Effects of an Intensive Short-term Diet and Exercise Intervention: Comparison Between Normal Weight and Obese Children

Christian K. Roberts¹, Ali Izadpanah¹,², Siddhartha S. Angadi¹ and R. James Barnard²

¹Exercise and Metabolic Disease Laboratory, Translational Section, School of Nursing, University of California, Los Angeles, CA.
²Department of Integrative Biology and Physiology, University of California, Los Angeles, CA.

Running title: Lifestyle Change in Normal Weight and Obese Children

Communicating author:
Christian K. Roberts, Ph.D.
Exercise and Metabolic Disease Laboratory
Translational Sciences Section
UCLA School of Nursing
700 Tiverton Ave.
Los Angeles, CA 90095-6918
croberts@ucla.edu
Abstract

Lifestyle intervention programs currently emphasize weight loss secondary to obesity as the primary determinant of phenotypic changes. We examined whether the effects of a short-term lifestyle intervention program differs in normal weight vs. overweight/obese children. 19 overweight/obese (O, BMI=33.6±1.9 kg/m²) and 14 normal weight (N, BMI=19.9±1.5 kg/m²) children participated in a 2-week program consisting of an ad libitum high-fiber, low-fat diet and daily exercise (2-2.5 hr). Fasting serum samples were taken pre- and post-intervention for determination of lipids, glucose homeostasis, inflammatory cytokines, and adipokines. Only O lost weight (3.9%), but remained overweight/obese (32.3±1.9 kg/m²). Both groups exhibited significant intervention-induced decreases (p<0.05) in serum insulin (N: 52.5% vs. O: 28.1%, between-group p=0.38), homeostatic model assessment for insulin resistance (N: 53.1% vs. O: 28.4%, p=0.43), leptin (N: 69.3% vs. O: 44.1%, p=0.10), amylin (N: 28.7% vs. O: 26.1%, p=0.80), resistin (N: 40.0% vs. O: 35.1%, p=0.99), plasminogen activator-inhibitor-1 (N: 30.8% vs. O: 25.6%, p=0.59), interleukin (IL)-6 (N: 58.8% vs. O: 48.5%, p=0.78), IL-8 (N: 46.0% vs. O: 42.2%, p=0.49), and tumor necrosis factor-α (N: 45.8% vs. O: 40.8%, p=0.99). No associations between indices of weight change and phenotypic changes were noted. A short-term, intensive lifestyle modification program is effective in ameliorating metabolic risk factors in normal weight and overweight/obese children. These results suggest that obesity per se was not the primary driver of the phenotypes noted and dietary intake and physical inactivity induce the phenotypic abnormalities. These data may have implications for the weight-loss independent management of cardiometabolic risk in pediatric populations.

Keywords: Physical activity, nutrition, metabolic, cytokines

Introduction
Obesity is associated with a variety of chronic diseases, including, but not limited to coronary artery disease, hypertension, diabetes and certain forms of cancer. In the US, 34% of adolescents ages 12 to 19 years of age are classified as “overweight” or “obese” (BMI > 85th percentile) (13). Unhealthy lifestyle factors, such as physical inactivity/lack of exercise training, and high-refined-carbohydrate, high-fat diet consumption begin in childhood and contribute to both the development of obesity and other chronic diseases (17), and thus it is unclear whether obesity per se or the associated lifestyle factors are underlying causes of the cardiovascular and metabolic dysfunction and the related development of chronic disease.

