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Research Article

Black seed oil massage and post-exercise inflammatory responses in male elite taekwondo athletes

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Abstract

The present study aimed to compare the effects of three weeks black seed oil massage on serum concentrations of IL-6, IL-10, CRP, and CPK in elite male taekwondo athletes. Fifteen elite male taekwondo athletes participated in this quasi experimental study. Three intervention groups were included: massage, black seed oil, and massage combined with black seed oil. No true control group (receiving neither intervention) was included, which represents a major limitation of the study design. All interventions were administered once weekly for three consecutive weeks. Participants in the massage group received a 30 45 min Swedish massage; the black seed oil group underwent topical application of 5 10 mL of black seed oil; and in the combined intervention group, Swedish massage was performed using black seed oil as the massage medium. Statistical analysis demonstrated a significant main effect of time for both IL-6 and IL-10($p \leq 0.001$). A significant group×time interaction was observed for these two inflammatory markers($p \leq 0.007$), although the overall between-group main effect was not statistically significant. Bonferroni revealed that at the first week post-test, serum IL-6 and IL-10 levels were significantly higher in the black seed oil group than in the massage plus black seed oil group ($p \leq 0.05$). Furthermore, within-group analyses showed that IL-6 and IL-10 levels significantly increased from baseline to all subsequent post-intervention measurements in all three groups ($p \leq 0.05$). The lower IL 6 and IL 10 response observed in the combined massage and black seed oil group during the first week is an isolated exploratory finding that may reflect Type I error or chance variation; it should not be interpreted as evidence of clinically relevant modulation. Given the absence of a true control group and the severely underpowered sample (N=12, 4 per group), these results should be considered preliminary and hypothesis generating.

Key Words: Nigella sativa; Massage; Taekwondo; Cytokines; Interleukin-6; Interleukin-10

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Introduction

Elite performance in combat sports, particularly in taekwondo, requires repeated exposure to high intensity training sessions that impose substantial physiological and mechanical stress on the athlete's body. Such demanding training loads are frequently accompanied by exercise induced muscle damage (EIMD) and a transient systemic inflammatory response, both of which are considered inevitable consequences of repeated maximal or near maximal efforts (Chen, Liao et al. 2017, Peake, Neubauer et al. 2017). The nature of taekwondo practice characterized by explosive kicking maneuvers, rapid changes of direction, repetitive jumping actions, and powerful concentric eccentric muscular contractions places considerable strain on skeletal muscle fibers. As a result, disruption of sarcolemmal membranes, ultrastructural microtrauma, and leakage of intracellular proteins and enzymes into the bloodstream commonly occur, among which creatine phosphokinase (CPK) is recognized as one of the most sensitive biochemical indicators of muscle membrane disturbance and exercise related tissue damage (Brancaccio, Lippi et al. 2010).

Beyond structural muscle disruption, strenuous athletic training also initiates a highly coordinated immunological response designed to facilitate tissue repair, metabolic adaptation, and restoration of homeostasis. This process is accompanied by the release of several inflammatory mediators, particularly cytokines that exert both pro-inflammatory and anti-inflammatory actions. Among these, IL6 is widely regarded as an early responsive cytokine that rises sharply following intense muscular work and contributes to both inflammatory signaling and energy substrate regulation, whereas IL10 serves as a compensatory anti-inflammatory cytokine involved in limiting excessive immune activation and promoting recovery (Kashani, Keshavarz et al. 2022). In parallel, CRP, an acute phase reactant synthesized by the liver in response to cytokine stimulation, has been extensively used as a systemic marker reflecting the magnitude of post-exercise inflammation in trained individuals. Monitoring fluctuations in these biochemical

variables therefore provides valuable insight into the athlete's recovery status, inflammatory burden, and readiness for subsequent performance (Trexler, Eagle et al. 2025).

Given the critical role of recovery in preserving competitive performance, minimizing cumulative fatigue, and reducing the risk of overreaching, sports scientists and practitioners have increasingly focused on interventions capable of attenuating post-exercise inflammatory disturbances. Among the available modalities, massage therapy remains one of the most commonly implemented recovery techniques in elite sport settings due to its practicality, low cost, and widespread athlete acceptance. Systematic evidence indicates that post-exercise massage may contribute to reductions in perceived soreness, circulating muscle damage markers, and selected inflammatory mediators, potentially through enhanced local blood flow, stimulation of lymphatic return, reduction of tissue edema, and modulation of immune cell trafficking within damaged muscle. Moreover, mechanistic investigations have shown that massage can attenuate nuclear inflammatory signaling pathways and influence molecular responses associated with muscle regeneration, suggesting that its benefits may extend beyond simple mechanical relaxation (Poppendieck, Wegmann et al. 2016). Swedish massage, characterized by its application of effleurage, petrissage, friction, tapotement, and vibration techniques, has been hypothesized to attenuate inflammation and muscle soreness through multiple physiological mechanisms, including enhanced lymphatic drainage, improved local circulation, reduced tissue edema, and modulation of neutrophil infiltration into damaged muscle tissue (Sinaga, Saleh et al. 2026). While the precise molecular mechanisms underlying massage-induced recovery remain incompletely elucidated, emerging evidence suggests that massage therapy may influence inflammatory signaling pathways and cytokine expression patterns in exercised skeletal muscle (Crane, Ogborn et al. 2012). However, the existing literature presents conflicting findings regarding the efficacy of massage in modulating systemic inflammatory markers, with some studies reporting beneficial effects on cytokine profiles while others demonstrate minimal or transient alterations (Ntoumas, Karatzaferi et al. 2025). These inconsistencies highlight the need for further investigation into the potential synergistic effects of combining massage with complementary therapeutic agents that possess intrinsic anti-inflammatory properties.

Despite these promising observations, the literature remains inconclusive regarding the consistency of massage induced changes in circulating inflammatory biomarkers, as several investigations have reported only modest or short lived physiological effects. This inconsistency suggests that massage alone may not always be sufficient to generate a robust anti-inflammatory response, particularly in highly trained athletes exp-osed to repeated strenuous workloads (Kooti, Hasanzadeh-Noohi et al. 2016). Consequently, there is growing interest in combining traditional manual recovery approaches with bioactive natural compounds possessing antioxidant and anti-inflammatory potential in order to enhance therapeutic efficacy. In this regard, black seed oil (Nigella sativa), some medicinal plant extract rich in thymoquinone and other phenolic constituents, has attracted increasing scientific attention because of its documented anti-inflammatory, immunomodulatory, and tissue protective properties (Chatterjee, Saha et al. 2025). The concurrent application of massage and black seed oil may therefore provide a dual action recovery strategy by integrating the circulatory benefits of manual manipulation with the biochemical effects of a phytotherapeutic agent. However, evidence regarding the influence of such a combined intervention on exercise induced inflammatory responses in elite combat athlete's remains scarce, warranting further empirical investigation. Importantly, the present study employs a quasi-experimental design without a true control group; thus, causal inferences regarding intervention effectiveness must be made with caution, and observed changes may partly reflect normal training related adaptations.

