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Heart rate reactivity mediates the relationship between trait gratitude and acute myocardial infarction



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ARTICLE INFO	A B S T R A C T
Keywords: Gratitude Cardiovascular Stress Heart rate reactivity Cardiovascular disease Myocardial infarction MIDUS	<i>Objective:</i> This study examines the relationship between trait gratitude and acute myocardial infarction. A burgeoning body of literature suggests that gratitude can play a role in regulating individual's cardiovascular responses to stress which in turn, may reduce the incidence of cardiovascular disease such as acute myocardial infarction. However, to date no research has examined these effects. <i>Method:</i> This study used the Mid-Life in the United States dataset (MIDUS; N = 1031) to assess these relationships. Participants completed a standardised cardiovascular stress-testing laboratory protocol and were assessed at a second time-point; on average 6.7 years later. <i>Results:</i> Results from logistic parallel mediation models suggest that trait gratitude was found to be significantly associated with reduced risk of acute myocardial infarction through the mechanism of increased heart rate reactivity mediated this relationship. <i>Conclusions:</i> These findings suggest that gratitude may be associated with certain aspects of physical health. Specifically, our study reveals a potential link between gratitude and cardiovascular reactivity, which could be a mechanism through which trait gratitude contributes to reductions in the risk of myocardial infarction. As such, this study highlights the potential utility of positive psychological factors, such as gratitude, in promoting cardiovascular health.

1. Evaluating the cardiovascular stress buffering effects of trait gratitude

Myocardial infarctions, also known as heart attacks, are defined by the WHO as the "demonstration of myocardial cell necrosis due to significant and sustained ischaemia" (Mendis et al., 2011). In the United States, it is estimated that 660,000 patients suffer heart attacks for the first time each year and that 1 in 7 deaths are due to acute myocardial infarction (Mozaffarian et al., 2016; Smilowitz et al., 2017). Furthermore, the rate at which myocardial infarction occurs has also been found to be growing (Kuhn et al., 2022), leading researchers to call for cost-effective policies and interventions in order to meet the UN's goal of reducing premature mortality due to non-communicable deaths by a third (Roth et al., 2020).

Positive psychological constructs such as optimism, purpose in life, and positive thoughts (Boehm & Kubzansky, 2012) have been identified as potential low-cost areas of intervention that have positive

associations with cardiovascular health (Boehm, 2021; Celano et al., 2017; Kubzansky et al., 2018; Park et al., 2016; Sin, 2016). Similarly, gratitude has been identified as a potentially useful area of intervention (Gallagher et al., 2020a). Gratitude can be conceptualized at both state and trait levels (Wood et al., 2010). As a state, gratitude refers to momentary feelings of appreciation for the good things one has in their life. As a trait, gratitude refers to a predisposition to notice and appreciate what is good in the world (Wood et al., 2010). The potential value of gratitude lies in it being a straight-forward, low-cost, and clinically usable intervention (Boggiss et al., 2020; Wood et al., 2010). Recent research has found evidence that gratitude can play a role in cardiovascular health (Cousin et al., 2021; Redwine et al., 2016), and in modulating the cardiovascular response to acute stress (Cousin et al., 2021; Gallagher et al., 2020; Leavy et al., 2023).

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1.1. Stress buffering and cardiovascular reactivity

One pathway by which positive constructs may influence health is by buffering the negative effects of psychological or perceived stress (Boehm et al., 2011). This is called the stress-buffering hypothesis, and it proposes that positive emotions can mitigate negative reactions to stress, and thus protect individuals from the potential deleterious effects of stressful events (Fredrickson et al., 2000; Gellert et al., 2018; Pressman et al., 2019).

Stress is a major risk factor in the development of cardiovascular illness (Phillips & Hughes, 2011; Steptoe & Kivimäki, 2012), comparable to risks associated with obesity and hypertension (Osborne et al., 2020). In the context of stress and cardiovascular health, it is important to consider cardiovascular reactivity due to its well-established relationship with cardiovascular disease (Phillips & Hughes, 2011). Cardiovascular reactivity refers to the magnitude of the change between an individual's baseline cardiovascular state and their state during acute psychological stress (Carroll et al., 2012). Until recently, research predominantly considered heightened cardiovascular reactions to stress to be associated with increased risk of onset and progression of cardiovascular disease (Hughes & Lü, 2017; Phillips & Hughes, 2011). For example, exaggerated cardiovascular reactivity has been associated with negative health outcomes such as atherosclerosis, hypertension, and coronary heath disease mortality (Carroll et al., 2012; Hocking Schuler, O'brien, 1997; Jennings et al., 2004; Phillips & Hughes, 2011; Yuenyongchaiwat, 2015) and myocardial infarction (Canto et al., 2012; Krantz et al., 1991; Manuck et al., 1992; Sundin et al., 1995).

