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The Effects of a Single Bout of Resistance Activity at Two Intensities with Different Recovery Periods on IL-6, ACTH, and BDNF Levels in Female Bodybuilders

Azar Momtaz[1]
Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran

Ramin Shabani[2]
Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran

Alireza Elmieh[3]
Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran

ABSTRACT

Introduction: Resistance exercise enhances physical performance and metabolic health, but responses vary based on intensity and recovery. This study investigated how different resistance training intensities and recovery periods affect interleukin-6 (IL-6), adrenocorticotropic hormone (ACTH), and brain-derived neurotrophic factor (BDNF) in female bodybuilders.

Material & Methods: Twelve healthy female bodybuilders (25–35 years) completed two weekly sessions for four weeks: moderate intensity (75% 1RM) with 1- or 2-minute recovery (MLRT1/MLRT2) and high intensity (85% 1RM) with 1- or 2-minute recovery (HLRT1/HLRT2). Blood samples were taken 36 hours post-exercise after fasting. Serum IL-6, ACTH, and BDNF levels were analyzed via SPSS (v27; $p < 0.05$).

Results: High-intensity training (85% 1RM) with 1-minute recovery significantly reduced IL-6 ($p = 0.0001$) and ACTH ($p = 0.0001$) while increasing BDNF ($p = 0.0001$) compared to lower intensities.

Conclusion: HLRT with short recovery did not impair immunity and was linked to lower IL-6/ACTH and higher BDNF, suggesting anti-inflammatory and neurotrophic benefits in female bodybuilders.

Keywords: Adrenocorticotropic hormone (ACTH), Brain-derived neurotrophic factor (BDNF), Female bodybuilders, Interleukin-6 (IL-6), Resistance exercise.

***Correspondence:** Raminshabani@iau.ac.ir
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1. Introduction

Resistance activity is recognized as one of the most effective methods for improving physical performance and metabolic health. This form of exercise not only enhances muscle strength and hypertrophy but also exerts significant effects on the immune and nervous systems. However, the body's response to resistance exercise, particularly in female bodybuilders, is influenced by various factors, including activity intensity and recovery duration. Among these, pro-inflammatory factors such as interleukin-6 (IL-6), the inflammatory marker ACTH, and brain-derived neurotrophic factor (BDNF) mediate the bidirectional communication between immune activation and neural circuitry.

Exercise-induced metabolic stress triggers the immediate release of interleukin-6 (IL-6), a key mediator of inflammatory signaling cascades. Research demonstrates that vigorous physical activity induces fluctuations in circulating IL-6 levels. Studies have specifically documented significant IL-6 elevation in female bodybuilders following high-intensity resistance exercise like weightlifting, with levels potentially remaining elevated for several hours post-exercise. This temporal pattern suggests IL-6 plays an acute role in exercise response, potentially facilitating muscle repair and recovery processes (1, 2). The exercise-induced IL-6 response appears particularly pronounced in bodybuilders engaging in high-intensity resistance activity. Current evidence indicates this elevation represents a physiological adaptation to muscle microtrauma and inflammation (3). Notably, chronically elevated IL-6 levels may contribute to metabolic syndrome, insulin resistance, and cardiovascular pathologies - significant health concerns in athletic populations. Mechanistically, IL-6 exhibits intensity-dependent regulation during exercise initiation (4)This cytokine mediates its biological effects through two distinct receptors: membrane-bound gp130 (mgp130) and membrane-bound gp80 (mgp80). Both receptors initiate signaling through direct Janus kinase (5) interaction, with all downstream pathways requiring JAK phosphorylation. IL-6 receptor organization and its three primary signaling cascades (6).

ACTH-mediated HPA axis activation represents a fundamental biological interface connecting physiological stress with systemic inflammatory control. This neurohormone demonstrates significant elevation in female bodybuilders, primarily due to the intense physical demands and the multidimensional stress load (psychological, metabolic, and neurological) of competitive athletic preparation activity. Such elevation triggers cortisol release and subsequent HPA axis activation, which may lead to clinical manifestations including sleep disturbances, anxiety, and central adiposity (7). Corticotropin-releasing hormone (CRH) is essential for stress adaptation by mediating hypothalamic-pituitary-adrenal (HPA) axis activation, which coordinates behavioral and autonomic stress responses. CRH neuronal activation depends on neural afferents from ascending brainstem monoaminergic projections and limbic output pathways, triggering sequential CRH synthesis and release. This subsequently stimulates anterior pituitary secretion of adrenocorticotropic hormone (ACTH)(8). CRH gene expression is subject to NF-κB transcriptional regulation, establishing an inflammatory-stress signaling interface (9)

