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Adiposity, stress, and stigmatization: A biopsychosocial approach to cardiovascular disease prediction

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Adiposity, Stress, and Stigmatization: A Biopsychosocial Approach to Cardiovascular Disease Prediction

by

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ABSTRACT

The purpose of the present study was to use a biopsychosocial approach to investigate the role of body image concerns and appearance-related evaluation stress in the relationship between adiposity and cardiovascular reactivity and recovery. Participants included 106 Caucasian female undergraduates at the University of South Florida. The laboratory procedure consisted of resting baseline, speech preparation and delivery, and recovery phases. Participants also completed a variety of body image questionnaires. To manipulate appearance-related evaluation stress, participants were randomly assigned to present their speech to a video camera or an audio recorder. Overall adiposity was measured as body mass index (BMI) and central adiposity was measured as waist-to-hip ratio (WHR). Greater adiposity was associated with more weight-related anxiety during the speech task, regardless of speech condition (BMI: $r = .54$, $p < .001$; WHR: $r = .44$, $p < .001$). Additionally, those in the video condition reported more weight-related anxiety ($M = 9.28$, $SD = 7.74$) during the speech task than those in the audio condition ($M = 3.31$, $SD = 5.61$; $F(1,99) = 19.73$, $p < .001$). Significant relationships between adiposity and CVR and recovery emerged for several outcome measures with central and overall adiposity predicting different CVR patterns (all $ps < .05$). There was no main effect of speech condition on CVR or recovery (all $ps > .05$). Significant interactions between

adiposity and speech condition emerged for some of the outcome variables as well (all *ps* < .05). Additionally, body image concerns mediated the relationship between adiposity, speech condition, and CVR for SV and CO reactivity. The results suggest that adiposity may have a robust effect on physiological reactivity and recovery independent of psychological processes that co-occur. The results also suggest that those with high adiposity experience more weight-related anxiety during evaluation, even when the evaluation is not intended to induce body image concerns. Finally, the results suggest that body image concerns may act to suppress reactivity among those with high levels of weight-related anxiety. Implications and future directions for research are discussed.

Introduction

Cardiovascular disease (CVD) is defined as any disease of the heart or blood vessels and includes coronary artery disease, stroke, hypertension, and others. According to the American Heart Association (AHA), CVD is the number one cause of mortality in the United States and is estimated to cost over $400 billion in 2008 (Rosamond et al., 2008). Furthermore, the AHA's prevalence estimates suggest that one out of every three adults has some form of CVD. Thus, the high cost and prevalence of CVD makes it a significant public health concern. As such, many health researchers have focused on pinpointing different risk factors for CVD. Many of these risk factors have emerged from biological, psychological, and social approaches, each of which has offered unique insight into CVD prediction.

Biological Approach: Adiposity and CVD

One common biological approach has focused on the role of adiposity (i.e., body fat) in CVD prediction. There are two major methods in which adiposity is assessed: overall obesity (peripheral adiposity) and central or abdominal obesity (visceral adiposity). The most common measures of overall obesity are weight and body mass index (BMI), which is a weight-to-height ratio where weight in kilograms is divided by the square of height in meters $\left(\text{Weight (kg)}^2 \middle/ \text{Height (m)}\right)$. Waist-to hip ratio (WHR) and waist circumference (WC) are the most common indices of central adiposity.

It is well established that obesity is an independent risk factor for CVD (Hubert, Feinleib, Mcnamara, & Castelli, 1983; Lee & Lip, 2003; Rosamond et al., 2008). Some

data have suggested that obesity is related to CVD through indirect mechanisms such as diabetes mellitus, hypertension, dyslipidemia, and metabolic syndrome (Rao, Donahue, Pi-Sunyer, & Fuster, 2001; Vega, 2001). Other data have shown that overall obesity (as measured by weight) prospectively predicts left ventricular mass and left ventricular hypertrophy, both of which are predictors of cardiovascular mortality (Murdison et al., 1998). Although research focusing on the obesity–CVD relationship is suggestive, it has been argued that overall obesity (as measured by weight or BMI), is a weak measure of cardiovascular risk because it fails to account for body composition and fat distribution. In addition, some researchers argue that the literature on the relationship between obesity and CVD is inconsistent, which makes drawing conclusions about the impact of obesity on CVD difficult (Wingard, 1990). As a result, measures of central adipose tissue distribution—such as waist circumference (WC) and waist-to-hip ratio (WHR)—are beginning to replace BMI to more accurately index cardiovascular risk.

In a brief review, Broom (2006) indicated that using a measure of central adiposity may be better at predicting cardiovascular risk than using a measure of overall obesity. Longitudinal studies have demonstrated that independent of BMI, greater central adiposity, as measured by WHR, is a significant predictor of cardiovascular disease and mortality (Lapidus et al., 1984; Larsson et al., 1984). In one study, Nicklas et al. (2004) investigated the relationship between visceral adiposity and incidence of myocardial infarction. Precise measures of visceral adiposity were taken using a computed tomography scanner. The results showed that at follow-up, women who suffered from myocardial infarction had greater concentrations of visceral adipose tissue at baseline compared to their healthy counterparts. Thus, central adiposity appears to be an important

biological marker for assessing CVD risk. However, while focusing on biological mechanisms that link obesity to CVD is important, it can be argued that using this approach solely and in the absence of psychological and social factors may be incomplete.

Psychological Approach: Stress and CVD

Stress is one psychological factor that may play a role in the adiposity-CVD relationship. Research has indicated that stressors such as employment burdens, and socioeconomic difficulties are highly correlated with CVD (Lee & Lip, 2003). Significant job strain has been shown to predict sub-clinical atherosclerosis in non-symptomatic men (Hintsanen et al., 2005), and acute life stressors such as bereavement, natural disasters, and trauma are associated with increases in cardiac events (Rozanski, Blumenthal, & Kaplan, 1999). However, to date, it is still unknown whether stress itself has a direct effect on the development of CVD, if stress has an influence by acting on other risk factors, or if stress is merely a correlate of other processes involved in etiology.

One theory posits that the magnitude of cardiovascular reactivity (CVR) to a laboratory stressor may be an indication of CVD risk enhancement (Krantz & Manuck, 1984; Manuck & Krantz, 1986). Presumably, the magnitude of CVR in the laboratory is an index of how an individual's cardiovascular system responds to stressful stimuli outside the laboratory. It is thought that over time, this chronic sheer stress on the cardiovascular system can lead to an increased risk for developing CVD. Empirical data have provided support for this theory, which is often referred to as the "reactivity hypothesis". For example, the results of a study by Waldstein et al. (2004) demonstrated that in healthy elderly adults, elevated blood pressure (BP) reactivity in response to a

laboratory stressor was associated with a greater likelihood of having a stroke. This relationship emerged independent of participants' resting BP levels. In addition, it has been shown that stress-induced BP reactivity in response to a mental stressor is positively related to the development of atherosclerosis 7 years later (Jennings et al., 2004). Further, it has been shown that individuals with high BP (i.e., those who were hypertensive) have significantly greater heart rate and diastolic blood pressure (DBP) reactivity to stress than those without hypertension (Rostrup, Westheim, Kjeldsen, & Eide, 1993). It has also been demonstrated that DBP reactivity is related to carotid intima media thickness in hypertensives, which is an indication of CVD progression (Gianaros et al., 2002).

Cardiovascular reactivity shows the ability to predict some preclinical CVD states as well, some of which include augmented left ventricular mass and atherosclerosis (Treiber et al., 2003). Furthermore, Everson et al. (2001) demonstrated that men who exhibit exaggerated systolic blood pressure (SBP) reactivity show a 72% greater risk for stroke occurrence when compared to less reactive men. Even in those with preclinical coronary heart disease, it has been shown that those with exercise-induced occult ischemia are 21 times more likely to be in the top quartile of CVR during mental stress (Kral et al., 1997). Thus, there is much evidence to support the theory that heightened CVR to stress may be a predisposing factor to the development of CVD.

In addition to heightened CVR to stress, prolonged cardiovascular recovery from stress may also be a risk factor for CVD development. Theoretically, prolonged elevations in cardiovascular functioning after stress exposure could be as detrimental as the magnitude of reactivity during stress. In a meta-analytic review, Hocking Schuler and O'Brien (1997) showed that individuals who were considered high-risk for hypertension

demonstrated delayed recovery from stress when compared to those who were low-risk. Additionally, it has been shown that those who demonstrate delayed physiological recovery from stress have higher cartotid intima-media thickness compared to those who return to baseline levels (Steptoe, Donald, O'Donnell, Marmot, & Deanfield, 2006). Thus, it appears that not only is it important to investigate the magnitude of change that occurs in cardiovascular functioning during stress, but also the rate and degree of recovery following stress.

Social Approach: Stigmatization and CVD

Another approach to predicting CVD involves social mechanisms. In particular, social stigma may play an important role in disease development. It has been suggested that, "…being a target of stigma and discrimination is associated with poor health and negative outcomes…" (Puhl & Brownell, 2003, p. 54). There are several potential explanations for the associations among stigma, discrimination, and poor health. One explanation is that stigmatized individuals may have low levels of social support. Small social networks and low levels of social support (Rozanski et al., 1999) as well as disruption in social cohesion (Lett et al., 2005) have been shown to be associated with an increased incidence of CVD.

Another explanation for the relationship between social stigma and overall health may involve the role of self-esteem. Severely stigmatized individuals may have lower self-esteem, particularly if the stigmatizing condition is perceived as internal and controllable (Crocker & Garcia, 2005; Crocker & Major, 1989). Some research has shown a relationship between self-esteem and health-outcomes. For example, Forthofer, Janz, Dodge, and Clark (2001) showed that higher perceived self-esteem is related to

improved functioning in elders with CVD. Thus, in addition to social support, self-esteem may play an important role in diminishing the progression of CVD, especially among stigmatized individuals.

A Biopsychosocial Approach: The Need for Integration

While each of the aforementioned areas within the CVD literature is compelling, much of the research investigating cardiovascular risk factors has looked at these factors independently. Clearly, having different explanations behind CVD development is valuable. However, using a multidisciplinary (i.e., biopsychosocial) approach lends itself to a more comprehensive evaluation of CVD development. Much research has indicated that adiposity, CVR and recovery, and stigma are interrelated, which highlights the degree to which these associations are not mutually exclusive. Thus, a more integrative approach to CVD prediction may be more appropriate.

Adiposity and Stress Reactivity/Recovery. Obese individuals' pattern of exaggerated CVR to stress may increase their CVD risk. Research has indicated that there is a relationship between overall obesity and CVR (Davis, Twamley, Hamilton, & Swan, 1999). In addition, there appears to be a positive relationship between BMI and prolonged CVR recovery (Steptoe & Wardle, 2005). This relationship between obesity and delayed cardiovascular recovery from stress has also been shown in animal studies (e.g., Sedová et al., 2004).

There is also compelling evidence in support of the relationship between central adiposity and heightened CVR to stress and prolonged cardiovascular recovery from stress. Waldstein, Burns, Toth, and Poehlman (1999) demonstrated that individuals with a high WC have greater overall CVR than individuals with a low WC. These individuals

also showed significantly lower HDL cholesterol levels as well as higher fasting glucose levels when compared to those with a smaller WC. In another study by Goldbacher, Matthews, and Salomon (2005), adolescents with a greater WC exhibited greater blood pressure reactivity to laboratory stressors than adolescents with a smaller WC. This pattern of reactivity was independent of BMI. Furthermore, prolonged cardiovascular recovery appears to be longitudinally related to increases in WHR over a three year time period (Chen, Matthews, Salomon, & Ewart, 2002; Steptoe & Wardle, 2005). Thus, there is clear evidence for the positive relationship between adiposity and heightened CVR to and prolonged recovery from stress.

