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Hypothesis

Cardiovascular Disease and Psychiatric Comorbidity: The Potential Role of Person Cognition

Britta A. Larsen and Nicholas J. S. Christenfeld

Department of Psychology, University of California, San Diego, 950 Jolla, CA 92093-0109, USA

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Abstract

The high comorbidity between psychiatric disorders and cardiovascular disease is little known about the processes linking the two. One plausible mechanism with psychiatric disorders to ruminate on stressful events. This rumination, can extend the psychological and physiological effects of stress to disease etiology. In this paper, we discuss the potential role of person cognition between psychiatric illness and cardiovascular disease. Rumination on stress, which in turn has been found to predict future cardiovascular disease, characterizes mood and anxiety disorders may then contribute to cardiovascular disease instead through extending activation, increasing the risks for cardiovascular disease.

1. Introduction

The high comorbidity between psychiatric disorders and cardiovascular disease in recent scientific literature [1 - 6]. Mood and anxiety disorders in

research showing that those displaying symptoms of anxiety or related morbidity and mortality [2, 7]. Those with the highest level in risk for fatal coronary heart disease [7], and those with clinical risk for cardiac incidents even as much as ten years following the is a link between these disorders, the connection itself is not well u

One explanation for this comorbidity is that chronic disease, such as restriction of activities, fear of impending mortality, and other depressive symptoms have also been found to predict coronary heart cardiac symptoms (see [9]), suggesting a possible bidirectional behavioral link, as behaviors associated with development of CVD isolation, are also associated with mood and anxiety disorders [4] well demonstrated, and in many cases can explain their cooccur connections persist even when controlling for these important health further underlying traits or behaviors common to mood and anxiety mechanisms in the development of CVD.

Another significant risk factor for CVD is psychological stress (see morbidity and all-cause mortality [10] and is recognized as a risk cardiovascular diseases [11]. During acute stress, a number of heart rate and blood pressure. According to the reactivity hypothesis response over time can result in weakened arteries, plaque build those experiencing stress more frequently or to a greater degree myocardial infarction, and other cardiovascular disorders.

While the majority of stress literature has focused on the magnitude duration of reactivity is also important. McEwen [13] has suggested "area under the curve" in which cardiovascular levels are elevated elevation that lingers after stress has passed, is more influential in the acute cardiovascular response. Consequently, it is important cardiovascular parameters are elevated above baseline, but the area [14, 15].

The inability to return quickly to baseline following elevated cardiac risk factor for cardiovascular disease. From this perspective, the generalized anxiety disorder (GAD) [16] could help to explain the found to exhibit lower heart rate variability (HRV), suggesting low parasympathetic nervous system [16]. This inflexibility could prevent to future disease.

Another way of thinking about inflexibility is not in physiological terms being able to return to physiological baseline, it is also important baseline—that is, to no longer experience the cognitive or affective the field, such as life event scales, measure the duration of object of an individual's subjective affective and physiological experience of stress strictly coincides with the occurrence of a stressor. autonomic inflexibility can prevent recovery from arousal, psychological stressors into long subjective ones. Consequently, the duration of the duration of one's cognitive and affective response to it.

Such an approach suggests a mechanism through which the boundaries of a stressful event, namely, prolonged cognitive activity anticipation. Brosschot et al. [17] have labeled this extended co

have suggested that the resulting extended duration of physiological response may be necessary for—the development of serious health consequences following acute psychological stress.

This explanation is particularly attractive when exploring cardiac disorders, since conditions such as GAD, depression, and Obsessive Compulsive Disorder (OCD) are characterized by an inability to shift attention away from troubling thoughts. This is particularly true for OCD, where the obsession persists after the stressor has ended, and anxiety, which can induce stress, is a common feature. The inability to shift focus in a timely manner and adapt to circumstances so not only marks psychiatric disorder, but also contributes to physical health problems. The hallmark of these psychiatric disorders could also be hyper-reactivity surrounding stressors (or, in the absence of concrete stressors, a general state of hyper-reactivity) which is deleterious for cardiovascular health.

