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Social support, social participation, and cardiovascular reactivity to stress in the Midlife in the United States (MIDUS) study

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ABSTRACT

This study tested two hypotheses of associations between dimensions of social connectedness and cardiovascular reactivity to acute stress: (1) high social support predicts diminished cardiovascular responses to stress (i.e., the stress-buffering model of social support), and (2) diminished cardiovascular responses predict lower social participation, a form of motivated behaviour. Participants (N = 606) in the main Midlife in the United States study completed measures of social support and social participation and underwent psychophysiological stress testing. In unadjusted analyses, social support was positively, rather than inversely, associated with reactivity. Results withstood adjustment for several control variables, but not for depressive symptoms, which was associated with diminished reactivity. Further, diminished reactivity was associated with lower social participation, but not in fully adjusted models. No robust evidence was observed for either the stress-buffering model, or for an association between diminished reactivity and lower social participation. The implications for our understanding of links between social connectedness and cardiovascular reactivity are discussed.

1. Introduction

It is by now well-established that social connectedness has implications for health across the lifespan. Social connectedness reflects the extent to which one has meaningful, close, and constructive relationships with others (i.e., individuals, groups, and society) (O'Rourke & Sidani, 2017). This may include subjective perceptions of social relationships (e.g., relationship quality), as well objective or structural measures (e.g., volunteering; church attendance). Meta-analytic evidence indicates that high social connectedness is associated with reduced rates of mortality (Holt-Lunstad, Smith, & Layton, 2010), with effect sizes comparable to that of well-established lifestyle factors, such as smoking. Early studies found that associations were particularly salient for cardiovascular disease mortality, relative to other causes (Kawachi et al., 1996), leading to a focus on relevant biomarkers including cardiovascular reactivity (CVR). Cardiovascular reactivity refers to the arithmetic difference between baseline measures of cardiovascular function and the elevation in these brought about by a stressor (Allan & Scheidt, 1996). The usual function of this elevation is to prepare an organism to respond behaviourally to the stressor; however, behavioural readiness to engage with stressors is viewed as less relevant for challenges requiring psychological rather than physical responses (Hughes, 2013). Therefore, reactivity to psychological stress may involve a degree of cardiovascular responding beyond that which is metabolically necessary, with consequences for the development of cardiovascular disease (Carroll, Phillips, & Balanos, 2009; Obrist, 1981). Exaggerated CVR to stress has been consistently implicated in adverse cardiovascular outcomes (Chida & Steptoe, 2010), including hypertension (Carroll, Ginty, Painter et al., 2012) and cardiovascular disease mortality (Carroll, Ginty, Der et al., 2012). Unsurprisingly, then, identifying social connectedness factors that "buffer" individuals from exaggerated CVR has been a focus of substantial research.

1.1. Social support and the stress-buffering hypothesis

One key dimension of social connectedness is perceived social support, broadly conceptualized as the perception that a person is cared for and loved, esteemed, and a member of a network of mutual obligations (Cobb, 1976). The stress-buffering model proposes that during periods of high stress, perceiving support to be available reduces maladaptive responses to the stressor, by enhancing a person's perceived ability to cope, while the main effects model suggests that support is

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beneficial even in the absence of stress or challenge (Cohen & Wills, 1985; Uchino et al., 2018). Meta-analytic evidence suggests that laboratory-based social support lowers exaggerated CVR to stress (Thorsteinsson & James, 1999), while others report associations between perceived social support and lower resting cardiovascular function (Creaven, Howard, & Hughes, 2013), and ambulatory levels (Bowen et al., 2013, 2014).

Collectively, these findings underlie the stress-buffering model as applied to reactivity (i.e., the social support-reactivity hypothesis, which asserts that supportive others enhance health prospects by preventing or attenuating harmful physiological responses to stressors, such as excessive or prolonged CVR; [Lepore, 1998]). However, although support has been described as a "well-known buffer" of stress reactions (Uchino et al., 2018), a persistent thread of null and mixed findings has been observed. For example, using the Midlife in the United States (MIDUS) biomarker data, Lee and Way (2019) found the benefits of social support for reduced inflammation were restricted to participants with high self-esteem; for those with low self-esteem, no advantage was observed. Bowen et al. (2013) reported benefits for emotional and informational support in reducing ambulatory blood pressure, but not for tangible, belonging or global support. These mixed findings are consistent with qualitative research on coping with chronic stressors; for instance, several studies of patient samples describe support as partially beneficial but at times unwanted, overbearing, or ineffective (e.g., Brennan & Creaven, 2016; Habenicht, Gallagher, O'Keeffe, & Creaven, 2018).

