



Expanding stress theory: Prolonged activation and perseverative cognition

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Summary Several theories of the stress-disease link have now incorporated prolonged activation. This article argues that these theories still lack an important element, that is, the cognitive nature of the mechanism that causes stress responses to be sustained. The perception of stress and the initial response to it do not automatically lead to prolonged activation. The active cognitive representations of stressors need to be prolonged in order to extend their physiological concomitants. We call this mediating process perseverative cognition, and it is manifested in phenomena such as worry, rumination, and anticipatory stress. We summarize evidence suggesting that these phenomena are indeed associated with physiological activation, including cardiovascular, endocrinological and immunological parameters. This evidence is still far from sufficient, due to the many methodological insufficiencies in the studies involved. Nevertheless, it makes clear that cognitive phenomena characterized by perseverative cognition may be likely candidates to mediate the effects of stress sources on somatic disease.

We also argue that there is a dearth of evidence supporting the role of prolonged activation. There are a limited number of studies demonstrating prolonged activity related to stressors and emotional episodes, and their methodologies often do not allow unambiguous conclusions. Even more important, the crucial assumption that prolonged activation actually leads to pathogenic states and disease has received hardly any attention yet and therefore is still largely unsupported. There are only a few studies that showed that anticipatory responses and slow recovery from stress predicted disease states.

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1. Prolonged activation and stress theory

Psychological stressors can codetermine the development and course of somatic disease (e.g. Krantz and McCeney, 2002). Most stress scientists would agree that a major part of this influence is

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caused by prolonged physiological activity due to stressors, and not or not alone the activity during stressors. Only prolonged activation can lead to the pathogenic state that eventually can lead to organic disease (Linden et al., 1997; Ursin and Eriksen, 2004). Prolonged physiological stress activity comes in three forms: anticipatory responses to (potential) stressors, slow recovery from stressors, and recurrent activity related to past stressors. Prolonged activity, or duration of the stress response, is prominently present in the early stress theory of Selye (1950). However, during the last 50 years stress scientists did not consequently adopt prolonged activation as a major element in their theories and research. Only a few theoretical models have done so. Ursin and co-workers introduced the concept of 'sustained activity' in the early 1980s (Ursin and Murison, 1983). Unfortunately, the implications of this concept did not appear to be sufficiently recognized by others. Much later, in the 1990s, McEwen (1998) launched his allostatic load theory, and Linden et al. (1997); Brosschot and Thayer (1998); Sluiter et al. (2000) attempted to put stress recovery back on the research agenda.

In this theoretical article, we will discuss how prolonged physiological activation can expand stress theory. We will explore the possible reasons for the failure of major stress theories to incorporate prolonged activity. Thereafter, we will focus on an important lacking element: the cognitive nature of the psychological mediator between stressors and prolonged activation. Finally, we will summarize available evidence with respect to some of the major assumptions of the prolonged activation model.

2. Missing elements in stress theory

There are some likely explanations for the failure to include measures of prolonged activation. One is that studying prolonged activity is more costly, both economically and time wise, than studying activity during stressors or immediately before or after stressors. There are also some methodological and statistical issues that need to be resolved, especially with recovery (see Linden et al., 1997). Still, we believe that a more important reason to neglect prolonged activation is related to the natural inclination of researchers to follow the existing theoretical and experimental paradigms, instead of critically examining their premises. For example, a major assumption underlying most of these paradigms involves the 'reactivity hypothesis' that holds that frequent and strong responses

to stressors lead to pathogenic wear and tear in organisms and ultimately to disease. The reactivity hypothesis obviously ignores this crucial element of prolonged activation and, not surprisingly, failed to hold up against empirical evidence (see Schwartz et al., 2003). In a reactivity model, the short-duration physiological spikes are thought to play the primary pathogenic role. In contrast, a prolonged activation model represents an 'allostatic load' model (McEwen, 1998), which may be conceptualized better in terms of the 'area under the curve'. In such a model, the total amount of stress-induced physiological activation, over time, is regarded as the primary pathogenic pathway.

An unfortunate consequence of the dominance of reactivity-based theories is that most researchers still use instruments that fail to capture the most central factor, the duration factor; that is, stress responses ahead of the stressor and after it, sometimes far ahead of it and long after it. Even though as far back as in the 1980s Ursin and co-workers incorporated the notion of negative outcome expectancy in their theory, as an important determinant of prolonged activity (Ursin and Murison, 1983), the dominant instruments used in stress research were not focussed on the future. Instead, instruments measuring life events, daily hassles, and various specific stressors, such as work-related stress factors and marital stress factors, all focus on the past. That is, they ask individuals about their experiences in the last week, month or year. At best, they ask about individuals' appraisal of these past experiences, and their interaction with their personality or other dispositional characteristics, such as coping style. None of them, at least not the best known and most widely used, ask about future stressors and anticipation of them. This is astonishing. Any layman would agree that in normal daily life our tense fears and hopes about the future consume at least as much time as those about the past. As the saying goes, 'looking ahead of things is already half the pleasure of it'. However, looking ahead of stressful events is—at least—half the misery of it! Consequently, it is likely that anticipatory stress responses account for a large part of stress-related prolonged activation.

Another group of important stress instruments that seem to have neglected prolonged activation involve coping behaviour. Apart from the huge conceptual problems with the concept of coping (see e.g. Ursin and Eriksen, 2004), it is doubtful if common tests of coping realistically reproduce actual coping behaviour. Coping tests usually measure the extent to which individuals exhibit certain coping behaviours, but not the time spent on stressful doubting about which coping strategy

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