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EFFECTS OF BODY MASS INDEX ON PARASYMPATHETIC NERVOUS SYSTEM REACTIVITY AND RECOVERY FOLLOWING ORTHOSTATIC STRESS

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Abstract: Vagally mediated heart rate variability (vmHRV), defined as the beat-to-beat fluctuations in a heart series mediated by the vagus nerve, serves as a non-invasive index of parasympathetic nervous system (PNS) activity. Lower resting state vmHRV is associated with greater body mass index (BMI), providing a psychophysiological pathway linking obesity with health and disease. However little research has been conducted to examine how BMI may influence PNS reactivity to orthostatic stress. The present study sought to explore this in a sample of 59 individuals (44 females, mean age = 24.37 years, age range 19-65 years). VmHRV was measured throughout the 5-minute baseline (sitting), orthostatic (standing), and recovery (sitting) conditions. Individuals were stratified into low (BMI < 20), moderate (BMI 20-25), and high (BMI > 25) BMI groups. Results indicate that the high BMI group had a greater decrease in vmHRV from baseline to standing in comparison to the moderate BMI group. Furthermore, the low BMI group showed lower vmHRV during recovery compared to baseline, suggesting that these individuals did not fully recover from the standing position. Taken together, these results extend previous literature showing that those with low and high BMI can show different yet maladaptive patterns of vmHRV in response to orthostatic stress. .

Key words: Heart rate variability, body mass index, orthostatic, autonomic nervous system, parasympathetic nervous system.

Introduction

The autonomic nervous system (ANS) regulates many vital bodily systems, including the cardiovascular, endocrine, and inflammatory systems. The parasympathetic (PNS) branch of the ANS is of particular importance as it represents the body's integrative system to adaptively self-regulate and maintain homeostatic balance (1, 2). Vagally mediated heart rate variability (vmHRV), defined as the rapid beat-to-beat fluctuations in heart rate mediated by the vagus nerve, serves as a non-invasive index of PNS activity (1, 2). In a resting state, greater vmHRV is associated with better overall health, in addition to adaptive and flexible physiological responses to an ever-changing environment, with lower vmHRV being associated with disease and all-cause mortality (1-3).

Body mass index (BMI) remains an important indicator of physical health condition. Given that adaptive patterns of PNS activity are essential in maintaining good physical health (1-3), understanding how BMI impact PNS activity is of interest. Converging evidence suggests that greater BMI is associated with lower resting PNS activity as index by lower resting vmHRV, providing a psychophysiological pathway linking obesity with health and disease (4-6).

Given that obesity is associated with poorer health outcomes and that lower resting vmHRV is typically indicative of maladaptive physiological regulatory abilities (1-3), it is plausible that those with higher BMI may experience maladaptive patterns of PNS activity in response to physical stressors in comparison to those with normal BMI. However little research has been conducted to examine how BMI may influence PNS reactivity indicated by changes in vmHRV to changing environmental demands. One study found that in moderately obese subjects (BMI 26-40), vmHRV was lower in an orthostatic (standing) position compared to non-obese (BMI < 25) individuals (7). Similarly, another study found lower vmHRV to be associated with greater BMI while standing (8). However, studies have not addressed how BMI affects changes in vmHRV from rest to standing and back to rest. This is important, as the normal BMI and otherwise healthy individual should show a decrease in vmHRV and an increase in heart rate from rest to stand and a return to baseline levels when resting again. One study sought to examine BMI influences on vmHRV patters from rest to standing but results were not significant (9). Thus, the influence of BMI on PNS reactivity during orthostatic stress is not well understood given the conflicting aforementioned investigations. Moreover, research suggests that underweight individuals may experience poor health outcomes similar to obese individuals (10). However, research has yet to examine vmHRV reactivity differences between underweight and normal weight individuals.

Thus, the present investigation examines individual patterns of vmHRV as a function of low, moderate, and high BMI throughout baseline (seated rest), orthostatic (standing), and recovery (seated rest) positions. Overall, we sought to better understand the link between poor health outcomes and individuals who are over/underweight by directly assessing how

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BMI may influence PNS reactivity.

Materials and Methods

The study was approved by the Institutional Review Board of SRH University in Heidelberg, Germany (data collection site), and all participants provided written informed consent prior to their assessment. A total of 59 apparently healthy Caucasian students (44 females, mean age = 24.37 years, age range 19-65 years) were included in the trial. Subjects' body measures (weight and height) were taken and BMI was obtained according to common calculation (kg/m^2). Subjects reported (i) no medication intake (that may influence vmHRV) and (ii) no current suffering from chronic or acute disease.

