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Stress and Disease: A Structural and Functional Analysis

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Abstract

Chronic stress, a validated health risk factor, remains an ambiguous construct in spite of years of research. We propose that chronic stress is best understood as a series of acute stress responses, and that these responses become maladaptive when they occur frequently or are of long duration. We focus on the factors that contribute to chronic stress: whether the presence of a stressor, real or imagined, is long-lived and/or frequent (repeated activation), the degree to which the stressor is perceived as a threat even when no longer present (low or slow adaptation), and the extent to which the duration of responding is prolonged (delayed or failure to return to homeostasis). Importantly, we examine how perseverative cognitions (such as rumination and worry) contribute to chronic stress by creating and sustaining acute stress responses, largely via influencing activation, adaptation, and return to homeostasis. Finally, we discuss the implications of our stress model: that interventions can be ideographically tailored to address how an individual is experiencing chronic stress; that researchers may be better able to identify specific characteristics of chronic stress that relate most strongly to poor health; and that moderators of chronic stress may function through these contributing factors rather than via general effects on the system.

Introduction

Everyone ''knows'' that stress is bad for you – we are told that it can make you ill and even kill you. Research generally supports this view; stress is related to the risk of cardiovascular disease, HIV/AIDS, and major depression, with emerging evidence linking stress to upper respiratory tract infections, asthma, herpes viral infections, autoimmune diseases, and wound healing (e.g. Cohen, Janicki-Deverts, & Miller, 2007). Additionally, the literature describing plausible biological pathways whereby chronic stress may influence disease onset and/or progression is rapidly growing (see Miller, Chen, & Cole, 2009; for review). Recent reviews have concluded that stress is related to complex alterations in both endocrine (e.g. Miller, Chen, & Zhou, 2007) and immune processes (e.g. Byrne-Davis & Vedhara, 2008; Segerstrom, 2010), and highlighted large individual differences in response to stress. Some work also suggests that stress may impair normal genetic processes, notably DNA expression and repair (e.g. Flint, Baum, Chambers, & Jenkins, 2007; Kiecolt-Glaser, Stephens, Lipetz, Speicher, & Glaser, 1985).

The Scope of This Paper

In spite of the ubiquity of stress as a construct, and the tremendous amount of research on stress in behavioral medicine and health psychology, it remains a challenge to clearly label, model, and understand. Providing comprehensive reviews on the extensive complement of stress theories is beyond the scope of this paper; where appropriate, we direct the reader to source material. The overarching purpose of this paper is to synthesize the major extant perspectives so as to provide a simple schematic for helping to understand

the structure and function of stress, and how it may connect our experiences of the environment to patterns of risk and resilience. Notably, our objective is to provide a heuristic model by which stress has an activating, and perhaps a *sustained* activating, effect on the body's physiological systems that are sensitive to stress and that are implicated in the development of disease. We focus on clarifying the respective domains of acute and chronic stress, and identifying the situations and processes whereby acute stress can become chronic, the contributing factors that give rise to chronic stress, and the unique role that our thoughts play in both creating and sustaining stress and its physiological effects.

It is important to note that there are many other pathways by which stress may influence health. Stress may produce changes in beliefs and attitudes, or other aspects of one's psychological makeup; these in turn may feed back into an individual's risk and resilience by altering the manner in which he or she perceives and/or responds to the environment. Stress is also clearly related to behavior, and a huge array of behaviors has substantive impact on the risk for disease and dysfunction. For example, diet, exercise, and smoking can be influenced by stress, but also important are social/interpersonal interactions and decisions, health-seeking and health care decisions, adherence to medical regimens, and a range of other behaviors that are strongly related to health. In this paper, however, we focus on direct psychophysiological effects.

What Is Acute Stress?

One source of ambiguity in the stress literature concerns the difference between acute and chronic stress. Although most people are confident that they can identify something as ''stressful,'' for example a fight with a spouse or an unhappy marriage, where acute stress ends and chronic begins is much less clear; likewise, there is little in the way of a theoretical framework for differentiating the two constructs.

