Social participation and cardiovascular stress-response adaptation: Evidence for enhanced stress tolerance

Tracey Keogh¹ and Siobhán Howard¹

¹University of Limerick

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Abstract

Diminished cardiovascular reactivity is a proposed marker of motivational dysregulation and is related to a range of adverse behavioural and health outcomes. Social participation represents the frequency an individual engages in social activities, is a form of motivated behaviour, and has been recently linked to lower cardiovascular responses to acute psychological stress. With recent work emphasizing the importance of assessing adaptation of the cardiovascular response to recurrent stress, the aim of the current study is to build on previous work by examining the relationship between social participation and cardiovascular stress response adaptation. Analyses were conducted using a general linear model and previously obtained data from the Pittsburgh Cold Study 3 (PCS 3). Two hundred and thirteen participants (M = 30.13; SD = 10.85) completed a social participation measure and had their systolic and diastolic blood pressure (SBP, DBP) and heart rate (HR) monitored across two separate standardized stress testing sessions. In line with previous research, lower social participation was related to lower cardiovascular responses to stress. Findings also indicate that higher levels of social participation were associated with greater blood pressure habituation to recurrent stress. Given that cardiovascular habituation to recurrent stress signifies good health, the implications of these findings are discussed. Key words: social participation, cardiovascular reactivity, cardiovascular habituation, blood pressure, Pittsburgh Cold Study.


Tracey M. Keogh, PhD¹,²* and Siobhán Howard, PhD¹,²

¹Department of Psychology, Centre for Social Issues Research, Study of Anxiety, Stress and Health Laboratory, University of Limerick, Limerick, Ireland

²Health Research Institute, University of Limerick, Castletroy, Limerick, Ireland

*Corresponding author

Tracey Keogh
Department of Psychology
University of Limerick
Castletroy
Limerick
Ireland

Contact: tracey.keogh@ul.ie/ +353 (0) 61 23 3618

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Abstract

Diminished cardiovascular reactivity is a proposed marker of motivational dysregulation and is related to a range of adverse behavioural and health outcomes. Social participation represents the frequency an individual engages in social activities, is a form of motivated behaviour, and has been recently linked to lower cardiovascular responses to acute psychological stress. With recent work emphasizing the importance of assessing adaptation of the cardiovascular response to recurrent stress, the aim of the current study is to build on previous work by examining the relationship between social participation and cardiovascular stress response adaptation. Analyses were conducted using a general linear model and previously obtained data from the Pittsburgh Cold Study 3 (PCS 3). Two hundred and thirteen participants ($M = 30.13; SD = 10.85$) completed a social participation measure and had their systolic and diastolic blood pressure (SBP, DBP) and heart rate (HR) monitored across two separate standardized stress testing sessions. In line with previous research, lower social participation was related to lower cardiovascular responses to stress. Findings also indicate that higher levels of social participation were associated with greater blood pressure habituation to recurrent stress. Given that cardiovascular habituation to recurrent stress signifies good health, the implications of these findings are discussed.

Key words: social participation, cardiovascular reactivity, cardiovascular habituation, blood pressure, Pittsburgh Cold Study.

1. INTRODUCTION

It is well-established that heightened physiological responses to stress pose a risk for future disease outcomes, and studies examining correlates of cardiovascular reactivity (CVR) to psychological stress have proven a fruitful avenue of research in psychosomatic medicine spanning the last four decades. Exaggerated cardiovascular responses are associated with hypertension (for reviews see Chida & Steptoe 2010; Turner et al., 2020), atherosclerosis (Barnett et al., 1997; Matthews et al., 1998), and cardiac mortality (Carroll et al., 2012).

In recent years, diminished cardiovascular responses to stress have been found to be related to a range of adverse health and behavioural outcomes; including obesity (Phillips, 2011), poor health (Phillips et al., 2009), carotid intima-media thickness (Ginty et al., 2016), cardiac-related hospitalizations (Sherwood et al., 2017), increased mortality risk (Kupper et al., 2015), poorer cognitive function (Ginty et al., 2012), substance abuse (Lovenberg et al., 2000), smoking (al’Absi et al., 2003), personality factors (e.g., Hughes et al., 2011; O’Riordan et al., 2022), depression (Keogh et al., 2022; Shier et al., 2021) and both proximal (Carroll et al., 2005) and distal (Keogh et al., 2023; Lovenberg, 2013) negative life events.