However, recent research has suggested that blunted or 'too low' reactivity can also be associated with a range of adverse outcomes (O'Riordan et al., 2022). For example, in individuals with poorer cardiovascular health, blunted reactivity predicts a range of adverse cardiovascular outcomes such as cardiac arrest, cardiovascular hospitalization and death, angina, and myocardial infarction (Ahern et al., 1990; Kupper et al., 2015; Sherwood et al., 2017). One posited explanation for these relationships is that lower reactivity reflects the inability of the cardiovascular system to produce an appropriate response, which may be due to a pre-existing condition, for example (O' Riordan et al., 2022).

1.2. Gratitude and cardiovascular reactivity

Gratitude has been associated with coping more successfully with stress and adversity (Wood et al., 2010), and gratitude expressions are positively related to emotional regulation strategies such as reappraisal (Bryan et al., 2018) as well as goal-directed activities, which reduce the frequency and intensity of stress (Wood et al., 2007). Research has only recently begun to investigate physiological aspects of these relationships, with research to date showing that state gratitude has a significant, inverse relationship with cardiovascular reactivity (Gallagher et al., 2020; Ginty et al., 2020). As heightened reactivity has traditionally been associated with poorer cardiovascular outcomes (Carroll et al., 2012) - although as we have discussed, blunted reactivity can be also this suggests that gratitude may play a protective role for physical health. This association is consistent with the cognitive model of stress which posits that an individual's internal resources and characteristics influence how one copes and manages with stress (Folkman & Lazarus, 1988), with positive emotions playing an important, restorative role in this model (Folkman, 2008).

Thus, gratitude's relationship with lower stress has been proposed as a potential mechanism by which gratitude is indirectly associated with cardiovascular health (Schache et al., 2019). Gratitude may not have a direct relationship with cardiovascular health, but rather operate through mechanisms such as improving health behaviours, improving physiological functioning, and buffering the negative effects of stress on physical health (Boehm, 2021; Schache et al., 2019; Soo et al., 2018).

1.3. Trait gratitude and cardiovascular health

Although three studies have shown that *state* gratitude has an inverse relationship with cardiovascular reactivity (Gallagher et al., 2020a; Ginty et al., 2020; Leavy et al., 2023), a recent psychophysiological study found an association between *trait* gratitude and an increase in reactivity (Gallagher et al., 2021). As there is an established relationship between increased reactivity and cardiovascular disease (Carroll et al., 2012), this may lead to the confusing claim that trait gratitude may actually be worsening cardiovascular health. The inconsistencies in previous research may reflect the more recent discovery that both blunted reactivity – too low – and exaggerated reactivity – too high – may result in poorer health outcomes (O' Riordan et al., 2022; Whittaker et al., 2021).

Nonetheless, it has yet to be established whether the increase in reactivity associated with *trait* gratitude has any relationship with cardiovascular health outcomes. Our research explored this by examining the prospective, indirect relationship between trait gratitude, reactivity and myocardial infarctions. Myocardial infarction is a serious health problem which causes substantial morbidity and mortality (Chi & Kloner, 2003), with studies estimating that the a significant portion of sudden deaths globally are caused by myocardial infarctions (Solomon et al., 2005; Zaman & Kovoor, 2014). Exploring how gratitude may be associated with reductions in the likelihood of the occurrence of myocardial infarctions contributes to programmes of research in both preventative cardiology and positive psychology which examine how positive psychological constructs such as gratitude can cultivate cardiovascular health, including the occurrence of myocardial infarctions (Kubzansky et al., 2018; Labarthe et al., 2016).

Our research aimed to clarify whether trait gratitude is indirectly associated with cardiovascular health through reactivity. We evaluated the claim that gratitude, as a positive emotion, is statistically associated with a lower rate of myocardial infarction through its capacity to modulate blood pressure reactivity and heart rate reactivity (Cousin et al., 2021; Gallagher et al., 2020b; Schache et al., 2019). Thus, our study is novel in that we test these cardiovascular reactivity pathways to understand whether trait gratitude is associated with the likelihood of suffering acute myocardial infarction. Moreover, we use a longitudinal study design and evaluate the occurrence of myocardial infarctions as an outcome. In so doing, we hope to clarify how increases in reactivity associated with trait gratitude are related to the occurrence of acute myocardial infarctions. As such, this study proposes:

Hypothesis 1. Systolic blood pressure reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

Hypothesis 2. Diastolic blood pressure reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

Hypothesis 3. Heart rate reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

2. Method

2.1. Study overview and design

This study made use of the publicly available Mid-life in the United States study (referred to as MIDUS) dataset (Radler, 2014). Between 1995 and 1996 the first wave of the Mid-life in the United States study was carried out using telephone interviews and questionnaires with over 7000 participants. The aim of these studies was to investigate the roles of behavioural, psychological, and social factors in understanding age-related differences in both mental and physical health. Detailed information on retention and response rates can be found in Radler and Ryff (2010). Participants were contacted to participate in a second wave

in 2004 (MIDUS 2; N = 4963). These MIDUS 2 participants were invited to complete a separate biological assessment called the MIDUS 2 Biomarker Project (N = 1054) (Radler, 2014). The primary reasons for not participating in this biological assessment were (1) participants did not wish to travel to the clinic, (2) had family obligations, (3) were too busy, or (4) were not interested (Dienberg Love et al., 2010). Between 2013 and 2014, MIDUS 3 (N = 3295) completed a third wave of data collection on the same sample. Of the 1255 participants who were part of the Biomarker Project, 945 were retained at MIDUS 3. The present study uses participants who completed MIDUS 2, the MIDUS 2 Biomarker Project and MIDUS 3. Detailed information on the study protocol and measures are found in Ryff and colleagues (2018).