We consider acute stress to be a discrete process that begins with the occurrence of a stimulus, which may arise in the individual's environment (e.g. being criticized by one's boss), or may be generated from within (e.g. the *memory* of being criticized by one's boss). To reiterate, people are able to re-create events and experiences in their minds based on memory of past or anticipated future events, and acute stress responses can occur in response to these symbolic representations of stressors (e.g. Brosschot, Gerin, & Thayer, 2006; Sapolsky, 2004; Ursin & Eriksen, 2004). Following the occurrence, the stimulus must be appraised as a threat in order for it to be classified a stressor and produce the stress response (see Lazarus, 1966; Lazarus & Folkman, 1984). Thus, to the person who does not feel that the boss' criticism is a threat, there is no stress response. Finally, the threat appraisal produces a set of physiological responses that cause deviation from homeostasis, such as by activating the fight-or-flight response (e.g. Canon, 1914). Seminal work in this area broadly characterized this as a non-specific shift towards sympathetic arousal and a release of neuroendocrine and immune products (Canon, 1914; Selye, 1956/1978), a response designed to facilitate the ability of the organism to cope with the environmental challenge by re-allocating biological resources and priorities. The point at which the body returns to resting/ normative levels or homeostasis signals the termination of the acute stress response. A simple schematic of the acute stress process can be seen on the left hand side of Figure 1. Of note, going forward, when we label something as acute stress we are referring to the end product of this process – the stress response.

Some qualifications are in order. First, to return to appraisal, it is important to consider the perceived emotional valence of the stimulus. For example, engaging in exercise or

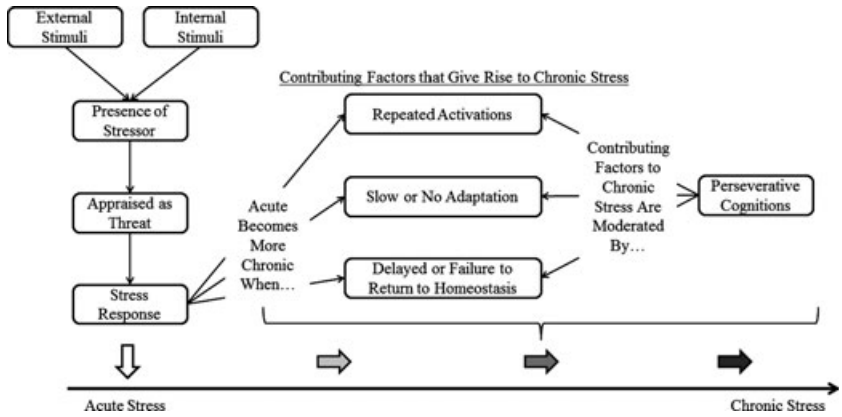

Figure 1 Conceptual model of acute and chronic stress.

being surprised by good news also causes physiological responding that may mimic the stress response. Yet, there is reason to believe that the responding to negative and positive events may not be equivalent – a point Selye (1974) noted when he made distinctions between ''eustress'' and ''distress''. As mentioned above, a component of our definition of acute stress is that the appraisal of the stressor is negative (holding the potential for threat or harm; Lazarus, 1966; Lazarus & Folkman, 1984), and some research shows distinct physiological responding to positive versus negative events (e.g. immunological responses, Yamaguchia et al., 2004; Zeier, Brauchli, & Joller-Jemelka, 1996). Thus, we eliminate stimuli that cause transient emotional or physiological activation but that are appraised as positive from our discussion of acute and chronic stress. Second, we posit that a singular, non-recurring, acute stress response is not implicated in the development of chronic disease. Rather, as we expand below, it is only when the stress response occurs ''too often'' and persists for ''too long'' that disease risk develops. Many forms of acute stress actually serve positive functions in that the motivational energy of acute stress can help the individual manage environmental threats and demands. For example, jumping out of the way of a speeding bicycle involves the body's responses to an acute stressor, but its effects are not likely to persist for more than a few minutes. In fact, acute physiological activation typically provides the resources needed to escape the threat, normatively followed with a rapid return to pre-stress levels when the incident has passed. An exception would be a situation in which the strain placed on the body from an extreme acute stress experience causes a biological overload, such as sudden cardiac death (see Steptoe & Brydon, 2009).

