



Introductory paper: Cardiovascular reactivity at a crossroads: Where are we now?

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ABSTRACT

A key foundation for research into the link between stress and ill-health has been the reactivity hypothesis; cardiovascular reactivity to psychological stressors, if prolonged or exaggerated, can promote the development of cardiovascular disease. This has been extended, such that by implicating stress in the aetiology of heart disease, it is plausible that psychosocial stress correlates can influence disease risk. Effects of several risk factors and stress buffers on reactivity have been investigated, but such research warrants scrutiny. Considerable literature links heightened reactivity to objective cardiovascular outcomes, but the evidence extending the reactivity hypothesis to other health outcomes and correlates of heart disease is less clear. Here, we explore the external, concurrent, internal, and construct validity of theories arising from the reactivity hypothesis. We aim to highlight theoretical and methodological considerations to guide future research, and clarify the contribution of the reactivity hypothesis to explaining the consequences of stress.

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The notion that psychological stress can damage physical health has been popular for centuries. In recent decades, empirical evidence for this notion has emerged from epidemiological and psychophysiological research. The reactivity hypothesis: that cardiovascular reactivity to psychological stressors, if prolonged or exaggerated, can promote the development of cardiovascular disease (Obrist, 1981), has been considered something of an all-encompassing mechanism. Heightened cardiovascular reactivity, primarily the increase in heart rate and blood pressure observed in response to stress, is posited to result in changes to the structure and functioning of the heart, which in turn promotes sustained hypertension. Indeed, there is much prospective evidence to support the association between heightened cardiovascular reactivity and hypertension (Carroll et al., 1995, 2001; Everson et al., 1996; Markovitz et al., 1998; Matthews et al., 1993; Newman et al., 1999; Treiber et al., 1997). Theoretical considerations of how negative health results from reactivity-related adaptations of the cardiovascular system have also been corroborated by an extensive empirical literature linking heightened reactivity to hard outcomes, including atherosclerosis, myocardial infarction, increased left ventricular mass, and coronary heart disease mortality (Allen et al., 1997; Barnett et al., 1997; Everson et al., 1997; Georgiades et al., 1997; Jennings et al., 2004; Kamarck et al., 1997; Kapuku et al., 1999;

Lynch et al., 1998; Matthews et al., 2006; Murdison et al., 1998; Treiber et al., 2003). Taken together, the literature on cardiovascular reactivity offers an exciting and powerful paradigm for studying the psychosomatic impact of stress.

By implicating stress in the etiology of heart disease, the reactivity hypothesis renders plausible the suggestion that psychosocial stress moderators or correlates can influence disease risk. Accordingly, the impact of several risk factors and stress buffers on reactivity has been investigated. Over the past three decades, a substantial research literature has demonstrated how personality types and traits (Chen et al., 2005; Chida and Hamer, 2008; Kibler and Ma, 2004; Lepore, 1995; Phillips et al., 2005; Suls and Wan, 1993), psychosocial contingencies (Burns and Katkin, 1993; Kamarck et al., 1995, 1998), social support interactions (Allen et al., 2002; Chen et al., 2005; Christenfeld et al., 1997; Gerin et al., 1995; Uchino et al., 1992), stressor tasks (Allen et al., 1991; Kamarck et al., 2000), and task perceptions (Carroll et al., 1986; Kelsey et al., 1999), are each associated with particular patterns of cardiovascular reactivity that elucidate potential mechanisms by which responses to stress might influence disease aetiology and/or progression.

However, when considering the current status of cardiovascular reactivity research, it is important that expectation not displace evidence. Given their reliance on biological, psychological, and social factors, reactivity paradigms are inherently complex and many issues remain unclear. As such, research on cardiovascular reactivity can be said to be facing a crossroads: while investigators might wish to continue to chart the correlates of reactivity

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in order to bolster our theories regarding the correlates of disease, such an approach may lead to diminishing returns unless the very nature of reactivity itself is more completely understood. Accordingly, both the reactivity hypothesis and its related psychosomatic disease models warrant rigorous scrutiny. Many important reactivity-related factors should be considered, including naturalistic reactivity, recovery following reactivity, habituation of the reactivity response, mechanisms underlying reactivity, and different classifications of reactivity. Therefore, in this special issue, the papers presented seek to explore the external, concurrent, internal, and construct validity of theories arising from the reactivity hypothesis, and the various mechanisms by which reactivity might relate to disease.

One of the assumptions of the reactivity hypothesis is that reactivity to acute stress tasks in the laboratory reflects our usual reactivity to stress in real life (Turner et al., 1990), and that such repeated reactivity is detrimental to health (Obrist, 1981). However, it is only through testing this relationship between laboratory and life reactivity that we can be confident that this is the case. In one of the papers in this special issue, Johnston et al. describe the findings on ambulatory reactivity and demonstrate that the associations between perceptions of stress and cardiovascular reactivity measured in the laboratory are also observed in real life stress situations. Although the two types of cardiovascular responses to stress are correlated, it is often the case that the responses obtained in real life are larger than those obtained in the laboratory. However, further research is needed to examine the strength of the associations between real life stress reactivity and cardiovascular disease.

