Plasma Pentraxin 3 and Glucose Kinetics to Acute High-Intensity Interval Exercise versus Continuous Moderate-Intensity Exercise in Healthy Men

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Title: Plasma Pentraxin 3 and Glucose Kinetics to Acute High-Intensity Interval Exercise versus Continuous Moderate-Intensity Exercise in Healthy Men

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Abstract

Pentraxin 3 (PTX3) is mainly synthesized and released by neutrophils to help regulate innate immunity. While plasma PTX3 concentrations are associated with improved glucose metabolism and overall metabolic health, evidence has shown that significant elevations in plasma glucose downregulates circulating levels of PTX3. To examine whether or not this relationship would be altered in response to exercise, this study investigated the kinetics of the plasma glucose and PTX3 response following high-intensity interval exercise (HIIE) and continuous moderate-intensity exercise (CMIE). It was hypothesized that the increased concentrations of plasma glucose following HIIE would be associated with the attenuated plasma PTX3 response compared to CMIE. Eight healthy male subjects participated in both HIIE and CMIE protocols administered as a randomized, counterbalanced design. Linear mixed models for repeated measures revealed that the overall plasma glucose response was greater following HIIE compared to CMIE (protocol x time effect: $p = 0.037$). To the contrary, although the plasma PTX3 response was only higher at 19 minutes during HIIE than CMIE (protocol x time effect: $p = 0.013$), no relationships were observed between plasma glucose and PTX3 in either baseline or in response to both exercise protocols, as indicated by area under the curve “with respect to increase” analysis. Our results indicate that exercise-mediated plasma PTX3 concentrations are independent of plasma glucose response. In addition, the present study suggests that the neutrophil-mediated innate immune response, as indicated by plasma PTX3 response, may be activated earlier during HIIE compared to CMIE.
Keywords: High-Intensity Interval Exercise; Continuous Moderate-Intensity Exercise; Pentraxin 3; Glucose

Introduction

Pentraxin 3 (PTX3) is an acute phase reactant commonly examined for its capacity to regulate innate immune function and protect against cardiovascular disease (Dias et al. 2001, Salio et al. 2008, Norata et al. 2009). More recently, plasma PTX3 concentrations have been shown to be associated with improved glucose metabolism and overall metabolic health (Yamasaki et al. 2009, Chu et al. 2012, Miyaki et al. 2014, Slusher and Huang 2016, Escobar-Morreale et al. 2017, Slusher et al. 2017, Weiss et al. 2017). For example, elevated fasting plasma PTX3 concentrations are associated with increased insulin sensitivity following intravenous (0.3 g/kg) and oral glucose administration (75 g) in humans (Osorio-Conles et al. 2011). It has further been suggested that PTX3 may enhance insulin sensitivity and facilitate glucose uptake into peripheral tissues as a mechanism to protect against metabolic disease (Weiss et al. 2017), a posit supported in metabolically healthy and diabetic mice expressing a positive association of skeletal muscle PTX3 protein levels with insulin-responsive glucose transporter (GLUT4) (Miyaki et al. 2014). Interestingly, Escobar-Morreale et al. (2017) have recently demonstrated that an oral glucose challenge (75 g) lowers circulating concentrations of plasma PTX3 in humans.

