# The Influence of Aerobic Exercise Training on Adipokine Concentrations in Obese Men

*Review*

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## Abstract

The rise of obesity and physical inactivity has elicited changes in body composition that negatively influence adipokine concentrations. While literature is limited to the vast majority of adipokines, several have been shown to play key roles in metabolism and inflammatory pathways. As a result, various comorbidities (e.g., hypertension, hypercholesterolemia, osteoporosis, diabetes mellitus, sarcopenia) have become more prevalent in individuals who possess an overabundance of adipose tissue. This may be attributed to the increase of pro-inflammatory adipokines as adipose tissue increases. However, some recent interventions have gained popularity [e.g., resistance training, aerobic exercise training (AET), nutrition] and have been shown to be effective in improving body composition in obese populations. More recently, studies have shown resistance training could be effective at improving pro- and anti-inflammatory adipokine concentrations and regulation (e.g., leptin, adiponectin, omentin-1, visfatin) by altering lean-muscle mass/fat-mass ratio. However, there have only been a few studies assessing how AET could also be effective in managing adipokine concentrations. Therefore, the focus of this review is to build a comprehensive understanding of the benefits of AET's influence on pro- and anti-inflammatory adipokine concentrations in obese males.

***Key Words***: Adipokines, Aerobic Exercise, Obese Males, Omentin-1, Adiponectin, Leptin.

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## Introduction

According to the Centers for Disease Control and Prevention (CDC), approximately 43% of adult males in the United States (US) are considered obese[1]. Over the last two decades obesity has increased 12% (e.g., 30.5% to 42.4%)[1]. Increased obesity rates have been correlated with increased overall costs in the US healthcare system[2]. The US spends nearly 173 billion dollars annually towards obesity and obesity-related diseases [e.g., Type 2 diabetes mellitus (T2DM), cardiovascular disease (CVD), hypertension, chronic kidney disease (CKD)][2,3]. Although various factors are associated with increases in obesity (e.g., stress, sleep, environment), physical inactivity and poor dietary habits are the most common factors[3]. Obesity is an issue due to the increased accumulation of excess adipose tissue leading to a production of both positive and negative physiologically active substances, known as adipokines[4].

Adipose tissue acts as an endocrine organ by secreting adipokines from adipocytes that are involved in several physiological processes (e.g., metabolic homeostasis, inflammation)[5,6]. Adipokines are produced from normal, dysregulated, premature and mature adipocytes within adipose tissue[6]. They are categorized into two subgroups (i.e., pro- or anti-inflammatory), each with its own physiological role[6]. Pro-inflammatory adipokines [e.g., leptin, resistin, interlukin-6 (IL-6), and tumour necrosis factor alpha (TNF-α)] have been extensively studied in published literature and potentially serve as mediators for various chronic diseases (e.g., T2DM, CVD, metabolic syndrome)[5,7]. Conversely, literature has noted that anti-inflammatory adipokines (e.g., adiponectin, omentin) play positive roles in reducing oxidative stress and systemic inflammation[7]. It is postulated that a balancing cascade may exist where anti-inflammatory adipokines have prospective roles in the reduction of pro-inflammatory adipokines[7]. Nevertheless, the dysregulation of adipokine secretion from adipose tissue accumulation causes low-grade systemic inflammation leading to the development and maturity of obesity-related diseases[6].

Numerous aids and interventions [e.g., physical activity, nutrition, resistance training (RT), aerobic exercise training (AET)], have been investigated to identify a meaningful way to moderate the detrimental impacts of an overproduction of pro- and anti-inflammatory adipokine concentrations on metabolic disorders[4,8]. AET, or endurance training, is a primary mechanism used by individuals to lose weight. With this loss of weight, there is an anticipated expectation that fat mass will be lost. Without adequate nutritional and protein intake, as well as RT, weight reduction can lead to reduced. muscle mass. Previous studies have examined the potential benefits of exercise as a mediator of adipokines, specifically assessing the effects of RT and combined RT/AET on adipokines in various populations (e.g., peri- and post-menopausal women, metabolic syndrome)[4,9]. However, there is a paucity of published literature assessing AET's relationship with body composition among adult males. Several studies acknowledge that both RT and AET could potentially influence adipokine mediators in overweight and obese individuals with excess adipose tissue[4,10]. Previous research suggests that reductions in adipose tissue and overall weight in overweight men can be attributed to AET [11,12]. However, it is unknown if there are any advantageous effects from performing higher intensity AET, in comparison to lower intensity AET.

