

Pentraxin 3 Reactivity to Acute Exercise in Obese Individuals

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Introduction and Purpose

- Pentraxin-3 (PTX-3) has recently been identified as a biomarker of vascular inflammation that is mainly released by macrophages and endothelial cells (Ogawa et al., 2010) as well as associated with cardiovascular disease (CVD) (Jenny et al., 2009)
- PTX3 deficiency promotes vascular inflammation, atherosclerosis, and heart damage (Norata et al. 2009)
- High circulating PTX-3 levels have been found in obese individuals with low arterial distensibility (Miyaki et al., 2010), whereas other studies showed decreased plasma PTX-3 in obese populations (Ogawa et al., 2010)
- Regular aerobic exercise enhances resting plasma PTX-3 production, suggesting that exercise may provide anti-inflammatory and cardioprotective actions to prevent the risk of CVD (Miyaki et al., 2011)
- The purpose of this study was to investigate whether obese subjects would elicit a greater plasma PTX-3 reactivity following acute intense exercise compared to the normal-weight subjects
- Possible relationships of exercise-induced PTX-3 with cardiorespiratory fitness level and insulin sensitivity were also examined

Methods

- Twenty-one subjects (9 obese and 12 normal-weight) were asked to fast overnight and to participate in a maximal exercise testing
- Baseline measurements of resting heart rates and blood pressure were measured, along with calculations of body mass index (BMI)
- Blood samples were collected for measurements of PTX-3, insulin, and glucose prior to and immediately following exercise, post-exercise one hour, and post-exercise two hours. The area-under-the-curve (AUC) for PTX-3, insulin, and glucose were calculated
- HOMA-IR (index of insulin resistance) was calculated was quantified according to (Matthews et al 1985) using the following equation: $HOMA-IR = \text{Glucose (mmol/L)} \times \text{Insulin } (\mu\text{IU/mL}) / 22.5$

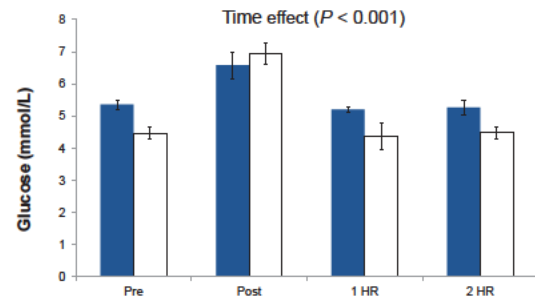
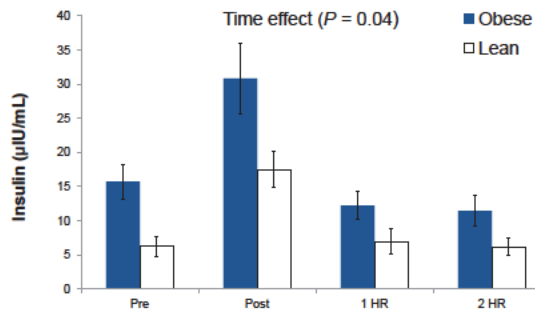
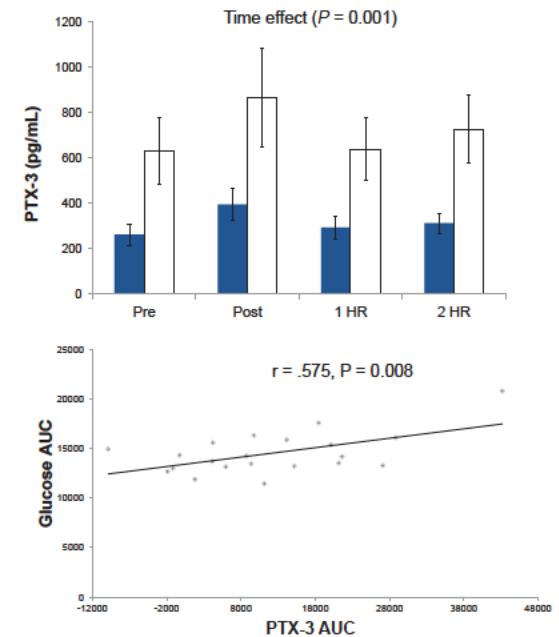
Statistical Analysis

- Differences between obese and normal-weight groups in baseline variables were computed by independent t tests
- A 2 by 4 (group \times time) repeated-measures ANOVA were used to analyze PTX-3, insulin, and glucose responses between obese and normal-weight groups
- Pearson product-moment correlations were utilized to examine the relationships of PTX3 with glucose and insulin. The α level was set at $P \leq 0.05$

Results

Table 1: Participant anthropometric characteristics and baseline markers

Variables	Obese (n=9)	Non-Obese (n=12)	P Value
Age (years)	21.56 (± 0.73)	23.17 (± 0.63)	0.109
Gender (Male/Female)	4/5	6/6	0.801
Height (m)	1.676 (± 0.03)	1.696 (± 0.03)	0.675
Weight (kg)	97.88 (± 6.3)	64.24 (± 3.5)	< 0.001
BMI (kg/m ²)	34.59 (± 1.3)	22.11 (± 0.47)	< 0.001
Systolic Blood Pressure (mmHg)	127.1 (± 3.7)	107 (± 3.6)	0.001
Diastolic Blood Pressure (mmHg)	84.22 (± 2.2)	67.5 (± 2.9)	< 0.001
Resting Heart Rate (BPM)	71.11 (± 2.8)	67.33 (± 2.7)	0.354
VO _{2max} (mL/kg ⁻¹ /min ⁻¹)	31.47 (± 1.9)	46.66 (± 2.3)	< 0.001
Fasting Pentraxin-3 (pg/mL)	262.8 (± 47)	631.6 (± 149)	0.034
Fasting Insulin (μ IU/mL)	15.75 (± 2.5)	6.227 (± 1.4)	0.006
Fasting Glucose (mmol/L)	5.356 (± 0.15)	4.457 (± 0.19)	0.001
HOMA-IR	3.727 (± 0.60)	1.279 (± 0.32)	0.003



Conclusions

- Our results showed that exercise elicited similar elevations in PTX3, insulin, and glucose in both obese and normal-weight subjects
- Furthermore, PTX3 area-under-curve (AUC) was significantly correlated with glucose AUC, even when controlled for BMI and fitness level
- These findings suggest glucose may potentially regulate PTX3 response to exercise
- Further investigation is needed to verify the impact of substrate utilization on exercise-induced PTX3 elevation

References

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