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Chapter 3: *Expanding Stress Theory:
Prolonged Activation and Perseverative
Cognition*

ABSTRACT

Several theories of the stress-disease link have 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 shows that cognitive phenomena characterized by perseverative cognition may be 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. A limited number of studies demonstrate prolonged activity related to stressors and emotional episodes, and their methodologies often do not allow unambiguous conclusions. It is even more significant that 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. Only a few studies show that anticipatory responses and slow recovery from stress predict disease states.

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PROLONGED ACTIVATION AND STRESS THEORY

Psychological stressors can codetermine the development and course of somatic disease (Krantz and McCeney, 2002). Most stress scientists would agree that a major part of this influence is 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 leads 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) and Sluiter et al. (2000) attempted to put stress recovery back on the research agenda.

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

MISSING ELEMENTS IN STRESS THEORY

There are some possible explanations for the failure to include measurements 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. Also some methodological and statistical issues need to be resolved, especially concerning recovery (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, fails to hold up against empirical evidence (Schwartz et al., 2003). In a reactivity model, the shortduration 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; i.e. stress responses ahead of the stressor and after,

sometimes far ahead and long after. Even though as far back as 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 workrelated 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'. However, looking ahead of stressful events is—at least—half the misery! 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 behavior. Apart from the huge conceptual problems with the concept of coping (e.g. Ursin and Eriksen, 2004), it is doubtful if common tests of coping realistically reproduce actual coping behavior. Coping tests usually measure the extent to which individuals exhibit certain coping behaviors, but not the time spent on stressful doubting about which coping strategy to choose. They also do not measure the extent to which individuals are unsure about their choice and will soon terminate one coping endeavour in favour of another, and so on. This pondering, brooding, and feverish vacillation between several coping options are an essential facet of ineffective coping behavior, but it is overlooked by common coping tests. This is especially regrettable because it is this dynamic facet of coping that might be responsible for a large part of prolonged stress activity, as we shall argue below. Ursin and co-workers came close to these dynamics of coping in their Cognitive Activation Theory of Stress (CATS: Ursin and Eriksen, 2004). In this prolonged activation theory, they conceptualise coping as 'positive outcome expectancy', which would only lead to a short stress response, 'training' the biopsychological organism to be more efficient in dealing with future challenges. Non-coping, in their view, is equal to 'negative outcome expectancy' (Fig. 1), 'straining' the organism instead. Still, they did not describe the actual behavioral manifestation of 'negative outcome expectancy', or for that matter, propose a mechanism that prolongs activation.

MEDIATOR OF PROLONGED ACTIVATION: PERSEVERATIVE COGNITION

What prolongs physiological activation, either in advance of a stressor, or afterwards? By lacking the prolonged activation element, the leading stress theories have obstructed the development of a hypothesis of this mediator. Even those theories that incorporated the prolonged activation element (Selye, 1950; Linden et al., 1997; Brosschot and Thayer, 1998; McEwen, 1998; Sluiter et al., 2000; Ursin and Eriksen, 2004) have not explicitly hypothesized a cognitive mechanism that really prolongs activation due to stressors or their perception. For example, although CATS theory (Ursin and Eriksen, 2004) proposes a psychological mediator, that is,

negative expectancy, the theory does not account for the mechanism that causes negative expectancy to be prolonged. It is important to realize that a stressor, or its perception (i.e. negative outcome expectancy) does not lead to prolonged activation in and of itself, but only when the stressor itself or its perception is prolonged. The average physiological response during a stressor is a 'medium sized' biological response, comparable to those occurring during moderate exercise. This kind of response recovers quickly when not instigated otherwise. Thus, something other than metabolic needs keeps on instigating the organism to respond after termination of a psychological stressor. Similarly, huge anticipatory responses far ahead of a stressor can also not be explained by metabolic needs at the moment of anticipation. And finally, modulating stress factors such as perceived uncontrollability, deficient coping styles, low social support and personality dispositions such as hostility, also do not produce prolonged activation of themselves. In short, there must be a mechanism mediating between stressors and stress factors on the one hand and prolonged activation on the other.