In contrast, brain-derived neurotrophic factor (BDNF) plays critical roles in neuronal growth, synaptic plasticity, and cognitive enhancement. However, the differential effects of resistance activity intensity and recovery duration on circulating BDNF levels in female bodybuilders remain insufficiently characterized. Randomized trials demonstrate significantly greater serum BDNF elevation following aerobic versus resistance interventions (10). Exercise modality-specific effects on BDNF remain debated. Some randomized trials report indistinguishable serum BDNF elevations following either resistance or endurance activity regimens (11)However, other studies report significant BDNF elevation specifically following resistance exercise (12). Notably, even low-intensity resistance activity has been shown to elevate circulating BDNF levels (13).

Significant sex-based differences exist in inflammatory responses to resistance activity, with distinct immunological profiles observed between male and female athletes (14). Given the paucity of research examining resistance activity effects in female populations, comprehensive investigations are imperative to characterize the precisely quantified immunomodulation of inflammatory axis components (15). Given the unresolved questions about optimal resistance activity parameters, this investigation compares two clinically prevalent activity protocols (85% 1RM/1-min vs 75% 1RM/2-min) to determine their differential effects on key physiological markers (IL-6, ACTH, BDNF) at 36 hours post-exercise in trained female bodybuilders. These findings will inform evidence-based programming for this athlete population.

2. Methodology

2.1. Materials and methods

This study was an applied research project conducted quantitatively. Methodologically, it represents a quasi-experimental study. The study assessed the impact of high-load resistance activity (HLRT) on levels of IL-6, BDNF, and ACTH in female bodybuilders. The study design involved one group with four repeated measures. The intervention comprised four distinct exercise protocols, each administered weekly over one month. Participants' menstrual cycles were monitored throughout the study period, with all activity sessions timed to

correspond with appropriate cycle phases. Blood samples were collected 36 hours post-exercise after a 10-hour overnight fast. All venipuncture procedures were performed in a clinical pathology laboratory.

2.2. Participants

The study participants comprised twelve female bodybuilders selected via convenience sampling. With an age of 35±5 years and a body mass index of 25±1.6, with a history of at least three years of bodybuilding. The inclusion criteria for the study included not using supplements and steroids in the past 6 months, and not consuming alcohol or smoking during the activity period. If they did not commit, they were excluded from the sample size. The participants - female volunteer bodybuilders - signed a consent form after a briefing session on the objectives and how to perform the study in compliance with ethical principles. This study was conducted in strict accordance with ethical guidelines for human research. All methods were approved by the Ethics Committee of Islamic Azad University, Rasht Branch, with the number IRIAU.RASHT.REC.1402.047. Also, the research registration number on the Iranian Clinical Trials Registry (IRCT) website is: IRCT20240305061178N1. Sample size was determined using G*Power software. Sample size was determined using G*Power software (with α = 0.05, df = 12, effect size = 3, and power = 85%).

2.3. Measurements

Repetition Maximum (1RM) Assessment: The 1-repetition maximum (1RM) was determined as the maximal load that could be lifted once with proper technique through a full range of motion. Participants underwent standardized 1RM testing protocols for each exercise, following a warm-up period comprising 5–10 repetitions at 50% of estimated 1RM. Subsequent attempts, Loads were progressively increased by 5-10% between trials separated by 3–5-minute recovery, with 3–5-minute rest intervals between trials until 1RM determination was identified within 3–5 attempts. This method demonstrates high test-retest reliability (ICC > 0.90) in resistance-trained populations. To ensure participant safety and maintain testing validity, certified spotters were present for all maximal lift attempts. Participants were instructed to avoid Valsalva maneuvers, and testing was immediately terminated if any signs of compromised form or distress were observed. Before testing, all participants received standardized verbal and written instructions emphasizing proper lifting technique and the avoidance of Valsalva maneuvers. 36 hours after each exercise session, after a ten-hour fast, blood sampling was performed in a medical laboratory. Blood samples were centrifuged at 4,000 rpm and frozen at -20°C. At the end of the one month, blood samples were sent together to the Passaged Histogen Research Institute in Tehran for analysis and were analyzed using the ELISA method.