Thus, the primary purpose of this review is to provide a better understanding of how AET influences body composition and adipokine concentrations. This review examines all studies using AET to improve body composition in obese men. Additionally, all adipokines measured in the included studies will be assessed to see if there were positive or negative changes in adipokine concentrations pre- and post-AET.

*Anti-inflammatory Adipokines to Consider in AET and Body Composition*

Adiponectin is an anti-inflammatory adipokine responsible for regulating numerous endocrine functions (e.g., glucose levels, insulin sensitivity)[13]. Adiponectin is inversely related to obesity. Thus, weight loss could stimulate an increase in adiponectin concentration[13]. Increased adiponectin concentration may help mitigate physiological disruptions (e.g., insulin resistance) common in obese individuals[13]. Because obese individuals have lower circulating levels of adiponectin than average weight individuals, increased obesity-induced inflammation could lead to the progression of further obesity-related diseases (e.g., T2DM) and an upregulated concentration of pro- inflammatory adipokines (e.g., Leptin, TNF-α)[13]. Omentin-1 (i.e., intelectin-1) resides in visceral adipose tissue, and also has anti-inflammatory effects [14]. Omentin-1 has been found to elicit anti-inflammatory and anti-oxidative roles that aid in the prevention of metabolic diseases [e.g., type 2 diabetes mellitus (T2DM), coronary artery disease (CAD)] [28]. High intensity (e.g., ≥ 60% heart rate max, ≥ 60% $VO_{2max}$) AET has been shown to have favourable relationships with adipokines such as omentin-1 [18,29]. Additionally, Omentin-1 promotes improvements in endothelial function by combating inflammatory actions mitigating the development of atherosclerosis[14]. It is also influential in lipolysis and lipid storage in adipose tissue. Further, its role in lipid metabolism can diminish the progression of insulin resistance [14]. Omentin-1 shares similar properties as adiponectin leading to speculation that it acts as a positive adipokine. However, individuals with chronic diseases associated with increased levels of adipose tissue (e.g., CVD, T2DM) elicit lower concentrations of this adipokine [14]. Omentin-1 provides potential benefits in combating chronic diseases but lacks knowledge in clinical data. Therefore, research assessing adipokine concentrations in clinical populations needs to be expanded.

*Pro-inflammatory Adipokines to Consider in AET and Body Composition*

Leptin is stored and secreted in adipose tissue, and an overabundance of leptin can lead to leptin resistance [15]. Ultimately leptin resistance can result in a proinflammatory cascade that causes increases in body mass and obesity-related diseases [15]. Leptin augments the production of pro-inflammatory adipokines (e.g., TNF-α, IL-1). Decreases in adiposity reduces leptin concentrations resulting in decreased inflammation[16]. Leptin can also stimulate increases of other pro-inflammatory cytokines (e.g., IL-6, TNF-α)[15]. When pro-inflammatory cytokines such as TNF-a are

stimulated by leptin, they in turn further stimulate the increases in leptin causing a circulating cascade of inflammation[15]. Furthermore, TNF- α is a proinflammatory adipokine secreted by adipose tissue responsible for negatively regulating aspects of glucose and lipid metabolism [15]. It is commonly found in obese individuals and plays a role in the downregulation of anti-inflammatory adipokines, leading to insulin resistance. Obradovic et al.[15] stated that hyperinsulinemia can result in increased leptin concentrations that modulate the expression of TNF- α, leading to an inflammatory cascade. Because TNF- α dysregulates the expression of adiponectin leading to insulin resistance contributing to the circulating cascade of inflammation which may lead to prominent obesity-related diseases (e.g., T2DM, metabolic syndrome)[15].

