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What is the role of Stress Cardiovascular Reactivity in Health Behaviour Change? A
Systematic Review, Meta-Analysis and Research Agenda

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Objective: The stress reactivity hypothesis posits that the extremes of exaggerated and low or blunted cardiovascular reactivity (CVR) to stress may lead to adverse health outcomes via psychophysiological pathways. A potential indirect pathway between CVR and disease outcomes is through health-related behaviour and behaviour change. However, this is a less well understood pathway. **Design:** A registered systematic review was undertaken to determine the association between cardiovascular reactivity (CVR) and health behaviour change, as well as identify mediators and moderators. Eight papers that met the inclusion criteria, focused on smoking cessation and weight loss, were identified. **Results:** Pooling data from studies exploring the prospective relationship between CVR (as systolic blood pressure) and smoking cessation found that exaggerated CVR was associated with smoking relapse (Hedges' $g = 0.39$, $SE = 0.00$, 95% CI 0.38 – 0.40, $p < .001$; $I^2 = 0\%$; $N = 257$) but did not find evidence that CVR responses were associated with changes in weight. In order to advance our understanding of reactivity as a modifiable determinant of health behaviour change, our review recommends exploring the association between CVR and other health behaviours, to determine the influence of blunted reactivity versus low motivational effort identify mediators and moderators and determine the focus of interventions.

Keywords: cardiovascular reactivity, reactivity hypothesis, stress, behaviour change, smoking cessation, weight

Psychological factors are postulated to influence health through two broad pathways: direct and indirect. Thousands of studies and many reviews have examined these pathways, but with respect to cardiovascular reactivity, few have examined the interplay of direct and

indirect pathways as influences on health'. In this review we examine the relationship between cardiovascular reactivity (a direct pathway) and health behaviour changes in smoking status and weight (indirect pathways).

Psychological direct pathways influencing health

Reactivity Hypothesis

Research has largely focused on the effects of cardiovascular stress reactivity on health outcomes, rather than the processes of health behaviour change. For several decades, stress and health research has focused on the reactivity hypothesis, which proposes that prolonged or exaggerated cardiovascular reactivity (CVR) to psychological stressors can promote the development of cardiovascular disease (Blascovitch & Katkin, 1993; Obrist, 2012). Over time, heightened and prolonged cardiovascular reactivity is posited to change cardiovascular structure and functioning, which can lead to a number of adverse health outcomes. These include sustained hypertension (Obrist, 2012), cardiac and vascular hypertrophy (Lovallo and Gerin, 2003), oxidation of low-density lipoproteins (Raitakari et al., 1997), increased serum concentrations of both pro-inflammatory cytokines (Georgiades, 2007) and blood insulin (Nazzaro et al., 2002). Whilst empirical studies provide support for negative health outcomes as a direct pathway from acute stress reactivity-related adaptations of the cardiovascular system, few have looked at the influences of health behaviour in this context.

Exaggerated Cardiovascular Reactivity

Exaggerated cardiovascular reactivity refers to empirically demonstrable cardiovascular response patterns to acute stress that are higher than those observed during states of homeostatic function during stress. Exaggerated reactivity is associated with a range

of negative health outcomes, which include hypertension (Carroll et al., 2009; Carroll et al., 2001; Everson et al., 1996; Markovitz et al., 1998; Matthews et al., 1993; Newman et al., 1999; Treiber et al., 2003), atherosclerosis (Barnett et al., 1997; Everson et al., 1997; Lynch et al., 1998; Matthews et al., 1998), increased left ventricular mass and/or hypertrophy of the heart (Georgiades et al., 1997; Kapuku et al., 1999; Murdison et al., 1998), and cardiovascular disease mortality (Carroll et al., 2012b). Both qualitative reviews and meta-analyses suggest an associated pathway between exaggerated reactivity and poor future cardiovascular health status and outcomes (Chida and Steptoe, 2010; Gerin et al., 2000; Schwartz et al., 2003; Taylor et al., 2006; Treiber et al., 2003).

