d = 0.09), uric acid (p = 0.22, d = 0.30); albumin (p = 0.66, d = 0.10); triglycerides (p = 0.28, d = 0.26); glycemia (p = 0.35, d = 0.22); and total cholesterol (p = 0.12, d = 0.38). Except for total proteins, in which significant reductions were identified post-training (p = 0.01, d = 0.67), and creatinine which increased when compared to the baseline values (p = 0.05, d = 0.55).

Some significant correlations were identified (Figure 4). The HRVcv correlated positively with internal load (r = 0.46, p = 0.04) and negatively with variation in albumin (r = -0.47, p = 0.04) and total proteins (r = -0.59, p = 0.01). Furthermore, cortisol positively correlated with total proteins (r = 0.61, p = 0.009), CK positively correlated with albumin (r = 0.53, p = 0.03); creatinine negatively correlated with uric acid (r = -0.62, p = 0.008); and TNF-α positively correlated with uric acid (r = 0.56, p = 0.01) and creatinine (r = 0.92, p = 0.01).

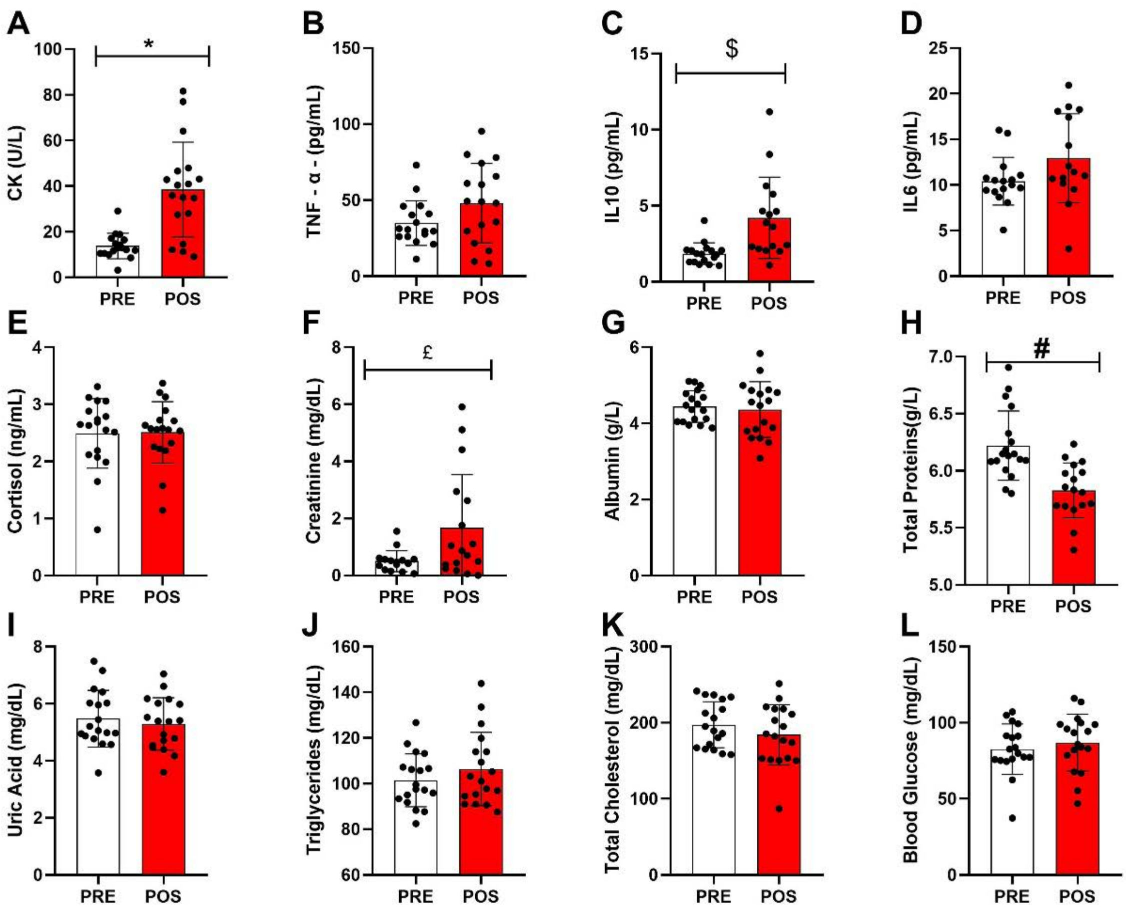

Figure 3. Biochemical and physiological variables. **Panel A** - Creatine Kinase (CK): * significant differences (PRE: 13.78 ± 5.569 U/L; POST: 38.51 ± 20.79 U/L; t = 4.611, df = 17, p = 0.0002, d = 1.09). **Panel B** - TNF-α (PRE: 38.78 ± 21.41 pg/mL; POST: 52.39 ± 31.39 pg/mL; t = 1.969, df = 17, p = 0.06, d = 0.46). **Panel C** – IL10: $ significant differences (PRE: 1.85 ± 0.71 pg/mL; POST: 4.20 ± 2.67 pg/mL; t = 2.908, df = 17, p = 0.01, d = 0.75). **Panel D** - IL-6 (PRE: 12.43