Our hypothesis is that perseverative cognition is such a mediator (Brosschot and Thayer, 2003; 2004; see Fig. 1). We have defined perseverative cognition as: 'The repeated or chronic activation of the cognitive representation of stress-related content'. Sources of stress will only lead to prolonged activation when individuals cognitively perseverate about these stress sources, to some extent and for some period. Thus, perseverative cognition might help to convert the immediate psychological and physiological concomitants of life events and daily stressors into prolonged physiological activation of several of the body's systems, which in turn is necessary for the development of a chronic pathogenic state that can lead to disease. As such, perseverative cognition can be thought of as a final psychological pathway by which stress exercises its deleterious effects on the body's systems. It does this by virtue of its propensity to prolong the stressor itself, in a representational form that continues to activate the organism. In terms of CATS theory, negative expectancy is the proposed key factor that produces a physiological activation in the first place, but only when an individual continues to endorse this negative outcome expectation by perseverative cognition, that it will finally lead to prolonged activation. In other words, if one does not worry about the negative outcomes, or maintains an active cognitive representation of it in another way, it will not lead to prolonged activation. What our theory adds to CATS, is the dynamics of this central subjective stress response.

Is there any evidence that perseverative cognition is related to physiological activation and disease outcomes? Perseverative cognition is a central element in cognitive phenomena such as worry and rumination. These phenomena have been major issues in psychopathology and have recently drawn attention in somatic fields too. We have recently reviewed available evidence of worry and rumination-related somatic outcomes (Brosschot and Thayer, 2004). Due to space limits, we will only give a short summary here with the number of studies that reported one or more relevant findings. Trait worry as well as episodes of worry were related to general somatic complaints (two studies), and trait worry predicted a second myocardial infarct (one study). Indirectly, worry is related to poor sleep (e.g. Nicassio et al., 1985), and poor sleep quality in its turn to increased mortality (Dew et al., 2003). Natural episodes of worrying were found to be related to increased cortisol (one study), to high heart rate (two studies) and low heart rate variability (HRV) (one

study). Several laboratory studies showed that experimental worry was related to low HRV (two studies) and that angry rumination was associated with slow blood pressure recovery (four studies). Finally, trait worry and rumination were associated with increased resting blood pressure levels (two studies), less natural killer cells (two studies), higher cardiovascular activity (three studies), and high cortisol values during natural or experimental stressors or during recovery from stressors (one study). In the latter study (Roger and Najarian, 1998) trait rumination was a stronger predictor of cortisol levels than neuroticism, suggesting that a tendency to engage in perseverative cognition represents an independent dimension. Not all of these studies controlled for the effects of health behavior. Worrying or ruminating may have led to for example increased smoking or coffee consumption that may have caused increased physiological activity. Thus, it was not always clear whether and to what extent the prolonged activity related to perseverative cognition was a direct consequence of the cognitive representation of the stressor, as our theory would predict.