What is Chronic Stress?

In considering our view of acute stress, the questions that become important are, what are the individual and environmental factors that give rise to repeated and/or persistent experiences of acute stress, what factors may delay a return to homeostasis, and why extended stress responses pose a health risk? These questions, although concerned with acute stressors, also begin to characterize chronic stress. In brief, we argue that chronic stress, and associated health risk, occur when the stress response is ''too often'' or ''too long'' and produces a sustained effect on biological systems. The meaning and implications of those statements, however, is complex and requires elaboration.

Many common conceptualizations of chronic stress consist of persistent and undesirable arrangements of the environment, including low socioeconomic status, being in prison, or caring for a disabled child or parent. These are often labeled as ''chronic stress'' insofar as the apparent presence of the stressor and, perhaps, the stress response, persist over time. Yet this definition is insufficient, as within those situations some individuals will be less affected than others. Moreover, this definition does not account for all aspects of topography of the response, for example, such as a person having a stress response but failing to return to homeostasis even after the event has terminated (e.g. Baum, O'Keefe, & Davidson, 1990). Clearly, then, chronic stress is more than just a property of objective environmental characteristics.

What should be becoming clear at this point is that we view a pure distinction between acute and chronic stress as a false dichotomy. To be more specific, we assert that chronic stress is more properly thought of as a continuum from a single, brief acute stress response to the cumulative burden of repeated activations that in its most extreme form represents a persistent stress response with little or no reprieve (as we schematically outline in Figure 1). Thus, our focus becomes understanding the conditions that lead the acute responding to being ''too often'' and ''too long,'' or rather to have a greater degree of ''chronic-ness.''

There have been several efforts to classify what ''too often'' and ''too long'' may consist of (e.g. Baum et al., 1990). Baum and colleagues suggest that the most ''classic'' chronic stress situation is when a stressor itself persists for a long duration, but they suggest additional classifications such that the appraisal of the stressor as a threat can either be brief (acute) or can continue (chronic), and the stress response to a stressor is either short-lived (acute) or long-lived (chronic). Their broader point was that chronic stress cannot simply be defined as *X* number of stressors that persists for *Y* number of minutes, but rather that stressors may have a whole constellation of effects (both directly and indirectly related to the initial stressor) that will either result in the body returning to homeostasis or remaining in a persistent stress response over time. Building from this, we consider a ''pure'' acute stress experience one where the stressor presents itself for only a brief amount of time, where that stressor is perceived as a threat for only as long as the stressor is present, and where the body returns to homeostasis shortly after the stressor is removed. In turn, we extend this logic by suggesting that a chronic stress experience begins to arise when one or more of the above conditions are not met.

Given that there are different contributing factors that produce chronic stress, it is likely that not all experiences of chronic stress are identical. One theory that also posited different contributing factors to chronic stress was McEwen's (1998) allostatic load theory; put simply, allostatic load is a representation of the number of times a person has cycled through a stress response. McEwen suggested that one could have a large allostatic load for different reasons, including an environment that caused repeated activations, a situation in which an individual was unable to adapt in a normal fashion to environmental stressors, and when a person never recovers to their baseline state. We find these different sources a useful starting framework to describe chronic stress; we construe these as contributing factors to chronic stress and label them repeated activations, low or slow adaptation, and failure to return to homeostasis. Where we attempt to extend this theory is that allostatic load is primarily concerned with the effects on biological systems, whereas our focus is on the factors that give rise to chronic stress. Thus, our discussion of chronic stress attempts to extend this work by evaluating how each of the conditions that deviate from a ''truly'' acute stress response may contribute to chronic stress (see Table 1; Figure 1). One final comment prior to moving on is that, although we discuss each