Even though the magnitude of cardiovascular reactivity is one of the key outcomes measured in acute stress studies, the original reactivity hypothesis also considers the role of prolonged cardiovascular responses to stress, in other words, the extent of a stress response over time before cardiovascular indices return to baseline levels. This phenomenon has been termed recovery, and has been shown to predict future cardiovascular outcomes (Stewart and France, 2001). In the paper presented by Larsen and Christenfeld, the cognitions and behaviours that have been found to influence recovery, and the extent of their effects, are discussed. Future research requirements in this field are also highlighted. A further aspect to the impact of cardiovascular reactivity over time relates to the possibility that cardiovascular responses to stressors adapt across repeated exposures. Patterns of cardiovascular adaptation to repeated stress may be important in elucidating the longer-term impacts of stress on heart disease mechanisms, with the capacity to adapt representing an important aspect of individual differences in disease risk. In the paper presented by Hughes et al., adaptation patterns across several cardiovascular parameters were found to be associated with psychometric measures of neuroticism, with persons low in neuroticism benefiting most from cardiovascular adaptation.

It is also important to examine whether or not the reactivity hypothesis can be extended to health outcomes other than those associated with the cardiovascular system, and to examine potential pathways and mechanisms through which such reactivity might relate to a broader range of diseases and disorders. In the paper authored by Phillips, a variety of data is presented showing that risk factors associated with cardiovascular disease (depression, obesity) do not appear to contribute to such risk via heightened cardiovascular reactivity. Data is also presented showing that lower cardiovascular reactivity can prospectively predict poorer self-reported health, increased depressive symptoms, and increased risk of obesity. This would suggest that, with the exception of cardiovascular outcomes, high cardiovascular reactivity may not always be associated with negative health outcomes and behaviours. In contrast, Brydon's paper in this issue examines the links between stress reactivity, obesity, and cardiovascular disease,

and examines the possible mechanisms through measuring plasma leptin, cardiovascular, and cytokine responses to acute laboratory stress in women. The cross-sectional links between abdominal adiposity and stress responsiveness of inflammatory cytokines and the adipokine, leptin, support the notion that certain stress responses in the context of obesity may be mechanisms by which obesity contributes to the development of cardiovascular disease. Further, in the context of higher basal leptin levels, but not necessarily obesity, greater inflammatory cytokine and cardiovascular reactivity to stress may also be a pathway to poor cardiovascular health.

Although the impact of high or low cardiovascular reactivity is typically interpreted in terms of their meaning for long-term risk of cardiovascular disease or of related immune disorders, it may be that reactivity interacts with, or comprises part of, important ancillary mechanisms underlying different psychological functions such as emotionality or addiction. In this context there may be an optimal range of cardiovascular reactivity that reflects normal psychophysiological function, with high and low reactivity indicating maladaptive deviations from the norm. The paper by Lovallo presents an overview of research exploring the links among the cardiovascular system, the limbic system, and the prefrontal cortex, and suggests that cardiovascular reactivity may indeed offer an empirical index of cortico-limbic function that, in turn, yields important insights regarding a range of clinical psychiatric conditions.

As well as issues of construct validity and internal validity, research on cardiovascular reactivity faces important considerations relating to external validity. The reactivity hypothesis has spawned a plethora of experimental laboratory studies examining psychosocial predictors of reactivity in order to implicate such factors in disease etiology. One factor that has received much scrutiny within this tradition is social support. However, although there are several controlled studies comparing reactivity of participants in receipt of social support with that of participants in control conditions, the complexity of the social psychological circumstances in which reactivity experiments take place presents several impediments to their interpretation. By considering some of the key psychosocial elements of cardiovascular reactivity research designs, the paper by Uchino et al. presents an extensive consideration of the pertinent variables that may confound, or even help explain, the heterogeneous findings in this important research area. Such consideration of psychosocial factors helps to ensure that laboratory models of how social support affects cardiovascular reactivity more closely match the complexity of real-world contexts in which relationships and interactions help people to cope with stress.

The special issue concludes with an integrative commentary by Carroll, which draws together the research presented and comments on the validity of the assumptions of the reactivity hypothesis, its validity, and the extent to which it can be used to explain mechanisms between stress, social relations, and disease, and underlie a broader set of health outcomes than originally envisaged. In summary, this special issue aims to highlight: what is currently known regarding the reactivity hypothesis; where this field is heading in terms of developing our understanding of the link between reactivity and psychosomatic disease; and important theoretical and methodological considerations to guide future research. As such, it seeks to evaluate and expand upon current and previous reactivity literature, so that the contribution of the reactivity hypothesis to explaining psychosomatic consequences of stress can reach its full potential.

References

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