In the pages that follow, we discuss studies that examine the connection between mood disorders and as a precursor to cardiovascular disease. (Some studies use “rumination” ; in this paper the two will be used interchangeably). We will explore the connections between rumination and long-term health consequences, the role of perseverative cognition in delayed recovery following acute stress, and rumination as a possible mechanism in the link between psychiatric disorders and cardiovascular disease.

2. Perseverative Cognition and Psychopathology

As described above, those with anxiety disorders have been found to have difficulty shifting focus away from them, as well as many other psychopathologies such as depression, OCD, and inflexibility; specifically, those with anxiety and depression are particularly vulnerable to perseverative cognition.

Research has shown that perseverative cognition is not only a symptom of psychiatric disorders, but also a risk factor for physical health problems. Nolen-Hoeksema, found that trait rumination, measured using a self-report questionnaire, predicted future episodes of depression and anxiety. While women are at greater overall risk for depression, studies show that men are more likely to ruminate [19]. Consistent with this, laboratory studies show that men with high levels of trait rumination are more likely to be depressed than nondepressed controls, depressed participants showed significant increases in rumination when assigned to ruminate on a stressful incident, and men with high levels of trait rumination showed significant increases in negative mood [20]. Furthermore, men with high levels of trait rumination are more likely to experience depressive episodes [21], and learning to not ruminate appears to prevent depression.

Another recent study found that cognitive inflexibility may be associated with psychopathology, particularly depression, and anxiety. In a study where participants were subjected to serotonin depletion in the prefrontal cortex, participants showed continued fixation on the previous paradigm, and showed continued fixation on the previous paradigm, suggesting that this depletion rather than serotonin depletion impeded their ability to learn a new task, suggesting that this depletion rather than serotonin depletion impeded their ability to shift focus and change their paradigm. This is consistent with the psychological disorders characterized by serotonin dysregulation.

3. Rumination and Long-Term Health

Several prospective studies have examined the connection between rumination and long-term health, generally finding evidence of a positive link between them. For example, a study found that rumination was associated with worry across five domains (social conditions, health, finances, self-

heart conditions and followed them for 20 years. They found that t an approximately 50% increased risk for total coronary heart di remained highly significant when accounting for possible confound over financial and health matters leads to elevated risk for total CF

It is possible that worry could be a result of rather than a cause o of worry in the relationship is supported by the fact that this link than worry about health conditions. If worry were only the result c as cardiovascular disease, one would expect the strongest relati worry over health conditions. This, however, was not the case perseverative cognition in cardiovascular health, as social conditi lead to extended worry or rumination. While financial and health endpoints (such as paying off a debt or reducing one' s blood pr indicators that the trouble has passed. It may be unclear, for ex resolved, making those involved particularly prone to rumination a that worry can predict future health outcomes, they do not speak that was the critical aspect in producing these health problems.

4. Rumination and Recovery from Psychological Str

The effects of worry and rumination on recovery from stress h studies. Generally, these have found that worry and rumination findings at least suggest a possible explanation for the relat cardiovascular reactivity has measured responses from both the s Roughly speaking, sympathetic response, typically measured with increased physiological arousal, while parasympathetic response, the body to baseline levels following sympathetic reactivity.

As mentioned previously, several studies have examined stress anxiety disorders [16]. The majority of these have assessed paras rate variability (HRV), which has been found to predict cardiovasc of these studies have found that those with clinical anxiety symptc variability following stressors [16, 27, 28], suggesting a delayed that those with anxiety are prone to slower cardiovascular recover any cognitive processes. It could be, for example, that lowered va which then causes an individual to ruminate.

Most laboratory studies with nonclinical populations have analyze have found that those high in trait rumination and worry, but i psychiatric disorders, exhibit slower heart rate and blood pressu [31]. Results from this line of research are mixed, however [32, methods of measurement. These studies use many different meas of them explicitly investigate whether participants engage in rumin tendency to ruminate are informative, it seems that assessments stress in the lab could be much more useful. Such measures cou recovery and speak to the validity of trait rumination measures include Likert-type scales for statements such as "I keep thinkin [34], which could easily be validated by assessing whether part about themselves do in fact continue to think about laboratory stre

Another problem facing these laboratory studies is determining wh a distinct recovery process, or are due to differences in initial impeded recovery, this could be a result of these stressors eli

recovery would simply be a function of reactivity rather than a d possible that those with anxiety and depression experience longe reactivity during actual stressors.