Table 1 Contributing factors to chronic stress and how perseverative cognitions moderate them

Contributing factors to chronic stress	Conditions that give rise to factors	Why factors are negative (stress response is…)	How perseverative cognition moderates factors
Repeated activations	Presence of stressor* Appraisal	Too often	Self-generates additional stressors
Low or slow adaptation	Appraisal* Presence of stressor Duration of response	Too often and too long	Contributes to continued appraisal
Delayed or failure to return to homeostasis	Duration of response*	Too long	Further delays recovery

*Indicates the mechanism we believe to be the primary source contributing to the form.

contributing factor independently, it is certainly possible that individuals can experience multiple factors contributing to chronic stress at the same time.

Repeated activations

We posit that the first contributing factor to chronic stress is the frequency of acute stressors. We argue that this includes what has often traditionally been considered chronic stress (e.g. caregiving, low SES), but can be better characterized as a context that serves to generate more frequent stressors and thus acute stress responses, whether in the form of the same stressor or a series of thematically related stressors (arising from the environmental context). Repeated activations can also result from many ''unconnected'' acute stressors, or some combination of both (i.e. activations both from the environmental context as well unrelated stressors). Furthermore, as we note in Row 1 of Table 1, even when the environment is not producing stressors, repeated activations can be caused by individuals perceiving neutral or benign stimuli as threatening, producing stress responses even in the absence of ''real'' external stressors, thus making appraisal important in this contributing factor to chronic stress. Repeated activation is thus negative primarily because it causes an individual to have a stress response too often.

Low or slow adaptation

It is often the case that when a stressor is presented repeatedly or persists for some time, psychoaffective and physiological responses will habituate (i.e. show a decrease in the elicited response). Our second contributing factor to chronic stress is when the individual does not adapt in this typical fashion to repeated or persistent environmental stressors. Lack of adaptation may reflect either a lack of habituation to repeated exposure to the same stressor, and/or continuing to show a physiological response even when the stressor should no longer be judged as threatening. Thus, on a basic level, low or slow adaptation also is influenced by how long a stressor is present (especially in the case of lack of habituation), but we suggest the primary mechanism of action is the persistent appraisal of stressors as threatening (see Row 2 of Table 1). Following this notion, the duration of response is also relevant here as continued (re)appraisals of a stressor can result in continued stress responding. Low or slow adaptation will thus result in prolonged exposure to negative psychoaffective and physiological changes both because a person is put in a stress state for too often and for too long.

Delayed or failure to return to homeostasis

Our final contributing factor to chronic stress is when subsequent to the ''end'' of the stressor a person either takes longer to recover to homeostasis than is needed to address the stressor, or even fails to return to homeostasis and ''resets'' to a slightly more activated biological resting state. This slow (or impaired) recovery occurs in response to frequent and/or persistent stress responses, as the body ''interprets'' these as reflective of an environment that is characterized by greater challenges and thus requires more biological resources and greater priorities assigned to meet potential threats (McEwen, 1998). Moreover, resting levels for homeostasis may ''rise'' over time (i.e. greater basal activation). Failure to return to homeostasis thus contributes to chronic stress primarily because it places a person in a stress state for too long (see row 3 of Table 1).

The health implications of chronic stress

An important question remains – why is chronic stress harmful? If acute stress responses are frequent and/or persistent, an individual is consistently under the strain of responding to environmental contingencies and challenges. The seminal work of Hans Selye, and the influential work of Bruce McEwen more recently, help to explain why repeated acute stress responses can lead to poor health and increased risk of disease and dysfunction. Both Selye, in his General Adaptation Syndrome (GAS) model, and McEwen, with allostatic load theory, posit that the initial acute response to a stressor is highly adaptive. In contrast, when such acute stress responses are sufficiently persistent, so as to not allow the body to ''recover'' from them (i.e. return to homeostasis), continuous episodes of acute responding become biologically negative. Briefly, the GAS states that after an initial alarm phase (what we would call ''acute responding''), the continued presence of a stressor eventually leads the body to limit its degree of responding (resistance) and ultimately to fail to address a stressor at all (exhaustion) which can lead to disease (Selye, 1956/1978). Recall that in our model, the continued presence of a stressor may be due either to its continued physical presence or to continued mental representations.