± 6.63 pg/mL; POST: 19.08 ± 16.71 pg/mL; t = 1.584, df = 17, p = 0.13, d = 0.37). **Panel E** – Cortisol (PRE: 2.49 ± 0.60 ng/mL; POST: 2.51 ± 0.53 ng/mL; t = 0.3809, df = 17, p = 0.70, d = 0.09). **Panel F** – Creatinine: £ significant differences (PRE: 0.500 ± 0.369 mg/dL; POST: 1.661 ± 1.878 mg/dL; t = 2.115, df = 17, p = 0.05, d = 0.55). **Panel G** – Albumin (PRE: 4.44 ± 0.41 g/L; POST: 4.36 ± 0.73 g/L; t = 0.4357, df = 17, p = 0.66, d = 0.10). **Panel H** – Total Proteins: # significant differences (PRE: 6.22 ± 0.30 g/L; POST: 5.82 ± 0.24 g/L; t = 2.859, df = 17, p = 0.01, d = 0.67). **Panel I** – Uric acid (PRE: 5.476 ± 0.994 mg/dL; POST: 5.291 ± 0.919 mg/dL; t = 1.252, df = 17, p = 0.22, d = 0.30). **Panel J** – Triglycerides (PRE: 101.4 ± 11.58 mg/dL; POST: 106.4 ± 16.08 mg/dL; t = 1.113, df = 17, p = 0.28, d = 0.26). **Panel K** – Total cholesterol (PRE: 197.4 ± 30.19 mg/dL; POST: 184.0 ± 39.52 mg/dL; t = 1.593, df = 17, p = 0.12, d = 0.38). **Panel L** – Blood glucose (PRE: 82.65 ± 16.57 mg/dL; POST: 86.87 ± 18.69 mg/dL; t = 0.9479, df = 17, p = 0.35, d = 0.22).

The evaluated correlations indicate that HRV exhibited the strongest associations (Figure 5), showing one positive correlation with Internal Load and two negative correlations with Total Proteins and Albumin. Cortisol was associated with Total Proteins but were not correlated with HRV. Regarding muscle damage, CK was correlated with Albumin but not with HRV. These findings suggest a potential interaction between Load, Autonomic Response, Metabolic Stress, and Hormonal Response.

