Research Article

# DeLorme-Watkins or high-intensity pyramidic training protocol: Which one has more effectiveness on adiponectin and TNF-α?

**Keyvan Ahmadi Dehrashid**[1*]

## Abstract

This study aimed to investigate the effects of two different resistance training protocols on TNF-α and adiponectin in young overweight men. Sixty healthy overweight men (BMI ≥ 25 kg/m², age 20–30 years) were randomly assigned to three groups: Experimental Group 1 (EG1), Experimental Group 2 (EG2), and Control Group (CG). EG1 performed the DeLorme-Watkins protocol, consisting of 3 sets of 10 repetitions at 50%, 75%, and 100% of 10RM, while EG2 performed HIPT, consisting of 3 sets (set 1: 6 repetitions at 70% of 1RM; set 2: 4 repetitions at 80% of 1RM; set 3: 2 repetitions at 90% of 1RM). Training lasted 8 weeks. TNF-α and adiponectin levels were measured pre- and post-intervention. One-way ANOVA with Tukey post hoc tests and paired-samples t-tests were used to analyze inter- and intra-group differences (α≤0.05). TNF-α significantly decreased in EG1 (P=0.04), while adiponectin levels significantly increased in both EG1 (P=0.02) and EG2 (P=0.03) at post-test compared to pre-test. Additionally, TNF-α levels were significantly lower in EG1 than in CG at post-test (P=0.01). Both resistance training protocols exerted beneficial effects on inflammatory and anti-inflammatory markers, potentially contributing to cardiovascular disease prevention. However, the DeLorme-Watkins protocol resulted in a significantly greater reduction in TNF-α levels compared to HIPT, whereas no significant between-group difference was observed for adiponectin.

*Key Words*:Resistance exercise training, TNF-α, Adiponectin, Inflammatory markers, Cardiovascular disease

## Introduction

Cardiovascular diseases (CVDs) are increasingly recognized as chronic inflammatory disorders (Ruparelia et al., 2017). Inflammation plays a central role in all stages of atherosclerosis, including initiation, progression, and plaque rupture (Golia et al., 2014). Excessive body fat accumulation exacerbates inflammation and thrombosis (Koliaki et al., 2019), as hypertrophy and hyperplasia of adipose tissue stimulate the production of pro-inflammatory cytokines such as C-reactive protein (CRP) and tumor necrosis factor-alpha (TNF-α), while reducing the synthesis of adiponectin — an adipokine with anti-inflammatory and anti-atherogenic properties (Klöting & Blüher, 2014; Landgraf et al., 2015; Mathieu et al., 2010). Therefore, obesity represents a chronic low-grade inflammatory state. Physical inactivity, increasingly prevalent due to industrialization, has contributed to a global rise in overweight and obesity (Rosengren, 2021). Individuals with excess body fat typically exhibit elevated inflammatory markers and reduced adiponectin levels, collectively promoting CVD development (Cobos-Palacios et al., 2022).

Among the signaling pathways linking obesity and inflammation, TNF-α plays a central role by promoting the expression of multiple pro-inflammatory cytokines. Its effects are largely mediated through activation of the nuclear factor kappa B (NF-κB) pathway, which is markedly upregulated in obese individuals (Carlsen et al., 2009). Activation of NF-κB by TNF-α amplifies inflammatory signaling, contributing to early atherogenesis and, over time, vascular occlusion (Couch et al., 2022; Dhiman et al., 2021; Schütze et al., 1995). Thus, TNF-α, via NF-κB activation, represents a key mechanistic link between adiposity-induced inflammation and cardiovascular disease progression. Conversely, adiponectin exerts protective cardiovascular effects by downregulating TNF-α and enhancing endothelial function (Ouchi & Walsh, 2007). Conversely, adiponectin exerts protective cardiovascular effects by counteracting the pro-inflammatory actions of TNF-α. It downregulates TNF-α expression and inhibits NF-κB–mediated

1. Assistant Professor, Department of Physical education and sport sciences, Payame Noor University (PNU), Tehran, Iran.

*Author for correspondence: ahmadi.kaiwan@pnu.ac.ir

K A D: 0000-0003-2159-9027

**Received 15 December 2025; Accepted 16 February 2026**

inflammatory signaling, thereby reducing CRP production, lowering adhesion molecule expression, and enhancing endothelial function (Ouchi & Walsh, 2007).