Echoing this notion, allostatic load suggests that the negative health effects of stress are generally not due to dramatic stressful events in one's life, but rather in response to the frequent, albeit typically more minor, stressors in one's daily life that cause a physiological response (McEwen, 1998; McEwen & Stellar, 1993). McEwen draws on allostasis (Sterling & Eyer, 1988) which described the adaptive mechanisms of the body to physiologically respond to stressors in the environment and to maintain this responding until the stressor is resolved. Summarized simply, McEwen proposes that frequent and repeated episodes of allostasis (i.e. one's allostatic load) produce disease risk because the body changes and adapts in response to frequently being in a stressed state, but that these modifications place increased strain on the body thus increasing biological and disease risk (see McEwen, 2006; McEwen & Seeman, 2006). The essential conceptual feature of these theories in the context of this paper is that it is the burden produced by the totality of our stress responses that poses risk; this burden is not a single episode, but rather the cumulative index of all responses over time. In other words, chronic stress is bad because it keeps an individual in a fight or flight state, the cumulative burden of which can be harmful (''too often'' and ''too long'').

Perseverative Cognitions as a Potent Influence on Chronic Stress

We have alluded to an additional process that moderates the contributing factors to chronic stress: perseverative cognitions. As we have noted, individuals have the ability to

(re)create stressors in their minds through symbolic representations of the actual event. For example, a person may continue to ruminate about the argument that he had with his boss earlier in the day, despite the fact that he is no longer at work; it is similarly common to worry about things that might occur in the future. We propose that this capacity for cognitions that perseverate after the stressor is no longer present thus influences and interacts with the contributing factors to chronic stress, resulting in the generation of chronic stress in several distinct ways (see last column of Table 1; right side of Figure 1). First, reliving past stressors or imagining future stressors can serve as its own source of stress, as physiological systems are activated along with these thoughts (e.g. Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006). Second, perseverative cognitions can increase the likelihood that individuals will appraise stimuli as threatening, even when they are neutral or after a stimulus has become harmless (through processes such as lowered perceived control, reappraisal, and threat regulation; e.g. Brosschot et al., 2006; Matthews & Funke, 2008; Taylor et al., 2008). Third, perseverative cognitions can powerfully extend the duration of a stress response by (symbolically) maintaining the stressor presence after it is objectively no longer present; thus extending the stress experience (e.g. Brosschot et al., 2006).

In sum, we propose that perseverative cognitions allow for a range of pathways by which cognitions and affect related to the stress experience can persist even after the ''actual'' stressor – the event that occurs in one's environment - has ended and is no longer present; a similar pattern will occur even when the stressor has never (or will never) occur – such as for imagined future events. This perseveration, in turn, can result in a failure to shut off physiological responses and return to homeostasis. Given that perseverative cognitions typically produce a stress response when the stressor/threat is not actually present, the capacity of the fight-or-flight system to remove or resolve the threat is greatly reduced. Accordingly, individuals may experience (self-generated) stressors and acute stress responses more often and for longer periods of time. Overall, then, we assert that perseverative cognitions are a potent pathway that can give rise to and sustain chronic stress, even in the absence of external/environmental stressors.

Implications of This Chronic Stress Model

Our chronic stress model posits that ''stress'' resides not in the initiating event itself (the argument, the memory or anticipation of the argument), nor in the appraisal of the event as a threat or not a threat; these are necessary precursors, but do not themselves encompass the processes that contribute to chronic disease. Rather, the persistent exposure to the negative psychoaffective state and the concomitant physiological deviation from homeostasis comprises what we refer to as ''chronic stress.'' Our synthesis of the stress literature describes the nature – and resultant topography – of chronic stress as highly variable. If one accepts this more nuanced conceptualization of chronic stress, several implications emerge for designing and implementing tailored interventions to reduce chronic stress, examining whether all contributing factors to chronic stress equivalently lead to disease risk, and understanding how individual difference characteristics lead to differential responding to stressors.