Blunted Cardiovascular Reactivity

Blunted cardiovascular reactivity is by definition an empirically demonstrable cardiovascular response pattern which is comparatively lower than typical states of homeostatic function during stress. Historically, the emphasis placed on the pathway between exaggerated reactivity and negative health outcomes implied that low or blunted reactivity to acute stress was a benign, adaptive or even protective response (Gerin et al., 2000). Recent experimental evidence has instead suggested that low cardiovascular reactivity to stress may also have serious adverse consequences for health outcomes and health-related behaviour change. For example, comparatively low levels of cardiovascular reactivity to acute psychological stress are associated with smoking status (al'Absi, 2006; al'Absi et al., 2005; Girdler et al., 1997; Phillips et al., 2009; Sheffield et al., 1997), exercise addiction (Heaney et al., 2011), Schwerdtfeger & Rosenkaimer, 2011; York et al., 2007), eating disorders (Ginty et al., 2012), poorer cognitive function (Ginty et al., 2011; Ginty et al., 2012), as well as alcohol and other substance addiction status (Brenner and Beauchaine, 2011; Lovallo, 2005;

Lovallo et al., 2000; Panknin et al., 2002). Such small cardiovascular responses to stress have also been shown to be associated with poorer health outcomes, including poorer self-reported health (De Rooij and Roseboom, 2010; Phillips et al., 2009), obesity (Carroll et al., 2008) and depression (Brinkmann et al., 2009; Carroll et al., 2007; de Rooij et al., 2010; Phillips et al., 2011; Rottenberg et al., 2007). Lower cardiovascular stress responses have also been associated with personality traits that are indicative of future negative health outcomes, such as Type D personality (Howard et al., 2013) and neuroticism (Hughes et al., 2011), which have been associated with an elevated risk of future cardiovascular events. The mechanism associated with blunted reactivity is unknown and it is important for studies to distinguish between reactivity that is blunted (a sub-normal response pattern) and low reactivity, which may be biologically expected in response to certain events and situations. Reactivity may be low due to lower motivational effort or motivational dysfunction, reduced awareness or perception of stress, task difficulty level or a reduced physiological level of difficulty in responding (Phillips, et al., 2012).

The extremes of exaggerated and blunted reactivity are considered maladaptive responses to stress (the reactivity hypothesis, Blascovich & Katkin, 1993) and could promote allostatic overload. Exaggerated and blunted reactivity have been theorised to reflect different levels of effort and motivation (Lovallo et al., 2011), with blunted reactivity purported to reflect lower motivational effort and exaggerated being indicative of high levels of effort. More recently, the revised reactivity hypothesis (Phillips et al., 2012; Phillips, Ginty, Hughes, 2013) has theorised that stress responses represent a systems-level response to threats to homeostasis, further suggesting that the midrange of stress intensity may be the most adaptive response to constantly challenging environmental demands. Deviations from the normative midrange response to stress are purported to signal poor systems integration

and therefore diminished homeostatic control. By extension, the most optimally healthy response to stress may be a moderate level of cardiovascular reactivity, described in terms of a 'robust stress response' (Lovallo, 2011) and as an indicator of resilience (al'Absi, 2018), which has important implications for the design of health psychology interventions and therapeutic approaches. Whilst the majority of empirical and theoretical work to date has examined the association between stress CVR and health outcomes, less attention has been given to the relationship between CVR and the enactment and maintenance of health-related behaviour change. It is possible that the poor affective and behavioral regulation associated with low or exaggerated reactivity in response to stress (Lovallo, 2011), may be a direct pathway to poor health behaviors (e.g. smoking or excessive consumption of alcohol) as coping responses, which in the long term have a cumulative and direct influence on poorer health outcomes. In addition to exaggerated or blunted stress reactivity, poor stress recovery (defined as sustained cardiovascular activation above baseline levels during the post-task recovery period) also has important implications for health status and outcomes, which may be mediated through poor health behaviours. Chida and Steptoe (2010)'s meta-analysis showed that greater reactivity and poor recovery from stress were associated longitudinally with poor cardiovascular status (including elevated blood pressure, hypertension, left ventricular mass, subclinical atherosclerosis, and clinical cardiac events). Incident hypertension and increased carotid intima-media thickness were more consistently predicted by greater stress reactivity and poor stress recovery, respectively, whereas both factors were associated with higher future systolic and diastolic blood pressures. Such findings suggest that greater responsiveness to acute mental stress and slower recovery have adverse effects on an individual's future cardiovascular risk status, supporting the use of methods of managing stress responsiveness in the prevention and treatment of cardiovascular disease. It is therefore

important to understand how our acute stress responses might be implicated not only in health outcomes, but also how they influence health-related behaviours.

