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Blood Pressure and Emotional Responses to Stress: Perspectives on Cardiovascular Reactivity

Clayton Hilmert* and Lexi Kvasnicka
North Dakota State University

Abstract

It has long been thought that increases in blood pressure in response to stress are associated with emotional responses to stress. The health implications of such an association are clear; excessive emotional reactivity leads to excessive cardiovascular reactivity (CVR), which is associated with cardiovascular disease. However, the data do not support a strong association between CVR and emotional responses to acute stress. This lack of support has lead to research that interprets CVR to stress in at least three different ways: (1) as a potential contributor to disease development, (2) as an index of active coping, or (3) as a multidimensional construct that is affected by cognitive appraisals of a situation. In this article, we review these separate perspectives on CVR and suggest that a multidimensional perspective of CVR and emotional responding to stress may help integrate the CVR – health, effort, and appraisal points of view.

> *…we may feel nearly sure that any sensation or emotion, as great pain or rage, which has habitually lead to much muscular action, will immediately influence the flow of nerve-force to the heart, although there may not be at the time any muscular exertion* (Darwin, 1898, p. 74).

A long held belief articulated here by Charles Darwin in *The Expression of Emotion in Man and Animals* (originally published in 1872) is that strong negative emotions directly influence the activity of the cardiovascular system. Despite this commonly held intuition, it has been difficult to demonstrate associations between emotions and cardiovascular reactivity (CVR) to stress. This is particularly true in laboratory stress-response research where reported correlations between emotional responses to stress and CVR are generally weak and inconsistent (*r*s range from 0.13 to 0.34; Feldman et al., 1999). Thus, the underlying psychology of CVR is unclear, and this has lead to inconsistent interpretations of cardiovascular responses to stress and the implications of these responses.

There are various possible reasons why correlations between negative emotional and cardiovascular responses to stress have been weak and inconsistent. Traditional measurement (Hilmert & Kvasnicka, forthcoming) and experimental methods (Gerin et al., 1999) may not be optimal for detecting this association. On the other hand, negative emotions may not be as closely linked to CVR as assumed. Other variables, like effort, may determine CVR (Wright & Kirby, 2001) and negative emotion may be unrelated or a minor determinant. It is also possible that associations between negative emotions and CVR are moderated by other variables, so that in certain circumstances, there is a stronger association than in others.

Because large magnitude CVR in response to stress has been implicated in the development of cardiovascular disease (Lovallo & Gerin, 2003), a great deal of research has sought to identify the individual and situational factors predictive of these responses. This way, we may be able to better identify those at risk of CVR-related disease. A better

understanding of the psychology underlying CVR that is harmful to health would potentially lead to improved interventions, allowing interventionists to identify the emotional, cognitive, or motivational factors that should be targeted.

There is an abundance of laboratory research relevant to considering the underlying psychology of CVR. This review is by no means intended to be comprehensive. Instead, our aim is to provide an overview of what has become a theory-rich area of study with important implications for health and to provide a framework within which some of the complexities of this area are highlighted. To these ends, we briefly discuss the most common measures of CVR and negative emotion. Then, we outline three of the primary perspectives taken in CVR research and consider the role of negative emotional responses in each perspective. We suggest an integrative, multidimensional perspective on CVR and psychological responses to stress that accounts for past inconsistencies in negative emotion and CVR findings. Finally, we consider some methodological and practical implications of this perspective.

CVR and Negative Emotion Measures

To examine how emotions and physiology are affected by an acute experience of stress, research has primarily relied on the laboratory setting. This is because it is difficult to manipulate acute stress outside the lab and it is difficult to measure physiological and emotional arousal during acute stress without a manipulation (cf. Ming et al., 2004). A set of laboratory tasks that has elicited reliable physiological and emotional responses is referred to as the Trier Social Stress Test (TSST) (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST has two components. First, participants prepare and perform a speech to an audience of evaluators. Second, participants perform mental arithmetic in front of the evaluators while the experimenter indicates calculation mistakes. Blood pressure and heart rate values are recorded during a pretask resting baseline period and during the stressful task. A common way to then calculate reactivity is to subtract the average baseline values from the corresponding average task values.