Biochemical Analysis: Serum levels of pro-inflammatory markers, including interleukin-6 (IL-6), adrenocorticotropic hormone (ACTH), and brain-derived neurotrophic factor (BDNF) were quantitatively measured using enzyme-linked immunosorbent assay (ELISA) according to standard protocols.

2.4. Intervention

2.4.1 Exercise program

The experimental protocol comprised four distinct training conditions incorporating two intensity levels: a moderate-intensity condition at 75% of one-repetition maximum (1RM) and a high-intensity condition at 85% 1RM.The protocol incorporated two distinct recovery intervals (1-minute and 2-minute durations) implemented across four successive weeks of training. Participants maintained their regular workout regimen twice weekly throughout the study period.

The progressive resistance exercise protocol, detailed in Table 1, was implemented over four weeks with systematic variations in intensity and recovery periods.

Table 1. *Exercise Training Protocol*

Parameter	Week 1 & 2	Week 3 & 4
Training Intensity (%1RM)	75% of 1RM	85% of 1RM
Total Session Time	Week 1: 100 min (2 min recovery)	Week 3: 80 min (2 min recovery)
	Week 2: 90 min (1 min recovery)	Week 4: 70 min (1 min recovery)
Inter-set Recovery	Week 1: 2 min	Week 3: 2 min
	Week 2: 1 min	Week 4: 1 min

2.5. Statistical Methods

Data were analyzed using SPSS version 27 (IBM Corp., Armonk, NY, USA). Normality of distribution was assessed via the Shapiro-Wilk test. A repeated-measures ANOVA was applied for normally distributed data to evaluate within-group differences, followed by Bonferroni-adjusted post hoc tests for pairwise comparisons. Statistical significance was set at $p < 0.05$.

3. Results

Demographic characteristics of study participants are presented in Table 2.

Table 2. *Demographic Characteristics of Female Bodybuilders Participating in the Study*

Variable	Mean ± SD
Age (years)	35 ± 5
Bodybuilding experience (years)	3 ± 0.7
Body Mass Index (kg/m²)	25 ± 1.67

Significant time-dependent variations emerged across all measured parameters ($p < 0.01$), as detailed in Table 3 (mean ± SD).

Table 3. *Repeated-Measures ANOVA Results for Exercise Protocol Effects on Biomarkers*

Dependent Variable	MLRT1 Mean ± SD	MLRT2 Mean ± SD	HLRT1 Mean ± SD	HLRT2 Mean ± SD	Analysis of variance P value	F
IL-6	۰/۶۷۴۹±۰/۰۷	۰/۴۱۵۲±۰/۰۳	۰/۴۰۸۲±۰/۰۳	۰/۳۷۶۵±۰/۰۲	*۰/۰۲۵	114/7
ACTH	۰/۸۵۳۳±۰/۱۸	۰/۹۴۳۳±۰/۴۴	۰/۸۹۱۳±۰/۰۵	۰/۷۰۷۶±۰/۱۷۹۸	*۰/۰۰۱	174/5
BDNF	2766 ± 042/0	0/4524 ±041/0	0/506 ± 035/0	0/702±078/0	*۰/۰۰۱	135/3

HLRT1: High-load (85% 1RM) with 1-min recovery
HLRT2: High-load (85% 1RM) with 2-min recovery
MLRT1: Moderate-load (75% 1RM) with 1-min recovery
MLRT2: Moderate-load (75% 1RM) with 2-min recovery
Significant at $p < 0.05$

Based on the results of the ANOVA and Bonferroni post-hoc tests, the level of the pro-inflammatory factor IL-6 significantly decreased 36 hours after exercise ($P = 0.0001$). This reduction was associated with increased exercise intensity and shorter recovery time. The most pronounced reduction was observed following the HLRT1 protocol (70% 1RM + 2-min recovery) vs. other protocols ($p < 0.0001$).

