

## Muscular strength, aerobic capacity, and adipocytokines in obese youth after resistance training: a pilot study

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### RESEARCH

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### ABSTRACT

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#### Background

Exercise has shown positive training effects on obesity-related inflammation, however, resistance training has shown mixed results concerning adipocytokine levels.

#### Aims

The purpose of this pilot study was to explore the effects of resistance training on blood adipocytokine concentrations in obese youth, with specific examination of the relationship between these biomarkers and improved fitness (i.e., aerobic capacity, muscular strength).

#### Methods

Fourteen obese adolescents ( $16.1 \pm 1.6$  y; BMI:  $32.3 \pm 3.9$  kg/m<sup>2</sup>) participated in a 16-week resistance training intervention. Body composition, fasting blood

concentrations of interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF- $\alpha$ ), adiponectin, and leptin were measured pre- and post-training. Aerobic capacity was assessed via a maximal discontinuous exercise test. The rate of gain in muscular strength was calculated as the slope of progression in 1-repetition maximum throughout the intervention.

#### Results

Resistance training increased lean mass (total, trunk) and decreased per cent body fat (total, trunk). The training also caused moderate clear decreases in IL-6 and TNF- $\alpha$  concentrations. A small increase in adiponectin was also observed before and after intervention. When the group was stratified by changes in aerobic capacity, there were substantially larger decreases in leptin levels for those with improved capacity. Correlation analyses also revealed a negative relationship between log-transformed leptin and aerobic capacity at rest. Improvement in quadriceps strength was positively correlated with IL-6 and TNF- $\alpha$ , while improvement in shoulder adductor strength was positively correlated with IL-6 only.

#### Conclusion

Resistance training improved adipocytokine markers, which were partially associated with improved physical fitness. Specifically, the relationship between strength improvements and IL-6 and TNF- $\alpha$  suggests an exercise-induced signalling pathway that results in overall adaptive decreases in systemic inflammation in obese youth.

#### Key Words

Exercise, paediatric obesity, inflammation, fitness

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## What this study adds:

### 1. What is known about this subject?

Obesity and physical activity have both been associated with adipocytokine levels, though often with contradictory effects. Specific improvements are often seen with aerobic activity; however, obese individuals can be disadvantaged by physical constraints. Resistance training is well tolerated by the obese population, but has shown mixed results for improving adipocytokine levels in this group.

### 2. What new information is offered in this study?

Resistance training improved pro-inflammatory adipocytokine levels in obese youth; these changes could result from the adaptive mechanism of IL-6 to muscular strength and fitness gains. The overall decrease in pro-inflammatory markers suggests that resistance training could have immune-modulating effects, which may impact insulin sensitivity.

### 3. What are the implications for research, policy, or practice?

Resistance training could be a good starting point for introducing obese youth to exercise, especially those who are limited by physical and cardiovascular capacity. The relationship between improved adipocytokine concentrations and markers of physical fitness emphasise the importance of including both improved aerobic capacity and muscular strength as targeted goals when prescribing exercise to obese youth.

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## Background

Obesity is characterised by excess total body fat, which influences metabolic processes and predisposes an individual to chronic non-communicable disease. Paediatric obesity has been associated with elevated levels of inflammatory adipocytokines, such as interleukin 6 (IL-6) and tumour necrosis factor alpha (TNF- $\alpha$ ).<sup>1</sup> IL-6 has been linked to cardiovascular disease via provasculopathic activities and mediates insulin resistance through an increase in TNF- $\alpha$  receptors.<sup>2</sup> Previous research suggests that decreased adiponectin levels seen in obesity may contribute to impaired cardiovascular function,<sup>3</sup> as well as development of insulin resistance.<sup>4</sup> Although leptin resistance has been associated with diet-induced obesity, elevated levels of leptin in an obese state have also been considered a link between obesity, insulin resistance, and atherosclerosis.<sup>5</sup> In general, an increase in systemic inflammation has been linked to increased risk of