Physical activity is an essential component to regulate glucose metabolism and has been shown to dose-dependently reduce the risk for metabolic disease by up to 30% (Kyu et al. 2016). Interestingly, improved glucose homeostasis following short-term (i.e., 7 days) and long-term (i.e., 3-6 months) dietary and/or physical activity intervention correlates with elevated plasma PTX3 concentrations (Chu et al. 2012, Weiss et al. 2017). While our laboratory and others have
further shown that acute exercise ≥ 60% of an individual’s cardiorespiratory fitness level (assessed by maximal oxygen consumption; VO\textsubscript{2max}) increases plasma PTX3 concentrations (Nakajima et al. 2010, Huang et al. 2014, Slusher et al. 2015), this response is independent of the glucose response following maximal exercise (Slusher and Huang 2016). However, the capacity of plasma glucose to alter PTX3 concentrations during exercise, and potentially vice versa, may be influenced by the intensity of exercise employed. For example, continuous moderate-intensity exercise (CMIE; e.g., ≤ 60% VO\textsubscript{2max}) facilitates the migration of the GLUT4 protein to the plasma membrane and transverse tubules in contracting skeletal muscle to facilitate glucose uptake independently of insulin (Douen et al. 1990, Goodyear et al. 1991, Marliss and Vranic 2002). Likewise, the rate of glucose uptake by skeletal muscle is matched by hepatic metabolism during CMIE, resulting in little to no net changes in plasma glucose concentrations, whereas at high exercise intensities (i.e., ≥ 80% VO\textsubscript{2max}), glucose production exceeds skeletal muscle uptake and results in elevated plasma glucose concentrations (Marliss and Vranic 2002). Moreover, a single session of high intensity-interval exercise (HIIE), characterized by repeated bouts of brief, vigorous exercise separated by periods of recovery, would elicit an elevation in plasma glucose, not typically observed during CMIE (Adams 2013). Therefore, this study examined the kinetics of the plasma glucose and PTX3 response at various time points during and throughout recovery from HIIE and CMIE. Given the capacity of plasma glucose to decrease circulating PTX3 concentrations (Escobar-Morreale et al. 2017), it was hypothesized that the increased concentrations of plasma glucose would be associated with the attenuated (less robust) plasma PTX3 response following acute HIIE compared to CMIE.

**Materials and Methods**
Subjects

Eight healthy, physically inactive, male subjects were recruited to participate in this study. Prior to participation, all subjects provided their informed consent and completed a medical history questionnaire. Subjects were excluded from participation if they had been previously diagnosed with any inflammatory or metabolic disease (e.g., cardiovascular disease, diabetes), orthopedic limitations, or were under the current administration of medication known to alter inflammatory or metabolic profiles. Additional exclusion criteria included the use of tobacco products (cigarettes, cigars, chewing tobacco), and consumption of more than 10 standard alcoholic beverages/week (i.e., 14 grams of pure alcohol/drink; 350 mL of beer, 150 mL of wine, and 44 mL of liquor). Finally, females were excluded to reduce the influence of gender on the relationship between plasma PTX3 and glucose (Escobar-Morreale et al. 2017). The University’s Institutional Review Board approved the experimental procedures for this investigation.

Assessment of Peak Oxygen Consumption

Three testing sessions comprised the data collection and each subject arrived to the laboratory at the same time of day for each visit. In addition, subjects were instructed to fast for at least 3 hours and to abstain from alcohol and caffeine intake for 24 hours and intense physical activity for at least 24 hours prior to each laboratory visit. Upon arrival, subjects rested quietly in a seated position for at least five minutes to obtain resting heart rate and systolic and diastolic blood pressure using a chest strap HR monitor (Polar T31, Polar Electro, Kempele, Finland) and sphygmomanometer (752M-Mobile Series, American Diagnostic Corporation, Hauppauge, NY), respectively. In addition, subject’s height and weight were assessed using standard medical
devices to calculate body mass index. Subsequently, each subject participated in a graded exercise test on an electronically-braked cycle ergometer (Lode Excalibur Sport, Model #909901, Groningen, Netherlands) designed to elicit peak oxygen uptake as assessed by open-circuit spirometry (ParvoMedics Metabolic Measurement System, Sandy, UT, USA). Carbon dioxide (VCO₂) and oxygen (O₂) were measured throughout the assessment of peak oxygen consumption and averaged every 15 seconds to calculate respiratory exchange ratio (RER: VCO₂/VO₂). Prior to the beginning the assessment of peak oxygen consumption, the cycle ergometer was adjusted to correspond to the dimensions of the subject; seat height, handlebar height and distance from nose of the seat to the center of the handlebars. These dimensions were recorded and employed across all three laboratory visits (visits two and three described below).