Through these mechanisms, adiponectin mitigates obesity-associated inflammation and contributes to the prevention of early atherogenesis and vascular dysfunction (Ouchi & Walsh, 2007). Adiponectin exerts protective effects against obesity-associated inflammation primarily by modulating TNF-α activity. In cardiac cells, it reduces TNF-α production through the induction of cyclooxygenase-2 (COX-2) and prostaglandin E2 (PGE2), thereby protecting the myocardium from ischemia-reperfusion injury (Takemura et al., 2007; Wang et al., 2020). In macrophages, adiponectin attenuates TNF-α and interleukin-6 (IL-6) production by inhibiting NF-κB signaling, while simultaneously enhancing anti-inflammatory mediators such as IL-10 and TIMP-1 (Lira et al., 2012). Furthermore, adiponectin prevents macrophage-to-foam cell transformation (Huang et al., 2008), and reduces cholesterol ester accumulation by downregulating scavenger receptor class A (SR-A) expression (Ouchi et al., 2001). Through cAMP–PKA–dependent pathways, adiponectin further suppresses NF-κB activation and promotes endothelial nitric oxide synthase (eNOS) phosphorylation, supporting vascular function and reducing inflammatory risk (Chen et al., 2003; Chen et al., 2017). Collectively, these mechanisms highlight how adiponectin directly counteracts TNF-α–mediated inflammation, providing a biological basis for investigating interventions, such as resistance training, that may enhance adiponectin levels and reduce inflammatory markers in overweight individuals.

A growing body of evidence supports the beneficial effects of physical exercise—particularly resistance training—on inflammatory and anti-inflammatory markers, including TNF-α and adiponectin (Gondim et al., 2015; Markofski et al., 2014; Montrezol et al., 2014). Resistance training has been shown to favorably modulate systemic inflammation, making it a key component of public health strategies for overweight and obese individuals (Strasser et al., 2012). For example, Santiago et al. (2018) reported significant reductions in TNF-α following resistance training compared with controls (Macêdo Santiago et al., 2018). Similarly, Montrezol et al. (2014) observed that resistance training increased adiponectin levels and decreased ICAM-1 concentrations in overweight subjects (Montrezol et al., 2014). Eskandari et al. (2021) demonstrated reductions in TNF-α after both upper- and lower-body resistance training, (Eskandari et al., 2021). While Shultz et al. (2015) found that 16 weeks of resistance training in overweight women decreased TNF-α and increased adiponectin levels (Shultz et al., 2015). Despite these promising findings, few studies have directly compared the effects of different resistance training protocols on TNF-α and adiponectin in young overweight men. Therefore, the present study aimed to investigate and compare the effects of the DeLorme-Watkins and HIPT resistance training protocols on inflammatory and anti-inflammatory markers, providing insights into optimal strategies for reducing obesity-related inflammation.

## Materials and methods

### Study design

The statistical population of this study consisted of healthy young men classified as overweight. From all volunteers, 60 overweight but otherwise healthy men (BMI ≥ 25 kg/m²), aged 20–30 years, who had not engaged in any regular exercise for at least one year prior to the study, were recruited. None of the participants had any known chronic, cardiovascular, or immune-related disorders, nor were they taking any medications or experiencing musculoskeletal limitations. Health status was verified through completion of the Beck Health Questionnaire, and all participants provided written informed consent prior to participation. During the initial session, anthropometric measurements (height, weight), as well as one-repetition maximum (1RM) and ten-repetition maximum (10RM) tests, were performed. Proper execution of each resistance exercise was also demonstrated and practiced under supervision. Participants were randomly assigned into three groups:

• Experimental Group 1 (EG1; n = 20)

• Experimental Group 2 (EG2; n = 20)

• Control Group (CG; n = 20)

### Exercise program

The intervention period lasted 8 weeks, with four alternating training sessions per week. Subjects in experimental group 1 (EG1) performed progressive resistance training based on the DeLorme–Watkins method (Westcott & Faigenbaum, 2003).