The first line of defense against obesity-related disease has been weight loss, as the currently held view in lifestyle intervention programs emphasizes that weight loss secondary to obesity is the primary determinant of phenotypic changes (16, 23). However, it is unclear if obesity and the subsequent weight loss are the primary drivers of the phenotypic modifications. Along these lines, we previously demonstrated reversal of metabolic syndrome (4) and amelioration of a variety of atherosclerotic (18) and metabolic (10) risk factors, including markers of endothelial dysfunction, oxidative stress, inflammation and fatty acid species in overweight/obese youth with lifestyle modification. As the intervention was short-term, this allowed us to study lifestyle effects prior to reversal of obesity. The changes occurred despite small changes in weight, and correlation analysis indicated that changes in phenotypic markers were independent of weight loss. These findings suggested that lifestyle changes may be the underlying drivers of changes in metabolic and cardiovascular phenotypes. This led us to test the validity of overweight/obesity status as a primary cause of metabolic abnormalities by investigating the effects of an identical lifestyle intervention in normal weight compared with overweight/obese children.
Thus, the present study was designed to examine the efficacy of a short-term, daily exercise and plant-based *ad libitum* diet intervention program on serum endocrine and cytokine markers. We examined its effects on interleukin (IL)-8, IL-10, IL-1 receptor antagonist ((IL-1ra), IL-6, tumor necrosis factor-α (TNF-α), plasminogen activator-inhibitor (PAI)-1, resistin, amylin and leptin differs in normal weight versus obese children. We hypothesized that normal weight and obese children would respond similarly to the lifestyle intervention program irrespective of baseline obesity status, suggesting that dietary intake and lack of exercise/physical activity are the underlying causes of the phenotypic abnormalities noted.

**Methods**

**Subjects**

Normal weight and overweight/obese children (classified by the CDC sex-specific BMI-for-age percentiles) voluntarily participated in the Pritikin Longevity Center 2-week residential lifestyle modification program where a plant-based diet was provided *ad libitum* and daily exercise (2-2.5 hr) was performed. Pre- and post-intervention data were obtained from 19 overweight (O) children aged 8 to 17 (mean 13.1±0.5 yr, 9 male and 10 female, mean BMI: 33.6±1.9 kg/m², BMI percentile: 95.2±1.4%) and 14 normal weight (N) children aged 9 to 15 (mean 11.2±0.5 yr, 6 male and 8 female, 19.9±1.5 kg/m², 55.5±7.9%) who participated in the two week program. All subjects in the O group had a BMI >85th percentile and 13 of the 19 were >95th percentile (obese) according to CDC BMI-for-age percentile standards. All 14 subjects in the N group were considered to be at a healthy weight (>5th and <85th percentile), according to CDC guidelines. None of the subjects were using drugs or therapies for obesity, and none had prior histories of
disease or injury that would prevent daily exercise. Consent to participate in a research program was obtained from the parents; all agreed to provide data for the study, and the project was approved by the University of California, Los Angeles Institutional Review Board.

**Diet and Exercise Intervention**

Participants in the program received a complete physical examination and underwent a 14-day diet and exercise intervention as previously described (4, 10, 18). The plant-based, *ad libitum* diet contained 12-15% of calories from fat (polyunsaturated/saturated fatty acid ratio = 2.4:1), 15-20% of calories from protein, and 65-70% of calories from primarily unrefined-carbohydrate, high in dietary fiber (>40g per/day). Carbohydrates were primarily in the form of high-fiber whole grains (≥ 5 servings/day), vegetables (≥ 4 servings/day) and fruits (≥ 3 servings/day). Protein was primarily derived from plant sources, along with nonfat dairy (up to 2 servings/day) and lean animal protein (fish and fowl) served (in 3½ oz. portions) 4 days/week and in soups or casseroles (2 days/week). All foods except animal derived protein sources were served *ad libitum*. The exercise intervention consisted of 2 - 2.5 hr of supervised activity per day, including gym-based exercises, swimming, tennis, and beach games, intended to encourage physical activity in the subjects. Blood samples were drawn after a 12-hour overnight fast on days 1 and 12 of the intervention. The blood was separated by centrifugation and serum was shipped on dry ice to UCLA where it was stored at -80°C until analysis. Height and body weight were used to calculate BMI and were also assessed on these days using a stadiometer and calibrated scale. BMI was calculated as weight (kg)/height (m²).

**Determination of Serum Lipids, Glucose, Insulin, HOMA-IR, and QUICKI**
Total cholesterol, triglycerides (TG), high-density lipoprotein (HDL), and glucose levels were measured at a national commercial laboratory (Quest Diagnostics, Miami, FL) using standardized techniques. Low-density lipoprotein (LDL) was calculated as described by the Friedewald formula (7). Total-cholesterol/HDL and LDL/HDL ratios were also calculated. Insulin was quantified in duplicate using Luminex xMAP Multiplex (Millipore, Billerica, MA). The degree of insulin resistance was estimated with the use of the homeostatic model assessment for insulin resistance (HOMA-IR), calculated as the product of the fasting plasma insulin level (µU/ml) and the fasting plasma glucose level (mmol/L), divided by 22.5. Insulin sensitivity was also estimated by the quantitative insulin-sensitivity check index (QUICKI), as defined by 1/(log[fasting insulin (µU/ml)] + log[fasting glucose (mg/dl)]).

