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Diminished cardiovascular stress reactivity is associated with lower levels of social participation.

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Abstract

Diminished cardiac reactions to acute psychological stress are associated with adverse behavioral and health-relevant outcomes. It has been proposed that diminished cardiac reactivity may be a marker for deficits in motivational functioning both at the biological and behavioral levels. Social participation reflects the frequency with which individuals participate in social events and has motivational components. As such, it is a distinct construct from other constructs such as social integration, which measures the number of social roles an individual has. Additionally, lower levels of social participation and diminished reactivity have been associated with similar adverse health outcomes. Therefore, it is possible that diminished cardiac reactivity is associated with lower levels of social participation. We aimed to examine whether diminished cardiovascular reactivity in response to an acute lab stressor was associated with reported social participation. The analyses were conducted using publicly available data from the Pittsburgh Cold Study 3 (PCS3). The PCS3 was a prospective viral-challenge study, which included participants completing an in-lab social evaluative stressor (N=202, Age= M= 29.71, SD= 10.66) and measuring cardiovascular responses at baseline and in response to the stressor. Separate regression analyses for each cardiovascular variable (SBP, DBP, MAP, and HR) demonstrated that lower cardiovascular reactivity was associated with less social participation. These associations were still evident following adjustment for respective baseline cardiovascular levels, age, sex, race, depressive symptomology, body mass index, socioeconomic status, smoking status, and levels of social integration (R^2 changes: ≥ .017; *p*s ≤ .02) The findings provide initial evidence that blunted cardiac reactivity may be a precursor to low levels of social participation.

Extreme cardiovascular responses to acute psychological stress are associated with adverse health outcomes [1,2]. Individuals who show exaggerated cardiovascular reactions to acute psychological stress are more likely to develop hypertension [3], atherosclerosis [4], and increased left ventricular mass [5]. They are also at an increased risk for cardiovascular

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disease mortality [6]. However, recent evidence suggests that diminished cardiovascular reactions to acute psychological stress are also associated with adverse health outcomes, mainly behavioral outcomes [2,7,8]. For example, diminished levels of cardiovascular reactivity are associated with lower levels of behavioral perseverance [9–10], and higher levels of depressive symptomology [11–15]. It has been proposed that diminished reactivity is a marker of a dysfunction of neural systems that support motivated and goal directed behavior and that it may be associated with outcomes that reflect, whether explicitly or inadvertently, variations in psychological effort and motivation [2,7.15].

Another behavioral outcome, which involves motivation, is social participation, or the frequency with which an individual engages in social activities with others [16].The act of social participation requires motivated effort, which is distinct from social integration. Social integration reflects the number of social roles an individual has, yet is also associated with a host of health-relevant outcomes [17]. An individual may have a high number of social roles, however, these roles do not necessarily require motivation and may or may not be desired by the individual. Given that diminished cardiovascular activity is associated with reduced motivated behavior, lower levels of perseverance, and higher levels of depression, it is possible that individuals with diminished cardiovascular responses to stress are less likely to engage in social participation.

Interestingly, many of the observed relationships between social participation and health are the same as the those observed between cardiovascular reactivity and health [1,2,18]. For example, higher levels of social participation are related to better self-rated health [19–20], less cognitive decline [21–22], and lower depressive symptoms [23–24]. Similarly, diminished cardiac reactivity to acute psychological stress is associated with worse self-rated health [25–26], greater cognitive decline [27,28], and higher depressive symptoms [29,30, 31]. Therefore, it is possible that in addition to being a marker of adverse health and behavioral outcomes, diminished reactivity may also be a characteristic of individuals who are less likely to engage in social participation. Extending from this, it is plausible that diminished cardiac reactivity to acute stress may precede lower levels of social participation, however, this relationship has yet to be examined. If diminished cardiac reactivity is associated with low levels of social participation, it could be another behavioral pathway through which diminished reactivity affects mental and physical health.

The aim of this study was to examine the association between cardiovascular responses to acute psychological stress and social participation. Based on the motivational component required of social participation and the similar relationships between health and social participation and health and cardiovascular reactivity, it was hypothesized that low cardiovascular reactivity would be associated with lower social participation.

Methods

The data in this manuscript were from the Pittsburgh Cold Study 3 (PCS3). The PCS3 was a prospective viral challenge study with data collected between 2007–2011. The sample consisted of 213 healthy volunteers ages 18–55 (Mean=30.1; SD=10.9) from the Pittsburgh, Pennsylvania metropolitan area. Data were collected by members of the Laboratory for the

Study of Stress, Immunity, and Disease at Carnegie Mellon University. This lab is directed by Sheldon Cohen, Ph.D. Data were obtained from the Common Cold Project website (www.commoncoldproject.com; grant number NCCIH AT006694).

