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State level income inequality affects cardiovascular stress responses: Evidence from the Midlife in the United States (MIDUS) study☆

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ABSTRACT

Rationale: The slow and insidious effects of income inequality on health means that their effects can be difficult to reveal, taking many years to become apparent. These effects can also be experienced differently according to subjective status and ethnicity making the relation between income inequality and health difficult to understand. Cardiovascular reactions to acute stress are indicative of future health outcomes. *Objective:* To examine whether short to medium term income inequality affected cardiovascular responses to acute stress whilst accounting for ethnic groups and subjective status. *Method*: Participants state of residence was available for 1155 people who participated in the MIDUS biomarker data project. This detail was used to merge the relevant US state level inequality data 1, 5, 10 and 15 years prior to the MIDUS biomarker data project which assessed cardiovascular responses to acute stress. *Results:* Our analysis demonstrated an association between inequality 5, 10 and 15 year prior and cardiovascular reactions to acute stress. Subjective community status and Black and minority ethnic group membership interacted to affect the association between inequality and cardiovascular reactions. *Conclusions:* In states where income inequality was high, less healthy cardiovascular responses were evident. However lower subjective community status and Black and Ethnic minority group members interacted with income inequality such that their impact was variable contingent on state level inequality. These findings extend the literature on income inequality and health and particularly highlights a psychophysiology pathway linking income inequality and health.

1. State level Income Inequality affects cardiovascular stress responses: Evidence from the Midlife in the United States (MIDUS) study

There is a growing body of research documenting the negative social, political and health consequences of high income inequality (Jay et al., 2019; Pickett and Wilkinson, 2015; Wolf et al., 2014). Focusing on health outcomes, the literature on the relationship between income inequality and health (IIH) shows considerable evidence of a negative relationship between higher income inequality and poorer self-rated health, shorter life expectancy, higher rates of infant mortality, and poorer mental well-being (Detollenaere et al., 2018). Research has linked income inequality to increased risk of diseases such as cardiovascular heart disease (Baum et al., 1999; Mobley et al., 2006). A number of biobehavioural pathways for the relationship between IIH have been explored. Income inequality has been linked to increased inflammation in otherwise healthy women (Clark et al., 2012). Lifestyle habits, such as smoking and diet, have also been linked to income inequality (Mobley et al., 2006). Another pathway of relevance is altered cardiovascular reactivity as a consequence of psychological stress. This latter pathway is the focus of the present research. In addition, there is evidence that income inequality creates and amplifies differences between socioeconomic groups such that those in lower socioeconomic groups are particularly at risk of coronary heart disease. For example, lower income groups show a 1.53 increase in their risk compared to higher income groups (Franks et al., 2011). That is why we also focus on the role of status group variables in the IIH relationship.

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2. Cardiovascular reactivity and income inequality

Even though there are many possible pathways to explain why there is a link of income inequality to health (Wilkinson and Pickett, 2017), one likely explanation is that income inequality increases stress. Consistent with this, many studies link income inequality to people's self-reported levels of stress and wellbeing (e.g., Oishi and Kesebir, 2015; Schneider, 2012). Even though there is value in these studies, it is also important to provide evidence that income inequality impacts physiological responses to stress—such as cardiovascular reactivity. Cardiovascular reactivity measures the physiological changes from baseline in response to a stressor. Several prospective studies have found that individuals who display exaggerated responses to acute psychological stress are at a greater risk of future hypertension, atherosclerosis, and cardiovascular mortality (Carroll et al., 2012; Chida and Steptoe, 2008; Gerin et al., 2000; Kamarck et al., 1997). A failure to mount a sufficient response, also known as blunted reactivity, can also be seen as problematic. It is related to depressive symptoms, addiction, obesity, and poorer self-reported health (Keogh et al., 2021; Phillips and Hughes, 2011) which are all known risk factors for heart disease (Hamieh et al., 2019) in both initially healthy and diseased populations (O'Riordan et al., 2022). Both blunted and exaggerated responses suggest that stress drives 'wear and tear' on the body (Glei et al., 2007; Guidi et al., 2021). Thus, given the links between income inequality and heart disease, altered cardiovascular reactivity to acute stress may be one pathway by which income inequality increases cardiovascular health and disease (Black et al., 2017; Carroll et al., 2012; Chida and Steptoe, 2008).

Here we respond to a recent call for examination of the physiological pathways linking inequality and health (Scheepers and Ellemers, 2019) to examine a potential psychophysiological stress mechanism of the income inequality-health relationship. Ryan et al. (2021) used an experimental paradigm and found evidence for heightened stress reactivity in response to income inequality. This approach also chimes with recent research highlighting the interacting effects of biological and social factors such as group membership and network position on cardiovascular reactivity and allostatic load (Gallagher et al., 2014, 2021). Here, we focus on both group and status factors to facilitate a better understanding of how income inequality may be harmful (Blázquez-Fernández et al., 2018). Specifically, we investigate the relationship between income inequality and cardiovascular reactivity whilst paying due regard to two potentially interacting group factors: subjective community status (SCS) and Black and minority ethnic group membership (BME). Before unpacking these processes further, to explain the approach we have taken, we consider key methodological considerations when studying income inequality and health.