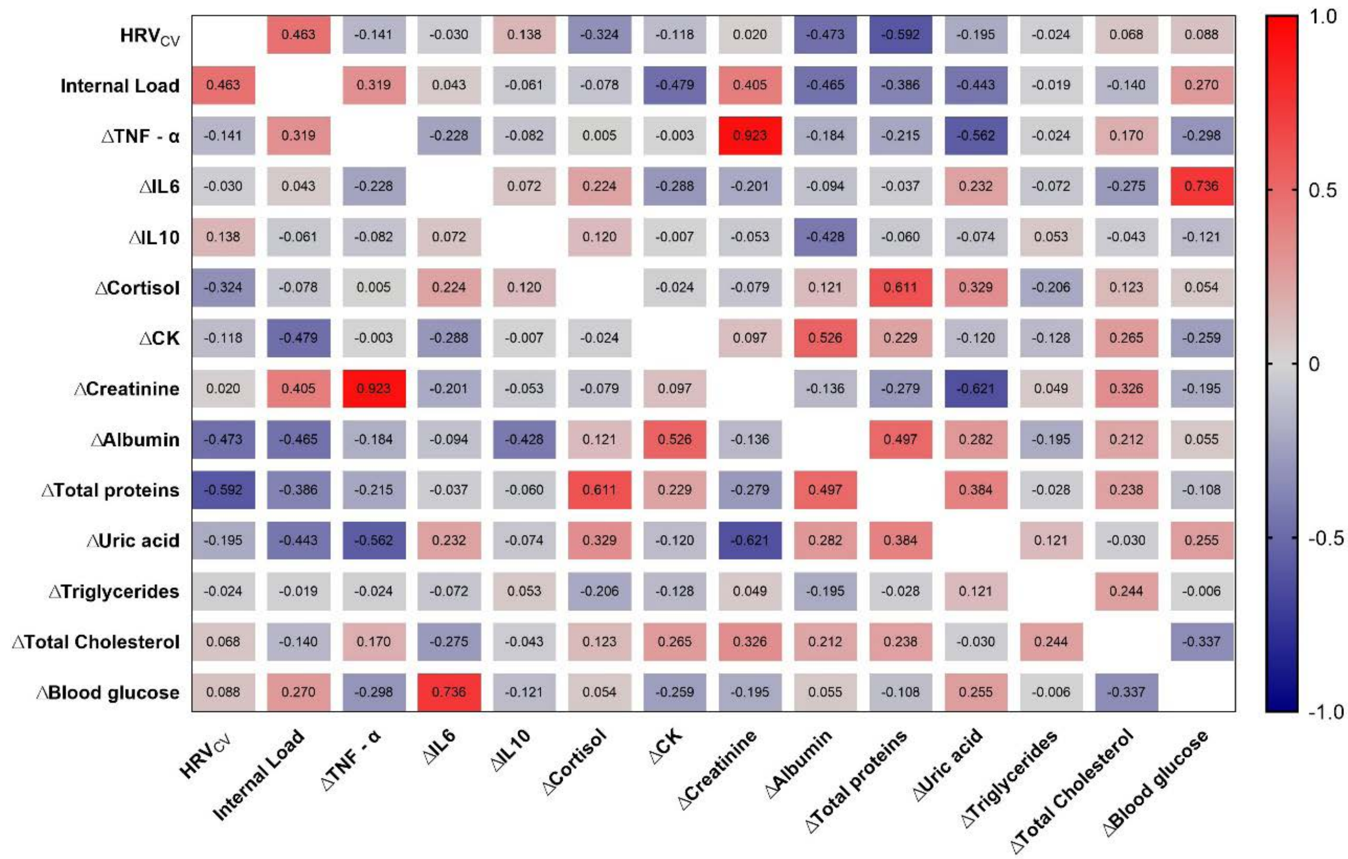

Figure 4. Heatmap representing the correlations between biochemical variables, internal load, and heart rate variability HRV_{CV}. The colors indicate the direction and magnitude of the correlations, with red tones representing positive correlations and blue tones indicating negative correlations. The quadrant contains the corresponding Pearson correlation values. The analyzed variables include inflammatory biomarkers (ΔTNF-α, ΔIL-6, ΔIL-10), hormonal markers (ΔCortisol), metabolic markers (ΔCreatinine, ΔAlbumin, ΔTotal Proteins, ΔUric Acid, ΔTriglycerides, ΔTotal Cholesterol, ΔBlood Glucose), and a muscle damage marker (ΔCK), correlated with HRV_{CV} and internal training load.

