Received: 18 December 2020 | Revised: 4 May 2021 | Accepted: 11 May 2021

DOI: 10.1111/psyp.13872

ORIGINAL ARTICLE

PSYCHOPHYSIOLOGY SPR WILEY

Attenuated cardiovascular reactivity is related to higher anxiety and fatigue symptoms in truck drivers

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Funding information
The data presented in this paper were collected as part of the baseline measures from the "Structured Health Intervention For Truckers (SHIFT)" randomized controlled trial, which is funded by the NIHR Public Health Research Programme (reference: NIHR PHR 15/190/42). SAC, JAK, AS and NJP are supported by the NIHR Leicester Biomedical Research Centre—Lifestyle theme. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health and Social Care. The first author (AG) has received funding for their PhD Studentship from the Colt Foundation (reference: JD/618). The Colt Foundation had no role in study design; election, synthesis, and interpretation of data; writing of the report; or the decision to submit the manuscript for publication

Abstract

Depression and anxiety have been linked with reduced stress-induced cardiovascular reactivity (CVR), which could be indicative of autonomic dysregulation. Less is known about the association between work-related fatigue and CVR. Truck drivers experience high levels of depression, anxiety, and fatigue, with repeated psychophysiological stressors on the road, yet little is known about the effects of these conditions on their CVR. Three hundred eighty six truck drivers completed the Hospital Anxiety and Depression Scale (HADS) and the Occupational Fatigue Exhaustion/Recovery Scale (OFER-15). Systolic (SBP) and diastolic (DBP) blood pressure and heart rate (HR) were measured at rest and during a stressor protocol to measure CVR. Multivariate regression analyses were used to determine relationships between variables and adjusted for nine key covariates. Higher symptoms of persistent fatigue were related to a reduced SBP reactivity ($\beta = -.236$, $p = .009$) and reduced DBP reactivity ($\beta = -.257$, $p = .005$), whereas there was a positive trend between acute fatigue and DBP reactivity ($\beta = .169$, $p = .052$). Higher symptoms of anxiety were related to a reduced SBP reactivity ($\beta = -.164$, $p = .016$). This study demonstrated in a population of truck drivers that both anxiety and persistent fatigue were related to an attenuated SBP reactivity in a combined model, whereas there was a positive trend between acute fatigue solely and DBP reactivity. These novel findings may have serious implications for cardiovascular disease risk in truck drivers, and future research should attempt to establish the causal effect of these associations and the underlying physiological mechanisms.

KEYWORDS

anxiety, blood pressure, cardiovascular reactivity, depression, fatigue, stress, heart rate, stress reactivity

1 | INTRODUCTION

Most research on cardiovascular stress reactivity has traditionally concerned elevated cardiovascular responses and its association with disease pathogenesis (Lovallo & Gerin, 2003), whereas a reduced stress reactivity was assumed to be acceptable, even necessary, for good health (Lovallo, 2005; Phillips et al., 2013). However, recent research shows that an attenuated cardiovascular response is equally indicative of dysregulation of the stress reactivity system as it deviates from the homeostatic norm. Reduced cardiovascular reactivity (CVR) has similarly been associated with other negative health implications such as obesity (Carroll et al., 2008), poor self-reported health (Phillips et al., 2009), depression and anxiety symptoms (Carroll et al., 2007; de Rooij et al., 2010; Salomon et al., 2009; York et al., 2007), which are associated with the development of cardiovascular disease (CVD) (Akil & Anwar Ahmad, 2011; Cohen et al., 2015; Fan et al., 2008). It is therefore now understood that dysregulated CVR can lead to poor health outcomes, and therefore, a moderate response closer to the median is optimal (Carroll et al., 2009; Lovallo, 2013).

Recent studies, which used large cohort samples and adjusted for covariates, have predominantly found significant negative associations between depression and cardiovascular stress reactivity (Carroll et al., 2007; Phillips, 2011; de Rooij, 2013; York et al., 2007), suggesting that those with higher rates of depression display lower CVR to stress. Similar results have been found for anxiety, with studies finding negative correlations between anxiety and cardiovascular stress reactivity (Carroll et al., 2007; de Rooij, 2013; Souza et al., 2015; Young et al., 1998) and meta-analytic data demonstrating the same effect (Chida & Hamer, 2008).

Less research has been conducted into the effects of fatigue on stress reactivity, despite fatigue being a somatic symptom in the diagnosis of depression (American Psychiatric Association, 2013). One study found that as self-reported fatigue increased during a stressor task, systolic blood pressure (SBP) reactivity decreased (Nolte et al., 2008). Similarly, another study that assessed naturally occurring fatigue found higher self-reported fatigue was associated with reduced SBP reactivity to a stress task (Lagory et al., 2011). Likewise, participants that underwent sleep restriction also saw a lower reaction to stress reactivity (Lü et al., 2018; O'Leary et al., 2013). Research suggests that there is an association between fatigue and an increased risk of developing CVD and cardiovascular-related events (Melamed et al., 2006). To date, no study has looked at the association between work-related fatigue and cardiovascular stress reactivity.