Each session consisted of three sets of 10 repetitions with 1–2 minutes of rest between sets:

• Set 1: 10 repetitions at 50% of 10RM

• Set 2: 10 repetitions at 75% of 10RM

• Set 3: 10 repetitions at 100% of 10RM

To ensure progressive overload, the 10RM was reassessed every two weeks, and training loads were adjusted accordingly. This approach ensured that participants consistently trained at the intended intensity throughout the 8-week intervention. Subjects in experimental group 2 (EG2) performed high-intensity pyramidal resistance training (HIPT) based on 1RM (Mang et al., 2021).

Each session included three sets with 1–3 minutes of rest:

• Set 1: 6 repetitions at 70% of 1RM

• Set 2: 4 repetitions at 80% of 1RM

• Set 3: 2 repetitions at 90% of 1RM

The training circuit consisted of eight resistance exercises targeting the major muscle groups: quadriceps, hamstrings, biceps, triceps, chest, back, legs, and forearms. The exercise stations included the lying leg press, lying leg curl, barbell biceps curl, cable triceps extension, barbell bench press, seated cable row, calf raise machine, and barbell wrist curl. Proper exercise technique was demonstrated by the investigator and supervised throughout the sessions to ensure safety and adherence to the training protocol. For EG1, the 10RM was reassessed every two weeks, and training loads were adjusted accordingly to ensure progressive overload throughout the 8-week intervention. For EG2 (HIPT), progressive overload was ensured by adjusting training intensities according to the predetermined percentages of 1RM, which were reassessed at week 4 to reflect changes in maximal strength, thereby maintaining systematic progression over the 8 weeks. Participants in the control group maintained their usual daily routines and did not participate in any structured exercise program during the 8-week intervention.

### Ethical considerations

This study was conducted in accordance with the ethical standards of the Institutional Research Committee and the 1964 Helsinki Declaration. All participants provided written informed consent prior to participation.

### Laboratory measurements

All participants followed their habitual diet throughout the study period. To assess serum levels of TNF-α and adiponectin before and after the intervention, fasting blood samples (5 mL) were collected from the antecubital vein between 8:00–9:00 a.m. After a 10-hour overnight fast. Serum TNF-α concentrations were determined using a commercially available ELISA kit from the same manufacturer according to the manufacturer's instructions, and Serum adiponectin concentrations were measured using a commercially available ELISA kit (Cat. No. CK-E10871; Hangzhou Eastbiopharm Co., Ltd., Hangzhou, China) according to the manufacturer's instructions. Measurements were performed using a Cobas Integra Analyzer (Roche Diagnostics, Germany) according to the manufacturer's instructions.

### Statistical analysis

Data normality was verified using the Kolmogorov–Smirnov test. Between-group differences in TNF-α and adiponectin concentrations were analyzed using one-way ANOVA followed by Tukey's post hoc test. Within-group (pre–post) changes were evaluated using paired-sample t-tests. Statistical significance was accepted at $p \leq 0.05$. All analyses were performed using SPSS software version 20 (IBM Corp., Armonk, NY, USA).

## Results

Sixty participants were randomly assigned into three groups. Their baseline characteristics, including height, weight, age, and BMI, are presented in Table 1.

Paired-sample t-tests revealed a significant decrease in TNF-α levels in Experimental Group 1 (EG1) at post-test compared to pre-test (P=0.04). Adiponectin levels increased significantly in both Experimental Groups 1 and 2 (EG1 and EG2) following the 8-week intervention (P=0.02 and P=0.03, respectively), whereas no significant changes were observed in the Control Group (CG) for either TNF-α (P=0.68) or adiponectin (P=0.62) [Table 2].

One-way ANOVA indicated no significant differences in TNF-α or adiponectin levels among the groups at baseline, confirming homogeneity prior to the intervention (TNF-α: F=0.121, P= 0.886; adiponectin: F=0.025, P=0.976). Following the 8-week intervention, one-way ANOVA revealed a significant effect of group on TNF-α levels (F=4.093, P=0.022), whereas adiponectin levels did not differ significantly between groups (F=2.254, P=0.114). Tukey's post hoc analysis showed that TNF-α levels in EG1 were significantly lower than those in the control group (P=0.019), while no other pairwise comparisons reached statistical significance [Tables 3–4].