2.2. Participants

1255 individuals participated in the in the MIDUS 2 Biomarker project, comprising two subsamples: the longitudinal sample (N = 1054) and the Milwaukee sample (N = 201) (Ryff et al., 2010). The Milwaukee sample does not have data at MIDUS 3, and hence, was not included in the present study. Thus, we used 1054 from MIDUS 2 (1255–201 = 1054). Of these, 945 were retained at MIDUS 3. We also excluded 20 individuals who completed a different protocol and 13 individuals who had heart attacks prior to the MIDUS 2 study (to ensure that the sample only included individuals who suffered heart attacks after the biological assessment). This resulted in a sample of 912 participants used in the present study. Ages ranged from 35 to 86 (M = 57.06, SD = 10.97). 55.71% were female, 22.6% had high school education or less, and the mean number of chronic conditions was 2.16 (SD = 2.13), with 432.9% reporting a hypertension diagnosis and 9.6% reporting a diabetes diagnosis.

2.3. Measures

Occurrence of acute myocardial infarction was measured in telephone interviews in both MIDUS 2 and MIDUS 3. Participants were asked if they had any heart trouble and if so, whether they had been diagnosed as having had a heart attack. To establish which participants had suffered acute myocardial infarction between being assessed at MIDUS 2 and MIDUS 3, participants who reported heart attacks at MIDUS 2 were excluded. This left only participants who suffered heart attacks between MIDUS 2 and MIDUS 3.

Cardiovascular measurement. Heart rate was measured using a beatto-beat electrocardiogram (ECG). Beat-to-beat analog ECG signals were collected and then digitised at a sampling rate of 500 Hz. This was conducted using a 16-bit National Instruments analog-to-digital board attached to a micro-computer. Heart rate was then calculated as the average of all valid inter-beat intervals and then translated to beats-perminute. Systolic and diastolic blood pressure were recorded using a Finometer monitor (Finapres Medical Systems, Amsterdam, Netherlands) which accurately assesses absolute blood pressure (Schutte et al., 2003). A finger cuff was placed on the middle finger of the non-dominant hand and an arm cuff on the upper arm on the same side. In the MIDUS 2 dataset, this resulted in two baseline averages corresponding to the mean blood pressure readings for the first and last 6 min of the baseline period. Similarly, the data set contains two averages for the stress tasks.

Cardiovascular reactivity is defined as the arithmetic difference between task and baseline averages (Gallagher et al., 2020). In line with previous research, we computed reactivity for systolic blood pressure, diastolic blood pressure and heart rate (Gallagher et al., 2020b; Ginty et al., 2020). In the MIDUS 2 dataset, an average score for two stress tasks - the Stroop task and an Arithmetic task - is provided. To arrive at a single, overall score for these stress tasks, we took the average of the average scores for each of these tasks, as this has been suggested to increased reliability and generalizability (Kamarck & Lovallo, 2003). We then calculated the cardiovascular reactivity. To do this, we subtracted the average baseline (or 'resting') score from the overall stress score for systolic blood pressure, diastolic blood pressure, and heart rate.

Trait Gratitude was assessed using two items from the Subjective Well-Being scale (McCullough et al., 2002) which were extracted from the gratitude questionnaire (GQ-6) (Jans-Beken et al., 2015). Participants were asked to rate their agreement with two statements on a 7-point Likert scale (1 = strongly disagree to 7 = strongly agree). These statements were: "I have so much in life to be thankful for", and "I am grateful to a wide variety of people." Cronbach's alpha for this scale was 0.73. This was measured as part of MIDUS 2.

Stress tasks. The stress tasks comprised a Stroop task and an arithmetic task. For the Stroop task participants were seated in front of a computer and coloured words appeared on screen. These words either matched the colour or did not and were subsequently judged as either congruent or incongruent (i.e., the word "red" written in red letters was congruent, but the word "red" written in yellow letters was incongruent). Participants used a keypad to respond to find the colours of the letters, not the name of the colour. Participants were also informed that the computer "will score your responses for speed and accuracy. If you don't respond quickly enough, it will score your response as incorrect and present a new problem." This task lasted 6 min and was followed by a 6-minute recovery period.