Besides concerns relating to null or mixed effects, studies of perceived social support and stress reactivity are often characterized by methodological issues including restricted sample sizes or insufficient adjustment for confounding variables (Howard, Creaven, Hughes, O'Leary, & James, 2017). Further, social support-reactivity studies often operationalize support differently from those large-scale studies linking social connectedness to health. Much population-based evidence for the benefits of support relies on psychometric measures of perceived social support (rather than individual instances of enacted support, as used in laboratory settings). However, few studies have evaluated associations between perceived social support in everyday life (i.e., using psychometric measures) and stress reactivity. In one exploratory study, Howard et al. (2017) reported associations between perceived quality of social support and lower CVR in the laboratory, with no associations for perceived quantity of social support, again, illustrating mixed findings. In sum, given the mixed findings and relatively few studies evaluating psychometric measures of social support, large-scale studies evaluating the stress-buffering hypothesis as it pertains to social support and stress reactivity are needed.

1.2. Blunted cardiovascular reactivity

Besides these methodological issues, the social support-reactivity hypothesis is complicated by accumulating evidence that diminished, as well as exaggerated, cardiovascular responses to acute stress are linked to adverse health outcomes, including depression, obesity, and poorer self-reported health (Lovallo, 2011; Phillips, 2011; Phillips, Ginty, & Hughes, 2013), as well as lower cognitive functioning (Ginty, Phillips, Roseboom, Carroll, & deRooij, 2012). Additionally, diminished cardiovascular responses to stress are associated with other behaviours that may impact social interactions such as addiction, impulsivity, and low levels of perseverance (e.g., al'Absi, 2006; Bibbey, Phillips, Ginty, & Carroll, 2015; Evans et al., 2016; Bennett, Blissett, Carroll, & Ginty, 2014; Ginty, Brindle, & Carroll, 2015). Based on this body of work, both exaggerated and diminished responses are now viewed as maladaptive, with exaggerated responses implicated in the development of cardiovascular disease, while diminished responses are linked to other adverse health outcomes (Gianaros & Jennings, 2018; Lovallo, 2011; Phillips et al., 2013). Recently, diminished reactivity has been proposed as a marker of dysfunction of neural systems that underlie motivated and goal-directed behaviour, and is thus thought to be associated with outcomes that reflect variations in motivation and behavioural engagement (Carroll, Ginty, Whittaker, Lovallo, & de Rooij, 2017; Ginty, Kraynak, Fisher, & Gianaros, 2017). While this line of work does not directly apply to our understanding of the social support-reactivity hypothesis, at least one recent study is relevant. Using data from the Pittsburgh Cold Study 3, John-Henderson, Counts, Sanders, and Ginty (2019)) evaluated whether diminished reactivity predicted social participation (defined as frequency of engagement in 16 social activities such as visiting friends), a structural dimension of social connectedness involving a motivational component. This study found that diminished CVR was associated with lower social participation, following adjustment for pertinent demographic and lifestyle variables. Although intended to evaluate links between diminished reactivity predicting motivated behaviour, the findings, if replicated, indicate that reactivity serves as a marker for social participation, an important dimension of social connectedness.

1.3. Summary and hypotheses

In sum, arguments for the stress-buffering effects of social support on CVR are complicated by mixed or limited evidence for the overall theoretical model and methodological concerns relating to CVR research. Additionally, recent research suggests that diminished CVR may be a marker of reduced social participation (John-Henderson et al., 2019). To date, no studies have attempted to compare the social support-reactivity hypothesis (which suggests that high social support, a dimension of social connectedness, predicts lower CVR) with the more recent work examining the negative consequences of diminished CVR (i.e., John-Henderson et al. (2019) suggesting that lower CVR predicts low social participation, also a dimension of social connectedness), within the same sample. Thus, clarifying the association between these dimensions of social connectedness and CVR is warranted. This was the overall aim of the present study.

Two important hypotheses are identified. First, that perceived social support predicts "buffered" (i.e., lower) CVR to acute psychological stress. Second, that diminished CVR to stress predicts lower levels of social participation. Therefore, we sought to (a) evaluate the social support-reactivity hypothesis within a large-scale, midlife sample and (b) replicate the recent findings reporting associations between blunted reactivity and lower social participation (John-Henderson et al., 2019).