## Discussion

**Table 1**. Baseline characteristics of participants by group (Mean ± SD). *Significant at $p \leq 0.05$

| Group | N | Age (years) | Height (cm) | Weight (kg) | BMI (kg/m²) |
|---|---|---|---|---|---|
| Experimental 1 (EG1) | 20 | 24.50 ± 2.92 | 176.15 ± 7.79 | 90.54 ± 8.22 | 29.02 ± 2.16 |
| Experimental 2 (EG2) | 20 | 23.85 ± 2.81 | 177.40 ± 10.87 | 88.41 ± 10.36 | 27.92 ± 1.46 |
| Control (CG) | 20 | 23.90 ± 3.30 | 179.25 ± 10.13 | 92.02 ± 9.81 | 28.46 ± 1.41 |
| P Value | — | 0.75 | 0.14 | 0.59 | 0.48 |

*Significant at $p \leq 0.05$

Note: Data are presented as mean ± standard deviation. P values were calculated using one-way ANOVA to compare baseline differences among groups. No significant differences were observed between groups (P > 0.05).

**Table 2**. Adiponectin and TNF-α levels (mean ± SD) before and after the intervention

| Biomarker | Group | Pre-Test | Post-Test | P Value |
|---|---|---|---|---|
| TNF-α (pg/mL) | Experimental 1 | 6.83 ± 0.96 | 6.18 ± 1.16 | P = 0.04* |
| | Experimental 2 | 6.75 ± 1.32 | 6.44 ± 0.93 | P = 0.39 |
| | Control | 6.93 ± 1.10 | 7.01 ± 0.65 | P = 0.68 |
| Adiponectin (µg/mL) | Experimental 1 | 6.14 ± 2.36 | 7.67 ± 2.05 | P = 0.02* |
| | Experimental 2 | 5.99 ± 2.29 | 7.40 ± 2.29 | P = 0.03* |
| | Control | 6.07 ± 1.93 | 6.29 ± 2.21 | P = 0.62 |

*Significant at $p \leq 0.05$

Note: Paired-sample t-tests were used to compare pre- and post-test values within each group. $P \geq 0.05$ indicates no statistically significant change

**Table 3.** One-way ANOVA for TNF-α and adiponectin at pre- and post-test.

| Variable | Test | Between Groups SS | df | Mean Square | F | P Value |
|---|---|---|---|---|---|---|
| TNF-α (pg/mL) | Pre-test | 0.315 | 2 | 0.157 | 0.121 | 0.886 |
| | Post-test | 7.268 | 2 | 3.634 | 4.093 | 0.022* |
| Adiponectin (µg/mL) | Pre-test | 0.240 | 2 | 0.120 | 0.025 | 0.976 |
| | Post-test | 21.701 | 2 | 10.851 | 2.254 | 0.114 |

*Significant at $p \leq 0.05$

Notes:

• Pre-test results indicate no significant differences among groups (P > 0.05).

• Post-test TNF-α shows a significant difference, while adiponectin differences were not significant.

**Table 4.** HSD post hoc comparisons for TNF-α at post-test

| Comparison (I–J) | Mean Difference (I–J) | Std. Error | P Value | 95% CI Lower | 95% CI Upper |
|---|---|---|---|---|---|
| EG1 – EG2 | -0.264 | 0.298 | 0.651 | -0.981 | 0.453 |
| EG1 – CG | -0.834 | 0.298 | 0.019* | -1.551 | -0.117 |
| EG2 – EG1 | 0.264 | 0.298 | 0.651 | -0.453 | 0.981 |
| EG2 – CG | -0.570 | 0.298 | 0.144 | -1.287 | 0.147 |
| CG – EG1 | 0.834 | 0.298 | 0.019* | 0.117 | 1.551 |
| CG – EG2 | 0.570 | 0.298 | 0.144 | -0.147 | 1.287 |

*Significant at $p \leq 0.05$

Notes:

• TNF-α post-test levels in EG1 were significantly lower than CG (P = 0.019).