Earlier research focused on changes in heart rate as the primary CVR measure (see Fowles, 1982 for a review). More recently, blood pressure has been the focus of reactivity studies. Although systolic blood pressure and diastolic blood pressure (and to a lesser extent, heart rate) tend to follow similar patterns of reactivity, systolic blood pressure seems to be the most reactive component of CVR to psychosocial stress (Hilmert, Christenfeld, & Kulik, 2002a; Obrist, 1981; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). The theoretical and practical implications of focusing on these different components are not necessarily well established (Kamarck & Lovallo, 2003; Treiber et al., 2003). Therefore, unless otherwise noted, in the present article, CVR refers to (primarily systolic) blood pressure responses to stress in the lab.

Negative emotional responses to acute stress are often measured immediately after a laboratory stressor and sometimes immediately before the stressor so emotional reactivity, or changes in emotions can be assessed. Frequently, measures are standardized emotion scales (e.g., the State Trait Anxiety Inventory; Spielberger, 1985) or measures designed for the particular study (e.g., 'How anxious did you feel during your speech?').

Reliance on these largely unidimensional measures of multidimensional constructs may be one reason research has found only small to modest correlations between CVR and negative emotional responses to stress. That is, there are multiple determinants of blood pressure (e.g., heart rate, vascular resistance, stroke volume). High CVR in different individuals and situation may reflect changes in underlying physiological components

(Kasprowicz, Manuck, Malkoff, & Krantz, 1990) that are differentially associated with negative emotion. Also, self-reported assessments of discrete emotions like anxiety or frustration may not capture the complexities of emotional responses to stress (Russell & Barrett, 1999). Different dimensions of negative emotional responses to stress may be differentially related to CVR. In later sections, we consider these measurement issues in more detail and discuss how taking a multidimensional approach to CVR (e.g., Tomaka, Blascovich, Kelsey, & Leitten, 1993) and emotional reactivity may help clarify CVR and emotional response associations.

Perspectives on CVR

In the CVR literature, we identified three primary perspectives. These perspectives are largely independent, based on antecedent, concurrent, or subsequent factors associated with CVR. For illustrative purposes, a systematic inspection of abstracts published in the PsycINFO database over the last 5 years (May 2004–May 2009) revealed over 600 abstracts describing nonclinical laboratory studies of CVR. Just over 200 of these abstracts gave clear information about how differences in CVR were interpreted. The majority of these abstracts (73.7%) took a health perspective, interpreting higher CVR as a health risk or indicator of risk. A sizeable minority of the studies (16.0%) took an 'active-coping' perspective, interpreting CVR as proportional to the amount of effort or 'active coping' exerted in response to a stressor and health risks were not mentioned. The third most common perspective (7.2%) took a cognitive appraisal perspective distinguishing between cardiovascular responses associated with appraisals of threat versus challenge. The remaining 3.1% of studies mentioned multiple determinants of CVR (e.g., effort and emotion).

Each of these perspectives leads to different, often contrasting hypotheses about the psychology underlying CVR to stress which we believe complicates interpretations of the implications of CVR to stress. Interpretations of results from a given perspective often seem to depend on previously established associations between the independent variable and health rather than direct evidence. This can be problematic when an alternative perspective can provide a contrasting interpretation of underlying mechanisms and implications of the same CVR.

In the remainder of this section, we consider representative research from each of three perspectives and discuss the limitations and insights of each perspective as they pertain to examining associations between negative emotions and CVR. We argue that a better understanding of the underlying psychology of CVR may be achieved by integrating some key components of each perspective. Furthermore, we believe that a better understanding of the psychology of CVR may improve our understanding of health-related implications.

The health perspective

A thorough review of CVR research motivated by 'the reactivity hypothesis' (Krantz & Manuck, 1984) was presented in a 5-article Reactivity Special Section of *Psychosomatic Medicine* in 2003 (introduced by Linden, Gerin, & Davidson, 2003). The authors of these articles generally agreed that there is notable support for the idea that repeated large-magnitude CVR contributes to the development of cardiovascular disease (Lovallo & Gerin, 2003; Schwartz et al., 2003; Treiber et al., 2003). Psychosocial stressors were associated with CVR and the development of cardiovascular disease in studies involving cynomolgus macaques (Manuck, Kaplan, Adams, & Clarkson, 1988). Macaques that had the greatest cardiac

responses to repeated social hierarchy reorganization (a highly stressful condition for macaques) developed more severe atherosclerosis than those with less pronounced responses to hierarchy disruptions. There have also been prospective human studies linking higher CVR to greater risk of disease (Matthews, Woodall, & Allen, 1993; Ming et al., 2004).