Stigma and Reactivity/Recovery. Research supports a relationship between experiencing elements of stigma (e.g. unfair treatment, need for acceptance by others, and harassment) and heightened cardiovascular stress responses. Additionally, it has been shown that the more adolescents seek peer approval, the more they show heightened CVR during a social stressor (Chen et al., 2002). The authors suggest that, "…striving to be accepted by others heightens the stressfulness of discussing an interpersonal problem situation…" (p. 22). Thus, it is plausible that lack of social approval (e.g., being stigmatized) may be particularly stressful for those who strive to be accepted by others. In another study, Glynn, Christenfeld, and Gerin (2002) found that those who were exposed to emotional stressors (i.e. exposed to stressors that involved harassment and aversive stimulus avoidance) displayed prolonged cardiovascular recovery when compared to those exposed to non-emotional stressors. Further, those who engaged in emotional stressors showed later BP elevations during directed rumination while those who engaged in non-emotional stressors did not show this same pattern of responding.

Taken together, the evidence suggests that experiencing elements of stigma may enhance cardiovascular risk through heightened reactivity and/or prolonged recovery.

Adiposity and Stigma. A large body of literature suggests that obese individuals are heavily stigmatized and are victims of a wide variety of discrimination (Puhl & Brownell, 2001). This stigmatization and associated discrimination may have deleterious psychological impacts. For example, Carr and Friedman (2005) demonstrated that those who were classified as obese II (BMI of 35-39.9) and obese III (BMI of 40+) reported significantly lower levels of self-acceptance than normal weight individuals. This relationship was mediated by the perception that one had been discriminated against due to weight or appearance. Another study looked at the relationship among the Protestant Ethic, feeling overweight, and psychological well-being (Quinn & Crocker, 1999). The authors define the Protestant ethic as "…an ideology that includes the belief that individual hard work leads to success and that lack of success is caused by the moral failings of self-indulgence and lack of self-discipline" (p. 403). Women were primed with either the Protestant ideology or an inclusive ideology. Overweight women in the Protestant ethic condition had lower levels of psychological well-being and felt worse about their appearance than overweight women in the inclusive condition. Other research has shown that not only does weight stigmatization predict poorer psychological functioning and self-esteem, but it also predicts increased body dissatisfaction (Friedman et al., 2005). Body dissatisfaction is a potential byproduct of weight stigmatization and has important psychological implications, especially for those with excess adiposity. Research has shown that among women, higher BMI and WHR are associated with greater body dissatisfaction (Bailey, Goldberg, Swap, Chomitz, & Houser, 1990; Sarwer,

Allison, & Berkowitz, 2004). Further, it has been found that among college females with high scores on the Beck Depression Inventory (BDI), those with high WHRs show greater body dissatisfaction than those with low WHRs (Joiner, Schmidt, & Singh, 1994).

It is possible that the positive association between BMI, WHR, and body dissatisfaction is due to others' perceptions of those with excess adiposity. There are clear societal values that influence how obese individuals are perceived. The health care field is one area in which weight bias and negative attitudes toward obese individuals have been found (Fabricatore, Wadden, & Foster, 2005). Additionally, Furnham, Petrides, and Constantinides (2005) conducted a study in which they had male and female undergraduates rate drawings of female figures on different desirability features. The drawings depicted six different WHRs and three different levels of body weight (underweight, average weight, and overweight). The three levels of body weight represented differential BMI categories. The results revealed that the figure that represented average weight and a low WHR of 0.7 was rated highest for attractiveness, health and flirtatiousness. Further, this same figure was rated second highest for fertility and youthfulness. This study suggests that a female's BMI and WHR may influence others' perceptions of her desirability. Thus, the degree to which a female with excess adiposity experiences weight stigmatization may result in an increase in her body dissatisfaction.

The Current Study. Among the many CVD risk factors currently identified by the American Heart Association, obesity appears to be of particular importance, as it affects approximately 64 million individuals per year (Rosamond et al., 2008). Obese individuals often suffer from deleterious biological, psychological, and social influences, all of which

can have a significant impact on their lives. Thus, those with excess adiposity represent a unique population through which a biopsychosocial approach to CVD prediction can be studied.

It has been established that excess adiposity, stress reactivity/recovery, and stigma are individually related to cardiovascular risk. However, little research has been conducted to elucidate the mechanisms through which these factors enhance risk. In those with excess adiposity, are there interactive effects of adiposity and stress resulting from weight stigmatization to increase cardiovascular risk? If so, what mediates this relationship? The aforementioned research suggests that those with excess adiposity are perceived negatively whereas those with less adiposity are viewed more favorably. Recent research has demonstrated that those who are more concerned with appearance-based rejection (i.e., appearance based rejection sensitivity: Appearance-RS) are more likely to have lower self-esteem and feelings of loneliness and rejection (Park, 2007). Accordingly, it is presumable that individuals with excess adiposity appraise evaluation from others as stressful. This "appearance-related evaluation stress" may be particularly intense for those with both excess adiposity and body image concerns.

The present study will attempt to extend previous research by examining the independent and interactive effects of adiposity and appearance related evaluation stress on CVR and recovery. Five main hypotheses were proposed. First, it was hypothesized that there would be a main effect of adiposity such that greater adiposity (as measured by BMI and WHR) would be associated with greater CVR and prolonged recovery. Second, it was hypothesized that there would be a main effect of appearance-related evaluation stress such that those who experience more evaluation stress (as manipulated by stressor

condition) would show greater CVR and prolonged recovery. Third, weight-related anxiety was predicted to partially mediate the relationship between adiposity and exaggerated CVR and recovery. Fourth, it was predicted that weight-related anxiety would fully mediate the relationship between evaluation stress and greater CVR and recovery. Finally, it was predicted that there would be an interaction between adiposity and evaluation stress (i.e., speech condition) on CVR and recovery. The pattern of the interaction was expected to be such that participants with greater adiposity (i.e., high BMI and WHR) and who experience more intense evaluation stress (i.e., being videotaped) will show greater CVR and prolonged recovery when compared to participants with lower adiposity and less intense evaluation stress.

Method

Participants

Participants were 106 White female undergraduates aged 18 to 52 years (M = 20.13, SD = 4.03) from the University of South Florida (USF). Overall, the sample consisted of mostly freshman who were educated in the U.S. and fell in the healthy range on adiposity measures (see Table 1 for complete demographic data).

Table 1

Sample Demographics

	M (SD)	Min	Max
BMI	24.83 (5.79)	18.14	50.86
WHR	0.81 (0.07)	0.69	1.08
	Frequency (%)		
Class			
Freshman	44 (43.10)		
Sophomore	24 (23.50)		
Junior	13 (12.70)		
Senior	18 (17.60)		
Post-Bachelors	3 (2.90)		
U.S. Citizen?			
Yes	101 (99.00)		
No	1 (1.00)		

K-12 Education in U.S.?	
Yes	101 (99.00)
No	1 (1.00)

Four participants were dropped from analyses due to missing or incomplete data, resulting in a final sample size of 102. Participants were recruited via Sona Systems, USF's online participant pool. Any participant who reported having a diagnosis of heart disease, having an arrhythmia, being pregnant, or taking medication that affects the cardiovascular system was excluded from the study. Participants received course extra credit as compensation for their involvement in the study.

Measures

Body Image and Stigmatization Measures. Due to the nature of the manipulation in this study, several trait measures of physical appearance concerns and experiences with stigmatization were administered. These measures were used to confirm that random assignment resulted in equivalent groups on trait physical appearance concerns as well as experience with stigmatizing situations.

Physical Appearance State and Trait Anxiety Scale (PASTAS). The PASTAS (Reed, Thompson, Brannick, & Sacco, 1991) was administered to assess both stable and variable body weight/shape concerns. Each portion of the PASTAS consists of 16 items, which ask participants to rate the extent to which they feel anxious, tense or nervous about various body characteristics using a 5-point Likert scale that ranges from "not at all" to "exceptionally so". Both the state and trait portions of the PASTAS have been shown to have high reliability coefficients, with Chronbach's α ranging from .82 to .92.

In addition, the weight scale from trait portion of the PASTAS has shown high convergent validity with other body image measures (e.g. $r = .74$ with the Body Dissatisfaction subscale of the Eating Disorders Inventory). An example item from the state portion of the PASTAS is, “Right now, I feel anxious, tense, or nervous about my waist.” The PASTAS is divided into two subscales, the weight-related subscale and the “other” subscale. The weight-related subscale addresses weight-related physical appearance concerns (e.g., waist) whereas the “other” subscale addresses non-weight related physical appearance concerns (e.g., nose). For all major analyses including the PASTAS, only the weight-related subscale was used.

Eating Disorders Inventory – Body Dissatisfaction Subscale (EDI-BD). In addition to the PASTAS, participants completed the Body Dissatisfaction subscale of the Eating Disorders Inventory (EDI-BD; (Garner, Olmstead, & Polivy, 1983). The EDI-BD is a 9-item scale that measures the extent to which an individual believes various body parts associated with excess adiposity are too large. Participants rate their body parts using a 6-point Likert scale that ranges from “always” to “never.” This subscale has shown evidence for good criterion-related and construct validity as well as showing high internal consistency reliability (Chronbach’s $\alpha \geq .90$). A sample item from the EDI-BD is as follows: "I think that my thighs are too large.”

Appearance-based Rejection Sensitivity Scale (ARS Scale). The ARS Scale measures the degree to which individuals have anxious expectations about others rejecting them based on their appearance (Park, 2007). The ARS Scale contains 15 items, which describe different scenarios that an individual might encounter. Using a 6-point Likert scale, participants are to rate the degree to which they feel concerned about being

rejected because of how they look and how likely it is that others would find them less attractive. The ARS Scale has shown strong psychometric characteristics with high internal consistency and test-retest reliability values (Chronbach's $\alpha = .90$, $r = .69$, $p < .001$, respectively). Additionally, the ARS Scale is highly correlated with other appearance-based scales (Appearance Contingency of Self Worth $r = .48$, $p < .001$), which provides evidence of good convergent validity. An example item from the ARS Scale is as follows: "You are trying on clothes at a department store and notice that you are a few pounds heavier than last week. How concerned or anxious would you be that others might be less attracted to you because of your physical appearance?" Participants respond using the 6-point Likert scale ranging from 1: "very unconcerned" to 6: "very concerned." Then, participants respond to the second portion of the item, which states, "I would expect that others would find me less attractive." Again, participants answer using the 6-point Likert scale, which ranges from 1: "very unlikely" to 6: "very likely."

Stigmatizing Situations Questionnaire (SSQ). The SSQ (Annis, Cash, & Hrabosky, 2004; Myers & Rosen, 1999) is a 2-part questionnaire. Part A (SSQ-A) of the questionnaire contains 50 items and assesses the frequency with which individuals have experienced weight-related stigmatizing situations. Part B (SSQ-B) of the questionnaire contains 99 items and assesses an individual's coping responses to the stigmatizing situations endorsed in part A. An example item from the SSQ-A is, "Parents or other relatives telling you how attractive you would be, if you lost weight." An example item from SSQ-B is, "I avoid going out in public because I am afraid people will make comments about my size." Participants provide frequency ratings for both sections of the SSQ using a 10-point Likert scale that ranges from "never" to "daily." The SSQ has

demonstrated strong psychometric characteristics with reliability estimates ranging from α = .94 to .95 and validity analyses showing the ability of the SSQ to discriminate between those with a BMI of 40+ and those with a BMI of < 40. The SSQ-B contains 21 subscales, which encompass a wide variety of coping responses.