**Scientific Methods**

This narrative review examined all existing literature through several databases (i.e., PubMed, EMBASE, and EBSCO). All databases were searched from inception through May 30th, 2024. Keywords and phrases included combinations of: "Aerobic Training," "Body Composition," "Obesity," and "Adipokines." A detailed search criterion with keywords input and the total number of articles found from each source can be found in Table S1. After removing all duplicated articles found through these databases, articles were screened against the eligibility criteria (Table 1). References from the selected articles were also screened for relevant studies fitting the eligibility criteria (e.g., males, BMI ≥ 30kg/m$^2$). A detailed schematic of all four articles including all relevant information (e.g., participant demographics, training status, aerobic exercise protocols, adipokine outcomes), can be found in the Table 2 and Table 3.

**Table 1.** Inclusion and exclusion criteria for study eligibility.

**Research Question:** How does endurance training influence adipokines through changes to body composition in obese populations?

| Parameter | Inclusion | Exclusion |
|---|---|---|
| Population | ≥ 18 years of age<br>Assessment of obese male populations<br>BMI ≥ 30 kg/m$^2$ | Physical disabilities<br>Patients with various chronic diseases (e.g., CVD, CAD cancer, arthritis, diabetes, nonalcoholic fatty liver disease, metabolic syndrome, dyslipidemia)<br>Lacks distinction between recruited overweight and obese males<br>BMI was not used as a classification of Obesity |
| Intervention | Must specific participation in an aerobic exercise training program (≥ 4 weeks) | Effect cannot be determined between resistance training and aerobic training<br>Utilization or comparison with a supplement<br>Lacks adequate description of training protocol |
| Outcome | Biochemical measurements of any currently classified adipokines<br>(e.g., adiponectin, resistin, omentin, visfatin, leptin, etc.)<br><br>Physiological parameters<br>(e.g., RER, $VO_{2max}$, body composition) | No biochemical measurements sampled<br>Lack of pre- and post-assessment measures (e.g., body composition measures, $VO_{2max}$) |
| Study Design | Any peer-reviewed trials (e.g., RCT's, Crossover, etc.) performed on humans and published in English<br>Must assess pre- and post-aerobic exercise training | Abstracts<br>Conference proceedings<br>Unpublished Thesis<br>Unpublished Dissertation<br>Systematic Reviews<br>Meta analysis |

BMI = body mass index; RCT = randomized controlled trial; $VO_{2max}$ = maximal oxygen consumption; RER = respiratory exchange ratio; CVD = cardiovascular disease; coronary artery disease; kg/m$^2$ = kilogram per meters squared

**Table 2.** Aerobic exercise protocols and intensities.

| References | Participants | Training Status | Exercise Protocol | Exercise Intensity | Intervention Compliance |
|---|---|---|---|---|---|
| Besse-Patin et al. [17] | Obese men (n=11) | Sedentary | 12-week HIIT exercise on cycle ergometer and running<br><br>Intensity increased 5% every 4wk interval<br><br>1-hour long sessions | 35%-85% $VO_{2max}$ | 8-wk, ET program (Cycling and Running); > 85% |
| Atashak et al. [18] | Healthy obese men (n=30) | Sedentary | 3 nonconsecutive days/week<br>Interval cycling at 85%-95% HRamx | 85%-95% HRmax | 12-wk, supervised HIIT exercise program; 100% |
| Parastesh et al. [16] | Obsese men (n=23) | Sedentary | 12-wk cross training protocol 3d/week<br><br>60%-65% first 5 wks for 25-30min<br><br>65%-70% wk 6<br>35-40 min<br><br>70%-75% wk 7<br>50-55 min<br><br>75%-80% by wk 12<br>50-55 min | 60%-65% HRmax first 5 weeks<br>Increased by 5%-10% each week for weeks 6-12<br>HRmax was 80% by week 12 | 12-wk, supervised AET program; (not stated) |
| Krause et al. [19] | Obese men (n=24) | Sedentary | 1st 8wks: 3 days/wk outdoor walking for 30min according to corresponding HR zone,<br><br>2nd 8wks: 3 days/wk outdoor walking for 30min at adjusted HR zone | 25, 35, 45, 60% $VO_{2max}$ | 16-wk, unsupervised AET; 92% (44 ± 7 of 48 total walking sessions) |

Abbreviations: ET = endurance training; $VO_{2max}$ = maximal oxygen consumption, HIIT = high-intensity interval training; Hrmax = heart rate max; wk = week; h = hour; min = minute; HR = heart rate