The issue of psychological influences (e.g., fatigue, anxiety, and depression) on CVR to stress is particularly relevant for truck drivers. Truck drivers are exposed to psychological stressors in their working environment daily, such as prolonged driving, traffic, dangerous road users, and navigating down narrow roads while endeavouring to meet chronic time targets (Passey et al., 2014). A recent systematic review found prolonged driving elicits a physiological stress response, including increases in urine catecholamine and cortisol concentrations after driving for long hours (Antoun et al., 2017). Truck drivers are at an elevated risk of developing CVD (Apostolopoulos et al., 2010) and also have a higher than national average prevalence of fatigue, anxiety, and depression (Chalmers & Lal, 2016; Guest et al., 2020; Varela-Mato et al., 2017). Negligible research has been conducted into the impact of these factors on their cardiovascular responses to stress amongst this at-risk group.

1.1 | Aims and hypothesis

This study aimed to investigate whether depression, anxiety, and fatigue were related to cardiovascular responses to acute stress tasks in a sample of truck drivers. It was hypothesized that there would be a negative relationship between self-reported depressive, anxiety, and fatigue symptomology, and cardiovascular stress reactivity, and that higher symptoms of depression, anxiety, and fatigue would be associated with attenuated cardiovascular responses to stress.

2 | METHOD

2.1 | Participants and selection

Participants were recruited as part of a cohort of long-haul truck drivers taking part in the "Structured Health Intervention For Truckers" (SHIFT) trial, which consisted of 386 participants recruited across 25 depots from the Midlands region of the United Kingdom. Full details of the trial protocol and measurements have been reported elsewhere (Clemes et al., 2019). In brief, drivers were recruited to voluntarily take part in a 12-month health intervention within their workplace. Drivers were excluded from the main trial if they suffered from clinically diagnosed CVD, had haemophilia or any blood-borne viruses, or suffered mobility limitations that prevent them from increasing their daily activity levels. The data reported in this study are from a 2-hr health assessment undertaken during the baseline phase of the trial and prior to intervention allocation and administration. Participants were asked to fast from all food and drink (except water) for at least 4 hr before the health assessment, which was conducted at the beginning of their shift. Where possible, participants underwent the health assessment in a room alone with the experimenter, where this was not possible (<25% of the time), a screen was put up to divide the room and offer privacy. Ethical approval was obtained from the Loughborough

University Ethics Approvals (Human Participants) Sub-Committee (reference: R17-P063). Written informed consent was obtained from all participants.

2.2 | Procedures and measures

Participants completed a comprehensive health questionnaire, which included information on lifestyle behaviours and medication use. This was followed by a health assessment, conducted by trained researchers within drivers' workplace environment before their shift. Details of all measurements taken are reported elsewhere (Clemes et al., 2019). The measurements relating to the analyses reported in this paper are described below, and a methodological schematic illustration is shown in Figure 1.

2.3 | Anthropometrics

Anthropometric measurements were undertaken, with stature measured using a portable stadiometer and body mass measured using Tanita DC-360S body composition scales. Body Mass Index (BMI) was subsequently calculated. The classification of healthy, overweight, and obese weight status were ascribed using the World Health Organization (WHO) classifications (World Health Organization, 2000).

2.4 | Psychological measures

2.4.1 | Depression and anxiety

Depression and anxiety symptoms were self-reported using the validated Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983), which has been previously used in similar research (de Rooij et al., 2010). The HADS can be used to indicate the presence of clinical depression or anxiety symptoms. This questionnaire consists of two subscales, seven questions for anxiety symptoms (HADS-A) and seven questions for depressive symptoms (HADS-D). The Cronbach's α for the HADS-A and HADS-D for this sample was calculated as .78 and .72, respectively. Each answer is scored between 0 and 3, with total scores for each construct ranging from 0 to 21. For each construct a score of 7 or less indicates no symptoms, whereas scores of 8–10, 11–14, and 15–21 indicate the presence of mild, moderate, and severe symptoms, respectively (Bjelland et al., 2002).