Following informed consent, participants sat in a comfortable chair for 5-mintues for a resting (baseline) period, followed by a 5-minute orthostatic (standing) period, and finally another 5-minute resting (recovery) period. Continuous inter-beat-intervals (IBI) was measured throughout the experiment at sampling frequency of 1000 Hz using a Polar RS800CX portable device, providing a temporal resolution of 1 ms for each R– R interval. The device's transmitter consists of a stable polyamide case with electrodes attached to an elastic belt fixated to the chest of the subjects. Device-specific software (Polar ProTrainer 5) was used to transfer recordings to a personal computer. IBI data were imported and analyzed with "Kubios HRV - Heart Rate Variability Analysis Software" (Biosignal Analysis and Medical Imaging Group, University Kuopio, Finland, Version 2.0, (11)) and analyzed. The square root of the mean squared difference of successive RR intervals (RMSSD, ms) was calculated and is considered to be a stable and valid (12) time-domain index of vmHRV. Autoregressive estimates were also calculated, yielding high frequency power HRV (HF-HRV, 0.15-0.4 Hz). In the present study RMSSD correlated highly with HF power throughout all phases of the experiment (each $r > .88$, $p < .001$), and as such we report vmHRV results using RMSSD – results were identical using HF-HRV (results not shown). Data on vmHRV measures was skewed and successfully log-transformed to meet assumptions of linear analyses. Individuals we stratified into three separate BMI groups: (i) low BMI (BMI < 20 | n = 14) ; (ii) moderate BMI (BMI = 20-25 | n = 33), (iii) high BMI (BMI > 25 | n = 12). StatsSoft Statistica 6.0 (StatSoft, Inc.,Tulsa, OK) was used to conduct a 2-way (1 between factor and 1 within factor) analysis of variance (ANOVA) to examine differences in vmHRV patterns between BMI groups. Experimental phases (baseline, standing, recovery) were used as the within factor and BMI groups (low, moderate, high) as the between factor. Because these are physiological data, within and within-between ANOVA interactions are reported using multivariate ANOVA tests (Wilks' Lambda (λ) and associated degrees of freedom), and preplanned contrasts were used as a direct test of differences in vmHRV throughout the experiment (13, 14). All tests were two-tailed with significance set at alpha of 0.05.

Results

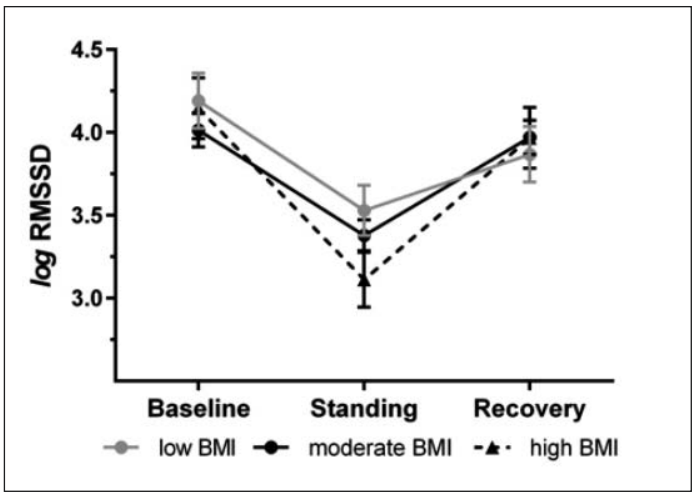
Adjusted means and standard deviations (for age and gender) of vmHRV in each position split by BMI group are displayed in Table 1. In this sample, individuals' BMI ranged from 17.16 - 32.35 kg/m^2.

Table 1
Adjusted Means and Standard Deviations of vmHRV by BMI Group throughout Experimental Positions

	n	Baseline-rest vmHRV	Standing vmHRV	Recovery-rest vmHRV
Low BMI	14	4.19 (.17)	3.53 (.15)	3.87 (.17)
Moderate BMI	33	4.02 (.10)	3.38(.09)	3.97(.10)
High BMI	12	4.15(.18)	3.11(.17)	3.97 (.18)
Total	59	4.12 (.09)	3.35 (.07)	3.94 (.08)

Note: The table above provides adjusted means for age and gender (standard deviation in brackets) values of vmHRV (ln-RMSSD) for resting, standing, and recovery phases by BMI group. Body mass index (BMI) was calculated in kg/m2 and individuals were stratified into three groups: low BMI (BMI < 20 | n = 14); (ii) moderate BMI (BMI = 20-25 | n = 33), (iii) high BMI (BMI > 25 | n = 12).