**Table 3.** Pre- and post-training body composition measurements of obese training groups.

| | **Pre-training** | | | | | |
|---|---|---|---|---|---|---|
| **References** | **BMI** | **Wt (kg)** | **FM (kg)** | **BF (%)** | **FFM (kg))** | **$VO_{2max}$ (ml/kg/min)** |
| Besse-Patin et al. [17] | 32.6 ± 2.3 | 102.3 ± 6.5 | 36.3 ± 6.3 | - | 62.1 ± 6.3 | 44.8 ± 3.8 ┼ |
| Atashak et al. [18] | 31.1 ± 1.2 | 93.2 ± 5.1 | - | 29.7 ± 5.4 | - | - |
| Paratesh et al. [16] | 31.2 ± 0.1 | 110.3 ± 7.6 | - | - | - | - |
| Krause et al. [19] | - | - | - | 31.4 ± 3.7 | | 39.3 ± 5.8 |
| | **Post-training** | | | | | |
| **References** | **BMI** | **Wt (kg)** | **FM (kg)** | **BF (%)** | **FFM (kg))** | **$VO_{2max}$ (ml/kg/min)** |
| Besse-Patin et al. [17] | 32.7 ± 2.5 | 102.6 ± 7.4 | 35.5 ± 6.8 | - | 63.3 ± 7.4 | 47.9 ± 6.0 ┼ |
| Atashak et al. [18] | 30.1 ± 1.5* | 89.9 ± 3.8* | - | 27.1 ± 5.1* | - | - |
| Paratesh et al. [16] | 28.7 ± 0.8* | 100.8 ± 7.0* | - | - | - | - |
| Krause et al. [19] | - | - | - | 30.5 ± 3.0 | - | 40.8 ± 4.7 |

All data are expressed as Mean ± SD; significant difference between pre- and post-training program ($p < .05$) = *, ($p < .01$) = **. $VO_{2max}$ assessed as $VO_{2max}$/fat free mass = ┼. Abbreviations: BMI = body mass index; FM = fat mass; BF = body fat; FFM = fat free mass; $VO_{2max}$ = maximal oxygen consumption; kg = kilograms; y = years; kg/m2 = kilograms per meters squared, mL/kg/min = milliliters per kilogram per minute; Wt = weight.

**Table 4.** Aerobic exercise and adipokines assessed.

| References | Dietary Protocol | Sampling Method/Biomarker | Adipoki-nes Analyzed | Outcome |
|---|---|---|---|---|
| Besse-Patin et al. [17] | Maintain usual dietary habits | OVNF (10-12hrs); Plasma and serum samples | Adiponectin, Leptin, RBP4, IL-6, Apelin, FGF21 | No significant changes were seen in any adipokines from pre- to post-training. |
| Atashak et al. [18] | Maintain usual dietary habits | OVNF (10hrs); Serum samples were taken from the ACV between 700 and 900hrs. Samples were allowed to clot at room temperature for 10min and then centrifuged at 3000rpm for 15min | Omentin-1, LCN-2 | HIIT significantly increased levels of LCN-2 (p = .002) while decreasing omentin-1 (p = .001) |
| Parastesh et al. [16] | Maintain usual dietary habits | OVNF (10-12hrs); 1st sample taken prior to 1st exercise session and 2nd sample taken 48hrs after the last exercise session. Serum samples were taken between 600 and 700hrs. Samples were centrifuged at 3000rpm for 20min | Leptin | Proceeding 12-weeks of training, AET had significantly lower leptin concentrations (p = .001) in the obese exercise group than in the obese control. |
| Krause et al. [19] | No dietary control | OVNF; Plasma and serum blood samples were taken from the ACV. Samples were centrifuged at 1000g for 15min | Adiponectin, Leptin, TNF-α, IL-6 | There were no significant differences seen in any adipokine for either obese MIT training or obese LIT training. No differences in body composition were seen in any groups. |

Abbreviations: ET = endurance training; $VO_{2max}$ = maximal oxygen consumption; OVNF = overnight fast; HIIT = high-intensity interval training; HRmax = heart rate max; ACV = Antecubital vein; AET = Aerobic exercise training; MIT = moderate intensity training; LIT = lower intensity training; T2DM = type 2 diabetes mellitus; wk = week; h = hour; min = minute.