2.4.2 | Fatigue

Fatigue symptoms were self-reported using the validated Occupational Fatigue Exhaustion/Recovery Scale (OFER-15) (Winwood et al., 2005, 2006). The OFER-15 questionnaire is a specific work-related measure of fatigue within the last few months. It comprises 15 questions, with five questions relating to chronic fatigue, five to acute fatigue, and five to persistent fatigue. In this questionnaire, acute work-related fatigue is principally mediated through work-related factors and measured as fatigue felt at the end of the shift. Persistent fatigue suggests the accumulation of low recovery from high levels of acute fatigue, and chronic work-related fatigue is a maladaptive consequence of unrelenting persistent fatigue, resulting in declining interest, reduced concentration, and negative emotions.

Each question was scored between 0 and 6 where 0 is *strongly disagree* and 6 is *strongly agree*. Scores were then processed to ascertain a 0–100 scale, where 0 indicated minimal fatigue and 100 indicated maximal fatigue. The Cronbach's α for each measure of fatigue in this sample has been calculated be >.84, identical to other population samples (Winwood et al., 2006).

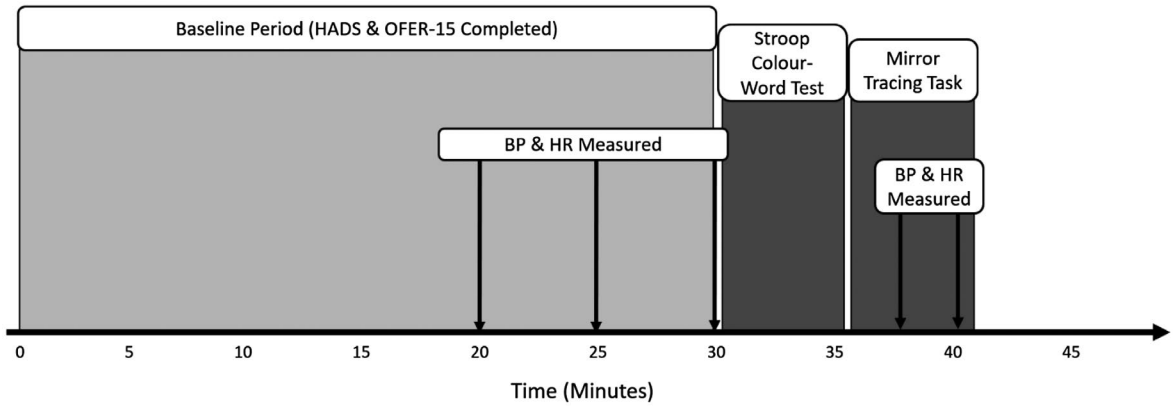

FIGURE 1 Schematic illustration of the trial protocol. BP, blood pressure; HR, heart rate; HADS, Hospital Anxiety Depression Scale; OFER-15, Occupational Fatigue Exhaustion/Recovery Scale

2.5 | Cardiovascular measures

Cardiovascular measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), and HR were measured at rest and under stress, using the validated automated Omron (HEM-907) sphygmomanometer (White & Anwar, 2001), placed on the brachial region of the left arm. Researchers ensured that arms were rested at heart level, and feet were flat on the floor for all measures. For resting measures, a reading was taken after 20 min rested in a seated position where participants were asked to fill in study questionnaires, with 5-min intervals between each measurement. The mean resting SBP, DBP, and HR readings were calculated from the three measurements.

2.6 | Psychological stress tasks

Cardiovascular perturbations to acute stress were induced using two well-known stressor tasks, previously used in similar research to stimulate a physiological stress response (Hamer & Steptoe, 2012).

A 5-min version of the Stroop Colour-Word test was used as the first stress task. The Stroop Colour-Word test is run using computer-based software, which displays colored words, and the participant is required to correctly identify the color of the word and not what the conflicting word says (MacLeod, 1991). This test compiled three stages, which became incrementally more difficult. Before each stage, participants were given standardized instructions and a short practice to ensure that they understood the requirements of the task.

The Stroop test was followed immediately by the Mirror Tracing task (Campden Instruments Auto Scoring Mirror Tracer 58024E), which has been routinely used to induce stress elsewhere (Feldman et al., 1999). The Mirror Tracing task involves tracing an adonized star pattern using a metal tipped stylus with the right hand continuously for 5 min; however participants are only to use the reflection of the star in an adjacent mirror for reference. The machine beeped if the metal tipped stylus left the star pattern, and each mistake was recorded on the machine. Participants were told to aim for at least five complete stars in the timeframe.

BP and HR were measured during the Mirror Tracing task at 2 min 15 s and at 4 min 35 s into the task to measure psychophysiological reactivity to acute stress. The mean stress induced SBP, DBP, and HR readings were calculated from the two measurements.