Participants provided informed consent and received $1000 for their participation of the entire protocol. The study was approved by the Carnegie Mellon University and University of Pittsburgh Institutional Review Boards. The exclusion criteria for the larger project were: regular medication regimen, previous nasal surgery, psychiatric hospitalization within the last 5 years history of chronic illness or any psychiatric disorder treated within one year of study enrollment, abnormal clinical profile (discovered via urinalysis, complete blood count or analysis of blood chemistry), human immunodeficiency virus seropositivity, current pregnancy or lactating, use of steroids or immunosuppressants within three months of trial, participation in another study involving psychological questionnaires and or investigational products within the last 30 days or plans to participate in such research while enrolled in the current study, cold or flu-like illness within 30 days prior to the infection with virus as part of this project, living with someone who has chronic obstructive pulmonary disease or an immunodeficiency, previous hospitalization for a flu like illness and allergies to eggs or egg products. In the current study, we focused on physiological responses to a stress task during a session was completed before virus inoculation and its relation to self-reported social participation. Social participation was measured in a questionnaire administered 6 weeks prior to the quarantine period.

For the stress task, participants attended an individual session that started between 3:00 pm and 9:00 pm at the University of Pittsburgh. Participants were asked to abstain from alcohol for 48 hours, from exercise and nonprescription medications for 24 h, from eating and drinking (except water) for 2 h, and from smoking for 1 h prior to the session. Upon arriving at the lab, participants were interviewed to ensure they had followed these instructions.

At the beginning of the stress protocol, participants were asked to sit quietly for 20 minutes (Habituation phase). Immediately following the Habituation phase, participants performed a modified version of the Trier Social Stress Test (TSST) [32]. They were told they had to deliver a 5-minute videotaped speech defending themselves against an alleged transgression (shoplifting or traffic violation) and a 5-minute mental arithmetic task. The protocol included an Anticipatory phase in which the participants had 5 minutes to prepare their speech. Immediately following the Anticipatory phase, the participants performed their speech and completed the arithmetic task (Stress phase). Finally, the participants were asked to rest quietly for 50 minutes (Recovery phase).

Participants were fitted with automated blood pressure (BP) cuff for measurement of systolic and diastolic blood pressure (SBP and DBP), mean arterial pressure (MAP) and heart rate (HR). During the 20-minute habituation period, HR and BP were measured 4 times (every 120 seconds) during the last 6 minutes. During the 15-minute Stress phase, HR and BP were measured every 2 minutes, yielding 9 readings (start of Stress phase, 2 minutes, 4 minutes, 6 minutes, 8 minutes, 10 minutes, 12 minutes, 14 minutes and 16 minutes). While the stress task itself took approximately 15 minutes, there was some time allotted for transitions between tasks. As such, the final HR and BP readings were taken at 16 minutes after the

beginning of the Stress phase. Three of these readings were taken during the speech preparation phase, 3 were taken during the speech delivery phase, and 3 were taken during the mental arithmetic phase. Finally, during the recovery phase, HR and BP were measured every 120 seconds for the first 15 minutes (8 readings) and then every 5 minutes (7 readings).

Measures

Cardiovascular Reactivity.—The study included two stress task sessions. However, only data from the first session are reported since the aim was not to examine habituation in physiological responses to a repeated task. Cardiovascular stress reactivity was defined as the difference between the mean averages during stress and baseline for each of the four cardiovascular variables.

Social Participation Measure.—Using a 6-point frequency scale (1= did not do this at all in the past year, 2= 1–5 times this past year, 3= once a month or every two months, 4= once every two or three weeks, 5= once a week, 6= more than once a week), respondents indicated how often they engaged in 16 social activities during the past 12 months (e.g., visited friends, participated in a competitive sport, went to a restaurant/bar/coffee shop with others). Responses were summed to reflect total social participation. The measure had good reliability in this study (α=0.77).

Social Integration.—Using data from the social network index, a measure of social integration was calculated. This is a measure of the number of different types of high contact social roles in which individuals participate. High contact roles are defined as those for which the respondent reports engaging at least once every two weeks. For example, for a participant to be assigned the role of “parent”, they must indicate that they talk to at least one of their children at least once every two weeks. Social integration is computed as the sum of all high contact roles [17,33]. Social integration was used as a covariate to examine if any relationships observed were due to active participation (with a motivational component) or simply from being involved in a number of social roles.