Further, diminished CVR is a proposed marker of dysfunction in brain regions responsible for motivation and goal directed behaviour (Carroll et al., 2017), and is related to higher levels of behavioural disengagement (Ginty et al., 2020). According to the self-determination theory (SDT) of motivation, satisfying basic psychological needs; (i.e., autonomy, competency and relating to others), can facilitate motivation (Deci & Ryan, 2008; 2012; Ryan & Deci 2000). In an educational context for example, methods of participation and engagement are employed to enhance motivation and increase academic performance (Christenson, Reschly, & Wylie, 2012). A core component of SDT is relatedness, which is a need to feel connected to others, and participating in a socially supportive environment is vital for psychological well-being (Chen et al., 2015) and performance (Deci & Ryan, 2012).
The social context in which one operates can influence types of motivation and goal-directed behaviors (Chen et al., 2015). Social participation is one form of motivated behavior that has been recently linked to lower cardiovascular responses to acute psychological stress (John-Henderson et al., 2019).

Social participation is characterized by engagement across formal and informal social activities and is measured by how often a person engages in activities pertaining to social, religious, and organizational-related events. Actively engaging in social roles and activities is necessary for our overall health and survival, with reports that connecting with others in a social context predicts survival among an older sample (Maier & Klumb, 2005), and reduces the risk of depression (Choi et al., 2021). It is not surprising then that higher levels of social engagement are related to better mental and physical health outcomes, including quality of life and general well-being (Dahan-Oliel, Gelinas, & Mazer, 2008).

Notably, social participation is related to many known correlates of diminished CVR, including depression (Croezen et al., 2015), self-reported health (Lee et al., 2008) and cognitive decline (Bourassa et al., 2017). Research shows that social participation is linked to cognitive function, with greater levels of social engagement reportedly related to lower risk of dementia (Wang et al., 2002; Zhou et al., 2018). Low levels of social participation predict risk of coronary heart disease (CHD), even after controlling for confounds (Sundquist et al., 2001). This may in part reflect non-adherence to medications, with lack of motivation identified as a barrier to active participation in treatment among cardiac patients (Baroletti & Dell’Orfano, 2010). In fact, many of the aforementioned outcomes related to lower reactivity, utilise treatment programs with social elements that require a degree of effort, motivation, and engagement. While one study using a traditional stress protocol found diminished cardiovascular responses became less pronounced when accounting for motivation (Keogh et al., 2021), how motivated behavior is related to adaptation to stress over time is unknown.

Assessment of CVR in a single stress paradigm may be less informative than examination of cardiovascular adaptation to recurrent stress. Examining patterns of cardiovascular stress-response habituation may offer a more ecologically valid assessment of an individual’s stress tolerance, by assessing cardiovascular responses to recurrent stress (Howard & Hughes, 2012) and thereby revealing important health-related differences in how individuals respond to stress (Hughes, Liu, & Howard, 2018). Both exaggerated and diminished responses to stress are related to risk of adverse outcomes (Phillips, Ginty, & Hughes, 2013; Whittaker et al., 2021). While an initial mounting of the stress response produces necessary and purposeful physiological changes, if sustained over time, this can signify poorer health consequences. Hughes et al. (2018) suggest that patterns of habituation are useful in understanding health components of CVR, in predicting health outcomes and can reveal psychosocial factors that influence stress.

While lower levels of social participation have been shown to be related to diminished CVR to stress (John-Henderson et al., 2019), no research has examined whether social participation influences an individual’s capacity to adapt to recurrent stress. Previous work has focused on components of social participation, such as social support (e.g., perceived social support; Howard & Hughes, 2012; social network; Hughes, 2007; social connectedness; Creaven et al., 2020), and identified these variables as important moderators of the initial stress response. Motivated behavior is required for successful engagement in treatment programs; and greater engagement in social activities is related to positive health outcomes (both physical and mental; Santini et al., 2020).