2.7 | Statistical analysis

Variables were checked for normal distribution using the Kolmogorov–Smirnov test of normality, with normally distributed variables reported as means (standard deviation [*SD*]) and non-normally distributed variables reported as medians (interquartile range [IQR]). The baseline cardiovascular measures were determined as a mean of the 3 readings at rest, and the stress cardiovascular measures were determined as a mean of the 2 readings during the stress protocol. To create a CVR measure for SBP, DBP, and HR, the mean resting SBP, DBP, and HR measures were subtracted from the mean stress-induced measures.

The HADS and OFER-15 Scale were analysed as continuous scores. The relationship between CVR and symptoms of depression, anxiety, and fatigue was tested using multivariate regression analysis. Models were adjusted for potential confounding variables determined a priori, based on previous associations with CVR (de Rooij et al., 2010). These were added to the regression models as covariates in Step 2 of the model and included age, sex, BMI, use of anti-hypertensive medication, use of beta blockers, smoking, alcohol consumption, shift pattern, weekly hours worked, and mean resting SBP/DBP/HR. Antipsychotic drug use was not included as a covariate as no participants reported using these. Statistical analyses were conducted using IBM SPSS Statistics 25. Outliers were defined as >2 *SD* from the mean, checked for physiological plausibility, and removed from the dataset if appropriate. Occasional missing data are reflected in the reported degrees of freedom. Relationships were considered statistically significant if p values were $\leq .05$.

3 | RESULTS

3.1 | Participant characteristics

Full sample characteristics are shown in Table 1. Three hundred and twenty-two participants provided valid data for all resting and stress protocol cardiovascular measurements. Table S1 shows full sample characteristics only for those who completed all cardiovascular measures of reactivity ($N = 322$). A sensitivity analysis revealed there were no significant differences in age, sex, BMI, depression, anxiety, and fatigue values between those included in the analyses compared with those with missing DBP or HR reactivity data ($p \geq .181$), but there was a significant difference in age ($p = .012$) between those included in the analyses and those with missing SBP reactivity data (see Table S2). For missing data, 45 participants were missing SBP reactivity data (29 participants were outliers and 16 did not provide all measures of SBP); 38 participants were missing DBP reactivity data (20 participants were outliers, and 18 did not provide all measures of DBP), and 39 participants were missing HR reactivity data (13 were removed due to being outliers, and 26 did not provide all measures of HR).

The median (range) score on the HADS-D questionnaire was 3 (1–13), and the HADS-A questionnaire was

TABLE 1 Sample characteristics

	Median (IQR)[a], Mean (*SD*), or % (*N*)	Number of missing data
Age (years)	49.5 (13.6)[a]	2
Sex (% male)	98.7% (381)	0
Average working hours (hours/week)	48.0 (5.0)[a]	1
Ethnicity		1
White European (%)	93.9% (361)	
Other (%)	6.4% (24)	
Highest level of education		18
GCSEs (%)	65.5% (241)	
A-level (%)	12.8% (47)	
University graduate (%)	6.3% (23)	
Other (%)	12.8% (47)	
Shift patterns		1
Morning (%)	63.4% (244)	
Afternoon (%)	11.9% (46)	
Night (%)	17.7% (68)	
Rotating shifts (%)	7.0% (27)	
Cardiovascular measures		
Resting SBP (mmHg)	129.7 (11.5)	27
Resting DBP (mmHg)	81.8 (11.3)	22
Resting HR (mmHg)	67.8 (9.6)	17
Stress task SBP (mmHg)	142.8 (21.5)[a]	36
Stress task DBP (mmHg)	91.3 (9.7)	30
Stress task HR (mmHg)	73.0 (14.1)[a]	32
SBP reactivity (mmHg)	14.0 (15)[a]	45
DBP reactivity (mmHg)	9.3 (7.5)	38
HR reactivity (mmHg)	5.3 (7.7)[a]	39
Hospital Anxiety and Depression Scale (HADS)		
Anxiety symptoms (0–21)	4.88 (3.18)	3
Depression symptoms (0–21)	3.88 (2.91)	3
Anxiety score ≥8 (%)	20.6 (79)	3
Depression score ≥8 (%)	13.1 (53)	3
Occupational Fatigue Exhaustion/Recovery Scale (OFER-15)		
Chronic symptoms (0–100)	36.7 (40)[a]	6
Acute symptoms (0–100)	50.0 (33)[a]	2
Persistent symptoms (0–100)	43.3 (33)[a]	4
BMI (kg/m^2)	29.76 (6.29)[a]	5
Healthy weight (%)	10.8% (41)	
Overweight (%)	40.9% (156)	
Obese Class 1 (%)	31.8% (121)	
Obese Class 2 (%)	12.3% (47)	
Obese Class 3 (%)	4.2% (16)	
Medication		
Anti-hypertensive medication (%)	9.1% (35)	0
Beta blockers (%)	3.4% (13)	5

(Continues)