Covariates

Depressive Symptoms.—The Trait Affect Questionnaire was administered twice during the PCS3 [33,34]. It consisted of 25 items and produced 5 subscales of negative affect, including depression. The depression subscale consisted of three items including sadness, unhappiness, and depression. It was administered once 7–8 weeks before the quarantine period and again immediately preceding the quarantine period. As a measure of depressive symptoms, we utilized the score from the measurement immediately preceding the quarantine period. The measure had good reliability in this study (α=0.87).

Body Mass Index.—Body mass index was calculated using participants’ height and weight recorded at the reactivity session.

Socioeconomic Status.—As a measure of current socioeconomic status, participants reported their current annual income. Reported income was categorized across 13 categories

(1= less than $5,000, 2= $5,000-$9,999, 3= $10,000-$14,999, 4= $15,000-$19,999, 5= $20,000-$29,999, 6=$30,000-$39,999, 7= $40,000-$49,999, 8=$50,000-$59,999, 9= $60,000-$74,99, 10=$75,000-$99,999, 11= $100,000-$124,99, 12=$125,000-$149,999, 13= $150,000 or more).

Smoking Status.—Participants reported whether they were a current smoker (0= no, 1= yes).

Statistical analyses

Analyses were conducted using SPSS version 25 (IBM Corp, USA). Repeated-measures ANOVAs, using baseline and stress task values, were undertaken to confirm that the TSST perturbed the cardiovascular system. The associations between cardiovascular reactivity and social participation were analyzed using multiple linear regression with social participation as the dependent variable first in models that adjusted only for respective baseline cardiovascular levels. Then analyses were run in models that additionally adjusted for age, sex, race (dummy coded as 1 = White, 0 = Other), depressive symptoms, body mass index, socioeconomic status, and smoking status. These covariates were selected based on previously documented associations between these variables and cardiovascular reactivity [2,35–37]. Lastly, to ensure that the social participation results were independent of social integration, a measure of number of high contact social roles, a third model additionally adjusting for social integration was run for each of the cardiovascular reactivity variables.

Results

Descriptive statistics are listed in Table 1 and bivariate correlations between main variables of interest are listed in Table 2. In this sample, social participation was associated social integration (r=.48).

Cardiovascular Stress Reactivity

Repeated measures ANOVA (baseline, task) indicated that the TSST significantly perturbed SBP, $F(1, 205) = 418.40$, $p < .001$, $_p eta^2 = .671$; DBP, $F(1, 205) = 567.84$, $p < .001$, $_p eta^2 = .735$; MAP, $F(1, 205) = 680.47$, $p < .001$, $_p eta^2 = .768$; and HR, $F(1, 205) = 356.70$, $p < .001$, $_p eta^2 = .635$. The summary statistics are presented in Table 3.

Cardiovascular stress reactivity and Social Participation

Cardiovascular stress reactivity for all variables of interest (SBP, DBP, MAP, and HR) were positively associated with social participation; the lower the cardiovascular reactivity the lower the social participation. Effect sizes and other statistics of these associated are presented in Table 4. Table 4 also shows that, firstly, adjustment for respective baseline cardiovascular activity and then, additionally, for age, sex, race, depressive symptoms, body mass index, socioeconomic status, and smoking status did not abolish the associations between cardiovascular reactivity and social participation for any of the variables. Although the effect sizes became smaller, in all cases the associations remained statistically significant. Similarly, when additionally adjusting for social integration, the associations between cardiovascular reactivity and social participation remained statistically significant.

Conclusion

Our analyses indicate that diminished cardiovascular reactivity in response to an acute laboratory stressor is associated with lower levels of self-reported social participation. The associations remained significant when controlling for important variables related to both acute psychological stress and social participation (e.g., age, gender, socioeconomic status) and when controlling for how many social contacts an individual has. These findings are in line with a growing body of research which indicates that diminished cardiovascular reactions to stress are associated with impairments in behaviors that require motivation [2,9,10].

Diminished cardiovascular reactivity is believed to be a marker of a dysfunction of the neural systems, which support motivated and goal directed behavior [2,38]. Blunted cardiovascular reactors exhibit reduced activation during acute psychological stress in areas involved in autonomic regulation [38,39]. Areas of the brain implicated in autonomic processing also comprise a network vital for motivated behavior [40]. It has been proposed that individuals with diminished reactivity to stress have a wider biological disengagement which prevents full engagement in activities that require motivated behavior [41]. Therefore, if blunted reactivity is a marker of a wider biological disengagement, it is possible that individuals with diminished cardiovascular reactivity are less able to engage in social participation because of differences in the function of neural systems which are critical to the promotion of social engagement and participation.

