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Best practices for stress measurement: How to measure psychological stress in health research

Alexandra D Crosswell and Kimberly G Lockwood

Abstract

Despite the strong evidence linking psychological stress to disease risk, health researchers often fail to include psychological stress in models of health. One reason for this is the incorrect perception that the construct of psychological stress is too vague and broad to accurately measure. This article describes best practices in stress measurement, detailing which dimensions of stressor exposures and stress responses to capture, and how. We describe when to use psychological versus physiological indicators of stress. It is crucial that researchers across disciplines utilize the latest methods for measuring and describing psychological stress in order to build a cumulative science.

Keywords

health psychology, measurement, psychological distress, quantitative methods, stress

Introduction

Epidemiological studies confirm that both experiencing a greater number of stressful events and reporting high perceived stress over long periods of time are associated with worse mental and physical health, and mortality (Epel et al., 2018). The association between greater stressor exposure and increased disease risk has been replicated with many different types of stressor exposures (e.g. discrimination, caregiving, work stress) and a range of aging-related health outcomes (e.g. cardiovascular disease, metabolic syndrome, mortality). The mechanistic pathways underlying these associations have also been detailed (Boyce, 2015; McEwen, 2015; Miller et al., 2009). Despite this compelling evidence, however, health researchers often measure stress using unvalidated measures or select a single type of stress to measure, thus either missing entirely or underestimating the role stress plays in predicting disease onset or progression.

One of the main reasons for the lack of sophisticated measurement and inclusion of psychological stress in health models may be the incorrect assumption that stress is too broad and nebulous of a construct to accurately measure. It is true that psychological scientists too often fail to specify what they mean when using the term "stress" or other variants such as "stressor," "acute stress," "stress response," and "stress biomarker." Social and behavioral scientists tend to use the term loosely, often failing to define it clearly in a manuscript and using it to refer to a range of experiences, from living in poverty to giving a public speech to current negative mood. Kagan (2006) pointed out this lack of specificity, providing a fair critique of the state of the literature. The lack of specificity in language, however, does not represent a true lack of specificity in theoretical or methodological approaches. Although psychological stress researchers have made great strides in differentiating different forms of stress in recent decades, the problem is rather that the language used in journal articles has not always accurately reflected these advancements—and these advancements have been kept within a small, specialized subset of researchers. Thus, the purpose of this article is to provide health researchers across disciplines with a useful update on best practices for measuring stress and offer suggested language for how to describe stress-related constructs with more granular language.

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Fundamentals of stress measurement

The term "stress" is an umbrella term representing experiences in which the environmental demands of a situation outweigh the individual's perceived psychological and physiological ability to cope with it effectively (Cohen et al., 2016). One important distinction in studying stress is to differentiate between exposures to stressful events and the responses to these events. Stressful events or "stressors" are discrete events that can be objectively rated as having the potential to alter or disrupt typical psychological functioning, such as losing your job or getting divorced. Stress responses are the cognitive, emotional, and biological reactions that these stressful events evoke.

Measuring stressor exposures versus stress responses

Stressor exposures can be measured with self-report questionnaires such as a life events checklist, assessed by an interviewer, or objectively determined based on proximity to an event (e.g. living in NYC during the September 11 terrorist attacks). The Life Events and Difficulties Schedule (LEDS; Brown and Harris, 1978) is a structured interview protocol that is considered the gold standard for assessing stressor exposure across someone's lifetime. This interview protocol is time intensive in both the data collection and data processing stages. To streamline the process of capturing stressor exposures across the life span, a computer-assisted methodology was developed (e.g. The Stress and Adversity Inventory [STRAIN]; Slavich and Shields, 2018). In both the LEDS and the STRAIN, participants are asked whether they have experienced a range of stressful life events at any point in their life. For each endorsed stressor, they are asked follow-up questions to provide greater context about the experience (e.g. how old were you when it happened, how long did it go on for, how stressful or threatening was it). The LEDS requires a trained interviewer to administer the measure, while the STRAIN can be completed either by an interviewer or by participants themselves. The LEDS also relies on blind raters to score the severity of a stressor using this contextual information, while the STRAIN relies on the participants reporting of event severity. The STRAIN's automated structure of follow-up questions allows the respondent to complete the interview much more quickly than the LEDS and reduces data processing time. Both measures provide a comprehensive assessment of stressor exposures across the lifespan, and use different methods to determine the severity of these experiences.