TABLE 1 (Continued)

	Median (IQR)[a], Mean (*SD*), or % (*N*)	Number of missing data
Alcohol Intake		0
Never (%)	8.3% (32)	
Monthly or less (%)	18.1% (70)	
2–4 times a month (%)	37.3% (144)	
2–3 times a week (%)	29.0% (112)	
4 or more times a week (%)	7.3% (28)	
Cigarettes		0
Never smoked (%)	39.6% (153)	
Ex-smoker (%)	40.9% (158)	
Current smoker (%)	19.4% (75)	
Of those:		
1–5 cigarettes/day (%)	17.3% (13)	
6–10 cigarettes/day (%)	17.3% (13)	
11–15 cigarettes/day (%)	33.3% (25)	
16–20 cigarettes/day (%)	28.0% (21)	
>20 cigarettes/day (%)	4.0% (3)	

[a]Data presented with the median (interquartile range). Total sample size = 386.

4 (0–18). For HADS-D, 330 (86.2%) participants had no symptoms of depression (scored ≤7), 46 (12.5%) had mild symptoms (scored 8–10), and 7 (1.3%) had moderate symptoms (scored 11–14). No participants had severe symptoms of depression. For HADS-A, 304 (79.4%) participants had no symptoms of anxiety (scored ≤7), 60 (15.7%) had mild symptoms (scored 8–10), 18 (4.7%) had moderate symptoms (scored 11–14), and 1 (0.3%) had severe symptoms (scored ≥15).

3.2 | CVR

Across all participants, all cardiovascular measures (SBP, DBP, and HR) significantly increased in response to the stressor protocol ($p \leq .001$).

3.3 | Fatigue

A significant negative relationship between persistent fatigue symptoms and SBP response to the stress protocol ($\beta = -.166$, 95% CI −0.140 to −0.032, $p = .002$) was observed (Table 2), indicating that for every point increase on the OFER-15 scale, SBP reactivity decreases by 0.166 mmHg. There was also a significant negative relationship between persistent fatigue symptoms and DBP reactivity ($\beta = -.131$, 95% CI −0.080 to −0.008, $p = .016$) and HR reactivity ($\beta = -.174$, 95% CI −0.083 to −0.020, $p = .001$) to stress after adjusting for covariates (Table 2).

There were no significant relationships between chronic fatigue and SBP reactivity ($\beta = -.070$, 95% CI −0.084 to 0.018, $p = .200$) or DBP reactivity ($\beta = -.065$, 95% CI −0.054 to 0.013, $p = .232$), but there was a significant negative relationship between chronic fatigue and HR reactivity ($\beta = -.151$, 95% CI −0.069 to −0.012, $p = .006$) (Table 2).

There were no significant relationships between acute fatigue and SBP reactivity ($\beta = -.085$, 95% CI −0.099 to 0.011, $p = .144$) or DBP reactivity ($\beta = -.037$, 95% CI −0.049 to 0.023, $p = .485$), but there was a significant negative relationship between acute fatigue and HR reactivity ($\beta = -.113$, 95% CI −0.064 to −0.002, $p = .038$) (Table 2).

3.4 | Anxiety

A significant negative relationship between anxiety symptoms and SBP response to the stress protocol ($\beta = -.175$, 95% CI −1.015 to −.252, $p = .001$) was observed (Table 2), indicating that for every point increase on the HADS-A scale, SBP reactivity decreases by 0.175 mmHg. There were no significant relationships between anxiety symptoms and DBP or HR ($p \geq .111$) responses to the stress protocol, after adjusting for covariates.

TABLE 2 Multivariate regression analysis of depression, anxiety, and fatigue symptoms relationship with SBP, DBP, and HR reactivity to the stressor protocol in separate models

	Unstandardized B	Standardized coefficient β	p	Adjusted R^2	Model F	Model p
Systolic blood pressure						
HADS depression[a]	−0.312	−0.077	.161	0.069	3.228	<.001*
HADS anxiety[a]	−0.633	−0.175	.001*	0.094	4.100	<.001*
Persistent fatigue[a]	−0.086	−0.166	.002*	0.092	4.045	<.001*
Chronic fatigue[a]	−0.033	−0.070	.200	0.066	3.089	.001*
Acute fatigue[a]	−0.044	−0.085	.114	0.070	3.281	<.001*
Diastolic blood pressure						
HADS depression[b]	−0.128	−0.046	.394	0.059	2.913	.001*
HADS anxiety[b]	−0.208	−0.086	.111	0.063	3.057	.001*
Persistent fatigue[b]	−0.044	−0.131	.016*	0.051	2.654	.003*
Chronic fatigue[b]	−0.021	0.017	.232	0.056	2.813	.002*
Acute fatigue[b]	−0.013	−0.037	.485	0.057	2.847	.001*
Heart rate						
HADS depression[c]	0.034	0.015	.786	0.022	1.763	.067
HADS anxiety[c]	−0.119	−0.058	.295	0.024	1.822	.056
Persistent fatigue[c]	−0.052	−0.174	.001*	0.052	2.844	.002*
Chronic fatigue[c]	−0.041	0.015	.006*	0.048	2.662	.004*
Acute fatigue[c]	−0.033	−0.113	.038*	0.035	2.231	.016*

[a]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting SBP.