2.1. Challenges associated with studying the negative consequences of income inequality

Income inequality is thought to create social contexts that are difficult for everyone to live in, rich and poor alike (Wilkinson and Pickett, 2010). Kawachi and Kennedy (1997) found that mortality is higher in more unequal societies. As well as this impact on health, Jetten et al. (2017) suggest that income inequality affects the social and political life in a society. Central to this, as Paskov et al. (2013) argue, is that there is more social competition and status anxiety amongst both the rich and the poor in more unequal societies. Income inequality is also related to reduced socioeconomic diversity in social networks (Bakker et al., 2019; Tammaru et al., 2020). In the short term, this is evident in the restricted social networks that are evident in the mobile phone data of migrant communities for example (Bakker et al., 2019). In the longer term, it is evident in the neighbourhood socioeconomic segregation that emerges with changing patterns of income inequality (Tammaru et al., 2020). Over time, these types of social processes are those that are likely to lead to a breakdown in social cohesion and segregation which disproportionately impacts low income and minority ethnic groups. The resulting health impact is unlikely to be observable instantaneously but is nonetheless corrosive within a society over time (Blakely et al., 2000). Methodologically, this presents a challenge because testing the relationship between income inequality and health may require the measurement of income inequality long before observing any effects on indices of health.

There are other practical challenges when studying the relationship between income inequality and health. Even within a single country, there is social and spatial complexity in income and wealth distribution at neighbourhood, city, and regional levels. Comparatively, rich people can live in poor cities and vice versa (Marmot, 2015). Furthermore, sociocultural factors such as minority and ethnic group memberships interact with income inequality in determining the residential spaces people ultimately occupy (Balsa-Barreiro et al., 2022). For example, it may be more tenable for a low income minority group member to work in a poorer city with lower pay because of lower associated living costs, than in a wealthier city with higher pay where living costs are higher. The challenges for comparisons between countries are no less complicated where estimating differential effects of income inequality on health are undertaken (Tammaru et al., 2020). Between country differences in income inequality are complicated by differences in GDP across countries, differences in national health systems, ethnic heterogeneity of the population and economic development (Marmot, 2015). Given these practical challenges and the lack of availability of comparable and relevant biometric health data across several countries, here, we use the Midlife in the United States (MIDUS) data series to test our ideas within one country, the USA. The availability of biometric data represents a fruitful approach to understanding the complex bio-behavioural pathway between income inequality and health. Data on state level income inequality facilitates the exploration of IIH relationship whilst also allowing us to attend to the role of ethnic and income group status on cardiovascular reactivity.

3. Income inequality and group status

Existing research highlights that income and ethnic group matter to the IIH relationship. Even though Wilkinson and Pickett (2010) argue that inequality is stressful for everyone, there is increasing evidence that the effect of income inequality is contingent on group status (Scheepers et al., 2009). High status groups have been found to experience more collective angst and more opposition to immigration in the face of high (compared to low) inequality (Jetten et al., 2017). This is attributed to a 'fear of falling'. On the other hand, Ahern and Galea (2006) found higher incidences of depression in lower (compared to higher) income groups which they attributed to lower status groups having fewer resources to cope with stress than their higher income counterparts. And those who experience the greatest stress, such as low status or minority groups, are the groups that have been found most likely to display dysregulated cardiovascular responses to stress (Evans and Kim, 2007).

Importantly too, in this research, it is an individual's subjective belief about their status that is a better predictor of health than objective status measures (Adler et al., 2000; Singh-Manoux et al., 2005; Wakefield et al., 2016) with evidence that income group or poverty do not fully account for this relationship (Adler and Snibbe, 2003). Instead, those who subjectively see their socioeconomic status (SES) as low are found to have worse health outcomes and higher rates of mortality than their higher SES counterparts. Furthermore, subjective view of status has been demonstrated to impact health over and above material circumstances (Adler et al., 1994). Correlational evidence for the SES-health relationship has been supplemented by experimental evidence showing that cardiovascular indices were responsive to experimentally manipulated changes in subjective social status (Pieritz et al., 2016). More recently, subjective status within a community level has been shown to predict health (Zell et al., 2018) suggesting that where an individual feels they stand in their own community is as important as where they feel they stand in society as a whole.

In the US, one important determinant of people's subjective perceptions of their own economic and community status relates to people's racial or ethnic group membership (Arbona and Jimenez, 2014). This is one reason why minority ethnic group membership has been found to be reliably predictive of chronic stress (Chen et al., 2004; Juster et al., 2010; Steptoe and Feldman, 2001; Vliegenthart et al., 2016) as well as cardiovascular reactivity (Shen et al., 2004). Nevertheless, the relationship between ethnic and racial group membership and stress may not be straightforward and ethnic minority group membership might also be protective of health. In particular, income inequality which drives ethnic group segregation (Bakker et al., 2019) may minimise available high-income comparators and protect minority group members' subjective status. In turn, comparatively higher subjective community status (SCS) derived from the tendency to compare oneself to ethically similar others may act to protect disadvantaged minority group members' health. Given the potentially complex relationships between majority and minority ethnic group membership and subjective community status we have undertaken an analysis that allows us to explore possible interacting effects between these factors.