Materials and Methods

Study design

The present study was conducted using a quasi-experimental design with a pretest and multiple posttest assessments to evaluate the longitudinal effects of three distinct recovery interventions over a three week training period. This design lacks a true control group that receives neither massage nor black seed oil, which limits our ability to attribute observed biomarker changes specifically to the interventions as opposed to normal training adaptations, circadian variations, or placebo effects. Participants were allocated into three intervention groups to facilitate between group comparisons, but the absence of a nonintervention control group must be acknowledged as a principal methodological limitation. An additional major limitation is the small sample size (N=12 final, 4 per group), which renders the study severely underpowered to detect meaningful differences. The findings should therefore be considered exploratory and hypothesis generating. All measurements were conducted at standardized and predetermined time points to ensure the reliability and validity of the data collected throughout the research process.

Ethical Considerations

This research was conducted in full compliance with the ethical standards outlined in the Declaration of Helsinki and its subsequent amendments. The study protocol received formal approval from the Ethics Committee of the University of Tehran, International Campus, under approval number (Ethics Code: IR.

UT.PSYEDU.REC.1404.075). All potential participants received comprehensive verbal and written information regarding the study objectives, procedures, potential risks, and benefits prior to enrollment. Written informed consent was obtained from each participant, or from their legal guardians in the case of minors, before any study-related procedures were initiated. Participants were explicitly informed of their right to withdraw from the study at any time without providing justification and without facing any consequences or prejudice to their relationship with the Taekwondo Federation. All personal data were anonymized using unique participant codes to ensure confidentiality, and access to identifiable information was restricted to the principal investigator. The study procedures were designed to minimize any potential discomfort or risk to participants, and all interventions were administered by qualified professionals in a safe and supportive environment. Also all required permissions were obtained.

Participants and sampling procedure

To address the need for comprehensive participant characterization, we collected detailed demographic, training history, and competitive experience data. All participants were active members of the Iranian national taekwondo team with a minimum of three years of systematic training experience. The mean training history across all participants was 5.3±2.1 years (range: 3–9 years).

The statistical population of this study comprised elite male taekwondo athletes who were active members of the national team. Following Morgans table guidelines for sample size estimation in experimental research, a total of 15 eligible athletes were initially selected through voluntary participation and were subsequently allocated into three experimental groups of five participants each, consisting of a massage only group, a black seed oil-only group, and a combined massage with black seed oil group. It should be noted that each group experienced one participant dropout during the course of the intervention period, resulting in a final analytical sample of 12 participants who successfully completed all phases of the study protocol. The participant's age range was deliberately restricted to 17 to 22 years to ensure physiological homogeneity and to control for age-related variations in inflammatory and recovery responses to exercise and intervention (Figure 1).

It is critically important to acknowledge that the final analytical sample of N=12 (4 per group) is severely inadequate for detecting meaningful differences between groups. No formal a priori power analysis was conducted to justify the sample size; the use of "Morgan's table guidelines" does not substitute for a proper power calculation based on expected effect sizes and desired statistical power. With only four participants per group, the study is severely underpowered to detect any but the most massive eff--ects. The effect sizes observed for non-significant findings (ES=0.033-0.066) suggest that the study lacked sensitivity to detect clinically meaningful differences. Consequently, all findings should be interpreted as preliminary pilot data requiring confirmation in larger-scale investigations. The training protocol comprised standardized taekwondo-specific sessions conducted three times per week throughout the three-week intervention period, for a total of nine training sessions. All training sessions were supervised by the same certified national team coaching staff and were identical in structure, intensity, volume, and content across all three intervention groups to ensure that the only systematic difference between groups was the recovery intervention received.

Each training session lasted approximately 120 minutes and followed a consistent structure: (1) a 15-minute warm-up consisting of light jogging, dynamic stretching, and mobility exercises; (2) 45 minutes of technical skill development, including fundamental kicks (roundhouse, side kick, back kick, and hook kick), combinations, and footwork drills performed at moderate to high intensity; (3) 30 minutes of sparring drills, including situational sparring (offensive and defensive scenarios) and full-contact controlled sparring consisting of three 3-minute rounds with 1-minute rest intervals; (4) 20 minutes of conditioning work, including plyometric exercises (box jumps, depth jumps, and hurdle hops), agility drills (cone drills, ladder drills), and high-intensity interval running (10×100-meter sprints with 30-second recovery); and (5) a 10-minute cool-down consisting of light stretching and breathing exercises. The intensity of training was monitored using the Rating of Perceived Exertion (RPE) scale, with target RPE values of 15–18 (hard to very hard) during the technical and sparring components, and 16–19 during conditioning work. Heart rate monitoring was conducted on a subset of participants (n=3 per group) using wrist-worn heart rate monitors to confirm that training loads were comparable across groups; mean heart rate during the high-intensity portions of training ranged from 165 to 185 beats per minute, with no significant differences between groups.

All training sessions were standardized to the same time of day (4:00–6:00 PM) to control for circadian influences on physiological responses. Participants were instructed to maintain their usual training habits outside the prescribed sessions, and no additional structured exercise was permitted. Compliance with the prescribed training protocol was confirmed through attendance records and coach verification; all participants who completed the study attended 100% of the scheduled training sessions.

Expanded dietary control information

No formal dietary logs, calorie counts, or nutritional controls were implemented in this study. Participants were instructed to