[b]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting DBP.

[c]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting HR.

*Statistically significant based on multivariate regression analysis ($p \leq .05$).

3.5 | Depression

There were no significant relationships between depressive symptoms and SBP ($p = .161$), DBP ($p = .394$) or HR ($p = .786$) when examined independently from anxiety or fatigue, and after adjusting for covariates (Table 2).

3.6 | Combined model of fatigue, anxiety, and depression

The combined model of fatigue, anxiety, and depression was analyzed by incorporating them all in the same multivariate linear regression model and adjusting for covariates (Table 3). When examining the combined model, the negative relationship between persistent fatigue and SBP reactivity remained significant ($\beta = -.236$, 95% CI −0.210 to −0.031, $p = .009$), as did the negative relationship between HADS-A and SBP reactivity ($\beta = -.164$, 95% CI −1.057 to −0.110, $p = .016$). However, there was no relationship with HADS-D and SBP reactivity.

The negative relationship between persistent fatigue and DBP reactivity also remained significant ($\beta = -.257$, 95% CI −0.144 to −0.025, $p = .005$), and there was a positive trend between acute fatigue and DBP reactivity ($\beta = .169$, 95% CI −0.001 to 0.110, $p = .052$).

No independent variables remained significantly related to HR reactivity when put in the combined model (all $p \geq .055$). Though there was a trend toward significance in the relationship between depressive symptoms ($\beta = .113$, 95% CI −0.007 to 0.624, $p = .055$) and persistent fatigue ($\beta = -.161$, 95% CI −0.099 to 0.005, $p = .074$) with HR reactivity.

4 | DISCUSSION

The present analyses have shown that in a sample of truck drivers from the United Kingdom, symptoms of persistent fatigue were negatively associated with SBP, DBP, and HR reactivity to stress, and both acute and chronic fatigue negatively related to HR stress reactivity. Symptoms of anxiety were negatively associated with SBP reactivity. When all

TABLE 3 Multivariate regression analysis of depression, anxiety, and fatigue symptoms relationship with SBP, DBP, and HR reactivity to the stressor protocol in a combined model

	Unstandardized B	Standardized coefficient β	p	Adjusted R^2	Model F	Model p
Systolic blood pressure						
				0.099	3.334	<.001*
HADS depression[a]	0.156	0.038	.550			
HADS anxiety[a]	−0.583	−0.164	.016*			
Persistent fatigue[a]	−0.120	−0.236	.009*			
Acute fatigue[a]	0.034	0.068	.432			
Chronic fatigue[a]	0.045	0.095	.258			
Diastolic blood pressure						
				0.061	2.414	.002*
HADS depression[b]	−0.048	−0.018	.798			
HADS anxiety[b]	−0.172	−0.075	.271			
Persistent fatigue[b]	−0.084	−0.257	.005*			
Acute fatigue[b]	0.055	0.169	.052			
Chronic fatigue[b]	0.008	0.027	.320			
Heart rate						
				0.058	2.425	.003*
HADS depression[c]	0.309	0.113	.055			
HADS anxiety[c]	−0.081	−0.039	.568			
Persistent fatigue[c]	−0.047	−0.161	.074			
Acute fatigue[c]	0.008	0.029	.737			
Chronic fatigue[c]	−0.028	−0.103	.217			

[a]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting SBP.

[b]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting DBP.

[c]Individual main effects adjusted for age, BP medication, beta blockers, sex, BMI, smoking status, alcohol intake, shift pattern, weekly working hours, and mean resting HR.

*Statistically significant based on multivariate regression analysis ($p \leq .05$).

independent variables were combined in the same model, the negative relationship between persistent fatigue and both SBP and DBP reactivity remained significant, as did the negative relationship between anxiety symptoms and SBP reactivity. No significant relationships between fatigue or anxiety and HR reactivity withstood. Interestingly, there was also a positive trend toward significance between acute fatigue and DBP reactivity.