It is not completely clear whether large cardiovascular responses to stress actually contribute to the development of disease or are indicators of risk for development, perhaps because of individual differences in personality, such as tendencies to be hostile (Smith, 1992) or genetic predispositions toward high CVR (De Geus, Kupper, Boomsma, & Snieder, 2007; Selby et al., 1991) or other related forms of stress reactivity (e.g., effort or emotional reactions). However, there is mounting evidence that large-magnitude cardiovascular responses, when repeated over a long period, have a cumulative wear-and-tear effect on the cardiovascular system (Lovallo, 2005; also see McEwen, 1998).

Repeated large-magnitude CVR episodes are believed to contribute to the development of cardiovascular disease by causing damage to the blood vessels, which leads to an inflammatory response. Frequent damage to the blood vessels and a chronic inflammatory response can contribute to the development of atherosclerosis, hypertension, and, increase risk of a heart attack or stroke (von Kanel, Kudielka, Preckel, Hanebuth, & Fischer, 2005; Lovallo & Gerin, 2003; Manuck et al., 1988; Treiber et al., 2003).

Because the effects of CVR on health are accumulative and occur over a long term, few studies of CVR actually involve clinical outcomes. Therefore, interpretations of the health implications of a given study are regularly based on relative levels of CVR, with conditions eliciting higher CVR interpreted as the most harmful to health. This assumption suggests that the association between the magnitude of CVR and its health–harmful effects are a gradient, such that any increase in CVR is harmful and the extent of harm depends on the extent of increase.

Another possibility is that CVR is harmful only after it reaches a certain point. Increases in cardiovascular functioning are clearly necessary to meet the energy requirements of muscles and organs, and such metabolically appropriate adjustments may not be harmful to cardiovascular health. Conversely, CVR in anticipation of dealing with a stressor (Feldman, Cohen, Hamrick, & Lepore, 2004; Obrist, 1981) or that is greater than what is needed to distribute the oxygen respired during a stressor (Rousselle, Blascovich, & Kelsey, 1995) may be excessive and harmful.

Large cardiovascular responses before and during stress may be a byproduct of evolution, preparing and sustaining physically strenuous behavioral responses. The theory of 'allostatic load' explains that the physiological responses of our ancestors were adaptive when stress responses regularly consisted of physically fighting or fleeing predators (McEwen, 1998; Sapolsky, 2004). Today, our behavioral stress responses consist of little fighting, fleeing, or otherwise physically taxing responses, but our physiological responses still tend to prepare us for sustained physical activity. It is believed that our 'somatically-uncoupled,' excessive physiological responses are harmful to health (McEwen, 1998; Obrist, 1981).

That metabolically excessive CVR more harmful than CVR to meet metabolic demands is important for understanding when CVR is most harmful and why an often-necessary physiological adjustment can be harmful to health. This threshold model of how CVR affects health suggests that there are elevations in CVR that are not harmful and others that are harmful. Thus, it may be important to determine whether the level of CVR elicited in a given study meets or exceeds metabolic demand. However, very few studies do this, and in the absence of direct evidence establishing CVR as excessive or linking CVR in a given study to disease, interpretations of just how

harmful CVR elicited by an independent variable is, should be made with some caution.

An area of research in which the previously established link between the independent variable and health has influenced interpretations of CVR involves the effects of social support. Epidemiological evidence has shown that the absence of support is associated with more negative health outcomes than the presence of social support (House, Landis, & Umberson, 1988). Therefore, in a number of studies showing that the presence of a supportive person during a stressful speech task reduces CVR, the effect of the independent variable is interpreted as buffering the negative health effects of higher CVR caused by stress (Christenfeld, Gerin, Linden, Sanders, et al., 1997; Cohen & Wills, 1985; Kamarck, Manuck, & Jennings, 1990; Lepore, Allen, & Evans, 1993). Given previously established associations with health outcomes, this interpretation seems sound. However, if the reduction in CVR is not adequate enough to make it metabolically appropriate, or if the CVR elicited when support is absent is not at a level that is harmful to health, then this interpretation, that social support benefits health by attenuating CVR to stress, is questionable.