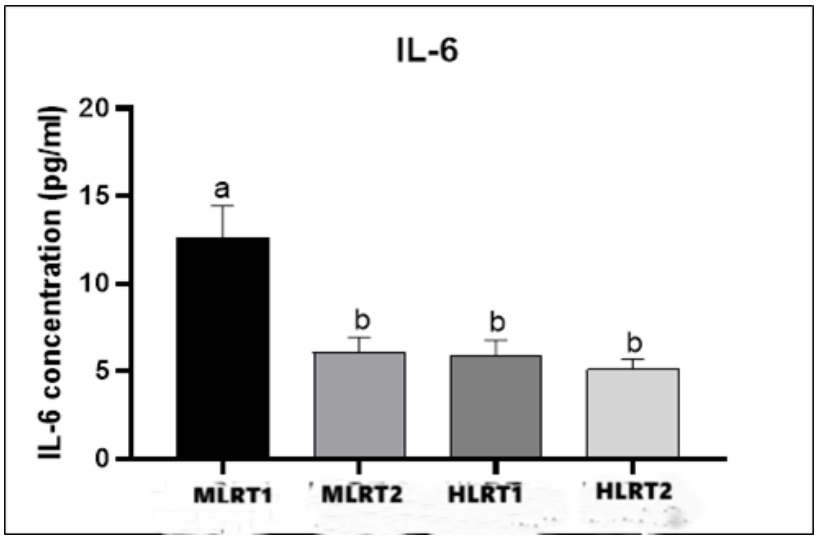

Figure 1. *Mean IL-6 Levels Following Exercise Protocols*
Significant Reduction:
Protocol MLRT1 (75% 1RM + 2min rest) showed:
Markedly lower IL-6 vs. all other protocols ($p < 0.05$)
MLRT1: 75% 1RM + 2min rest/MLRT2: 75% 1RM + 1min rest/HLRT1: 85% 1RM + 2min rest/HLRT2: 85% 1RM + 1min rest

ANOVA with Bonferroni post-hoc testing revealed a significant dose-dependent reduction in ACTH levels ($P = 0.0001$) 36 hours post-exercise, with: (1) Progressive decline at higher loads (85% > 75% 1RM) and (2) Greater suppression with shorter recovery (1-min < 2-min intervals).

The most pronounced ACTH suppression occurred following high-load protocols, except when comparing MLRT2 (70% 1RM + 2-min recovery) versus HLRT2 (85% 1RM + 2-min recovery), where no significant difference was observed ($P > 0.05$).

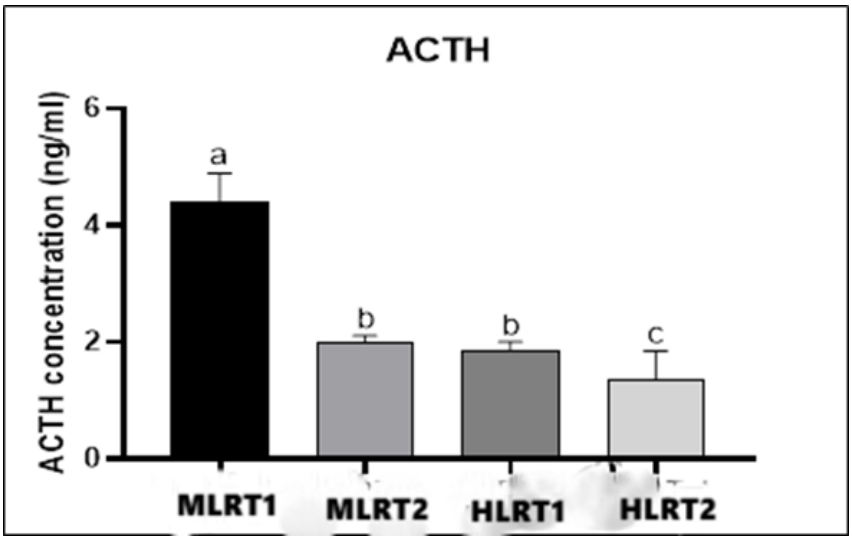

Figure 2. *Mean ACTH Levels Following Exercise Protocols*
*ACTH levels 36h post-exercise. (a) MLRT1 (75% 1RM + 2-min rest) showed the greatest stress-hormone reduction vs. all protocols ($p < 0.0001$). (c) HLRT2 (85% 1RM + 1-min rest) also significantly lowered ACTH ($p < 0.01$). Data = mean ± SD; *$p < 0.05$, **$p < 0.01$, **$p < 0.001$.
MLRT1: 75% 1RM + 2min rest/MLRT2: 75% 1RM + 1min rest/HLRT1: 85% 1RM + 2min rest/HLRT2: 85% 1RM + 1min rest

ANOVA with Bonferroni post-hoc analysis revealed a significant increase in BDNF levels ($P \leq 0.0001$) 36 hours post-exercise, with:(1) Progressive elevation at higher loads, and (2) Greater enhancement with shorter recovery intervals