PROLONGED ACTIVATION: EVIDENCE FOR ITS EXISTENCE AND ITS DISEASE RISK

The foregoing suggests that perseverative cognition may be a mediator of stress related prolonged activation. However, is there any proof for prolonged activation itself, and even more so, proof that it predicts ill health? Because of its absence in stress theories, prolonged activity has not often been an explicit research goal of scientific inquiry. Not surprisingly therefore, only a relatively small number of laboratory and real life or ambulatory studies have provided evidence for prolonged activity. Recent reviews of laboratory studies revealed that in particular emotional stressors lead to slow recovery after stress (Linden et al., 1997), especially when stressors contain both uncontrollable and social-evaluative elements (Dickerson and Kemeny, 2004). We have recently reviewed evidence of prolonged cardiovascular activation in ambulatory stress studies (Pieper and Brosschot, 2005). We distinguished three types of prolonged activation (anticipatory, recovery and recurrent) related to discrete and chronic stress sources, as well as negative emotional episodes. Natural disasters were excluded as being too uncommon in the life of most people. The combined data from the reviewed studies tentatively suggested that these three sources of stress were related to prolonged cardiovascular activity of various durations, including sleep periods. Endocrinological and immunological real life stress studies are not yet systematically reviewed from the prolonged activation perspective, but a few examples should be mentioned here. For example anticipating a stressor was associated with increased salivary cortisol (Smyth et al., 1998), plasma cortisol (Lacey et al., 2000) and salivary immunoglobulin A (Spangler, 1997), and anticipating a stressful working day was related to higher salivary cortisol awakening response (Kunz-Ebrecht et al., 2004). For more examples the reader is referred to Kristenson et al. (2004). For many of the reviewed studies strong conclusions could not be drawn, since they were hampered by methodological shortcomings with respect to prolonged activation. For example, the exact beginnings and endings of stressors were seldom clear and neutral or rest episodes were not checked for the presence of stressors other than the stressor of interest (e.g. work stress or caregiver stress). Further, not all studies controlled for health behavior such as smoking or drinking, that could act as

alternative causes of prolonged activation. These drawbacks are mostly due to prolonged activity not being the primary focus of study.

Several studies reported prolonged physiological effects of stress sources on sleep. We consider these findings as particularly important because sleep is a major and natural recovery period. A particularly noteworthy example is a study by Hall et al. (2004) who demonstrated that anticipating an oral speech that had to be delivered upon awakening in the morning appeared to increase sympathovagal balance throughout the whole preceding sleep period. If normal daily stressors will be proven to have substantial prolonged physiological effects even during sleep, these effects may account for a considerable part of the effects of stressors on health.

Last but not least, given the existence of prolonged stress-related activity, is there any proof that it actually predicts disease? The answer is yes, but the available evidence is still very meagre. We found five studies, all of which used laboratory stressors that were embedded in larger cardiovascular epidemiological studies. This meagre result is perhaps not surprising given the relatively recent revival of interest in prolonged activity and the costly and time-consuming nature of epidemiological studies. High anticipatory blood pressure responses to a stressor predicted hypertension 4 years later (Everson et al., 1996), and delayed blood pressure recovery from a stressor predicted hypertension 3 and 5 years later (Stewart and France, 2001; Borghi et al., 1986, respectively). Delayed heart rate recovery predicted overall mortality five (Nishime et al., 2000) and 6 years later (Cole et al., 1999) in cardiac patients, and high resting heart rate 5 years later (Treiber et al., 2001), while in the latter study blood pressure recovery did not predict hypertension. Delayed heart rate recovery, but not blood pressure recovery, predicted mortality in atherosclerotic patients 6 years later (Ellis et al., 2004). Importantly, most of these studies corrected for reactivity during the stress task, and for biobehavioral variables such as gender, age, smoking, body mass index, parental history of hypertension and disease severity. On the other hand, only one study (Borghi et al., 1986) used a psychological stressor, while the others used physical stressors, such as a bicycle ergometer, or did not differentiate between the two kinds of stress (e.g. Treiber et al., 2001). Summarizing, evidence for a crucial assumption of the prolonged activation model is still limited. Importantly, prolonged activation values in real life however, and due to a purely psychological stressor (preferably during sleep), have not yet been linked to later development of disease.

CONCLUSION

Prolonged physiological activation is an essential element in a theory of the effects of psychological stress on somatic disease. After having been neglected for nearly half a century, several major stress theories have now incorporated prolonged activation. Nevertheless, an important element seems to be lacking in prolonged activation theory. This element is the cognitive nature of the psychological mediator between stressors or stress-factors and prolonged activation. We argued that stressors or their perception do not automatically lead to prolonged activation. The cognitive representation of stressors needs to be activated in order to extend their physiological concomitants. We call this mediating process perseverative cognition, and it is manifested in phenomena such as worry and rumination, or anticipatory stress. We summarized 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 shows that perseverative cognition may be a likely candidate for the mediator of the effects of stress on somatic disease. Therefore, theoretical models of stress and disease should not only account for prolonged activation, but also for its production by cognitive perseverative processes.

