C-Reactive Protein and the Disease Analog Model May Identify Predisposed Pre-Obese African-American Women

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Introduction

While the obesity rate in the Unites States has been reported to have hit a plateau, the overall percentage of obese Americans remains alarmingly high (27% self-reported, 33% population estimate). While the subgroup with the highest 2010 obesity rate is Black, non-Hispanic women (41.9%), there remains a disparity in the research with regards to this population group. The implication of an elevated obese population puts a strain on health care, overall quality of life, and is associated with a number of other comorbidities. Given this background, pilot work to evaluate a disease analog model for obesity would be useful with the potential for identifying seemingly normal-weight individuals who are most susceptible to developing obesity.

Graded exercise tests are the most common diagnostic tool utilized to identify individuals with underlying cardiac conditions. Analogous to how a graded exercise test is used in the clinical cardiac setting, it may be possible that a similar model can be implemented to identify the most predisposed normal weight individuals based on biomarkers of chronic inflammation associated with obesity following an acute exercise bout. We hypothesized that obesity represents a chronic disease state, and that exercise may represent an acute state of physiological perturbation in predisposed but otherwise normal weight individuals. In this model, post-exercise biomarker values for chronic disease (such as C-reactive protein, CRP) in predisposed individuals would be similar to the resting values of our obese subjects. In this investigation, we evaluated the ability of the plasma biomarker C-reactive protein to serve as an indicator of a predisposition toward obesity in apparently healthy African-American females.

Methods

Subjects: Participants in this pilot investigation were six apparently healthy women (age = 26 ± 4 yrs, height = 165 ± 2 cm, weight = 69 ± 4 kg, BMI = 25 ± 1 kg·m⁻²), and seven obese women (age = 37 ± 4 yrs, height = 166 ± 2 cm, weight = 93 ± 6 kg, BMI = 33 ± 2 kg·m⁻²). Participants were categorized as non-obese or obese (BMI>30 kg·m⁻²) based on BMI measurements. Prior to participation all subjects completed an informed consent document that was approved by the university Human Subjects Review Board.

Protocol: Participants reported to the Exercise Physiology Laboratory on one occasion in which anthropometric measurements were obtained, and a graded exercise test was performed using the Bruce protocol. Blood samples were obtained at rest, immediately following exercise, and 1-hour after the exercise bout. Whole blood was centrifuged to separate plasma, and stored at -80°C for subsequent analysis. Plasma C-reactive protein was measured using a commercially available kit (Signosis, Inc., Sunnyvale, CA).

Results

The resting C-reactive protein value for obese African-American women was 6.13 ± 1.27 mg·L⁻¹ (SEM), which was significantly greater than non-obese participants (2.29 ± 1.16 mg·L⁻¹) (P = 0.03). As we hypothesized that following exercise, predisposed individuals would display values similar to the obese group at rest, we evaluated each non-obese participant individually (see figure 1). We found that one apparently healthy participant (subject 3) did produce post-exercise CRP values that were similar to the mean of the obese group.



Figure 1. C-reactive protein following a graded exercise test (Post) and 1-hour following the bout (1H Post) in apparently healthy non-obese African American women (N = 6). The line represents the mean CRP value of the obese group.

Discussion

The purpose of this pilot study was to evaluate if a disease analog model utilizing the biomarker C-reactive protein could identify African-American women susceptible to developing obesity. We theorized that exhaustive exercise could induce acute changes in predisposed individuals that would be similar to the chronically elevated CRP levels observed in obese women. We observed an elevated level of CRP in one apparently healthy individual, which suggests that this disease analog model could show promise in identifying individuals with the propensity for developing a chronic disease such as obesity. While the subject number in this pilot investigation is admittedly small, the fact that we did observe a change in one individual shows potential. Further investigation with a much larger subject pool is warranted, as is a longitudinal study to follow individuals identified with a possible predisposition to observe whether the chronic disease does in fact develop. In addition, a greater panel of chronic disease biomarkers should be employed to determine whether single factors are appropriate or if a disease analog index could be developed.