Recent findings suggest that low social participation is related to lower CVR (John-Henderson et al., 2019); perhaps diminished stress responses, a proposed marker of motivational dysregulation, engenders lower levels of social participation in social activities, giving rise to poorer health outcomes. Given both social participation and diminished cardiovascular reactivity are associated with poorer health outcomes (e.g., Bourassa et al., 2017; Croezen et al., 2015; Phillips et al., 2013; Sundquist et al., 2001), extending on this work to examine how engagement in social activities can influence cardiovascular habituation to recurring stress is warranted.

The aim of the present study was to (a) examine the association between CVR and social participation during a second stress task exposure, and (b) assess the relationship between social participation and cardiovascular
habituation across two acute psychological stress tasks, a method proposed to increase reliability (e.g., Kamarck et al., 1992; Kamarck & Lovallo, 2003). Based on previous research (John-Henderson et al., 2019), it was hypothesized that lower levels of social participation would be associated with lower cardiovascular reactivity. Given recent work found that cardiovascular responses to stress became less pronounced when accounting for motivation (Keogh et al., 2021) and social participation is a form of motivated behaviour, the current study hypothesized that higher levels of social participation would be related to greater habituation.

2. METHOD

2.1 Study overview

Data for the current study were drawn from the Pittsburgh Cold Study 3 (PCS3); a prospective viral challenge conducted by researchers from the Laboratory for the Study of Stress, Immunity, and Disease (2016) at Carnegie Mellon University. This laboratory is under the directorship of Sheldon Cohen, PhD and data was accessed via the Common Cold Project website (www.commoncoldproject.com; grant number NCCIH AT006694). The aim of the PCS3 was to expose participants to the rhinovirus 39 (RV39) that causes the common cold and evaluate the development of infection during a 5 day quarantine period. A novel feature of the PCS3 was inclusion of CVR assessment across two separate standardised stress testing sessions, while also obtaining psychological, social, behavioural and health measurements.

Data collection took place between 2007 and 2011; participants responded to an advertisement in a newspaper and then completed a telephone interview, followed by an in person health screening. Those who met any of the following criteria were excluded from participating; taking regular medications (e.g., antidepressants), a history of chronic illness/psychiatric disorder within 1 year of study enrolment, previous nasal surgery, prior hospitalization due to flu-like illness, hospitalization due to a psychiatric illness within the last 5 years, cold/flu-like symptoms 30 days prior to quarantine, pregnancy or planning to be pregnant within 3 months of study enrolment, use of steroids/immunosuppressants within 3 months of the trial, living with someone diagnosed with chronic obstructive pulmonary disease or immunosuppressant, human immunodeficiency virus seropositivity, abnormal clinical profiles observed via blood count, blood chemistry or urinalysis, those who participated in a study 30 days prior to enrolment that involved psychological questionnaires/investigational products or intend to while enrolled on the PCS3, and those allergic to eggs/egg products.

Healthy volunteers with viral specific antibody titers ≥ 4 were considered eligible for inclusion in the study. These participants completed biological assessments (e.g., saliva cortisol) and baseline psychological questionnaires and were administered nasal drops containing RV39 and followed for a brief 5 day quarantine to monitor the development of infection. Blood was collected approximately 28 days after exposure to the virus for serologic assessment. Participants were instructed to refrain from taking non-prescription medications and physical exercise 24h prior to the stress testing sessions as well as abstaining from alcohol for 48h, eating and drinking (except for water) for 2h and smoking 1h before attending the laboratory. Both oral and written consent were obtained prior to the initial telephone screening and both visits. Participants who completed the study received $1,000 for their participation, with an additional $60 to those who provided hair samples. The study was approved by the University of Pittsburgh and Carnegie Mellon University internal review boards.

2.2 Participants

The total sample included 213 participants from Pittsburgh, Pennsylvania who ranged in age from 18 to 55 (M = 30.13, SD = 10.85) years. The sample was predominantly Caucasian (66.7%) and consisted of 90 females. From the total sample, outliers deviating +/- 4 SD s from the mean on CVR parameters were also excluded (n = 7; Visit 1; n = 2; Visit 2). A further 14 were removed for missingness on physiological measures across the two visits (Visit 1 = 7, Visit 2 = 7). This left a total sample of 190 participants and demographic information for the sample can be found in Table 1.