We also argued there is a dearth of evidence supporting the role of prolonged activation. Some studies demonstrate prolonged activity related to stressors and emotional episodes. These studies were not primarily focussed on the issues above, and therefore lacked methodologies that were adequate for their investigation. Even more important, one crucial assumption underlying prolonged activation theories has received hardly any attention in research yet, and therefore is still largely unsupported. This assumption states that prolonged activation (anticipatory responses and slow recovery) from stress predicts pathogenic states and disease. Only a few studies showed this, but these were human laboratory studies, and nearly all of them pertained to physical stress. Thus, even this little evidence might not generalize to the real world, and not to psychological stress. On the other hand, several studies have demonstrated that need for recovery, which is seen as a subjective index of prolonged activation, mediates the effect of work stress on health complaints and sickness leave (Sluiter et al., 2003), and cardiovascular disease (Van Amelsvoort et al., 2003), effects that seemed reversible by extending rest periods (Schuring et al., 2004). Still, as yet no real life findings are reported showing that slow physiological recovery after psychological stress predicts somatic disease. Finding such evidence in epidemiological studies that preferably use real life psychological stressors would provide an important impetus for testing the prolonged activation theories discussed in this article (Selye, 1950; Linden et al., 1997; Brosschot and Thayer, 1998; McEwen, 1998; Sluiter et al., 2000; Ursin and Eriksen, 2004).

A final important issue about the nature of these processes pertains to sleep. Given the fact that sleep is perhaps the major restorative period in human life, it is at the same time a major 'opportunity' for prolonged activation. The studies mentioned above that show prolonged activation during sleep (e.g. Hall et al., 2004) make clear that at least part of perseverative cognition is not consciously carried out. It is likely that conscious perseveration is not a prerequisite for prolonged physiological activation. At present, little is known about the physiological effects of unconscious processing of distressing information, apart from some studies that show changes in cerebral activity, startle reflexes, skin conductance and the sexual system (Öhman and Mineka, 2001). Studies showing more substantial effects on parameters that are more relevant for somatic disease, such as endocrinological effects or cardiac effects are still rare (Ruiz-Padial et al., 2003). We believe that this is yet another important target for future studies. It is a challenge to show clinically relevant effects of a cognitive process that might be the mediator of prolonged stress responses during a crucial restorative phase: sleep.

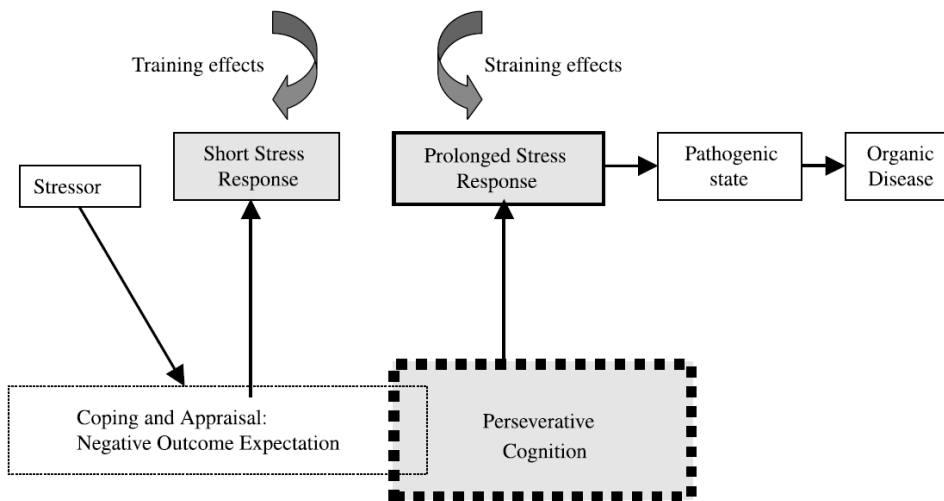


Figure 1: Model of prolonged stress-related activation, including perseverative cognition as a mediator between stress factors and prolonged stress responses.

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