2025. *Journal of Exercise & Organ Cross Talk*. 2025; 5 (1): 1-6. doi: 10.22122/jeoct.2025.522762.1151

**Research Article**

# Acute hormonal and myokine responses to traditional vs. circuit resistance training in high-BMI and low-BMI males

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## Abstract

This study examined the acute hormonal and myokine responses to traditional and circuit resistance training in young males with distinct BMI-based groups. Twenty sedentary men aged 20–30 years were classified as high-BMI (BMI >29.9) or low-BMI (BMI <18.5). Each participant completed both traditional resistance training (TRT) and circuit resistance training (CRT) protocols in a crossover design, with a one-week washout period. Sessions included multi-joint upper and lower body exercises at comparable intensities. Blood samples were taken immediately before and after each session. Serum levels of testosterone, cortisol, myostatin, and follistatin were assessed using ELISA kits. A repeated-measures ANOVA was used to compare within- and between-group changes across time and training modality. Baseline cortisol levels were significantly higher in low-BMI individuals (P=0.037), while testosterone levels showed no initial difference between groups (P>0.05). Post-TRT, testosterone levels increased significantly in high-BMI individuals compared to low-BMI individuals (P=0.017), with both training types elevating testosterone and cortisol across all participants (P<0.05). CRT led to a significant reduction in myostatin and increase in follistatin in high-BMI individuals (P<0.05), while only follistatin increased significantly in low-BMI individuals after TRT (P<0.05). These results suggest that CRT promotes superior anabolic signaling in high-BMI individuals, while TRT is more effective at enhancing testosterone response. Myokine and hormonal responses appear to be body-type-dependent and training-modality-specific. Practitioners designing hypertrophy-focused programs should tailor training protocols to the athlete's BMI-based group to optimize muscle adaptation and endocrine outcomes. And also, trainers should individualize hypertrophy programs based on body composition to optimize both hormonal responses and muscle adaptation.

*Key Words:* Resistance training, Testosterone, Cortisol, Myostatin, Follistatin, Circuit training, Somatotype

## Introduction

Resistance training (RT) is widely recognized for its role in promoting muscular hypertrophy, strength development, and favorable metabolic adaptations (Phillips et al., 2012). Among its modalities, traditional resistance training (TRT), involving sequential sets with rest intervals and circuit resistance training (CRT), characterized by high-density, continuous exercise have shown distinct physiological impacts, especially on hormonal and cellular signaling responses (Alcaraz et al., 2008). These physiological responses are not only determined by training mode but also appear to vary based on individual morphological characteristics such as BMI-based group (Nindl et al., 2001).

Body composition classification, based on the BMI-based proxy (note: not formal Heath-Carter method) method, classifies individuals as low-BMI (lean and linear), mesomorphic (muscular), or high-BMI (rounded and adipose) (Carter & Heath, 1990). These body types differ not only in appearance and metabolism but also in endocrine profile and responsiveness to mechanical and metabolic stressors (Chao et al., 2011). For example, previous studies have shown that low-BMI individuals exhibit greater sympathoadrenal responses to aerobic exercise, while high-BMI individuals show greater insulin resistance and heightened cortisol levels at rest (Velasquez et al., 2018). Hormones like testosterone and cortisol are central to the anabolic–catabolic balance post-exercise and play a decisive role in muscle remodeling, recovery, and adaptation (Crewther et al., 2006). In addition to classical hormones, myokines, specifically myostatin and follistatin—have emerged as critical regulators of muscle growth. Myostatin acts as a potent inhibitor of hypertrophy by limiting satellite cell proliferation and protein synthesis (McPherron et al., 1997), while follistatin antagonizes myostatin by binding to and neutralizing its function, thereby promoting muscle growth (Amthor et al., 2004). Resistance training has been shown to modulate these signaling molecules, but the effects appear to differ based on training intensity, duration, and participant characteristics such as adiposity or lean mass (Walker et al., 2015; Kim et al., 2005).

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**Received 11 January 2025; Accepted 28 March 2025**

Recently, few studies have simultaneously examined both endocrine (testosterone, cortisol) and molecular (myostatin, follistatin) responses to acute resistance exercise, particularly in individuals with contrasting BMI-based groups. Moreover, the relative effectiveness of CRT versus TRT in inducing anabolic responses across body types remains unclear. Given that myostatin and follistatin are now recognized as therapeutic targets for sarcopenia, cachexia, and obesity-related muscle loss (Lee, 2021), understanding how they respond to exercise in different body types has both athletic and clinical implications.