**Determination of Serum Cytokines and Metabolic Markers**

Serum IL-8, IL-10, IL-1ra, IL-6, TNF-α, PAI-1, resistin, amylin and leptin were measured in duplicate using specific Luminex xMAP Multiplex kits (Millipore, Billerica, MA) according to the manufacturer’s instructions. Serum adiponectin (ACRP) was measured in duplicate using enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, Minneapolis, MN). For a particular assay, all subject samples were assayed on the same day and according to the kit inserts recovery was generally 85-112% and the coefficient of variation <10%.

**Statistical Analysis**

Statistical analyses were performed with Graph Pad Prism (GraphPad, San Diego, CA) and STATA (StataCorp, College Station, TX). Pearson correlations between changes in anthropometrics and metabolic markers were performed in Prism. To determine the effect of
the intervention for each group and compare the changes between the normal weight and obese group we used a linear mixed model for repeated measures in STATA. All data are expressed as mean ± SE unless otherwise noted. A p value of < 0.05 was considered statistically significant.

Results

Anthropometric Characteristics, Blood Pressure, Lipids, Glucose and Insulin (Table 1)

Anthropometric and metabolic data are summarized in Table 1. Body weight, BMI, BMI percentile and waist circumference (WC) were significantly different at baseline between the two groups (all p<0.01). After the 2-week program, there was a non-significant weight reduction in N and O lost more weight (% decreases, N: 2.3% vs. O: 3.9%, N vs. O p<0.01) but remained obese (BMI percentile: 92.5±2.4% post vs. 95.2±1.4% pre, BMI: 32.3±1.9 vs. 33.6±1.9 kg/m²). WC decreased similarly in both groups (N: 4.8% vs. O: 4.3%, p=0.69), but the reduction was not significant in the N group (p=0.07).

Resting heart rate (RHR) and systolic and diastolic blood pressure (SBP and DBP) (N: SBP: 0.9%, DBP: 7.6%, RHR: 7.8% vs. O: SBP: 8.1%, DBP: 7.4%, RHR: 15.4%, all N vs. O p>0.10) had comparable decreases in both groups; but only significantly in the O group (N:DBP: p=0.09, SBP: p=0.8, RHR: p=0.082).

Total-C (N: 23.3% vs. O: 20.8%, p=0.84), LDL (N: 29.4% vs. O: 24.5%, p=0.81) and TG (N: 41.1% vs. O: 37.5%, p=0.63) all had similar significant decreases in both groups, p<0.01. Serum
HDL (N: 5.0% vs. O: 2.1%, p=0.38) decreased non-significantly in both N (p=0.07) and O (p=0.44).

Insulin (N: 52.5% vs. O: 28.1%, p=0.38) decreased in both groups, while glucose did not change significantly in N (1.3% decrease, p=0.66) or O (5.5% increase, p=0.05), nor was there a difference between the N and O (p=0.10). However, HOMA-IR (N: 53.1% vs. O: 28.4%, p=0.43) had similar significant decreases in both groups, p<0.05, and QUICKI (N: 9.3% vs. O: 4.9%, p=0.12) increased significantly in both groups, p=0.01, mainly driven by the decrease in insulin.

**Cytokines, Adipokines and Endocrine Markers (Figures 1, 2 and 3)**

All biomarkers were similar at baseline between the two groups except for PAI-1 (p<0.01), leptin (p<0.001), and IL-1ra (p<0.05). Serum PAI-1 (N: 30.8% vs. O: 25.6%, p=0.59), resistin (N: 40.0% vs. O: 35.1%, p=0.99), amylin (N: 28.7% vs. O: 26.1%, p=0.80) and leptin (N: 69.3% vs. O: 44.1%, p=0.10) decreased significantly (p<0.01) and similarly in both groups. Adiponectin (N: 29.3% vs. O: 41.8%, p=0.78) increased in both groups, but was only statistically significant in O (O: p<0.01, N: p=0.12).