After a 10-minute warm-up at 50 watts (W), peak exercise test consisted of one-minute stages beginning at a workload of 100 W. Workload was increased an additional 25 W every minute. However, once participants achieved an RER of 1.05 or self-selected cadence decreased by 10 rpm or more, the workload was increased by 10 W every minute to ensure that volitional exhaustion was due to the obtainment of maximal oxygen consumption and not due to the subject’s inability to continue exercising from localized skeletal muscle fatigue. Finally, HR and rating of perceived exertion (RPE) using the Borg 15-point scale were recorded during the last 15 seconds of each exercise state. Max power (W_max) were collected throughout the exercise testing protocol. W_max was determined according to the following formula (Kuipers et al. 1985):

\[ W_{\text{max}} = W_{\text{com}} + \frac{t}{T \times W} \]

- \( W_{\text{com}} \) is the power of the last completed stage of the VO₂peak test
- t is the number of seconds into the uncompleted stage
- T is stage length
- W is the power output of the uncompleted stage.

**Exercise Testing Session**

Sessions two and three occurred a minimum of 48 hour after the assessment of peak oxygen consumption and consisted of participation in a HIIE or CMIE protocol administered as a randomized, counterbalanced design no less than 48 hours and no more than two weeks apart. Each exercise protocol began with a 10-minute warm-up at 50 W, immediately followed by either; HIIE – 10 high-intensity intervals of cycling for 60 seconds at 90% \( W_{\text{max}} \) separated by 2 minutes of active rest, or CMIE – 28 minutes of continuous exercise at 60% \( W_{\text{max}} \). Total work output (kilojoules [kJ]) was calculated from the percentage of \( W_{\text{max}} \) for each exercise protocol (HIIE = 90%; CMIE = 60%) and multiplied by total exercise time (in seconds) (HIIE = 600 s; CMIE = 1680 s) / 1000. Of note, the electronically-braked cycle ergometer continuously adjusted resistance based upon the subject’s cadence to maintain the appropriate percentage of \( W_{\text{max}} \) specified for each exercise protocol.

**Measurements of PTX3 and Glucose**

Upon arrival for sessions two and three, an intravenous catheter was inserted into each subject’s antecubital vein for the collection of whole blood samples (6 mL) in a tube containing \( K_2 \) ethylenediaminetetraacetic acid (BD Vacutainer, Franklin Lakes, NJ, USA). Whole blood samples were collected prior to (Pre) participation in the 10-minute warm-up, immediately following each high intensity interval and corresponding times during the CMIE (minutes 1, 4, 7,
10, 13, 16, 19, 22, and 25), immediately upon completion (Post), and 30 and 60 minutes into recovery (R30 and R60, respectively) from both HIIE and CMIE exercise protocols. Whole blood was immediately centrifuged for 15 minutes at 3000 rpm (1000 x g) at room temperature and stored at -80°C for further analyses of plasma glucose (Cayman Chemical Company, Ann Arbor, MI, USA) and PTX3 (R&D Systems, Minneapolis, MN, USA) in duplicate with enzyme-linked immunosorbent assay (ELISA) kits and according to manufacturer’s instructions.

**Statistical Analyses**

Data analyses were performed using the Statistical Package for the Social Sciences (SPSS version 23.0). Independent *t*-tests were conducted to compared resting plasma glucose and PTX3 concentrations prior to participation in HIIE and CMIE, and to compare total work between exercise protocols. A two protocol (HIIE vs. CMIE) by thirteen time point (Pre, intra-exercise [E] minutes E1, E4, E7, E10, E13, E16, E19, E22, and E25, Post, R30, and R60) linear mixed model for repeated measures analysis of variance was utilized to examine changes in plasma glucose and PTX3 concentrations. Significant effects were further analyzed with Fisher’s LSD post hoc comparisons. In addition, to examine for the potential influence that differences in total work performed between the HIIE and CMIE protocols may have exerted on the plasma glucose or PTX3 responses, analysis of variances were performed with and without controlling for total work output (kJ). To assess the intensity of the exercise-induced plasma glucose and PTX3 response relative to pre-exercise conditions, area under the curve “with respect to increase” (AUCi) was calculated according to Pruessner et al. (2003). Pearson’s correlations were utilized to examine the relationship of plasma glucose and PTX3 concentrations prior to and in response to both HIIE and CMIE protocols (AUCi). In addition, to better understand the potential impact...
of the plasma glucose response on PTX3 concentrations during and throughout recovery from exercise, Pearson’s correlations were utilized to examine the relationship between peak levels of plasma glucose and PTX3, and the time points that peak concentrations were observed to have occurred, during HIIE, and separately, during CMIE. Finally, a post-hoc power analysis was conducted using program G*Power (version 3.1.9.2) for primary outcome measures. Based on the differences of mean and standard deviation with an alpha level of 0.05 at the time points (E25 for glucose and E19 for PTX3; assessed by paired t-tests) where the difference was observed between HIIE and CMIE, the overall sample size of 8 subjects in this study achieved an adequate power (> 80%) for all outcome variables. Statistical significance was set at $p \leq 0.05$.