One key study explored several of the issues stated above by orth stressful tasks. Glynn et al. [35] monitored cardiovascular reactiv Two stressors (a cold pressor task and a shock-threat task) were while the other two (mental arithmetic with harassment and physi reactivity. Additionally, the mental arithmetic and shock threat w cold pressor and exercise tasks were meant to be predominantly p function of reactivity magnitude, one would expect the tasks elicit Alternatively, if recovery is an affective and cognitive process rath the tasks higher in emotionality to result in slower recovery. The arithmetic with harassment tasks elicited the greatest initial arou arithmetic and shock threat—that showed delayed recovery, supp task influenced recovery more than did the initial magnitude of res

In order to investigate further whether this continued activation second study in which all participants completed a mental arithr half were told to relax and sit quietly, while the other half were gi from thinking about the stressor. Again, those who were allowe elevation of SBP, while those who were distracted recovered quick these findings, showing that cognitive distraction can expedite subject to ruminate prevents recovery [29, 31].

These findings underscore several important points. First, they em processes, and given that they can respond independently, the fo for the latter. While stressors like the shock threat could be cor small initial change in CVR, they could be disproportionately delea are long lasting. Secondly, these findings also highlight the role of measured in terms of the duration of the actual stressor, but if actual experience of stress—and resulting physiological reactivity could be that worrying over social conditions predicted greater risk these problems were more emotionally upsetting and generated r inadequate finances. Finally, these findings emphasize that the del is a cognitive rather than physiological effect. While emotional eve arousal to nonemotional tasks, recovery from such tasks, such as cognitive focus.

Other studies have shown that the physiological effects of stress c event. While there is a good deal of research on reactivity related examining the effects of anticipating concrete events. Though th increased cardiac activation in those anticipating stressors compa that physiological effects of stress can even be manifest during slk both that perseverative cognition needs not to be conscious, a impaired sleep quality [36]. The data connecting anticipation of str extremely limited.

5. Cardiovascular Recovery and Long-Term Health C

While the allostatic load theory predicts that extended stress activ leads to long-term negative health, longitudinal studies with hea Several prospective studies have examined whether recovery from

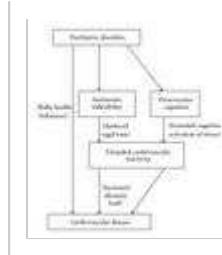
mortality. The results from several of these studies show quite a few minutes, can predict health years later. Two of these studies that showed that slow recovery from acute cardiovascular reactivity were more likely to die. Stewart and France found that this effect held even when controlling for other factors. recovery and reactivity are distinct risk factors. Treiber et al. [39] found that following a stressor independently predicted heart rate and blood pressure values. Another laboratory study investigated the role of reactivity in children whose parents either did or did not have heart disease [40]. The authors found that those who had two parents with heart disease were more likely to inherit “heart disease” from their parents, but in some cases slow recovery that eventually lead to cardiovascular disease.

Two other studies also found that slow recovery from acute cardiovascular reactivity was due to perseverative cognition; cardiovascular reactivity was due to physical fitness, independently. Importantly, however, the Cole et al. study found that controlling for initial health status, including weight, blood pressure, and health. This suggests that delayed recovery, whether due to physical fitness or perseverative cognition, has consequences independent of other cardiovascular risk factors.

While these studies suggest a connection between recovery and health, additional research. Specifically, prospective studies are needed to determine whether rumination can lead to heart disease in otherwise physically fit individuals. Such studies are needed for disease to develop. Such studies should investigate whether perseverative cognition contributes to physical disease, which would likely be in the form of physiological flexibility—that is, normal sympathetic and parasympathetic responses, and by doing so extend sympathetic responses, decrease parasympathetic responses, and thus activate the cardiovascular system. In this case, perseverative cognition would lead to that certain people experience autonomic inflexibility due purely to perseverative cognition. The resulting extended activation could both trigger cognitive fixation and cardiovascular system. Perseverative cognition in this model could serve more as a marker than a cause of future disease. Further research is needed to elucidate which individual differences act as risks for heart disease.