The arithmetic task used the Morgan and Turner Hewitt mental arithmetic task, which requires participants to complete several addition and subtraction problems (Turner et al., 1986). A problem was presented on screen with an equal to sign and participants pressed a key to indicate whether the answer presented was correct or incorrect. Problem difficulty varied. If participants gave a correct answer they were subsequently presented with a more difficult problem. If an incorrect answer was given, a less difficult problem was subsequently presented. Participants were informed that if they did not answer sufficiently quickly their answer would be scored as incorrect (Coyle et al., 2020). This task lasted 6-minutes and was followed by a 6-minute recovery period.

Perceived stress was assessed at baseline and after each stress task. Participants were verbally asked by the researcher for a stress rating from 1 to 10, with 1 being not stressed at all and 10 being extremely stressed. These were captured to confirm that the stress tasks were psychologically stressful. Such items have been used in similar studies (e.g., Gallagher et al., 2021). We computed an overall average for both stress tasks.

Control variables were selected based on their well-established relationships with cardiovascular reactivity and cardiovascular health. These control variables used were: socio-economic status (Coughlin & Young, 2020), diabetes (Jacoby & Nesto, 1992), body mass index (BMI) (Bucholz et al., 2012), age, sex (Canto et al., 2012), and high blood pressure (Creaven et al., 2020a), and whether participants had ever smoked (Elkhader et al., 2016).

Education was used as a proxy for socioeconomic status and coded as 'high school or less', 'some college', and 'college and higher'. It was measured at MIDUS 2. Diabetes was assessed by asking participants had they ever been diagnosed with diabetes. It was coded as either 'yes' or 'no' and it was measured at the MIDUS 2 biomarker project. BMI was calculated by dividing weight by height squared it was measured at the MIDUS 2 biomarker project. Smoking status was coded as ever having been a smoker or not it was measured at the MIDUS 2 biomarker project.

2.4. Procedure

Participants at MIDUS 2 were admitted for a two-day hospital stay in one of three participating sites. On day one, they completed selfadministered questionnaires which assessed various psychological constructs and demographic questions, as well as a 45-minute medical exam which included a medical history and physical exam. After breakfast on the second day, participants received a standardised experimental protocol examining the response to cognitive challenges similar to stressors experienced in everyday life. The session lasted 90 min. Participants sat quietly for 11 min for a formal baseline before undergoing their first cognitive stress task followed by a 6-minute recovery period followed by the second cognitive stress task and a 6-minute recovery. The stress tasks were presented in random order.

Following the stress tasks, participants were asked to hand in their completed self-administered questionnaires and were then debriefed. This protocol has been outlined in detail elsewhere (e.g. Dienberg Love et al., 2010, Ryff et al., 2011), and includes further details on the collection of blood samples, urine samples, saliva samples, the measurement of respiration, and heart rate variability. We have chosen to focus on those sections relevant to the current study.

2.5. Data reduction and analysis

R version 4.2.0 was used to prepare the data. We used MPlus (version 8.2) for all analyses. Checks for normality and assumption checking were carried out using inspection of Q-Q plots, histograms, and Shapiro tests, where all variables had p-values > .05. Manipulation checks were carried out using paired-samples t-tests to confirm that the stress tasks increased blood pressure. Exploratory comparisons between participants suffering myocardial infarctions and those who did not were conducted were conducted using independent samples t-tests. In cases where equal variances were not assumed, Welch's two-sample t-test is reported which can result in degrees of freedom which are smaller or in decimal form (Whitlock & Schluter, 2015).

To test the hypotheses, we conducted a logistic regression parallel mediation model in Mplus using a maximum likelihood estimator. Gratitude (assessed at MIDUS 2) was entered as the predictor variable, systolic and diastolic blood pressure reactivity, and heart rate reactivity (assessed during the MIDUS 2 Biomarker Project) were entered as parallel mediators, and myocardial infarction (assessed at MIDUS 3) was entered as a dichotomous outcome variable. All previously mentioned control variables were entered as control variables.

Maximum likelihood estimation was used and was appropriate as it makes use of all available data, meaning participants with some missing data were not excluded. 27% of observations were missing for systolic and diastolic reactivity, and 12% for heart rate reactivity. As Dong and Peng (2013) and Newman (2014) recommend, when > 10% of observations are missing, full information maximum likelihood should be used to ensure unbiased estimates. Additionally, to account for the binary outcome, the mediation model was estimated using logistic regression (Feingold et al., 2019).

Preacher and Hayes (2008) recommend using bias corrected confidence intervals to test indirect effects. Following these recommendations, we made use of 1000 bootstrapped samples with bias corrected 95% confidence intervals. As our mediators were entered in parallel, each of our three indirect effects were assessed while accounting for the other two. This is important for building parsimonious models (Preacher & Hayes, 2008), and reducing parameter bias owing to omitted variables (Judd & Kenny, 1981). Standardised estimates are reported for direct effects with continuous outcomes. Estimates for categorical outcomes are reported in log-odds scale. This includes estimates for indirect effects which are also reported in odds ratio scale meaning that they are regarded as statistically significant if the confidence intervals do not intersect zero.