3.1. The present research

Following initial evidence that the effects of income inequality on cardiovascular reactivity (a key marker of future cardiovascular health, Chida and Steptoe, 2008), are more likely to become apparent over time, we conducted a secondary analysis of the MIDUS data series. Specifically, we explored whether US State level income inequality prospectively predicts cardiovascular reactivity in response to a lab based stress task. We explored whether level of income inequality in participant's home state 5 years prior increases cardiovascular reactivity in response to standardised stress tasks (H1). Here we chose five years prior for our central research question as a major review of 79 studies of analysis of US national level data suggests that the detrimental effects of income inequality became apparent within 3–5 years (Zheng, 2012). Because income inequality does not act in isolation and has been found to be reliably linked to subjective status in society, we anticipated that subjective community status will moderate the relationship between income inequality and cardiovascular reactivity (H2). We hypothesised that higher subjective community status would reduce any negative cardiovascular effects of income inequality. Third, because minority ethnic group status may both be detrimental and protective of health, we hypothesised that BME group membership and subjective community status would interact to affect the relationship between income inequality and cardiovascular reactivity (H3). We refrained from specifying the nature of this interaction given the opposing patterns of results that have been observed for BME group membership in past research.

Finally, though our central research question related to the effect of inequality 5 years prior, we conducted a set of supplementary analyses using participants' home state level income inequality 1, 10 and 15 years prior to data collection. This allows us to consider whether these effects endure over time (H4) which, as Pickett and Wilkinson (2015) note, few studies have done previously. Due to space limitations these additional analyses are presented in the supplemental materials.

4. Method

4.1. Participants and recruitment

The cardiovascular reactivity data was taken from wave two (N = 5164) of the Midlife in the United States (MIDUS) dataset, collected in 2004. MIDUS is a longitudinal dataset aimed at exploring the health and daily life of adults in America, specifically those in mid to later life. Data was available for a total of 1155 participants who completed the psychophysiological sessions. Of these, for 1042 valid blood pressure scores were recorded and 1135 had valid heart rate measurements in response to the stress tasks. Participant age ranged from 35 to 86, with a mean age of 57.32 (SD = 11.55). Participants had a mean Body Mass Index (BMI) of 29.77kg/m2 (SD = 6.63).

Participants were originally recruited to the study using random digit dialling. We were particularly interested in wave two due to the associated biomarker data collected between 2004 and 2009. As part of this project, a sample of the original participants completed a battery of physiological tests, such as cortisol testing and cardiac reactivity to stress tasks.

To be eligible for this involvement in the biomarker project, participants had to be a resident in the US. Participants from the first MIDUS survey (collected in 1994–1995) were re-contacted and invited to participant in this second wave which included both a phone interview and a questionnaire via regular mail, for a total of two surveys completed per participant. The self-administered questionnaire was 114 pages long, and the phone interview lasted approximately 45 min. Once a participant had completed both assessments, they were sent a recruitment pack for the biomarker project in the mail. This contained a letter and brochure explaining the biomarker project. A follow up call was arranged in the following weeks to schedule an appointment at their assigned testing centre.

There were three testing sites: the University of California Los Angeles, the University of Wisconsin, and Georgetown University. As such, there may have been a large distance for participants to travel to participate. The exhaustive battery of physiological tests, including two separate psychological stress tasks required an overnight stay as part of the project (Ryff et al., 2019). The extent of this time commitment is no doubt responsible for the reduced biomarker data sample. A more detailed description of the complete biomarker procedure can be found in Love et al. (2010) and Ryff et al. (2019).

4.2. Design

For the purposes of this paper, three datasets were merged prior to analysis. A Gini Coefficient statistic calculated by Frank (2014) using tax data from the Internal Revenue Service was used as indicators of state level income inequality, 1, 5, 10 and 15 years prior to MIDUS data collection. This approach is consistent with multiple other studies that have explored income inequality in the USA (Bloome, 2015; Jacobs and Dirlam, 2016; Schneider et al., 2018). After compiling a dataset of inequality in each US state from 2014 to 1983, we matched this information to the state of residency for each participant 1, 5, 10 and 15 years prior to the date of their data collection (Blakely et al., 2000; Kondo et al., 2012; Zheng, 2012. See supplementary materials for exact years). This data set was then merged with data from wave two of the MIDUS survey, including measures of subjective community status and BME group membership, and with MIDUS biomarker data, which included cardiovascular reactivity.

The dependent or cardiovascular reactivity variables accessed via the biomarker database included were systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR). Subjective community status and Black and ethnic minority group membership were explored as variables that might intervene in the relationship between inequality and CVR. In case of any earlier or later onset of negative consequences (Blakely et al., 2000), the role of state level income inequality 1, 10 and 15 years prior to participants' cardiovascular reactivity to stress was also considered. Control variables were chosen based on well researched and established relationships with health and physiological responses, including BMI, smoking status, gender and whether or not participants took blood pressure medication.

4.3. Predictor measures

4.3.1. Measure of income inequality

The current study uses the Gini coefficient statistic to measure income inequality in participants' home state in the United States of America. This is one of the most common measurements of income

inequality (De Maio, 2007; Kawachi and Kennedy, 1997) and calculates income inequality on a scale of 0–1, where 0 denotes perfect equality. For the sake of brevity, we refer to income inequality as inequality in describing our results.

4.3.2. Subjective community status

Subjective Community Status was measured using a version of the MacArthur Subjective Social Status Scale (Adler et al., 2000). Participants ranked themselves on a 10-point ladder, and respondents were asked to rank themselves in the community most meaningful to them. This measure has been found to have good test-retest reliability (Cundiff et al., 2013; Operario et al., 2004). A total of 194 respondents did not respond to this item.

Black and Minority Ethnic Group Membership (BME).