Furthermore, some studies including those from our lab have reported a CVR-increasing effect of social support during stress (Allen, Blascovich, Tomaka, & Kelsey, 1991; Anderson & Lawler, 1998; Hilmert, Kulik, & Christenfeld, 2002b; Hilmert et al., 2002a; Raynor, Cerrone, Finney, Pro, & Kamarck, 1996). Based on the reactivity hypothesis then, we would conclude that in these studies, relative to no support, social support increases the risk of developing cardiovascular disease by augmenting CVR to stress. This interpretation, however, is not consistent with epidemiological evidence linking social support to better health outcomes (House et al., 1988) and we have no direct evidence that the extent to which social support increases CVR is in a health–harmful range. Thus, the health-related implications of these effects must be interpreted with caution and alternative pathways linking social support to health in these situations should be considered.

In terms of associations between negative emotions and CVR, the health perspective makes no explicit hypotheses. General models of stress and health include negative emotional responses as the bridge between stress and physiology (Lazarus & Folkman, 1984), and negative emotions have been independently associated with risk of cardiovascular disease. Although physiology tied to negative emotions is hypothesized to be a pathway to disease (Gallo & Matthews, 2003; Grippo & Johnson, 2002; Irwin, 2002; Kubzansky, 2007; Kubzansky & Kawachi, 2000), evidence linking negative emotional responses and CVR to stress is not very supportive. Taken alone, the health perspective offers relatively little insight thus far. However, when considered with other perspectives on CVR, the health perspective highlights a valuable association, between CVR and risk of disease.

The active-coping perspective

Research falling under this perspective focuses on CVR as reflecting the amount of 'active coping' an individual exerts in response to situational demands rather than the implications of such responses. Much of this research can be traced back to the seminal nonhuman and human work of Paul Obrist (1981). Obrist believed there was little adaptive utility afforded by 'affective, motivational, and even attentive states,' but felt that coping responses were adaptive. Obrist (1981) claimed that CVR was not simply an index of effort, but a reflection of how much coping an organism was exerting.

In defining coping, Obrist said that it could be viewed along a dimension of 'activity.' Active coping was contrasted with passive coping, which was defined as the response of

an organism to a stressor when no recourse for action was possible (Obrist, 1981). It usually consisted of freezing and enduring, along with decrements in cardiovascular functioning (decreased heart rate and sometimes blood pressure). Beyond a contrast with passive coping and distinguishing it from affect, motivation, cognition, and effort, active coping is a bit vague. That is, it is possible that active coping is a combination of multiple dimensions, including affect, motivation, attention, and effort. Regardless, active coping has often been operationalized as physical activity in response to a stressor, like escaping an impending electric shock (Elliott, 1969) and, despite Obrist's claim, effort has been highlighted as a key component of active coping (Wright, Contrada, & Patane, 1986; Wright & Kirby, 2001).

Tasks commonly used in stress response research today require relatively little physical activity; having participants give a speech, subtract numbers verbally, attempt to influence others, or play a video game all require minimal physical activity (Gerin, Litt, Deich, & Pickering, 1995, 1996; Hilmert et al., 2002a; Smith, Ruiz, & Uchino, 2000; Wright & Kirby, 2001). Nevertheless, the quality or amount of talking (Hilmert et al., 2002a) and the physical vigor one applies to playing a video game could be interpreted as, at least part of, the active coping an individual exerts.

One focus of research influenced by an active coping perspective has been the relative contribution of active coping to determine the magnitude of CVR (Wright & Kirby, 2001, 2003). In an early example involving negative emotion, participants in an easy task condition had plenty of time to avoid an electric shock by removing their hand from a shock plate. In a moderately difficult condition, participants had less time to remove their hand from the shock plate, thus more effort or active coping was required to avoid the shock. Participants in an impossible task condition had their hands strapped to the shock plate and could not avoid the shock (Elliott, 1969; also, classical aversive condition paradigms; e.g., Obrist, 1981). The key result of this study was that increases in cardiovascular functioning were greater under moderately difficult compared to easy and impossible task conditions (Obrist, 1981). Because negative emotion was assumed to be highest and effort lowest in the impossible task condition (as there is no recourse for action), relatively low CVR lead to the conclusion that CVR must be proportional to the amount of effort (or active-coping) exerted and not the amount of emotion experienced (e.g., Brehm & Self, 1989; Elliott, 1969; Obrist, 1981; Wright & Kirby, 2001). However, without a high effort, low emotion (no threat of shock) condition, it is impossible to know whether emotion and effort jointly influenced CVR in the moderately difficult and stressful (avoiding shock) condition.