An individual's response to the stressor sometimes matters more than mere exposure to it, particularly when it comes to the impact of the stressor on physical health. For example, caregiving for a family member with a debilitating illness is often considered a chronic stressor because of the constant physical and emotional demands. There is a significant amount of research examining the impact of being a dementia caregiver, in particular, given the large increase in the number of family dementia caregivers as the population ages in the United States. In fact, the Alzheimer's Association estimated in 2018 that there were over 16 million family caregivers providing an estimated 18.5 billion hours of care to people with Alzheimer's or other dementias (Alzheimer's Association, 2019). Empirical evidence has shown that family caregivers of Alzheimer's patients have worse physical and mental health compared to age-matched non-caregivers (Kiecolt-Glaser et al., 1987; Vitaliano et al., 2003). However, not every caregiver's health is damaged by their caregiving role (Roth et al., 2015). This may be because the negative impact of caregiving is caused by individuals' *subjective response* to the caregiving situation, not from the mere exposure of being a caregiver. Thus, a better predictor of health decline would be the degree to which caregivers report high levels of psychological burden from their caregiving role. Empirical evidence supports this perspective; for example, Alzheimer's caregivers who reported emotional distress or physical strain from caregiving had 63 percent greater mortality than caregivers who reported no distress (Schulz et al., 1999).

Stress responses can be measured with self-report measures, behavioral coding, or via physiological measurements. These responses include emotions, cognitions, behaviors, and physiological responses instigated by the stressful stimuli. One of the simplest ways to measure stress responses is through self-reports of perceived stress related to a specific stressor or to one's life circumstances (Cohen et al., 1983). For example, the Perceived Stress Scale is a 10-item self-report measure that captures an individual's perception of how overwhelmed they are by their current life circumstances. Responses to acute stressors have traditionally been studied in controlled laboratory settings in order to capture responses that unfold within minutes of stressor exposure (e.g. emotional and physiological reactivity to an acute stress task). A commonly used acute stress paradigm is the Trier Social Stress Test (TSST), a standardized laboratory stress task in which participants give a speech and perform mental arithmetic in front of judges (Kirschbaum et al., 1993). The TSST reliably evokes an acute stress response for the majority of participants. Outside of the laboratory, new technology has enhanced our ability to capture real-time stress responses in daily life using mobile phones and wearables, which many researchers are now doing. Considering the impact of both stressor exposure and stress responses on health may improve the prediction of health outcomes, as many models of stress propose that the stress response mediates the effect of stress exposures on health outcomes (McEwen, 1998; Wheaton et al., 2013).

Table 1. Types of stress by timescale.

Type of stress	Definition	Relevance for health
Chronic stress	Chronic stressors are prolonged threatening or challenging circumstances that disrupt daily life and continue for an extended period of time (minimum of one month).	People under the chronic stress are at greater risk of chronic illness, mortality, and accelerated biological aging (Epel et al., 2018; Holt-Lunstad et al., 2015; Nyberg et al., 2013).
Life events	Life events are time-limited and episodic events that involve significant adjustment to one's current life pattern, such as getting fired, being in a car crash, or the death of a loved one. Some life events can be positive (e.g. getting married, moving to a new place), and some become chronic (e.g. disability caused by car crash).	Exposure to more stressful life events is linked with poorer mental health in addition to development and progression of cardiovascular disease, as well as mortality due to cardiovascular disease and cancer (Chida et al., 2008; Cohen et al., 2007; Steptoe and Kivimäki, 2013).
Traumatic life events	Traumatic life events are a subclass of life events in which one's physical and/or psychological safety is threatened.	Experiencing a greater number of traumatic events across the life course is consistently associated with worse health and mortality (Gawronski et al., 2014; Keyes et al., 2013; Krause et al., 2004; Rosengren et al., 2004).
Daily hassles (i.e. daily stressors)	Interruptions or difficulties that happen frequently in daily life such as minor arguments, traffic, or work overload, and that can build up overtime to create persistent frustration or overwhelm.	Greater emotional responses to these daily hassles are associated with worse mental and physical health (Almeida, 2005; Charles et al., 2013; Chiang et al., 2018; Sin et al., 2015).
Acute stress	Short-term, event-based exposures to threatening or challenging stimuli that evoke a psychological and/or physiological stress response, such as giving a public speech.	Greater cardiovascular reactivity to acute stressors has been prospectively associated with increased risk of cardiovascular disease (Brosschot et al., 2005; Chida and Steptoe, 2010; Steptoe and Marmot, 2005).

Selecting stress measures

Due to constraints on participant burden and other considerations, difficult choices about which type of stress to measure need to be made by researchers. Common types of psychological stress measured using self-report questionnaires in adult samples are major life events, traumatic events, early life stress exposure, and current chronic or perceived stress in various domains (i.e. loneliness, marital discord, experiences of discrimination, work stress, financial strain, neighborhood safety and cohesion, and current perceived stress). The choice of which type of stressor exposure to measure depends on what is most relevant to the study population, the specific research question, and the hypothesized mechanisms linking that stress type to the outcome of interest. To begin the selection, consider first what is the most relevant stress type(s), given the sample's demographic makeup. For example, measures that capture religious persecution or combat exposure would be particularly important for a sample living in a conflict zone, while the amount of overwhelm related to being a parent (parenting stress) may be most relevant for a sample of mothers caring for their child who has an autism spectrum disorder. In both cases, it would also be important to measure types of stressors that may not be directly related to the circumstances—such as levels of loneliness and financial strain. Capturing a range of stressor types reduces the likelihood that the individual's psychological and social distress is underestimated.

