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Relationship between obesity, negative affect and basal heart rate in predicting heart rate reactivity to psychological stress among adolescents

Andres E. Park [a], Pauline Huynh [a], Anne M. Schell [b,*], Laura A. Baker [a]

[a] Department of Psychology, Seeley G. Mudd Building Room 501, University of Southern California, 3620 South McClintock Ave., Los Angeles, CA 90007, United States
[b] Department of Psychology, Occidental College, 1600 Campus Rd, Los Angeles, CA 90041, United States

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ABSTRACT

Reduced cardiovascular responses to psychological stressors have been found to be associated with both obesity and negative affect in adults, but have been less well studied in children and adolescent populations. These findings have most often been interpreted as reflecting reduced sympathetic nervous system response, perhaps associated with heightened baseline sympathetic activation among the obese and those manifesting negative affect. However, obesity and negative affect may themselves be correlated, raising the question of whether they both independently affect cardiovascular reactivity. The present study thus examined the separate effects of obesity and negative affect on both cardiovascular and skin conductance responses to stress (e.g., during a serial subtraction math task) in adolescents, while controlling for baseline levels of autonomic activity during rest. Both obesity and negative affect had independent and negative associations with cardiovascular reactivity, such that reduced stress responses were apparent for obese adolescents and those with high levels of negative affect. In contrast, neither obesity nor negative affect was related to skin conductance responses to stress, implicating specifically noradrenergic mechanisms rather than sympathetic mechanisms generally as being deficient. Moreover, baseline heart rate was unrelated to obesity in this sample, which suggests that heightened baseline of sympathetic activity is not necessary for the reduced cardiovascular reactivity to stress.

1. Introduction

The ability of the body to increase energy outlay under stress is an important adaptive process. In particular, in the well-functioning organism, heart rate increases under both physical and psychological stresses (Rieger et al., 2014). Past research has indicated that certain characteristics of individuals are associated with a reduced ability to increase cardiac function under stress. One frequent finding is that individuals with a higher body mass index (BMI), a measure of general adiposity as indicated by weight in kilograms divided by height in meters squared, show lowered heart rate (HR) reactivity under conditions of psychological stress, such as increased mental workload (Carroll et al., 2008; Phillips et al., 2012, 2013). One of the more prominent explanations suggested for this relationship to obesity has been a blunted sympathetic nervous system response (Carroll et al., 2008; Phillips, 2011; Phillips et al., 2013; Singh and Shen, 2013), specifically due to high basal cardiovascular activity, which could dysregulate beta-adrenergic receptor response (Singh and Shen, 2013; Phillips et al., 2012). Another explanation, possibly related to the first, lies in an obese individual's higher level of chronic stress, which might result in impaired hypothalamic–pituitary–adrenal (HPA) axis activity and thus the body's ability to maintain allostasis (Singh and Shen, 2013).

A second set of findings relates reduced cardiac activity under stress to negative affect, particularly depression and social inhibition (de Rooij et al., 2010; Phillips, 2011; Phillips et al., 2013; Carroll et al., 2007). Such reduced reactivity has been associated with dysregulation and suboptimal functioning of the areas of the brain related to motivation, as similar studies indicate a blunted response to reward (Phillips et al., 2013). As with the blunted HR reactivity seen among the obese, blunted HR response in negative affect has been related to decreased beta adrenergic receptor responsiveness (Phillips et al., 2012), perhaps due to high basal sympathetic activity as evidenced by higher concentrations of plasma noradrenaline, and thus to a reduced capacity to respond to stress (Phillips et al., 2013; Yu et al., 2008). Gillespie and Nemeroff (2005) and Xu et al. (2011) suggest that adiposity, through a chain reaction of increased cytokines, decreased glucocorticoids, and increased cortisol concentrations, dysregulates the HPA axis, an impairment that is often associated with depression.

There appears to have been little attention to the possibility that these two correlates of reduced HR reactivity—obesity and negative affect—are themselves intertwined in their effects on HR reactivity. To our knowledge, there have been few studies that examined the relationship between obesity and negative affect. Leckie and Withers (1967) reported higher levels of depressed mood among the obese, and Pasco

* Corresponding author at: Psychology Dept., Occidental College, 1600 Campus Road, Los Angeles, CA 90041, United States. Tel.: +1 323 259 2798.
E-mail address: schell@oxy.edu (A.M. Schell).

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et al. (2013) also observed a positive correlation between BMI and negative affect; see Preiss et al. (2013) for a recent review. In cultural beauty standards in America, the obese in particular are looked upon as not only being unattractive, but also are likely to be seen as lazy, less intelligent, and less motivated (Ayers, 2008; Xu et al., 2011; Puhl et al., 2013; Hebl et al., 2012; Bradley et al., 2008). Therefore, not surprisingly, persons of high body mass index, particularly the obese, are more likely to be depressed and socially inhibited due to social ostracization and bullying (Phillips et al., 2013; Xu et al., 2011; Bradley et al., 2008). Phillips (2011) investigated the effects of both BMI and depression on cardiovascular reactivity and found that when each was used as a covariate in the analysis of the effects of the other, both higher BMI and higher depression were associated with lower levels of reactivity. However, the relationship between depression ad obesity was not itself examined.

The question therefore arises whether high BMI and negative affect independently act to reduce heart rate reactivity, or whether they are both manifestations of essentially the same process. One purpose of the present investigation was to determine if these factors are in fact independent predictors of HR reactivity under psychological stress.