Although Obrist (1981) demonstrated that CVR occurs in the absence of physical activity (i.e., 'cardiac-somatic uncoupling') and supposed that this excessive cardiovascular functioning was bad for health, he was unsure whether CVR elicited by active-coping put one at risk for developing cardiovascular disease (see our discussion of the Health Perspective above). Because effort and coping are not notable risk factors for disease, it is rare for a study that interprets CVR as reflecting effort or active coping to consider potential health outcomes. In addition, few of these studies provide direct evidence of an association between CVR and effort.

Therefore, in parallel with strategies used in research motivated by the health perspective, higher CVR elicited by an independent variable that is not associated with risk for disease is frequently interpreted as reflecting effort or active coping (Smith, Allred, Morrison, & Carlson, 1989; Wright & Kirby, 2001). For instance, in a study that manipulated self-efficacy or confidence in the ability to successfully complete a task, participants in the high self-efficacy condition had higher CVR than participants in the low self-efficacy

condition (Gerin et al., 1996). The interpretation of these findings was that participants with high self-efficacy were exhibiting more active coping (cf., Wright & Kirby, 2001). If we instead consider the implications of these results from a health perspective of CVR (Lovallo & Gerin, 2003), then the greater active coping exerted and CVR exhibited by those with high self-efficacy put them at greater risk of developing cardiovascular disease than those with low self-efficacy.

To further illustrate our point, other studies have shown that high self-efficacy is associated with lower CVR during a stressor than low self-efficacy. This pattern has been interpreted as indicating the experience of greater stress and risk for disease in those with low self-efficacy (Hilmert et al., 2002a), or as indicating that those with low self-efficacy need to exert more effort than those with high self-efficacy to achieve the same goal (Wright & Kirby, 2001). Without direct evidence linking feelings of stress (emotions) or effort to CVR in these studies, it is not clear why a variable like high self-efficacy has been associated with higher and lower CVR than low self-efficacy.

It is difficult to make sense of results across experiments showing opposite effects of a variable on CVR. Such results suggest that there may be a moderating variable causing the determinant of CVR (e.g., self-efficacy) to increase effort under some circumstances and to contribute to risk of disease (and perhaps feelings of stress or negative emotions) in other situations, or to simultaneously vary in impacting (increasing or decreasing) effort and conferring (increasing or decreasing) risk.

In a pair of relevant studies, we replicated findings that social support reduces CVR to stress (Hilmert et al., 2002b) and we showed that the presence of the experimenter moderated this effect such that when the experimenter was absent rather than present during a speech, social support increased CVR (Hilmert et al., 2002b). In a separate study, we replicated the CVR-increasing effect of social support and measured effort by counting the number of words produced during each speech. Results indicated that the effect of social support on CVR was mediated by the number of words produced (effort exerted) during the speech and there was no indication that self-reported anxiety mediated these effects (Hilmert et al., 2002a). Thus, social support increased effort thereby increasing CVR. But, it is still not clear whether social support is also an increasing risk of CVR-related disease in these situations or whether social support is a decreasing effort when it has a CVR-attenuating effect (Christenfeld et al., 1997; Gerin, Pieper, Levy, & Pickering, 1992; Kamarck et al., 1990).

The findings that higher CVR is associated with active coping, effort, and poor health outcomes are difficult to reconcile. Two possible alternatives to claiming that all CVR is effort related and bad for health are that CVR is associated with effort and other behavioral or psychological stress responses (e.g., negative emotion) and that higher CVR is sometimes bad for health and sometimes it is not. Research concerning CVR and cognitive appraisals of stressful situations provides some useful insights regarding the latter possibility.

The cognitive appraisal perspective

When faced with a stressful situation, it is believed that an individual makes two automatic appraisals (Lazarus & Folkman, 1984). The primary appraisal is of the demands of the situation. The secondary appraisal is of one's resources to cope with the demands. Challenge appraisals are generally made when individuals feel that their resources meet or outweigh the demands of the situation. Threat appraisals are made when individuals feel that the demands of the situation outweigh their resources. Threat and challenge appraisals

may be associated with different amounts of effort and emotional responses to stress as well as cardiovascular responses with disparate implications for health.