Within the MIDUS, participants were asked to respond to an item about ethnic/racial origins using the item 'What are your main racial origins – that is, what race or races are your parents, grandparents, and other ancestors?' In line with prior work using the MIDUS dataset, responses to this question were first dichotomised as White ($N = 988$) or Black ($N = 51$). A small number of individuals belonged to other minority ethnic groups categories ($N = 59$). A total of 64 people stated they did not know their ethnicity ($N = 7$) or did not respond to the question ($N = 57$). See Table 1 for a full breakdown. In line with previous research, we compared White ($N = 988$) respondents with all Black and Minority Ethnic groups (BME; $N = 110$) (Allen et al., 2019; Boylan et al., 2015; Boylan and Ryff, 2015; Tsenkova et al., 2013). A comparison of White and Black ($N = 51$) respondents is presented in the supplemental results.

4.4. Dependent measures: stress task and cardiovascular testing

For the reactivity measure, a Finometer was used to take non-invasive blood pressure readings for systolic and diastolic blood pressure. This is considered a highly accurate measure due to the use of both a finger and wrist cuff and it is a validated measure for cardiovascular reactivity according to the British Hypertension Society protocol (McMahon et al., 2020; Schutte et al., 2004).

An electrocardiogram (ECG) was used to gather beat to beat heart rate data continuously throughout the psychophysiology protocol. Heart rate was calculated as the average of all the valid 'RR intervals'. RR intervals are defined as the time between R waves, with R waves being defined as "the first upward deflection of the electrocardiogram following the Q wave arising from ventricular depolarization" (p. C2, Ryff et al., 2019). These were then converted from millisecond readings to beats per minute.

Two stress tasks were presented to participants while blood pressure and heart rate measures were taken: the MATH task (Turner et al., 1986) and a Stroop test. Reactivity to the Stroop and MATH were used as a single CVR as per previous research (Bibbey et al., 2013). The Stroop test is considered an effective laboratory stressor and has been used as a stress task in other research (Šiška, 2002; Teixeira et al., 2015). Baseline cardiovascular measures were collected whilst seated and resting approximately 11 min commencement of the protocol. The two stress tasks were of 6 min duration, with a 6 min recovery period between. Following the second task participants were allowed a final recovery period which also lasted 6 min. Combining the average reactivity across the two tasks meant the baseline and the task were closer to each other in terms of number of readings (11 min for baseline, 12 for tasks. Reactivity scores were calculated by subtracting mean SBP during the stress tasks (MATH and Stroop) from baseline ratings of SBP, in line with prior research (Gallagher et al., 2014). DBP and HR were calculated in the same way. More detail on both these stress tasks can be found in Ryff et al. (2019) and Keogh et al. (2021).

Of the original sample, 1255 completed the biomarker project, 713 women (56.8%) took part in the study and 542 men (43.2%). Data was available for a total of 1155 participants who completed the psychophysiological sessions. Twenty-six participants were excluded due to completing a different protocol for the stress tasks (Love et al., 2010; Ryff et al., 2019) and all physiological data were missing for 41 participants. A further 41 participants had no BP data and a further 31 participants were missing all of the blood pressure baseline or task data. Within task performance was assessed with continuous physiological monitoring. Where there was valid data of 180 s duration for either of the two baseline or task readings, data was imputed using mean performance for that task. Valid reactivity scores were available for 1042 participants in relation to blood pressure and 1135 in relation to heart rate.

Table 1
Ethnic composition of sample.

Ethnicity	Frequency	Percent
White	988	85.5
Black/African American	51	4.4
Native American/Alaska Native Aleutian Islander	15	1.3
Asian	4	.3
Native Hawaiian or Pacific Islander	3	.3
Other	30	2.6
Don't know	7	.6
Missing	57	4.9
Total	1155	100

4.5. Procedure on day of biomarker measurement

Participation in the biomarker project involved an overnight stay in one of the three testing centres. During this stay, participants took part in a battery of tests including a medical history and physical exam. The psychophysiology portion of the stay took part in the morning of day 2. In order to collect both blood pressure and beat to beat heart rate data, participants were connected to a Finometer Pro and an electrocardiogram (ECG).

The two stress tasks were administered: a Stroop test and a mental arithmetic task, the Morgan and Turner Hewitt (MATH) task (Turner et al., 1986), and the overall psychophysiological lab session took 90 min in total (Keogh et al., 2021). The Stroop test involves the presentation of a colour word on a computer screen. This word was either displayed in the same colour as the word presented or in a different colour. Participants were asked to respond as quickly as possible identifying the colour of the font rather than the word. The MATH task involved a math question with the answer appearing on the screen. Participants were then required to indicate whether the answer was correct or not.

To avoid human error in administering the tasks, both tasks were delivered on a computer. The order of the tasks was also randomised, so some participants did the Stroop task first and others did the MATH task first. The difficulty of the Stroop and MATH task were responsive to participant's performance so that the task remained stressful regardless of how well or poorly a participant performed. For example, if a participant was more accurate in the Stroop task, the difficulty increased. In the MATH task, all participants started on difficult level 3. The difficulty level would then go up or down depending on how well the participant performed.