Other Measures

Health Questionnaire. Upon arrival to the laboratory, participants completed a brief health questionnaire to assess factors that may influence cardiovascular activity. Participants were asked to record most recent meal-time, smoking behavior, caffeine consumption, any current medications, and whether they have been diagnosed with or are aware of any heart condition. This questionnaire served as an additional screen for exclusion criteria.

Pre- and Post-Task Appraisal Questionnaires. These questionnaires were used to assess participants' subjective appraisal of the speech before and after completion. This questionnaire is modeled after that which was used in a previous study of challenge and threat appraisal (Tomaka, Blascovich, Kelsey, & Leitten, 1993). These questionnaires include 4 items with a 5-point Likert response scale. The four items ask participants to appraise the extent to which the upcoming (or just completed) speech task is demanding, threatening, and stressful as well as how able they are/were to cope with the task (1 = not at all, 5 = very).

Condition Awareness Questionnaire. At the end of the laboratory task, this questionnaire was administered to confirm participants' awareness of the condition to which they were assigned. In this measure, participants were asked to respond to a single-item that stated, "My speech was recorded using…" The response choices were: a) An

audio recorder, which was placed in front of me on the table, and b) A video camera, which was placed in front of me on the table.

Physiological Recording Apparatus.

Systolic (SBP) and diastolic (DBP) blood pressure were measured using an Accutorr Plus non-invasive blood pressure monitor (Datascope Corp., Mahwah, NJ) according to published guidelines (Shapiro et al., 1996). Heart rate was measured via electrocardiogram (EKG) using silver-silver chloride electrodes with a modified lead II configuration. The EKG signal was amplified using a Biopac EKG amplifier (Biopac Instruments Inc., Goleta, CA). The sympathetically-mediated cardiac and vascular responses that contribute to increases in blood pressure and heart rate were measured via impedance cardiography (ICG). ICG was collected using disposable electrode mylar tape. The ICG signal was processed using a Biopac EBI100C Cardiac Impedance monitor according to published guidelines (Sherwood et al., 1990). The EKG and impedance (Z0) signals was digitized, acquired and stored using a PC and the AcqKnowledge data acquisition software provided by Biopac. ICG provides assessment of stroke volume (SV; The amount of blood in milliliters ejected by the heart on an average heart beat), cardiac output (CO; The amount of blood in liters ejected by the heart in a minute in L/min), and pre-ejection period (PEP: The time between ventricular depolarization and ejection of blood from the heart, which provides a measure of cardiac contractile force in milliseconds).

Evaluation Stress – Speech Task

Participants were instructed to develop and present a speech for traffic court about receiving a ticket for failing to stop at a stop sign. Participants were presented with the

scenario that they were driving down the street and suddenly saw a police car in their rear view mirror. When pulled over, the officer told them that they failed to stop at a stop sign about a block before. They were convinced that there was no stop sign. However, when they go back to the incident site with the police officer, they saw that there was a stop sign, but it was obstructed from view by a large tree. After being presented with this scenario, participants were informed that they had two minutes to prepare a three-minute long speech and that the researcher would tell them when to begin speaking. They were told that their speech is to convince the traffic court judge that they should not have to pay the ticket. They were also told that the quality of their speech would be evaluated based on style, clarity, and the degree to which their argument was convincing. Participants were informed that they would be presented with a study card to facilitate their preparation before they were required to speak. This type of speech task has been used successfully in previous studies to elicit reactivity.

Experimental Protocol

The data collection for this study was broken down into two parts. First, as soon as participants enrolled in the study, they completed the trait portion of the PASTAS, the EDI-BD, and the ARS Scale online via the Sona Systems website. Once the online portion was completed, participants were eligible for the second part of the study, which was conducted in the laboratory. Upon arrival to the laboratory, participants completed a comprehensive informed consent. Participants then completed the Health Questionnaire were instructed to remove all jewelry, to turn off their cell phones, and to visit the restroom if necessary before being connected to the physiological equipment.

Once participants were ready, they were connected to the physiological equipment. First, four bands of disposable electrode mylar tape were adhered to participants' necks and torso (two bands around the neck and two around the torso). The neck bands were placed as low on the neck as possible without being placed on the shoulders and were no fewer than 3cm apart. The top torso band was placed approximately 1 inch below the zyphoid process and the bottom torso band was placed at least 3cm from the top band. The research assistant then measured and recorded the distance between the bottom neck and top torso electrode bands. Next, the EKG electrodes were placed on the participant using a modified lead II configuration. An alcohol prep pad was used to clean the upper right chest just below the distal end of the right collarbone, and the lower left of the abdomen just below the ribcage. An adhesive silver-silver chloride electrode was placed on each of the alcohol-prepped areas once they dried. Participants were then escorted into the laboratory chamber and were instructed to sit in a comfortable chair. At this time, the research assistant attached leads to the mylar electrode bands as well as the EKG electrodes. The research assistant then instructed the participant to expose her non-dominant arm for placement of the automatic blood pressure cuff. The blood pressure cuff was then placed at least 1/2 to 1-inch above the elbow with the microphone placed over the brachial artery. The air hose was then attached to an extension hose that is connected to a blood pressure monitor outside the experimental chamber.

Once participants were connected to the physiological equipment, they were instructed to sit comfortably and relax while they watched a travel guide video about

Hawaii. This rest period allows participants to acclimate to the lab and provides the researcher with an assessment of resting cardiovascular levels.

After the baseline period, participants were randomly assigned to one of two speech conditions: an audiotape condition and a videotape condition. Those in the audiotape condition were informed that their speech would be audio recorded for later evaluation. Participants in the videotape condition were informed that their speech would be video recorded for later evaluation. Each participant was instructed to provide a "practice recording" before the speech task began to ensure that the recording equipment was working. The "practice recording" was then played back in front of the participants to demonstrate what their speech would sound or look like (i.e., those who were audiotaped heard how they sounded on tape and those who were videotaped saw how they looked on camera). After the "practice recording" was played back, participants completed the Pre-Task Appraisal Questionnaire. Participants were then handed their study card and instructed to prepare their speech. The video camera and television monitor remained on during the preparatory period. Thus, those in the video condition were able to see themselves displayed on the television throughout the preparatory period. Once the two-minute preparatory period had passed, the research assistant returned to the room with the participant, took the study card, and began recording. For the video condition, the television monitor was turned off so participants could not see themselves during the speech delivery. The research assistant then left the room and told the participant to await instruction for when to begin speaking.

Participants were instructed to begin speaking once the experimenter had prepared the physiological recording program and were instructed to stop speaking after the 3-

minute speech period has elapsed. If the participant stopped speaking at any time during the 3-minute speech period, she was prompted to continue speaking until instructed to stop. After the participant was instructed to stop speaking, the research assistant entered the room and turned off the recording apparatus. The participant was then instructed to sit quietly and wait for the next task. The participants' physiological measures were recorded for the entirety of the ten-minute recovery period.

After the speech recovery period, participants were instructed to complete the final questionnaires, which included the Post-Task Appraisal Questionnaire, the state version of the PASTAS with modified instructions, and the Condition Awareness Questionnaire. Then, participants were disconnected from the psychophysiology equipment and escorted from the experimental chamber. At this time, the research assistant collected the participant's anthropometric measurements including weight, height, waist circumference, and hip circumference.

Data Quantification and Reduction

Three BP recordings were taken at minutes 6, 8, and 10 of the 10-minute baseline rest period. ICG and EKG data were collected continuously during the last 5 minutes of the baseline rest period. BP was collected during both minutes of the preparatory period and EKG and ICG data were collected continuously. During the speech task, BP was collected during minutes 1 and 3 and EKG and ICG data were collected continuously. During recovery, five BP readings were recorded and were collected every-other minute starting at minute 1. That is BP was recorded at minutes 1, 3, 5, 7, and 9 of the recovery period. ICG and EKG data were collected continuously during the recovery period.

The ICG values (i.e., SV, CO, and PEP) were obtained by ensemble-averaging the dZ/dt waveform for each minute of data collected using MindWare IMP 2.56 software (MindWare Technologies, Ltd., Gahanna, OH). The data were screened for artifact by visual inspection of the ensemble-averaged dZ/dt waveforms. MindWare was also used to calculate HR from the EKG signal. The vascular resistance component of blood pressure, total peripheral resistance (TPR) was estimated using the formula TPR = (MAP/CO) * 80 in arbitrary units, where MAP is mean arterial pressure (SBP + (2 * DBP))/3.

To calculate reactivity, SBP and DBP readings were analyzed separately and averaged for each segment of the laboratory procedure. Thus, each participant had a calculated averaged SBP and DBP value for the baseline, speech preparation, and speech task periods. Difference scores between each of the task segments and the baseline period were calculated as well. Thus, each participant will had a composite SBP and DBP change score (i.e., reactivity score) from baseline to each portion of the laboratory task. Reactivity scores for the HR, SV, CO, PEP and TPR were calculated in a similar fashion. Each segment was averaged to create an aggregate score and change scores were computed.

Time-to-recovery was used to index the degree to which a participant returned to baseline levels (i.e., recovered) after the speech task. To calculate time-to-recovery, participants' SBP, DBP, HR and impedance-derived values during recovery were compared to their respective baseline data. Participants were considered "fully recovered" if their values returned to baseline levels during the recovery period. Baseline scores were divided by recovery values on a minute-by-minute basis to get a percentage of overall recovery for each minute. The minute in which the participant reached full

recovery was used as the time-to-recovery value. If a participant did not reach “full recovery” by the end of the 10-minute recovery period, 10 minutes was used as her score.

Results

Random Assignment Check and Preliminary Analyses

All data were analyzed using the SPSS statistical software program. A series of between-groups ANOVAs with speech condition as the between-subjects factor was conducted to evaluate whether random assignment resulted in equivalent groups. No group differences were found on any demographic variables or trait physical appearance concerns (see Table 2).

Table 2

Random Assignment Results

	Audio Mean *(SD)*	Video Mean *(SD)*	*p*
Age	20.38 (5.20)	19.86 (2.23)	.514
BMI	24.84 (5.73)	24.82 (5.91)	.990
WHR	.81 (.08)	.81 (.06)	.896
PASTAS – Trait, Weight Subscale	13.10 (7.24)	12.28 (7.85)	.586
PASTAS – Trait, Other Subscale	2.83 (3.40)	2.74 (2.69)	.887
PASTAS – Trait, Total	15.92 (9.50)	15.02 (9.43)	.631
EDI: Body Dissatisfaction	24.33 (9.74)	23.82 (11.11)	.807
ARS Scale Average	15.17 (6.99)	14.57 (8.07)	.688
SSQ-A Total	.42 (.42)	.51 (.63)	.417

Table 3 provides a correlation matrix for the major study variables.

Table 3

Correlations Among Major Study Variables (N = 102)

	1.	2.	3.	4.	5.	6.	7.
1. BMI	--	.66***	.54***	.43***	.51***	.27***	.39**
2. WHR		--	.44***	.36***	.34***	.22*	.38***
3. State PASTAS: Weight Subscale			--	.36***	.41***	.32**	.36***
4. Trait PASTAS: Weight Subscale				--	.81***	.52***	.54***
5. EDI-BD					--	.54***	.50***
6. ARS						--	.38***
7. SSQ-A							--

Note. BMI = Body Mass Index, WHR = Waist-to-Hip Ratio, PASTAS = Physical Appearance State Trait Anxiety Scale, EDI-BD = Eating Disorders Inventory-Body Dissatisfaction Subscale, ARS = Appearance-based Rejection Sensitivity scale, SSQ-A = Stigmatizing Situations Questionnaire-Part A

*$p < .05$

**$p < .01$

***$p < .001$

A Pearson's correlation analysis was conducted to test the assumption that there would be a positive association between adiposity and state PASTAS. The analysis confirmed this assumption (BMI: $r = .54$, $p < .001$; WHR: $r = .44$, $p < .001$). Additionally, a one-way ANOVA was conducted to examine whether the speech task manipulation had an effect

on state PASTAS scores. The analysis revealed that those in the video condition had higher state PASTAS scores ($M = 9.28$, $SD = 7.74$) during the speech task than those in the audio condition ($M = 3.31$, $SD = 5.61$; $F(1,99) = 19.73$, $p < .001$). This analysis indicates that those in the video condition experienced higher levels of weight-related anxiety during the speech than those in the audio condition.