[INSERT TABLE 1 HERE]
2.3 Material

2.3.1 Demographics

Self-report questionnaires were administered prior to the quarantine period to collect socioeconomic and demographic information (e.g., sex, age, educational attainment, employment status).

2.3.2 Social participation

Social participation was measured 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 2 months; 4 = once every two or three weeks; 5 = once a week; 6 = more than once a week) to measure how often in the past 12 months participants engaged in 16 social activities (e.g., went to movies, visited friends, attended a religious service). A total social participation score was calculated by summing all the items. The total scale achieved an overall $\alpha = .82$ for the current study and therefore demonstrated internal consistency.

2.4 Cardiovascular Assessment

The PCS3 included two stress task sessions with CVR findings for the first testing session being reported elsewhere (John-Henderson et al., 2019). Given that the aim of the current study was to examine cardiovascular habituation to recurring stress, data from both sessions were analysed, with CVR on Task 2 and cardiovascular habituation on all parameters being reported. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured in both sessions using an automated blood pressure cuff placed above the brachial artery of the non-dominant hand (Critikon Dynamap® Vital Signs Monitor 1846SX, GE Healthcare, U.S.). During the session a total of 28 readings were statically taken; four recordings initiated during the last six minutes of baseline (14, 16, 18, 20), nine taken every two minutes of the stress task (0, 2, 4, 6, 8, 10, 12, 14, 16) and fifteen recorded during the recovery period (8 readings every 2 min for the first 15 minutes and then every 5 minutes yielding another 7 readings).

2.5 Stress Task Procedure

The two separate laboratory visits were conducted in the University of Pittsburgh approximately 6 weeks apart. Each session lasted approximately 2 1/4 hr and occurred between the hours of 3pm and 9pm. Both stress testing sessions had identical protocols. At the start of each stress session, participants were asked a series of questions to determine that pre-testing instructions had been followed; they were also instructed to sit quietly and upright in a chair for a 20-min baseline period. Following the formal baseline period, participants completed a modified version of the Trier Social Stress Test (TSST; Kirschbaum et al., 1993) which consisted of a public speaking and a mental arithmetic task. For the speech task, participants were instructed that they would have 5 minutes to prepare a defense speech for an alleged transgression (i.e., traffic violation/shoplifting; counterbalanced across the two visits). They then had 5 minutes to deliver the speech which they were told was being recorded and evaluated. For the 5 minute mental arithmetic task, participants were instructed to subtract 13 from 1,022 or 1039 depending on the session and to continue subtracting 13 from subsequent answers as quickly and as accurately as possible. If subtraction errors were made, participants received instructions to start from the beginning.

2.6 Data analyses

Data were screened for outliers prior to analyses. Mean levels of SBP, DBP, and HR were computed across each phase in both stress testing sessions to yield baseline, task, and reactivity measures for cardiovascular parameters. Cardiovascular responses were averaged across the tasks as previously reported elsewhere (e.g., Tyra et al., 2020). Reactivity scores for each cardiovascular parameter were determined using the delta method (subtract the mean baseline from mean task scores for visit 1 and visit 2), with a total of six reactivity values were calculated (SBP, DBP, HR reactivity visit 1 and SBP, DBP, HR reactivity visit 2).
Cardiovascular habituation scores were determined as the difference in reactivity on cardiovascular parameters between the first and second task (reactivity Task 1 – reactivity Task 2), with a significant decrease in responses across exposures indicating habituation. A series of repeated-measures ANOVAs were used to examine if the task successfully elicited a stress response for both stress task 1 and 2. Additional repeated-measures ANOVAs were conducted to determine whether there were differences in CVR from the first to the second stress testing session (i.e., habituation).

Correlational analyses evaluated the unadjusted associations between parameters of CVR and social participation. Hierarchical multiple regression analyses were undertaken to examine the association between CVR and social participation at the second stress exposure. Social participation was entered as the dependent variable and the first model adjusted for baseline activity. The second model adjusted for confounds age, smoking status, and body mass index, which were selected based on associations with previously documented covariates (Creaven et al., 2020; John-Henderson et al., 2019). In the third model, a measure of high social roles was entered to ensure any observed effects were independent of social integration.