cardiovascular disease and type 2 diabetes in obese individuals.<sup>2</sup>

A low level of cardiorespiratory fitness is predictive of type 2 diabetes and cardiovascular disease in obese young adults.<sup>6</sup> Conversely, there have been significant relationships reported between physical activity levels and adipocytokines.<sup>7</sup> Previous research has also found that participants with obesity-related inflammation show a positive training effect on inflammatory biomarkers after an exercise intervention.<sup>7</sup> Although most improvements have been seen with aerobic activity,<sup>7</sup> obese individuals can be disadvantaged by physical (i.e., body weight) and cardiovascular constraints (i.e., aerobic capacity). Resistance training has been shown to be well tolerated by this population,<sup>8</sup> primarily as a result of the greater absolute strength shown in obese youth.<sup>9</sup> Resistance training has improved markers of metabolic syndrome, but these interventions have provided mixed results for adipocytokine levels.<sup>10-12</sup> Previous research has primarily focused on older populations; therefore, the purpose of this pilot study is to examine the implications of resistance training interventions on adipocytokine levels in obese youth, with a specific focus on the relationship to improved aerobic capacity and muscular strength.

## Method

Fourteen obese adolescents (n=8 females; n=12 Caucasian, one Polynesian, one Asian; 16.1 $\pm$ 1.6 y; BMI: 32.3 $\pm$ 3.9 kg/m<sup>2</sup>) participated in a four-month resistance training exercise intervention. A health screening was completed; participants were excluded if there was a recent history of cardiovascular conditions, neuromusculoskeletal disease, or musculoskeletal injury or surgery within the past six months, which would have affected their ability to safely complete the exercise sessions. Prior to study commencement, participants and their parents/guardians were made aware of any risks associated with the study and gave their written informed assent and consent, respectively. All protocols were approved by Queensland University of Technology Human Research Ethics Committee.

## Intervention

The 16-week resistance training intervention was completed on three non-consecutive days per week. The 60-minute training session consisted of 1-2 sets of 15 exercises performed on pneumatically controlled progressive resistance training equipment (Ab Hur Oy, Kokkola, Finland). Exercises primarily targeted the upper extremity (chest press, chest curl, biceps curl, triceps curl, push-up, curl down, deltoid press, pull-down, latissimus

dorsi curl) and lower extremity (leg press, quadriceps extension, hamstring curl, hip abduction, hip adduction), with less focus on the abdomen (abdominal curl). Intensity was based on a predicted one-repetition maximum (1-RM), as outlined in the American College of Sports Medicine guidelines.<sup>13</sup> Assessment of 1-RM was completed at baseline and then re-assessed every four weeks, to account for any strength gains. Resistive load increased fortnightly from 60–85 per cent 1-RM over the initial 12 weeks, then stabilised at 85 per cent 1-RM for the last four weeks. The number of sets alternated weekly, so that resistive load was increased when only one set was to be completed during the training session. Participants were to be removed from the study if they missed three consecutive training sessions, or 10 per cent of the training sessions over the 16-week period; however, all participants completed the intervention (100 per cent retention rate).

### Assessments

Measures of adipocytokines and aerobic capacity were conducted pre- and post-intervention. Height was measured to the nearest 0.1cm with a Harpenden stadiometer (Holtain Ltd, Crymych, Wales). Body mass was measured to the nearest 0.1kg using an electronic scale. Body mass index (BMI=kg/m<sup>2</sup>) was calculated from height and weight measurements. Assessment of regional body composition was completed using dual energy x-ray absorptiometry (DXA; Lunar Prodigy Advance, GE Healthcare, Madison, WI).

Twelve-hour fasting blood samples were analysed with specific focus on biomarkers that related to cardiometabolic health, including interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF- $\alpha$ ), high sensitivity C-reactive protein (hsCRP), adiponectin, and leptin. IL-6 and TNF- $\alpha$  were measured using a high sensitivity cytokine Milliplex kit (Millipore, Billerica, MA). A Siemens BNII instrument and corresponding Siemens Cardiophase hs-CRP reagent were used to analyse hs-CRP. Serum adiponectin was measured using a Procarta kit (Panomics Fremont, CA) and serum leptin was measured using an in-house multiplex assay and measured on the Luminex 100 platform (Luminex, Austin, TX).

The effect of training on maximal aerobic capacity (VO<sub>2</sub>max) was determined via a maximal discontinuous graded exercise protocol on an electronically braked cycle ergometer (Lode Excalibur Sport; Gronigen, The Netherlands).<sup>14</sup> The initial workload for all participants was 50W and each subsequent stage increased in workload by 25W. Each stage was five minutes in length, consisting of 3.5 minutes of cycling followed by a 1.5 minute rest. Participants were required to maintain a cycling cadence of 60–70rpm. When a cadence of 60rpm could not be maintained, the stage was considered incomplete. The

participant was then given several minutes rest before a second attempt was made at the previous workload. If the participant completed the stage, then the workload increased by 25W and the test continued as described above. When the participant was unable to complete a stage during the second attempt, the testing session was terminated. For participants who did not achieve the defined VO<sub>2</sub>max as previously published,<sup>15</sup> the highest value was taken as peak VO<sub>2</sub> for all subsequent analyses.