Stressor and stress response characteristics

In addition to identifying stressor type(s) of interest, there are several key measurement considerations when choosing specific measures of stress to include in studies or analyzing existing stress measure data. These considerations include characteristics of the stressor or response (e.g. timescale, types of stressor response) as well as measurement characteristics (e.g. life stage of exposure and measurement assessment window). We briefly describe these aspects below (see Epel et al., 2018 for further discussion).

Timescale of the stressor

Stressors generally take place along the following timescales: chronic stressors, life events, daily events/hassles, and acute stress. Table 1 provides definitions for each of these timescales. It is important to note that naturalistic experiences of stress rarely fall neatly into one category. For example, death of a loved one is often considered a major life event but, depending on the cause of death, may also be considered a chronic stressor, such as if the family member was sick for years or months before the death. Similarly, arguments with a spouse may be considered an acute stressor, but if they happen every day they may be considered chronic. There is a significant amount of gray area between categories. Researchers should first make a

thoughtful attempt to pick the category that best aligns with the stressor and with the way that stressor type has been described in past research, and then describe the exposure with as much specificity as possible.

Types of stress response

Responses to stressor exposures provide additional useful information beyond measuring stressor exposure alone. Stress responses include psychological, behavioral, cognitive, and physiological reactions related to the stressor exposure that can occur before, during, or after the exposure. Psychological stress responses include specific emotions triggered by the stressor, as well as efforts to regulate that emotion (Gross, 2002). Behavioral responses include coping behaviors such as smoking or seeking social support. Cognitive responses include appraisals of the exposure (e.g. as a threat versus challenge; Blascovich and Mendes, 2010) and perseverative cognitions (e.g. rumination Brosschot et al., 2005). Physiological responses include immune, autonomic, neuroendocrine, and neural changes related to stressor exposure. Further details about the various stress responses deserve more attention than can be described here (Epel et al., 2018). As a part of selecting stress measures, researchers should identify the type of stress response that is most relevant for their research question and sample. Often, studies will assess multiple types of stress responses simultaneously.

Additional characteristics of the stressor

There are additional stressor exposure attributes that can be described and captured to thoroughly assess the exposure. These include, but are not limited to, duration, severity, controllability, life domain, the target of the stressor (e.g. self, close other), and the potential of the stressor to elicit specific harmful emotional responses (e.g. social status threat). Lack of control, social status threat, and stressor severity have been identified as potent attributes that predict worse outcomes across a range of stressor types and scenarios.

Characteristics of stress measurement

Life stage during stressor exposure

In addition to the timescale of the stressor, another important characteristic of stressor exposure is the developmental or life stage during which the stressor occurs. Knowing the person's age during the exposure informs hypotheses about which psychological and biological processes the stressor may have impacted. This is because developing systems are more open to environmental cues and are thus more likely to be impacted by stress exposure. "Sensitive periods" are specific time points in the life course during which physiological systems are maximally influenced by external environmental factors, and thus stressor exposure can have a particularly strong influence on development (Knudsen, 2004). Sensitive periods during which stress may have the greatest effect are likely: prenatal (Van Den Bergh et al., 2005; Weinstock, 2001), before age 5 (Zeanah et al., 2011), during puberty (Fuhrmann et al., 2015), entry into parenthood (Saxbe et al., 2018), and during menopause (Gordon et al., 2015). Identifying and measuring stress during sensitive periods could greatly increase our understanding of who is at risk for the negative effects of stress, the mechanistic pathways linking stress exposure to health decline, and where and how to focus intervention efforts.

Measurement assessment window

The window of measurement is also essential to consider to avoid measurement error and improve specificity in hypotheses. Measures can ask about stressors and stress responses across a wide range of time frames, such as in the present moment, over the course of that day, the past week, the past month, the past year, in childhood, or across the entire lifespan. For example, there are fundamental differences in a measure that ask participants to report on stress exposure in the past month versus across their lifespan. The latency between stressor exposure and measurement is crucial, as retrospective autobiographical reports are prone to bias and error, especially when there have been years or decades since the exposure in question (Bradburn et al., 1987; Hardt and Rutter, 2004). In addition to the latency between exposure and measurement, several other factors can impact the accuracy of retrospective reports, such as mental state at the time of recall and the emotional salience of a given memory (Shiffman et al., 2008). This may lead to overestimating the frequency of emotionally salient stressors and underestimating the frequency of more mundane, daily stressors (Bradburn et al., 1987; Shiffman et al., 2008). For these reasons, it can be beneficial to measure stressor exposure and responses in close proximity to their occurrence whenever possible.