Lastly, hierarchical multiple regression analyses were conducted to examine the association between social participation and cardiovascular habituation scores (reactivity task 1 – reactivity task 2). In line with previous research (Creaven et al., 2020; John-Henderson et al., 2019), we first adjusted for respective baseline values, followed by the aforementioned confounds. All analyses were conducted using SPSS v28 (IBM Corp, USA).

3. Results
3.1 Descriptive statistics
Descriptive statistics of study variables are noted in Table 1.

3.2 Manipulation check
Results from several repeated measures ANOVAs (baseline, task) confirmed an increase in baseline to task on all cardiovascular parameters for SBP, \( F(1, 189) = 432.25, p < .001, \eta_p^2 = .696 \), DBP, \( F(1, 189) = 573.38, p < .001, \eta_p^2 = .752 \) and HR, \( F(1, 189) = 441.64, p < .001, \eta_p^2 = .700 \) for visit 1; and SBP, \( F(1, 189) = 426.77, p < .001, \eta_p^2 = .693 \), DBP, \( F(1, 189) = 607.93, p < .001, \eta_p^2 = .763 \) and HR, \( F(1, 189) = 338.20, p < .001, \eta_p^2 = .642 \) for visit 2. Changes in raw cardiovascular scores across the study phases are displayed in Table 2.

Further repeated measures ANOVAs revealed statistically significant changes in mean CVR between visit 1 and visit 2 for SBP, \( F(1, 189) = 7.65, p = .006, \eta_p^2 = .039 \), and DBP, \( F(1, 189) = 14.42, p < .001, \eta_p^2 = .071 \), but not for HR \( (p = .139) \). As can be seen in Table 3, there was a significant decrease in SBP reactivity and DBP reactivity from task 1 to task 2, indicating habituation of blood pressure responses across both repeated exposures to the stress task.

Given that this was a viral challenge study, a series of independent \( t \)-tests were also conducted to examine possible between group differences (exposed to virus vs. not exposed to virus) on CVR and habituation to stress. Analyses revealed no group differences in SBP, DBP or HR reactivity (all \( p s > .05 \)) or habituation from Visit 1 to Visit 2 for all CVR parameters (all \( p s > .05 \)).

3.3 Cardiovascular reactivity at visit 2 and social participation
Correlational analyses confirmed that lower social participation was related to lower CVR, for SBP, \( r(190) = .167, p = .021 \), and HR reactivity, \( r(190) = .248, p < .001 \) at Visit 2. Similarly, in hierarchical multiple regression, diminished SBP and HR reactivity were related to lower levels of social participation in model 1; no associations for DBP reactivity were
observed. The associations observed for HR reactivity withstood adjustment for confounds (age, smoking status, body mass index and baseline values) in model 2. However, this association did not withstand adjustment for further control variables in model 3. Information on full models can be found in Table 4.

3.4 Social participation and cardiovascular habituation

Correlational analyses indicated that social participation was positively associated with cardiovascular habituation for SBP, \( r(190) = .200, p = .006, \) and DBP \( r(190) = .238, p < .001; \) social participation was not associated with HR adaptation. Similarly, hierarchical multiple regressions controlling for the aforementioned confounds revealed that social participation was positively associated with SBP habituation, \( \beta = 0.223, t = 3.01, p = .003, \) and DBP, \( \beta = 0.273, t = 3.95, p < .001. \) As can be seen in Table 5, addition of social integration did not eradicate the observed effects, indicating that the reported associations are due to active engagement in social activities irrespective of high contact social roles.

4. Discussion

The primary aim of the current study was to extend previous work on CVR and social participation by examining the associations between these dimensions at multiple exposures to acute psychological stress. This study evaluated associations between (1) CVR to a non-novel stress task and social participation; and (2) explored the influence of social participation on cardiovascular habituation to recurrent stress.