Cytokine changes also exhibited similar responses between the N and O groups. IL-6 (N: 58.8% vs. O: 48.5%, p=0.78), IL-8 (N: 46.0% vs. O: 42.2%, p=0.49), and TNF-α (N: 45.8% vs. O: 40.8%, p=0.99) decreased significantly in both groups, p<0.05. IL-1ra decreased non-significantly in both groups (N: 32.8% vs. O: 19.9%, p=0.90). IL-10 did not change in either group (N: 7.0% decrease vs. O:4.9% increase, p=0.77).
Additionally, we did not detect any significant correlations between changes in waist circumference, body weight or BMI with changes serum cytokines, adipokines, or endocrine markers (all \( r \) values ranged between -0.34 and 0.41, all \( p>0.12 \)).

**Discussion**

Currently there is the perception that obesity is causally implicated in the pathogenesis of dyslipidemia, insulin resistance, inflammation, and other features related to metabolic health. It is certainly the case that adipose tissue, depending on location and characteristics, may exacerbate various risk factors. For example, it is well-established that visceral fat contributes to worsening of metabolic health (6, 25). However, if obesity is the primary cause, then: 1) reversal of obesity would be required to reverse phenotypic abnormalities, and 2) subjects who were not obese would not respond similarly to obese subjects. Regarding the first point, in previous seminal studies, we demonstrated that short-term lifestyle modification could ameliorate metabolic syndrome phenotypes in both men (19) and children (10). In addition, a variety of cardiovascular disease risk factors were improved in men (19, 20), women (28) and children (4, 18), despite modest weight loss and subjects remaining overweight/obese by BMI classification. Furthermore, when we correlated changes in body weight or BMI with changes in phenotypic outcomes, no significant associations were noted. We did, however, find significant correlations between various fatty acid species and inflammatory factors (10).

Regarding the second point, the present study was designed to test the hypothesis that responsiveness to a short-term, daily physical activity and plant-based *ad libitum* diet
An advantage of using a short-term intervention is that lifestyle changes can be assessed independent of obesity reversal. The findings of the current study indicated that effects were similar in both overweight/obese and normal weight subjects. This occurred even for metabolic outcomes that exhibited significant differences at baseline (PAI-1, leptin, and IL-1ra). Furthermore, the normal weight subjects had similar responses in measured phenotypes/metabolic markers to the overweight/obese subjects despite not being overweight/obese at baseline or exhibiting weight loss. The potential contributing lifestyle factors involved in the changes seen in the metabolic profile have been discussed previously (10) and likely include the decrease in saturated/trans fats and refined-sugar consumption and the increase in omega-3 fatty acids and nutrients (vitamins, minerals, phytochemicals, and fiber) from the diet, and the increase in physical activity, all of which can alter inflammatory and oxidative processes. Interestingly, the changes in serum cytokines, adipokines and endocrine markers were not associated with changes in body weight, BMI or waist circumference in either group. One explanation for the similar effect is that both the normal weight and obese subjects improve phenotypes related to lipid levels, insulin resistance, adipokines, etc., but the obese subjects have a genetic predisposition (1) to gain weight more readily compared with normal weight subjects. It is known that many obese subjects are not metabolically unhealthy (24), while many normal weight individuals (by BMI) are metabolically unhealthy (29). Thus, long-term, it is possible that with continued lifestyle modification obesity may be reversed, but even if not, a metabolically healthy, obese phenotype can develop, as was noted in the short-term with the obese subjects enrolled in the current study. Furthermore, given that weight loss attempts are typically associated with a high-degree of recidivism in adult and pediatric populations (5, 8);
lifestyle modification that focuses on the normalization of metabolic phenotypes may be of significant value in the pediatric population.