**Results**

Subject descriptive characteristics and peak oxygen consumption values are presented in table 1. No differences were observed in the baseline levels of plasma glucose or PTX3 concentrations between HIIE and CMIE ($t_{[14]} = 0.460, p = 0.653$; $t_{[14]} = -0.174, p = 0.864$, respectively). Total work was significantly lower in response to HIIE compared to CMIE ($164.89 \pm 20.72$ kJ versus $307.79 \pm 38.69$ kJ, respectively; $t_{[14]} = 9.21, p \leq 0.001$). A linear mixed model for repeated measures revealed a significant Protocol * Time interaction effect for the plasma glucose response to HIIE compared for CMIE with and without controlling for differences in total work ($F_{[12, 124.606]} = 1.923, p = 0.037$; $F_{[12, 124.565]} = 1.932, p = 0.036$, respectively; Figure 1A). More specifically, the plasma glucose response was greater 25 minutes into exercise and 60 minutes into recovery following HIIE compared to CMIE, whereas CMIE showed a higher level at 1 minute into exercise. In addition, HIIE significantly increased plasma glucose concentrations 25 minutes into and immediately following exercise relative to baseline,
whereas CMIE significantly decreased plasma glucose concentrations 10 minutes into exercise \((p \leq 0.05)\). A linear mixed model for repeated measures also revealed a significant Protocol \(\times\) Time interaction effect for the plasma PTX3 response to HIIE compared to CMIE with and without controlling for differences in total work \((F_{[12, 107.301]} = 2.266, p = 0.013; F_{[12, 107.584]} = 2.272, p = 0.013\), respectively; Figure 1B). Plasma PTX3 concentrations were elevated 19 minutes into HIIE compared to CMIE, whereas CMIE elevated plasma PTX3 concentrations from 22 minutes into CMIE through 30 minutes into recovery from exercise. Finally, no relationship was observed between plasma glucose and PTX3 either at baseline or in response (as assessed by AUCi analysis) to HIIE \((r = -0.132, p = 0.755; r = 0.141, p = 0.739\), respectively) and CMIE \((r = -0.321, p = 0.438; r = 0.011, p = 0.980\), respectively). Likewise, no relationships were observed between peak plasma glucose and PTX3 concentrations, or the time points that peak concentrations were observed, in response to HIIE \((r = -0.104, p = 0.804; r = 0.290, p = 0.486\), respectively) or CMIE \((r = -0.465, p = 0.245; r = 0.425, p = 0.294\), respectively).

**Discussion**

The primary purpose of this study was to measure the plasma glucose and PTX3 kinetic responses following HIIE and CMIE. Consistent with the hypothesis presented by Adams (2013), plasma glucose concentrations increased above baseline during (E25) and immediately following completion of HIIE, whereas plasma glucose concentrations decreased at E10 and remained unaltered compared to resting values during and throughout recovery from CMIE. Previous research has demonstrated that hepatic glucose production exceeds glucose uptake by skeletal muscle in response to high intensity exercise (i.e., 87% VO\(_{2\text{max}}\)), whereas the balance in glucose production and uptake is relatively balanced during CMIE (Marliss and Vranic, 2002).
Likewise, this difference in glucose production and uptake continues to be observed during the initial stages of recovery from exercise to a great extent following higher exercise intensities (Marliss and Vranic, 2002), potentially explaining the differential glucose kinetics observed in response to HIIE and CMIE in the present study.