4.6. Approach to analysis

SPSS version 24 was used to analyse the data for the current study. Because we were interested in the direct relation between inequality and health as well as the interaction of subjective community status and BME group membership, we used Hayes' PROCESS macro (version 3). We used PROCESS model 3 with 5000 bootstrapping (Hayes, 2013) to test the main effect of income inequality (H1), and the interaction effects of inequality and subjective community status (H2), and inequality, subjective community status and BME (H3) (see Fig. 1 for a conceptual

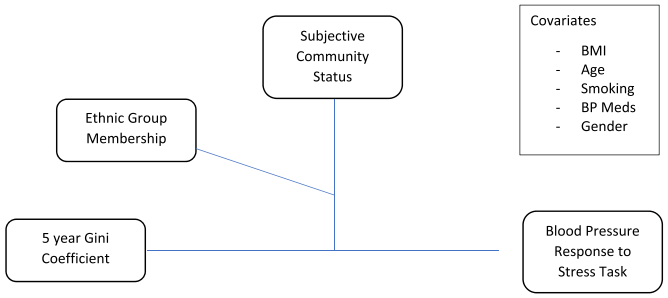

Fig. 1. Conceptual model. Relationship between inequality and biological responses to the stress tasks.

representation of the hypotheses). To consider the durability of any effects (H4) we examined state level inequality 1, 5, 20 and 15 years prior to data collection entered as the primary predictor. This analysis was undertaken for each outcome variable, systolic blood pressure, diastolic blood pressure and heart rate, separately. Control variables were chosen based on well researched and established relationships with health and physiological responses, including gender, BMI, smoking status, and whether or not participants took blood pressure medication.

We conducted a post hoc power analysis using G*Power 3 (Faul et al., 2007). Given the size of the sample and the inclusion of three predictor variables, this analysis indicated we had sufficient power (0.98) to detect a small effect size in the 0.02 range and (>0.99) to detect a moderate effect in the 0.15 range. In line with current advice, we offer variance explained (R^2) and change in variance explained (R^{2chng}) as indicators of effect size (Fairchild et al., 2009; Hayes, 2013). In line with current best practice, we have reported the extent to which variables of interest had missing values (Sidi and Harel, 2018) and we indicate how missing values were dealt with in our CVR variables.

5. Results

Table 2 indicates that the MATHS and Stroop manipulations perturbed the cardiovascular system of participants. Scores on the Stroop and MATHS task were higher than biometric indicators obtained during baseline. Blood pressure showed stronger perturbation in response to the stress tasks than heart rate. Table 3 indicates the relationships between all variables. Due to its demonstrable relationship with health (Adler and Snibbe, 2003), we used a subjective measure of community status. In our correlations matrix, however, we also outline the relationship between these variables and more objective indicators of social status such as educational level and adjusted household income. Subjective community status is significantly related to SBP ($r = -.15$, $p < .01$), DBP ($r = -0.08$, $p < .05$) and HR ($r = 0.11$, $p < .01$) reactivity (see Table 3).

H1. Inequality and Cardiovascular Reactivity to Stress

Participants home state level inequality 5 years prior did not directly predict SBP reactivity response to stress, $B = -134.61$, $SE = 118.72$, $p = .26$, 95% CI [−367.63, 98.42]. Nor did we find support for the prediction that state level inequality should directly affect DBP reactivity, $B = -48.59$, $SE = 47.69$, $p = .31$, 95% CI [−142.20, 45.02] or HR reactivity in response to stress, $B = -1.15$, $SE = 40.66$, $p = .98$, 95% CI [−80.95, 0.78.66].

Table 2
Mean (SD) cardiovascular reactivity at baseline and stress tasks.

	SBP (mmHg)	DBP (mmHg)	HR (bpm)
Baseline	124.40 (18.93)	61.34 (11.81)	73.28 (17.65)
Stressor			
Average	136.45 (21.62)	67.17 (12.20)	76.57 (11.25)
Stroop	138.02 (22.13)	68.02 (12.33)	77.10 (11.46)
MATH	134.22 (21.75)	66.54 (12.24)	76.03 (11.24)

H2. Subjective Community Status Intervenes in the Relation Between Inequality and CVR

Controlling for covariates, subjective community status (SCS) directly affected the relation between inequality and SBP reactivity in response to the stress tasks, $B = -31.93$, $SE = 14.56$, $p = .03$, 95% CI [−60.52, −3.35, see Table 3]. Overall, reactivity was higher amongst those with lower SCS (see Table 2 and Fig. 2). In line with H2, inequality and SCS interacted to affect SBP reactivity, $B = 51.83$, $SE = 25.22$, $p = .04$, 95% CI [2.32, 101.4]. As Fig. 2 illustrates, high state inequality and high SCS was associated with lower reactivity. On the other hand, low state inequality and high SCS was associated with higher reactivity. SCS was not linked to reactivity in states where the GINI coefficient was within one standard deviation of the US mean. Note that our three-way interaction (H3) was also significant qualifying these main and two-way effects (Tabachnick et al., 2007).

We found no support for the hypothesis that inequality five years prior and SCS interacted to affect DBP, $B = -9.28$, $SE = 5.85$, $p = .11$, 95% CI [−20.76, 2.21] or HR, $B = -1.24$, $SE = 5.05$ $p = .81$, 95% CI [−11.15, 8.67] reactivity to stress (see Table 3).