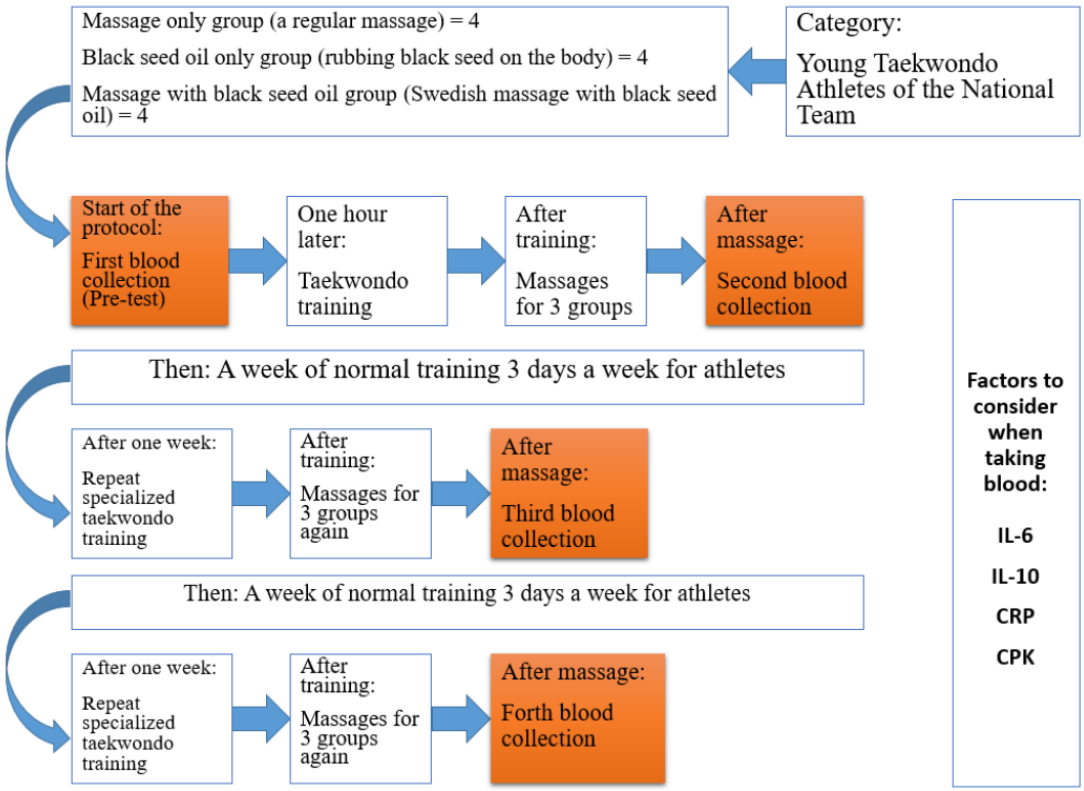

Figure 1. Schematic representation of the study design, participant allocation, intervention protocols, and blood collection time points across the three experimental groups.

maintain their regular dietary and hydration habits throughout the study period and to avoid any significant changes in food intake, alcohol consumption, or the use of nutritional supplements. While we acknowledge that this lack of strict dietary control represents a significant limitation, we provided participants with verbal and written guidelines regarding the importance of dietary consistency to minimize variability in inflammatory markers. Specifically, participants were advised to consume similar meals on training days, to maintain consistent carbohydrate and protein intake, and to avoid excessive consumption of processed foods, high-fat meals, and sugary beverages that could influence inflammatory responses. However, no objective verification of dietary intake (e.g., food frequency questionnaires, 24-hour dietary recalls, or food diaries) was conducted.

Inclusion and exclusion criteria

Stringent inclusion criteria were established to ensure the internal validity of the study findings. All participants were required to be actively engaged in taekwondo training for a minimum of three years prior to the study, with consistent participation of at least three training sessions per week. Additionally, eligible participants had no documented history of cardiovascular or respiratory diseases, no existing orthopedic injuries that could be exacerbated by the training protocol, and confirmed abstinence from antioxidant supplements and ergogenic aids throughout the study period. Regarding exclusion criteria, participants were removed from the study if they developed any illness, particularly upper respiratory tract infections, that could influence systemic inflammatory markers. Furthermore, the occurrence of any musculoskeletal injury during the intensive taekwondo training sessions, inability to complete the prescribed training regimen, consumption of medications known to affect inflammatory or muscle damage markers, or voluntary withdrawal of consent at any stage of the research process were all considered valid grounds for exclusion from the final analysis.

Intervention protocols

Swedish massage intervention

The massage intervention administered to the relevant experimental groups consisted of a standardized Swedish massage protocol delivered once per week over three consecutive weeks, with each session lasting between 30 and 45 minutes depending on the specific muscle groups targeted during that session. This duration range was not random; it reflected the number of muscle groups that required treatment based on each athlete's individual report of tightness and the therapist's clinical judgement. However, we accept that this introduces significant variability in the total mechanical stimulus received, and we have now listed this as a key confounding factor in the Limitations sec-

-tion. All massage procedures were performed by a licensed and experienced sports massage therapist to ensure technical consistency and professional application of the techniques. The protocol specifically targeted the major muscle groups that are predominantly engaged during taekwondo training and competition, including the quadriceps, hamstrings, gastrocnemius, shoulder girdle muscles, and the lumbar region of the lower back. The Swedish massage technique incorporated the five fundamental strokes sequentially applied to optimize therapeutic outcomes. The session typically commenced with effleurage, consisting of long, gliding strokes performed with the palms and fingers in the direction of venous return to warm the muscle tissue and enhance local circulation. This was followed by petrissage, involving rhythmic kneading, compression, and rolling of the muscle bellies to release myofascial tension and improve tissue pliability. Friction techniques, characterized by deep, circular pressure applied with fingertips or thumbs, were employed to address specific areas of muscle tightness and to penetrate deeper connective tissue layers. Tapotement, consisting of rhythmic percussion movements such as hacking and cupping, was applied to stimulate muscle spindles and enhance neuromuscular activation. Finally, vibration techniques involving fine, oscillatory movements were utilized to promote muscle relaxation and soothe nerve endings. The pressure intensity was modulated between moderate and deep levels based on individual participant tolerance and specific muscle condition, with each technique applied for approximately 5 to 10 minutes per targeted muscle group.

Black seed oil preparation and application protocol

The black seed oil utilized in this investigation was derived from cold-pressed seeds of Nigella sativa, commonly known as black cumin or black seed, which was specifically selected to preserve the integrity of its bioactive constituents, most notably thymoquinone, which is widely recognized for its potent anti-inflammatory, analgesic, and antioxidant properties. The oil was stored under controlled conditions in an opaque, airtight container maintained at ambient room temperature throughout the study duration to prevent photo-oxidation and preserve its therapeutic efficacy. Prior to each application session, the oil was warmed to body temperature of approximately 37 degrees Celsius by placing the container in a warm water bath, a procedure intended to enhance dermal absorption and provide an additional soothing sensation during application. The target areas for oil application, comprising the lower back, lower extremities, and shoulder regions, were first cleansed using a mild, alcohol-free cleansing solution to remove surface impurities, sweat, and sebum that might otherwise impede optimal transdermal penetration of the active compounds. A standardized volume of approximately 5 to 10 milliliters of warmed oil, adjusted according to the surface area being treated, was then applied uniformly to the designated areas using gentle, circular massage motions for approximately 10 minutes to facilitate mechanical distribution and absorption. We acknowledge that the 5 10 mL range is relatively broad; this variability was based on individual differences in the treated surface area (e.g., lower back vs. entire lower extremities), which is a common practice in topical application studies. However, we concede that this lack of strict dose standardisation per body surface area or weight is a methodological limitation that may have contributed to inter individual variability in transdermal uptake. Following the active application phase, participants remained in a resting state for an additional 10 minutes to allow complete dermal penetration of the remaining oil residue. All participants were systematically screened for hypersensitivity or allergic reactions to black seed oil or its constituents prior to the initial application, and throughout the intervention period, no adverse dermatological reactions were reported by any participant.