Effect of Adiposity and Speech on Psychological Variables

State PASTAS as Outcome. A series of hierarchical multiple regression analyses was conducted using adiposity, speech condition, and their interaction as predictors and state PASTAS score as the criterion. Trait PASTAS was entered as a covariate for all analyses to account for dispositional weight-related anxiety. Separate analyses were conducted for BMI and WHR. The covariate was entered into the model on step 1. Adiposity and speech condition were entered into the model on step 2. The interaction between adiposity and speech condition was entered into the model on step 3. When BMI was used as the adiposity predictor, significant main effects of BMI and speech condition were found such that higher BMI and the video condition were associated with higher state PASTAS scores ($B = .59$, $SEB = .10$, $t = 5.73$, $p < .001$; $B = 6.15$, $SEB = 1.07$, $t = 5.74$, $p < .001$, respectively). However, there was no interaction between BMI and speech condition on state PASTAS scores ($p > .05$). When WHR was used as the adiposity predictor, significant main effects of WHR and speech condition were found such that the higher WHR and the video condition were associated with higher state PASTAS scores ($B = 34.71$, $SEB = 8.53$, $t = 4.07$, $p < .001$; $B = 6.07$, $SEB = 1.15$, $t = 5.30$, $p < .001$, respectively). There was no interaction between WHR and speech condition on state PASTAS scores ($p > .05$).

Task Appraisals as Outcomes. A series of hierarchical multiple regression analyses was conducted using adiposity, speech condition, and their interaction as predictors and pre- and post-task appraisals as the criterion variables. Separate analyses were conducted for BMI and WHR. Adiposity and speech condition were entered into the model on step 1. The interaction between adiposity and speech condition were entered into the model on step 2. No significant effects of adiposity, speech condition, or the interaction between adiposity and speech emerged for pre- or post-task appraisals (all *ps* > .05).

Results for Adiposity and Speech Predicting Reactivity and Recovery

A series of hierarchical multiple regression analyses was conducted to evaluate the main effect and interaction hypotheses. BMI and WHR were standardized and combined to create an aggregate adiposity variable. The correlation between the aggregate adiposity variable and BMI and WHR was large ($r = .91$, $p < .001$ for both), suggesting that the aggregate variable was an adequate measure of adiposity. Baseline physiological values were used as a covariate in analyses of reactivity data, because baseline levels can influence reactivity due to the law of initial values (i.e., starting values can influence degree of change if there are upper and lower limits on level). Reactivity scores were used as a covariate in recovery analyses, because degree of change during the task can influence time to recovery (i.e., the greater the change during the task, the more time it should take to recover). Covariates were entered into the model on step 1. The aggregate adiposity variable and speech condition (video, audio, dummy coded as 0 and 1, respectively) were entered into the model on step 2. The interaction between adiposity

and speech was entered as the predictor on step 3. CVR during the preparatory and speech phases as well as time-to-recovery were entered as the criterion variables.

For the preparatory period, significant effects were found for the main effect of adiposity on DBP reactivity (B = -1.27, SEB = .64, t = -1.99, $p < .05$) and SV reactivity (B = -3.03, SEB = 1.10, t = -2.75, $p < .01$). For the speech period, significant effects were found for the interaction between adiposity and speech on DBP reactivity (B = -3.67, SEB = 1.81, t = -2.027, $p < .05$), the main effect of adiposity on CO reactivity (B = .41, SEB = .19, t = 2.16, $p < .05$), the main effect of adiposity on PEP reactivity (B = -4.14, SEB = 1.79, t = -2.32, $p < .05$), and the main effect of adiposity on TPR reactivity (B = -64.35, SEB = 23.08, t = -2.79, $p < .01$). No other significant effects were found.

Rationale and Method for Subsequent Analyses

There was no interaction between adiposity and speech condition on state PASTAS scores, which suggests that the speech condition did not differentially enhance weight-related anxiety among those with high adiposity. There was also no main effect of adiposity or speech condition or their interaction on task appraisals. Additionally, there was no main effect of speech condition on reactivity or recovery for any outcome variables for any of the study phases. Taken together, these results suggest that any effect found on reactivity and recovery in the present study is due to adiposity and the overall stress of delivering a speech, not body image concerns. When the aggregate adiposity variable was used, significant main effects of adiposity and significant interactions between adiposity and speech condition emerged for some of the outcome variables. However, as previously mentioned, research has suggested that central and overall adiposity provide independent information about CVD risk and contribute differently to

reactivity and recovery. Thus, any interpretation based on the results of the analyses using the aggregate adiposity measure may be inappropriate. To be consistent with current literature, all further analyses were conducted using one adiposity variable as a covariate and the other as the predictor to evaluate the independent effect of central and overall adiposity on reactivity and recovery.

Method for Regression Analyses. A series of hierarchical regression analyses was conducted to examine the main effect and interaction hypotheses. When BMI was used as the adiposity predictor, WHR was entered as a covariate. Likewise, when WHR was used as the adiposity predictor, BMI was entered as a covariate. Again, baseline values were used as a covariate in reactivity analyses and reactivity scores were used as a covariate in recovery analyses. Covariates were entered into the model on step 1. Adiposity (BMI and WHR) and speech condition were entered into the model as predictors on step 2. The interaction between adiposity and speech condition were entered into the model as the predictor on step 3.

Method for Mediation Analyses. A series of mediation analyses using the method outlined by Preacher and Hayes (2008) was conducted to investigate both mediation hypotheses. In this method, the analysis tests the overall mediation without relying on the assumptions and restrictions of the causal path method (see Preacher & Hayes, 2008 for more information). Another benefit of this approach is that it allows for testing mediation in the presence of covariates. This method uses confidence intervals to determine the significance of the overall model. All confidence intervals presented are bias-corrected. The mediation analyses included the same covariates as the hierarchical multiple regression analyses. Additionally, trait PASTAS was entered as a covariate to account for

dispositional weight-related anxiety. The weight subscale was used in all analyses using PASTAS scores.

Main Hypotheses

Main Effect of Adiposity (H_1). The first hypothesis was that there would be a main effect of adiposity on CVR and recovery such that higher adiposity would predict greater CVR and longer recovery time. When BMI was used as the adiposity predictor, there was partial support for the first hypothesis. Consistent with the hypothesis, a significant main effect of BMI was found for HR (B = .38, SEB = .19, t = 2.07, p = .04) and PEP (B = -.79, SEB = .36, t = -2.11, p = .04) reactivity during the preparatory period. Likewise, there was a significant main effect of BMI on SV (B = .91, SEB = .35, t = 2.60, p = .01), CO (B = .15, SEB = .04, t = 3.53, p = .001), and PEP (B = -.85, SEB = .42, t = -2.02, p = .05) reactivity during the speech task. Contrary to the first hypothesis, there was a significant main effect of BMI on TPR reactivity during the speech task such that higher BMI predicted greater decreases in TPR during the speech (B = -20.57, SEB = 4.96, t = -4.15, p = .001). Finally, in partial support of the first hypothesis, there was a significant main effect of BMI for SV recovery time such that higher BMI predicted a slower return to baseline SV levels (B = .18, SEB = .08, t = 2.41, p = .02). No other significant main effects were found with BMI as the adiposity predictor.

When WHR was used as the adiposity predictor, the first hypothesis was partially supported. Contrary to the first hypothesis, there was a main effect of WHR on CO reactivity during the preparatory period such that higher WHR predicted less CO reactivity during the preparatory period (B = -6.61, SEB = 2.22, t = -2.98, p = .004). Consistent with the hypothesis, was a significant main effect of WHR on TPR reactivity

during the preparatory period such that higher WHR predicted greater increases in TPR (B = 945.96, SEB = 280.35, t = 3.37, p = .001). Contrary to the first hypothesis, there was a main effect of WHR on SV recovery time such that higher WHR predicted faster return to baseline SV levels (B = -14.81, SEB = 5.51, t = -2.69, p = .009). No other significant main effects for WHR were found.

Main Effect of Speech Condition (H_2). The second hypothesis was that there would be a significant main effect of speech condition such that those in the video condition would demonstrate greater CVR and prolonged recovery. Contrary to the second hypothesis, no significant main effects for speech condition were found for prep reactivity, speech reactivity, or recovery time (all ps > .05).

State PASTAS as a Mediator Between Adiposity and CVR/Recovery (H_3). The third hypothesis was that the effect of adiposity on CVR and recovery would be partially mediated by state PASTAS scores. When BMI was used as the adiposity predictor, a significant mediation emerged for CO reactivity during the speech task (lower 95% CI = -.08, upper 95% CI = -.002, bias corrected). The IV-to-mediator path was significant such that higher BMI was associated with higher state PASTAS scores (t = 2.67, p = .009). The mediator-to-DV path was significant such that higher state PASTAS scores were associated with less CO reactivity (t = -2.45, p = .02). Overall, the effect of BMI on CO reactivity was strengthened when state PASTAS scores were accounted for (total effect: t = 3.15, p = .002; direct effect: t = 3.77, p < .001). The pattern of this mediation suggests that higher state PASTAS served to suppress the effect of BMI on CO reactivity. Weight-related body image concerns did not mediate the relationship between BMI and any other outcome variable during any other phase.

When WHR was used as the adiposity predictor, state PASTAS did not mediate the relationship between WHR and CVR or recovery for any of the experimental phases.

State PASTAS as a Mediator Between Speech Condition and CVR/Recovery (H_4). The fourth hypothesis was that the main effect of stressor condition on CVR and recovery time would be fully mediated by state PASTAS scores. Two significant mediations emerged for the effect of speech condition on CVR. A significant mediation emerged for SV reactivity during the preparatory phase (lower 95% CI = -4.31, upper 95% CI = -.24, bias corrected). The IV-to-mediator path was significant such that the video condition was associated with higher state PASTAS scores than the audio condition ($t = 4.68$, $p < .001$). The mediator-to-DV path was marginally significant such that higher state PASTAS scores were associated with less SV reactivity during the preparatory phase ($t = -1.91$, $p = .06$). Overall, the effect of speech condition on SV reactivity during the preparatory phase was such that the video condition was associated with less SV reactivity than the audio condition; however, this effect was not significant (total effect: $t = -1.12$, $p = .27$). Although the effect of speech condition on SV reactivity was not significant, the strength of the effect of speech condition on SV reactivity was reduced when state PASTAS scores were accounted for (direct effect: $t = -.19$, $p = .85$).