Indirect pathways

Psychological factors also impact on health through indirect pathways, namely through health behaviours including smoking and behaviours that impact on weight and energy balance (e.g. snacking, physical activity). This review proposes that the indirect pathways themselves are subject to direct physiological influences of cardiovascular reactivity. For example, it is well established that perceived stress plays a role in the early withdrawal stage of smoking cessation, however there are few studies looking at the psychophysiological predictors of the maintenance of smoking cessation or other areas of health behaviour change.

CVR and Smoking

Stress plays an important role in understanding the influences that drive smoking cessation and continued smoking. Abstinence from smoking is associated with negative psychophysiological states including craving, anxiety, depression, restlessness, irritability, and difficulty concentrating (Hughes & Hatsukami, 1986). The intensity of withdrawal symptoms is influenced by situational demands of acute stressors, demanding challenges, or smoking-related cues, as well as the person's disposition and skills (e.g. coping resources, history of psychopathology), their level of nicotine dependence and time since the last cigarette or intake of nicotine. The detrimental effects of abstinence on mood may exacerbate the experience of stress and continued smoking (Shiffman, 2005). However, the role of psychophysiological responses to behavioural challenges and its impact on relapse risk following abstinence from smoking are less known.

CVR and Weight Changes

Both adiposity and the development of obesity have been shown to be linked with psychological distress and stress reactivity. Abdominal obesity has been associated with both exaggerated (Goldbacher et al., 2005; Steptoe and Wardle, 2005) and blunted reactivity (Hamer et al., 2007; Carroll et al., 2008; Phillips, 2011; Phillips et al., 2012). Low cardiovascular reactivity has been shown to be associated with a greater body mass index, more abdominal adiposity and greater likelihood of being obese. Low reactivity may be attributable to a blunted sympathetic nervous system response to acute challenge (Carroll, Philips & Der, 2008; Philips et al., 2012); there is reasonably consistent evidence that the sympathetic nervous systems of individuals who have become obese, are less responsive to stimulation (Carroll et al., 2008; Tentolouris et al., 2006), although this is characterized by high basal sympathetic nervous system activity (Carroll et al., 2008; Tentolouris et al., 2006), and higher basal cortisol levels (Bjorntorp, 1993; Rosmond & Bjorntorp, 2000).

The Present Review

Whilst it is widely acknowledged that exaggerated and low or blunted reactivity have negative effects on health outcomes (Philips, et al., 2011), the role of CVR as a predictor of health behaviour change is less well understood. A better understanding could guide intervention development e.g. by identifying the crucial time point for intervention, as well as a means of predicting less successful health behaviour change and identifying who might benefit from more intensive intervention, in addition to helping expand our understanding of modifiable determinants of health behaviour. The purpose of this systematic review was to review the available evidence on the associations between cardiovascular response to laboratory induced acute psychological stress and future health behaviour change status by

analyzing all of the available prospective studies. We also aimed to examine whether post-stress recovery is related to subsequent behaviour change success.

Method

Search Strategy

We conducted a systematic review to explore the association between cardiovascular reactivity and health behaviour change. The protocol was pre-registered with PROSPERO (CRD42017073485).

Information Sources

We searched Medline, PsychINFO, PsychArticles, Web of Science, PubMed Central, PubMed Central (Europe), ScienceDirect, and EBSCO databases. We used search terms from each of the PICO domains, including participants ('adults'), Interventions (Cardiovascular reactivity, reactivity hypothesis, CVR, blunt* reactivity, blood pressure, stress reactivity, reactive, heart rate variability, exaggerated cardiovascular reactivity, systemic resistance reactivity, total peripheral resistance (TPR) or rate-pressure product (RPP), pulse transit time reaction, finger pulse amplitude, mean arterial pressure), Comparison (Clinical trial*, cohort, case control, observational, experiment*) and Outcomes (Health*, behaviour*, behavior*change, physical activit*, exercise, sedentary, smoking cessation, relapse, stress, diet, obesity, self-report* health, alcohol,motivation, self-regulation).

For our full search strategy and search terms, see table 1 in the online supplementary materials and our Open Science Framework page (<https://osf.io/vb2qr/>)

This review has been reported in line with AMSTAR II criteria (Shea, Reeves, Wells, Thuku, Hamel, Moran et al.2017) for systematic reviews, to allow for evaluation and to

reduce the potential for bias in the reporting of this review. For full reporting on our review methods, including the screening procedure, data handling and extraction processes, in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Protocols (PRISMA-P) guidelines (Moher, Liberati, Tetzlaff & Altman, 2009) see table 2 in the online supplementary materials for a summary of the *PRISMA 2009 statement of methodological reporting*.