The topical route was chosen deliberately to mirror real-world sports massage practice, where oils are applied locally to the skin overlying the exercised muscles. This approach was intended to isolate local (peripheral) effects rather than systemic actions, and to avoid the gastrointestinal and hepatic first-pass metabolism that would occur with oral administration. Nevertheless, we recognise that transdermal bioavailability of thymoquinone is limited, which may have attenuated the expected anti-inflammatory response.

Combined massage and black seed oil intervention

Participants allocated to the combined intervention group received the identical Swedish massage protocol described previously, with the critical distinction that the warmed black seed oil served as the lubricating medium throughout the entire massage session. This approach ensured that the mechanical benefits of soft tissue manipulation were delivered concurrently with the transdermal administration of the bioactive compounds present in the oil, potentially allowing for synergistic effects between the two modalities. The same certified massage therapist administered all sessions for this group, and the duration, frequency, and technical application of massage strokes remained consistent with the massage-only group to ensure comparability of results across experimental conditions.

Blood sampling and biochemical analysis

Venous blood samples were collected at four distinct time points throughout the experimental period to track longitudinal changes in the selected biochemical markers. The first sampling session was conducted prior to the initial intervention and training session to establish baseline values for each participant. Subsequent sampling sessions were scheduled following the completion of the first, second, and third weeks of the intervention protocol, pr-

-ecisely 48 hours after the last training session. This time point was selected to capture sub-acute inflammatory and muscle damage responses, as it represents a compromise between allowing sufficient time for tissue repair processes to evolve and avoiding the immediate post exercise peak fluctuations that are known to occur within the first few hours. However, we fully acknowledge that this single time point has inherent limitations: IL 6 typically peaks at 1–2 hours post exercise and may have already declined by 48 hours, whereas CRP peaks at 24–48 hours and may be at or near its maximum at the time of sampling. Thus, the 48 hour window may have captured IL 6 near its nadir while capturing CRP at its peak or early declining phase. This methodological choice was driven by practical considerations in an elite athlete population (training schedules, recovery periods, and federation constraints) and the need to standardise sampling across all participants. Nevertheless, we recognise that measuring only one post exercise time point limits our ability to characterise the full kinetic profile of these biomarkers. To control for the well-documented influence of circadian rhythms on endocrine and inflammatory parameters, all blood collection procedures were consistently performed during the morning hours between 8:00 and 10:00 AM (Figure 1). Participants were instructed to maintain their regular dietary and sleep patterns prior to each At each time point, 10 milliliters of whole blood were drawn from the right antecubital vein with participants in a comfortable seated resting position, and all venipunctures were performed by a qualified laboratory technician using standardized phlebotomy techniques to minimize procedural variability and participant discomfort.

Serum and plasma preparation

Following collection, blood samples were immediately transferred to the collaborating analytical laboratory for processing. Samples designated for serum analysis were placed in gel barrier tubes containing clot activator and allowed to stand at room temperature for approximately 15 minutes to permit complete clot formation. Subsequently, all samples underwent centrifugation at 3500 revolutions per minute for 10 minutes, a process that effectively separated the cellular components from the serum or plasma fraction. The resulting supernatant was carefully aliquoted into sterile microtubes using micropipettes, taking care to avoid contamination with the buffy coat layer. Serum and plasma aliquots were then stored at minus 80 degrees Celsius until the day of biochemical analysis to preserve analyte stability and prevent degradation from repeated freeze-thaw cycles.

Measurement of biochemical markers

All biochemical analyses were performed using enzyme-linked immunosorbent assay technique (ELISA) with commercially available kits specifically validated for the measurement of human inflammatory and muscle damage markers. Interleukin-6 concentrations in plasma samples were quantified using a highly sensitive ELISA kit with a detection range spanning 15.6 to 500 picograms per milliliter, an analytical sensitivity of 5.6 picograms per milliliter, and an intra-assay coefficient of variation ranging from 5.4 to 7.8 percent, indicating acceptable precision for research applications. Plasma interleukin-10 levels were similarly measured using a dedicated ELISA kit providing a broader detection range of 4.9 to 3000 picograms per milliliter, with a sensitivity of 4.1 picograms per milliliter and intra-assay coefficients of variation between 2.3 and 2.5 percent, demonstrating excellent reproducibility. For the assessment of systemic inflammation, serum C-reactive protein concentrations were determined using a commercial immunoturbidimetric kit manufactured by Pars Azmoon Company of Iran, which exhibited a detection sensitivity of 0.1 milligrams per liter. Serum creatine phosphokinase activity, serving as an indirect marker of muscle membrane integrity and exercise-induced muscle damage, was measured using an enzymatic kit also provided by Pars Azmoon Company with a sensitivity of 0.2 milligrams per liter. All assays were performed in duplicate according to the manufacturer's instructions by the same laboratory technician who was blinded to the group allocation of samples to prevent analytical bias, and standard quality control procedures were implemented throughout the analytical process. In contrast, the intervention administrator (massage therapist) and the participants could not be blinded to group allocation because the black seed oil has a distinct odour and appearance. Consequently, performance bias and detection bias cannot be ruled out.

Statistical analysis

All statistical analyses were conducted using appropriate software for biomedical research. The Shapiro-Wilk test was employed to assess the normality assumption for all continuous outcome measures. To evaluate the effects of the interventions across the three experimental groups over the four measurement time points, a two-way analysis of variance with repeated measures on the time factor was performed for each dependent variable. Bonferroni-adjusted post-hoc tests were conducted for pairwise comparisons to identify specific time points and group differences while controlling for the inflation of type I error associated with multiple comparisons. The threshold for statistical significance was established at an alpha level of 0.05 for all analyses, and all reported p-values are two-tailed. However, it must be emphasized that the statistical analyses were conducted on a severely underpowered sample. The absence of statistically significant group differences may reflect Type II error rather than true equivalence of interventions. The effect sizes reported (ES=0.033-0.066 for group main effects) should not be interpreted as evidence of no meaningful differences, but rather as an indication that the study lacked suff-

Table 1. Mean and standard deviation of descriptive characteristics of research samples.

Variable	Age (year)	Height (m)	Weight (kg)	BMI (kg/m^2)
Mean±SD	21.45±3.25	1.72±0.07	71.18±9.15	23.85±1.78

-icient sensitivity to detect such differences if they existed.