2. Methods

2.1. Study overview and design

In 1995–1996, the first wave of the MIDUS study (N = 7108) was conducted to investigate the role of behavioural, psychological, and social factors in understanding age-related differences in physical and mental health, using telephone interviews and self-administered questionnaires. Participants were contacted to take part in the second wave (MIDUS 2; N = 4963) in 2004, and the third wave (MIDUS 3; N = 3295) in 2013. Detailed information on retention rates is reported by Radler and Ryff (2010). MIDUS 2 participants were invited to complete an additional biological assessment, known as the Biomarker Project (N = 1054). The primary explanations given for non-participation in the Biomarker Project were that participants: (1) did not want to travel to the clinic, (2) had other family obligations (such as caregiving), (3) were too busy, or (4) were not interested (Love, Seeman, Weinstein, & Ryff, 2010); however, the sample were representative of the larger participant pool on the majority of demographic characteristics. Biomarker Project participants were more likely to have a college degree, and less likely to smoke (Love et al., 2010). Of the 1054 Biomarker Project participants, 945 were retained at MIDUS 3. The present study avails of data from Biomarker Project participants who completed the psychophysiology session and who were retained at MIDUS 3.

2.2. Participants

Biomarker Project participants ranged in age from 35 to 86 years (M = 58.04, SD = 11.62); 55 % were women (N = 477). There was no significant difference in age between men and women, $t(1052) = 1.68$, $p = .09$. Of 1054 participants, 970 attended the psychophysiology session. These participants were, on average, younger than those who did not complete the session (M = 57.79, SD = 11.57 years vs. M = 60.93, SD = 11.92 years; $p = .02$). The first 26 Biomarker Project participants completed an extended version of the stressors before the overall protocol was shortened (see Ryff, Seeman, & Weinstein, 2018).

Our sample for analysis is based on participants who undertook the brief form of the reactivity protocol, and who had sufficient cardiovascular data to calculate CVR (i.e., SBP, DBP, or HR data for at least one baseline epoch and one stressor epoch [i.e., participants with responses to either the Stroop or MATH were included]). Participants had complete data for our control variables (with the exception of medication use), and for total social support and social participation scores at both MIDUS 2 and at MIDUS 3. Given relatively high levels of missingness for medication use, a dummy variable was computed to indicate that cases were missing medication data, and these cases were retained for analysis. The final sample for analysis was 606.

2.3. Psychological stress testing

The stress-testing protocol ran for approximately 90 min and has been outlined in detail elsewhere (Coyle et al., 2020; Love et al., 2010). Testing took place in the morning. Participants sat quietly for an 11-minute resting baseline period, after which they undertook the first cognitive stress task lasting 6 min. This was followed by a 6-minute recovery period and then the second cognitive stress task also lasting 6 min. The psychological stressors were the Stroop colour/word interference task, and a mental arithmetic task, presented in random order. For the Stroop task, a word was presented on a computer screen, either of a congruent or incongruent colour (e.g., the word "yellow" written in yellow letters versus the word "yellow" written in blue letters). Participants used a keypad to respond to the answer that corresponds to the colour of the letters, rather than the colour name. The Morgan and Turner Hewitt (MATH; Turner et al., 1986) mental arithmetic task required participants to complete a number of addition and subtraction problems. An arithmetic problem was presented on the screen, followed by the word "equals" and an answer to the problem. Participants pressed a key (corresponding to yes or no) to indicate whether the answer was correct. The difficulty of problems varied across five levels. All participants began the task at level 3 and the difficulty level of each subsequent problem was determined by the accuracy of their response on the preceding trial. Participants rated their stress levels verbally on a scale from 1 (*not stressed at all*) to 10 (*extremely stressed*) at baseline, and once during each stressor.

2.4. Cardiovascular assessment

Heart rate (HR) was measured using a beat-to-beat electrocardiogram (ECG). Beat-to-beat analog ECG signals were collected and then digitized at a sampling rate of 500 Hz by a 16-bit National Instruments analog-to-digital (A/D) board installed in a microcomputer. In the MIDUS 2 dataset, HR is calculated as an average of all valid RR intervals for a specified length of time, and has been converted from RR interval units to beats per minute units. Both systolic and diastolic blood pressure (SBP; DBP) were recorded using a Finometer monitor (Finapres Medical Systems, Amsterdam, Netherlands), which accurately assesses absolute blood pressure (Schutte, Huisman, Van Rooyen, Oosthuizen, & Jerling, 2003) and meets the validation criteria of the Association for the Advancement of Medical Instrumentation. A finger cuff was placed on the middle finger of the non-dominant hand, and an arm cuff was placed on the upper arm on the same side.