## Discussion

*Adipokine Changes in Relation to Changes in Body Composition*

From the four articles reviewed, two reported significant decreases in weight including decreases in body fat percentage [16,18]. Following a 12-week protocol Atashak et al.[18] noted BMI, weight, and body fat percentage decreased in the healthy obese group performing high-intensity interval training (HIIT) ($p$=.001, *ES*:.458). Effect size (ES) was calculated for each variable with the equation: ES = (mean post – Mean Pre) / SD. Individuals performing HIIT also had significant increases in concentrations of anti-inflammatory adipokine LCN-2 ($p$ = .002, *ES*:.382), and decreased of omentin-1 ($p$ = .001, *ES*: .558)[18]. Despite the claim that HIIT training caused changes in these adipokine concentrations, however it is possible that the changes were related to the decline in fat mass resulting from the completion of the HIIT program. This finding would support the notion that circulating adipokines are likely related to changes in body composition, and not exercise [20,21]. Parastesh et al. [16] assessed healthy and obese men (e.g., sedentary compared to AET) performing supervised AET for 12 weeks [16].ES in this study was set to 1.2 with an error type I (α = 0.05) and an error type II (Power = 0.85). Participants performed AET 3 days per week on fixed days (e.g., Monday, Wednesday, Saturday) lasting roughly 1.5-hours. Training regimens were progressed in difficulty relative to their maximum heart rate (MHR)[16]. MHR was increased weekly by 2-5% on a weekly basis – starting at 60-65% MHR and ending on the 12th week at 75-85% MHR. Although fat mass relative to weight was not reported, the exercise group decreased total abdominal fat ($p$ = .005, *ES*:1.2) more than the sedentary group [16]. Additionally, leptin concentrations were significantly lower in the exercise group ($p$ = .001, *ES*: 1.2) compared to the control [16]. These findings support a relationship between leptin concentrations and the amount of fat mass an individual possesses [22,23]. More research is needed to

assess leptin concentrations and other pro-inflammatory adipokines (e.g., TNF-α) that are modulated during an acute training protocol among obese individuals[16].

Besse-Patin et al.[17] was the only study that included fat free mass and fat mass along with BMI measurements. The study failed to identify significant declines between pre- and post-AET without controlling for the participants' diet. Furthermore, researchers saw no changes in adiponectin, leptin, RBP4, IL-6, Apelin, or FGF21. This result suggests changes in adipokines are mainly attributed to changes in body composition regardless of AET [24]. Likewise, Krause et al. [19] showed adipokines (e.g., adiponectin, leptin, TNF-α, IL-6) in their obese control groups had no significant changes, along with no changes in body composition following their intervention. This indicates that positive changes in pro- and anti-inflammatory adipokine concentrations likely require a decline in fat mass. Other published studies suggest that obese individuals lack adipokine responsiveness to exercise compared to healthy populations[4,25].

Published literature assessing adipokine concentrations in obese males is limited. Examination of other samples has shown there is an association between adipokine and fat concentration[9,22]. Additionally, current literature on adipokine concentrations in obese males lacks specific criteria to determine how "obese" was operationally defined. This has led to the inclusion of overweight individuals, and those not meeting CDC obesity guidelines[26]. Further, even the most well-known adipokines (e.g., adiponectin, leptin) have not been consistently assessed by the articles examined in this review [27]. Studies should more consistently examine specific adipokines, body composition, and exercise modalities employed. Understanding the specific adipokines that are controlled through various forms of exercise (e.g., by means of altering body composition), would enhance knowledge related to the low-grade systemic inflammation that obesity causes.