Results

Descriptive characteristics of study subject showed in table 1. Two-way ANOVA result showed significant main time effect (p≤0.001, ES=0.838) and group by time interaction effect (p=0.002, ES=0.427) but not group main effect (p=0.550, ES=0.033) for IL-6. The results of the Bonferroni post-hoc test showed that IL-6 levels increased significantly in the black seed oil group compared to the massage+black seed oil group in the post-test of the first week (p=0.030). There was no significant difference in other comparisons (p≥0.05). Also, the results of the within-group comparisons showed a significant increase in IL-6 levels in the post-test of the first, second and third weeks compared to the pre-test of the first week in all three groups (p≤0.05) (Fig2. A). Similarly for IL-10 Two-way ANOVA result showed significant main time effect (p≤0.001, ES=0.750) and group by time interaction effect (p=0.007, ES=0.371) but not group main effect (p=0.328, ES=0.060). The results of the Bonferroni post-hoc test showed that IL-10 levels increased significantly in the black seed oil group compared to the massage + black seed oil group in the post-test of the first week (p=0.050). There was no significant difference in other comparisons (p≥ 0.05). Also, the results of the within-group comparisons showed a significant increase in IL-10 levels in the post-test of the first, second and third weeks compared to the pre-test of the first week in all three groups (p≤0.05) (Fig2. B).

Nevertheless, result of Two-way ANOVA showed there was no significant main group effect (p=0.968, ES=0.002; p=0.861, ES=0.008), main time effect (p=0.999, ES=0.001; p=0.474, ES=0.066) or group by time interaction effect (p=0.984, ES=0.027; p=0.845, ES=0.069) for CRP and CPK respectively (Fig2. C, D). It is important to note that while statistically significant findings were observed for certain within-group and interaction effects, these results must be interpreted with considerable caution given the small sample size (N=12). The lack of significant group main effects and the small effect sizes observed (ES=0.033-0.066) may reflect insufficient statistical power to detect meaningful differences rather than true equivalence of the interventions. The significant within-group increases over time should be interpreted cautiously as they may partly reflect normal training-related adaptations in the absence of a true control group.

Discussion

The present findings must be interpreted within the context of several important methodological limitations. First, the quasi-experimental design without a true control group precludes causal attribution of the observed changes to the interventions specifically. Second, the severely underpowered sample (N=12, 4 per group) substantially limits the reliability and generalizability of all findings. Third, the following mechanistic interpretations—while grounded in established literature—are speculative in the context of this study, as we did not directly measure intracellular signaling pathways, gene expression, or tissue-level molecular changes. Our data consist solely of circulating biomarker measurements, which provide indirect insight into systemic inflammatory responses but cannot elucidate the underlying molecular mechanisms. With these caveats firmly in mind, the observed time-dependent increases in IL-6 and IL-10 across all groups confirm that intensive taekwondo training elicited a coordinated inflammatory response. However, rather than representing maladaptive inflammation, these elevations are consistent with the physiological myokine response to high-intensity intermittent exercise, reflecting transient immune signaling and metabolic adaptation to repeated muscular contractions.

The present study demonstrated significant time-dependent increases in IL-6 and IL-10 across all intervention groups, confirming that intensive taekwondo training elicited a coordinated inflammatory response. These findings are in line with previous evidence indicating that high-intensity intermittent exercise stimulates a rapid release of myokines from contracting skeletal muscle fibers. Such cytokine responses are considered a natural physiological consequence of repeated muscular contractions, metabolic stress, and transient tissue disruption induced by strenuous athletic activity. Therefore, the observed elevation in these inflammatory markers reflects the body's acute attempt to regulate tissue repair, immune signaling, and metabolic adaptation following demanding training sessions (He, Tian et al. 2018, Nieman and Wentz 2019). IL-6 is now recognized not merely as a pro-inflammatory cytokine (Sommatis, Mocchi et al. 2026) but as a pleiotropic myokine released in response to glycogen depletion and calcium-mediated signaling within myocytes (Calle-Ciborro, Espin-Jaime et al. 2023). It activates AMP-activated protein kinase (AMPK) and p38 MAPK pathways, promoting glucose uptake and lipid oxidation while simultaneously initiating anti-inflammatory cascades through IL-10 induction. The parallel rise in IL-10 observed in the present study supports the concept of a tightly regulated inflammatory balance in trained athletes, where anti-inflammatory mediators are upregulated to prevent excessive tissue damage and to facilitate regeneration. Thus, the cytokine elevations detected in this investigation likely reflect physiological adaptation rather than maladaptive inflammation. However, the small sample size (N=12, 4 per group) limits the statistical power

