Behaviour Research and Therapy 101 (2018) 20–29

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Everyday stress response targets in the science of behavior change

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ARTICLE INFO

Article history:
Received 4 March 2017
Received in revised form
13 August 2017
Accepted 25 September 2017
Available online 28 September 2017

Keywords:
Stress
Ecological momentary assessment
Daily diary
Health behavior
Physical activity
Sleep
Science of Behavior Change (SOBC)

ABSTRACT

Stress is an established risk factor for negative health outcomes, and responses to everyday stress can interfere with health behaviors such as exercise and sleep. In accordance with the Science of Behavior Change (SOBC) program, we apply an experimental medicine approach to identifying stress response targets, developing stress response assays, intervening upon these targets, and testing intervention effectiveness. We evaluate an ecologically valid, within-person approach to measuring the deleterious effects of everyday stress on physical activity and sleep patterns, examining multiple stress response components (i.e., stress reactivity, stress recovery, and stress pile-up) as indexed by two key response indicators (negative affect and perseverative cognition). Our everyday stress response assay thus measures multiple malleable stress response targets that putatively shape daily health behaviors (physical activity and sleep). We hypothesize that larger reactivity, incomplete recovery, and more frequent stress responses (pile-up) will negatively impact health behavior enactment in daily life. We will identify stress-related reactivity, recovery, and response in the indicators using coordinated analyses across multiple naturalistic studies. These results are the basis for developing a new stress assay and replicating the initial findings in a new sample. This approach will advance our understanding of how specific aspects of everyday stress responses influence health behaviors, and can be used to develop and test an innovative ambulatory intervention for stress reduction in daily life to enhance health behaviors.

Stress, broadly defined, is the imbalance between perceived environmental, psychological, and/or social demands and an individual's perceived capacity to adapt (Lazarus & Folkman, 1984). Large, prolonged, and frequent responses to everyday stress (e.g,. arguments, work overload) can influence the risk for adverse long-term health outcomes, including a wide range of disease outcomes (e.g., cardiac, metabolic, neurologic, and others; e.g., Bose, Oliván, & Laferrère, 2009; Johansson et al., 2010; Piazza, Charles, Sliwinski, Mogle, & Almeida, 2013; Rosengren et al., 2004). Put simply, stress is a process that begins with an external or internal stimulus (e.g., a real or imagined experience; a stressor), which, when perceived as harmful or threatening (i.e., threat appraisal), results in a stress response (Miller, Gordon, Daniele, & Diller, 1992; Smyth, Zawadzki, & Gerin, 2013). Stress responses can result not only from infrequent major life events or traumas, but also from minor yet frequent everyday occurrences, such as concerns about work, interpersonal conflicts, or unexpected events that disrupt daily life (Almeida, 2005). Responses to everyday stress consist of robust, short-term (e.g., same day) effects on emotional, behavioral, and physical functioning (e.g., Almeida, Piazza, Stawski, & Klein, 2010; Zautra, 2005), which may increase long-term vulnerability to morbidity and mortality (e.g., Charles, Piazza, Sliwinski, Mogle, & Almeida, 2013; Mroczek, Stawski, et al., 2015; Piazza et al., 2013).

In this paper, we present a conceptual overview of our approach to developing an everyday self-report stress response assay to

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https://doi.org/10.1016/j.brat.2017.09.009
0005-7967/

measure stress response targets, which are comprised of stress response components (described more fully below) and stress response indicators (how components are indexed). Our focus on targets and assays, of those that are relevant to understanding stress responses in daily life, adopts the experimental medicine, mechanism-focused approach to behavior change research proposed by the National Institute of Health's Science of Behavior Change (SOBC) program (scienceofbehaviorchange.org; https://commonfund.nih.gov/behaviorchange/index). The SOBC program is designed to enhance our understanding of human behavior change that cuts across a broad range of health-related behaviors, and integrates basic and translational science and cuts across many disciplines. The goal is to collectively establish the foundation for a unified science of behavior change by supporting research to improve our understanding of human behavior change and using this knowledge to improve and enhance behavioral interventions.