The noted benefits on metabolic, cardiovascular and inflammatory biomarkers independent of weight loss are not surprising, and it is apparent that the relationships between body weight and lifestyle are complex. For example, Phillips et al. (15, 27) provided different diets to obese young adults and noted that despite both groups exhibiting significant weight loss and decreased blood pressure, a “low-fat” diet improved, while a “low-carbohydrate” diet worsened endothelial function. In addition, Bradley et al. (3) noted that a low-fat diet improved, while a low-carbohydrate diet worsened augmentation index. Petersen’s group (11, 14) noted that with a short-term decrease in daily physical activity there was decreased insulin sensitivity and aerobic fitness, despite modest weight loss. Interestingly, in the Diabetes Prevention Program, those who only met the physical activity goal, but not the weight loss goal had a 44% decrease in diabetes incidence (9), while in the Finnish Diabetes trial achieving 4 hours/week of physical activity led to a reduction in diabetes risk in subjects who did not lose weight (26).

Also of interest in our cohort is the fact that baseline and percent change in several of the biomarkers were similar in normal weight and obese children, suggesting similar cardiovascular disease risk profiles. The Pathobiological Determinants of Atherosclerosis in Youth study demonstrated coronary atherosclerosis in both normal weight and obese males but more severe in the obese (12). It is possible that the mechanisms by which normal weight and obese subjects improve in an intervention of this type differ; however, this would require further investigation using molecular techniques to establish potential differences.
The current study has important strengths and limitations to consider. The major strength of the study is the monitoring permitted by the study. Monitoring food intake and physical activity reduces the need to query subjects about their compliance or to rely on food and activity questionnaires. Further, all exercise sessions were supervised facilitating adherence to the diet and activities. Additionally, the diet was ad libitum – a major advantage in cases where overeating is an issue – and thus, this is a more realistic program to implement into the daily lives of children rather than intentional caloric restriction. A limitation of the present study is the lack of body composition data to determine the fat mass and lean mass of both groups. Additionally, we did not look at diet and exercise independently and therefore cannot attribute the changes directly to either aspect of the intervention.

Overall, short-term, intensive lifestyle modification is effective in ameliorating several metabolic risk factors similarly in both normal weight and overweight/obese children. Furthermore, because the normal weight children were not obese and we did not find any associations between changes in indices of obesity and our serum measures; baseline obesity or weight loss per se were not the primary drivers leading to the phenotypic changes noted. These findings suggest that dietary intake and exercise/physical activity changes may be the underlying causes of the phenotypic changes noted. Additionally, the normal weight subjects exhibited metabolic abnormalities, likely due to their ongoing diet and lifestyle habits. Even though current public health recommendations are centered on overweight/obesity and the metabolic abnormalities associated with the same, we have demonstrated that there may be room for improvement in the metabolic profiles of individuals who are not overweight/obese. Given that body weight and weight change are poor surrogates for lifestyle (2), these findings reinforce the need to remind
even normal weight individuals about healthy lifestyle choices. Furthermore, therapies driven by BMI classification and weight loss in young patients may lead to missed opportunities to counsel them on the benefits of weight loss-independent effects of lifestyle modification, including proper diet and physical activity (21, 22). Overall, the results support the need for larger, randomized, long-term studies to investigate the impact of lifestyle modification on disease outcomes independent of weight loss.

Author Contributions
C.K.R. researched data, wrote the manuscript, and designed the study. A.I. researched data, wrote the manuscript, and designed the study. S.S.A. reviewed/edited manuscript. R.J.B. contributed to discussion and reviewed/edited manuscript. C.K.R. has full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Disclosure Statement
R.J.B. receives consulting honorarium from the Pritikin Longevity Center.

Grants
This work was supported by the American Heart Association (BGIA #0765139Y to C.K.R.), the National Heart, Lung and Blood Institute (P50 HL105188 to C.K.R.), the National Institute of Diabetes and Digestive and Kidney Diseases (DK090406 to C.K.R.).
References


**Figure and Table Legends**

**Table 1.** Anthropometric and lipid measures in normal weight and obese children undergoing a 14-day diet and exercise intervention. All data are expressed as mean ± SE. ‡p<0.01, *p<0.05. Baseline differences between the normal weight and obese group at are indicated as † p<0.05.

**Figure 1.** Effect of diet and exercise intervention on serum concentration of the cytokines IL-8, IL-6, TNF-α, IL-10, and IL-1ra in normal weight (filled bars) and obese (open bars) children. All data are expressed as mean ± SE. ‡p<0.01, *p <0.05 post-intervention vs. pre-intervention. Baseline differences between the normal weight and obese group at are indicated as † p<0.05. The baseline difference between normal weight and obese group for IL-10 was p=0.12.