*Adipokines Response to Intensity of Aerobic Exercise Training*

While it is generally hypothesized adipokines are correlated to the amount of an individual's adipose tissue, future studies should explore the acute changes occurring from AET[9,27]. Krause et al.[19] compared two groups of individuals performing AET (Obese v. T2DM). Groups were split into two different intensities of AET (60% of estimated $VO_{2max}$ [n = 6] and 35% of estimated $VO_{2max}$ [n = 6])[19]. The present review only compared exercise intensities among obese male groups. Krause[19] did not identify significant changes in body composition or adipokines based on AET intensities of obese men in the sample. However, results of the study should be interpreted with caution due to the various limitations identified (e.g., absence of diet control, small sample size). This study did not control or monitor participants' diets during the 16-week AET program, posing a significant concern for the reported results[19]. Due to the lack of a caloric deficit, it is unknown if participants in this study would not likely experience changes in body composition or adipokine concentrations[19]. This ultimately created apprehension towards the interpretation or usage of their results for this review.

Ouerghi et al.[29] reported significant increases in concentrations of omentin-1 ($p < 0.01$) after 8-weeks of high intensity AET (e.g., 100-110% maximal aerobic velocity) in both healthy and obese groups. It was also noted that omentin-1 failed to demonstrate changes after moderate intensity exercise in obese males. This result suggests that exercise intensity plays an important role in the secretion of omentin-1[29]. Atashak et al.[18] reported similar findings to following a 12-week high intensity interval training (HIIT). Specifically, participants performed HIIT on a Monark Ergometer[18] at starting at 85% of their MHR, and ending at week 12 performing at 95% of their MHR[18]. After completing the protocol increased concentrations of omentin-1 were noted in obese males[18]. Furthermore, it is evident that there may be a positive relationship between performing high intensity AET and the release of specific adipokines this obese male population.

Currently, studies identifying changes in adipokines from varying intensities of AET in Class I-III obese males are scarce. Future studies should aim to assess the acute changes in adipokines from different intensities of AET. Additional needs for future acute and chronic study designs are presented on Table 4. When assessing acute changes, it is recommended researchers utilize $VO_{2max}$ as the standard to quantify the varying intensities of AET. Likewise, the type of modality assessed should be carefully considered for this population. Depending on the nature of participants' obesity, stationary cycling may appear to be a safer and more consistent means of assessing adipokine concentrations. In accordance with current literature on acute assessments, it is also recommended that venous blood samples be taken at baseline, 1-hour, 2-hours, 3-hours, and 24-hours post-exercise to assess changes in adipokines across the time points.

**Table 5.** Future study design recommendations.

| Study Design | Acute | Chronic |
|---|---|---|
| **Study Duration** | Monitored metabolic study day (3-4Hrs); Acute bout assessments (1-2Hrs) | 6Wks – 16Wks of monitored AET |
| **Venous serum or plasma blood samples** | Baseline, 1Hr, 2Hr, 3Hr, and 24Hr | Baseline, 6Wk, and 12Wk |
| **AET modality** | Cycling (stationary or outdoor), Running, Jogging, Rowing | Cycling (stationary or outdoor), Running, Jogging, Rowing |
| **Aerobic exercise measurements** | $VO_{2max}$ or sub-max $O_2$ consumption | $VO_{2max}$ or sub-max $O_2$ consumption |
| **Dietary recommendations** | Diet should be controlled several days prior to assessment (utilization of dietary records) | Diet should be controlled throughout duration of the AET study protocol (utilization of dietary records) |
| **Body Composition Measurements** | Body composition should be assessed using a DEXA (to account for visceral adipose tissue) | Body composition should be assessed using a DEXA (to account for visceral adipose tissue) |

*Abbreviations: Hr = hour; Wk = week; AET = aerobic exercise training; $VO_{2max}$ = maximal oxygen consumption; sub-max $O_2$ = submaximal oxygen consumption; DEXA = dual-energy x-ray absorptiometry

*Strengths & Limitations*

There were several limitations of the review that should be addressed. The primary limitation is the lack of current literature examining adipokine responses among obese males that meet the minimum Class I, CDC criteria for obesity (≥ 30 kg/m$^2$) [26]. Several studies adequately fit the model on face value. However, the inclusion of overweight males (25 – 29.9kg/m$^2$) in addition to obese males were cause for exclusion from this review. Moreover, differences between studies in training intensities employed could play a role in adipokine concentrations reported. Finally, differences in the nutrition of participants between studies may have influenced changes in body composition (e.g., calorie deficit). This may have affected adipokines differently in that some participants may have lost more fat mass than others due to being in a greater caloric deficit. Another limitation to the review was that studies included all contained either small sample sizes or didn't specify the number of participants in the study. It is imperative to ensure an adequate sample size was recruited in order to generalize the data to the population. Due to the lack of participants, it makes it difficult to generalize the findings in the studies. Furthermore, all studies failed to control for diet and had varying intensities, thus making it difficult for interpretation of whether diet or the chosen intensity was the cause in changes in adipokine concentrations.