Key first steps in the experimental medicine approach are identifying intervention targets that produce desired behavior change and developing tools (i.e., assays) for measuring such intervention targets. Specifically, our targets are everyday stress responses and we propose a stress response assay that will offer the conceptual and operational definitions of three specific components of everyday stress responses (reactivity, recovery, and pile-up) as they unfold in real-time and in individuals' natural environments (e.g., physical location, activity, presence of others, social interactions, etc.). Moreover, we allow for the stress response to have separate indicators (negative affect and perseverative cognitions) that might be differentially important across the stress response components. We then describe a set of coordinated analyses to assess these stress response targets and identify their links to the enactment of daily health behaviors. Finally, we outline how such information can be used for novel intervention development that leverages the within-person assessment model, along with between person information, to identify moments of unique risk (e.g., for a given stress response component or for a particular health behavior).

1. Proposing stress response targets

Although everyone experiences stress, there is considerable variation in the nature of stress responses and their effects on health behaviors and health outcomes, both between and within individuals. A sensitive and informative stress assay should, therefore, not only identify *who* is broadly at risk for stress-related dysfunction (i.e., between-person effect; shows broad differences between individuals), but also identify situations and times *when* people are at risk (i.e., within-person effect; reveals dynamic processes within individuals). Our approach is within-person because it emphasizes measuring relatively short-term emotional and cognitive responses to stress in order to identify moments of risk. In particular, we look at initial responses to a stressor (i.e., stress reactivity), the persistence of responses after initial reactivity (i.e., stress recovery), and the temporal patterns of repeatedly experiencing (and/or responding to and recovering from) stressors (i.e., pile-up of stress responses). As such, we posit that a within-person approach that captures ecologically valid data at many time points in daily life will reveal the everyday stress response process as it unfolds naturally in time and in context. Moreover, this can be done, on average, across individuals as well as within individuals. Taken together, the results will provide a more comprehensive and nuanced picture of within-person stress processes.

Our assay will measure two distinct domains that comprise the stress response. The first domain identifies the *stress response components*, which comprise the three temporal dimensions just introduced: Reactivity, Recovery and Pile-up (collectively, RRP). In other words, we posit that stress response components – RRPs – are distinguished by the magnitude (reactivity), persistence (magnitude and/or length of recovery) and frequency (pile-up) that reflect stress responses. The second domain assesses *stress response indicators*, which reflect the emotional and cognitive aspects of the stress response. From the broad range of possible options we have selected affective and cognitive response indicators that can provide valid, reliable, feasible, and timely measurements of stressor reactivity, recovery, and pile-up and that are conceptually and empirically linked to stress-related outcomes and processes. Specifically, our assay will measure responses (e.g., to reported stressors) using two indicators: (a) negative affect [NA] and (b) perseverative cognitions [PCs] (e.g., worry and rumination; Brosschot, Gerin, & Thayer, 2006) in an attempt to broadly capture responses to both externally and internally generated stressors. Previous work has shown within-person associations between everyday stressors and increases in NA (e.g., Almeida, 2005; Smyth et al., 1998) and PCs (e.g., Moberly & Watkins, 2008). NA and PCs negatively impact engagement in health behaviors (including both physical activity and sleep behavior; e.g., Anton & Miller, 2005; Brummett, Babyak, et al., 2006; Clancy, Prestwich, Caperon, & O'Connor, 2016; Farmer, et al., 1998; Jerstad, Boutelle, Ness, & Stice, 2010; Jones, O'Connor, McMillan, & Ferguson, 2007). In addition, NA and PCs can be reliably measured and exhibit short-term within-person variability (Murry, Allen & Trinder, 2002; Stone, Smyth, Pickering, & Schwartz, 1996; Watson, Clark, & Tellegen, 1988) making them malleable targets for interventions designed to produce proximal changes to health behaviors.