BDNF levels were significantly elevated following high-intensity protocols with brief recovery intervals ($p<0.0001$), except between MLRT2 and HLRT1 ($p = 0.0676$).MLRT-1:75%1RM,2 min; MLRT-2: 75%1RM,1 min; HLRT-1: 85%1RM,2min; HLRT-2: 85%1RM,1min

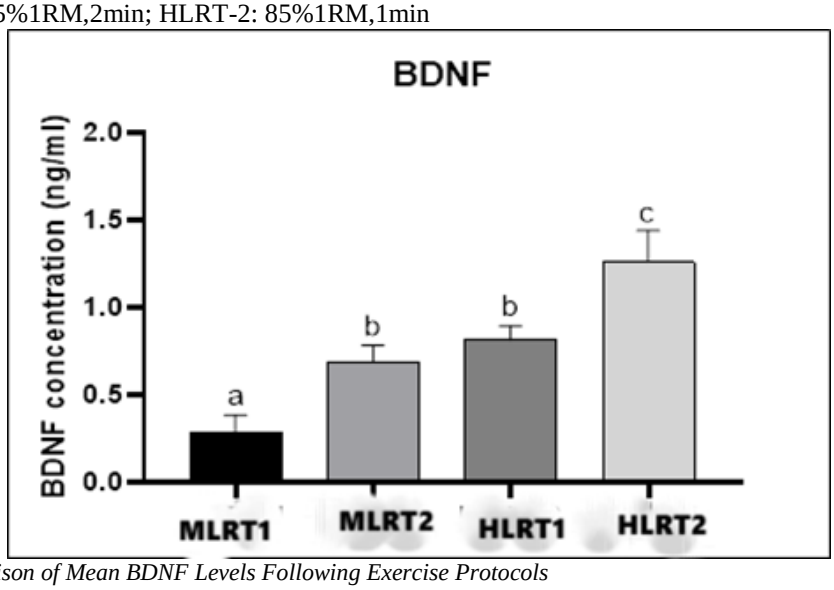

Figure 3. *Comparison of Mean BDNF Levels Following Exercise Protocols*
*"BDNF levels 36h post-exercise. HLRT2 (85% 1RM + 1-min rest) elicited the greatest increase in BDNF compared to other protocols ($p < 0.0001$). Data = mean ± SD; **$p < 0.001$."
MLRT-1:75%1RM,2min; MLRT-2: :75%1RM,1 min; HLRT-1: :85%1RM,2min; HLRT-2: 85%1RM,1min

4. Discussion

This study examined the effects of a bout of resistance activity at two intensities (75% and 85% of one-repetition maximum) and two recovery periods (one and two minutes) on proinflammatory (IL-6), inflammatory (ACTH), and neurotrophic (BDNF) factor levels in female bodybuilders. Results indicated that both activity intensity and recovery duration significantly influenced these biomarkers.

Our findings indicate that, 36 hours post-exercise, the pro-inflammatory factor IL-6 significantly decreased with higher exercise intensity and shorter recovery periods. Proven conclusions can be seen that even single sessions of resistance activities can effectively regulate performance indicators in athletes(16). Muscle contraction force production was positively associated with IL-6 gene expression levels ($\beta=0.65$, SE=0.12) (17). Resistance exercise can induce muscle damage, typically triggering a cytokine production-related physiological inflammatory response. However, cytokine responses may vary depending on exercise type, intensity, duration, and session

recovery time. The primary mechanism involves reducing protein degradation(18). During resistance exercise, IL-6 levels rise acutely post-exercise but subsequently stimulate the release of anti-inflammatory markers(19). Repeated acute IL-6 surges accumulate over time in trained individuals, shifting the physiological state from pro-inflammatory to anti-inflammatory (20). Notably, the impact of resistance exercise on pro-inflammatory factors differs in adapted bodybuilders. Adaptation reduces pro-inflammatory factor production. Pro-inflammatory markers such as IL-6 and TNF-α peak 24 hours post-exercise, subsequently stimulating the release of anti-inflammatory factors like IL-10, suppressing IL-6 and TNF-α levels after this timeframe (21).

A key finding of the current study is the elevation of anti-inflammatory factors observed 36 hours post-exercise (during blood sampling), which correlated with a decline in pro-inflammatory markers. In exercise, IL-6 regulation is intensity-dependent (22). Both receptors directly engage Janus kinase, and all three signaling pathways depend on phosphorylation for activation(23).