This is the first study which examines the association between constructs of occupational fatigue (measured via the OFER-15 scale) and cardiovascular stress reactivity, and as a result, presents a valuable contribution to the field. It is somewhat unclear why we found a significant negative association between persistent fatigue and SBP, DBP, and HR reactivity, but only a negative association between acute fatigue and chronic fatigue with HR reactivity only. Further, when entered into the combined model, only a positive trend toward significance between acute fatigue and DBP reactivity remained. This particular finding was unanticipated, though could be explained by short-term (acute) fatigue resulting in mental depletion, which could increase the perceived difficulty of the task, and consequently task effort, which manifests a stronger cardiovascular response in DBP (Wright et al., 2008). Plausibly, as fatigue becomes prolonged (persistent/chronic), the sympathetic activity compensates less, and this may be where autonomic dysregulation occurs, which may explain our associations. It is also possible that those who self-reported higher persistent and chronic fatigue were less motivated on the day and perceived the task as too difficult which led to them engaging less with the stress protocol, resulting in decreased CVR (Wright et al., 2008). However, this would be disputed by many studies who still found significant associations after adjusting for ratings of perceived stressfulness (Bibbey et al., 2013; Yuenyongchaiwat et al., 2017) and task performance (Carroll et al., 2007; Yuenyongchaiwat et al., 2017). This would

indicate the concept that attenuated cardiovascular stress reactivity may be a marker of neural hypoactivation in the brain, due to the under-recruitment of brain systems that require motivated action during stress exposure (Carroll et al., 2017; Ginty et al., 2013; Holsen et al., 2011); however, in this study, we were unable to corroborate this. Further, more mechanistic work would be needed to determine the mechanisms that may underlie our results.

The percentage of our sample which displayed clinical levels of symptoms for anxiety (HADS-A $\geq$ 8) were lower than a comparative normal population sample of adults in the United Kingdom (Breeman et al., 2015), which would appear to contradict the literature (Guest et al., 2020). However, there is considerably less research into anxiety prevalence among truck drivers compared with depression, and our study adds valuable findings to this under-researched topic. HADS-A scores were comparable with other cohort studies (Carroll et al., 2007; de Rooij et al., 2010). As hypothesized, the negative association between SBP reactivity and HADS-A scores seen in our analyses was consistent with similar studies (Carroll et al., 2007; de Rooij et al., 2010; Young et al., 1998; Yuenyongchaiwat et al., 2017). It is known that other psychological disorders related to anxiety (e.g., panic disorders) are associated with a decreased sensitivity of the beta-adrenergic receptor and decreased stimulation of the beta-adrenergic system and could explain why we saw a significant negative association between anxiety symptoms and SBP reactivity but not DBP in our active stressor task. Future research in passive stress tasks such as the cold-pressor test may be useful to assess the alpha-adrenergic system in truck drivers (Yuenyongchaiwat et al., 2017).

The percentage of our sample, which displayed clinical levels of symptoms for depression (HADS-D $\geq$ 8), were comparative with a general population sample of adults in the United Kingdom (Breeman et al., 2015). Also, these scores were similar to other cohort studies which used the HADS-D questionnaire (Carroll et al., 2007; de Rooij et al., 2010). Globally, truck drivers have been known to exhibit higher than average levels of depression (Guest et al., 2020). This further highlights that truck drivers are an at-risk group which requires further attention, particularly due to the well-known links between depression and future CVD (Hare et al., 2014). Contrary to the hypothesis, there was no association between HADS-D scores and any of the CVR parameters, in contrast to other studies (Carroll et al., 2007; de Rooij et al., 2010; Salomon et al., 2009; York et al., 2007). However, we did observe a trend toward significance for HR reactivity in the combined model, which would follow the findings from the literature. Other studies which have used the same stressor tasks have similarly found no associations between depressive symptoms and CVR (Wang et al., 2016). Our Cronbach α of internal consistency was considered acceptable for this cohort, but future studies which look at depressive symptoms in truck drivers may want to consider the use of alternative tools to measure depressive symptomology.

The association between lower SBP reactivity and elevated persistent fatigue and anxiety symptoms is noteworthy as lower cardiovascular responses have been associated with negative health outcomes (Phillips et al., 2013). This is exacerbated by the association between anxiety and the onset of CHD, independent of classic risk factors (Dhar & Barton, 2016; Roest et al., 2010). This relationship may become cyclical in truck drivers, as the poor cardiometabolic profile of truck drivers such as high prevalence of obesity can result in chronic inflammation, which in turn can affect autonomic function. Fatigue has also been linked to chronic inflammation, affecting both the periphery and the CNS (Lee & Giuliani, 2019). This may provide a mechanism underlying reduced SBP reactivity seen in drivers with higher persistent fatigue and anxiety symptoms but requires further investigation.