We hypothesize these indicators are proximal mechanisms that derail people's health behaviors (e.g., efforts to be more active, less sedentary, and get a good night's sleep). There are, of course, many other indicators that could be used and we do not assert that these are the only useful indicators. Quite to the contrary, we hope to identify and present a general conceptual framework that can readily be applied to other indicators. Hence, using the primary domains, our initial potential *stress response targets* consist of the six possible combinations of response *components* and *indicators*; that is, reactivity, recovery, and pile-up will be separately identified for NA and PC.

Fig. 1 illustrates the distinction among reactivity, recovery and pile-up, using our two primary stress response indicators, NA and PC. (Although not depicted to keep the figure simple, we believe that the response patterns for each indicator will be largely distinct from one another.)

Reactivity reflects the highest degree of initial increase following stressor occurrence. Recovery is operationalized by estimating the degree to which elevations post-stressor persist following the event. Response pile-up refers to repeated patterns of reactivity and recovery depicted in this figure that occur within a narrow time interval (e.g., within a day or across adjacent days) that reflect repeated "hits." Importantly, these components may differ depending on the indicators (e.g., high levels of NA reactivity do not necessitate high levels of PC reactivity). By measuring specific temporal components of the everyday stress response process (reactivity, recovery and pile-up) across two indicators as they unfold in real-time, we aim to identify specific contributors (or particularly "toxic" combinations of contributors) to poorer health behavior. Such contributors (or combinations thereof) can also be more specific and – in theory – efficient targets for interventions designed to break the link between stress responses and unhealthy behaviors. An important feature of this framework is the relative independence of RRPs. As shown in Fig. 1, reactivity may be large and recovery complete (as for Stressor 1) or reactivity may be relatively small and recovery incomplete (as for Stressor 2). Pile-up reflects the number or frequency of stress responses (e.g.,

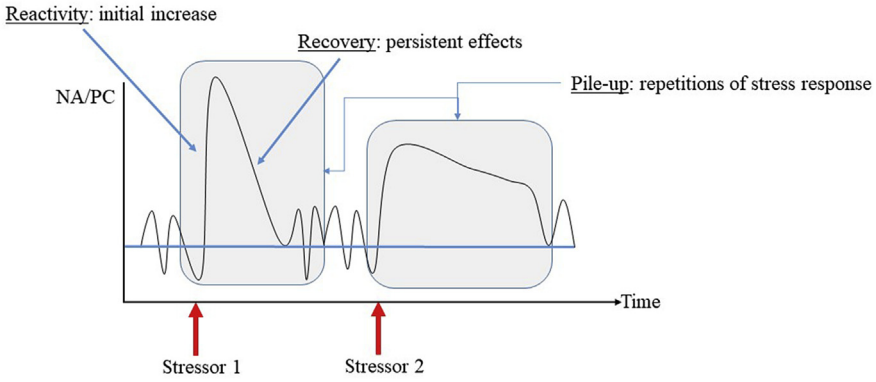

Fig. 1. Everyday stress response components.

reactivity-recovery cycles) within a given temporal window (e.g., the same day, over a week).

2. Basic challenges for operationalizing stress response targets

Numerous end of day [EOD] and ecological momentary assessment [EMA] studies have examined everyday stressors and have made preliminary attempts to infer reactivity; which, at a basic level, can be characterized as a change on a response indicator (i.e., NA or PC) from pre-stressor to the time following the onset of a stressor. Yet, even this basic definition of reactivity means something different depending on the type of study. For example, EOD diary studies usually operationalize stress reactivity as the difference between levels of NA on stressor days compared to days on which no stressor was reported (e.g., Almeida, 2005). Because reports at the end of the day do not distinguish between peak NA in the moments following a stressor and persistently elevated NA in the hours following a stressor, this approach may conflate reactivity with lack of recovery (Scott, Ram, Smyth, Almeida, & Sliwinski, 2017; Sliwinski & Scott, 2014). Designs, such as EMA, that obtain temporally finer grained measurements usually define reactivity as the difference in levels of a response indicator between moments on which a recent stressor is reported compared to moments on which no stressor is reported. This approach assumes that moments on which no stressor is reported—even those following the report of a stressor—represent a "stress-free" baseline. Such assumptions, however, may not be entirely warranted. For example, using post-stressor measurements before complete recovery has transpired contaminates the estimate of baseline with a prior stress response component ([lack of] recovery). In turn, this would result in biased (in this example, systematically underestimated) estimates of reactivity because of (again in this example) inflated or elevated baselines. Thus, one might elect to construct an estimate of baseline only from non-stress moments on non-stress days, or conceptually related approaches. More generally, determining and selecting the appropriate "baseline" in daily life is a challenging but essential task, given the dynamic interplay between stressors and the potential indicators of a stress response (RRP, in either or both NA and PC).