The experimental studies examining acute stressor exposure and responses, there are additional considerations with the measurement assessment window. Because the timing of stressor exposure is controlled, researchers can begin measuring psychological, behavioral, and physiological states prior to the stressor exposure and continue measuring throughout and after exposure. By measuring responses before, during, and after exposure, researchers can access (and predict) anticipation of and recovery from the stressor exposure.

Summary of steps for selecting stress measures

There are of course numerous considerations for selecting the appropriate stress measure for your study. In sum, researchers should identify the type or types of stress that are most relevant to their research question and sample. Stress measure selection should then be refined based on characteristics of the stressor and/or stress response that the

Table 2. Summary of steps for choosing appropriate stress measures.

Steps for choosing an appropriate measure of psychological stress.
1. Determine the type(s) of stress you intend to capture based on your research question and the uniqueness of your sample.
2. Determine the timescale of the stressor exposure and how you will capture objective exposure. For the exposure variable, in particular, you may need to develop your own measure based on the uniqueness of your sample.
3. Identify which types of stress responses you are able to assess in your study design (e.g. psychological, behavioral, cognitive, physiological).
4. Determine the life stage in which the stressor occurs and choose a measure appropriate for that particular life stage.
5. Identify additional characteristics of the stressor that are important to capture (e.g. severity, controllability, target of the stressor) and how these will be assessed (e.g. objective reviewer, participant report, a priori study design).
6. Consider your measurement assessment window and select measures that are specific to the time frame of exposure and/or response you intend to capture.
7. Look for well-validated scales that capture these aspects. It is common to use multiple scales to capture different aspects of the stress exposure and stress response, and the range of stress types that might be relevant for your sample. The Stress Measurement Network Toolbox provides validated and curated stress measures (https://stresscenter.ucsf.edu/).

researcher intends to measure, such as the timescale, the type(s) of stress responses the researcher is interested in, and other attributes of the stressor (e.g. duration, severity, controllability). Selection of stress measures should also account for measurement characteristics, such as the life stage during stressor exposure and the measurement assessment window (e.g. framing of questions, timing of assessment relative to occurrence of the stressor).

Beyond these stress-specific considerations, researchers should also follow general best practices for measure selection. For example, validated scales should be used when available. The Stress Measurement Network Toolbox provides a resource for validated measures of different types of stress that has beeen curated by experts (https://stressmeasurement.org). Measures should also be selected based on the uniqueness of the sample, and hile validated scales are preferred, some samples or exposures may require researchers to develop a new scale or modify an existing scale to fit their needs. These practical steps for selecting a stress measure are summarized in Table 2.

Compelling evidence linking stress to physical health

The types of stress that have the most consistent and compelling relationships with disease risk and mortality are acute stress reactivity, early life stress, work or occupational stress, and social isolation/loneliness. A comprehensive review of these literatures is outside the scope of the present article; however, the following section highlights foundational studies linking these stress types physical health, with a particular emphasis on cardiovascular disease (because it is the leading cause of death in developed countries) and mortality. Effect sizes are included where possible, as are references to reviews and meta-analyses for further reading. Of note, we do not review the literature here on the impact of cumulative life stress (aggregate number of stressor exposures and/or intensity of stress responses over one's life course). Despite initial compelling work on the impact of cumulative life stress on cardiovascular disease outcomes, this area of research is still in its infancy, with a need for measurement approaches to be unified across research studies to allow for building of a collective science (Albert et al., 2013; Slopen et al., 2018).

Research on acute stress reactivity and physical health

Decades of research have shown that heightened cardiovascular reactivity and delayed recovery to acute stressors are prospectively associated with increased cardiovascular disease risk (Brosschot et al., 2005; Chida and Steptoe, 2010; Steptoe and Marmot, 2005). One of the earliest studies in this area was a longitudinal study of healthy adult men (age 45–55; $n=279$) in which those classified as "hyper-reactors" (defined as >20 mmHg increase in diastolic blood pressure to the cold pressor acute stress task) were 2.4 times more likely to have a myocardial infarction or die from cardiovascular disease in the following 20 years than men who showed a rise of <20 mmHg (Keys et al., 1971). Cortisol and inflammatory responses to acute stressors have also been shown to prospectively predict incident hypertension (Hamer and Steptoe, 2012; Steptoe et al., 2016). Heightened reactions and prolonged recovery time periods may be driven by perseverative cognitions before (worrying) and after (rumination) stressor exposure (Brosschot et al., 2005, 2006). Despite the evidence linking reactivity to disease outcomes, the clinical meaningfulness of these associations is still debated (Treiber et al., 2003). Importantly, a blunted response to an acutely stressful situation (sometimes termed a "hyporeactive response"), is also linked to worse health (Carroll et al., 2017). For example, in a sample of 725 healthy adults from the Dutch Famine Birth Cohort Study, decreased cardiovascular and/or cortisol response to the acute stressor was associated with obesity, risk of becoming obese, depressive symptoms, anxiety, and poor self-rated and functional health