2.5. Social support

For the social support variables, the respondent was asked how much (i) family members (ii) friends, and (iii) spouse (where relevant) "care about you", "understand the way you feel", and how much you can "rely on them", and "open up to them", with four response options: *not at all* (1), *a little* (2), *some* (3), *a lot* (4). For spousal support two additional items asking how much the spouse "appreciates you" and how much "you can be yourself" around him or her were also included. This resulted in three social support scales assessing support from family members, friends, and spouse. A social support score was calculated by taking the mean of the three support scales where participants had a valid score on at least two out of the three scales (as per Elliot, Heffner, Mooney, Moynihan, & Chapman, 2018). Therefore, being married did not automatically result in a higher social support score. Cronbach's α for this scale in MIDUS 2 was .86, and in MIDUS 3 was .84.

2.6. Social participation

Several items relating to social participation are included in MIDUS. Participants are asked to state how many times in a typical month they attend three types of activities (meetings of unions or other professional groups; meetings of sports or social groups; and meetings of any other groups [not including any required by their job]). Participants are also asked to report the hours per month they spend doing each of four types of formal volunteer work (hospital, nursing home, or other health-care-oriented volunteering; school or other youth-related volunteering; volunteering for political organizations or causes; and volunteering for any other organization, cause or charity). Participants are also asked how often they attend religious or spiritual services, and how often they attend/participate in church/temple activities (on a 6-point scale from *at least daily*, to *never*), resulting in 9 relevant social participation items. To generate a social participation score across items with different response options, we dichotomized responses to these items (i.e., attends each activity at least once per month vs. never attends; engages in each type of formal volunteering each month, vs. never engages in that type; and attends services and activities at least once per month vs. less often). A mean social participation score was generated for participants who provided a response on five or more of the nine items (cases with insufficient data were not retained for analysis). Cronbach's α for this scale in MIDUS 2 was .69, and in MIDUS 3 was .72.

2.7. Control variables

Control variables were selected a priori (see Harrell, 2000) based on well-established relationships with CVR and cardiovascular health. Race was coded as White or non-White. Education was used as a proxy for socio-economic status and was coded as 'high school or less', 'some college' and 'college degree or higher' (at MIDUS 2). Medication was coded as taking, not taking,or missing data for taking medication for high blood pressure/hypertension.

The remaining control variables were assessed at the time of biological data collection. Participants' heights and weights were measured, and body mass index (BMI) computed by dividing weight by height squared. Smoking status was coded as smoker or non-smoker (former smokers were categorized as non-smokers). Given associations between depression and both social participation (Croezen, Avendano, Burdorf, & van Lenthe, 2015) and CVR (Carroll, Phillips, Hunt, & Der, 2007; Phillips, 2011), depressive symptoms were included. These were measured using the 20-item Centre for Epidemiological Studies Depression Inventory (CESD). Sample items include "I was bothered by things that usually don't bother me" and "I did not feel like eating; my appetite was poor" measured on a scale from 0 *(Rarely or none of the time)* to 3 (*Most or all of the time)* (Cronbach's α = .88).

2.8. Statistical analyses

Data were analysed using SPSS (IBM, version 24, SPSS Inc., Chicago, IL, USA). Cardiovascular reactivity was computed as the difference between average baseline measures and average stress measures (across the two stress tasks, where data for both were available). Reactivity to the two stress tasks were averaged based on previous research suggesting that averaging across stress measures increases reliability and generalisability of results (Kamarck & Lovallo, 2003; Kamarck et al., 1992; Kamarck, Jennings, Stewart, & Eddy, 1993). Linear regression analyses evaluated associations between social support at MIDUS 2 and CVR as assessed in the MIDUS 2 Biomarker Project. This analysis represented the optimal test of the social support-reactivity hypothesis. It was hypothesised that higher levels of social support would predict lower levels of CVR. Linear regression analyses were also employed to evaluate associations between stress reactivity (in the Biomarker Project of MIDUS 2) and social participation (at both MIDUS 2 and at MIDUS 3 – to evaluate CVR as a predictor of social participation). It was hypothesised that lower levels of CVR would be associated with lower levels of social participation.

In line with the approach adopted by John-Henderson et al. (2019), we first adjusted for baseline cardiovascular activity, and then for other control variables (demographic variables and depression). Multi-collinearity of the predictors was analysed using tolerance statistics and the variation inflation factor (VIF). Tolerance statistics < .20 and/or VIF of five and above indicate a multi-collinearity problem (O'Brien, 2007). In the present study, the tolerance statistics for final adjusted models ranged from .70 to .98, with the VIF ranging from 1.02 to 1.42, indicating that there were no problems with multi-collinearity. Associations were regarded as statistically significant at $p < .05$. The findings of our final models did not change when analyses were repeated excluding extreme values identified for SBP, DBP, and HR reactivity; therefore, analyses using the larger sample are reported.