As expected, lower CVR (SBP & HR) was evident to non-novel stress, which in turn was related to lower levels of social participation. For HR reactivity, this association remained significant in adjusted models controlling for baseline values, smoking status, age, and body mass index. These findings are in line with previous work examining CVR and elements of social connectedness that require active engagement (Creaven et al., 2020; John-Henderson et al., 2019) and support the suggestion that lower CVR may be a marker for motivational dysfunction.

Further, the findings indicate that higher levels of social participation were significantly associated with greater blood pressure (SBP & DBP) habituation on repeated exposure to stress. These associations withstood adjustment for several control variables (i.e., respective baseline cardiovascular activity, age, body mass index, smoking status, and a measure of social integration (i.e., total number of high contact social roles); indicating that the observed effects were due to active engagement in social activities rather than the number of social networks that individuals may have.

This is the first study to show that social participation, a form of motivated behaviour, is associated with distinct patterns of blood pressure (i.e., SBP, DBP) habituation to multiple exposures to repeated stress over time. The ability to habituate to the second stress exposure reflects a tolerance for stress and an ability to adapt, with potentially important health implications (Howard & Hughes, 2013; Hughes et al., 2018). Social participation protects against the negative impact of isolation (Kawachi & Berkman, 2000), is linked to improved health ratings, more support, sense of fulfilment, and community involvement (Lee et al., 2008). Lower levels of social participation are linked to increased number of chronic health conditions (Santini et al., 2020), and social connectedness is related to lower mortality risk (Holt-Lunstad, Smith, & Laylor, 2010). A key component of social connectedness is perceived social support, which may increase coping ability (Creaven et al., 2020).

The response profile observed in the current study is consistent with previous research that found social support was related to cardiovascular habituation (Howard & Hughes, 2012). Individuals who engage more in social activities may perceive having greater amounts of social support available to them if needed. It seems reasonable to suggest that greater participation in social activities, like high social support, is beneficial to health by buffering against the negative aspects of stress. Treatment programs targeting many of the health and behavioural correlates of CVR (e.g., high blood pressure, obesity, depression, smoking, substance
abuse) could benefit from exploring the contextual nature of motivation to increase buy-in or engagement in these programs.

One notable finding, unrelated to the construct of social participation, but important to note for reactivity research, was the absence of habituation for HR, across repeated exposure to the same stressor. Despite significant habituation of the blood pressure response, HR did not habituate. Heart rate showed equivalent elevations on exposure to task 1 as to task 2. This is notable, although given the nature of the stress task (speech), may not be entirely unsurprising. While the task itself involved similar stress-tasks (i.e., speech and mental arithmetic), the topic of the speech did vary between exposures, therefore could not be considered identical. An interesting paper from Trehub and Curran (1979) has shown that babies showed habituation of the cardiac response to syllables that remained constant across trials, but not to those that changed; perhaps subtle alterations in in speech task also prohibit the adaptation of the HR response to speech tasks of different topics.

This study has some notable strengths. First, we extend on previous research examining associations between CVR and social participation at the first stress exposure in the PCS3, to include analysis across multiple exposures to stress, and provide a more comprehensive representation of stress profiles. Second, to the best of our knowledge we are the first to examine the influence of social participation on cardiovascular habituation to acute psychological stress between sessions. Indeed, much of the research on adaptation of the cardiovascular stress-response to-date has focused on within-session habituation (Griffin & Howard, 2021; O’Súilleabháin, Howard, & Hughes, 2018a), or even within-task exposure (Liu et al., 2018; O’Súilleabháin, Howard, & Hughes, 2018b). However, this is one of the first studies to examine cardiovascular stress-response adaptation across multiple exposures that span weeks, not minutes. In this paradigm, habituation and social participation are related to several positive outcomes which are outlined above; and the current findings suggest that increased social connectedness may buffer individuals from the cardiotoxic effects of stress.

