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Comment: The Emotional Basis of Toxic Affect

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Abstract

I focus on some differences between negative emotional states and how they are coped with in explaining different cardiac risks. The different cognitive, motivational, and physiological characteristics of emotions imply different appraisals of the negative event, and different resources to cope with the event. Cardiovascular activity depends on these different appraisals and coping strategies. For example, cortisol levels have shown to be differently associated with anger and fear responses to social stress. In addition, different ways to regulate one's emotions are also associated with different bodily responses that may increase or decrease cardiac risks. Future research should not only examine different emotions to stressors, but also more long-term regulation strategies and coping resources, such as self-esteem.

Keywords

cardiac disease, emotion regulation, fight flight

Toxic affect refers to the idea that negative emotional states can have consequences for one's physical health. The narrative review of available studies here (Suls, 2018), shows that both anxiety and depression have been demonstrated as independent risk factors for people with both premature and existing cardiac diseases. The review also shows that the effects differ depending on the nature of the negative emotional states, but it is unclear why each of these different emotional states can lead to potential heart problems. In this comment, I would like to focus on some differences between these negative emotional states that may be relevant for heart-related health outcomes.

It may be useful first of all, to distinguish between emotions, affective dispositions, and psychopathological disorders. Whereas emotions are immediate reactions to a specific threat, affective dispositions are generalized tendencies to react in a certain way and can become part of one's personality. Psychopathological affective disorders are extreme and dominant affective tendencies that also occur in neutral situations. Such long-term states, however, do not come out of the blue, and have often evolved from the frequent experience and dysfunctional regulation of similar underlying emotions. Hence, depression is likely built on sadness, hostility on anger, and anxiety on fear, and it may be useful to reflect on whether to measure dispositional states or immediate emotions.

The different cognitive, motivational, and physiological characteristics of emotions may tell us something about their relation with hypertension as one of the factors leading to cardiac problems. Previous research has shown that stress is associated with higher cortisol levels, but this relation widely varies across tasks and contexts (Dickerson & Kemeny, 2004). This is supported by other lines of research showing that bodily responses during stress depend on how one appraises the stressor (e.g., negative, unexpected, familiar) and one's own coping potential to deal with stress (low vs. high; Blascovich & Tomaka, 1996; Jamieson, Hangen, Lee, & Yeager, 2018; Lazarus, 1991). Two emotional reactions that have been distinguished are an angry, or fight; and a fear, or flight response (see also Carver & Harmon-Jones, 2009). Anger is based on appraisals of certainty, unfairness, and other-blame, resulting in the tendency to attack the threat. Individuals who are angry are motivated to confront and are certain that they have the resources



to do so. In other words, angry people do not doubt their coping potential and anger prepares the bodily system to fight.

Fear, on the other hand, implies that the stressful situation is seen as a threat, and this is based on appraisals of uncertainty, and low coping potential resources to deal with the negative event. In line with this, the body prepares for withdrawal, or freeze. Interestingly, however, anger and fear regulate stress in different ways, tailored on the specific stressor and the resources of the individual. Moons, Eisenberger, and Taylor (2010) for example, studied cortisol and other hormones in association with self-reports of anger and fear at different stages of a social stress task. One of their findings is that anger positively predicts poststressor cortisol levels, whereas the relation with fear is negative. Importantly, if anger was controlled for, no negative relation for fear was found, suggesting that emotion-specific characteristics, and not mere shared negative valence, are important. Further, fear, and not anger, was associated with higher levels of proinflammatory cytokines, a hormone that induces (social) withdrawal, such as decreased exploration, and promotes recovery through shutting the system off. The different functionality of emotions in stress is also emphasized in the biopsychosocial (BPS) model, where appraisals of the stressor as either challenge or threat have been associated with different cardiovascular reactivity (e.g., Mendes, Blascovich, Major, & Seery, 2001). Thus, one factor that may predict different cardiac outcomes is the prevalence of specific emotional responses that are associated with different cardiovascular responses, which may in turn differently affect blood pressure levels.

A second factor that links emotions and health outcomes relates to the (dys)regulation of emotions (e.g., Appleton & Kubzansky, 2014). Previous research has shown for example that the suppression of negative emotion expressions indeed decreases emotional expressions, but does not decrease emotional intensity, and in fact increases physiological responding compared to other, more cognitively oriented emotion regulation strategies, such as reappraisal (Gross, 2002). In addition, the failure to regulate one's negative emotions effectively costs energy and may further increase emotional experiences during subsequent events (Consedine, Magai, & Bonanno, 2002). In the case of anger, for example, people with an anger disposition, often referred to as hostility, do not necessarily express anger more often than nonhostile individuals. Hostile people have cynical attitudes and mistrust others, which may either lead to more frequent expressions or to more frequent suppression in order not to negatively affect daily interactions. Interestingly, individuals suppressing their anger are more prone to the development of coronary atherosclerosis than individuals expressing their anger (e.g., Anderson, Metter, Hougaku, & Najjar, 2006). More generally, the regulation strategy of emotional disclosure has also shown to be positively related to cardiovascular health (Frattaroli, 2006).

One conclusion drawn from the narrative review is that anxiety and depression appeared most frequently as predictors, whereas anger/hostility did not. The results are too inconsistent, however, to draw any firm conclusions with regard to the effect

of specific emotions. This lack of consistency may not only be due to a lack of studies or differences in measures and populations, however. First of all, the emotions underlying the different psychopathologies are associated with different physiological symptoms that may imply different risks for heart diseases. Second, negative affect (NA) also implies negative self-esteem, in addition to the prevalence of negative emotions, and appears to be an independent predictor in some studies. This may also suggest that negative self-esteem, rather than the prevalence of specific negative emotions, is the underlying factor that involves cardiac health risks. Negative self-esteem implies a general lack of coping potential which maybe a common factor in people reporting NA, as well as people reporting depression or fear. The literature on emotion regulation and the BPS model also suggest that resources and coping potential are relevant. High scores on depression, anxiety, and hostility measures may have ineffective emotion regulation in common, increasing emotion intensity and the use of bodily resources. Thus, future research should not only examine different emotions to stressors, but also more long-term regulation strategies and coping resources, such as self-esteem.

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