For each participant, a linear relationship was established between workload (W) and VO<sub>2</sub> (ml/min).<sup>16</sup> The slope and y-intercept of this line was used to describe oxygen economy. A reduction in the slope (ml/min/W) after the intervention is evidence of an improved economy, and hence improved aerobic function. The y-intercept provides an estimate of the oxygen cost (and energy expenditure) of seated rest. A reduced intercept would demonstrate a lower oxygen cost (i.e., aerobic function) at seated rest.

Pre- and post-intervention maximal strength measures were not taken. To provide an alternative estimate of the overall effect of training on muscular strength, a predicted 1-RM was collected at zero, four, eight, and 12 weeks. A linear relationship was established between collection points and predicted 1-RM; the slope of this line described the overall position and rate of improvement in muscular strength. For each exercise, the slope of all participants was positive, indicating improvements in muscular strength.

### Statistical analysis

Paired sample t-tests were performed to examine differences in total and regional body composition (per cent body fat and lean mass for total body and trunk), adipocytokine concentrations, and aerobic capacity before and after the intervention. Adipocytokine concentrations were log transformed prior to analysis to manage non-uniformity of residual error that could be present as a result of small sample size and a large distribution of concentration values. These calculated values were used for the independent t-test and correlational analyses.<sup>17</sup>

Adipocytokines with log-transformed values greater than 1.0 indicated an increase in concentration levels post-intervention; conversely, log-transformed values less than 1.0 indicated decreased concentrations post-intervention. Pearson's correlation analysis was used to examine relationships between muscular strength, aerobic capacity, and adipocytokine concentrations. Independent t-tests were used to determine differences in inflammatory markers between those whose aerobic capacity improved (decreased slope; n=6) and those with reduced or unchanged aerobic capacity (increased slope; n=8). All

statistical analyses were performed using SPSS 20.0 (IBM SPSS Inc., Chicago, IL).

## Results

Sixteen weeks of resistance training altered adipocytokine concentrations and body composition in obese youth (Table 1). Specifically, IL-6 and TNF- $\alpha$  concentrations were moderately decreased post-resistance training, while a clear substantial increase in adiponectin concentration was observed at study completion. Total and truncal lean mass also increased while total and truncal per cent body fat decreased after the intervention, however, only the reduction in truncal per cent body fat was non-trivial (Table 1).

There was no overall clear difference in aerobic capacity before and after the resistance training intervention. On an individual level, almost half (43 per cent) of the participants improved their aerobic capacity after completing the intervention; further analyses revealed changes in adipocytokine concentrations when participants were stratified by those who did or did not improve aerobic capacity. Participants who showed improved aerobic capacity had greater improvements in leptin levels ( $p=0.02$ ). Specifically, the log-transformed leptin values were unchanged in participants whose aerobic capacity did not improve ( $0.94 \pm 0.14$ ), whereas a decrease ( $0.56 \pm 0.29$ ) in leptin occurred in those who had improved aerobic capacity. Correlational analyses revealed a negative relationship between log-transformed leptin values and a marker of aerobic capacity,  $y$ -intercept of the linear workload- $\text{VO}_2$  relationship (Table 2). No other clear relationships existed between aerobic capacity (via slope or  $y$ -intercept) and adipocytokine concentrations.

Additional relationships were found between the inflammatory adipocytokines and muscular strength (Table 2). Improvements in quadriceps strength were positively correlated to log-transformed values of IL-6 and TNF- $\alpha$ , while improvements in shoulder adductor strength were positively associated with IL-6 only.

## Discussion

The purpose of this pilot study was to understand the effects of 16 weeks of resistance training on adipocytokine levels in obese youth, specifically as these changes relate to improvements in aerobic capacity and muscular strength. While the overall changes to adipocytokine concentrations is positive, the lack of control group in this pilot study makes it difficult to confirm the full contribution of the intervention to these improvements. Nevertheless, the obese youth seem to have improved systemic inflammation, by decreasing both IL-6 and TNF- $\alpha$  levels after the

intervention. The overall decrease in pro-inflammatory markers suggests that resistance training could have immune-modulating effects that may, in turn, influence insulin sensitivity. Specifically, both IL-6 and TNF- $\alpha$  play specific roles in compromised insulin sensitivity. Furthermore, it is believed that IL-6 negatively affects the secretion of adiponectin;<sup>18</sup> thus, the decrease in circulating IL-6 may have accounted for the observed increase in adiponectin after the intervention.