Implications for interventions

Our model provides a new way to think about the design and implementation of interventions; that chronic stress treatment be ideographically tailored to address the

contributing factors to chronic stress. For example, if chronic stress arises from frequent activations, we may look for interventions that allow a person to alter her or his environment or to anticipate future occurrences and identify available resources to deal with the stressor before it arises (e.g. proactive coping; Aspinwall & Taylor, 1997). In contrast, both negative appraisals and perseverative cognitions represent problematic adaptations for how individuals can perceive the environment. In this case, cognitive behavioral therapy might be most appropriate to help replace excessively negative thoughts with more positive and adaptive thoughts and behaviors, or to reduce the frequency and/or intensity of perseverative cognitions (see Meichenbaum, 1977). Finally, many interventions (e.g. diaphragmatic breathing) try to directly reduce sympathetic arousal by eliciting a relaxation response so as to reduce the amount of time a person is in a stress response state (see Benson, Greenwood, & Klemchuk, 1975). Here, as in other sections, we discuss these elements in isolation; however, we recognize that interventions often address multiple components of the chronic stress response. Mindfulness meditation approaches, for example, often include both appraisal and relaxation components (e.g. Baer, 2003). Although we hope that our model provides a conceptual framework for thinking about these issues, future work is clearly needed, notably, to test the efficacy of interventions tailored specifically to the address the contributing factor(s) to chronic stress a person is experiencing (especially relative to untailored interventions). Additionally, we believe that there is great promise in integrating this chronic stress model with intervention approaches that deliver appropriate intervention components tailored to unique moments and/or contexts of risk in daily life (e.g. when one is engaged in perseverative cognition; see Heron & Smyth, 2010; Smyth & Heron, 2011).

Is one contributing factor to chronic stress worse than the others?

With the different contributing factors to chronic stress laid out above, it becomes evident that chronic stress can be characterized by very different physiological patterns. These can include moderate activation that is consistent over long intervals, intense bursts that occur in sequenced spikes, jagged and unpredictable responses with little uniformity, and everything in between. As aforementioned, we generally accept that the total exposure to stress responses (roughly the intensity by duration, more properly the total area under the curve [AUC]) is a good estimate of chronic stress. Yet, it remains unclear if the physiological strain of responding to acute challenges is equal across these different response patterns (i.e. perhaps not all AUCs are created equal). We acknowledge of course that the various contributing factors to chronic stress may all be equally pathogenic, and the AUC would thus be the best estimate of chronic stress related disease risk; however, other possibilities also exist. It may be that rapid ''cycling'' between stress responses and quiescence produces more physiological strain and disease risk, or that the greater risk is due to duration of recovery from a peak that occurs during the ''acute'' phase to resting values. Alternatively, it may be any number of more complex emergent properties (e.g. some dynamic interaction of slope, variance, peak values, etc.); these are open, and exciting, questions for future research. As our characterization of the source, nature, and even ''shape'' of chronic stress states improves, future work is needed to understand how and when each type of chronic stress leads to disease risk.

Moderators of chronic stress

Our model also allows a more granular understanding of how moderators affect chronic stress; specifically that stress moderators likely do not influence the stress experience in its

entirety, but rather influence the factors that contribute to chronic stress and perseverative cognitions. It is beyond the scope of this paper to provide a thorough analysis of how our model impacts all possible moderators; instead, we use social support as an example and template for considering the relationship between other moderators and chronic stress. Increased social support is both associated directly with less stress and better health (Uchino, 2006) and has been described as a buffer to stress (Cohen, 1985), thus indirectly improving health. Our model suggests that the direct effect might particularly occur via reducing the number of stressors that may be present, but also by preventing an individual from engaging in perseverative cognitions (Puterman, DeLongis, & Pomaki, 2010). In contrast, buffering effects may occur primarily via a reduced likelihood that environmental demands are perceived as exceeding one's resources (i.e. influence appraisal). Thus, although both social support pathways reduce stress responses, they may function in different ways. More broadly, our model allows that a moderator may not exert a ubiquitous effect on all aspects of chronic stress, but rather influences (some subset of) the contributing factors that give rise to chronic stress. Future work on moderators may benefit by providing a more nuanced understanding of when and how a moderator exerts its effect.