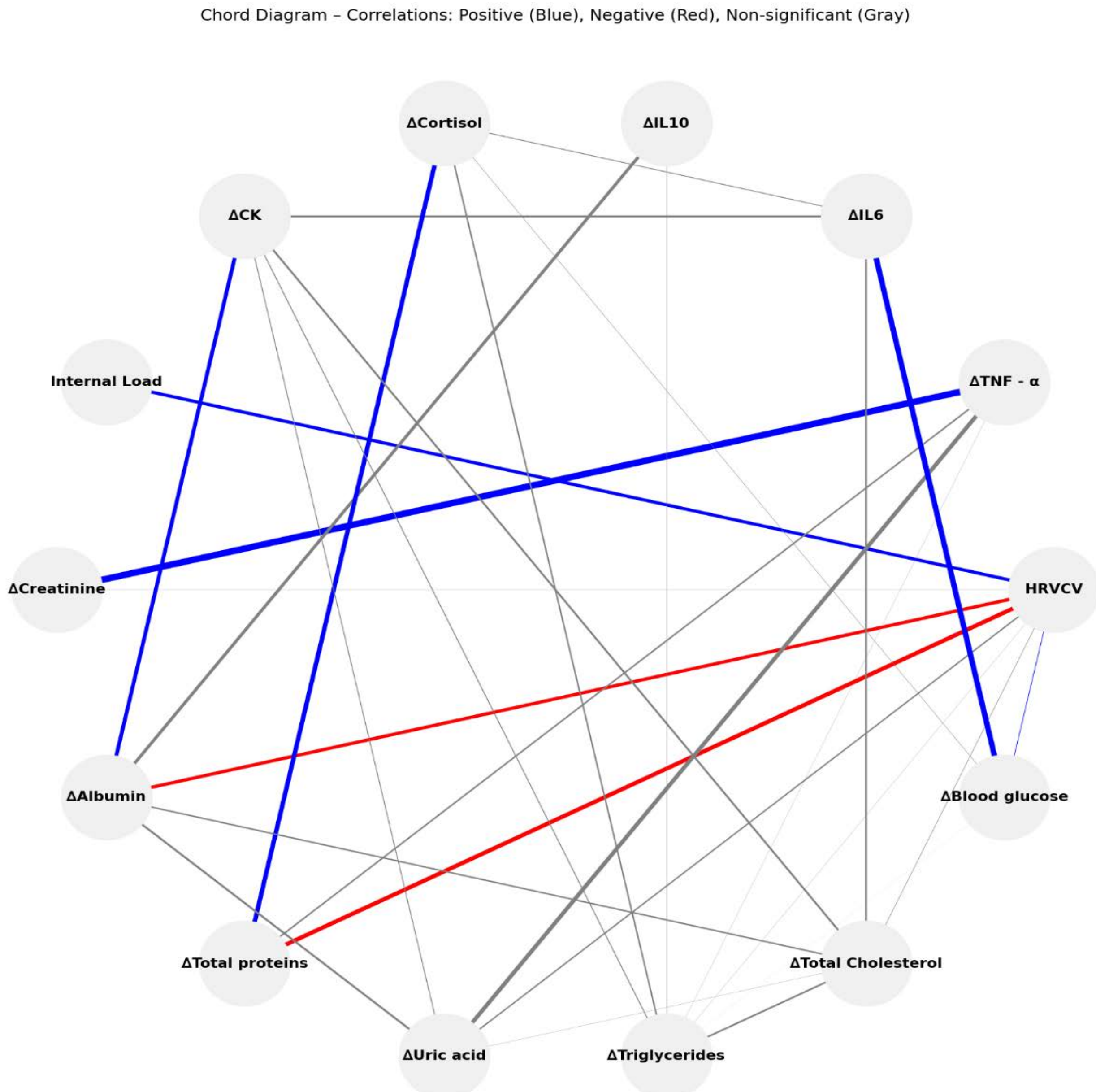

Figure 5. Chord diagram representing the significant correlations ($p<0.05$) between biochemical variables, internal load, and heart rate variability (HRV_{CV}). The blue lines indicate significant positive correlations, while the red lines represent significant negative correlations. The node size reflects the relative importance of the variables within the correlation network. The analyzed variables include inflammatory markers (ΔTNF-α, ΔIL-6, ΔIL-10), hormonal markers (ΔCortisol), metabolic markers (ΔCreatinine, ΔAlbumin, ΔTotal Proteins, ΔUric Acid, ΔTriglycerides, ΔTotal Cholesterol, ΔBlood Glucose), and a muscle damage marker (ΔCK), demonstrating their interrelationships with HRV_{CV} and internal training load.

Discussion

The aim of the present study was to investigate the effects of four weeks of systematic training with 15-day tapering period on HRV and circulating levels of CK, TNF-α, IL-6, IL-10, cortisol, albumin, total proteins, and creatinine in high-level judokas. In general, the main results obtained in the present study revealed that (i) both sRPE and total protein levels decrease throughout the training progress accordingly to the planning, while (ii) circulating levels of CK, IL-10, and creatinine were higher at post-training time point than before (baseline values). In an interesting way, (iii) HRVcv was positively associated with internal load and negatively correlated with albumin and total protein levels, besides (iv) cortisol correlated positively with total proteins, while (v) CK was positively associated with albumin, and (vi) TNF-α demonstrated strong positive correlations with creatinine and uric acid. These findings are discussed in light of previously reported physiological responses to training stress and recovery in combat sports.