Our findings indicate that, based on the results, ACTH levels significantly decreased 36 hours after exercise with increased exercise intensity and reduced recovery time. Consistent with our findings, Steiner et al. demonstrated that resistance activity reduces plasma ACTH levels in bodybuilder athletes (24). Similarly, Dovan et al. reported decreased ACTH alongside increased testosterone following resistance exercise (25). resistance activity was more effective than aerobic exercise in reducing both cortisol and ACTH levels(26). In contrast, a research study reported elevated ACTH levels following high-intensity resistance activity(27). Similarly, Bermejo et al. (2022) documented a significant increase in cortisol after acute resistance exercise(28). These divergent outcomes appear attributable to variations in exercise protocols and intensity levels.

ACTH, a key hormone in stress response, frequently shows elevated levels in female bodybuilders. This elevation stems from intense physical activity and exercise-induced stress, which can trigger increased cortisol production and altered ACTH secretion (29). The corticotropin-releasing hormone (CRH) mediates stress adaptation through the hypothalamic-pituitary-adrenal (HPA) axis, coordinating behavioral and autonomic stress responses. CRH neuron activation CRH neuronal activation is mediated by afferent signaling from both brainstem and limbic system structures, which coordinately regulate the sequential synthesis and release of CRH. This, in turn, stimulates anterior pituitary secretion of ACTH (30). CRH synthesis is modulated via the NF-κB signaling pathway (31).

Our results demonstrate that 36 hours post-exercise, BDNF levels increased significantly with higher exercise intensity and shorter recovery periods compared to the other three exercise protocols. These findings align with the cross-sectional study by Wang et al. (2020), titled "The Effect of Resistance Exercise on Inflammation and Neurotrophic Factors in Obese Elderly Women. The authors established that structured resistance exercise in obese elderly females significantly: (i) increases lean muscle mass, (ii) downregulates pro-inflammatory cytokines, and (iii) upregulates neurotrophic factor expression (32). Key findings demonstrated: (1) a significant increase in plasma brain-derived neurotrophic factor (BDNF) levels following exercise sessions, (2) concurrent measurements of vascular endothelial growth factor (VEGF) in serum samples, and (3) consistent neurotrophic responses irrespective of blood flow restriction (BFR) conditions (33). The observed increase in brain-derived neurotrophic factor (BDNF) may exert anti-inflammatory effects through tropomyosin receptor kinase B (Trk B) activation, subsequently inhibiting nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κ B) signaling(34). This mechanism is supported by in vitro studies showing a 40% reduction in IL-6 secretion following Trk B activation (35). BDNF, essential for neuronal maintenance and plasticity, increases significantly post-resistance activity in female bodybuilders (36).

In contrast to these studies, recent evidence suggests that resistance exercise program configuration significantly influences circulating brain-derived neurotrophic factor (BDNF) responses. Notably, light-load resistance activities may induce a more pronounced acute increase in BDNF compared to heavy-load resistance training (37). In other words, Current evidence indicates that heavy-load resistance training does not elicit significant increases in circulating brain-derived neurotrophic factor (BDNF) levels (38). This contrasts with light-to-moderate intensity resistance exercise, which consistently demonstrates measurable BDNF elevation (39). The observed discrepancies may stem from variations in participants' exercise history and/or differences in post-exercise blood collection timing across studies.

The study’s limitations include the lack of analysis across varying blood collection times and age groups, particularly in postmenopausal women, women without a bodybuilding history, and men. Baseline variables were also not measured.

5. Conclusion

This study demonstrates that high-intensity resistance exercise with shorter recovery periods elicits beneficial effects, including: Reduced levels of pro-inflammatory markers (IL-6) and ACTH, and Increased concentrations of neurotrophic factors (BDNF). results could guide the design of evidence-based training regimens for female bodybuilders by systematically regulating neurotrophic factor release and inflammatory markers. However, further research is required to investigate the long-term adaptations to such activity regimens.

Our findings suggest that structured resistance training likely simultaneously regulates inflammatory markers and neurotrophic pathways in elite female bodybuilders and provide an evidence-based physiological framework for: (1) optimizing training cycles, and (2) maintaining professional health in this specialized athlete population.

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Conflict of interests: The authors declare that there is no conflict of interest regarding the publication of this manuscript.

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