Plasma PTX3 concentrations were also differentially altered in response to HIIE compared to CMIE. For example, HIIE significantly increased plasma PTX3 concentrations at E19 relative to concentrations measured at the same time point during CMIE. To the contrary, while HIIE did not elicit a significant change in plasma PTX3 during and throughout recovery from exercise compared to baseline concentrations measured prior to participation in exercise, plasma PTX3 concentrations were significantly increased from E22 to 30 minutes into recovery from CMIE. Although recent evidence has demonstrated that elevations in plasma glucose concentration decrease circulating PTX3 concentrations in plasma (Escobar-Morreale et al. 2017), no relationships were observed in the overall (i.e., AUCi) plasma glucose and PTX3 response to HIIE or CMIE in the present study. Likewise, no relationships were observed in peak plasma glucose and PTX3 concentrations, or the time points that peak concentrations were observed, during and throughout recovery from either exercise protocol. These results further suggest that increased levels of plasma glucose, specifically during HIIE, may not play a role in modulating plasma PTX3 concentrations in response to physical activity.

Our laboratory and others have demonstrated that plasma PTX3 concentrations are positively associated with enhanced insulin-mediated glucose uptake under resting conditions (Yamasaki et al. 2009, Chu et al. 2012, Miyaki et al. 2014, Slusher et al. 2017). Similarly, Weiss et al. (2017) recently demonstrated that elevated concentrations of circulating PTX3 concentrations in response to long-term (i.e., 3-6 months) caloric restriction and/or physical
activity intervention are an independent predictor of improved insulin sensitivity following oral glucose challenge under resting conditions. However, the causality of the relationship between PTX3 with insulin and glucose responses to metabolic challenge remains unclear. For example, although Escobar-Morreale et al. (2017) demonstrates that oral glucose challenge decreased plasma PTX3 concentrations, the PTX3 response was associated with the reciprocal increase in insulin concentrations, suggesting that insulin, but not glucose, may potentially be responsible for decreasing plasma PTX3 concentrations in individuals with metabolic disease. On the other hand, regular participating in physical activity has been shown to increase plasma PTX3 concentrations at rest (Miyaki et al. 2011, 2012). Likewise, our laboratory and others have shown that PTX3 inhibits cellular production of pro-inflammatory cytokines (i.e., tumor necrosis factor alpha) and increases the production of anti-inflammatory cytokines (i.e., interleukin 10 and transforming grown factor beta) (Shiraki et al. 2016, Slusher et al. 2016). The PTX3-mediated alteration of inflammatory milieu may restore the capacity of insulin to mediate glucose uptake within skeletal muscle typically impaired under resting conditions in individuals with pro-inflammatory diseases, including type 2 diabetes mellitus (T2DM) (Hotamisligil et al. 1993, Miyaki et al. 2014). Therefore, while the results from the present investigation suggest that plasma PTX3 concentrations are independent of plasma glucose at rest and in response to acute exercise, the long-term anti-inflammatory capacity of physical activity, in part evidenced by increased plasma PTX3, may enhance the capacity of PTX3 to regulate glucose homeostasis.