Figure 1
BMI Group by Position Interaction



Note: Figure 1 depicts the strong trending interaction (λ = .841, F(4,106) = 3.00, r = .319, p = .058) of body mass index (BMI) group and position on vmHRV throughout the experiment. Means and standard errors are adjusted for gender and age (see Methods and Materials). Low BMI (BMI < 20 | n = 14); (ii) moderate BMI (BMI = 20-25 | n = 33), (iii) high BMI (BMI > 25 | n = 12). In comparison to the moderate BMI group, the low BMI group's vmHRV was lower at recovery compared to baseline (F(1,54) = 5.57, r = .306, p = .022), and the high BMI group's vmHRV showed a greater decrease in vmHRV from baseline to standing group (F(1,54) = 4.746, r = .284, p = .034).

Controlling for age and gender, repeated measure ANOVA results showed no significant main effect of BMI group (F(2, 55) = .166, r = .077, p = .848), a significant main effect of position (λ = .720 F(2,53) = 10.28, r = .529, p < .001), and a strong trending interaction between the two (λ = .841, F(4,106)

= 3.00, r = .319, p = .058) (Figure 1). Preplanned contrasted showed a significant quadratic trend overtime in each BMI group (each p < .001), such that participants' vmHRV decreased from baseline while standing, and increased when sitting again. However in the low BMI group, vmHRV during recovery was significantly lower than vmHRV during baseline (F(1,54) = 5.57, r = .306, p = .022), indicating that this group's vmHRV did not fully recover. Individuals in the moderate and high BMI groups did not show significant linear decreases from baseline to recovery, suggesting good vmHRV recovery from an orthostatic position.

The high BMI group showed a significantly greater decrease in vmHRV from baseline to standing in comparison to the moderate BMI group (F(1,54) = 4.746, r = .284, p = .034). Although there were no other significant contrasts between groups, a trend showed that the moderate BMI group had slightly higher vmHRV during the orthostatic position in comparison to the high BMI group (F(1,54) = 3.29, r = .240, p = .075).

Discussion

The present study sought to explore differences in vmHRV reactivity throughout a simple orthostatic stress test (baseline-rest, standing, recovery-rest) based on BMI profiles. To our knowledge, few studies have investigated the impact BMI may have on vagal activity when making simple movements, showing that those with greater BMI had lower vmHRV during an orthostatic position (7, 8). Our data replicate these previous findings, as the moderate BMI group showed slightly higher vmHRV in comparison to the high BMI group while standing. In contrast to previous research (9) and adding to the literature, those in the high BMI group showed a steeper decrease in vagal activity from baseline to standing, suggesting that those with high BMI have lower PNS activity when altering body positions. Thus, the present investigation, coupled with previous research, suggests that individuals with high BMI have lower PNS at rest (4-6) which is considered maladaptive (1-6), in addition greater withdraw of the PNS while standing, and possibly other physical activities (15, 16). We theorize that these maladaptive physiological responses may have a negative impact on overall health in these individuals.

Results also showed lower vmHRV during recovery compared to baseline in low BMI group, suggesting that these individuals' vmHRV did not fully recover from the orthostatic position as it did in individuals with moderate and high BMI groups. This suggests prolonged vmHRV reactivity in response to the orthostatic position, which can be considered maladaptive and harmful for health (3). Future research is needed to understand potential physiological mechanisms and time course underlying this phenomenon in those with low BMI.

Both age and gender can influence vmHRV (17, 18) and thus, were included as covariates in the current investigation. Importantly, the age-range for the current sample was between 19 and 65 years, with 46 individuals falling between 19 and 25, eight individuals between 25 and 30, three individuals between 30 and 40, and 2 individuals over 40. Therefore, it is important to note that interpretations and implications of the current study are applicable primarily to younger adults.

One limitation of the current study is that we did not control for body composition (i.e., fat vs. lean mass), and future investigations should include this variable as a covariate as the interpretation of BMI can be misleading depending on body composition (19).

Overall, the present investigation suggests that both low and high BMI can lead to maladaptive patterns in vmHRV reactivity under different conditions. In line with previous research (7, 8), those with high BMI show a greater decrease in vmHRV in response to standing and possibly other physical activities, whereas those with low BMI show prolonged decreased vmHRV following standing, as they did not recovery back to baseline within the 5-minute rest period. Future research needs to replicate the present findings extending the experimental paradigm to other physical activities.