3. Results

Descriptive statistics for study variables are reported in Table 1 and correlations are reported in Table 2. At the time of participation in MIDUS 2, the majority of our sample (76.6 %) reported having experienced at least one chronic health condition in the past 12 months. Of those chronic conditions reported, those occurring most frequently were high blood pressure/hypertension (23.1 %), joint/bone disease (22.3 %) and anxiety/depression (19.3 %). Women reported significantly greater numbers of chronic health conditions ($p < .001$), as well as marginally higher social support at MIDUS 2 and MIDUS 3, and social participation at MIDUS 3 (all $ps < .05$). Women were less likely to have completed a college degree (47 % vs. 57 %; $p = .008$). Social support and social participation measures in MIDUS 2 and MIDUS 3 were positively inter-correlated, and each was inversely correlated with depressive symptoms. Depressive symptoms were also positively correlated with BMI and number of chronic health conditions, and inversely correlated with age, and with each of SBP, DBP, and HR reactivity (i.e., depressive symptoms were associated with blunted reactivity). Chronic health conditions were inversely associated with social support, and with social participation in MIDUS 2 (but not in MIDUS 3).

One-way ANOVAs indicated that social participation varied by education level at MIDUS 2, $F(2, 603) = 20.07$, $p < .001$, $\eta^2 = 0.06$, and at MIDUS 3, $F(2, 603) = 13.55$, $p < .001$, $\eta^2 = 0.04$. For both waves, Tukey post-hoc tests showed that participants with 'college degree or higher' reported greater social participation ($M = .39$, $SD = .28$; $M = .37$, $SD = .27$, respectively) than those with 'high school or less' ($M = .24$, $SD = .23$; $M = .24$, $SD = .22$), or "some college education" ($M = .27$; $SD = .24$; $M = .27$, $SD = .27$) ($ps < .001$).

Table 1
Characteristics of final sample for analysis ($N = 606$).

Variable (measurement scale or reference category)	*M (SD)*	*n*	%
Age (years)	56.05 (10.28)		
Sex (women)		329	54.3
Race			
White		562	92.7
Black and/or African American		17	2.8
Other		16	2.6
Native American or Alaska Native Aleutian Islander/Eskimo		9	1.5
Asian		2	.3
Number of chronic health conditions	2.11 (2.00)		
Body mass index (kg / m2)	29.12 (5.78)		
Education		124	20.5
High school or less			
Some college education		168	27.7
Minimum college degree		314	51.8
Smoking (current smoker)		64	10.6
Medication for hypertension or high blood pressure			
Yes		131	21.6
No		408	67.3
Missing		67	11.1
Depressive symptoms	7.28 (7.15)		
	MIDUS 2	*MIDUS 3*	
Social support - *M (SD)*	3.49 (.45)	3.48 (.44)	
Social participation - *M (SD)*	.33 (.26)	.31(.27)	
Volunteering (%)			
Healthcare-oriented	10.9	11.1	
School or youth-oriented	26.8	21.8	
Political organisations	8.1	8.7	
Other	45.8	45.5	

Note: Age, body mass index, smoking, and depressive symptoms are reported at the time of participation in the Biomarker Project of MIDUS 2.

3.1. Cardiovascular reactivity manipulation check

Repeated measures (baseline, mean reactivity across stress tasks) ANOVAs confirmed that the stress tasks increased cardiovascular responses for SBP, $F(1, 605) = 1049.80$, $p < .001$, $\eta_p^2 = .63$; DBP, $F(1, 605) = 1574.12$, $p < .001$, $\eta_p^2 = .72$; and HR, $F(1, 605) = 571.76$, $p < .001$, $\eta_p^2 = .49$ (see Table 3).

3.2. Testing the stress-buffering hypothesis: social support predicting CVR

Social support, assessed in MIDUS 2, was positively associated with SBP and DBP reactivity, but not HR reactivity, when adjusting for respective baseline cardiovascular activity (see Table 4). With adjustment for additional control variables (i.e., sex, race, BMI, education, medication, and smoking status) this association remained significant for SBP reactivity, only. However, this association did not withstand additional adjustment for depressive symptoms. A number of control variables predicted CVR; in the overall models, higher depressive symptoms was associated with lower DBP reactivity ($\beta = -.12$, $t = -2.86$, $p = .004$); though not lower SBP reactivity ($\beta = -.08$, $t = -1.94$, $p = .053$). Age was positively associated with both SBP and DBP reactivity, and sex and smoking status were significant predictors of SBP (see supplementary material).

3.3. CVR predicting social participation

Next, we tested CVR in the Biomarker Project of MIDUS 2 as a predictor of social participation in MIDUS 2, and also in MIDUS 3. For MIDUS 2, diminished SBP and DBP reactivity were associated with lower social participation in Model 1; no association for HR reactivity was observed. However, these associations did not withstand adjustment for additional control variables in Model 2, 3, or 4. No associations were observed between CVR and social participation in MIDUS 3 (Table 5; see supplementary material for full models).