Almost half of the participants improved their aerobic capacity; however, there was no substantial difference before and after the intervention for the group. Given the established relationship between aerobic fitness and cardiovascular disease,<sup>6</sup> it was important to investigate aerobic fitness as a potential mechanism for improving cardiovascular health. Indeed, those who improved aerobic capacity showed decreases in leptin concentration. Previous research has shown an inverse relationship between leptin and aerobic fitness,<sup>19–20</sup> which is often dependent on simultaneous changes in body composition.<sup>19</sup> Although it was not the focus of this investigation, relationships were seen between leptin and truncal fat and lean mass (unreported) that would suggest the relationship between aerobic capacity and leptin is not wholly independent of subsequent body composition changes. Leptin has a strong positive relationship with oxidative stress that may be associated with cardiometabolic disease.<sup>21</sup> Conversely, previous research has shown that improvements in aerobic fitness decreases oxidative stress in obese adults, irrespective of changes in body composition.<sup>22</sup> The exercise-induced benefits to the anti-oxidant defense system<sup>22</sup> could directly affect the actions of leptin on phagocytic activity.<sup>18</sup> The reduction of leptin in an obese population (where leptin resistance prohibits the natural role of leptin in fatty acid oxidation)<sup>23</sup> can reduce risk of cardiovascular disease, specifically atherosclerosis and obesity-associated hypertension.<sup>5</sup>

Muscular strength improvements were seen only in association with pro-inflammatory adipocytokines. The positive relationship between muscular strength improvements and IL-6 were particularly strong, indicating a subsequent promotion of IL-6 with improved quadriceps and shoulder adductor strength. Adipose tissue contributes only 30 per cent of circulating IL-6 (thus classifying it as an adipocytokine); skeletal muscle is considered the primary secretion organ for this cytokine (with classification as a myokine).<sup>24</sup> Given the possible mediatory role of IL-6 in muscle hypertrophy,<sup>25</sup> it was promising to find a relationship between strength gains and IL-6 reductions. While we have no time-series data, we propose that this

putative mechanistic relationship would more likely have been stronger in the early stages of the intervention: previously sedentary participants would have the strongest reaction to exercise and pro-inflammatory mediators. It is suggested that as the sedentary participant adapted to regular exercise, IL-6 levels would stabilise. The moderate overall reductions of IL-6 after intervention would suggest an improved inflammatory environment for the obese youth. IL-6 is strongly correlated to TNF- $\alpha$ .<sup>26</sup> The muscle damage induced by strength gains in combination with increased IL-6 levels may explain the additional positive relationship between quadriceps strength and TNF-alpha levels.

This pilot study is limited by its small sample size and lack of a control group. In addition, as these results are from a primarily Caucasian cohort, it could affect the application of findings to other ethnicities. The resistance training intervention was designed to meet the recommended amount of resistance exercise per week.<sup>27</sup> Given the international agreement that resistance training is beneficial to health and fitness in youth,<sup>28</sup> it was encouraging to report 100 per cent exercise retention rate. The perfect retention rate indicates that the biggest obstacle may be in motivating youth to begin exercise, rather than continue exercise once begun.<sup>29</sup> Given the exploratory nature of this pilot study, it is important to re-examine the relationship between resistance training and metabolic profile in a larger research study containing a control group and greater range of ethnicities.

### Conclusion

The resistance training intervention produced overall improvements to biomarkers associated with adiposity, specifically those with a pro-inflammatory role; however, the lack of a control group makes it difficult to attribute these benefits to the intervention alone. Nevertheless, the improvement to pro-inflammatory markers could be a result of the adaptive mechanism of IL-6 to improvements in strength gain and muscular fitness seen during resistance training, although future research would need to include time-series data. Additional improvements to aerobic capacity can impact levels of major adipocytokines in obese youth, specifically leptin. The results of this study suggest that resistance training could be a good starting point for introducing obese youth to exercise, especially those who are limited by physical and cardiovascular capacity. However, it is important to include both improved aerobic capacity and muscular strength as primary outcomes when prescribing exercise to obese youth.