The second significant mediation emerged for CO reactivity during the preparatory phase (lower 95% CI = -.47, upper 95% CI = -.02, bias corrected). The IV-to-mediator path was significant such that the video condition was associated with higher state PASTAS scores than the audio condition ($t = 4.67$, $p < .001$). The mediator-to-DV path was marginally significant such that higher state PASTAS scores were associated with less CO reactivity ($t = -1.87$, $p = .07$). Overall, the effect of speech condition on CO

reactivity was not significant. However, the direction of the effect of speech condition on CO reactivity changed when state PASTAS scores were accounted for. That is, the initial pattern was such that the video condition was associated with less CO reactivity than the audio condition (total effect: t = -.51, p = .61). Once state PASTAS scores were accounted for, the pattern changed to reflect higher CO reactivity for the video condition than the audio condition (t = .34, p = .73). Similar to the mediation for SV reactivity, the strength of the effect of speech condition on CO reactivity was reduced when state PASTAS scores were accounted for.

Weight-related body image concerns did not mediate the relationship between speech condition and any other outcome variable during any other phase.

Interaction Between Adiposity and Speech Condition (H_5). The fifth hypothesis was that there would be an interaction between adiposity and speech condition such that those with higher adiposity and who were randomly assigned to the video condition would demonstrate greater CVR and prolonged recovery compared to those with less adiposity and who were randomly assigned to the audio condition. When BMI was used as the adiposity predictor, one significant interaction emerged. There was a significant interaction between BMI and speech condition for TPR reactivity during the preparatory period (B = 11.91, SEB = 5.82, t = 2.05, p = .04). Contrary to the hypothesis, the pattern of the interaction was such that higher BMI predicted greater decreases in TPR and this relationship was more pronounced in the audio condition than the video condition. Figure 1 provides a graphical depiction of this interaction.

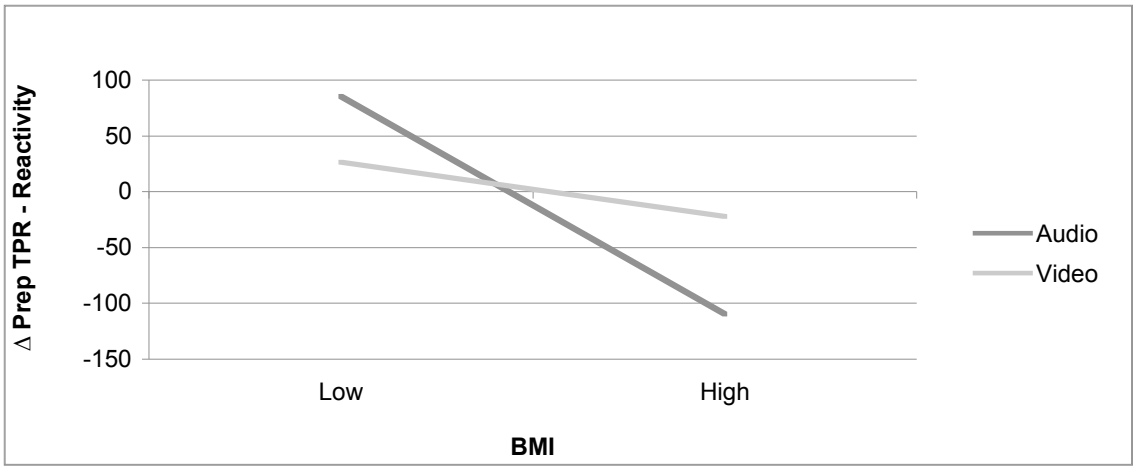

Figure 1. Interaction Between BMI and Speech Condition on TPR Reactivity During the Preparatory Period.

Table 4 summarizes the complete results for each step in the hierarchical multiple regression conducted for this analysis.

Table 4

Summary of Hierarchical Regression Analysis for BMI and Speech Condition Predicting TPR Reactivity During the Preparatory Period (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Baseline TPR	-.02	.04	-.06
WHR	331.50	210.26	.16
Step 2: Main Effects			
BMI	-12.18	3.86	-.43**
Speech Condition	6.01	28.85	.02
Step 3: Interaction			
BMI x Speech Interaction	11.91	5.82	1.00*

Note. $R^2 = .03$, $p > .05$ for Step 1; $\Delta R^2 = .10$, $p < .01$ for Step 2, $\Delta R^2 = .04$ $p < .05$ for Step 3

*p < .05

**p < .01.

No other significant interactions between BMI and speech condition were found for reactivity during the preparatory period, reactivity during the speech, or recovery time.

When WHR was used as the main adiposity measure, three significant interactions emerged. First, there was a significant interaction between WHR and speech condition for DBP reactivity during the speech task ($B = -57.34$, $SEB = 23.34$, $t = -2.46$, $p = .02$). Contrary to the hypothesis, the pattern of the interaction was such that there was no effect of WHR on reactivity in the audio condition and higher WHR predicted less DBP reactivity in the speech condition. Figure 2 provides a graphical depiction of this interaction.

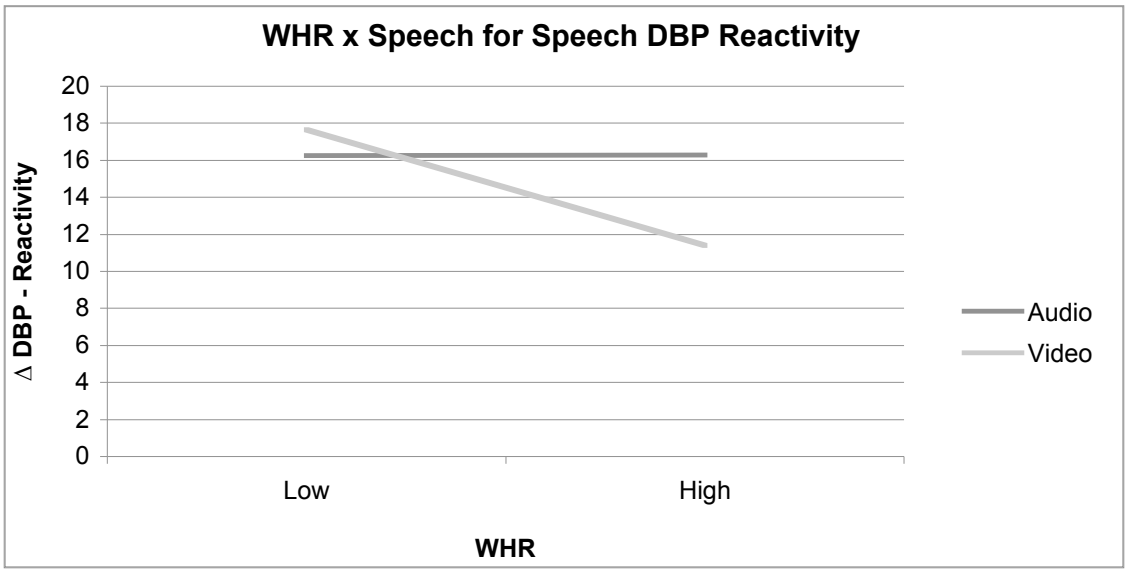

Figure 2. Interaction Between WHR and Speech Condition on DBP Reactivity During the Speech Task.

Table 5 summarizes the complete results for each step in the hierarchical multiple regression conducted for this analysis.

Table 5

Summary of Hierarchical Regression Analysis for WHR and Speech Condition Predicting DBP Reactivity During the Speech Task (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Baseline DBP	-.41	.14	-.28**
BMI	.14	.14	.10
Step 2: Main Effects			
WHR	-15.48	15.04	-.13
Speech Condition	-1.39	1.62	-.08

Step 3: Interaction			
WHR x Speech Interaction	-57.34	23.34	-2.82*

Note. $R^2 = .09$, $p < .05$ for Step 1; $\Delta R^2 = .02$, $p > .05$ for Step 2, $\Delta R^2 = .06$, $p < .05$ for Step 3

*p < .05

**p < .01.

Second, there was a significant interaction between WHR and speech condition for PEP recovery time (B = -19.19, SEB = 7.93, t = -2.42, p = .02). Again, the pattern of the interaction was not as hypothesized. For those in the audio condition, higher WHR predicted longer PEP recovery time. However, for those in the video condition, higher WHR predicted shorter recovery time. Figure 3 provides a graphical depiction of this interaction.

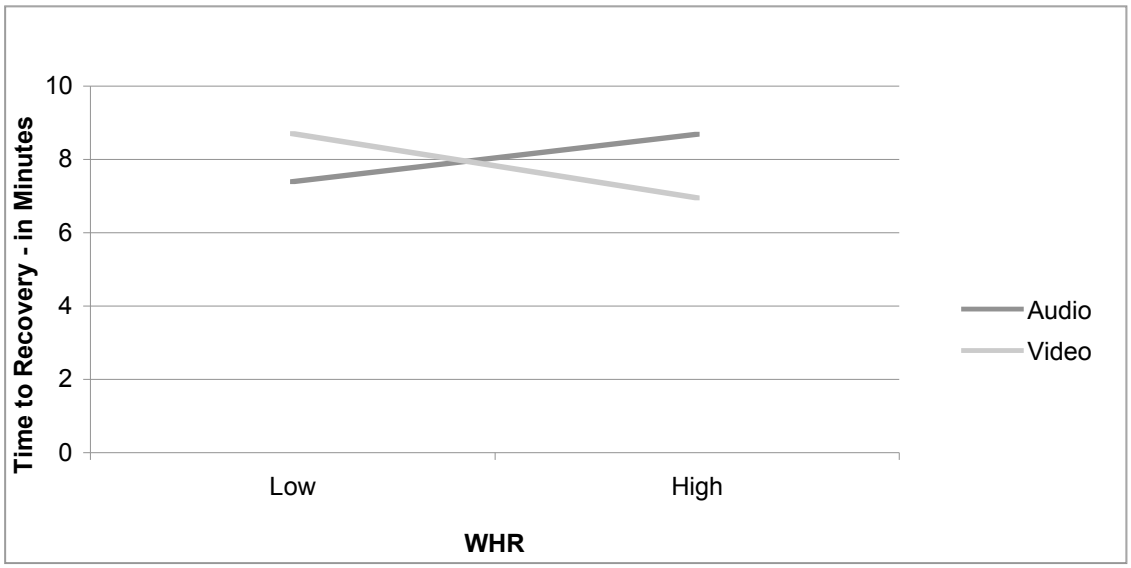

Figure 3. Interaction Between WHR and Speech Condition on PEP Recovery Time.

Table 6 summarizes the complete results for each step in the hierarchical multiple regression conducted for this analysis.

Table 6

Summary of Hierarchical Regression Analysis for WHR and Speech Condition Predicting PEP Recovery Time (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Reactivity PEP Value	-.09	.02	-.42***
BMI	-.02	.05	-.03
Step 2: Main Effects			
WHR	.61	5.26	.02
Speech Condition	-.26	.54	-.05
Step 3: Interaction			
WHR x Speech Interaction	-19.19	7.93	-2.71*

Note. $R^2 = .17$, $p < .001$ for Step 1; $\Delta R^2 = .002$, $p > .05$ for Step 2, $\Delta R^2 = .05$, $p < .05$ for Step 3

*p < .05

***p < .001.

Finally, there was a significant interaction between WHR and speech condition for TPR recovery time ($B = 19.02$, $SEB = 8.77$, $t = 2.17$, $p = .03$). The pattern of this interaction was partially consistent with the hypothesis. Overall, the pattern of the interaction was such that higher WHR predicted longer recovery time, but this relationship was only seen in the video condition. Figure 4 provides a graphical depiction of this interaction.