• No other post hoc comparisons reached statistical significance.

The present study demonstrated that resistance training effectively modulates inflammatory and anti-inflammatory biomarkers in young overweight men. Specifically, TNF-α levels decreased significantly in EG1, while adiponectin levels increased similarly in both EG1 and EG2 post-intervention. This indicates that while both resistance training protocols are effective in enhancing adiponectin, the main differentiating effect between the protocols lies in the reduction of TNF-α. The significant decrease in TNF-α highlights the anti-inflammatory potential of structured resistance training. Mechanistically, this reduction may be mediated through the inhibition of the NF-κB signaling pathway, a central regulator of pro-inflammatory cytokine expression, and activation of AMP-activated protein kinase (AMPK), which has been shown to suppress TNF-α production and improve metabolic function. Through these pathways, resistance training may attenuate adiposity-induced chronic inflammation and reduce early Atherogenic processes.

These findings are consistent with previous evidence supporting resistance training as a modulator of TNF-α and adiponectin. For instance, Cobos-Palacios et al. (2022) reported that weight loss interventions significantly decreased inflammatory markers, including the TNF-α, although adiponectin remained unchanged (Cobos-Palacios et al., 2022). Lucotti et al. (2011) observed a significant decrease in TNF-α and increase in adiponectin following aerobic exercise in type 2 diabetic patients (Lucotti et al., 2011). Zaidi et al. (2021) found that combined endurance-resistance training did not significantly alter TNF-α or adiponectin, though improvements in $VO_2$ peak correlated positively with adiponectin levels (Zaidi et al., 2021). Other studies report mixed findings regarding TNF-α and adiponectin. Jadhav et al. (2021) reported that exercise reduced leptin and IL-6 but had no significant effect on TNF-α or adiponectin (Jadhav et al., 2021). Shokri et al. (2021) found that 12 weeks of combined resistance-aerobic training increased adiponectin and decreased resistin, while TNF-α remained unchanged (Shokri et al., 2021). Lim et al. (2012) observed that neither high- nor moderate-intensity aerobic training significantly altered adiponectin levels (Lim et al., 2012). Gondim et al. (2015) reported that long-term regular exercise reduced adiponectin, leptin, and resistin in overweight individuals (Gondim et al., 2015). In contrast, Hopps et al. (2011) showed that combined aerobic-resistance training decreased pro-inflammatory markers, including TNF-α, while increasing anti-inflammatory cytokines such as IL-4, IL-10, and adiponectin (Hopps et al., 2011).

Focusing specifically on resistance training, Montrezol et al. (2014) reported significant reductions in ICAM-1 alongside increases in adiponectin (Montrezol et al., 2014), while Strasser et al. (2012) demonstrated reductions in CRP and increases in adiponectin after structured resistance exercise (Strasser et al., 2012). Markofski et al. (2014) found a 55% increase in adiponectin following 12 weeks of combined training. The discrepancies among studies likely stem from variations in participant characteristics, including age, baseline activity level, and health status, as well as differences in training intensity, duration, and modality (Markofski et al., 2014). Overall, the present findings suggest that resistance training can effectively reduce TNF-α via mechanistic pathways involving NF-κB inhibition and AMPK activation, contributing to the attenuation of systemic inflammation in overweight young adults, while improvements in adiponectin appear comparable across different resistance protocols.

## Conclusion

In summary, both resistance training protocols—DeLorme-Watkins and high-intensity Pyramidal Training (HIPT)—demonstrated beneficial effects on TNF-α as a pro-inflammatory marker and adiponectin as an anti-inflammatory adipokine. Both protocols similarly increased adiponectin levels, while the reduction in TNF-α was slightly more pronounced in the DeLorme-Watkins group. These findings suggest that resistance training can effectively modulate inflammatory and anti-inflammatory biomarkers, potentially contributing to the prevention of cardiovascular disease. The observed effects are likely mediated through mechanisms such as inhibition of NF-κB signaling and activation of AMPK pathways. It should be noted that this study had some limitations, including the inability to control participants' motivation, sleep quality and duration, and other lifestyle factors that may influence cardiovascular health. Future research is recommended to explore different exercise modalities, longer interventions, and additional mechanistic pathways that may impact inflammatory and anti-inflammatory markers.