Study Eligibility Criteria

In press and published (in English language) studies from 1995 to February 2020 were included. Our inclusion criteria was for studies of adult populations describing prospective studies including at least one of the following objective measurements of CVR in response to an acute stressor test: blood pressure (BP), heart rate (HR), total peripheral resistance (TPR) or rate-pressure product (RPP), pulse transit time reaction, finger pulse amplitude, pre-ejection period (PEP) or mean arterial pressure. On the basis of existing reviews of CVR and future health status, we defined an acute laboratory stressor as a task that lasted no more than one hour and did not serve a function outside the laboratory setting. Included studies must have measured changes in at least one of the following health behaviours using either objective measures or self-report: physical activity, sedentary behaviour, diet, smoking or drinking alcohol (our primary outcome measure). In terms of secondary outcome measures, we also sought to gather data on mediator and moderator variables known to be associated with reactivity, such as age, gender, co-morbidities, socio-economic status, smoking status, substance use and the role of medication use (Carrol et al., 2003; Philips et al; 2012)

Screening Procedure

One reviewer (AC) screened all retrieved records identified through initial searches against eligibility criteria in two stages. Initial screening was based on titles, abstracts and keywords and the second reviewer (DS) screened a random 20% of the total titles and abstracts to establish consensus. All studies were screened by one author (AC) and the final selection of studies to take forward to the next stage of the review were agreed by two reviewers (AC, DS). No additional studies were added to the corpus of studies at this stage. Following initial screening, full-text versions of all potentially relevant studies were retrieved and reviewed independently and screened against all inclusion and exclusion criteria by two reviewers (AC and DS) (see online supplementary material: figure 1 *PRISMA flowchart*).

Data Extraction and Selection

Data extraction occurred in two stages; (1) extracting general study data using a data extraction form and (2) assessing the quality and risk of study bias of the review studies using the National Heart, Lung and Blood Institute's Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies and the Cochrane risk of bias assessment tool.

Data Management

Before analysis, the following decisions were made; the duration of the follow up periods were calculated from the time when the participants received the CVR inductions. The follow up period was defined as the period during which contact ceased with participants from the end of the stress test, to the time of the measurement of the behaviour. Where studies reported findings from more than one stress test, we extracted data from acute psychological stress tests (e.g. Paced Auditory Serial Addition Test) in favour of physical stress tests (e.g. deep knee bends). Where two psychological stress tests were reported, we used findings from social psychological tests (e.g. public speaking, social challenge or

competition) over cognitive (e.g. maths tests, mirror tracing) psychological tests (Lepore, 1998). We extracted effect sizes between the cardiovascular response predictor during a) stress reactivity and b) stress recovery and subsequent health behaviour change outcomes. An effect size reflects the effect between the cardiovascular response predictor and health behaviour change outcome or was calculated from the difference in subsequent behaviour change outcomes between the high and low cardiovascular response groups. Separate data extraction was carried out for associations of stress recovery. We sought to retain data on the primary outcome measure only.

Meta-analysis technique

When primary studies provided sufficient data or such data were obtained by other means (personal communication with the author or indirect calculation i.e. zero effect used where the authors reported non-significant findings, Higgings, White & Wood, 2008), effect sizes were calculated for each of the outcomes. We examined the effects on each of the outcomes after intervention (i.e. the first time point following the stress test) and at follow-up (the last time point assessed). The conservative approach of Hedges' g was used to calculate effect sizes for each of the studies. However, in some cases, where there was insufficient data reported to calculate an effect size and the findings were not statistically significant, the results were then treated as having zero effect and the sample size was simply added to the total sample size when calculating the combined effect size. Because we compared the effects of CVR as a predictor of health behaviour change, we used random effects modeling (restricted maximum likelihood method).

Heterogeneity

Heterogeneity was investigated using a chi-squared test (significance level: 0.1) and Higgins I^2 statistics and was found to be high, which may limit the conclusions we can draw from our meta-analyses. In the spirit of open science, we want to provide full disclosure about the data we obtained and the analyses we conducted, as well as to highlight the limited conclusions that can be drawn from the meta-analysis thus far. Therefore, we report a narrative summary of the review study findings alongside commentary of how we can develop the evidence base in this area and to encourage replications and larger scale studies.