(De Rooij, 2013). In addition, there are several other reactivity patterns that have been hypothesized to represent maladaptive response profiles such as lack of habituation when exposed to repeated stressors of the same kind (see McEwen, 1998). Thus, the clinical meaningfulness of different stress reactivity profiles is largely debated.

Research on early life stress and physical health

The evidence linking early life stress to increased adult disease risk and mortality is strong. A foundational study in this area, the Adverse Childhood Experiences (ACE) Study, included nearly 10,000 adults and demonstrated that a greater number of self-reported retrospective adverse childhood experiences (e.g. physical abuse, living with an alcohol-dependent adult, witnessing violence) was positively associated in a graded relationship with the presence of ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease, after controlling for demographic factors (Felitti et al., 1998). Convincingly, reporting seven or more ACE was associated with three times the likelihood of heart disease compared to reporting no ACE (Dong et al., 2004). These findings have been so compelling that significant changes in clinical and educational settings have been undertaken in recent years to recognize the role that early trauma has on current and future cognitive, socio-emotional, and behavioral outcomes for both children and adults.

Research on work stress and physical health

Epidemiological studies consistently demonstrate associations between high work stress and worse physical and mental health. One of the most widely studied models of work stress is job strain, which is a combination of high demands (workload and intensity) and low control (Karasek, 1979). Decades of research has linked high job strain to anxiety and depression, increased blood pressure (BP), cardiovascular events, and metabolic syndrome (Chandola et al., 2006; Landsbergis et al., 2013; Madsen et al., 2017; Nyberg et al., 2013). An analysis of the Whitehall II study cohort found that chronic work stress was associated with coronary heart disease (CHD) risk, with the associations strongest in participants under 50 (RR = 1.68, 95% CI 1.17–2.42). Other components of work stress, such as effort-reward imbalance, also predict cardiovascular disease risk (Dragano et al., 2017).

Research on social isolation, loneliness, and physical health

A meta-analysis of decades of work on social isolation and loneliness found that being socially isolated, lonely, and/or living alone corresponded to an average of 29 percent, 26 percent, and 32 percent increased likelihood of mortality (Holt-Lunstad et al., 2015). The mortality risk for the most socially isolated adults in the National Health and Nutritional Examination Survey (hazard ratio (HZ) = 1.62 for men, HZ = 1.75) was found to be comparable to the risk of smoking (HR = 1.72 for men, HZ = 1.86) and having high BP (HR = 1.16 for men, HR = 1.32 for women) (Pantell et al., 2013). These strong relationships suggest that meaningful connection with others is an essential component of health and well-being. Several short measures have been created to capture this important social determinant of health, including a validated three-item measure of loneliness (Hughes et al., 2004).

Biological pathways from stress to disease

There are numerous plausible biological pathways linking stress to cardiovascular disease, with most of the current evidence pointing to stress-related alterations in the immune, autonomic, and neuroendocrine systems. The brain networks that orchestrate stress-induced changes in these peripheral systems have also been identified (Gianaros and Wager, 2015; Gianaros and Jennings, 2018), and can be described as the systems related to threat processing, safety processing, and social cognition (Muscatell and Eisenberger, 2012). One widely accepted stress-disease model is the "wear and tear" hypothesis (Charles et al., 2013; McEwen, 1998; Selye, 1956). This hypothesis is centered on the postulation that prolonged or repeated stress prematurely depletes the finite amount of "adaptational energy" of the organism, decreasing the body's ability to successfully adapt to environmental challenges (Selye, 1956). In this model, stressful events cause stress responses that involve activation of physiologic systems. In the short term, mobilizing physiological resources to respond to a discrete event or threat is adaptive. In the long term, however, frequent and repeated mobilization of these resources wears down these response systems and maladaptive patterns appear (McEwen, 1998). The "wear and tear" hypothesis is theoretically compelling, but currently lacks definitive empirical support. This is because we do not currently have data that demonstrates the slow degradation of multiple physiological systems over decades in humans, an effort that requires tremendous investment. Instead, most studies have chosen one or maybe two physiological systems to measure to try to capture degradation or maladaptive responses to stressors, thus providing support, but not direct evidence for the "wear and tear" hypothesis. Other potential pathways include stress-related changes in endothelial function, elevated chronic inflammation, metabolic dysfunction, changes in DNA repair, changes in gene expression, and telomere shortening. These are all exciting areas of research, some of which fit in to the "wear and tear" hypothesis (e.g. telomere shortening; Epel et al., 2004) and others that suggest alternate processes (e.g. biological embedding of early experiences; Miller et al., 2011). These pathways are relevant for numerous chronic diseases beyond cardiovascular disease.