Therefore, this study aimed to investigate the acute effects of traditional and circuit resistance training on serum testosterone, cortisol, myostatin, and follistatin levels in young sedentary males with high-BMI and low-BMI body types. By comparing hormonal and myokine responses across two distinct training modalities and BMI-based groups, this study offers new insights into how body composition shapes endocrine and muscular adaptation to resistance exercise.

## Materials and methods

### Subjects

This experimental randomized crossover design study aimed to examine acute hormonal and myokine responses under controlled conditions. Twenty sedentary but otherwise healthy young men (age: 24.1 ± 3.2 years, BMI: high-BMI individuals 30.4 ± 1.2 kg/m²; low-BMI individuals 17.8 ± 0.6 kg/m²) were purposefully selected based on Body type classification which in this study was based on BMI thresholds as a proxy for BMI-based group rather than the full BMI-based proxy (note: not formal Heath-Carter method) anthropometric method. Participants were therefore classified into 'high-BMI' and 'low-BMI' groups rather than precise BMI-based groups. Participants were stratified into two BMI-based group groups: high-BMI individuals (n=10) and low-BMI individuals (n=10). Eligibility criteria included no musculoskeletal injuries, no history of resistance training in the past six months, and no use of supplements, tobacco, or medications. Ethical approval was granted by the Shahid Beheshti University Ethics Committee (IR.SBU.REC.1403.018), and all subjects provided written informed consent prior to participation.

### Exercise training protocols

Traditional Resistance Training (TRT) and Circuit Resistance Training (CRT)—separated by a one-week washout period. A familiarization session was held before testing to standardize instruction and ensure participants understood the protocol. One-repetition maximum (1RM) values were estimated using the Brzycki equation based on submaximal testing to ensure participant safety. This method was chosen due to the sedentary status of participants, minimizing injury risk while providing reliable load estimations (Fasihiyan et al., 2023).

To account for workload discrepancies, the CRT and TRT protocols were designed to achieve an approximately equivalent training volume per exercise. This was accomplished by reducing the load intensity in the CRT protocol (50% 1RM) while increasing the number of repetitions (15 reps per set), in contrast to the TRT protocol which used higher intensity (70–80% 1RM) with fewer repetitions (8 reps per set). Although the structure and pacing of the workouts differed, the total mechanical work was equated across conditions to isolate the effects of training modality from total workload. We acknowledge that exact equivalence in neuromuscular demand is difficult to achieve, but this design allowed for a balanced comparison of circuit versus traditional formats within practical constraints. This equivalence was calculated using an iso-volume formula, where total training volume per exercise was estimated as the product of the number of repetitions and the amount of weight lifted (repetitions × load). This method allowed us to design CRT and TRT protocols with comparable total workloads, despite differences in training structure. While CRT used lower resistance and higher repetitions (15 reps at 50% 1RM), TRT employed higher resistance and fewer repetitions (8 reps at 70–80% 1RM), yielding a similar training volume across modalities.

Each workout began with 10 minutes of dynamic warm-up and light aerobic exercise. The TRT protocol involved 3 sets of 8 repetitions at 70–80% of 1RM for 8–12 repetitions of each exercise: (1) barbell bench press, (2) machine abdominal crunch, (3) leg press, (4) wide-grip front Lat pulldown, (5) machine back extension, and (6) barbell squat. Rest intervals were fixed at 120 seconds between sets and exercises. The CRT protocol used the same exercises but at 50% of 1RM, with 15 repetitions per movement. Each repetition followed a strict tempo of 1-1-1-1 (1 second eccentric, pause, concentric, pause), with 15–20 seconds rest between exercises and 2–3 minutes of active rest between circuits. The CRT protocol was repeated for three full-body rounds.

### Laboratory measurements

Blood samples (5 mL) were collected via venipuncture from the antecubital vein immediately before and 10 minutes after each session. Samples were centrifuged at 3, 000 rpm for 15 minutes, and serum was stored at –80°C for batch analysis. Serum testosterone and cortisol concentrations were measured using commercial ELISA kits (GeneBio Systems, Canada). Myostatin was assessed using the Human GDF-8/Myostatin ELISA Kit (RayBiotech, USA; Cat. No. ELH-GDF8), and follistatin was measured with the Human Follistatin Quantikine ELISA Kit (R&D Systems, USA; Cat. No. DFN00). All assays were run in duplicate, and optical density was read at 450 nm using a BioTek

ELx800 microplate reader.