Given that social participation requires motivation, lower levels of cardiovascular reactivity could give rise to lower levels of social participation resulting in poorer health outcomes. This is supported by the overlap in negative health outcomes associated with both low cardiovascular reactivity and social support (e.g., depression, cognitive decline, poorer self-rated health). Additionally, social participation has potential health benefits. Social participation is viewed as social sharing of individual resources through active participation in social activities and is believed to exert positive effects on health by providing a sense of meaning and increasing access to social support [42]. Future work is needed to understand whether cardiovascular reactivity associates with future social participation and whether levels of social participation may affect downstream health outcomes by shaping psychosocial factors and resources.

There are important limitations to this research. First, since the research is cross-sectional, causality cannot be inferred [43]. For example, it is possible that social experience may shape cardiovascular reactivity or more specifically that lack of social engagement could lead to diminished reactivity. The current origins of diminished reactivity are unknown [2]. Longitudinal or experimental research examining cardiovascular reactivity and social participation is needed to help establish directionality. While analyses adjusted for depressive symptomology, the scale used was not a clinically validated scale. Future research should aim to replicate and extend these findings using a clinically validated depression scale. Further, while cardiovascular reactivity was measured in real-time in a lab setting, the measure of social participation reflects how often participants engaged in a variety of social activities during the last 12 months and is self-report. As noted previously,

this data cannot speak to whether cardiovascular reactivity would predict future levels of social participation. However, previous research does indicate that levels of social participation and cardiovascular reactivity are relatively stable over time [44,45]. Future research should explore whether individuals who exhibit diminished cardiovascular reactivity are unsatisfied with their level of social participation and whether they are motivated to become more socially engaged.

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Highlights:

- Blunted Cardiovascular stress reactivity associates with lower social participation
- Relationship was independent of depressive symptoms and social integration
- Cardiovascular stress reactivity could be a precursor to social participation

Table 1.

Descriptive Statistics

Measure	n	M (SD)/%	Range
Age	202	29.7 (10.7)	18–55
Race	202	64.8% White	
Socioeconomic Status	202	3.9 (2.6)	1–13
Social Participation	202	52.5 (11.9)	25–86
Social Integration	202	5.2 (1.9)	2–10
Depressive Symptoms	202	.39 (.72)	0–3
Body Mass Index	202	27.4 (6.5)	16.9–50.7
Current Smokers	202	33.8%	
Social Support	202	29.2 (5.7)	6–36

Table 2.

Correlations between main variables of interest.

	MAP residual	DBP residual	SBP residual	HR residual
1. age	−0.07	−0.10	−0.08	−0.30**
2. sex	−0.20**	−.24**	−.19**	−.01
3. race	.28**	.25**	.36**	.21**
4. depressive symptoms	.04	.05	−.02	.02
5. social participation	.28**	.26**	.30**	.26**
6. social integration	−.07	.04	.11	.07

Note: MAP: Mean Arterial Pressure, DBP: Diastolic Blood Pressure, SBP: Systolic Blood Pressure, HR: Heart Rate. Sex: male= 0 , female = 1. Race: 1= White/Caucasian, 0= all others.

Table 3.

Mean (SD) Cardiovascular activity at baseline and during stress task exposure

	SBP mmHg	DBP mmHg	MAP mmHg	HR bpm
Baseline	114.8 (12.6)	67.5 (83)	84.8 (8.8)	69.2 (10.5)
Stress *	130.4(14.5)	77.9 (8.1)	98.3 (9.4)	80.0 (12.6)

* Stress differed from Baseline for all four variables, $p < .001$ in each case.

Table 4.

Regression Models for Social participation and Cardiovascular reactivity

	β	t	p	R^2 change
SBP reactivity				
Model 1	.31	4.45	<.001	.089
Model 2	.24	3.36	.001	.046
Model 3	.17	2.69	.008	.023
DBP reactivity				
Model 1	.28	3.85	<.001	.066
Model 2	.22	2.89	.004	.034
Model 3	.19	2.82	.005	.026
MAP reactivity				
Model 1	.30	4.25	<.001	.081
Model 2	.24	3.33	.001	.045
Model 3	.19	3.06	.003	.030
HR reactivity				
Model 1	.26	3.89	<.001	.068
Model 2	.18	2.52	.01	.026
Model 3	.14	2.28	.023	.017

Notes: Model 1 includes respective baseline cardiovascular activity; Model 2 additionally includes age, sex, race, depressive symptoms, body mass index, socioeconomic status, and smoking status; Model 3 additionally includes social integration.