3. Results

3.1. Descriptive statistics

Descriptive statistics for the study variables are reported in Table 1, and correlations are reported in the online supplementary materials. On average, 6.7 years elapsed between participation in the MIDUS 2 Biomarker project and MIDUS 3, with a minimum of 4 and maximum of

Table 1

Descriptive statis	stics of	sample.
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Name	Mean	SD	Min	Max	N (%)	Missing (%)
Heart attack (Yes)					23 (2.5)	0
Years between measure	6.67	1.30	4	9		0
Trait gratitude	6.29	0.81	2.00	7.00		0
Systolic blood pressure reactivity	13.87	10.60	-18.30	65.60		27
Diastolic blood pressure reactivity	6.52	4.03	-7.15	21.40		27
Heart rate reactivity	3.83	3.91	-6.40	29.40		12
Age	57.06	11.97	35.00	86.00		0
BMI	28.98	5.97	16.49	60.39		0
Education	2.33	0.82	1.00	3.00		0
High school or less					206	
					(22.6)	
Some college					198	
					(21.7)	
College degree					506	
minimum					(56.6)	
Diabetes (Yes)					87 (9.6)	0
Sex (Female)					483	0
					(55.71)	
High blood					297	1
pressure (yes)					(32.9)	
Number of chronic conditions	2.16	2.13	0.00	16.00		0
Ever smoked (Yes)					394	0
					(43.2)	

9 years. 2.5% of the sample reported suffering a heart attack between MIDUS 2 and MIDUS 3. At MIDUS 3, 76.32% reported having at least one chronic underlying condition in the past 12 months, with 7.7% reporting a diabetes diagnosis. Women reported a larger number of chronic health conditions at MIDUS 2, t(789.32) = 3.61, p < .001.

There was a high correlation between systolic blood pressure reactivity and diastolic blood pressure reactivity (N = 660, r = .79, p < .001). Trait gratitude had a positive and significant correlations with systolic, diastolic and heart rate reactivity (see Table A.1 in supplementary materials). The average heart rate reactivity and diastolic blood pressure were quite low in the sample (O' Riordan et al., 2022), see Table 2. Those who suffered heart attacks by MIDUS 3 had lower heart rate reactivity at MIDUS 2 than those who did not, t(14.35) = 3.89, p = .002; there were no significant differences for systolic or diastolic reactivity.

3.2. Manipulation checks

Paired samples t-tests were conducted on the baseline perceived stress and the mean of the perceived stress ratings for the two stress tasks, confirming that participants experienced stress during the tasks t (853) = 44.25, p < .001, d = 1.51. Paired samples t-tests between baseline cardiovascular measures and the cardiovascular stress responses averaged across the two stress tasks confirmed that the stress tasks increased cardiovascular responses for systolic blood pressure, t (661) = 33.67, p < .001, d = 1.31, diastolic blood pressure t(661) = 41.60, p < .001, Cohen's d = 1.62, and heart rate t(801) = 27.75, p < .001, Cohen's d = 0.98, See Table 4 for descriptive statistics and cardiovascular parameters.

3.3. Hypothesis testing

We hypothesised that gratitude would have an association with acute myocardial infarction through systolic reactivity, diastolic reactivity, and heart rate reactivity. We tested this using the MODEL INDIRECT Table 2

Comparison of reactivity scores (means, SDs) for those who suffered acute myocardial compared to those who did not.

Acute myocardial infarction	Ν	SBP reactivity mean	SBP reactivity SD	SBP reactivity min/max	DBP reactivity mean	DBP reactivity SD	DBP reactivity min/max	HR reactivity mean	HR reactivity SD	HR reactivity min/max
No	889	13.95	10.66	-18.3/65.6	6.54	4.06	-7.15/21.4	3.86	3.93	-6.4/29.4
Yes	23	9.68	5.9	2.35/22.95	5.51	2.21	2/10.8	2	1.65	-0.95/4.2

Note: SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

Table 3

Means and standard deviations for systolic blood pressure, diastolic blood pressure, and heart rate.

Name	Mean	SD
SBP baseline	123.9	19.2
DBP baseline	61.5	12.0
HR baseline	72.8	10.7
SBP pooled task	136.7	21.6
DBP pooled task	67.7	12.3
HR pooled task	76.6	11.3
SBP Stroop task	138.9	22.2
DBP Stroop task	68.4	12.5
HR Stroop task	77.2	11.6
SBP math task	134.9	21.6
DBP math task	66.8	12.
HR math task	76.0	11.2

Note: SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

command in Mplus version 8.2. Looking first to the direct effects, the paths from trait gratitude to systolic blood pressure reactivity ($\beta = 0.09$, p = 0.012), diastolic blood pressure reactivity ($\beta = 0.08$, p = 0.043), and heart rate reactivity ($\beta = 0.10$, p = 0.003) were statistically significant and positive in nature. Furthermore, there was no direct association between trait gratitude and the occurrence of acute myocardial infarction in the logistic regression analysis. Coefficients for the paths are illustrated in Fig. 1 and summarised in Tables 4 and 5.