Table 2
Correlations between main variables of interest ($N = 606$).

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Age	−.02	−.06	−.04	−.15**	.06	−.06	.14**	.14**	.12**	.06	.24**	.17**	−.10*
2. Sex	–	−.01	−.04	.03	.18**	−.02	.10*	.08*	.08	.08*	−.11**	−.01	.01
3. Race	–	–	.06	−.01	−.04	−.01	−.07	−.06	−.06	−.02	.02	.00	.01
4. BMI	–	–	–	.10*	.21**	−.05	−.10	−.08	−.06	−.03	−.00	−.06	−.06
5. Depressive symptoms	–	–	–	–	.26**	.09*	−.32**	−.32**	−.19**	−.16**	−.15**	−.17**	−.10*
6. No. of chronic conditions	–	–	–	–	–	.04	−.13**	−.09*	−.08*	−.02	−.08*	−.12**	−.10*
7. Smoking status	–	–	–	–	–	–	.01	−.09*	−.18**	−.20**	−.16**	−.09*	−.06
8. Social support (MIDUS 2)	–	–	–	–	–	–	–	.59**	.23**	.21**	.10*	.09*	−.01
9. Social support (MIDUS 3)	–	–	–	–	–	–	–	–	.19**	.18**	.08	.06	.00
10. Social participation (MIDUS 2)	–	–	–	–	–	–	–	–	–	.68**	.11**	.10*	.06
11. Social participation (MIDUS 3)	–	–	–	–	–	–	–	–	–	–	.03	.04	.02
12. SBP reactivity	–	–	–	–	–	–	–	–	–	–	–	.81**	.24**
13. DBP reactivity	–	–	–	–	–	–	–	–	–	–	–	–	.35**
14. HR reactivity	–	–	–	–	–	–	–	–	–	–	–	–	–

Notes. $^{**}p < .001$; $^{*}p < .05$. Sex: male = 0, female = 1. Race: white = 0, other = 1. Smoking status: never = 0, smoker = 1.

Table 3
Mean (*SD*) cardiovascular activity at baseline and during exposure to the stress tasks.

	SBP (mm Hg)	DBP (mm Hg)	HR (bpm)
Baseline	123.40 (18.46)	61.09 (11.84)	72.93 (10.70)
Stressor			
Average	138.02 (21.71)	67.78 (12.35)	76.79 (11.24)
Stroop	140.01 (22.34)	68.67 (12.43)	77.41 (11.47)
MATH	135.72 (21.77)	66.95 (12.46)	76.21 (11.23)

For the Stroop; $n = 573$ for SBP and DBP, and $n = 595$ for HR. For the MATH, $n = 592$ for SBP and DBP, and $N = 606$ for HR.

Table 4
Regression models for social support in MIDUS 2 as a predictor of cardiovascular reactivity ($N = 606$).

	β	*t*	*P*	R^2	R^2 change
SBP reactivity					
Model 1	.10	2.52	.012	.011	.010
Model 2	.08	2.11	.035	.108	.007
Model 3	.06	1.40	.161	.114	.003
DBP reactivity					
Model 1	.09	2.28	.023	.011	.009
Model 2	.07	1.79	.074	.050	.005
Model 3	.04	.82	.411	.063	.001
HR reactivity					
Model 1	−.01	−.23	.817	.002	< .001
Model 2	.00	−.00	.998	.027	< .001
Model 3	−.03	−.77	.441	.037	.001

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

4. Discussion

This study evaluated associations between (1) perceived social support and CVR to acute stress and (2) CVR and subsequent social participation, in a large, midlife sample. Notably, the majority of the sample reported at least one chronic health condition. However, given chronic health conditions were in effect, normative for this age group, it was not feasible to include only participants with no chronic conditions in our analyses, and adjustment for these in our full models did not alter the results observed. In terms of cardiovascular health, our sample could be considered generally healthy, with mean resting blood pressure and HR in the "normal" (i.e., non-hypertensive) range. However, epidemiological data indicates that cardiovascular disease risk increases incrementally with the blood pressure even within this "normal" range (Lewington et al., 2002). Drawing on this sample, our study contributes to the literature by comparing two models of associations between social connectedness and CVR, specifically, the stress-buffering model of social support, and the more recent model described by Carroll et al. (2017) and tested by John-Henderson et al. (2019), linking blunted reactivity with lower social participation.