For full information on the decisions taken regarding heterogeneity for the meta-analysis, see the online supplementary materials. In summary, we performed meta-analyses of studies with CVR as a predictor of behaviour change as individual weight and smoking cessation datasets.

Meta-bias

Confidence in cumulative evidence

The quality of evidence for primary outcomes from each of the review studies was assessed using the Quality Assessment Tool for Observational cohort and case control studies (National Heart, Lung, and Blood Institute, ND; see supplementary materials). The overall quality for the corpus of studies was judged as high to moderate quality, indicating a high to moderate level of confidence that the true effect lies close to the calculated effect. No studies were excluded on the basis of quality.

Data synthesis

Effect sizes were extracted for each study and where necessary they were calculated using means, standard deviations and sample sizes at baseline and post-intervention of experimental and control conditions (Decoster & Claypool, 2004). Where such statistics were

missing, we used F-statistics, t-values and p-values; we calculated effect sizes in this manner for Swan et al. (1993), al'Absi et al. (2005), Steptoe and Wardle (2005); Carroll et al. (2008) and Philips et al. (2012). Data was analysed using Meta-Essentials (van Rhee, Suurmond & Hak, 2017).

Results

A total of 1,259 potential articles were initially identified from the searches following the removal of duplicates. After screening the abstracts, 214 full text articles were retrieved and assessed for eligibility against the inclusion criteria. Eight papers, including data from an unpublished study by one of the review authors (DS), met the inclusion criteria and were included in the review for analysis (see online supplementary materials: table 3: *Summary of characteristics of included studies*). There was no evidence of publication bias (see online supplementary materials, figure 3).

Differences in Operational Definitions of Cardiovascular Reactivity

This review included a total number of 3,077 observations based on data from adult participants (18 years or older). Sample sizes varied from $n = 23$ to $n = 1,647$ and were conducted in UK ($k = 3$), the US ($k = 3$), Netherlands ($k = 1$) and Thailand ($k = 1$).

In order to better understand how cardiovascular reactivity has been operationalised, we extracted data on the study stress reactivity tests including: (1) the type of measures taken to index CVR and how reactivity was calculated, (2) study setting, and (3) nature of the acute stressor task (see online supplementary materials: *Table 3*).

Narrative summary: smoking cessation and CVR

In total, three studies have explored the prospective association between CVR and smoking relapse (N = 329). Swan et al. (1993) found that those who relapsed after a smoking quit attempt ($n = 173$) had significantly higher CVR on the cognitive stress challenge prior to the quit attempt compared with those who were abstinent after a year ($n = 59$) ($g = 0.39$, CI 0.37 - 0.41). Higher CVR was also associated with shorter time to relapse. Emmons et al. (1989) assessed CVR to stress approximately one week prior to the initiation of a smoking cessation program and 6 months following cessation ($n = 25$). They recruited a sample of smokers with high levels of nicotine addiction to attend a smoking cessation program consisting of six to eight sessions, with cessation occurring on a planned quit day early in the program. Relapse at 6 months was significantly associated with exaggerated stress responses at baseline ($g = 0.35$, CI 0.17 - 0.53).

In a non-intervention observational study of smoking cessation attempts, al'Absi et al. (2005) found small to moderate sized associations in favour of blunted CVR as a predictor of smoking relapse for SBP and DBP as an index of CVR ($g = -0.26$, SE = 0.03, CI -0.32 - -0.20, $n = 72$; $g = -0.73$, CI -0.80 - 0.66, $n = 72$, respectively) but not for HRR. In this study, in contrast to the other two studies where the stress reactivity testing was conducted pre-cessation, the laboratory stress session was conducted 24 hours after the onset of abstinence from smoking. Smokers who relapsed within 4 weeks after quitting demonstrated blunted BP responses to acute stress and experienced more intense craving during the baseline laboratory stress session.

Cardiovascular Reactivity and Smoking Cessation: Meta-analysis

Meta-analysis of the two intervention studies (Emmons et al., 1989; Swan et al., 1993) found a significant, medium effect for exaggerated CVR as a predictor of smoking

relapse (Hedges' $g = 0.39$, SE 0.00, 95% CI 0.33 – 0.45, $p < .001$; $Q = .22$, $I^2 = 0\%$; $N=257$), using SBP as a marker of CVR. DBP and HRR associations were not significant (see online supplementary materials *Figure 3*. Effect sizes, confidence intervals and forest plot of effect sizes for the impact of CVR (SBP) on smoking relapse rates).