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## PEER REVIEW

Not commissioned. Externally peer reviewed.

## CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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## ETHICS COMMITTEE APPROVAL

Queensland University of Technology Human Research Ethics Committee (HREC No: 0900001207).

**Table 1: Effect of a 16-week resistance training intervention on variables of aerobic capacity, body composition, and adipocytokines**

Variable	Pre-intervention <sup>1</sup>	Post-intervention <sup>1</sup>	Effect (95% CI) <i>p</i> value	Magnitude and direction of change <sup>2</sup>
Aerobic capacity (slope)	0.23 ± 0.05	0.24 ± 0.04	0.2 (-0.04, 0.02) 0.330	Trivial increase
Aerobic capacity (y-intercept)	1.11 ± 0.25	1.05 ± 0.21	0.24 (-0.05, 0.18) 0.260	Small decrease
Truncal lean mass (kg)	23.27 ± 5.07	23.80 ± 5.14	0.10 (-1.10, 36.37) 0.064	Trivial increase
Total lean mass (kg)	50.83 ± 10.90	52.53 ± 11.26	0.16 (-2.85, -0.56) 0.011	Trivial increase
Percentage Truncal Body Fat (%)	44.59 ± 5.82	43.23 ± 5.80	0.23 (0.09, 2.74) 0.038	Small decrease
Percentage Total Body Fat (%)	41.85 ± 6.52	40.69 ± 6.72	0.18 (0.33, 2.12) 0.006	Trivial decrease
IL-6 (pg/mL)	7.08 ± 5.09	1.89 ± 0.77	1.02 (1.54, 8.84) 0.011	Moderate decrease
TNF-α (pg/mL)	5.30 ± 3.04	3.47 ± 1.83	0.60 (-0.15, 3.81) 0.066	Moderate decrease
Adiponectin (µg/mL)	5.80 ± 4.02	7.33 ± 3.82	0.38 (-3.03, -0.03) 0.047	Small increase
Leptin (ng/mL)	30.42 ± 17.86	24.18 ± 13.82	0.35 (-1.55, 14.03) 0.103	Small decrease

<sup>1</sup>Data are means ± SD.

<sup>2</sup>The standardised difference is from the modified Cohen *d* effect size of Hopkins et al.,<sup>17</sup> where trivial, 0.0–0.2; small, 0.2–0.6; moderate, 0.6–1.2; large, 1.2–2.0; very large, 2.0–4.0; extremely large >4.0.

**Table 2: Results of correlational analysis between variables of muscular strength, aerobic capacity, and adipocytokine**

Adipocytokines	Slope	y-intercept	Biceps Brachii	Hamstrings	Pectoralis Major	Quadriceps	Deltoid	Triceps Brachii	Shoulder Adductors
Log-Transformed IL6	-0.268 <i>(0.454)</i>	0.430 <i>(0.215)</i>	0.332 <i>(0.349)</i>	0.233 <i>(0.518)</i>	0.211 <i>(0.559)</i>	0.701* <i>(0.035)</i>	0.160 <i>(0.660)</i>	-0.010 <i>(0.978)</i>	0.654* <i>(0.040)</i>
Log-Transformed TNF	-0.064 <i>(0.861)</i>	0.143 <i>(0.693)</i>	0.365 <i>(0.300)</i>	0.302 <i>(0.397)</i>	0.351 <i>(0.320)</i>	0.700* <i>(0.036)</i>	0.470 <i>(0.171)</i>	0.329 <i>(0.353)</i>	0.370 <i>(0.293)</i>
Log-Transformed Adiponectin	-0.075 <i>(0.836)</i>	0.021 <i>(0.954)</i>	-0.351 <i>(0.320)</i>	-0.453 <i>(0.189)</i>	-0.140 <i>(0.701)</i>	-0.506 <i>(0.165)</i>	-0.295 <i>(0.407)</i>	-0.237 <i>(0.509)</i>	-0.332 <i>(0.349)</i>
Log-transformed Leptin	-0.616 <i>(0.058)</i>	-0.637* <i>(0.048)</i>	0.154 <i>(0.672)</i>	-0.236 <i>(0.512)</i>	0.289 <i>(0.418)</i>	-0.238 <i>(0.537)</i>	0.115 <i>(0.751)</i>	0.105 <i>(0.773)</i>	-0.429 <i>(0.216)</i>

Note: \*P values are bracketed and italicized.