Associations between stress and immune system functioning are especially relevant given that the major diseases of aging in the United States are mediated, in part, through the immune system. The top three leading causes of death in the United States—cardiovascular disease, cancer, and chronic lower respiratory disease—all share the common thread of being characterized by elevated chronic inflammation (Aghasafari et al., 2019; Golia et al., 2014; Grivennikov et al., 2010). Because of this common thread, chronic systemic inflammation has become a recent focus of health research. Stress exposure has been examined extensively as a predictor of increased systemic inflammation. Indeed, elevated systemic inflammation has been found in those experiencing chronic stress like caregivers (Gouin et al., 2008), immediately after a stressful life event like death of a loved one (Cohen et al., 2015), historical stress like childhood adversity (Slopen et al., 2010, 2012), daily stress (Chiang et al., 2012), and in response to lab-based stress tasks (Marsland et al., 2017). A short-term inflammatory response to stress is thought to be adaptive because it involves recruiting immune cells to the site of a real or potential injury in order to heal wounds resulting from stressor exposure. However, when there is no wound to heal, as is the case with psychosocial stressor exposure, repeated or exaggerated inflammatory responses may cause long-term damage and contribute to disease processes (Black and Garbutt, 2002; Miller et al., 2002; Rohleder, 2014).

Is there an "objective" way to measure stress?

Stress and health researchers have searched for many years for a single biological indicator that someone is "under stress." However, there is no single stress-specific biomarker. This is likely because acute stress is not the only state that evokes reliable biological changes (e.g. increased heart rate and BP). Other non-acute stress states, such as feeling excited, focusing attention on non-negative affect inducing stimuli, or exercising, also trigger biological responses that are similar to those evoked by negative affect inducing acute stressors like increased heart rate and blood pressure. This is even true for what is often termed the "stress hormone," cortisol—not all cortisol increases are triggered by increases in psychological stress responses, nor does every experience that people perceive as "stressful" cause cortisol to rise (Dickerson and Kemeny, 2004).

While measuring stress-related biomarkers may not provide a perfect indicator of whether someone is under stress or not, there are still compelling reasons to include these biomarkers in research studies of stress and health. Stress-related biomarkers are objectively measured biological indicators of physiological processes that are either implicated in the pathway from stress to disease or serve as a marker of that process. In typical models of the stress-health relationship, the stressful event (X) leads to a biological change (Y) that then leads to the disease state or related outcome (Z). Stress-related biomarkers can be the variable inserted in any component (X, Y, or Z) of this model; examples of the stress-related biomarker in each part of this basic model are shown in Figure 1. In example A, the biomarker serves as a mediator, or a part of the causal pathway between a stressor and a health outcome. In example B, the biomarker serves as a predictor of stress-related psychosocial and behavioral processes that ultimately impact health outcomes. In example C, the biomarker serves as an outcome of psychological and physiological responses to a traumatic stressor. The way a biomarker is conceptualized (e.g. as a mediator, predictor, or outcome) depends on the research question and study methods. As such, choosing a stress-related biomarker to include in a study depends on the design of the study and the outcomes of interest. Table 3 provides further tools for how to choose the appropriate biomarker. It is also important to keep in mind that a biomarker may not be needed to answer a research question, despite the desire for a seemingly "objective" indicator of stress or stress reduction.

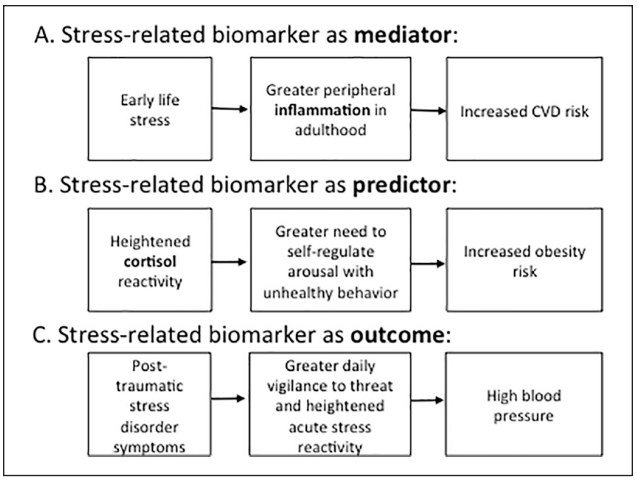

Figure 1. Examples of how stress-related biomarkers can be modeled as either the predictor, the mediator, or the outcome in research studies.