Figure 1 shows a simplified view of the comorbidity relationship. Cardiovascular disease and psychiatric disorders are interconnected than shown, but the figure is designed for illustration purposes. The central connection, in which psychopathology is associated with deficiencies in physiological flexibility, elevated cardiovascular reactivity following stress. The focus of the figure is on the left, which illustrates a cognitive pathway linking these phenomena to physiological deficit, but rather to cognitive fixation on negative experiences. This means mutually exclusive; rather, it is likely that all three (plus additional factors) are interconnected between psychopathology and cardiovascular disease.

Figure 1: This informal theoretical model shows the relationship between psychiatric disorders and cardiovascular disease. The mechanisms highlighted (the left and right) are additional cognitive pathways on the right, in which perseverative cognition leads to cognitive fixation on stressful experiences.



6. The Role of Flexibility in Health

The studies discussed above emphasize an often overlooked individual: flexibility. As the ability to adapt is an essential aspect of survival, the importance of flexibility in health is often overlooked. With much attention devoted to the duration of actual stressors, it is often the most deleterious for health. These stressors presumably result in the long-term negative consequences. However, the effects of a chronic stressor can be regulated by negative emotions, adjusted appraisals of threat, and altered coping responses. Those with inflexible coping responses could experience negative health outcomes from even brief stressors. In fact, it has been suggested that coping flexibility, which predicts effective coping responses to stress better than rigid coping strategies, predicts effective coping responses to stress better than rigid coping strategies. Lazarus [44] emphasized that coping is a shifting process and found that coping strategies fluctuated markedly not only between but also within individuals.

While the research on flexibility's impact on health is limited, there is evidence that flexibility impacts mental and physical health (see [45]). One laboratory study found that high flexibility predicted lower levels of distress 1.5 years later [46]. Flexibility and social support confers health benefits [47]. It is possible that support and flexibility act as different appraisals, or act as distractions to prevent rumination.

Flexibility may also be a common theme in various coping strategies. The Constructive Anger Behavior-Verbal scale (CAB-V), for example, involves expressing anger verbally in order to understand "the other person's point of view" [48]. As opposed to someone that they were angry, those high in constructive anger were more likely to understand when controlling for hypertension risk factors and psychosocial factors. Expressing anger in order to organize and understand them has also been found to improve health. Expressing anger could be partially due to the writing process offering new interpretations. Expressing anger in cognitive behavioral therapy (CBT), which focuses on cognitive restructuring, has been shown to be an effective treatment for depression and anxiety. Further, the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) study, which focused on restructuring specifically to address depression and anxiety in patients with coronary heart disease, have found that CBT can effectively address psychopathology in patients and is associated with improved cardiac health [52].

While these studies do not examine flexibility specifically, they do suggest that flexibility, understanding and embracing new perspectives, which could both be associated with better health.

7. Conclusions

As shown in the studies above, inflexibility, whether cognitive, emotional, or behavioral, is associated with mental and physical health. It is possible that the high comorbidity

disorders is attributable to a general state of inflexibility, leading to variability and vagal tone, and extended sympathetic arousal. And these areas perpetuates it in another. Perseverative cognition, for physiological arousal [35], and extended and/or more severe depressive

While the studies discussed here suggest a role of perseverative cognition in cardiovascular disease, clearly there are questions remaining to be answered. For recovery from stress, a condition which predicts future cardiovascular long-term health outcomes can in fact be predicted by differences in cognitive evidence of an autonomic inflexibility in those with GAD, it is unclear if a shift in cognitions to more constructive paths, might also play a role.

As perseverative cognition has been found to extend physiological health, a better understanding of the situational and individual factors is important. Generally, cognitive fixation suggests a lack of adaptability in the physical and social world.

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