Cardiovascular Reactivity and Weight Change

Five studies examined the association between CVR and weight changes ($N = 2,748$), four of which were population observational designs ($N = 2,725$), and one was an intervention study ($n = 23$; Endrighi et al., 2015). Phillips et al.'s (2012) Dutch Famine study ($n = 725$) reported that low stress reactivity during the acute stressor task was prospectively associated with an increased likelihood of becoming or remaining obese in the subsequent 4-7 years. Additionally, participants with high resting HR activity were more likely to be, become or remain obese (i.e. ≥ 30 kg/m²) in the next 4-7 years. This association remained significant even with adjustment for key potential confounders of CVR (e.g. gender, age, SES.). However, CVR was not found to be associated with any changes in BMI.

Similarly, in another large community sample ($n = 1,647$) the West of Scotland Study (Carroll, Phillips, & Der, 2008) found that low CVR (indexed as HR reactivity) was associated with an increased likelihood of becoming obese in the following 5 years ($g = -0.03$, $p < .001$). The largest increases in BMI and waist to hip ratio were in the youngest cohort of the sample, with the oldest cohort registering a slight decrease. Whilst CVR (HR reactivity) did not predict the rise in either BMI or waist-hip ratio in the five years between the third and fourth follow-ups, it was reported that HR reactivity predicted whether participants became obese or remained or became non-obese, OR (95% CI) = 0.97 (0.95 – 0.99), $p = .02$; with high HR reactivity associated with a reduced likelihood of becoming obese. Steptoe and

Wardle (2005)'s study of 225 civil servants found that neither the upward drift in BMI or waist-to-hip ratio over a 3-year follow-up period were associated with the earlier measures of cardiovascular reactivity (indexed by SBP, DBP, HR, cardiac index and TPR). In a sample of 102 healthy staff and students at a University in Thailand, Yuenyongchaiwat & Sheffield (*under review*; 2017) found that blunted reactivity (indexed by SBP, but not DBP, HR, CO or TPR) was associated with increases in BMI at 40 month follow up ($g = -0.31, p < .001$).

Meta-analysis

Given the variation in sample size and characteristics, as well as the differences in weight change outcome measures, we pooled data of the four observational studies (Philips, 2011; Carroll, et al. 2008; Yuenyongchaiwat, *under review*). The pooled effect estimate from the meta-analysis favoured an association between blunted CVR, as measured by SBP, and weight changes, but was not statistically significant with high heterogeneity (Hedges' $g = -0.08$, $SE = 0.07$, 95% CI $-0.32 - 0.15$, $p = 0.27$; $Q=219.99$, $I^2= 99\%$; $N = 2,699$) (see *Figure 4. Effect sizes, confidence intervals and forest plot of effect sizes for the impact of CVR (SBP) on weight changes*). DBP and HRR were not significant

Stress Recovery and Subsequent Behaviour Change Status

We found three studies reporting prospective analyses of stress recovery effects as a predictor of health behaviour change (weight changes: Steptoe & Wardle, 2005; Endrighi et al., 2015; smoking cessation, al'Absi et al., 2005). Poor stress recovery (defined as sustained cardiovascular activation above baseline levels during the post-task recovery period) was reported to be associated with behaviour change status in the overall analyses in all of the three studies that reported measures of stress recovery. In Steptoe and Wardle (2005)'s study, slower stress recovery effects of heart rate recovery (HRR) and DBP recovery predicted

increases in waist to hip ratio. However, in the Endrighi et al. (2015) study, no pre-post weight loss differences in SBP or DBP were observed during stress recovery, however improved HRR was associated with improved weight loss and changes in fat mass. HRR improved after weight loss at 9 weeks. In terms of smoking relapse, al'Absi et al. (2005) reported significant recovery effects between quitters and abstainers, with slower stress recovery being associated with relapse.

Mediator and moderator analysis

Overall, few studies reported moderators. Based on the limited number of included studies, the available statistical power for moderator analyses was limited and analyses were not conducted. However, our review identified potential moderators as: (1) gender, (2) study quality, (3) socio-economic status, (4) hypertension, (5) negative affective state (e.g., motivation, self-efficacy, emotional response- anxious and depressed mood), (6) age, (7) type of stress task (i.e. cognitive vs behaviour challenge tests) and (8) the type of reactivity measure. Additional moderators for smoking cessation include bupropion for smoking, polysubstance use and emotional disposition and for weight changes, these include early life adversity, abdominal obesity and appetite.