The training load was structured in a block format, with a reduction in randori load during the final two weeks. The internal load responses aligned with the planned progression, as evidenced by a significant decrease in internal load over the weeks. This adaptation is an expected response to the chronic prescription of training load.[20] While external load measurement provides insights into the work performed and the athlete's capabilities, internal load measurement is essential for determining the appropriate stimulus for optimal biological adaptation.[39]

Heart rate variability (HRVcv) fluctuated within normal limits throughout the training period, a response consistent with previous observations in well-trained athletes exposed to progressive training load reduction.[40] According to the literature, high internal loads typically result in a significant increase in HRVcv, reflecting greater autonomic disturbance due to fatigue.[9] In this study, the absence of significant HRVcv changes was expected, given the progressive reduction in training load over the weeks.

Muscle damage was inferred from sustained elevations in circulating creatine kinase, which is widely used as an indirect marker of exercise-induced muscle damage rather than a direct indicator of structural injury, in agreement with studies reporting sustained elevations of CK following periods of high training density in judo athletes.[11,15,30,41] Despite implementing a 15-day tapering phase, it may not have been sufficient to fully mitigate the muscle damage accumulated during the first two weeks. Some studies suggest that 1 to 2 weeks of tapering can significantly reduce CK levels.[23,42] Importantly, the magnitude of CK elevation observed here is consistent with mild-to-moderate muscle damage typically reported in high-level combat athletes during periods of increased training density.[11,15,30,41] However, we cannot rule out the possibility that the training load in the final two weeks was still enough to induce muscle damage, even with the reduction in internal load and the absence of autonomic disturbances.

Given the significant increase in muscle damage, a rise in pro-inflammatory cytokines (TNF-α, IL-6) was expected as part of the cellular repair process. In fact, the presence of pro-inflammatory cytokines, such as TNF-α play a key role stimulating the production of IL-6 and growth factors that trigger satellite cell proliferation and reconstruction of muscle fibers.[25] However, here, the circulating levels of these pro-inflammatory cytokines were unchanged. On the other hand, the systemic levels of the anti-inflammatory cytokine IL-10 increased post-training, which corroborates previous evidence describing the anti-inflammatory effects of repeated high-intensity exercise[12,43,44] but also may reflect an adaptive inflammatory response to training stress.[45,46] In agreement with these suggestions, a recent meta-analysis reported that training induces a decrease in pro-inflammatory cytokines and an increase in anti-inflammatory cytokines,[47] and also it is well-known that skeletal muscle functions as an endocrine organ by producing and releasing myokines during contraction, mediating metabolic changes and promoting an anti-inflammatory response.[12] Although the statistical analysis did not show significant differences, the tendency of reduction of IL-6/IL-10 ratio ($p=0.08$) found here not only reinforces our suggestions since it has been suggested that the ratio between pro- and anti-inflammatory cytokines analysis can provide more precise information regarding the individual´s (un)balance of systemic inflammatory status,[48,49] but also demonstrated that a short time of training (4 weeks) can be useful to promote a balanced inflammatory status. Hence, the significant increase in anti-inflammatory cytokine IL-10 indicates that the physiological systems effectively responded to muscle damage and initiated the recovery process anticipated during the tapering phase.

Metabolic biomarkers—including uric acid, albumin, triglycerides, blood glucose, and cholesterol— showed no significant post-training differences. However, an increase in creatinine and reduction in total serum protein post-training was identified. Judo combat has been shown to disrupt metabolic responses by increasing creatinine, uric acid, triglycerides, and cholesterol levels, likely due to glycogen depletion and increased lipid and protein metabolism.[50] Creatinine is a byproduct of phosphocreatine degradation in muscle and is typically produced at constant rate by the body. The increased creatinine excretion observed in this study may reflect a 'washout' effect, as previously described following intense exercise-induced changes in membrane permeability.[51] Some studies suggest that oxidative stress accelerates plasma protein degradation, particularly albumin, leading to lower total protein levels.[52] Overall, this study demonstrated an increase in muscle damage, creatinine, reduction in total serum protein, and enhanced anti-inflammatory response, this may suggest greater metabolic stress, but it appears to have been well managed physiologically by the athletes throughout the four-week training period. Taken together, the concomitant elevation in CK, creatinine, and IL-10, alongside preserved autonomic modulation, suggests a coordinated physiological response in which muscle damage, metabolic stress, and inflammatory regulation coexist within an adaptive, non-pathological range.