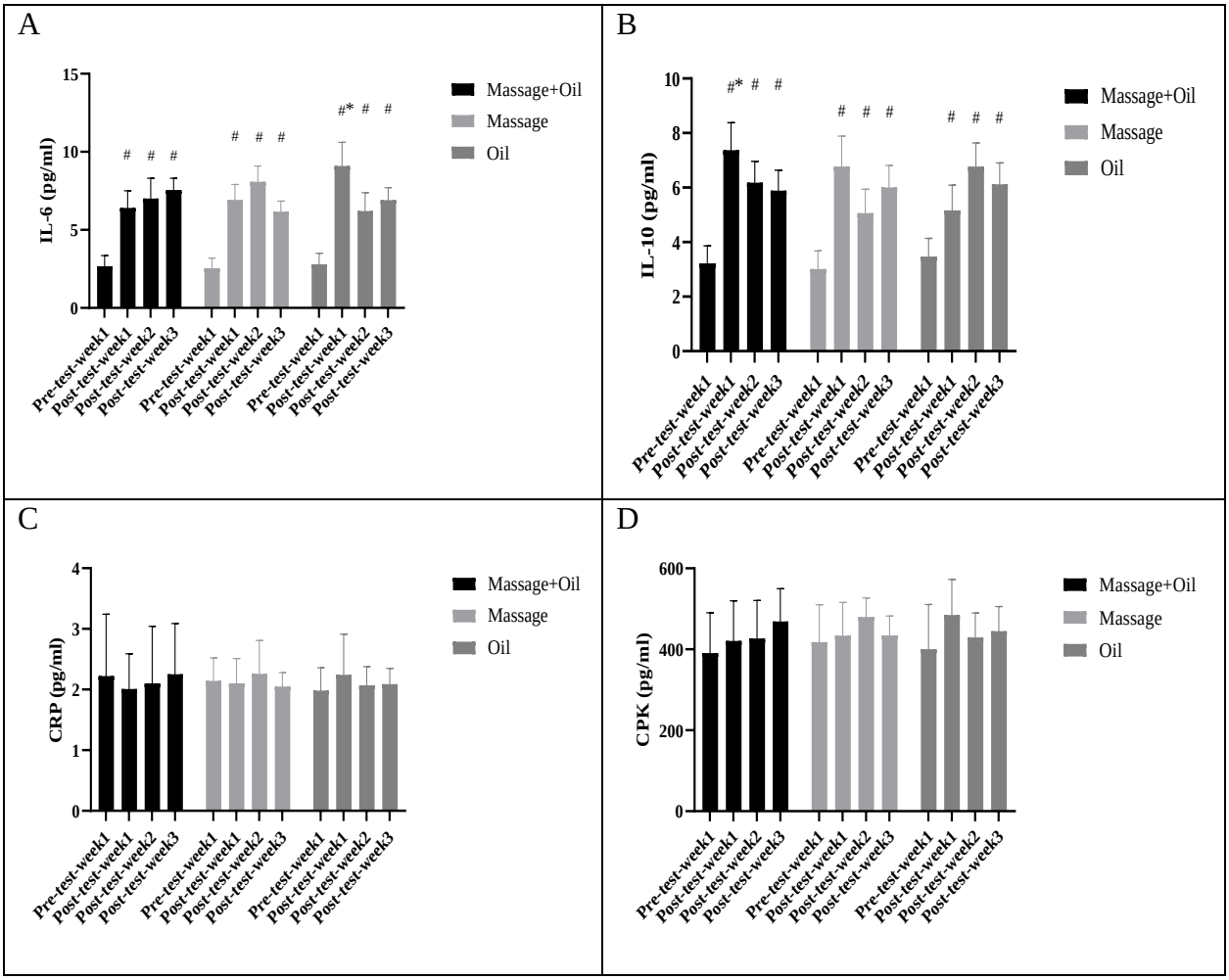

Figure 1. Results of two-way ANOVA test to examine between-group differences in IL-6, IL-10, CRP and CPK levels. # Indicates a significant difference within the group with the first week pre-test. * Indicates a significant difference between the black seed oil and massage + black seed oil groups.

to detect subtle differences between intervention groups, and these findings should be considered preliminary.

In comparing the present findings with previous massage related research, the results appear to be partially aligned while also showing some degree of divergence. The seminal work by Crane et al. reported that post-exercise massage reduced NF-κB activation and attenuated pro-inflammatory signaling within skeletal muscle tissue. This suggests that massage may exert beneficial anti-inflammatory effects by modulating intracellular stress pathways and limiting the magnitude of exercise induced inflammatory responses. However, although the current study similarly supports the regulatory role of massage in post-training recovery, the pattern and extent of cytokine alterations were not entirely identical, indicating that the effectiveness of massage may vary according to exercise type, training load, intervention duration, and athlete characteristics (Crane, Ogborn et al. 2012). Similarly, systematic analyses such as Poppendieck et al. (2016) concluded that massage may reduce delayed-onset muscle soreness, though effects on systemic inflammatory markers remain inconsistent (Poppendieck, Wegmann et al. 2016).

In contrast, the present study did not reveal a significant main group effect for IL-6 or IL-10, suggesting that once-weekly Swedish massage may not be sufficient to markedly blunt circulating cytokine concentrations in elite athletes. Although slight fluctuations were observed over the intervention period, these changes were not large enough to indicate a superior anti-inflammatory effect when compared with the other recovery strategies. This discrepancy between the current findings and previous massage based studies may be explained by several methodological and physiological differences, including variations in training status, timing of blood sampling, massage frequency, and the overall intensity of the imposed exercise stimulus. Elite athletes generally exhibit a repeated bout adaptation characterized by attenuated inflammatory reactivity, enhanced cellular resilience, and improved membrane stability. As a result of long term exposure to strenuous training, their physiological systems become more efficient at tolerating exercise induced muscle stress, which may substantially reduce the observable impact of external recovery modalities compared with recreationally trained or untrained individuals. Therefore, interventions such as low frequency Swedish massage may

provide only limited measurable changes in circulating inflammatory biomarkers when applied to highly conditioned athletes whose endogenous recovery mechanisms are already well developed.

The transient difference observed during week one between the black seed oil group and the combined massage plus oil group warrants further mechanistic interpretation. This early variation suggests that the physiological response to black seed oil may manifest more rapidly when the intervention is applied alone, whereas its effects in combination with massage may be influenced by additional recovery related factors. Nigella sativa contains thymoquinone, a major bioactive constituent that has been reported to inhibit NF-κB translocation, suppress cyclooxygenase-2 (COX-2) expression, and reduce reactive oxygen species (ROS) production. Through these pathways, black seed oil may contribute to limiting oxidative stress and moderating the early inflammatory cascade induced by repeated high-intensity exercise. (Kavyani, Musazadeh et al. 2023, Ahmad, Arif et al. 2024). At the cellular level, thymoquinone modulates Toll-like receptor (TLR) signaling and reduces phosphorylation of IκB kinase, thereby limiting downstream transcription of IL-6 (Abulfadl, El-Maraghy et al. 2018). It may also enhance the activity of antioxidant enzymes such as superoxide dismutase and glutathione peroxidase, thereby mitigating oxidative stress induced cytokine production and limiting secondary inflammatory damage. By strengthening the endogenous antioxidant defense system, black seed oil could theoretically reduce the accumulation of free radicals generated during repeated bouts of intensive exercise. However, the topical route of administration used in the present study introduces several important pharmacokinetic considerations that may have restricted the magnitude of this expected physiological benefit. Transdermal absorption is strongly dependent on factors such as molecular size, lipophilicity, skin hydration, and the permeability of the epidermal barrier. Therefore, it is plausible that dermal penetration of thymoquinone was limited, resulting in only minimal systemic bioavailability and a weaker than anticipated anti-inflammatory response. Interestingly, the greater cytokine increase observed in the oil-only group compared with the combined massage plus oil group during week one suggests that mechanical massage may have altered local microcirculation and interstitial fluid dynamics in a manner that influenced the transport of inflammatory mediators. Enhanced blood flow and lymphatic movement induced by massage could potentially facilitate a different pattern of cytokine clearance or redistribution into circulation. In addition, massage induced shear stress and mechanotransduction are known to modulate endothelial nitric oxide synthase (eNOS) activity and may influence immune cell trafficking within the affected tissues. Such physiological alterations may contribute to shaping the early inflammatory sign-aling process and partially explain the transient differences detected between the intervention groups during the initial phase of the study (Crane, Ogborn et al. 2012). Nevertheless, we reiterate that these mechanistic speculations are not supported by direct evidence from our study and should be treated as hypothesis-generating rather than confirmatory.