## What is already known on this subject?

Cardiovascular diseases (CVDs) are increasingly recognized as chronic inflammatory disorders. Inflammation plays a central role in all stages of atherosclerosis, including initiation, progression, and plaque rupture

## What this study adds?

Both resistance training protocols—DeLorme-Watkins and high-intensity Pyramidal Training (HIPT)—demonstrated beneficial effects on TNF-α as a pro-inflammatory marker and adiponectin as an anti-inflammatory adipokine.

**Organ Cross-Talk Tips:**

- The study highlights that exercise-induced changes in adiponectin (an anti-inflammatory hormone) and TNF-α (a pro-inflammatory cytokine) represent a direct modulation of signals originating from adipose tissue. These molecules are key messengers in the cross talk between fat tissue and other organs (such as skeletal muscle and the cardiovascular system), influencing systemic metabolism and inflammation.

## Acknowledgements

The author would like to express sincere gratitude to Payame Noor University for their support of this research.

## Funding

None.

## 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 This study was conducted in accordance with the ethical standards of the Institutional Research Committee and the 1964 Helsinki Declaration. All participants provided written informed consent prior to participation.

Informed consent Performed.

## Author contributions

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

## References

Carlsen, H., Haugen, F., Zadelaar, S., Kleemann, R., Kooistra, T., Drevon, C. A., & Blomhoff, R. (2009). Diet-induced obesity increases NF-κB signaling in reporter mice. Genes & nutrition, 4(3), 215-222. doi: https://doi.org/10.1007/s12263-009-0133-6

Chen, H., Montagnani, M., Funahashi, T., Shimomura, I., & Quon, M. J. (2003). Adiponectin stimulates production of nitric oxide in vascular endothelial cells. Journal of Biological Chemistry, 278(45), 45021-45026. doi: https://doi.org/10.1074/jbc.m307878200

Chen, Y., Zheng, Y., Liu, L., Lin, C., Liao, C., Xin, L., Zhong, S., Cheng, Q., & Zhang, L. (2017). Adiponectin inhibits TNF-α-activated PAI-1 expression via the cAMP-PKA-AMPK-NF-κB axis in human umbilical vein endothelial cells. Cellular Physiology and Biochemistry, 42(6), 2342-2352. doi: https://doi.org/10.1159/000480006

Cobos-Palacios, L., Muñoz-Úbeda, M., Gallardo-Escribano, C., Ruiz-Moreno, M. I., Vilches-Pérez, A., Vargas-Candela, A., Leiva-Gea, I., Tinahones, F. J., Gómez-Huelgas, R., & Bernal-López, M. R. (2022). Adipokines Profile and Inflammation Biomarkers in Prepubertal Population with Obesity and Healthy Metabolic State. Children, 9(1), 42. doi: https://doi.org/10.3390/children9010042

Couch, C., Mallah, K., Borucki, D. M., Bonilha, H. S., & Tomlinson, S. (2022). State of the science in inflammation and stroke recovery: A systematic review. Annals of physical and rehabilitation medicine, 65(2), 101546. doi: https://doi.org/10.1016/j.rehab.2021.101546

Dhiman, S., Kumar, I., Palia, P., Jamwal, S., & Kumar, P. (2021). TNF-α: A Benificial or Harmful Pathogenic Cytokine in Cardiovascular System. Journal of Drug Delivery and Therapeutics, 11(1), 114-120. doi: : http://dx.doi.org/10.22270/jddt.v11i1.4507

Eskandari, M., Asghari, H., Saghebjoo, M., & Kazemi, T. (2021). Short duration moderate resistance training reduces blood pressure and plasma TNF-α in hypertensive men: The importance role of upper and lower body training. Science & Sports, 36(1), e1-e11. doi: https://doi.org/10.1016/j.scispo.2019.12.005