H3. Income Inequality, SCS and Ethnic Group Interact to Affect CVR

In line with H3, inequality, SCS and BME group status interacted to predict SBP, $B = -48.67$, $SE = 21.5$, $p = .02$, 95% CI [−90.88, −6.46] and DBP, $B = 16.59$, $SE = 8.64$ $p = .04$, 95% CI [−33.55, −0.37]. The interaction effect for SBP is illustrated in Fig. 3. Among White majority ethnic group respondents, lower inequality and higher subjective community status was associated with less SBP reactivity. In this way, higher SCS can be seen to act as a protection against stress regardless of income inequality. However, the pattern was different BME participants in US states that experienced low and medium levels of inequality. In these states, higher subjective community status was associated with greater SBP reactivity (Fig. 3), $B = -173.45$, $SE = 64.37$, $p = .007$, 95% CI [−299.81, −47.09]. In terms of effect size, this interaction effect explains a further 1.5% in variance in cardiac reactivity over and above the significant effects of smoking, gender and age observed and any other main or interaction effects which combined explained 11% of the variance in cardiac reactivity.

For DBP, the indirect effects were also significant, including the overall interaction of inequality, subjective community status and BME group membership (H3) (Table 4), reactivity $B = 30.13$, $SE = 17.64$, $p = .088$, 95% CI [−4.5, 64.77]. In low and medium inequality states, higher subjective community status was associated with comparatively higher DBP reactivity to the stress tasks. In terms of effect size, this interaction effect explains a further 1% in variance in cardiac reactivity over and above the effects of smoking, gender and age and any other main or interaction effects which combined explained 5% of the variance in cardiac reactivity.

We found no support for H3 in relation to HR (see Table 4).

H4. The Temporal Reach of Effects of Inequality

The availability of state level GINI data 1, 5, 10 and 15 years prior to the MIDUS data collection allowed us to consider whether these the effects of inequality endure over time (H4). We found that our model was a good fit to the SBP data using participants' home state inequality 1 year, $R^2 = 0.11$, $F(12, 817) = 8.52$, $p < .001$, 10 years, $R^2 = 0.11$, $F(12, 817) = 8.26$, $p < .001$, and 15 years, $R^2 = 0.12$, $F(12, 817) = 8.43$, $p < .001$, prior to the completion of the MIDUS stress tasks. In all cases the model added 1% to the explanation of variance in SBP reactivity, again comparing well to the 11–12% variance explained by the full model which included significant covariates such as smoking, age, and gender. We found that our models were also a good fit to the DBP but for a shorter duration. The model using participants' home state inequality at 1 year $R^2 = 0.05$, $F(12, 817) = 3.43$, $p < .001$, and 5 years, $F(12, 817) = 5.12$, $p < .001$ were a good fit. The model fit for 10 years, $R^2 = 0.05$, $F(11,817) = 2.99$, $p = .08$, or 15 years, $R^2 = 0.05$, $F(11, 817) = 2.81$, $p = .09$ prior to the completion of the MIDUS stress tasks was not significant. In both the 1 and 5 year case, the model added 1% to the explanation of

Table 3
Bivariate correlations between Inequality, Cardiovascular Reactivity variables and indicators of Income and Group status.

	Inequality 1 year	Inequality 5 year	Inequality 10 year	Inequality 15 year	SBP Reactivity	DBP reactivity	HR reactivity	BME	SCS	Income per head household	Educational level
Inequality 1 year	x	.51*	.71*	.68*	.02	−.00	.04	.09*	.02	.03	−.01
Inequality 5 year	.50*	x	.77*	.75*	.06	.02.	−.05	.05	−.01	−.03	−.07ˆ
Inequality 10 year	.70*	.76*	x	.85*	−.00	−.02	.02	.07	−.01	−.07ˆ	−.08*
Inequality 15 year	.68*	.75*	.85*	x	.06	.03	−.01	.06	.01	−.06	−.04
SBP reactivity	−.02	.04	−.03	.04	x	.81*	−.26*	.01	−.14*	.00	−.01
DBP reactivity	−.03	.02	−.05	.01	.80*	x	−.34*	−.02	−.08ˆ	.03	.04
HR reactivity	.06	−.04	.06	.02	−.26*	−.35*	x	.03	.11*	.05	.03
BME Group	.10*	.02	.06	.04	.03	−.01	.05	x	.01	.02	.01
Subjective Community Status	.02	.02	.03	.04	−.14*	.09ˆ	.09ˆ		x	.12*	.17*

Simple correlations between all variables above the diagonal. Partial correlations below the diagonal control for educational level and household income per resident. In all cases * indicates $p < .01$ and ˆ indicates $p < .05$.

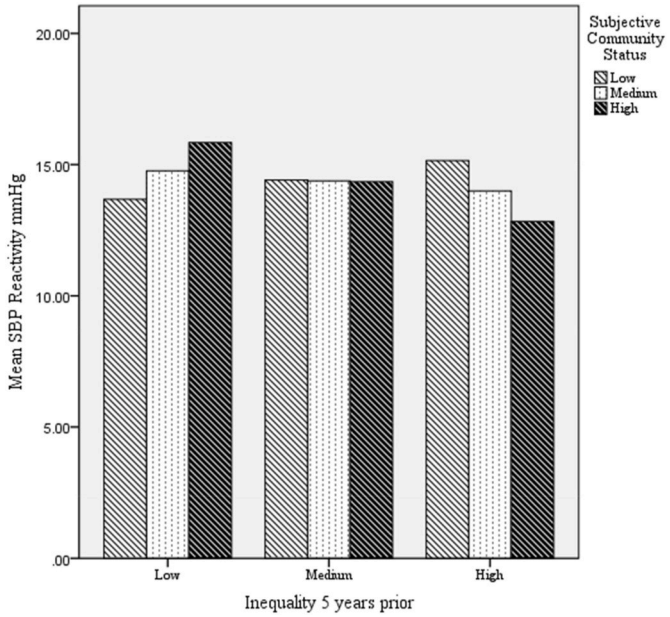

Fig. 2. Mean Systolic Blood Pressure Reactivity, State level inequality 5 years prior and Subjective Community Status.