Another important finding indicates that HIIE does not elicit a plasma PTX3 response immediately following or during recovery from exercise. Although the precise mechanisms responsible for the elevated plasma PTX3 concentrations typically observed in response to acute exercise remain unknown, Nakajima et al. (2010) have demonstrated that neutrophils are a
significant contributor to the systemic PTX3 response to acute exercise. Neutrophils synthesize PTX3 throughout development and store readily available, intracellular concentrations of PTX3 upon reaching maturation (Jaillon et al. 2007). These mature neutrophils release PTX3 into circulation following their activation as a protective mechanism to regulate the innate immune response and prevent the excess transmigration of neutrophils into the vascular endothelium (Jaillon et al. 2007, Deban et al. 2010). While Nakajima et al. (2010) further demonstrates that elevated PTX3 concentrations observed following acute continuous aerobic exercise are dependent upon intensity, the present study suggests that HIIE may have activated the neutrophil-mediated PTX3 response earlier compared to CMIE (observable at E19), which returns to baseline levels throughout the remaining bouts of exercise. However, no differences were observed in peak PTX3 concentrations, or the time points that peak concentrations were observed, between exercise protocols (data not shown). Our laboratory has recently shown that compared to CMIE, indices of neutrophil activation (i.e, plasma calprotectin and myeloperoxidase [MPO]) also appear to be lower throughout recovery from HIIE (Fico et al. 2018). These findings suggest that additional research is necessary to determine whether the intermittent nature, or the work-to-rest ratio of our HIIE protocol influences the mobilization (i.e., changes in neutrophil cell counts, and/or activation of circulating neutrophils, such as changing in intracellular PTX3, calprotectin, and MPO concentrations), and to what extent this response would influence the release of PTX3 and other stored regulators of the neutrophil-mediated innate immune response during and throughout recovery from exercise.

In conclusion, results from the present investigation demonstrate that while HIIE and CMIE differentially influence the plasma glucose and PTX3 responses during and in response to acute exercise, the observed plasma PTX3 response appears to be independent of glucose. Of
note, the present study consisted of healthy, young men, and future studies should consider the inclusion of populations that are at risk (i.e., obesity) or have previously been diagnosed with T2DM to better understand the dynamic relationship between plasma PTX3 and glucose. Furthermore, HIIE has gained increased attention in the literature as a time-efficient alternative to CMIE that elicits similar, if not superior, cardiovascular, metabolic, and neurocognitive benefits (Little et al. 2014, Connolly et al. 2016, Gillen et al. 2016, Tsukamoto et al. 2016). In light of these findings, the present study supports previous findings from our laboratory (i.e., calprotectin) that compared to CMIE, HIIE elicits these positive physiological benefits while stimulating an earlier neutrophil-mediated innate immune response that is no longer observable during recovery from exercise (Fico et al. 2018). It is therefore imperative that additional research be conducted to determine the underlying mechanisms responsible for the altered innate immune response following HIIE compared to CMIE, and in turn, the capacity to which these responses prevent and/or treat chronic health disorders.

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Conflicts of Interest

The authors declare that they have not conflicts of interest.

References

Adams, O. 2013. The impact of brief high-intensity exercise on blood glucose levels. Diabetes,


Figure Legends

**Figure 1.** Kinetics of the plasma glucose (panel A) and PTX3 (panel B) response at various time points during and throughout recovery from HIIE and CMIE. ¥ indicates a significant difference in plasma glucose or PTX3 concentrations between HIIE and CMIE at corresponding time points ($p \leq 0.05$); * indicates a significant difference in plasma glucose or PTX3 concentrations relative to pre-exercise in HIIE ($p \leq 0.05$); # indicates a significant difference in plasma glucose or PTX3 concentrations relative to pre-exercise in CMIE ($p \leq 0.05$). Data are presented as means ± SEM.
Fig 1 Kinetics of the plasma glucose (panel A) and PTX3 (panel B) response at various time points during and throughout recovery from HIIE and CMIE. ¥ indicates a significant difference in plasma glucose or PTX3 concentrations between HIIE and CMIE at corresponding time points ($p \leq 0.05$); * indicates a significant difference in plasma glucose or PTX3 concentrations relative to pre-exercise in HIIE ($p \leq 0.05$); # indicates a significant difference in plasma glucose or PTX3 concentrations relative to pre-exercise in CMIE ($p \leq 0.05$). Data are presented as means ± SEM.
Table 1. Subject descriptive characteristics and peak oxygen consumption values

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<td>Resting SBP (mmHg)</td>
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<tr>
<td>VO2peak (mL O₂ · kg⁻¹ · min⁻¹)</td>
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Note: Data are presented as means ± SD. BMI = body mass index; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; VO2peak = peak oxygen consumption.