Overall, perceived social support was associated with exaggerated, rather than buffered, reactivity in our models, prior to adjustment for depressive symptoms. The findings are arguably more consistent with the dual-effects model of social support (Teoh & Hilmert, 2018) rather than the stress-buffering model. This dual-effects model proposes that in some contexts, social support encourage the recipient to engage with stressors (Teoh & Hilmert, 2018) resulting in increased reactivity to those stressors. However, given stressor engagement was not explicitly assessed in MIDUS, this interpretation cannot be confirmed here. Rather than discard the stress-buffering hypothesis entirely, it is possible that in some contexts, actually enacted support (rather than global perceptions of support availability) can be effective in buffering stress responses (e.g., Thorsteinsson & James, 1999). In addition, perceived social support may be beneficial for subgroups of participants. For example, Lee and Way (2019) reported benefits for inflammation for participants high in self-esteem. However, the assumption that perceived social support exerts a stress-buffering effect for CVR, should not be universally applied, and as noted earlier, is inconsistent with a large body of qualitative work indicating mixed benefit for support for coping with chronic stressors such as illness (Brennan & Creaven, 2016; Habenicht et al., 2018). Moreover, as an optimal level of reactivity to challenge has not yet been established, interpreting a given response as "buffered" by social support, rather than simply "diminished" (and unaffected by social support), remains somewhat problematic.

The second aim of our study was to replicate and extend the recent findings linking blunted CVR to lower social participation (John-Henderson et al., 2019), drawing on the larger sample available from MIDUS. Diminished SBP and DBP were associated with lower social participation in unadjusted models, consistent with prior findings; however, these findings did not withstand adjustment for our full list of control variables, regardless of whether or not depressive symptoms were included. Importantly, John-Henderson et al. (2019) examined associations between CVR and social participation using social participation data collected 6 weeks prior to the stress protocol. Although the Pittsburgh Cold Study 3 offered an excellent test of the association between CVR and social participation within a relatively short timeframe, our prospective data (particularly, reactivity in the Biomarker Project of MIDUS 2 as a predictor of MIDUS 3 social participation) is a better test of whether blunted reactivity is a precursor to lower levels of social participation. Our findings suggest that this is not the case, at least, over the timespan assessed in MIDUS. This may partially explain divergent findings between our study and the Pittsburgh Cold Study

Table 5
Regression models for cardiovascular reactivity as a predictor of social participation (N = 606).

	MIDUS 2					MIDUS 3				
	β	*t*	*p*	R^2	R^2 change	β	*t*	*p*	R^2	R^2 change
SBP reactivity										
Model 1	.11	2.80	.005	.014	.013	.03	.64	.520	.001	.001
Model 2	.07	1.78	.075	.124	.005	−.02	−.37	.710	.088	< .001
Model 3	.06	1.42	.157	.143	.003	−.03	−.72	.471	.105	.001
Model 4	.05	1.19	.233	.167	.002	−.03	−.76	.449	.120	.001
DBP reactivity										
Model 1	.09	2.31	.021	.010	.009	.04	.98	.325	.002	.002
Model 2	.07	1.81	.070	.123	.005	.02	.47	.637	.088	< .001
Model 3	.05	1.34	.182	.142	.003	.00	.03	.976	.105	< .001
Model 4	.05	1.21	.225	.166	.002	.00	.07	.942	.119	< .001
HR reactivity										
Model 1	.06	1.38	.167	.003	.003	.02	.39	.693	.002	< .001
Model 2	.05	1.30	.194	.122	.003	.00	.07	.948	.093	< .001
Model 3	.04	.96	.339	.141	.001	−.01	−.25	.802	.109	< .001
Model 4	.04	1.10	.271	.166	.002	−.00	−.16	.871	.123	< .001

Notes: Model 1 includes respective baseline cardiovascular activity; Model 2 additionally includes age, sex, race, body mass index, education, medication, and smoking status; Model 3 additionally includes depressive symptoms; Model 4 additionally includes social support for the corresponding wave.

data. Notably, while John-Henderson et al. (2019) also controlled for depressive symptoms using a questionnaire measuring trait affect, our study used a validated measure of depressive symptomology. However, given the social participation-reactivity associations were eliminated in models adjusted for variables besides depression, this is not a likely explanation for divergent findings.

Importantly, as suggested by an anonymous reviewer, the associations studied here may have been reduced by including apparent confounders that in themselves have a true place in the relationships under investigation. For example, if depressive symptoms are associated with poor mood regulation, a potential correlate of blunted CVR, then controlling for depressive symptoms may remove true variance from the relationships under study. Similar arguments can be made for risky health behaviours (e.g., smoking), traditionally regarded as covariates in studies of exaggerated reactivity and cardiovascular outcomes, and now viewed as outcome variables in studies of blunted reactivity and motivational and behavioural (dis)engagement. Therefore, the curvilinear (rather than linear) model of associations between CVR and health outcomes poses challenges to our interpretation of CVR study findings; in this instance, to the interpretation of associations between CVR, social connectedness, and motivational and behavioural factors related to both of these key variables.