### Statistical analysis

Statistical analysis was performed using SPSS version 23. The Shapiro-Wilk test confirmed normality, and Levene's test verified homogeneity of variances. Two-way repeated-measures ANOVA was used to examine the interaction between time (pre/post) and group (high-BMI individual/low-BMI individual) for each biomarker. Bonferroni correction was applied for multiple comparisons during post hoc testing to control for Type I error. Significance was accepted at $p<0.05$.

## Results

All 20 participants completed both training protocols without adverse events. Baseline characteristics, including age and BMI, were significantly different between high-BMI and low-BMI groups ($p<0.01$), but no initial differences were found in serum testosterone, myostatin, or follistatin levels ($p>0.05$). However, baseline cortisol was significantly higher in low-BMI individuals compared to high-BMI individuals (24.5 ± 2.3 vs. 18.5 ± 2.0 μg/dL; $p=0.037$).

Both training protocols induced significant changes in hormonal and myokine concentrations, with modality- and BMI-based group-specific patterns. For testosterone, TRT produced a significantly greater increase in high-BMI individuals (from 3.1 ± 0.5 to 4.5 ± 0.6 ng/mL; $p=0.017$; Cohen's d = 2.8; 95% CI [1.09, 1.71]) compared to low-BMI individuals (from 3.2 ± 0.6 to 3.6 ± 0.5 ng/mL; $p=0.082$; Cohen's d = 0.7; 95% CI [0.03, 0.77]). CRT led to smaller but still significant increases in testosterone for high-BMI individuals ($p<0.05$), while changes in low-BMI individuals were not statistically significant (Fig. 1).

Cortisol levels rose significantly post-exercise in both groups and both protocols ($p<0.01$), but the response magnitude was higher in low-BMI individuals. In low-BMI participants, TRT increased cortisol from 20.1 ± 2.1 to 24.5 ± 2.3 μg/dL, while CRT raised it from 19.9 ± 2.0 to 23.2 ± 2.2 μg/dL (both $p<0.01$). High-Adipose subjects showed smaller increases under both conditions ($p<0.05$) (Fig. 2).

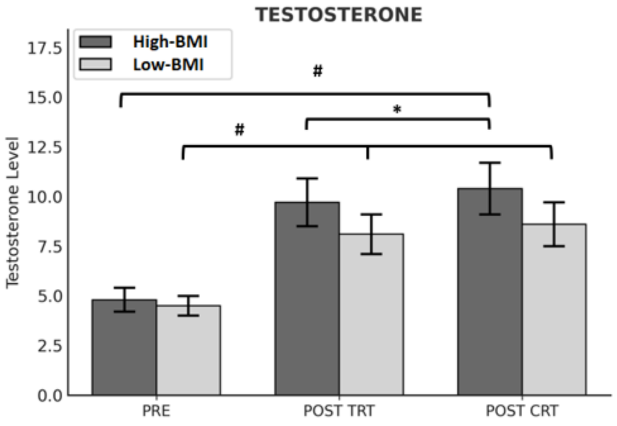

Figure 1. Changes in testosterone following PRE, POST TRT, and POST CRT in high-BMI and low-BMI participants. *p<0.05; #p<0.01.

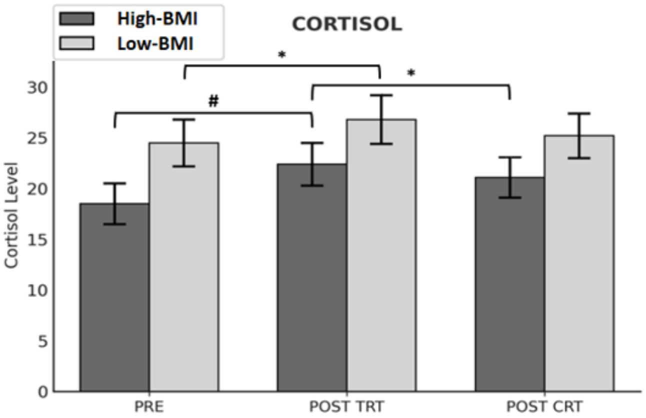

Figure 2. Changes in cortisol following PRE, POST TRT, and POST CRT in high-BMI and low-BMI participants. *p<0.05; #p<0.01.

Myostatin levels decreased significantly in high-BMI individuals following CRT (from 4.3 ± 0.5 to 3.1 ± 0.4 ng/mL; $p=0.003$; Cohen's d = -2.4; 95% CI [-1.51, -0.89]), while low-BMI individuals showed only minor, non-significant reductions. TRT led to modest myostatin suppression in both groups, but the change was only statistically significant for high-BMI individuals ($p<0.05$) (Fig. 3).