Gondim, O. S., Camargo, V. T. N. d., Gutierrez, F. A., Martins, P. F. d. O., Passos, M. E. P., Momesso, C. M., Santos, V. C., Gorjão, R., Pithon-Curi, T. C., & Cury-Boaventura, M. F. (2015). Benefits of regular exercise on inflammatory and cardiovascular risk markers in normal weight, overweight and obese adults. PloS one, 10(10), e0140596. doi: https://doi.org/10.1371/journal.pone.0140596

Hopps, E., Canino, B., & Caimi, G. (2011). Effects of exercise on inflammation markers in type 2 diabetic subjects. Acta diabetologica, 48(3), 183-189. doi: https://doi.org/10.1007/s00592-011-0278-9

Huang, H., Park, P. H., McMullen, M. R., & Nagy, L. E. (2008). Mechanisms for the anti-inflammatory effects of adiponectin in macrophages. Journal of gastroenterology and hepatology, 23, S50-S53. doi: https://doi.org/10.1111/j.1440-1746.2007.05284.x

Jadhav, R. A., Maiya, G. A., Hombali, A., Umakanth, S., & Shivashankar, K. (2021). Effect of physical activity promotion on adiponectin, leptin and other inflammatory markers in prediabetes: a systematic review and meta-analysis of randomized controlled trials. Acta diabetologica, 58(4), 419-429. doi: https://doi.org/10.1007/s00592-020-01626-1

Klöting, N., & Blüher, M. (2014). Adipocyte dysfunction, inflammation and metabolic syndrome. Reviews in Endocrine and Metabolic Disorders, 15(4), 277-287. doi: https://doi.org/10.1007/s11154-014-9301-0

Landgraf, K., Rockstroh, D., Wagner, I. V., Weise, S., Tauscher, R., Schwartze, J. T., Löffler, D., Bühligen, U., Wojan, M., & Till, H. (2015). Evidence of early alterations in adipose tissue biology and function and its association with obesity-related inflammation and insulin resistance in children. Diabetes, 64(4), 1249-1261. doi: https://doi.org/10.2337/db14-0744

Lim, K. I., Suk, M. H., & Shin, Y. A. (2012). Effects of acute aerobic exercise on circulating adiponectin and inflammatory makers in obese middle-aged women. Korean Journal of Health Promotion, 12(4), 203-210.

Lira, F. S., Rosa, J. C., Pimentel, G. D., Seelaender, M., Damaso, A. R., Oyama, L. M., & do Nascimento, C. O. (2012). Both adiponectin and interleukin-10 inhibit LPS-induced activation of the NF-κB pathway in 3T3-L1 adipocytes. Cytokine, 57(1), 98-106. doi: https://doi.org/10.1016/j.cyto.2011.10.001

Lucotti, P., Monti, L. D., Setola, E., Galluccio, E., Gatti, R., Bosi, E., & Piatti, P. (2011). Aerobic and resistance training effects compared to aerobic training alone in obese type 2 diabetic patients on diet treatment. Diabetes research and clinical practice, 94(3), 395-403. doi: https://doi.org/10.1016/j.diabres.2011.08.002

Macêdo Santiago, L. Â., Neto, L. G. L., Borges Pereira, G., Leite, R. D., Mostarda, C. T., de Oliveira Brito Monzani, J., Sousa, W. R., Rodrigues Pinheiro, A. J. M., & Navarro, F. (2018). Effects of resistance training on immunoinflammatory response, TNF-alpha gene expression, and body composition in elderly women. Journal of aging research, 2018. doi: https://doi.org/10.1155/2018/1467025

Mang, Z., Beam, J., & Kravitz, L. (2021). Pyramid Resistance Training Programs: Which Style is Most Effective? ACSM's Health & Fitness Journal, 25(6), 28-32. doi: https://doi.org/10.1249/FIT.0000000000000719

Markofski, M. M., Carrillo, A. E., Timmerman, K. L., Jennings, K., Coen, P. M., Pence, B. D., & Flynn, M. G. (2014). Exercise training modifies ghrelin and adiponectin concentrations and is related to inflammation in older adults. Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences, 69(6), 675-681. doi: https://doi.org/10.1093/gerona/glt132