Fewer EOD and EMA studies have explicitly examined the notion of recovery as it relates to the enduring emotional and cognitive effects of everyday stressors. One straightforward way to operationalize recovery is through examining lagged effects of stressors. For EOD studies this might reflect the effect of a stressor today on NA tomorrow (e.g., Charles & Almeida, 2006); for EMA studies this might reflect the effect of a stressor reported earlier in the day on mood reports made later in the day (e.g., Scott, Sliwinski, & Blanchard-Fields, 2013). More nuanced approaches examine the amount of time it takes for the stressor response to get back to baseline (Bergeman & Deboeck, 2014). The temporal window (hours vs. days) of recovery and cutoffs for determining lack of recovery (i.e., return to baseline) will be important considerations in operationalizing this target. Put another way, EMA studies may assess recovery over several hours (or can be aggregated to estimate change over days), but in EOD studies recovery is constrained to only be over days. As such, the temporal window of recovery may differentially be related to health behaviors and may influence appropriate intervention elements (i.e., interventions delivered at the moment or day level).

Pile-up reflects the patterning of stress reactivity and recovery responses that occur across a narrow time frame (e.g., within a day, across adjacent days). For example, pile-up could be viewed as patterning of response "bumps" using some sort of defined running count (e.g., over a week). Some previous work has operationalized pile-up using frequency counts of reported events (e.g., Almeida, 2005; Schilling & Diehl, 2014), but this approach appears to make two assumptions: (1) each event elicits a reaction and (2) individuals recover completely prior to encountering the next stressor. In our view, pile-up it is not simply total exposure or the amount of time a person spends in a state of "stress" (e.g., area under the curve), nor is it merely the intensity of the response (i.e., reactivity) and the time it takes to recover (see Smyth et al., 2013). Our view is that, broadly speaking, pile up reflects the frequency with which one is pulled out of homeostatic range of functioning (cf. Sterling & Eyre, 1988). As such, pile up could (for example) be viewed as the number of times people are exposed to a stressor, exhibit a reaction, and return to baseline within a specified temporal epoch (although alternatives certainly exist; for example, not requiring the presence of a novel stressor, simple counts of exposures, etc.). We note that these RRP indicators are not fully independent. For example, using the definition provided above for pile up, one cannot have pile up without reactivity. Pile up does, however, function differently analytically than reactivity and recovery – the former is essentially a count of "cycles" devoid of specifics of the target, whereas the latter two are conceptually more similar to change scores (with various levels of sophistication) based on the target indicator's value.

A final and critically important consideration is that most approaches to operationalizing components of the stress response, particularly reactivity, assume that people differ from each other, but do not consider the possibility that individuals may vary from themselves across time and context in their reactivity to or recovery from everyday stressors. In fact, there is some data to suggest there is substantial within-person variability in emotional responses to everyday stressors even over relatively short time scales

(Sliwinski, Almeida, Smyth, & Stawski, 2009). This issue is particularly relevant for the current project because our approach to intervention (see below) presumes that we can identify not only *who* tends to be highly reactive, but also that we can identify *when* a given person has experienced a high reactivity moment or prolonged recovery from a stressor.