For follistatin, CRT elicited the strongest response in high-BMI individuals, increasing from 5.7 ± 0.6 to 7.2 ± 0.9 ng/mL ($p<0.01$). Low-BMI Individuals experienced a significant follistatin increase following TRT (from 5.6 ± 0.6 to 6.1 ± 0.7 ng/mL; $p<0.05$ but the change post-CRT did not reach statistical significance) (Fig. 4).

Interaction effects from repeated-measures ANOVA revealed significant training type × BMI-based group interactions for testosterone ($F=5.81$, $p=0.024$), myostatin ($F=6.42$, $p=0.019$) and

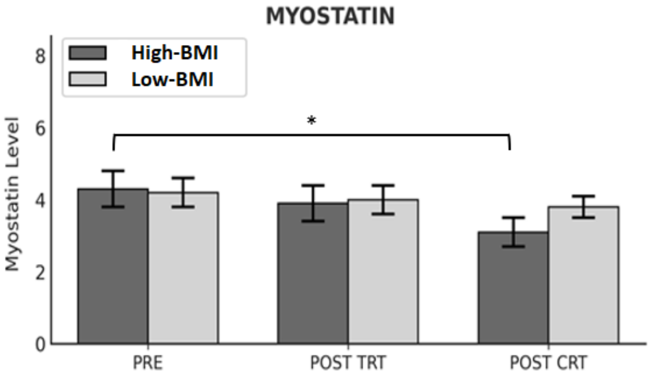

Figure 3. Changes in myostatin following PRE, POST TRT, and POST CRT in high-BMI and low-BMI participants. *p<0.05.

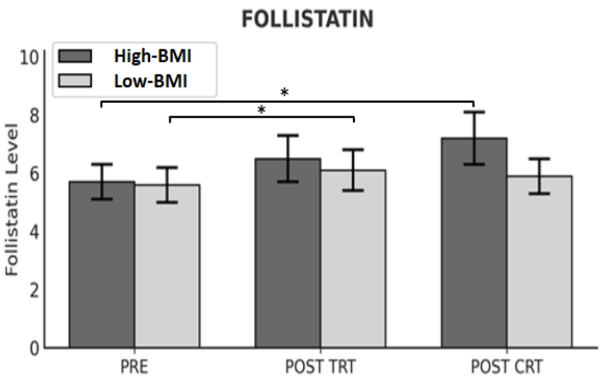

Figure 4. Changes in follistatin following PRE, POST TRT, and POST CRT in high-BMI and low-BMI participants. *p<0.05; #p<0.01.

follistatin (F=5.17, p=0.031), indicating that physiological responses varied by both body type and training modality. Significant changes between groups are annotated in figures using * and # to indicate statistical significance.

## Discussion

These findings suggest that acute hormonal and myokine responses to resistance training are appears to have influenced by both BMI-based group and exercise modality. The results suggest that high-BMI individuals, characterized by higher adiposity, exhibit more pronounced anabolic responses to both TRT and CRT protocols, particularly in testosterone and follistatin levels. This aligns with prior findings indicating that individuals with greater fat mass may respond differently to training-induced stress and endocrine signaling due to altered baseline hormonal environments (Chao et al., 2011; Velasquez et al., 2018).

Testosterone, a key anabolic hormone, increased significantly post-TRT in high-BMI individuals compared to low-BMI individuals, supporting the notion that heavier individuals may derive greater acute anabolic benefit from high-intensity, lower-volume protocols (Crewther et al., 2006). Although low-BMI individuals also showed increased testosterone, the change did not reach significance, potentially due to lower initial muscle mass and metabolic reserve. Cortisol, a catabolic hormone, rose significantly in both groups, with low-BMI individuals showing a more robust response. This observation may reflect increased physiological strain or sympathetic activation in leaner individuals, consistent with evidence of heightened sympathoadrenal activity in this group (Velasquez et al., 2018). Elevated cortisol could also partly explain the blunted testosterone rise in low-BMI individuals, given cortisol's known antagonistic effects on anabolic hormones (Nindl et al., 2001). Regarding myokines, CRT led to a notable decrease in myostatin and an increase in follistatin among high-BMI individuals, indicating a favorable anabolic shift. These changes suggest that CRT may serve as a more effective modality for improving the muscle-building environment in individuals with greater adiposity, likely due to its higher training density and metabolic demand (Alcaraz et al., 2008). The findings align with Kim et al. (2020) and Walker et al. (2015), who reported similar benefits in overweight individuals following circuit-style protocols. In contrast, TRT seemed to elicit a better follistatin response in low-BMI individuals, even in the absence of significant myostatin suppression. This suggests that traditional set structures might be more beneficial for leaner individuals by promoting a more sustainable anabolic signal without overwhelming metabolic stress. It is also possible that repeated CRT bouts in low-BMI individuals would induce chronic adaptations not captured in this acute design.