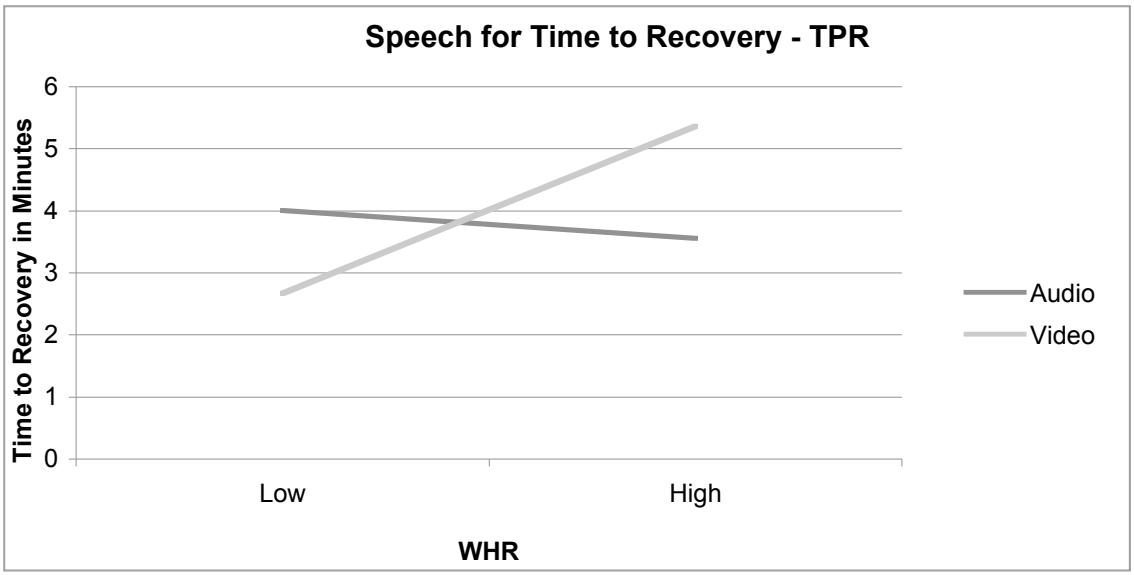

Figure 4. Interaction Between WHR and Speech Condition on TPR Recovery Time.

Table 7 summarizes the complete results for each step in the hierarchical multiple regression conducted for this analysis.

Table 7

Summary of Hierarchical Regression Analysis for WHR and Speech Condition Predicting TPR Recovery Time (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Reactivity TPR Value	-.01	.002	.28**
BMI	-.10	.06	-.14
Step 2: Main Effects			
WHR	5.91	6.04	.14
Speech Condition	.08	.59	.01
Step 3: Interaction			

WHR x Speech Interaction	19.02	8.77	2.55*

Note. $R^2 = .11$, $p < .01$ for Step 1; $\Delta R^2 = .009$, $p > .05$ for Step 2, $\Delta R^2 = .04$, $p < .05$ for Step 3

*p < .05

**p < .01.

Post-Hoc Analyses

A series of hierarchical regression analyses was conducted to examine whether there was an interaction between adiposity and task demand appraisals on reactivity and recovery. Participants' pre and post-task demand appraisal scores were classified as high or low using quartiles. Change in task demand appraisal category from pre-to-post task was used to place participants in one of 3 categories. Those whose category changed from low to high were placed in the "increase" category. Those whose category remained the same were placed in the "same" category. Those whose category changed from high to low were placed in the "decrease" category. As in the above analyses, baseline values were used as a covariate in reactivity analyses and reactivity scores were used as a covariate in recovery analyses. Covariates were entered into the model on step 1. Adiposity and demand appraisal category were entered into the model as predictors on step 2. The interaction between adiposity and demand appraisal category were entered into the model as the predictor on step 3.

There was an interaction between adiposity and demand appraisal category for HR reactivity during the preparatory period ($B = -4.58$, $SEB = 1.73$, $t = -2.65$, $p = .009$), PEP reactivity during the preparatory period ($B = 7.83$, $SEB = 2.86$, $t = 2.74$, $p = .007$), and PEP reactivity during the speech task ($B = 8.12$, $SEB = 3.22$, $t = 2.52$, $p = .01$). The pattern of these interactions was such that higher adiposity predicted more reactivity in

the “increase” category compared to the other categories. Figures 5, 6, and 7 provide graphical depictions of these interactions.

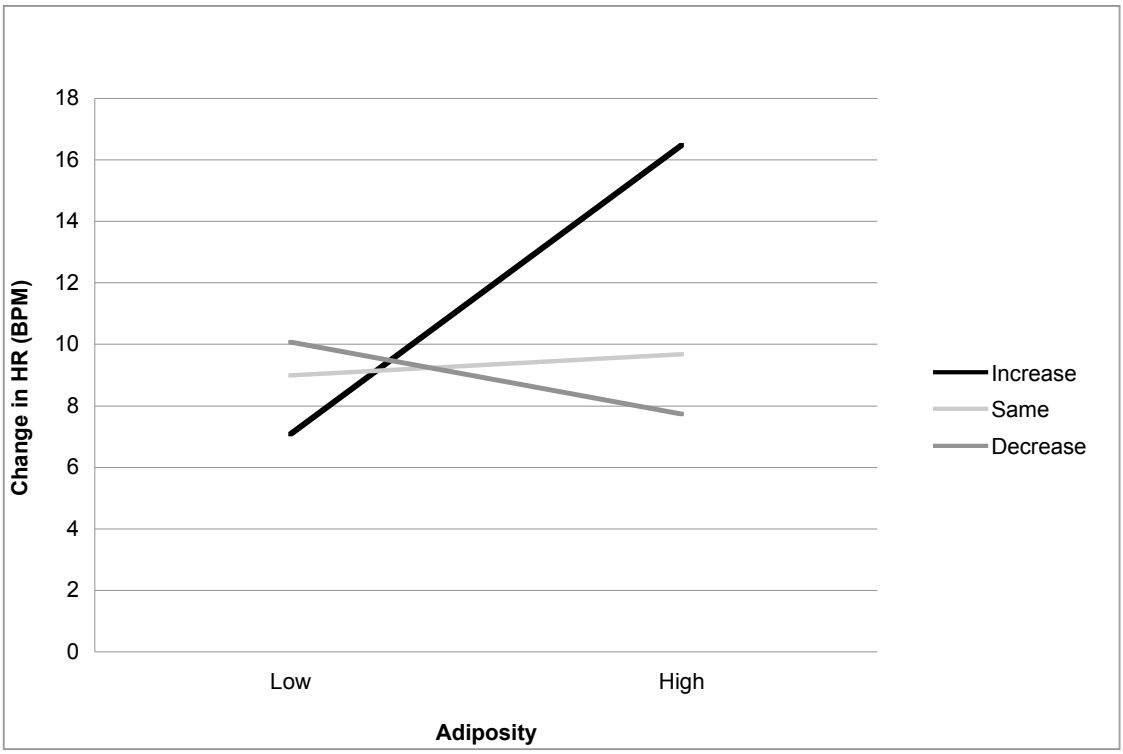

Figure 5. Interaction between Adiposity and Demand Appraisal Category on HR Reactivity During the Preparatory Period.

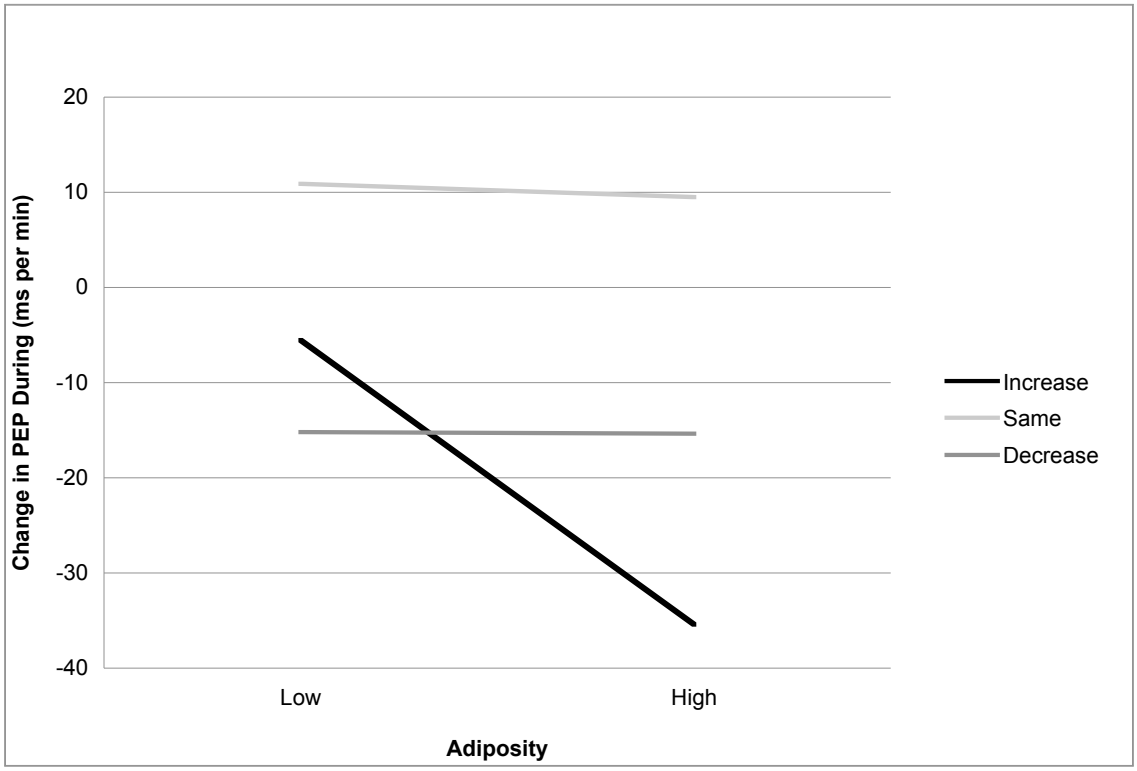

Figure 6. Interaction between Adiposity and Demand Appraisal Category on PEP Reactivity During the Preparatory Period.

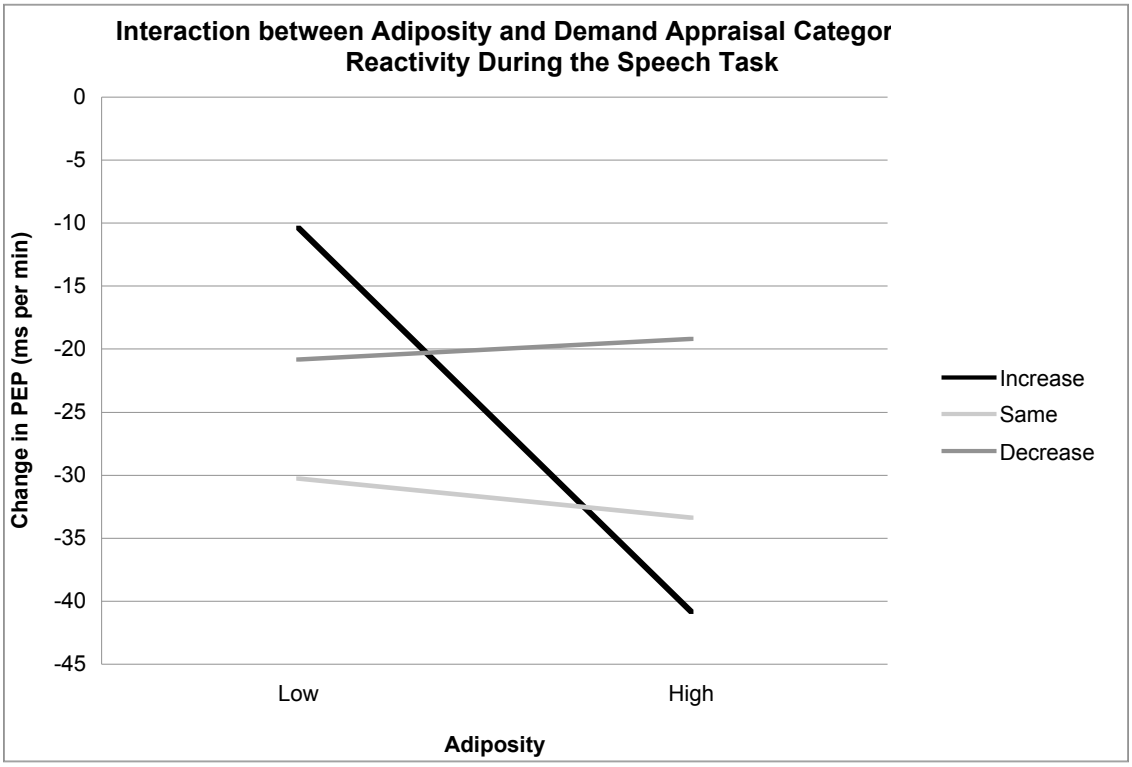

Figure 7. Interaction between Adiposity and Demand Appraisal Category on PEP Reactivity During the Speech Task.