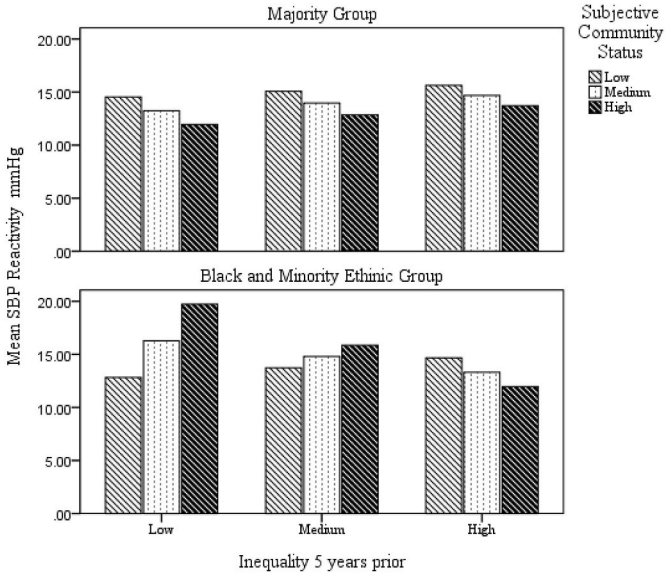

Fig. 3. Mean SBP reactivity and State level inequality 5 years prior by Ethnic group and Subjective Community Status.

variance in DBP reactivity again comparing well to the 5–6% variance explained by the full model which included significant covariates such as smoking, age and gender.

We did not find support that state level inequality directly affected SBP or DBP or HR at any time point.

6. Discussion

The current study explored the effect of income inequality on cardiovascular reactivity, with a particular emphasis on the idea that the effects of income inequality are best examined across time and whilst paying due regard to subjective status and ethnic group membership. Even though we found no support for a direct effect of income inequality on cardiovascular reactivity, there was evidence that this relationship was qualified by subjective status and ethnic group membership.

All in all, the effect of income inequality was complex. How respondents ranked themselves in their community and their BME membership was central to the relationship between income inequality and reactivity. Specifically, our findings show that, for majority ethnic group members (here White Americans), the cost of income inequality was highest amongst those who rated their own subjective status as lower while higher subjective community status was associated with lower blood pressure reactivity overall.

The pattern of effects for Black and Minority ethnic group members was different. The protection offered by high subjective community status for minority members was only evident for those residents in states that were highly unequal. In states with lower inequality (GINI less than .55) and average inequality (GINI between 0.56 and 0.61), higher subjective community status was associated with *higher* blood pressure reactivity in response to stress tasks. These findings highlight not only the importance of subjective status in driving health effects, but also the relevance of majority and minority ethnic group membership in understanding the health effects of inequality.

Stated another way, our findings seem to suggest that the protection afforded to White respondents who rank themselves highly in their communities is not evident amongst Black and ethnic minority respondents in comparatively more equal states. Counterintuitively perhaps, this may reflect greater experienced pressure and ambition to

Table 4
Main and interaction effects of 5 year Income inequality, Subjective Community Status (SCS) and Ethnic Group Membership (BME) on Systolic (SBP), Diastolic Blood Pressure (DBP) and Heart Rate (HR) reactivity.

Model inequality 5 year prior	SBP Reactivity				DBP Reactivity				HR Reactivity			
	B	Lr CI	Upr CI	t	B	Lr CI	Upr CI	t	b	Lr CI	Upr CI	t
Inequality	−134.61	−367.63	98.42	−1.13	−48.59	−142.2	45.02	−1.02	−1.15	−80.95	78.66	−.03
SCS	**−31.93**	**−60.52**	**−3.35**	**−2.19**	−9.28	−20.76	2.21	−1.59	−1.24	−11.15	8.67	−.25
Inequality x SCS	**51.83**	**2.32**	**101.34**	**2.05**	15.08	−4.8	34.97	1.49	1.97	−15.16	19.11	.23
BME	−87.96	−202.55	26.63	−1.51	−35.30	−81.34	10.73	−1.51	−2.75	−42.10	35.59	−.14
Inequality x BME	143	−54.77	342.20	1.42	57.57	−22.16	137.31	1.42	3.13	−64.86	71.12	.09
SCS x BME	**29.44**	**5.08**	**53.80**	**2.37**	**10.0**	**.21**	**19.78**	**2.01**	2.23	−6.25	10.71	.52
Inequality x SCS X BME	**48.67**	**−90.88**	**−6.46**	**−2.26**	**16.59**	**−33.55**	**−.37**	**−1.98**	−3.33	−17.99	11.34	−.45
Covariates												
Gender	***−2.03***	***−3.48***	***−.58***	***2.75***	−.08	−.66	.50	−.66	−.28	−.79	.22	−1.09
Age	***.16***	***.09***	***.23***	***4.60***	**.04**	**.01**	**.07**	**.01**	**.03**	**.00**	**.05**	2.17
BMI	−.01	−.13	.12	−.08	−.03	−.08	.02	−.08	.02	−.02	.07	.99
Smoking	***6.23***	***3.92***	***8.53***	***5.30***	**1.72**	**.79**	**2.65**	**.79**	**−1.22**	**−2.04**	**−.40**	−2.93
BP meds	−1.04	−2.88	.80	−1.11	.11	−.63	.64	−.63	**−.81**	**−1.44**	**−.17**	−2.50