In studies looking at CVR and threat and challenge appraisals, participants are often asked what their primary and secondary appraisals of a task are prior to performing the task (Tomaka et al., 1993). Some studies have reported that appraisals of threat were associated with greater blood pressure reactivity than appraisals of challenge (Blascovich & Mendes, 2000). This suggests that even in the controlled laboratory environment, participants facing the same stressor may have different appraisals of the stressor that are associated with different cardiovascular responses.

Additional physiological differences associated with threat and challenge have involved the components of blood pressure. Blood pressure is determined by a number of physiological parameters including cardiac output (a combination of heart rate and stroke volume) and vascular resistance. Participants who appraise a task as a challenge tend to have greater cardiac output responses than those who appraise a task as threatening (Tomaka et al., 1993). Participants who appraise a task as threatening tend to have an increase in vascular resistance (Tomaka, Blascovich, Kibler, & Ernst, 1997; Tomaka et al., 1993). Thus, although blood pressure changes may be equivalent, changes in the underlying components of blood pressure can differ. Furthermore, it is believed that an increase in vascular resistance (threat response) may be a key component to cause wear-and-tear on the cardiovascular system contributing to the development of disease (Dienstbier, 1989; Lovallo & Gerin, 2003).

Psychological differences between threat and challenge appraisals include more negative emotions associated with appraisals of threat (Tomaka et al., 1993, 1997) and more positive emotions (Tomaka et al., 1997) and increased positive well-being (Blascovich, Mendes, & Seery, 2002) associated with appraisals of challenge. Also, enhanced performance and higher heart rate associated with challenge appraisals may be because of greater effort exerted in this situation than in threat situations (Blascovich, Mendes, Tomaka, Salomon, & Seery, 2003; Blascovich et al., 2002; Wright & Kirby, 2003). Thus, untangling how CVR is associated with health, effort, and emotion may require simultaneous consideration of multiple dimensions of CVR and psychological (including behavioral) responses to stress.

A multidimensional stress response perspective

When experimenters elicit stress in the laboratory, psychological responses are likely complicated and multifaceted (Russell & Barrett, 1999; Shapiro, Jamner, Goldstein, & Delfino, 2001). Psychological responses to laboratory stressors may involve multiple emotions experienced simultaneously (Lane & Schwartz, 1987), various blends of emotional valence (positivity-negativity) and emotional energy (activation), or 'a complex set of interrelated subevents,' including core affect, behavior, attention, awareness, and cognition (Russell & Barrett, 1999). Variance in any dimension of responding could interact with other dimensions to predict CVR.

The two dimensions of psychological and behavioral responding to a stressor we have focused on are how negatively participants feel and how much effort they exert in response to the stressor. Although emotion and effort are often related (James, 1890), the association between them can be positive or negative (Hilmert & Roy, forthcoming). Emotional experiences are often conceived of as having emotional valence (e.g., feeling negative or positive) and activation (e.g., feeling energetic) components (Russell & Barrett, 1999). Therefore, when asked to perform a speech in front of an audience, it is

conceivable that some participants experience negative high-energy feelings and other participants experience negative low-energy feelings. While the former participants may put forth more effort to give a good speech in the hopes that it will alleviate their negative feelings, the latter participants may be more likely to withdraw and wait for the task period to end, only exerting enough effort to keep the experimenter happy.

From this point of view, a negative, high energy response will likely be associated with higher CVR than a negative low energy response, suggesting that the energy and effort dimensions of responding are keys to determine the magnitude of CVR. However, both feelings of negativity and feelings of energy (and resulting effort) may vary independently. It is possible that the intensity of negative emotion experienced by the low-energy or low-effort individual is unrelated to CVR while the intensity of negative emotions experienced by the high-energy or high effort individual is associated with CVR. In this case, the highest, potentially most health deleterious CVR would be that of the most highly negative and effortful response, like putting forth effort in a situation one still believes will be a disaster.

Although not a laboratory CVR experiment, a study that took a multidimensional approach to examine associations between moods and cardiovascular functioning examined average daily blood pressure and five representative moods (Shapiro et al., 2001). The authors characterized these moods as positive (happy), negative (stressed, anxious, angry), and energy related (tired). Analyses revealed that a number of interactions between moods and energy predicted CVR. Consistent with our suggestion, for participants who reported low 'tiredness' (high energy), higher reports of 'stress' were associated with higher blood pressure values. Participants who reported low tiredness or high stress alone had lower blood pressures than those whose energy and stress were high (Shapiro et al., 2001). The results of this study suggest that the amount of energy felt and resulting effort exerted in response to stress could interact with a negative emotion to determine cardiovascular functioning.