It is essential to explicitly acknowledge that the present study did not measure any intracellular signaling molecules (e.g., phosphorylated NF-κB, IκB kinase, MAPK, AMPK, or eNOS), gene expression, or tissue-level inflammatory markers. Our data are limited to circulating concentrations of IL-6, IL-10, CRP, and CPK measured in peripheral blood. These biomarkers provide an indirect and systemic reflection of inflammatory and muscle damage responses but cannot inform us about the specific molecular events occurring within skeletal muscle or other tissues. Any discussion of NF-κB, COX-2, TLR, AMPK, p38 MAPK, or eNOS pathways in the context of our findings is speculative and derived from the broader scientific literature, not from our experimental data. The observed between-group differences were small, transient, and observed only at a single time point (week one). Given the small sample size, the absence of a true control group, and the multiple comparisons performed, these findings may represent Type I error or chance variation rather than true biological effects. Therefore, all mechanistic interpretations should be considered preliminary and hypothesis-generating, requiring confirmation in studies specifically designed to measure these pathways directly.

With respect to CRP and CPK, the absence of significant changes in the present study is consistent with several investigations conducted in elite athletic populations. CRP synthesis in hepatocytes is primarily driven by sustained elevations in IL-6; therefore, transient cytokine fluctuations of modest magnitude may not be sufficient to trigger a measurable acute phase response. In the current context, although IL-6 levels increased over time, the magnitude and duration of this elevation were likely inadequate to induce a robust hepatic response. Furthermore, highly trained athletes often exhibit blunted CRP responses due to enhanced inflammatory regulation and more efficient homeostatic control mechanisms. Chronic exposure to intensive training leads to physiological adaptations that reduce excessive inflammatory activation and promote faster recovery following exercise-induced stress. As a result, biomarkers such as CRP may remain relatively stable despite repeated bouts of high intensity activity. This adaptive response may partly explain the lack of significant changes observed in CRP levels throughout the study period (Nieman and Wentz 2019). Similarly, the stability of CPK levels in this study may reflect the repeated-bout effect and structural adaptation of sarcolemma integrity described by Brancaccio et al (Brancaccio, Lippi et al. 2010).

In contrast, several studies conducted in untrained individuals or using eccentric exercise models have reported pronounced elevations in CPK following strenuous physical activity, reflecting greater exercise-induced muscle membrane disruption. The discrepancy between these findings and the present study likely reflects key differences in training status, exercise modality, and overall recovery capacity. Elite athletes typically demonstrate a higher degree of structural and functional adaptation within skeletal muscle, which enhances resistance to mechanical stress and accelerates post-exercise repair processes. Accordingly, the absence of significant alterations in CRP and CPK observed in the current investigation does not necessarily indicate a lack of physiological stress. Rather, it more plausibly reflects an efficient and well adapted recovery system in elite taekwondo athletes, characterized by improved muscle integrity, attenuated inflammatory signaling, and rapid restoration of homeostasis following intensive training.

A further important consideration is the timing of blood sampling. The single 48 hour post exercise measurement, while practical and standardised, may not have captured the full dynamic range of the cytokine responses. IL 6 is known to rise rapidly within 1–2 hours of intense exercise and typically returns toward baseline within 24 hours. Therefore, our 48 hour sampling likely missed the early peak of IL 6, potentially underestimating the acute inflammatory response and any differential modulation by the interventions. Conversely, CRP, which peaks at 24–48 hours, was measured at an appropriate time for its maximal response, although we cannot rule out that some participants were already in the declining phase. This single time point design precludes assessment of the complete kinetic profile and may have contributed to the absence of significant group differences. Future studies should incorporate multiple post exercise sampling points (e.g., immediately, 2 h, 24 h, 48 h, and 72 h) to capture peak responses and recovery trajectories more accurately.

Collectively, the current findings contribute to the growing yet still inconclusive body of literature on recovery modalities and their role in inflammatory modulation. While massage and black seed oil did not produce significant differences in systemic inflammatory or muscle damage markers when compared with each other, the observed interaction effect during the first week suggests the possibility of short-term modulation in cytokine kinetics at the early phase of recovery. This transient response may indicate that certain interventions exert time-sensitive effects that are more detectable in the initial adaptation period rather than across prolonged exposure. At the cellular level, both mechanical stimulation through mechanotransduction pathways influencing NF-κB and MAPK signaling and phytochemical intervention via thymoquinone mediated antioxidant and anti-inflammatory mechanisms theoretically converge on overlapping molecular targets involved in inflammation and oxidative stress regulation. Despite this mechanistic plausibility, the magnitude of exercise induced cytokine release in highly trained individuals may be sufficiently robust to mask or override the relatively subtle effects of these recovery strategies. However, the small sample size of this study (N=12) severely limits the ability to detect moderate or small effect sizes. The effect sizes observed for group main effects (ES=0.033-0.060) suggest that the study lacked sensitivity to detect clinically meaningful differences, and the significant findings may represent chance associations rather than true intervention effects. Future research should therefore consider more sensitive and comprehensive methodological approaches. Studies with larger sample sizes, increased intervention frequency, and extended monitoring periods may help clarify subtle physiological effects. In addition, incorporating a broader panel of inflammatory biomarkers such as TNF-α and IL-1β, alongside direct tissue-level assessments such as muscle biopsy analyses, would provide deeper insight into intracellular signaling adaptations and offer a more precise understanding of how different recovery modalities influence post-exercise physiological regulation.

While we observed statistically significant time-dependent increases in IL-6 and IL-10 across all groups, translating these changes into clinically or practically meaningful recovery outcomes requires careful scrutiny. In exercise immunology, there are no universally established thresholds for a "minimal clinically important difference" (MCID) or "minimal important change" for circulating IL-6 or IL-10, particularly in elite athletic populations. The biological variability of these cytokines is inherently high (intra assay CV: 5.4–7.8% for IL 6; 2.3–2.5% for IL 10), and inter individual fluctuations often exceed 20% due to circadian rhythms, hydration status, and glycogen availability. The observed within group increases, although statistically robust ($p \leq 0.05$), largely reflect the normal physiological myokine response to high intensity training rather than a pathologically elevated inflammatory state requiring intervention. Critically, we did not concurrently measure functional recovery outcomes such as countermovement jump performance, isometric strength recovery, perceived muscle soreness on a visual analog scale, or next day training quality. Without these objective or subjective performance anchored measures, the actual recovery relevance of a 10–20% fluctuation in IL 6 or IL 10 remains speculative. Furthermore, the effect sizes for time effects (ES=0.838 and 0.750) reflect the potent exercise stimulus itself, not an intervention driven clinical benefit. In elite athletes with well-developed endogenous adaptation mechanisms, even transient cytokine elevations are typically resolved within 24–48 hours and do not necessarily translate into delayed recovery or impaired subsequent performance. Therefore, we caution against overinterpreting these statistical p values as evidence of practic-

-ally meaningful recovery enhancement. Future investigations should integrate biomarker kinetics with functional performance batteries and athlete reported outcome measures to establish whether specific magnitudes of cytokine change reliably predict accelerated return to performance or reduced injury risk.