Ethical Standards: The study was approved by the Institutional Review Board of SRH University in Heidelberg, Germany (data collection site), and all participants provided written informed consent prior to their assessment.

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References

1. Thayer J, Lane R. A model of neurovisceral integration in emotion regulation and dysregulation. Journal Of Affective Disorders 2000;61:201-216
2. Thayer J, Åhs F, Fredrikson M, Sollers I, Wager T. Review: A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. Neuroscience And Biobehavioral Reviews 2012;36:747-756
3. Thayer JF, Sternberg E. Beyond heart rate variability. Annals of the New York Academy of Sciences. 2006 Nov 1;1088(1):361-72.
4. Molfino A, Fiorentini A, Tubani L, Martuscelli M, Fanelli F, Laviano A. Body mass index is related to autonomic nervous system activity as measured by heart rate variability. European Journal Of Clinical Nutrition 2009;63(10):1263-1265.
5. Koenig J, Jarczok M, Thayer J, Warth M, Ellis R, Bach C, Hillecke T. Body mass index is related to autonomic nervous system activity as measured by heart rate variability - A replication using short term measurements. Journal Of Nutrition, Health & Aging 2014;18(3):300-302 3p.
6. Koenig J, Windham BG, Ferrucci L, Sonntag D, Fischer JE, Thayer JF, Jarczok MN. Association strength of three adiposity measures with autonomic nervous system function in apparently healthy employees. The journal of nutrition, health & aging. 2015 Nov 1;19(9):879-82.
7. Piccirillo G, Vetta F, Marigliano V, Viola E, Santagada E, Ronzoni S, Cacciafesta M. Heart rate and blood pressure variability in obese normotensive subjects. International Journal Of Obesity And Related Metabolic Disorders: Journal Of The International Association For The Study Of Obesity 1998;22(8):741-750
8. Byrne EA, Fleg JL, Vaitkevicius PV, Wright JE, Porges SW. Role of aerobic capacity and body mass index in the age-associated decline in heart rate variability. Journal of Applied Physiology. 1996 Aug 1;81(2):743-50.
9. Kim JA, Park YG, Cho KH, Hong MH, Han HC, Choi YS, Yoon D. Heart rate variability and obesity indices: emphasis on the response to noise and standing. The Journal of the American Board of Family Practice. 2005 Mar 1;18(2):97-103.
10. Kushner RF (1993). Body Weight and Mortality. Nutrition Review 1993; 51(5):127-136.
11. Tarvainen MP, Niskanen JP, Lipponen JA, Ranta-Aho PO, Karjalainen PA. Kubios HRV–heart rate variability analysis software. Computer methods and programs in biomedicine. 2014 Jan 31;113(1):210-20.
12. Thayer J, Hansen A, Johnsen B. The Non-invasive Assessment of Autonomic Influences on the Heart Using Impedance Cardiography and Heart Rate Variability. Handbook Of Behavioral Medicine 2010:723

13. Rosnow RL, Rosenthal R. "Some things you learn aren't so": Cohen's paradox, Asch's paradigm, and the interpretation of interaction. Psychological Science. 1995;6(1):3-9.
14. Vasey M, Thayer J. The Continuing Problem of False Positives in Repeated Measures ANOVA in Psychophysiology: A Multivariate Solution. Psychophysiology 1987;24(4):479
15. De Meersman RE. Heart rate variability and aerobic fitness. American heart journal. 1993 Mar 31;125(3):726-31.
16. Rennie KL, Hemingway H, Kumari M, Brunner E, Malik M, Marmot M. Effects of moderate and vigorous physical activity on heart rate variability in a British study of civil servants. American Journal of Epidemiology. 2003 Jul 15;158(2):135-43.
17. Choi JB, Hong S, Nelesen R, Bardwell WA, Natarajan L, Schubert C, Dimsdale JE. Age and ethnicity differences in short-term heart-rate variability. Psychosomatic medicine. 2006 May 1;68(3):421-6.
18. Koenig J, Thayer JF. Sex differences in healthy human heart rate variability: A meta-analysis. Neuroscience & Biobehavioral Reviews. 2016 May 31;64:288-310
19. Prentice AM, Jebb SA. Beyond body mass index. Obesity reviews. 2001 Aug 1;2(3):141-7.