While there were notable limitations in this review, several strengths of the study should also be considered. All males in studies examined had no other known contraindicating chronic diseases, other than being obese. All individuals in the samples had high levels of fat mass without having any obesity-related disease interventions Additionally, the AET programs from the six studies we examined were all completed with ≥ 8-weeks of AET. Therefore, adaptations in participants were from chronic AET and not acute AET. Therefore, this contributes to a further discussion and approach of how adipose tissue functions and releases adipokines when undergoing changes in body composition with AET.

*Future Directions*

Proper identification of which adipokines to assess is recommended to be taken into consideration. While the more commonly assessed adipokines are adiponectin, visfatin, resistin, and TNF-α, there are more novel anti- and pro-inflammatory adipokines that have recently been examined in terms of RT and AET exercises. More specifically, Omentin-1 has shown pro-inflammatory properties when performing RT in healthy individuals and AET in male smokers [18]. One interesting direction would be to assess how Omentin-1 acutely or chronically responds to an AET program, especially in obese males [18,30(p1)]. Nevertheless, researchers should carefully determine the appropriate adipokines to assess in the study designs. As previously stated in the limitations, sample size and diets should be controlled for when assessing the implications AET has on adipokine concentrations. Ensuring diet is controlled for will allow researchers to distinguish if the changes were due to the protocol, or dietary modifications. Employing dietary recalls, along with counselling participants on preferred food consumption throughout the duration of the study

are ways diet can be accounted to ensure diet was not a contributing factor to adipokine changes. Finally, ensuring an adequate sample size is chosen for the study is imperative to the reliability and validity of the study. Additionally, the utilization of a larger sample size would enable researchers to make a more convincing comparison and generalizing the findings to the population.

**Conclusions**

Based on the examination of the papers assessed in this literature review, we can ascertain that inclusion of an AET program will likely improve body composition in obese males. Due to the various protocols and intensities utilized in the studies, it is difficult to conclude a concreate AET intensity that will stimulate an improvement in adipokines. According to the studies examined in this review, there were very little changes seen in body fat mass. Therefore, it is possible that without those body composition changes, no changes would be seen in pro- and anti-inflammatory adipokine concentrations. It is difficult to conclude what intensity of AET would stimulate the reduction in pro-inflammatory adipokines due to the mixed findings between the studies and the various protocols they utilized. However, we can postulate that the utilization of AET and dietary modifications to reduce bodyfat mass can result in reductions in pro-inflammatory adipokines, and an increase in anti-inflammatory adipokines as seen in the study conducted by Atashak et al. Though more research is needed to confirm this hypothesis. Future research is needed to identify if the lack of adipokine changes seen were from participants' diet, the intensity of their aerobic training, lack of body composition changes, or a combination of the three.

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**Table S1.** Database results.

| *Database* | *Keywords* | *Number of Articles* |
|---|---|---|
| **PubMed** | (Aerobic Training) AND (Body Composition) AND (Obesity) AND (Adipokines) | 195 |
| | AND ("Humans") | 167 |
| | AND ("English") | 165 |
| **EMBASE** | ('aerobic training' OR (aerobic AND ('training'/exp OR training))) AND ('body composition'/exp OR 'body composition' OR (body AND composition)) AND ('obesity'/exp OR obesity) AND ('adipokines'/exp OR adipokines) | 59 |
| | AND [humans]/lim | 57 |
| | AND [english]/lim | 54 |
| **EBSCO** | (Aerobic Training AND Adipokines AND Obesity AND Body Composition) | 562 |
| | Peer-Reviewed | 543 |
| | English | 501 |
| **TOTAL** | **(PubMed + EMBASE + EBSCO)** | 720 |
| | **After removing duplicates using Zotero software** | 674 |