Discussion

Whilst other reviews of cardiovascular stress reactivity (CVR) and health outcomes exist (Chida & Steptoe, Chida & Hamer; Carroll, et al., 2012; Philips et al., 2014), this paper describes the first systematic review of the role of CVR as a predictor of health behaviour change, namely smoking cessation and weight/adiposity changes. We aimed to quantify and synthesize prospective data examining behavioural and behaviour-related outcomes with CVR reactivity and recovery. Although we are limited in the conclusions we can draw from

the available literature, our review highlights lines of research enquiry to test the reactivity hypothesis in the context of health behaviour change maintenance.

Indirect pathways

Stress Reactivity and Smoking Cessation Relapse

Interventions should focus on supporting individuals in appraising and managing stress and the associated stress reactivity that occurs in everyday life, as well as addressing stress in the context of health behaviour change, particularly where the health behaviour might be used to cope with stress e.g. planning interventions to reduce stress-induced smoking or eating. Situational factors, such as the role of smoking cue reactivity, are also important in understanding the role of CVR in smoking cessation (Saladin et al., 2012). For example, individuals who remained abstinent for at least three months had lower heart rate responses to smoking-specific situations prior to cessation than those who relapsed (Abrams et al., 1998). Additionally, Niaura et al. (1989) combined a laboratory test of both stress and cue reactivity (and therefore excluded from our review) and found that low heart rate reactivity measures predicted smoking relapse three months later. Higher heart rate variability has also been found to be higher both pre-cessation and at the four-week follow up point among quitters compared to relapsers (Harte and Meston, 2013). Further analysis in this study found that successful quitters demonstrated increases in HRV over time, whereas relapsers did not exhibit changes in HRV. These studies of CVR responses provide some preliminary support for a potential curvilinear relationship between CVR responses and health behaviour change, whereby either extreme of responsivity is associated with less successful behaviour change and poorer long-term health outcomes.

There are additional reasons why focusing on stress appraisals and reactivity are important therapeutic smoking cessation targets. Acute psychological stressors increase smoking rates and the risk for relapse (Willis et al., 2002; Carey et al., 1993; Shiffman et al., 1996), which manifest as increased intensity in withdrawal symptoms (Koval & Pederson, 1999; Perkins & Grobe, 1992) and an increased desire to smoke (Perkins et al., 1994). Compared to non-smokers, smokers exhibit a blunted cortisol response to stress (al'Absi et al., 2003). These diminished systolic BP and cortisol responses to stress in smokers, which are independent of acute exposure to nicotine, indicate alterations in the stress response resulting from chronic exposure to nicotine that are independent of smoking withdrawal.

Stress reactivity and changes in adiposity

Three studies included in this review examining CVR and changes in weight reported small significant effects in favour of low levels of CVR predicting future weight gain (Philips et al., 2012; Carroll, Philips & Der, 2008), and one null finding. It is well known that weight and adiposity measures are affected by the interplay of a wide range of biopsychosocial factors over time, and whilst we identified some potential mediating and moderating influences worthy of further investigation e.g. age, SES, hormones (ACTH, leptin), we should also determine the relative contribution of factors other than stress CVR that also explain long-term weight changes. These may include physical factors of gender, leptin and smoking status, as well as automatic and reflective psychological processing factors. Accordingly, we recommend that stress CVR and adiposity changes are not studied in isolation, but rather future studies should examine the interplay of other known moderating and mediating influences on health behaviour change, in order to determine the processes and contexts in which stress reactivity impacts on health behaviour change.

An agenda for advancing CVR health behaviour change research

Our review highlights methodological priorities to advance our understanding of the direct and indirect effects of CVR on health.