Negative emotion may interact with other variables to predict CVR and its consequences in a number of ways. One possibility is that CVR is positively associated with negative emotion only under certain circumstances, for example, when participants feel energy or are putting forth effort, but not when a participant has withdrawn or disengaged. The magnitude of CVR may be greater when negative emotion and energy are high than when only energy is high and there is little negative emotion. This would suggest that there is a synergistic effect of these two variables on CVR (cf., Rousselle et al., 1995) and that a combination of negative emotion and energy may lead to the highest and most deleterious levels of CVR. It may also be that there are differences in how interactions between negative emotions and a moderator (e.g., energy) influence the components of CVR. There may be greater vascular resistance present in the high negative emotion, high energy response than in the high energy alone response.

Another possibility is that when activity related to stress is low, but anticipated to increase in the near future (i.e., the participant has not withdrawn), negative emotion is directly associated with CVR, but when active coping is occurring, the negative emotion–CVR link becomes obscured by other dimensions of stress responding (e.g., extent of effort or activity, sustained task engagement). A study in which cardiovascular and emotional reactions were assessed in anticipation of (during a preparation phase) and during a speech stressor found just this pattern (Feldman et al., 2004). There was a substantial correlation between negative emotion and CVR only during the anticipatory phase. It may be that variance in effort during the task performance which was not present during anticipation moderated the association between CVR and negative emotion.

If, within a given study, individual responses to stress vary substantially (Lazarus & Folkman, 1984; Tomaka et al., 1993) along multiple dimensions that interact to predict CVR, then significant variance in one interacting dimension (e.g., effort) would lead to weak correlations between another interacting dimension (e.g., negative emotion) and CVR. Between study differences in psychological response variability would lead to inconsistent findings (Feldman et al., 1999). Multidimensional examinations of CVR, behavioral, and psychological responses during laboratory stressors may help untangle when CVR is associated with negative emotion, and possibly when CVR is more harmful to health.

Implications for health. From a health perspective, one of the ultimate goals of discovering the individual and situational factors associated with higher CVR is identifying who is at risk of developing cardiovascular disease and the potential targets of preventive treatment. However, because, as we have suggested, much of the laboratory research on CVR from the health perspective relies on previously established associations between a predictor (e.g., hostility, lack of social support) and cardiovascular disease, few novel disease risk factors have been uncovered. On the other hand, these studies have provided a better understanding of a mechanism by which these risk factors may confer risk, by causing higher CVR. Thus, for individuals exhibiting tendencies to be hostile or lacking social support, interventions that target cardiovascular reactions may be implemented to reduce risk of cardiovascular disease.

From a multidimensional stress response perspective, risk factors like hostility and lack of social support may lead to higher CVR and confer risk because they tend to be associated with a certain combination of psychological and behavioral responses to stress, which we believe includes strong negative emotions. If this is the case, then interventions can target one or both of the interacting dimensions of stress responding to help mitigate risk of CVR-related disease. That is, if high negative emotion combined with high effort in a stressful situation elicits the most health–harmful forms of CVR, then intervention can target negative emotions specifically under these circumstances. For example, rather than aiming to alter general tendencies toward hostility or negative emotion, an intervention can aim to change how one feels about putting forth effort in stressful situations.

Effort could also be targeted. That is, if high effort combined with negative emotion is associated with harmful CVR, then we might conclude that it is better to not put forth effort when feeling negative emotions. However, we would suggest that repeated high negative emotion and low effort responses to stress may contribute to poor health through other non-CVR-related pathways. Evidence linking depression, which is associated with blunted, rather than excessive CVR (Carroll, Phillips, Hunt, & Der, 2007), to heart disease and other negative health outcomes through behavioral, immune, and neuroendocrine pathways may be informative in this case (Grippo & Johnson, 2002; Irwin, 2002; Smith & Ruiz, 2002).

Study Design Considerations

An important methodological consideration of research from each perspective is that a traditional laboratory design may not be optimal for detecting associations between CVR and negative emotions (Gerin et al., 1999; Lovallo & Gerin, 2003; Schwartz, 1999). In laboratory studies, researchers make every effort to reduce variability in psychological and physiological responses so that each level of the independent variable elicits the same responses from each participant. This helps isolate the effect of the independent variable

on CVR, the focus of the study. But by reducing variability in emotional and cardiovascular responses, strong associations between different levels of emotion and different amounts of CVR are difficult to detect (Gerin et al., 1999).