Summary of Model

We have suggested that chronic stress is best understood as a series of acute stress responses, and that these responses become maladaptive (i.e. become chronic stress) because they occur too often or have too long a duration. Importantly, we posit that chronic and acute stress are not specific and exclusive constructs, but instead represent different points on a temporal continuum of physiological responding. What differentiates these points – and allows us to determine when an acute stress situation starts to evidence chronicity, thus transitioning to chronic stress – are the following distinguishable factors that contribute to chronic stress: the presence of a stressor whether real or imagined is long-lived and/or frequent (repeated activation); the stressor is perceived as a threat even when it is no longer present or a threat (low or slow adaptation); and the duration of responding to a stressor is prolonged (slow or failure to return to homeostasis). We hope that this conceptualization allows for a more nuanced analysis of chronic stress, and believe it opens up several avenues of future work. For example, research examining if these different contributing factors to chronic stress produce similar disease risk. Interventions could use this model to ideographically tailor treatment to individuals' specific form of chronic stress, or perhaps even move to *in situ* tailoring that dynamically adjusts to the sources and nature of the chronic stress context. This model also suggests that moderators of chronic stress may function through these contributing factors rather than via a unitary or ubiquitous effect. Finally, we assert that that the full range of factors contributing to chronic stress are powerfully moderated by the extent to which a person engages in perseverative cognitions. In summary, we conclude with a quote – astonishingly from nearly 2000 years ago – that underscores this point:

> Thus it is that foresight, the greatest blessing humanity has been given, is transformed into a curse. Wild animals run from the dangers they actually see, and once they have escaped them worry no more. We however are tormented alike by what is past and what is to come. A number of our blessings do us harm, for memory brings back the agony of fear while foresight brings it on prematurely. No one confines his unhappiness to the present.

Seneca, Letter V from ''Letters from a Stoic,'' (0064 CE)

Short Biographies

Joshua M. Smyth received his PhD in Health-Social Psychology from Stony Brook University. He is Professor of Biobehavioral Health and Medicine at The Pennsylvania State University. His work encompasses three broadly defined areas: (1) What are the effects of stress on health? (2) Can we assess stress, affect, and health in an ecologically relevant manner? (3) Can psychological interventions improve health and well-being? His recent work synthesizes these areas to use real-time, ambulatory data capture to dynamically tailor the implementation and delivery of *in situ* interventions to promote health and well-being.

Matthew J. Zawadzki received his PhD in Social Psychology from The Pennsylvania State University. He is currently a post-doctoral fellow in Biobehavioral Health at The Pennsylvania State University. His research investigates how psychological processes affect health both indirectly (e.g. how beliefs and stereotypes influence the delivery of health care) and directly (e.g. how rumination raises blood pressure and predicts the onset and progression of cardiovascular disease). A recent publication of his in *Health Psychology* examines the effect of rumination and anxiety as mediators of the relationship between loneliness and poor sleep and depression.

William Gerin received his PhD in Social Psychology from Columbia University in 1984, and then completed a postdoc in cardiovascular epidemiology at Cornell Medical Center. He has worked since that time at Cornell, Mount Sinai, and Columbia University, and recently joined the faculty of the Department of Biobehavioral Health at The Pennsylvania State University. He has specialized, over the years, in three main areas: Measurement of blood pressure and diagnosis of hypertension, controlled clinical trials in non-pharmacological treatments for hypertension in minority populations, and the psychoaffective and physiological mediators of stressor effects on subclinical cardiovascular changes, and hypertension and cardiovascular disease. One of his next projects concerns the effects of social support as an intervention that may lower blood pressure in hypertensive patients.

Endnote

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