Tables 8, 9, and 10 summarize the complete results for each step in the hierarchical multiple regressions conducted for these analyses.

Table 8

Summary of Hierarchical Regression Analysis for Adiposity and Task Demand Appraisal Category Predicting HR Reactivity During the Preparatory Period (N = 102)

Variable	B	SEB	β
Step 1: Covariates			
Baseline HR	-.02	.08	-.03
Step 2: Main Effects			
Adiposity	.99	.91	.11

Change Category	-1.33	1.51	-.09
Step 3: Interaction			
Adip x Change Interaction	-4.58	1.73	-.26**

Note. $R^2 = .001$, $p > .05$ for Step 1; $\Delta R^2 = .02$, $p > .05$ for Step 2, $\Delta R^2 = .07$, $p < .01$ for Step 3

**p < .01.

Table 9

Summary of Hierarchical Regression Analysis for Adiposity and Task Demand Appraisal Category Predicting PEP Reactivity During the Preparatory Period (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Baseline PEP	.004	.09	.01
Step 2: Main Effects			
Adiposity	-2.73	1.59	-.172
Change Category	2.46	2.47	.10
Step 3: Interaction			
Adip x Change Interaction	7.83	2.86	.27**

Note. $R^2 < .001$, $p > .05$ for Step 1; $\Delta R^2 = .04$, $p > .05$ for Step 2, $\Delta R^2 = .07$, $p < .01$ for Step 3

**p < .01.

Table 10

Summary of Hierarchical Regression Analysis for Adiposity and Task Demand Appraisal Category Predicting PEP Reactivity During the Speech Task (N = 99)

Variable	B	SEB	β
Step 1: Covariates			
Baseline PEP	.05	.11	.05
Step 2: Main Effects			
Adiposity	-4.09	1.78	-.23
Change Category	2.72	2.76	.10
Step 3: Interaction			
Adip x Change Interaction	8.12	3.22	.24*

Note. $R^2 = .002$, $p > .05$ for Step 1; $\Delta R^2 = .06$, $p < .05$ for Step 2, $\Delta R^2 = .06$, $p < .05$ for Step 3

*p < .05.

Additionally, there was a main effect of demand appraisal category for PEP recovery time such that those in the "increase" category recovered faster than those in the other two categories ($B = 1.08$, $SEB = .47$, $t = 2.30$, $p = .02$). Figure 8 provides a graphical depiction of this main effect.

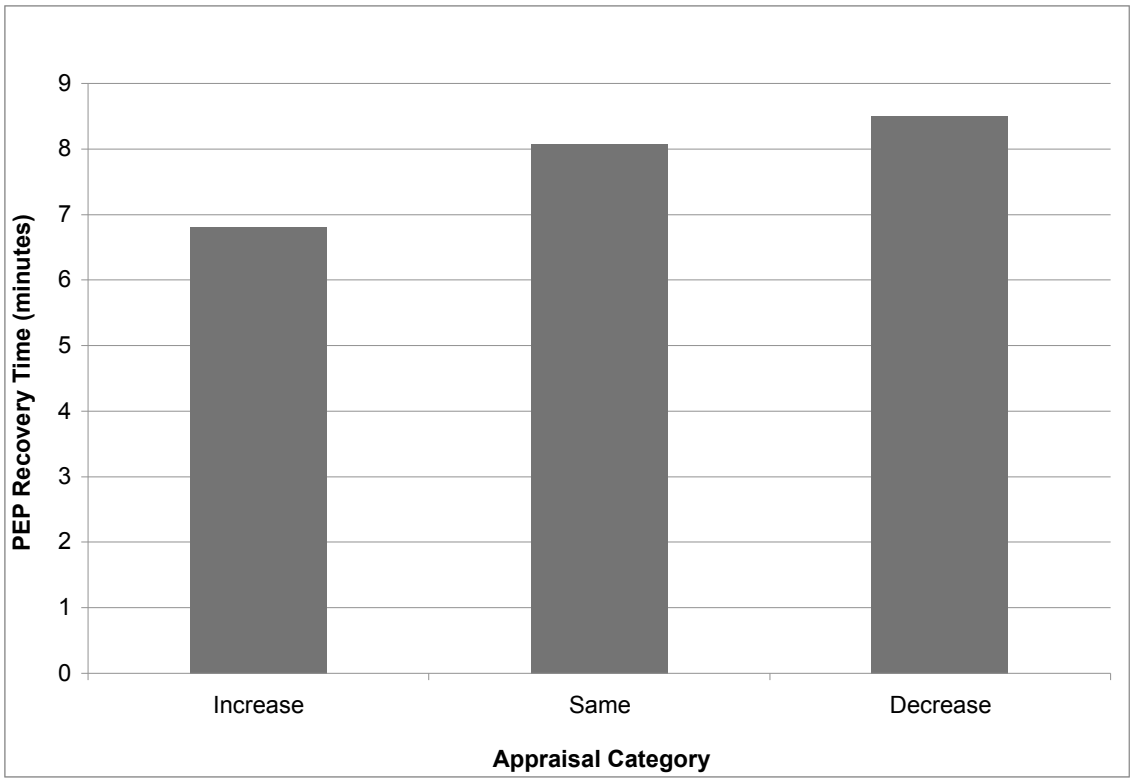

Figure 8. Differences in PEP Recovery Time by Appraisal Category.

Discussion

Results Summary

Overall, the results from the present study were mixed. There was a main effect of adiposity on CVR and recovery with BMI demonstrating better predictive utility than WHR. Additionally, BMI appeared to predict a cardiac reactivity pattern whereas WHR appeared to predict a vascular reactivity pattern. Weight-related anxiety during the speech mediated the relationship between BMI and CO reactivity during the speech task. Additionally, weight-related anxiety mediated the relationship between speech condition and SV and CO reactivity during the preparatory phase. The overall pattern of these mediations was such that higher state PASTAS scores acted to suppress the effect of adiposity and speech condition on reactivity. Finally, significant interactions emerged for several of the outcome variables. In general, the patterns of the interactions were such that higher adiposity predicted less reactivity and shorter recovery in the video condition. This pattern of interaction was contrary to what was expected.

General Discussion

In general, the results of this study suggest that appearance-related concerns and appearance-related evaluation stress may play a role in the adiposity-CVR/recovery relationship. Interestingly, it appears that higher weight-related anxiety may act to suppress the effect of adiposity and appearance-related evaluation stress on CVR/recovery. One potential explanation for this finding may be the role of task-difficulty and effort in the reactivity response. Some research has indicated that the

objective difficulty of the stress task as well as participants' effort can influence reactivity such that tasks with greater difficulty and that require more effort on behalf of the participant can increase reactivity (Richter, 2008; Waldstein, 1997; Wright & Dismukes, 1995). The present study found no main effect of speech condition on reactivity. Additionally, there was no effect of speech condition on appraisals of task demand, threat, or stress. This suggests that the video and audio speech conditions were perceived as equally demanding, threatening, and stressful. Thus, it is possible that those in the audio and video conditions did not differ in reactivity because the conditions were matched on difficulty. That is, perhaps developing and presenting a speech is such a potent stressor that the recording apparatus did not influence the degree of reactivity. Interestingly, the post-hoc analyses revealed that adiposity and task demand appraisals interact to influence degree of reactivity. Specifically, higher adiposity predicted greater reactivity among those whose pre-task appraisals were an underestimation of how demanding the task would be. This suggests that among those with high adiposity, those who underestimate the difficulty of a stressor may be at greater risk. Further, Richter et al. (2008) demonstrated that once the stress task reaches the threshold of impossibility, reactivity significantly decreases. The mediation analyses demonstrated that higher weight-related anxiety served to suppress the effect of BMI and speech condition on reactivity. Thus, perhaps those with high levels of weight-related anxiety during the speech demonstrated less reactivity because the task reached a difficulty level threshold, which mirrored that of impossibility.

Another potential interpretation of the results from this study is that adiposity has a robust effect on physiological reactivity independent of psychological processes that

co-occur. There were several main effects of adiposity on physiological responding that remained once weight-related anxiety during the speech was accounted for. Of particular interest was the finding that BMI and WHR predicted different reactivity patterns. While this finding was unexpected, it is not unique to this study. Other studies have demonstrated that overall adiposity is associated with cardiac reactivity whereas central adiposity is associated with vascular reactivity (e.g., Davis et al., 1999; Jern, Bergbrant, Björntorp, & Hansson, 1992). The authors suggest that these reactivity patterns may underlie important disease processes and these disease processes vary as a function of body fat distribution. It can be argued that while the psychosocial factors involved in the relationship between adiposity and well-being are important, they may be secondary to the direct impact adiposity exerts on physiological functioning. Such an interpretation highlights the importance of adiposity reduction over modifying psychosocial factors.

This study has several limitations that warrant addressing. First, the sample used consisted of all-white female undergraduate students. Thus, the results from this study have limited generalizability. Additionally, no direct measure of task difficulty was collected so any interpretations regarding task difficulty are speculative. Another limitation is that very few mediation analyses emerged as significant. It is possible that those that did emerge as significant reflect increased Type I error probability. Thus, any significant results from the mediation analyses should be interpreted with caution. Finally, the speech task may have been too potent to uncover subtle physiological changes that may occur when a woman is concerned about others' perceptions of her physical appearance. It is possible that a woman with body image concerns may be at heightened physiological arousal compared to her less-concerned counterpart, especially

when interacting socially with others. However, the speech task used in the present study may have been so demanding that it produced a greater physiological response than would be expected in a more realistic encounter. Conversely, there was a significant relationship between adiposity and weight-related anxiety during the speech task independent of speech condition. This suggests that even those in the audio condition experienced increases in weight-related concerns during the speech task. Thus, future studies should focus on different tasks and more subtle manipulations of body image concerns to further elucidate the psychological processes that underlie the relationship between adiposity and physiological responding.

Despite its limitations, the present study does provide useful insight into the role of psychosocial processes in the adiposity-CVR/recovery relationship. It is possible that when the stressor reaches a certain level of intensity, adiposity plays a more important role in the immediate physiological responses to the stress than the concomitant psychological processes. Additionally, it is possible that weight-related anxiety may increase during evaluation regardless of the intention of the evaluation (i.e., weight-related anxiety increased among those with higher adiposity in the audio condition as well). Thus, among persons under intense acute stress, those with greater adiposity may be at greater risk for poor outcomes than those with less adiposity. Future research should take these considerations into account when investigating the adiposity-stress-health outcome relationship.

References

Annis, N. M., Cash, T. F., & Hrabosky, J. I. (2004). Body image and psychosocial differences among stable average weight, currently overweight, and formerly overweight women: The role of stigmatizing experiences. *Body Image, 1*, 155-167.