Mathieu, P., Lemieux, I., & Després, J. P. (2010). Obesity, inflammation, and cardiovascular risk. Clinical pharmacology & therapeutics, 87(4), 407-416. doi: https://doi.org/10.1038/clpt.2009.311

Montrezol, F., Antunes, H., Almeida, V., Gomes, R., & Medeiros, A. (2014). Resistance training promotes reduction in blood pressure and increase plasma adiponectin of hypertensive elderly patients. J Hypertens, 3(185), 1-6. doi: https://doi.org/10.4172/2167-1095.1000185

Ouchi, N., Kihara, S., Arita, Y., Nishida, M., Matsuyama, A., Okamoto, Y., Ishigami, M., Kuriyama, H., Kishida, K., & Nishizawa, H. (2001). Adipocyte-derived plasma protein, adiponectin, suppresses lipid accumulation and class A scavenger receptor expression in human monocyte-derived macrophages. Circulation, 103(8), 1057-1063. doi: https://doi.org/10.1161/01.CIR.103.8.1057

Ouchi, N., & Walsh, K. (2007). Adiponectin as an anti-inflammatory factor. Clinica ch acta, 380(1-2), 24-30. doi: https://doi.org/10.1016/j.cca.2007.01.026

Rosengren, A. (2021). Obesity and cardiovascular health: the size of the problem. European Heart Journal, 42(34), 3404. doi: https://doi.org/10.1093/eurheartj/ehab518

Ruparelia, N., Chai, J. T., Fisher, E. A., & Choudhury, R. P. (2017). Inflammatory processes in cardiovascular disease: a route to targeted therapies. Nature reviews cardiology, 14(3), 133-144. doi: https://doi.org/10.1038/nrcardio.2016.185

Schütze, S., Wiegmann, K., Machleidt, T., & Krönke, M. (1995). TNF-induced activation of NF-κB. Immunobiology, 193(2-4), 193-203. doi: https://doi.org/10.1016/S0171-2985(11)80543-7

Shokri, E., Heidarianpour, A., & Razavi, Z. (2021). Positive effect of combined exercise on adipokines levels and pubertal signs in overweight and obese girls with central precocious puberty. Lipids in Health and Disease, 20(1), 1-14. doi: https://doi.org/10.1186/s12944-021-01588-5

Shultz, S. P., Dahiya, R., Leong, G. M., Rowlands, D. S., Hills, A. P., & Byrne, N. M. (2015). Muscular strength, aerobic capacity, and adipocytokines in obese youth after resistance training: A pilot study. The Australasian medical journal, 8(4), 113. doi: http://dx.doi.org/10.4066/AMJ.2015.2293

Strasser, B., Arvandi, M., & Siebert, U. (2012). Resistance training, visceral obesity and inflammatory response: a review of the evidence. Obesity reviews, 13(7), 578-591. doi: https://doi.org/10.1111/j.1467-789X.2012.00988.x

Takemura, Y., Walsh, K., & Ouchi, N. (2007). Adiponectin and cardiovascular inflammatory responses. Current atherosclerosis reports, 9(3), 238-243. doi: https://doi.org/10.1007/s11883-007-0025-4

Wang, D., Zhang, S., Liu, B., Wang, B., He, S., & Zhang, R. (2020). Anti-inflammatory effects of adiponectin in cigarette smoke-activated alveolar macrophage through the COX-2/PGE2 and TLRs signaling pathway. Cytokine, 133, 155148. doi: https://doi.org/10.1016/j.cyto.2020.155148

Westcott, W. L., & Faigenbaum, A. D. (2003). Strength training for kids: Practical guidelines and recommended resistance exercises you can use today to improve youth muscle strength by as much as 74 percent! IDEA Health & Fitness Source, 21(4), 36-44.

Zaidi, H., Byrkjeland, R., Njerve, I. U., Åkra, S., Solheim, S., Arnesen, H., Seljeflot, I., & Opstad, T. B. (2021). Adiponectin in relation to exercise and physical performance in patients with type 2 diabetes and coronary artery disease. Adipocyte, 10(1), 612-620. doi: https://doi.org/10.1080/21623945.2021.1996699