The first priority is to explore the role of CVR in relation to other types of health behaviour change, particularly as there is evidence to support the role of stress in other health behaviours. For example, in other addictive behaviours, the role of stress in the maintenance of and relapse to drug abuse is well-established (Sinha, 2001; Back et al., 2011; Garland, Franke & Howard, 2012) and blunted hypothalamic-pituitary-adrenal axis stress responses have been observed in individuals with alcoholic addiction and other substance over-users (Lovallo et al., 2011; Adinoff et al., 2005). Physical activity is known to change stress responses, as it appears to have an attenuating effect on CVR at resting levels, (de Geus and van Doornen, 1993; de Geus et al., 1993; Schuler and O'Brien, 1997) and higher cardio respiratory fitness levels are associated with HR responses to psychological stress (Clayton, 1991; Boutcher and Nugent, 1993; Spalding et al., 2000), although multiple meta-analyses and larger clinical studies have provided different conclusions (Crews and Landers, 1987; Forcier et al., 2006; Hamer et al., 2006; Jackson and Dishman, 2006; Sloan et al., 2011), due to study differences in participant demographics, research methodologies, and outcome measures.

Our review highlights the need for future high quality, well-powered epidemiological and experimental studies to test the relationship between CVR and health behaviour change as both linear and curvilinear, as well as to determine whether exaggerated or blunted cardiovascular responses are indeed associated with less successful health behaviour change,

which would extend the reactivity hypothesis. Previous studies have only used linear models to explore these associations (Carrol et al., 2008; Philips et al., 2011; Ginty et al., 2013).

We recommend that future studies seek to establish gold standard definitions and observational cut off points that distinguish between reactivity that is blunted (which implies sub-normality and may indicate motivational dysfunction) and comparatively low levels of reactivity (which may be biologically normal within a given context) in order to progress the science in this area. Empirical observations that suggest low levels of reactivity may be an indicator of low motivational effort or motivational dysfunction in the context of health behaviour change as low reactivity may signify motivation-contingent behaviors or tasks that require lower effort (Phillips et al., 2012). However, other explanations of low reactivity have included reduced awareness or perception of stress (Higgins & Hughes, 2012), the impact of experimental task difficulty (Richter & Gendolla, 2005). Blunted reactivity has been found to predict poor performance during motivation and effort contingent tasks, such as lung function spirometry (Carroll et al., 2013; Carroll, Bibbey et al., 2012; Crim et al., 2011). A recent study by Chaunry, Williams & Whittaker, (2019) added further evidence of blunted reactivity as a physiological marker of poor behavioural regulation, which may indicate why individuals with blunted reactivity are at increased risk of developing negative health outcomes as a result of weight increases and addictions. We also found variation in terms of the timing (baseline to peak reactivity and baseline to end of task) of reactivity measures; establishing consensus on standardised measurement definitions is key to developing a progressive science within stress reactivity work. Tests of the Reactivity Hypothesis usually assume that responses to acute stressors in the laboratory provide an indication of how individuals respond to stressful events in their daily life (Johnston et al., 2008). We would recommend incorporating laboratory tests of stress in the context of health behaviour change

(i.e. cue reactivity in terms of imagery of stressful situations that may lead to smoking relapse).

Many of the mechanisms behind the association between CVR and health behaviour change are unknown. Future work should seek to confirm the role of specific stress-response biological pathways (e.g., blunted HPA and for addiction, endogenous opioid system responses, al Absi, 2018). Whilst our review suggests potential mediator and moderator variables, future studies are needed to inform the direction of clinical and intervention studies to assess the likelihood of an individual to change their behaviour. From a health psychology intervention and therapeutic point of view, it is important to note that CVR and stress reactivity responses are modifiable through targeted psychological interventions (al'Absi, 2018). Should future research confirm the role of CVR as a robust predictor of health behaviour change, then this may be used to tailor interventions. For a full research agenda with recommendations for methodological and analytical techniques, see table 6 *A research agenda for CVR and health behaviour change research* in the online supplementary materials).

Conclusions

In summary, this paper summarises the available evidence of stress cardiovascular reactivity as a predictor of health behaviour change and suggests that the relationship is context and the health behaviour dependent. There was some evidence that both exaggerated and low levels of CVR were associated with less successful behaviour change, which suggests a possible curvilinear rather than linear relationship. The nature of the relationship between CVR and health behaviour change should be studied using the specific future directions from our review to examine the precise processes and mechanisms of the

association between CVR and health behaviour. Our review highlights the importance of understanding the role of CVR in health behaviour change and the need for well powered studies, using multiple measures of CVR. Studies are needed in relation to a range of health behaviours in order to develop efficacious interventions for the appraisal and management of stress reactivity in the context of enacting and maintaining health behaviour change. Future research should also capture the role of regulatory processes known to be associated with CVR, for example emotional regulation, as depression and anxiety are both associated with blunted reactivity.

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