A second issue arises from the fact that studies of reduced heart rate reactivity associated with either high BMI or negative affect appear to have largely included only adults. Obesity rates in the United States have grown to alarming levels, as approximately one third of adults and 17% of children and adolescents in the general population are now obese (Ogden et al., 2012, 2014). Furthermore, this growth has been paralleled in the rising rates of psychological distress in adolescents, such as major depressive disorder (Twenge et al., 2010). Because childhood and adolescent obesity and psychological distress are currently of considerable concern in American society (Pasco et al., 2013; Ayers, 2008; Xu et al., 2011; Hebl et al., 2012), it is important to know whether the reduction of heart rate reactivity associated with obesity seen in adults is also observable in younger individuals. Working with adolescents approximately 13–16 years old, Barnes et al. (1998) studied the effects of central adiposity (waist-to-hip ratio) on reactivity of systolic and diastolic blood pressure, cardiac output, and peripheral resistance, but not heart rate. They found that when BMI was statistically controlled, higher levels of obesity were associated with greater blood pressure reactivity. Goldbacher et al. (2005) studied adolescents 14–16 years old and also found that when BMI was used as a covariate, another measure of central adiposity, higher waist circumference, was positively associated with greater systolic and diastolic blood pressure reactivity to stress. Interestingly, BMI itself was negatively associated with blood pressure response — patients with lower BMI values were more responsive. Heart rate was measured in this study, but results were not reported. Thus, the second purpose of this study was to investigate the interrelationships of negative affect and BMI on heart rate reactivity in adolescents aged 14–16.

A third question of interest is the generality of the reduced reactivity to stressors seen in obesity or negative affect. Increased cardiovascular activity in response to stress is a component of increased sympathetic nervous system activity and/or vagal withdrawal, responses that are adrenergically mediated, but increases in response to stress would also be expected in other aspects of sympathetic activity, including in skin conductance level, which is entirely under sympathetic control but is cholinergically mediated (Boucsein, 2012; Dawson et al., 2007). The question thus arises as to whether the reduced reactivity seen in obesity and negative affect is limited to those sympathetic responses which are adrenergically mediated or whether it extends more broadly to include the skin conductance response.

The present study involved three research hypotheses. First, we predicted that the negative associations of HR reactivity to both BMI and negative affect seen in adults would also be found in adolescents. That is, we predicted that adolescents with higher BMI would have lower heart rate reactivity than those that have lower BMI, and that adolescents with higher negative affect would have lower heart rate reactivity than those with lower negative affect. Second, we predicted that high BMI and negative affect influence HR reactivity independently of one another, although perhaps through the same mechanism. Finally, we predicted that the negative correlations of BMI and negative affect with HR reactivity would not be seen for skin conductance reactivity, since skin conductance is cholinergically mediated at the receptor site (Boucsein, 2012) and therefore presumably would not be affected by the reduced beta adrenergic receptor responsiveness (Phillips et al., 2012) thought to be responsible for the reduced HR reactivity seen in obesity and in negative affect.

2. Method

2.1. Participants

The participants in the present analyses were participants in a longitudinal twin study at the University of Southern California (Twin Study of Risk Factors for Antisocial Behavior — RFAB). The participants were selected on the basis of being twins. The RFAB is a longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial and aggressive behavior from childhood to young adulthood. Participating families were recruited from the Los Angeles community and the sample is representative of the ethnic and socio-economic diversity of the greater Los Angeles area. To date four waves of data have been collected and analyzed and the total sample contains 1569 participants (780 twins and triplets). On the first assessment (Wave 1) the twins were 9–10 years old (N = 614, mean age = 9.59, s.d. = 0.58). On the second assessment (Wave 2), the twins were 11–13 years old (N = 445, mean age = 11.79, s.d. = 0.92). On the third assessment (Wave 3), the twins were 14–16 years old (N = 604, mean age = 14.82, s.d. = 0.83), and during Wave 4 the twins were 16–18 years old (N = 504, mean age = 17.22, s.d. = 1.23). Informed consent and assent were obtained from all participants. More detailed information regarding participant recruitment and design can be found elsewhere (Baker et al., 2002, 2006).

Analyses presented here are based on data collected during a single assessment when the participants were 14–16 years old (N = 490 with all necessary data, mean age = 14.6 years, s.d. = 0.56). The sample was composed of 51.1% females and 48.9% male and contained both monozygotic (MZ) and dizygotic (DZ) twins. 75% were twin pairs, with the remainder being singletons for which their co-twin's data on obesity, affect or autonomic responding were not available. In spite of the sample being composed of twin pairs, the present analyses were based on individuals and did not examine twin correlations since these were not directly related to the primary research questions. As described in the analysis section below, hierarchical mixed-model analyses were used for all analyses to account for the non-independence of observations due to the paired nature of the sample.

2.2. Recording autonomic variables

2.2.1. Cardiovascular responses

Heart rate (HR) was recorded using self-adhesive disposable electrocardiogram (ECG) electrodes. The electrode sites were cleaned with a cotton pad and 70% b/V isopropyl rubbing alcohol. Electrodes were then placed between the first and second ribs on either side of the chest. Alligator clips with 48 in. lead wires were then attached to the tabs of the electrodes. The ECG signal was recorded using a James Long system electrocardiogram amplifier. Data were stored for off line analysis of HR during both resting states and during tasks.

2.2.2. Skin conductance level (SCL)

SCL was also measured using the James Long system. Silver–silver chloride electrodes were secured on the distal phalanges of the index and middle fingers of the participants' non-dominant hands using adhesive electrode collars. The electrodes were then further secured