Regarding the indirect effects, (see Table 5), there were no significant indirect effects for either systolic blood pressure reactivity, $\beta = -0.079$, 95%CI [- 0.253, 0.015], or diastolic blood pressure reactivity $\beta = 0.032$, 95%CI [-0.050, 0.174]. Thus, our findings do not support Hypotheses 1 and 2. In support of Hypothesis 3, there was a significant indirect effect through heart rate reactivity, $\beta = -0.098, 95\%$ CI [-0.331, -0.010], meaning that, while statistically controlling for age, sex, BMI, high blood pressure, education, diabetes, whether or not

Table 4

Model Coefficients for the Parallel Mediation Model with Three Mediators and Covar
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						Consequent						
				Parallel	mediator						Outcome	
Antecedent	SBP reactivity			DBP reactivity			HR reactivity			Myocardial infarction		
	Coeff	SE	Р	Coeff	SE	Р	Coeff	SE	Р	Log odds	SE	Р
Baseline	-0.02	0.05	0.73	-0.03	0.04	.45	-0.03	0.04	.44			
Age	0.25	0.04	< .001	0.19	0.04	< .001	-0.08	0.04	.03	0.09	0.03	< .00
Sex	0.08	0.04	0.05	-0.01	0.04	.79	-0.02	0.04	.51	1.08	0.81	.18
BMI	0.05	0.04	0.18	-0.01	0.04	.85	-0.06	0.04	.08	0.06	0.04	.14
Trait gratitude	0.09	0.04	0.01	0.08	0.04	.04	0.10	0.03	< .001	-0.12	0.36	.74
Education										-0.43	0.30	.15
Ever smoked										0.09	0.53	.86
High blood pressure										-0.39	0.59	.52
Diabetes										0.51	1.19	.67
SBP reactivity										-0.07	0.04	.13
DBP reactivity										0.09	0.13	.48
HR reactivity										-0.21	0.13	.11
-	$R^2 = 0.0$	8		$R^2 = 0.0$	5		$R^2 = 0.0$	20		$R^2 = 0.45$		
	P - < 00	01		P = 0.00	12		P = 0.06	4		P - < 0.01		

Note: SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

participants had ever smoked, trait gratitude was associated with a lower likelihood of suffering a heart attack through its effect on heart rate reactivity. Furthermore, because the mediators were included in parallel, this effect also account for the effects of systolic and diastolic blood pressure reactivity.

3.4. Supplementary analyses

We conducted several additional analyses to ensure the robustness of our results and also to rule out alternative explanations. Further details and statistical output regarding the supplementary analyses can be found in the online supplement.

First, due to the high correlation between systolic and diastolic blood pressure reactivity, we conducted three additional mediation models to examine separately systolic blood pressure reactivity, diastolic blood pressure reactivity, and heart rate reactivity (using a Bonferroni adjustment and a more conservative alpha coefficient of.016 to assess significance). Findings showed that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction; there was also a significant indirect effect for systolic blood pressure reactivity, but no effect was detected for diastolic reactivity (see online supplement).

Second, we ran an additional model controlling for positive affect, as previous research suggests a relationship between positive affect, gratitude and cardiovascular health (Ginty et al., 2020; Pressman et al., 2019b; Schache et al., 2019). Findings show that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction, but no effect was detected for systolic or diastolic reactivity (see online supplement, tables A.2 and A.3).

Finally, we ran an additional model controlling for depressive affect, as previous research suggests a relationship between depressive affect, gratitude, and cardiovascular health (Bouzinova et al., 2015; Ginty et al., 2020). Findings show that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction, but no effect was detected for systolic or diastolic reactivity



Note: SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM). a indicates that the estimate is given in log odds scale. Otherwise, standardised estimates are reported. * Indicates p < .05

Fig. 1. Parallel mediation analysis of the relationship between trait gratitude and myocardial infarction through the cardiovascular reactivity parameters.

Table 5

Results of mediation analysis predicting myocardial infarction: indirect relationships between trait gratitude and myocardial infarction through three reactivity measures.

			BC 95%	CI
	Estimate	SE	Lower	Upper
Indirect effects	Predictor: gratitude	Trait		
Total indirect effect Unique effects:	-0.142	0.359	-0.662	0.766
1. Systolic blood pressure reactivity	-0.079	0.064	-0.253	0.015
2. Diastolic blood pressure reactivity	0.032	0.058	-0.050	0.174
Heart rate reactivity	-0.098	0.080	-0.331	-0.010

Note: BC 95% CI refers to the bias-corrected 95% confidence interval using 1000 bootstrap samples. All estimates are reported in log-odds scale; estimates with CIs that do not include zero are statistically significant and bolded.

(see online supplement, tables A.4 and A.5).