One area of research that requires particularly careful consideration of biomarker selection is when biomarkers are used as an outcome in psychosocial intervention trials. The scientific community is often eager to find an objective biological indicator that a psychosocial intervention can improve health; this is typically done by measuring improvement in a biomarker from pre- to post-intervention. There has been a trend in recent years toward using changes in biomarkers as an indicator of an intervention's success, rather than relying on subjective psychological reports of well-being. This approach is problematic for several

Table 3. Essential questions for following best practices in choosing an appropriate stress-related biomarker.

Questions to answer to help identify the right stress-related biomarker for your study:
1. What are the plausible biological pathways linking my stress predictors to my health outcome? The first step is to identify which physiological system is the likely candidate that is related to the health outcome of interest and that previous evidence has linked to stress or stress-related psychological processes.
2. What is the window of time that the stressor can plausibly have its impact for? If the stress response is short, is there a plausible reason it would have long-lasting impacts?
3. Is there a biomarker that captures functioning of the pathway identified in Question 1, and that reflects the appropriate timeline (Question 2)?
4. Is this biomarker associated with any end disease states relevant for my population of interest?
5. If you are proposing to use this biomarker for an intervention study, is the biomarker sensitive enough that it can change in the proposed intervention period window? Is it stable enough that the control condition would remain relatively stable during the intervention period? Would the expected change in the intervention group be clinically meaningful?
6. Are you able to collect the biomarker specimen well enough that is worth the subject burden and research cost? For example, while drawing blood is often the best way to capture many biomarkers, it is more invasive and requires more wet lab capacity than collecting saliva samples.
7. Is this biomarker needed to answer my research question or can this question be answered with a self-report or task-based measure? Biomarkers may not be needed despite initial excitement and desire to include a potentially "objective" indicator of stress or stress reduction.
8. For studies examining an acute stress response, what is the expected pattern of response? Complicating biomarker selection is that there is limited empirical evidence that identifies what a "bad" or "good" physiological acute stress response pattern is. This is because stress exposures take many forms, and thus the most adaptive response depends on a myriad of immediate contextual factors, such as what the goal of the arousal is.

reasons, including variability in baseline biomarker profiles, unknown reliability of biomarker assessment over time, unknown stability of these changes, and lack of evidence for the long-term impact of small changes in stress-related biomarkers on disease risk. Therefore, biomarkers should not replace self-report, behavioral, and cognitive outcomes as primary outcomes in psychosocial intervention trials aimed at reducing stress or related goals.

Variability in exposures and responses

Despite stress exposure being an inevitable part of life, not everyone develops stress-related illnesses at the same speed. One primary reason for this is that stress exposures are not distributed evenly across social groups. Women, young adults, members of racial-ethnic minority groups, divorced and widowed persons, and poor and working-class individuals report greater chronic stress and cumulative stress exposure across their lives (Thoits, 2010). In addition, recent research has demonstrated that both psychological and physiological stress responses vary remarkably within and between people. While the physiological systems that are activated in response to a stressor are generally universal and non-specific as initially proposed by one of the founders of the field of stress, Hans Selye (1956), the pattern of these responses vary considerably in terms of the degree of the system's activation and how long the systems are activated for. Individual-level differences and environmental contexts interact to influence the psychological and physiological stress response trajectories. These include socioeconomic and cultural factors, genetic and developmental factors, historical and current stressors, stable protective factors, and health behaviors. A model integrating these different levels of experience is presented by our group in detail in Epel et al. (2018) and reprinted here with permission (Figure 2).

Advanced statistical models can be used to examine variability in stress responses (both psychological and physiological) within and between people (Bryk and Raudenbush, 1987; McArdle and Epstein, 1987). Within-person variability in stress responses means that a person's response to a stressor within one life domain (e.g. work) does not necessarily predict how they will respond to a stressor within another life domain (e.g. family). Between-person variability means that different people respond to the same stressor in a variety of ways. As an example of variability in psychological stress responses, in a sample of 1,532 healthy adults from the Changing Lives of Older Couples prospective study, psychological responses to the death of one's spouse took on four discrete trajectories (e.g. chronic grief, chronic depression, temporary depression, resilient), suggesting that there is not one universal pattern for spousal grief (Galatzer-Levy and Bonanno, 2012). Cortisol can be used as an example of variability in physiological stress responses Cortisol generally increases in response to laboratory-based acute stress tasks if they are uncontrollable and characterized by social-evaluative threat (Dickerson and Kemeny, 2004), such as the TSST described earlier (Kirschbaum et al., 1993). However, around 30 percent of people do not mount a cortisol response, and there is tremendous variability in the size of the response. Individual-level predictors of this variability include age, gender, sex steroid levels, smoking, coffee, and alcohol consumption (Kudielka et al., 2009). Interestingly, these