To address this issue, Gerin et al. (1999) suggested using more open-ended tasks, such as 'talk with your spouse about whatever you want to.' While this sort of task may elicit a variety of responses, it seems unlikely to reliably elicit strong stress responses. Furthermore, given the multidimensional nature of CVR and psychological stress responses, it is unlikely that this reduction in experimental control will reveal strong correlations. Such a paradigm would require accounting for a variety of dimensions of responding and potential moderators of psychological dimension effects on CVR.

From another perspective, efforts to control stress response variability may not always be as successful as assumed. That is, even within a controlled experiment, variability may exist along important dimensions that have not been accounted for. As we noted, if unaccounted for response dimensions, such as effort exerted during a laboratory speech stressor, interact with emotional responses to predict CVR (Feldman et al., 1999), variability in the unaccounted for dimension would obscure emotion and CVR associations. Assessments of multiple dimensions of responding during more open-ended tasks may provide some clues. Independent, controlled manipulations of effort, emotion, and other facets of stress responding in a single experiment will likely be more difficult, but we believe more fruitful.

Summary and Conclusion

Large cardiovascular reactions to stress have been associated with the development of cardiovascular disease (Matthews et al., 1993; Ming et al., 2004), the exertion of effort (Hilmert et al., 2002a; Wright & Kirby, 2001) or active coping (Obrist, 1981), and cognitive appraisals of stressful situations (Tomaka et al., 1993). However, questions remain concerning the underlying psychology of CVR and the long-term implications of CVR. That is, it seems unlikely that all CVR causes damage and contributes to cardiovascular disease to some extent, but it is possible (Obrist, 1981). We have suggested here that integrating insights from relatively disparate perspectives on CVR may help answer a potentially relevant question regarding whether and when negative emotion and CVR to stress are associated.

A multidimensional perspective of CVR and stress responding is not entirely novel. Laboratories have concurrently examined cognitive and emotional reactions to stress in association with CVR (Feldman et al., 2004) and have considered how multiple physiological systems are impacted by stress (Kunz-Ebrecht, Mohamed-Ali, Feldman, Kirschbaum, & Steptoe, 2003; Larson, Ader, & Moynihan, 2001; Perna, Schneiderman, & LaPerriere, 1997). It is possible that cardiovascular, immune, and neuroendocrine responses are differentially associated with psychological responses and that interactions among these physiological variables differentially impact health. We believe that continued research that considers multiple dimensions of physiological and psychological responses to stress, accounting for emotions, motivation, effort, cognition, and interactions among the dimensions, may help provide answers to some of the remaining questions.

Short Biographies

Clayton J. Hilmert is currently Assistant Professor of Health and Social Psychology at North Dakota State University where he runs a social psychophysiology and health

laboratory, and an attitude and opinion formation social psychology laboratory. His primary research interests are in the effects of stress on health and in the social foundations of attitude and opinion formation. Recently his research has focused on the psychological underpinnings of physiological responses to acute stress, and interactions between environmental stress and physiology predicting pregnancy outcomes. Other recent work involves identifying social reasons for disagreement. Dr. Hilmert has authored or co-authored papers in several peer-reviewed publications including *Annals of Behavioral Medicine, Journal of Behavioral Medicine, Journal of Personality and Social Psychology,* and *Psychosomatic Medicine*. Dr. Hilmert completed his PhD in Experimental Social Psychology from the University of California, San Diego under the guidance of James A. Kulik and Nicholas Christenfeld, and he held a fellowship at the University of California, Los Angeles where he worked with Christine Dunkel-Schetter and Shelley E. Taylor.

Lexi R. Kvasnicka is a third year doctoral student in health and social psychology at North Dakota State University. Her primary interests involve cardiovascular reactivity in different environments, including during exercise and in response to stress, the effects of stress on pregnancy outcomes, and the health implications of accurate and inaccurate body image. Lexi's master's thesis explored how effort and emotions were associated with cardiovascular reactivity during exercise.

Endnote

* Correspondence address: Clayton Hilmert, Department of Psychology, North Dakota State University, Box 5075, Fargo, ND 58105-5075, USA. Email: clayton.hilmert@ndsu.edu

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