Notwithstanding these strengths, the present study is not without limitations. First, due to the observational nature of the study analyses, determination of causality cannot be inferred. Future research would benefit from a prospective approach to determine how patterns of cardiovascular adaptation predict health outcomes. In addition, a self-report measure of social participation was administered only once prior to the initial stress testing session, as such changes over time in social participation were not obtained. Findings may also reflect individuals who were motivated to participate in specific social contexts and not in others. However, the measure used represents engagement in a wide variety of both formal and informal social activities (e.g., work, education, religious and social events) across a 12 month period. It may be that individuals in the current study were motivated to engage socially, therefore future research may benefit from recruiting from populations characterised by deficits in motivation (e.g., depression) to examine the mechanistic influence of social participation in vulnerable populations. While associations were observed between social participation and HR reactivity when controlling for several confounds (i.e., age, smoking status, body mass index and baseline activity), statistically significant effects were not observed for HR habituation. However, notable effects remained for both SBP and DBP habituation even after controlling for confounds. Further, this study provided a comprehensive evaluation of patterns of cardiovascular adaptation, yet inclusion of hemodynamic factors (i.e., cardiac output, total peripheral resistance) would have provided a more detailed assessment, particularly in the context of the failure of HR to habituate to repeated exposure to the stressor. Lastly, given the inclusion criteria, the findings may be influenced by a sample comprised of healthy individuals. It is worth noting that the age of the sample recruited ($M = 30.13; SD = 10.85$), may be indicative of better health, with chronic conditions often considered normative in older samples (e.g., Keogh et al., 2022; Creaven et al., 2020). Nevertheless, many of the exclusion criteria applied included general restrictions (e.g., taking medications, pregnant) often applied in reactivity research (Keogh et al., 2021; O’Riordan et al., 2020).

To conclude, the present study provides confirmatory support that diminished cardiovascular stress responses
are associated with lower levels of social participation (John-Henderson et al., 2019). In addition, it provides a more comprehensive assessment of CVR across multiple stress exposures and extends our understanding of how social participation influences patterns of cardiovascular adaptation to recurrent stress. The findings demonstrate that individuals who actively participate in social activities show enhanced stress tolerance as evident by an adaptation of the cardiovascular stress-response; suggesting that a possible target for future health-promoting interventions may be to increase levels of social participation to promote better mental and physical health outcomes and enhance treatment success.

References


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Table 1.

**Participant demographics**

<table>
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<tr>
<th>Items</th>
<th>N</th>
<th>Mean (SD)</th>
<th>Range</th>
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<td>Age (years)</td>
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<td>BMI</td>
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<td>Doctoral</td>
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</table>

**Note:** N = 190 for all measures.

Table 2

Mean (SD) changes in cardiovascular parameters across the testing sessions.

<table>
<thead>
<tr>
<th></th>
<th>Baseline 1</th>
<th>Task 1</th>
<th>Baseline 2</th>
<th>Task 2</th>
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</thead>
<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td>114.77 (12.28)</td>
<td>130.25 (14.48)*</td>
<td>116.94 (12.27)</td>
<td>130.59 (14.83)*</td>
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<tr>
<td>DBP (mm Hg)</td>
<td>67.59 (7.97)</td>
<td>77.91 (8.13)*</td>
<td>68.70 (8.12)</td>
<td>77.50 (7.70)*</td>
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<tr>
<td>HR (bpm)</td>
<td>69.30 (10.78)</td>
<td>79.51 (12.20)*</td>
<td>71.47 (11.48)</td>
<td>81.03 (12.48)*</td>
</tr>
</tbody>
</table>

**Note:** SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate, bpm = beats per minute. * Significantly different from respective baseline values at p < .001.

Table 3

Cardiovascular reactivity at visit 1 and visit 2.

<table>
<thead>
<tr>
<th></th>
<th>Visit 1</th>
<th>Visit 1</th>
<th>Visit 2</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Range</td>
<td>Mean (SD)</td>
<td>Range</td>
<td>F</td>
<td>p</td>
<td>r_p^2</td>
</tr>
<tr>
<td>ΔSBP</td>
<td>15.48 (10.27)</td>
<td>-13.47-41.81</td>
<td>13.65 (9.10)</td>
<td>-10.33-41.47</td>
<td>7.66</td>
<td>.006*</td>
<td>.039</td>
</tr>
<tr>
<td>ΔDBP</td>
<td>10.33 (5.94)</td>
<td>-6.94-23.61</td>
<td>8.79 (4.92)</td>
<td>-4.36-23.031</td>
<td>14.42</td>
<td>&lt; .001*</td>
<td>.071</td>
</tr>
<tr>
<td>ΔHR</td>
<td>10.21 (6.70)</td>
<td>-4.64-29.72</td>
<td>9.57 (7.17)</td>
<td>-9.50-33.47</td>
<td>2.20</td>
<td>.139</td>
<td>.012</td>
</tr>
</tbody>
</table>