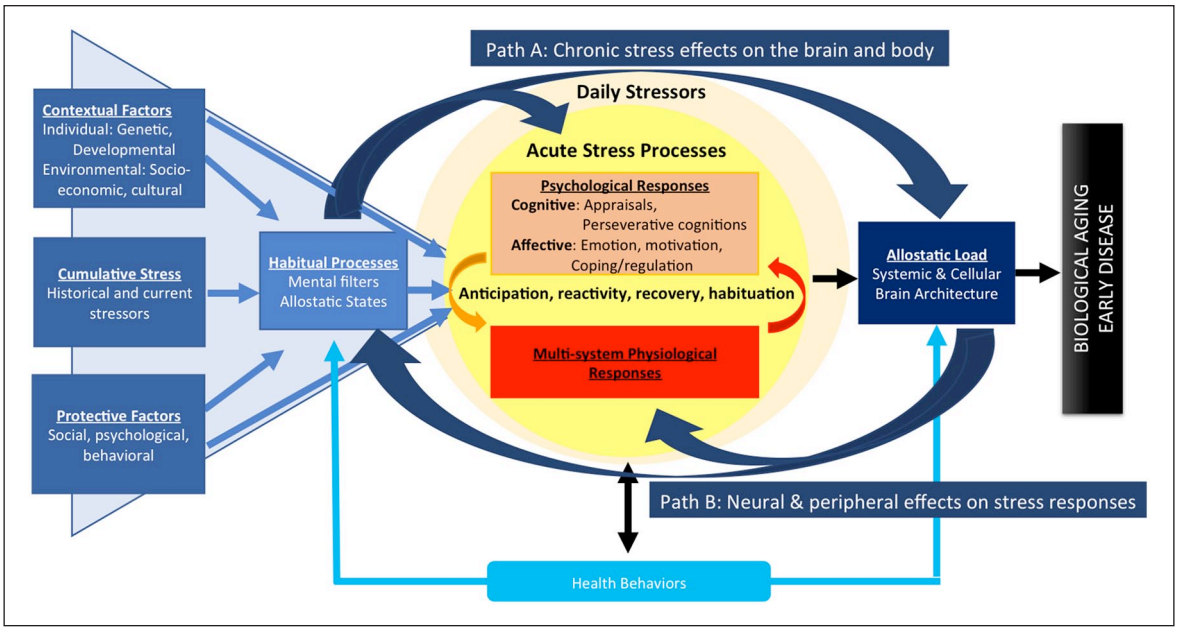

Figure 2. Transdisciplinary model of psychological stress: Integrating contextual, historical, habitual, and acute stress processes.
Figure 2 presents a transdisciplinary model that describes psychological stress as encompassing as a set of interrelated processes. The figure illustrates that stressors are experienced within the context of a person's life, represented by the contextual factors in the blue triangle. These contextual factors include individual-level characteristics such as personality and demographics, the environment in which one lives, current and past stressor exposures, and protective factors—all of which combine to determine the baseline allostatic state of physiological regulation, and the lens through which stressors are perceived and assigned meaning. Contextual factors and habitual processes together influence psychological and physiological responses to acute and daily stressors. These responses, if dysregulated, are thought to lead to allostatic load and ultimately biological aging and early disease. Reprinted from *Frontiers in Neuroendocrinology* (Epel et al., 2018).

differences are not driven by differences in the emotional responses to the task as acute stressors are not strongly correlated to the physiological responses. In a review of 49 acute stress studies, only 25 percent reported a significant correlation between the two emotional and physiological responses (Campbell and Ehlert, 2012).

Conclusion

Empirical evidence supports a strong relationship between psychological stress and disease development. These studies may be underestimating the impact of stressor exposure and the stress response on health, given that measuring these constructs has been challenging and limited. Recent work in the stress field has identified important aspects of psychological stress to capture in order to fully test the role that psychological stress plays in predicting disease; these include capturing the specific type(s) of stressor exposure, a wide range of psychological, cognitive, behavioral, and physiological responses to the exposure, and contextual and individual-level factors that moderate the impact of the exposure and response. In this article, we identified ways for researchers to improve the language specificity when describing stress measures and offered guidance on how to choose the appropriate stress measure. We encourage the adoption of more precise language when writing about stress in academic papers, more careful selection of stress measures, with a focus on validated measures when possible, and theoretically driven integration of mechanistic pathways linking stress to health outcomes. The ultimate goal of having sophisticated research on the relationship between stress, health, and well-being is to develop evidence-based ways to help people thrive in our stress-filled world.

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