4.1 | Strengths

Strengths of this study include the novelty and size of the sample. Our large sample size enabled us to adjust for a range of important covariates and retain statistical power. As mentioned, truck drivers are an under researched population group who experience high levels of psychological stressors when on the road. Our recruited sample is comparable with truck drivers in the United Kingdom (median age 49.5 years, 98.7% male), as truck driving is a male-dominated industry comprising an ageing workforce (Freight Transport Association, 2018). This is relevant as the burden of CVD is higher for men than it is for women (Townsend et al., 2015). Both the Stroop test and Mirror Tracing task are widely used psychophysiological acute stressor tasks, which can allow for comparable results between studies. A further benefit is that data collection took place in the participants' own worksite setting, alleviating some potential "white coat syndrome" effects, and potentially enhancing ecological validity. Another strength is that the depression, anxiety, and fatigue scores were quantified using widely utilized and validated measures.

4.2 | Methodological considerations

As with all studies, the results of our analyses should be viewed in light of the following considerations. Limitations of this study include the laboratory stress which may not mimic natural stressors such as driving, preventing generalizability, though it is argued the controlled setting where the task was administered removes some potential variability, which aids in determining associations. Future studies should aim to replicate our findings in a field

setting with "real-life stressors." Furthermore, the stressor task included only two measures of BP and HR, which meant it was difficult to remove an outlier if there was measurement variability; however, if there was an outlier (>2SD), the participant was removed from the analyses. No cardiovascular measures occurred during the Stroop task, so we were unable to determine the cardiovascular response to this task. The analyses reported herein were cross-sectional, inhibiting the identification of causality. Also, the stress tasks were not counterbalanced across participants, which therefore could have resulted in an order effect. Though the results were statistically significant, the overall effect sizes for HADS-A and persistent fatigue were relatively small; however, these effect sizes are in line with similar studies (de Rooij et al., 2010). We only assessed symptoms of depression and anxiety and did not address questions about previously diagnosed depression or anxiety, and anti-depressant, sedative, or anti-convulsant use. Additionally, to obtain a thorough understanding of the effects of depression, anxiety, and fatigue on cardiovascular stress reactivity, analyzing further markers of stress such as cortisol and plasma cytokine responses would have been beneficial. However, given the constraints on data collection in the workplace environment, and the opportunistic nature of our study, this was not possible. Likewise, given the main trial protocol, we were unable to complete beat-to-beat assessments of CVR; however, brachial BP monitoring and HR assessment have been extensively used to accurately assess cardiovascular stress reactivity (Carroll et al., 2007). It should also be noted that there is a general lack of consensus within the literature on what pre-determined threshold specifically defines a "blunted" CVR. Finally, a control group was not feasible due to the SHIFT trial protocol, which means this study is only generalizable to truck drivers, who are at high-risk of stress exposure. However, the effects of these associations with depression and anxiety have been similar to other population-based studies (Phillips, 2011). Future stress reactivity research should therefore focus on population-based studies particularly in self-reported fatigue measures.

5 | CONCLUSION

In conclusion, we found that in a population of truck drivers from the United Kingdom, there is a significant negative relationship between stress induced SBP reactivity, with anxiety and persistent fatigue symptomology, indicating that increased persistent fatigue or anxiety symptoms relates to lower SBP reactivity to stress. This study provides further evidence to the literature encompassing attenuated CVR and mental health. Future research should utilize prospective designs to establish the cause and effect of these associations, and this in turn will provide clearer guidelines in determining the focus of future health interventions to protect both mental and cardiovascular health.

ACKNOWLEDGEMENTS

The authors would like to thank all the truck drivers who participated in this study.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Amber J Guest: Conceptualization; Data curation; Formal analysis; Methodology; Writing-original draft. **Stacy A Clemes:** Funding acquisition; Project administration; Supervision; Writing-review & editing. **James A King:** Funding acquisition; Project administration; Supervision; Writing-review & editing. **Yu-Ling Chen:** Data curation; Project administration. **Katharina Ruettger:** Data curation; Project administration. **Mohsen Sayyah:** Data curation; Project administration. **Aron Sherry:** Data curation; Project administration. **Veronica Varela-Mato:** Data curation; Funding acquisition; Project administration. **Nicola J Paine:** Conceptualization; Formal analysis; Methodology; Supervision; Writing-original draft; Writing-review & editing.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the Supporting Information section.

TABLE S1 Sample characteristics $N = 322$

TABLE S2 Sensitivity analysis T-Test between Included and excluded participant's SBP/DBP/HR reactivity to the stress task, based on the characteristics presented below

How to cite this article: Guest AJ, Clemes SA, King JA, et al. Attenuated cardiovascular reactivity is related to higher anxiety and fatigue symptoms in truck drivers. *Psychophysiology*. 2021;58:e13872. https://doi.org/10.1111/psyp.13872