Overall, the interaction effects confirm that training prescriptions should not be universally applied but rather tailored to body composition characteristics. These insights are clinically relevant for strength coaches, physical therapists, and clinicians aiming to optimize muscle hypertrophy and recovery across varying BMI-based groups. However, the study measured hormonal responses only at 10 minutes post-exercise. Given that testosterone may peak between 15–30 minutes post-exercise, the chosen sampling window might have underestimated peak hormonal responses. Future studies should incorporate serial sampling at multiple time points to better capture the full hormonal response profile. Although previous studies suggested higher baseline cortisol in individuals with greater adiposity, our findings demonstrated higher resting cortisol in low-BMI participants. This discrepancy may be attributed to differences in sample characteristics, measurement protocols, or the psychological stress response in lean individuals. Further research is required to clarify the relationship between body composition and baseline cortisol levels.

Another important limitation is the timing of blood sampling. In this study, hormonal responses, particularly testosterone, were measured only at a single time point—10 minutes post-exercise. However, previous literature suggests that testosterone levels may peak between 15 and 30 minutes following resistance training. This timing mismatch may have led to an underestimation of peak hormonal responses. Future studies should include serial post-exercise sampling (e.g., 0, 15, 30, and 60 minutes) to more accurately characterize the temporal dynamics of hormonal fluctuations.

Future research should examine chronic adaptations to both CRT and TRT in larger cohorts, include mesomorphic individuals for comparative analysis, and investigate downstream effects on satellite cell activation and mTOR signaling to clarify the mechanisms behind the observed hormonal shifts.

## Conclusion

It is worth noting that acute hormonal and myokine responses to resistance training are significantly modulated by both BMI-based group and exercise modality. High-Adipose individuals exhibited more favorable anabolic profiles following circuit resistance training, characterized by increased testosterone and follistatin levels and reduced myostatin. Low-BMI Individuals responded better to traditional resistance training for increasing follistatin, though their testosterone response was less pronounced. These findings underscore the importance of tailoring resistance training protocols based on body composition to optimize hormonal and molecular environments for muscle growth and recovery.

## What is already known on this subject?

Resistance training induces hormonal and myokine responses that play critical roles in muscle adaptation, Traditional and circuit resistance training modalities produce different physiological effects, and Somatotype influences baseline hormonal profiles and exercise responsiveness.

## What this study adds?

This research explores of myokine dynamics in relation to BMI-based group-specific responses, providing new insights into body-type-dependent adaptations to resistance training. Also, high-adipose individuals may benefit from high-density circuit sessions spaced 48 hours apart to maximize follistatin elevation and minimize myostatin rebound. For low-BMI participants, ensure adequate post-exercise carbohydrate intake and emphasize relaxation techniques post-training to counteract excessive cortisol elevation.

Organ Cross-Talk Tips:

- Prioritize body-type-specific training to leverage hormonal and myokine pathways for improved muscular adaptations.
- For high-BMI individuals: Implement high-density, circuit-style protocols to maximize anabolic signaling and myokine balance.
- For low-BMI individuals: Utilize traditional training protocols emphasizing progressive overload with sufficient rest to support hormonal responses without excessive catabolic stress.
- Monitor training-induced hormonal fluctuations to refine and adjust programming strategies accordingly.

## Acknowledgements

The authors wish to thank all participants for their commitment and effort throughout the study. Appreciation is also extended to the staff at Shahid Beheshti University for their support in data collection and laboratory analyses.

## Funding

No sources of funding were sought or awarded for this study.

## 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 they have no conflict of interest.

### Ethical approval

This study's procedures were carried out under the Declaration of Helsinki regarding human research. Ethical approval was granted by the Shahid Beheshti University Ethics Committee (IR.SBU.REC.1403.018).

### Informed consent

Participants signed an informed consent form prior to participation in the study

## Author contributions

Conceptualization: S.K., M.F., M.N.; Methodology: M.F., M.N.; Software: S.K., B.B.; Validation: M.F., S.K. Formal analysis: S.K., M.F., M.N.; Investigation: S.K., B.B.; Resources: S.K., M.F., M.N.; Data curation: S.K., B.B.; Writing - original draft: M.F., S.K.; Writing–review & editing: S.K., M.F., M.N.; Visualization: M.F.; Supervision: M.F.; Project administration: M.F.; Funding acquisition: M.F.

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