Note. Fit for models SBP 5yrInequality $R^{2\ Chng} = 0.02$, $F(1, 817) = 5.86$, $p < .001$; DBP 5yrInequality $R^{2\ Chng} = 0.01$, $F(1, 817) = 3.69$ $p < .001$; HR 5yrInequality $R^{2chng} = 0.01$, $F(1, 897) = 0.20$, $p = .66$.
Bold indicates p value $< .05$ ***Bold and italics*** indicates p value less than 0.01.

succeed amongst high status minority ethnic group members in more equal states. One possible explanation for this effect is offered by Postmes and Branscombe (2002) who also found evidence that upwardly mobile Black respondents displayed worse health outcomes than their counterparts who remained embedded in Black communities. Pointing to a double jeopardy for minority racial groups, these findings suggests that higher status members of minority groups are less likely to reap health benefit from equality than majority group members. In general, our findings are consistent with previous research that demonstrated that both BME and subjective community status are central to the experience of inequality (Adler et al., 1994; Arbona and Jimenez, 2014). Extending existing knowledge further, we demonstrated subjective community and ethnic group status need to be accounted for to fully understand cardiovascular stress responses.

These findings are also an important addition to the literature as they highlight an important physiological pathway linking inequality and health —a pathway that is not evident in the literature to date. Our results indicate that prior experience of inequality affects people's physiological reactivity to everyday stress. Systolic blood pressure is a stronger predictor of cardiovascular disease and so our findings evidence that inequality is linked more strongly to systolic reactivity across time is important. We found evidence of this relationship between inequality and systolic blood pressure reactivity 1, 5, 10 and even 15 years after participants in the MIDUS studies. For sure, this relationship is also affected by ethnic group and subjective status. In our model these latter variables intervene in the relationship between income inequality and reactivity. This analysis adds to the growing body of research highlighting that effects of inequality on health and cardiovascular reactivity are complex and cumulative (Pickett and Wilkinson, 2015) across time. Our study adds to also in that it suggests that inequality is a form of chronic stress with long-term negative effects (Ryan et al., 2021). The reactivity we observe can be thought of as a form of wear and tear on the body increasing the long term allostatic load (Glei et al., 2007) of those living in unequal states. To our knowledge, we are the first to show this psychophysiological pathway linking income inequality and health in general through CVR.

The present study advances current research by providing further evidence that the social and political conditions that inequality creates is important for health. We particularly extend the literature by demonstrating that these effects can be shown in terms of biomarkers of health linked to allostatic load and stress. Furthermore, this study adds to the growing literature that the impact of inequality endures across time. Blakely et al. (2000) showed that the impact of inequality on self-rated health can take 5–15 years to emerge. We add to this by literature by being the first to demonstrate empirically that the impact of inequality on physiological responses is witnessed over time. This is useful for future work on inequality as it shows the importance of examining the role of inequality in the years prior to current experiences rather than in terms of people's current experience. We also demonstrate that the experience of inequality is influenced by subjective community and ethnic group status. This adds to the current literature by showing that inequality does not impact health in isolation, but rather in conjunction with status and contextual factors.

6.1. Limitations and future research

A limitation of our current study is the ratio of White to BME respondents. Our sample of BME respondents is small when compared to our sample of White respondents. However, adding to the strength of our findings is the fact that the effect was present also when BME versus Black only respondents were compared with Whites. The effects were also apparent across four time points and for both systolic and diastolic blood pressure. However, there was no evidence of a relationship between our variables of interest and heart rate. We believe that this may be because these tasks did not perturb heart rate to a high degree. We theorise the effects of subjective community and BME membership interact because of the reference group forces linked to ethnic group. However, there was no available measure for us to explore the degree to which respondents identified with their minority group or compared themselves to ethnically similar others. Future research would benefit from examining this explicitly, by potentially asking respondents how strongly they identify with their minority group membership to further uncover the mechanisms that may be driving these effects.

7. Conclusion

The current paper explored prospectively how inequality impacted cardiovascular stress responses. This analysis demonstrates that exposure to income inequality was also associated with heightened cardiovascular reactivity in particular SBP and DBP responses to stress, though this is a complex indirect relationship. BME group membership and subjective community status intervened to affect this relationship between inequality and cardiovascular reactivity to stress. To the best of our knowledge, our study is the first to demonstrate that inequality and group factors impact physiological stress responses in this way and as such our findings highlight an important biological pathway between income inequality and subsequent health. Our findings demonstrate that income inequality matters for health because it affects our ability to

mount cardiovascular response to stress. We contribute to the growing body of research that suggests inequality impacts downstream biomarkers of stress and health.

Credit author statements

Megan Ryan: Conceptualisation, Methodology, Data Curation, Formal, Analysis, Investigation, Resources, Writing- Original Draft. Stephen Gallagher: Conceptualisation, Methodology, Writing, Review and Editing. Jolanda Jetten: Conceptualisation, Methodology, Writing, Review and Editing. Orla Muldoon: Conceptualisation, Methodology, Validation, Visualisation, Writing- Original Draft, Visualisation.

Declaration of competing interest

The authors state they have no competing interests.

Data availability

The authors do not have permission to share data.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2022.115359.

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