Bailey, S. M., Goldberg, J. P., Swap, W. C., Chomitz, V. R., & Houser, R. F. (1990). Relationships between body dissatisfaction and physical measurements. *International Journal of Eating Disorders, 9*(4), 457-461.

Broom, I. (2006). Thinking around abdominal obesity and cardiovascular risk. *British Journal of Diabetes and Vascular Disease, 6*, 58-61.

Carr, D., & Friedman, M. A. (2005). Is obesity stigmatizing? Body weight, perceived discrimination, and psychological well-being in the United States. *Journal of Health and Social Behavior, 46*, 244-259.

Chen, E., Matthews, K. A., Salomon, K., & Ewart, C. K. (2002). Cardiovascular reactivity during social and nonsocial stressors: Do children's personal goals and expressive skills matter? *Health Psychology, 21*(1), 16-24.

Crocker, J., & Garcia, J. A. (2005). Self-esteem and the stigma of obesity. In K. D. Brownell, R. Puhl, M. B. Schwartz & L. Rudd (Eds.), *Weight bias: Nature, consequences and remedies* (pp. 165-174). New York: The Guilford Press.

Crocker, J., & Major, B. (1989). Social stigma and self-esteem: The self-protective properties of stigma. *Psychological Review, 96*(4), 608-630.

Davis, M. C., Twamley, E. W., Hamilton, N. A., & Swan, P. D. (1999). Body fat distribution and hemodynamic stress responses in premenopausal obese women: A preliminary study. *Health Psychology, 18*(6), 625-633.

Everson, S. A., Lynch, J. W., Kaplan, G. A., Lakka, T. A., Sivenius, J., & Salonen, J. T. (2001). Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke, 32*, 1263-1270.

Fabricatore, A. N., Wadden, T. A., & Foster, G. D. (2005). Bias in health care settings. In K. D. Brownell, R. M. Puhl, M. B. Schwartz & L. Rudd (Eds.), *Weight bias: Nature, consequences and remedies* (pp. 165-174). New York: The Guilford Press.

Forthofer, M. S., Janz, N. K., Dodge, J. A., & Clark, N. M. (2001). Gender differences in the associations of self esteem, stress and social support with functional health status among older adults with heart disease. *Journal of Women and Aging, 13*(1), 19-38.

Friedman, K. E., Reichmann, S. K., Costanzo, P. R., Zelli, A., Ashmore, J. A., & Musante, G. J. (2005). Weight stigmatization and ideological beliefs: Relation to psychological functioning in obese adults. *Obesity Research, 13*(5), 907-916.

Furnham, A., Petrides, K. V., & Constantinides, A. (2005). The effects of body mass index and waist-to-hip ratio on ratings of female attractiveness, fecundity, and health. *Personality and Individual Differences, 38*(8), 1823-1834.

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15-34.

Gianaros, P. J., Bleil, M. E., Muldoon, M. F., Jennings, J. R., Sutton-Tyrrell, K., Mccaffery, J. M., et al. (2002). Is cardiovascular reactivity associated with atherosclerosis among hypertensives? *Hypertension, 40*(5), 742-747.

Glynn, L. M., Christenfeld, N., & Gerin, W. (2002). The role of rumination in recovery from reactivity: Cardiovascular consequences of emotional states. *Psychosomatic Medicine, 64*(5), 714-726.

Goldbacher, E. M., Matthews, K. A., & Salomon, K. (2005). Central adiposity is associated with cardiovascular reactivity to stress in adolescents. *Health Psychology, 24*(4), 375-384.

Hintsanen, M., Kivimaki, M., Elovainio, M., Pulkki-Raback, L., Keskivaara, P., Juonala, M., et al. (2005). Job strain and early atherosclerosis: The cardiovascular risk in young Finns study. *Psychosomatic Medicine, 67*(5), 740-747.

Hocking Schuler, J. L., & O'Brien, W. H. (1997). Cardiovascular recovery from stress and hypertension risk factors: A meta-analytic review. *Psychophysiology, 34*, 649-659.

Hubert, H. B., Feinleib, M., Mcnamara, P. M., & Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham heart study. *Circulation, 67*(5), 968-977.

Jennings, J. R., Kamarck, T. W., Everson-Rose, S. A., Kaplan, G. A., Manuck, S. B., & Salonen, J. T. (2004). Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation, 110*(15), 2198-2203.

Jern, S., Bergbrant, A., Björntorp, P., & Hansson, L. (1992). Relation of central hemodynamics to obesity and body fat distribution. *Hypertension, 19*(6), 520-527.

Joiner, T. E., Schmidt, N. B., & Singh, D. (1994). Waist-to-hip ratio and body dissatisfaction among college women and men: Moderating role of depressed symptoms and gender. *International Journal of Eating Disorders, 16*(2), 199-203.

Kral, B. G., Becker, L. C., Blumenthal, R. S., Aversano, T., Fleisher, L. A., Yook, R. M., et al. (1997). Exaggerated reactivity to mental stress is associated with exercise-induced myocardial ischemia in an asymptomatic high-risk population. *Circulation, 96*, 4246-4253.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin, 96*(3), 435-464.

Lapidus, L., Bengtsson, C., Larsson, B., Pennert, K., Rybo, E., & Sjöström, L. (1984). Distribution of adipose tissue and risk of cardiovascular disease and death: A 12 year follow up of participants in the population study of women in Gothenburg, Sweden. *British Medical Journal, 289*, 1257-1261.

Larsson, B., Svärdsudd, K., Welin, L., Wilhelmsen, L., Björntorp, P., & Tibblin, G. (1984). Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *British Medical Journal, 288*, 1401-1404.

Lee, K. W., & Lip, G. Y. H. (2003). Effects of lifestyle on hemostasis, fibrinolysis, and platelet reactivity. *Archives of Internal Medicine, 163*, 2368-2392.

Lett, H. S., Blumenthal, J. A., Babyak, M. A., Strauman, T. J., Robins, C., & Sherwood, A. (2005). Social support and coronary heart disease: Epidemiologic evidence and implications for treatment. *Psychosomatic Medicine, 67*(6), 869-878.

Manuck, S. B., & Krantz, D. S. (1986). Psychophysiologic reactivity in coronary heart disease and essential hypertension. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck & R. B. Williams (Eds.), *Handbook of stress, reactivity, and cardiovascular disease* (pp. 11-34). New York: John Wiley & Sons.

Murdison, K. A., Treiber, F. A., Mensah, G., Davis, H., Thompson, W., & Strong, W. B. (1998). Prediction of left ventricular mass in youth with family histories of essential hypertension. *The American Journal of the Medical Sciences, 315*(2), 118-123.

Myers, A., & Rosen, J. C. (1999). Obesity stigmatization and coping: Relation to mental health symptoms, body image, and self-esteem. *International Journal of Obesity, 23*, 221-230.

Nicklas, B. J., Penninx, B. W. J. H., Cesari, M., Kritchevsky, S. B., Newman, A. B., Kanaya, A. M., et al. (2004). Association of visceral adipose tissue with incident myocardial infarction in older men and women. *American Journal of Epidemiology, 160*(8), 741-749.

Park, L. E. (2007). Appearance-based rejection sensitivity: Implications for mental and physical health, affect, and motivation. *Personality and Social Psychology Bulletin, 33*(4), 490-504.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879-891.

Puhl, R., & Brownell, K. D. (2001). Bias, discrimination, and obesity. *Obesity Research, 9*(12), 788-805.

Puhl, R., & Brownell, K. D. (2003). Ways of coping with obesity stigma: Review and conceptual analysis. *Eating Behaviors, 4*(1), 53-78.

Quinn, D. M., & Crocker, J. (1999). When ideology hurts: Effects of belief in the protestant ethic and feeling overweight on the psychological well-being of women. *Journal of Personality and Social Psychology, 77*, 402-414.

Rao, S. V., Donahue, M., Pi-Sunyer, F. X., & Fuster, V. (2001). Obesity as a risk factor in coronary artery disease. *American Heart Journal, 142*(6), 1102-1107.

Reed, D., Thompson, J. K., Brannick, M. T., & Sacco, W. P. (1991). Development and validation of the physical appearance state trait anxiety scale (pastas). *Journal of Anxiety Disorders, 5*, 323-332.

Richter, M., Friedrich, A., & Gendolla, G. H. E. (2008). Task difficulty effects on cardiac activity. *Psychophysiology, 45*, 869-875.

Rosamond, W., Flegal, K., Furie, K., Go, A., Greenlund, K., Haase, N., et al. (2008). Heart disease and stroke statistics--2008 update: A report from the American Heart Association statistics committee and stroke statistics subcommittee. *Circulation, 117*(4), e25-e146.

Rostrup, M., Westheim, A., Kjeldsen, S. E., & Eide, I. (1993). Cardiovascular reactivity, coronary risk factors, and sympathetic activity in young men. *Hypertension, 22*(6), 891-899.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*(16), 2192-2217.

Sarwer, D. B., Allison, K. C., & Berkowitz, R. I. (2004). Obesity: Assessment and treatment. In L. J. Haas (Ed.), *Handbook of primary care psychology* (pp. 435-453). New York: Oxford University Press.

Sedová, L., Bérubé, J., Gaudet, D., Dumont, M., Tremblay, J., Hamet, P., et al. (2004). Diet-induced obesity delays cardiovascular recovery from stress in spontaneously hypertensive rats. *Obesity Research, 12*(12), 1951-1958.

Shapiro, D., Jamner, L. D., Lane, J. D., Light, K. C., Myrtek, M., Sawada, Y., et al. (1996). Blood pressure publication guidelines. *Psychophysiology, 33*(1), 1-12.

Sherwood, A., Allen, M. T., Fahrenberg, J., Kelsey, R. M., Lovallo, W. R., & Doornen, L. J. P. (1990). Methodological guidelines for impedance cardiography. *Psychophysiology, 27*(1), 1-23.

Steptoe, A., Donald, A. E., O'Donnell, K., Marmot, M., & Deanfield, J. E. (2006). Delayed blood pressure recovery after psychological stress is associated with carotid intima-media thickness: Whitehall psychobiology study. *Arteriosclerosis, Thrombosis, and Vascular Biology, 26*, 2547-2551.

Steptoe, A., & Wardle, J. (2005). Cardiovascular stress responsivity, body mass and abdominal adiposity. *International Journal of Obesity, 29*, 1329-1337.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65*(2), 248-260.

Treiber, F. A., Kamarck, T. W., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*, 46-62.

Vega, G. L. (2001). Obesity, the metabolic syndrome, and cardiovascular disease. *American Heart Journal, 142*(6), 1108-1116.

Waldstein, S. R. (1997). Active coping and cardiovascular reactivity: A multiplicity of influences. *Psychosomatic Medicine, 59*(6), 620-625.

Waldstein, S. R., Burns, H. O., Toth, M. J., & Poehlman, E. T. (1999). Cardiovascular reactivity and central adiposity in older African Americans. *Health Psychology, 18*(3), 221-226.

Waldstein, S. R., Siegel, E. L., Lefkowitz, D., Maier, K. J., Pelletier Brown, J. R., Obuchowski, A. M., et al. (2004). Stress-induced blood pressure reactivity and silent cerebrovascular disease. *Stroke, 35*(6), 1294-1298.

Wingard, D. L. (1990). Sex differences and coronary heart disease: A case of comparing apples and pears? *Circulation, 81*, 1710-1712.

Wright, R., & Dismukes, A. (1995). Cardiovascular effects of experimentally induced efficacy (ability) appraisals at low and high levels of avoidant task demand. *Psychophysiology, 32*(2), 172-176.