**Figure 2.** Effect of diet and exercise intervention on serum concentration of the metabolic risk markers PAI-1, resistin, ACRP (adiponectin), leptin, and amylin in normal weight (filled bars) and obese (open bars) children. All data are expressed as mean ± SE. ‡p<0.01, *p <0.05 post-intervention vs. pre-intervention. Baseline differences between the normal weight and obese group at are indicated as † p<0.05.

**Figure 3.** Effect of diet and exercise intervention on percent changes in concentration of cytokines and metabolic risk markers in normal weight and obese children. No differences in changes between normal weight and obese group changes were noted post-intervention vs. pre-intervention. Percentage changes from baseline were calculated based on the geometric mean. All data are expressed as means. Error bars represent the 95% confidence interval.
Table 1. Anthropometric and metabolic phenotypes in normal weight and obese children

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Obese Subjects (n=19)</th>
<th>Normal Weight Subjects (n=14)</th>
<th>% Change</th>
<th>% Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Weight (kg)</td>
<td>94.0±7.4</td>
<td>90.3±7.1</td>
<td>-3.9$^\dagger$</td>
<td>-2.3</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>33.6±1.9</td>
<td>32.3±1.9</td>
<td>-3.8$^\dagger$</td>
<td>-2.3$^*$</td>
</tr>
<tr>
<td>BMI %tile</td>
<td>95.2±1.4</td>
<td>92.5±2.4</td>
<td>-2.8*</td>
<td>-6.9$^i$</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>97.2±5.7</td>
<td>93.0±4.8</td>
<td>-4.3$^\dagger$</td>
<td>-4.8</td>
</tr>
<tr>
<td>Resting HR</td>
<td>95±4</td>
<td>80±3</td>
<td>-15.4$^\dagger$</td>
<td>-7.8</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>125±4</td>
<td>115±2</td>
<td>-8.1$^\dagger$</td>
<td>-1.0</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>73±3</td>
<td>68±2</td>
<td>-7.4*</td>
<td>-7.6</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>81.3±2.0</td>
<td>85.8±1.3</td>
<td>5.5</td>
<td>-1.3</td>
</tr>
<tr>
<td>Insulin (µU/mL)</td>
<td>22.3±3.5</td>
<td>16.1±4.2</td>
<td>-28.1*</td>
<td>-52.5$^i$</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>4.8±0.8</td>
<td>3.4±0.8</td>
<td>-28.4*</td>
<td>-53.1$^i$</td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.31±0.01</td>
<td>0.33±0.01</td>
<td>4.9$^\dagger$</td>
<td>9.3$^i$</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>137.7±14.7</td>
<td>86.0±7.9</td>
<td>-37.5$^\dagger$</td>
<td>-41.1$^i$</td>
</tr>
<tr>
<td>Total-C (mg/dL)</td>
<td>166±6.2</td>
<td>132.0±5.8</td>
<td>-20.8$^\dagger$</td>
<td>-23.3$^i$</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>95.0±6.1</td>
<td>71.8±5.0</td>
<td>-24.5$^\dagger$</td>
<td>-29.4$^i$</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>44.0±2.2</td>
<td>43.1±2.7</td>
<td>-2.1</td>
<td>-5.0</td>
</tr>
<tr>
<td>Total-C/HDL</td>
<td>4.0±0.3</td>
<td>3.3±0.3</td>
<td>-18.2$^\dagger$</td>
<td>-19.0$^i$</td>
</tr>
<tr>
<td>LDL/HDL</td>
<td>2.3±0.2</td>
<td>1.8±0.2</td>
<td>-20.7$^i$</td>
<td>-24.0$^i$</td>
</tr>
</tbody>
</table>
FIGURE 1

**IL-8**

**IL-6**

**TNF-a**

**IL-10**

**Il-1ra**

![Bar charts showing changes in cytokine levels between pre and post states for normal weight and obese participants.](image_url)
FIGURE 2

PAI-1

Resistin

ACRP

Leptin

Amylin

Normal Weight

Obese