The third aim of our study was to consider links between reactivity, social support and social participation within the same sample. In our study (and in others), social support and social participation were positively inter-correlated. Although support and participation can be conceptualized as dimensions of social connectedness (and were positively inter-correlated in MIDUS) evidence for their discriminant validity is noted. Number of chronic health conditions was inversely associated with perceptions of social support, but not with social participation. Individuals with high need for support, owing to their poorer physical health status, may have higher expectations for support, placing them in a position where they are more likely to perceive their social support as inadequate. In contrast, social participation is not especially reliant on one's perceptions of social relationships. Although engagement in some activities such as volunteering may depend on one's health status, attendance at religious services or church activities is less likely to be compromised in the same manner. Substitutions for previous forms of social participation are arguably more feasible than replacements for intimate social relationships. Conceptually, social support may be viewed as a functional measure of connectedness, while social participation (and integration) comprise structural measures. Overall, then, it appears that social support and social participation merit consideration as distinct variables in social connectedness-reactivity research, consistent with prior arguments for the separability of functional and structural measures (Holt-Lunstad et al., 2010). This distinction is also important in the context of interventions to enhance social connectedness, with the aim of reducing loneliness and improving health. Although further research is needed, the null associations observed here raise questions about the importance of CVR as a mechanism linking social connectedness to health, at least for this midlife sample.

4.1. Limitations

We generated a social participation score using several individual items available from MIDUS; however, a comprehensively validated measure would have been preferable. Both social support and social participation data were collected via self-report, the optimal method of assessing *perceptions* of social support. Self-report data is arguably less optimal for social participation, where recall bias may influence people's reports of engagement in specific activities. However, it seems unlikely that participants would have difficulty recalling events such as attendance at religious services, for example. A further limitation relates to the study stressors; the stress tasks included in MIDUS can both be viewed as asocial, non-evaluative, stress tasks. This may explain the relatively muted cardiovascular reactions observed in response to these tasks, in comparison to other large-scale studies (e.g., the Dutch Famine Birth Cohort study; de Rooij, 2013). Importantly, this has implications for our test of the stress-buffering hypothesis, as the degree of stress experienced by participants, may have been relatively low, obscuring the potential for support to be of benefit under conditions of high stress. However, given the positive relationship between support and stress reactivity (i.e., the reverse of what is expected based on the stress-buffering model) this remains unlikely. Further, while we evaluated associations against the conventional p value of < .05, rather than a more stringent value, associations between social variables and reactivity were not significant in final models, in any case. Finally, the generalizability of our findings is limited owing to attrition between MIDUS 2 and MIDUS 3, as well as some degree of missing data for our variables of interest.

4.2. Future research

In terms of future research, including assessments of both social support and social participation may help clarify the associations between social connectedness and stress reactivity, in the context of competing theoretical models. In light of recent discussions on the

validity of psychometric measures (Hussey & Hughes, 2020), alternative analytical approaches should also be considered to compare reactivity across "clusters" of participants differing in social connectedness or participation. Given recent models such as the dual-effects model (Teoh & Hilmert, 2018), reactivity studies in this area should optimally include some measure of stressor engagement. In addition, comparisons of cross-sectional and longitudinal research would enhance our understanding of whether diminished reactivity only co-occurs with lower levels of social participation or whether it predicts social participation prospectively, particularly given blunted reactivity has been identified in longitudinal studies as a predictor of other key outcomes such as depression (Phillips, Hunt, Der, & Carroll, 2011). Aside from research with a social connectedness focus, additional studies evaluating links between reactivity and other measures of motivated behaviour may clarify the extent to which blunted reactivity reflects motivation dysregulation, as proposed by Carroll et al. (2017).

In sum, the present research demonstrated that neither the stress-buffering nor main effects models adequately capture the overall relationship between social support and CVR in the main MIDUS sample. Further, limited evidence for a prospective association between blunted reactivity and diminished social participation did not withstand comprehensive adjustment for control variables. Studies evaluating associations between social connectedness and stress-related variables should incorporate assessment of depressive symptoms, to determine the subgroups and contexts for which social connectedness is meaningful for health outcomes. Further, identifying which components of support are most effective in buffering stress, and for whom, may further clarify the extent to which the social support-reactivity hypothesis is a useful extension of the stress-buffering model.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.biopsycho.2020.107921.

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