**Note:** Δ indicates difference between task and baseline (i.e., reactivity). SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate and * denotes significant difference in cardiovascular reactivity from respective baseline at p < .01.
Regression models for cardiovascular reactivity as a predictor of social participation.

**Stress Exposure 2**

<table>
<thead>
<tr>
<th></th>
<th>$\beta$</th>
<th>$t$</th>
<th>$p$</th>
<th>$R^2$</th>
<th>$R^2$ change</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP Reactivity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>.167</td>
<td>2.31</td>
<td>.022*</td>
<td>.028</td>
<td>.028</td>
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<tr>
<td>Model 2</td>
<td>.102</td>
<td>1.37</td>
<td>.173</td>
<td>.077</td>
<td>.009</td>
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<tr>
<td>Model 3</td>
<td>.043</td>
<td>.660</td>
<td>.510</td>
<td>.290</td>
<td>.002</td>
</tr>
<tr>
<td><strong>DBP Reactivity</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>.063</td>
<td>.801</td>
<td>.424</td>
<td>.021</td>
<td>.003</td>
</tr>
<tr>
<td>Model 2</td>
<td>.001</td>
<td>.017</td>
<td>.987</td>
<td>.070</td>
<td>.000</td>
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<tr>
<td>Model 3</td>
<td>.011</td>
<td>.164</td>
<td>.870</td>
<td>.285</td>
<td>.000</td>
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<td><strong>HR Reactivity</strong></td>
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<tr>
<td>Model 1</td>
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<td>3.25</td>
<td>.001*</td>
<td>.069</td>
<td>.053</td>
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<td>2.00</td>
<td>.047*</td>
<td>.100</td>
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<tr>
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<td>.111</td>
<td>1.62</td>
<td>.106</td>
<td>.305</td>
<td>.010</td>
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</table>

*Notes*: Model 1 includes respective baseline values; Model 2 additionally includes age, body mass index, smoking status; Model 3 additionally includes a measure of social integration. * Denotes significant effect.

$N = 190$ for all models.

Table 5

Regression models for social participation as a predictor for cardiovascular habituation.

<table>
<thead>
<tr>
<th></th>
<th>$\beta$</th>
<th>$t$</th>
<th>$p$</th>
<th>$R^2$</th>
<th>$R^2$ change</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP Habituation</strong></td>
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<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>.201</td>
<td>2.82</td>
<td>.005*</td>
<td>.050</td>
<td>.040</td>
</tr>
<tr>
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<td>.223</td>
<td>3.01</td>
<td>.003*</td>
<td>.059</td>
<td>.033</td>
</tr>
<tr>
<td>Model 3</td>
<td>.229</td>
<td>2.69</td>
<td>.008*</td>
<td>.059</td>
<td>.037</td>
</tr>
<tr>
<td><strong>DBP Habituation</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>.282</td>
<td>4.17</td>
<td>&lt;.001**</td>
<td>.163</td>
<td>.078</td>
</tr>
<tr>
<td>Model 2</td>
<td>.273</td>
<td>3.95</td>
<td>&lt;.001**</td>
<td>.182</td>
<td>.069</td>
</tr>
<tr>
<td>Model 3</td>
<td>.281</td>
<td>3.55</td>
<td>&lt;.001**</td>
<td>.182</td>
<td>.056</td>
</tr>
<tr>
<td><strong>HR Habituation</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
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<td>.926</td>
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<td>.000</td>
</tr>
</tbody>
</table>

*Notes*: Model 1 includes respective baseline values; Model 2 additionally includes age, body mass index, smoking status; Model 3 additionally includes measure of social integration. * Denotes significant effect at $p < .01$; ** at $p < .001$. $N = 190$ for all models.