Correlational analyses provided integrative insights into the associations between training load and physiological responses to training. HRVcv showed a positive correlation with total internal load, suggesting that greater autonomic variation is linked to higher perceived load.[7] Conversely, HRVcv was negatively correlated with albumin and total protein levels, indicating that greater autonomic disturbance may be associated with reduced serum protein availability, possibly due to oxidative stress.

A positive association between cortisol and total serum protein explained 37.2% of protein level variation, suggesting that increased plasma protein availability may heighten oxidative stress, triggering a cortisol response.[53] However, in this study, total serum protein levels decreased post-training, while cortisol remained unchanged, reinforcing a positive adaptation to training.

Additionally, CK and albumin were positively correlated, with 28.0% of CK variation explaining albumin fluctuations, likely reflecting oxidative stress-related muscle damage.[50,54] Lastly, TNF-α and uric acid exhibited a negative association, suggesting that athletes with higher TNF-α levels had lower uric acid concentrations, possibly indicating efficient oxidative stress regulation due to their high training status.[55]

The observed correlations suggest a complex interplay between training load, autonomic response, metabolic stress, and hormonal regulation. The strong associations of HRV—positively correlated with Internal Load and negatively with Total Proteins and Albumin—align with previous research indicating that increased training stress can influence autonomic function and metabolic markers. The absence of a correlation between HRV and CK suggests that while autonomic regulation responds to training load, muscle damage markers may follow a different time course or be influenced by other factors, such as individual recovery capacity. Additionally, the association between Cortisol and Total Proteins, but not HRV, reinforces the idea that hormonal responses to training stress may be partially independent of autonomic fluctuations. The correlation between CK and Albumin further suggests that metabolic and muscle damage responses are interconnected. These findings highlight the need for a multifaceted approach to monitoring training adaptations, as different physiological systems may respond uniquely to variations in training load.

This study provides relevant contributions to training load monitoring in high-performance athletes, integrating physiological and biochemical assessments over a four-week period. The longitudinal design and inclusion of multiple variables, such as internal load, HRV, and metabolic and inflammatory biomarkers, offer a comprehensive perspective on training adaptations. Additionally, the elite-level sample enhances the applicability of these findings in competitive settings. However, a limitation of the present study is the exclusive use of time-domain HRV indices, which precludes a more detailed characterization of sympathetic–parasympathetic interactions that could be obtained through frequency-domain or non-linear analyses. Other limitations should be noted, including the absence of a control group, the exclusive focus on male athletes, and the lack of external factor monitoring, such as sleep and nutrition, which could influence physiological responses. Given the observational design and absence of a control group, all physiological interpretations are grounded in established literature and should be interpreted as associative rather than mechanistic. Despite these limitations, the findings offer practical insights to inform training monitoring practices and support evidence-based decision-making in high-performance judo. By describing the behavior of autonomic, metabolic, and inflammatory markers across systematic training and tapering, the present results may help contextualize training-induced stress and recovery dynamics, particularly in elite athletes. Future studies should expand upon these findings with longer follow-up periods, comparative designs, and more diverse samples to further elucidate training adaptations in combat sports. Nevertheless, within its methodological framework, this study contributes to a better understanding of physiological responses to training in judo. From a practical perspective, the present findings suggest that fluctuations observed in HRV and internal load measures can reflect underlying physiological responses similar to those demonstrated in this study. Therefore, even when biochemical monitoring is not feasible on a routine basis, HRV and sRPE may provide complementary information to support interpretation of athletes' training responses. Nonetheless, these interpretations should be made with appropriate caution, considering the study's limitations and avoiding excessive extrapolation beyond the specific context investigated.

The integration of internal load monitoring, HRV, and biochemical markers throughout a well-planned systematic training demonstrated that, despite an initially high training demand, judo athletes exhibited effective adaptations, maintaining autonomic balance and activating repair mechanisms, such as increased IL-10 in response to muscle damage, as indicated by elevated CK levels. Additionally, correlation analyses revealed that variations in HRVcv are associated with perceived exertion, showing a positive correlation with internal load and a negative correlation with albumin and total protein levels, suggesting that fluctuations in autonomic regulation may impact the availability of these markers. The positive correlation between cortisol variations and total protein, as well as between CK and albumin, along with the strong associations among TNF-α, creatinine, and uric acid, reinforces the interconnectedness between inflammatory processes and metabolic responses during training. This integrated approach enhances training individualization, enables early overload detection, and supports optimized recovery strategies, ultimately promoting sustainable and safe long-term competitive performance.

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