4. Discussion

Evidence from a growing body of research suggests that gratitude can be associated with cardiovascular health outcomes through its modulation of stress responses (Jans-Beken et al., 2020; Schache et al., 2019). However, the underlying processes have not been fully examined. We tested the potential indirect association between trait gratitude on acute myocardial infarction through systolic blood pressure reactivity, diastolic blood pressure reactivity, and heart rate reactivity. We found a significant indirect association between trait gratitude and myocardial infarctions, through heart rate reactivity, meaning that increased trait gratitude was associated with decreased risk of suffering a heart attack through increases in heart rate reactivity. However, we found no significant indirect effects through either systolic blood pressure reactivity or diastolic blood pressure reactivity. We also found significant, positive direct associations between trait gratitude and systolic blood pressure reactivity, diastolic blood pressure, and heart rate reactivity. There were no direct associations between trait gratitude and risk of myocardial infarction.

Looking first to the direct effects, the results of our present study suggest that trait gratitude was associated with increased reactivity, and this increase was associated with a reduced risk of suffering acute myocardial infarction. This finding is seemingly inconsistent with the stress buffering hypothesis insofar as trait gratitude has a positive relationship with all cardiovascular reactivity parameters and increases in cardiovascular reactivity are associated with negative outcomes (Phillips & Hughes, 2011). However, previous research has suggested that an increase in reactivity associated with trait gratitude may reflect moderate or healthy responses to stress (Gallagher et al., 2021). These authors suggest that responding to stress requires the mobilization of resources and positive emotions facilitate this. For example, happiness has previously been associated with increased cardiovascular reactivity (Framorando & Gendolla, 2019). Framorando and Gendolla (2019) suggest that emotions like happiness lead to individuals appraising tasks as less demanding and subsequently mobilizing higher effort, leading to higher blood pressure. This leads Gallagher and colleagues (2021) to suggest that gratitude may increase engagement. Thus, the result of our study may reflect the capacity of trait gratitude to buffer against the deleterious effects of stress by helping to mobilise resources complete challenging tasks, which is consistent with the stress buffering hypothesis.

Consistent with this line of reasoning, we found that increases in heart rate reactivity mediated the relationship between trait gratitude and a decreased risk of acute myocardial infarction. In the context of the previously discussed direct effects, this implies that – despite increases in reactivity – trait gratitude is associated with more positive cardiovascular outcomes, providing further evidence for gratitude playing a health-protective role. This is consistent with predictions that positive emotions like gratitude are associated with better health outcomes (Fredrickson, 2004; Jans-Beken et al., 2020; Schache et al., 2019).

Concomitantly, we found that individuals who suffered acute myocardial infarctions had significantly lower heart rate reactivity than those who did not. This is inconsistent with some previous research which reported positive associations between reactivity and risk of myocardial infarction (Carroll et al., 2012). However, while increased reactivity has previously been found to be cardio-toxic (Phillips & Hughes, 2011), some recent research suggests that blunted reactivity is

associated with an increased risk of myocardial infarctions (O' Riordan et al., 2022). For example, a study of 100 patients found that increased heart rate reactivity was associated with a reduced risk of cardiovascular morality (Kupper et al., 2015). This may reflect chronotropic incompetence, or the inability to increase heart rate to match cardiac output to metabolic demands (Brubaker & Kitzman, 2011; Kupper et al., 2015). Chronotropic incompetence has been associated with cardiovascular disease (Brubaker & Kitzman, 2011), including myocardial infarction (Savonen et al., 2008).

This may partially align with the suggestion that gratitude reduces cardiovascular disease by improving physiological function (Boehm, 2021; Schache et al., 2019), implying that increases in reactivity associated with trait gratitude reflect the adequacy of the cardiovascular system to respond to acute mental exertion. As such, higher trait gratitude may more holistically reflect healthier lifestyles and physiological functioning rather than stress coping resources (Boehm, 2021). For example, research has found that other positive constructs like optimism are associated with more frequent exercise, healthier foods and a lower likelihood of smoking cigarettes (Virani et al., 2020).

Although we found that heart rate reactivity mediated the relationship between trait gratitude and a decreased risk of acute myocardial infarction, we did not find indirect relationships between systolic blood pressure reactivity or diastolic blood pressure reactivity and risk of myocardial infarction, which is inconsistent with previous predictions. Nonetheless, there was also high correlation between both diastolic and systolic reactivity in our study. In a simulation study exploring how bootstrapping methods are impacted by correlated mediators, results showed that when mediators are highly correlated, there was a lower likelihood of the confidence intervals to include the true values of the correlated parameters (Beasley, 2014). To investigate this, we re-ran the analyses, while excluding systolic blood pressure reactivity or diastolic reactivity, and confirmed that the pattern of results remained the same. However, it is worth noting that when we look at our supplementary analyses, which included only systolic blood pressure reactivity as the mediating variable, this mediator emerged as a significant indirect pathway by which gratitude reduces acute myocardial infarctions. This is more consistent with past findings (Gallagher et al., 2020a), and may suggest that the high correlations between systolic blood pressure reactivity may have masked their effects. Nonetheless, this requires further investigation before any conclusions may be drawn.