Study limitations

The most critical limitation of the present study is its quasi-experimental design without a true control group. Although participants were allocated to three different intervention groups, the absence of a group receiving neither massage nor black seed oil makes it impossible to determine whether the observed time-dependent increases in IL-6 and IL-10 resulted from the interventions themselves or from normal physiological adaptations to continued intensive training, circadian rhythms, or placebo effects. Consequently, the significant main effects of time observed across all groups should be interpreted cautiously, and causality cannot be established from this study. Future research should employ randomized controlled trial designs with appropriate control groups to isolate the specific effects of each recovery modality.

An equally serious limitation is the severely inadequate sample size. The final analytical sample of N=12 (4 per group) following a 20% dropout rate is woefully insufficient for detecting meaningful differences between groups. The authors used "Morgan's table guidelines" to justify sample size, but no formal power analysis or effect size estimation was performed a priori. With only four participants per group, the study is severely underpowered to detect any but the most massive effects (Cohen's f>0.60). The authors' own effect sizes (ES=0.033-0.066 for non-significant findings) suggest that the study lacked sensitivity to detect clinically meaningful differences. This substantially increases the risk of Type II errors (false negatives) and limits the reliability and generalizability of the findings. Therefore, the results should be presented as preliminary/pilot data rather than definitive findings, and all conclusions should be tempered accordingly. Future adequately powered studies with a priori sample size calculations are essential to confirm or refute the observations reported here.

Additional critical limitations, beyond those already noted, include: (1) the variable volume of black seed oil (5-10 mL) and variable massage duration (30-45 min), which were not standardised per body weight or muscle group, thus potentially masking or exaggerating intervention effects; (2) the choice of topical over oral administration, which limits systemic bioavailability of thymoquinone and may have reduced the pharmacological impact; and (3) the complete absence of blinding for participants and the massage therapist, which introduces performance and detection biases. These factors collectively constrain the internal validity of our findings, and all results must be interpreted with extreme caution. In addition to the design limitations mentioned above, the statistical approach used in this study is inherently problematic. With only four participants per group, the assumptions of repeated measures ANOVA (sphericity, normality, homogeneity of variances) could not be adequately tested, and no Greenhouse–Geisser corrections were applied. Furthermore, the interpretation of Bonferroni adjusted post hoc comparisons following a non-significant omnibus group main effect is statistically inappropriate and greatly increases the risk of false positive findings. Therefore, all reported pairwise differences should be viewed as hypothesis generating, not confirmatory. Future studies should employ non parametric methods (e.g., Friedman test with post hoc Wilcoxon signed rank tests) or Bayesian approaches that are more suitable for small samples, and should always include a priori power calculations to ensure adequate sample size.

In addition to the design and power limitations discussed above, the present study is limited by its single post exercise blood collection time point (48 hours). Given that IL 6 peaks at 1–2 hours and CRP at 24–48 hours, this single time point may have missed the early IL 6 peak and may have captured CRP at a variable phase of its response. Without multiple serial measurements, we cannot determine the true area under the curve or the peak magnitude of these biomarkers, which limits our ability to detect intervention related differences. We acknowledge that the choice of 48 hours was a practical compromise rather than an optimal kinetic design, and this should be considered when interpreting the absence of significant group effects. Future investigations should employ repeated sampling schedules to capture the full temporal profile of exercise induced inflammatory and muscle damage markers.

Conclusion

In summary, while intensive taekwondo training elicited significant time-dependent increases in IL-6 and IL-10 consistent with exercise-induced immunomodulation, neither once-weekly Swedish massage, topical black seed oil, nor their combination produced significant or consistent alterations in systemic CRP or CPK concentrations. The isolated first-week cytokine differences were small, transient, and observed in a severely underpowered sample without a true control group; they should be interpreted exclusively as hypothesis-generating pilot data requiring independent replication. The primary conclusion is that, under the present experimental conditions, these short-term recovery interventions did not demonstrate clinically meaningful benefits on systemic inflammatory or muscle damage markers in elite taekwondo athletes. Given the severe methodological limitations most notably the absence of a control group and the final sample size of N=12 (4 per group)—all findings require confirmation in

larger, adequately powered randomized controlled trials before any practical recommendations for clinical practice or athletic recovery protocols can be made.

What is already known on this subject?

Intensive exercise, particularly in combat sports like taekwondo, induces a transient systemic inflammatory response characterized by elevated circulating cytokines (e.g., IL-6, IL-10) and muscle damage markers (CPK, CRP). Massage therapy has been proposed to attenuate post-exercise inflammation through mechanical and neural mechanisms, while Nigella sativa (black seed) oil exhibits anti-inflammatory and antioxidant properties in vitro and in animal models. However, evidence in elite athletes is sparse, and the potential synergistic effect of combining massage with topical black seed oil on inflammatory biomarkers has not been systematically examined.

What this study adds?

This pilot study provides preliminary, hypothesis-generating data on the effects of a 3-week once-weekly intervention (Swedish massage, topical black seed oil, or their combination) in elite male taekwondo athletes. Despite significant time-dependent rises in IL-6 and IL-10 across all groups no consistent between-group differences emerged for CRP or CPK. A transient, isolated first-week difference (higher IL-6/IL-10 in the oil-only vs. combined group) suggests a possible early modulation of cytokine kinetics, but this finding is undermined by the lack of a true control group, a severely underpowered sample (N=12, 4/group), and multiple methodological limitations. The study underscores that in highly conditioned athletes, endogenous recovery mechanisms may overshadow the measurable impact of such recovery modalities, and it highlights the urgent need for adequately powered, randomised controlled trials with serial biomarker sampling and functional outcome measures.

Organ Cross-Talk Tips:

- Exercise-induced myokine release (IL-6) from contracting skeletal muscle acts as a primary signal that concurrently stimulates systemic anti-inflammatory IL-10 production, illustrating a dynamic muscle-to-immune feedback loop.
- Topical black seed oil administration relies on transdermal absorption; massage may enhance local perfusion and lymphatic drainage, potentially altering interstitial cytokine trafficking and clearance, but limited thymoquinone bioavailability restricts systemic effects.

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Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest in the present research.

Ethical approval The study protocol received formal approval from the Ethics Committee of the University of Tehran, International Campus, under approval number (Ethics Code: IR.UT.PSYEDU.REC.1404.075).

Informed consent Animal study.

Author contributions

Conceptualization: Conceptualization: P.P.; Methodology: F.D.; Software: S.R.M; Validation: F.Sh.; Formal analysis: F.Sh.; Investigation: P.P.; Resources: F.D.; Data curation: S.R.M.; Writing-original draft: F.Sh.; Writing - review & editing: R.R.M.; Visualization: F.Sh.; Supervision: P.P., F.D.; Project administration: F.Sh.; Funding acquisition: S.R.M.

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