Moreover, we conducted additional supplementary analyses to ensure that our findings were specific to gratitude. We controlled for positive affect to demonstrate that trait gratitude continued to have a significant indirect effect on the risk of the occurrence of acute myocardial infarction. In addition, we checked that our findings were not just the reverse of the statistical effects of depressive affect by conducting supplementary analyses with depression as an additional control variable. Here, our findings continue to demonstrate that gratitude continues to have an indirect relationship with myocardial infarction through heart rate reactivity. Thus, we can be reasonably confident in the robustness of our findings. Taken together, our findings suggest that higher levels of trait gratitude are associated with higher heart rate reactivity, and through this increase are associated with a lowered risk of acute myocardial infarction.

4.1. Strengths and limitations

This study has several limitations. The MIDUS 2 survey only used two questions from the GQ-6 scale to assess gratitude, making it difficult to compare it to other studies which used the full scale. The present study only included individuals who suffered heart attacks and survived. There was no laboratory-based manipulation of gratitude. It would be preferable if there were more measurement periods as this would allow for a more complete picture of participant health, over time. A further limitation is that the stress tasks used to induce stress (i.e., the Stroop test and arithmetic task) can be viewed as non-evaluative, asocial, and low threat. This may help to explain why stress responses were relatively muted compared to other large studies (Creaven et al., 2020b). The present study did not examine the stress buffering hypothesis using the one-item self-report stress measure reported in MIDUS due to limitations associated with simple self-report measures of stress (Epel et al., 2018).

While gratitude has shown consistent beneficial effects for health (Boggiss et al., 2020; Hill et al., 2013), this is not to say that one should only practice gratitude for the putative health benefits. Rather, it is to acknowledge exploring the determinants of health mean, examining both negative and positive psychological constructs (Fredrickson, 2004). In this case, the gap in past research is that it has predominantly focused on gratitude and self-reported health (Boggiss et al., 2020; O'Connell & Killeen-Byrt, 2018), and the novelty of our paper is that we show how gratitude can also have effects on objective physical health outcomes in cardiovascular reactivity.

Finally, due to the design of the study, causal relationships cannot be inferred. However, this study also has several strengths and novel contributions. It makes use of a standardised and well-controlled laboratory-based stress tasks to assess reactivity. It is the first study to longitudinally assess the association between trait gratitude and the risk of heart attacks. Furthermore, it helps to clarify that increases in cardiovascular reactivity associated with trait gratitude do not necessarily result in poorer cardiovascular outcomes.

4.2. Future directions

It would be useful to extend the present analysis by examining other cardiovascular outcomes such as hypertension. Accordingly, it would also be useful to explore the extent to which trait gratitude impacts reactivity by reducing stress or by improving physiological functioning. For example, in previous stress-buffering models (Pressman et al., 2019a), one way by which gratitude may buffer the effects of stress is by interacting with how stressful the task is perceived. It would be useful to assess this relationship in a stress-testing protocol context, as well as assessing the relationship between trait gratitude and chronotropic incompetence. Additionally, with further research suggesting that the relationship between reactivity and health may be curvilinear (Phillips et al., 2013), it would be helpful to explore how gratitude is related to both exaggerated reactivity and blunted reactivity. It would be useful to address how gratitude, depressive affect, and positive affect influence each other's relationships to cardiovascular outcomes.

It is also recommended that future research investigate the association between gratitude and reactivity in the context of a randomised control trial using a gratitude induction. Finally, it would be useful to explore this relationship in a latent variable modelling framework in order to more accurately examine how systolic and diastolic reactivity may mediate the relationship between trait gratitude and acute myocardial infarctions.

Finally, these findings have clinical utility. Gratitude interventions are low-cost and easy to use (Wood et al., 2010). For example, gratitude lists whereby individuals write down three to five things for which they are grateful have been shown to have a number of beneficial effects (Kerr et al., 2015; Manthey et al., 2016). Previous research shows that the use of gratitude journals in cardiac samples improves outcomes (Redwine et al., 2016). Combined with the results of this study and previous work, gratitude may constitute a useful point of intervention for the improvement of cardiovascular health.

4.3. Conclusions

In conclusion, this study found that heart rate reactivity significantly mediated the relationship between trait gratitude and the occurrence of acute myocardial infarction. Higher trait gratitude was associated with lower likelihood of suffering acute myocardial infarction 6.7 years later, through changes in heart rate reactivity, even when controlling for age, sex, BMI, education, high blood pressure and diabetes. This suggests that gratitude may buffer the negative physiological consequences of stress and overall improving cardiovascular outcomes.

These novel findings help further clarify that increases in cardiovascular reactivity, associated with trait gratitude, do not necessarily result in poorer cardiovascular outcomes. They also demonstrate that positive psychological constructs have beneficial impacts of cardiovascular health. In sum it may be said that this study contributes to our understanding of how gratitude impacts physical health.

Declaration of generative AI and AI-assisted technologies in the writing process

Statement: The author(s) did not use generative AI technologies for preparation of this work.".

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.

Data availability

The Mid-Life in the United States dataset is publicly available.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.biopsycho.2023.108663.

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