In the process of developing our assay, we consider and compare various approaches to operationalizing reactivity, recovery and pile-up that will attempt to resolve some of the limitations described above. For example, we are exploring different approaches to estimating baseline for each person that (a) preserve the temporal ordering of pre- and post-stressor measurements, and (b) impose constraints on which pre-stressor measurements are used to define baseline in order to reduce contamination by incomplete recovery. Similar steps will be taken for recovery and pile-up; more generally, these multiple approaches to indexing each of our stress response components allow us to explicitly examine important conceptual and practical issues for stress theory (and, as described in our analysis plans below, we replicate these findings across multiple independent data sets to avoid capitalizing on chance or over-fitting the data).

3. Testing the impact of RRP on physical activity levels and sleep

Everyday stress responses can be construed as generally impacting health through two broad pathways. The first is that chronic stress can lead to biological dysregulation that alters immune (e.g., Segerstrom & Miller, 2004) and hormone function (Miller, Chen, & Zhou, 2007), potentially resulting in increased risk for disease and dysfunction. Although important and of great interest, this pathway falls outside of the scope of work on health behavior change. A second pathway, however, is that stress can impact distal health outcomes by cumulative effects emergent from their proximal influence on daily health behaviors (e.g., Ng & Jeffery, 2003). That is, when people are stressed, they may behave in unhealthy ways, such as being less active and sleeping less (Stults-Kolehmainen & Sinha, 2014; Åkerstedt et al., 2002). Over time, if these behavioral responses to stress are too intense, persistent, and/or frequent, they may lead to long term negative health outcomes (Smyth et al., 2013).

As proof of concept, we focus on how stress responses may undermine two important health behavior domains: (1) physical activity and (2) sleep. We, of course, recognize that physical activity and sleep may influence stress responses (and recursive dynamic processes no doubt exist), but our primary interest at this point is in identifying malleable stress response targets that reliably predict health behavior enactment – as these can be used as the basis for intervention to improve health behaviors. Unhealthy lifestyles have well-documented negative long-term health consequences but relatively little attention has been paid to the acute effect of everyday stress responses on engagement in activities, such as physical activity and sleep. Indeed, we adopt this approach from the perspective that individuals' emotional and cognitive responses to everyday stressors "in the moment" shape their health behavior decisions and enactment that day and contribute to their typical behavior that, over time, give rise to long-term health outcomes.

Physical activity. It is estimated that less than 4% of American adults meet public health recommendations for moderate-vigorous intensity physical activity (CDC, 2008; Troiano et al., 2008). Whereas physical activity improves mood and reduces the risk of chronic disease (Penedo & Dahn, 2005), physical inactivity is a major risk factor for cardio-metabolic (Buman et al., 2014; Matthews et al., 2012; Warren et al., 2010) and neurologic disease (Barnes & Yaffe, 2011) among other negative physical and mental health outcomes. Broadly speaking, stress generally disrupts exercise plans and lowers physical activity (Stults-Kolehmainen & Sinha, 2014); in particular, infrequent exercisers become less active (Lutz, Stults-Kolehmainen, & Bartholomew, 2010; Seigel, Broman, & Hetta, 2002) and increase their discretionary sedentary behavior (i.e., television watching) in response to stress (Mouchacca, Abbott, & Ball, 2013). In sum, we expect everyday stress responses to reduce the frequency of moderate-vigorous physical activity (unless people have strong physical activity habits) and increase the duration of sedentary behavior.

Sleep. Sleep deficiency is generally defined as "a deficit in the quantity or quality of sleep obtained vs. the amount needed for optimal health and well-being" (NHLBI, 2011). Sleep deficiency has direct deleterious effects on the autonomic nervous system, endocrine and metabolic function, and many indicators of cognitive function (e.g., Buxton & Marcelli, 2010; Spira, Chen-Edinboro, Wu, & Yaffe, 2014). Sleep deficiency is also associated with weight gain, obesity, diabetes, hypertension, cardiovascular disease, mental health, immune function, and early mortality (see Luyster, Strollo, Zee, & Walsh, 2012). Both chronic naturalistic and acute experimental stress are robustly associated with sleep deficiency (e.g., Kim & Dimsdale, 2007).

Additionally, studies indicate that days characterized by high stress or negative emotions are typically followed by nights of poorer sleep (Kalmbach, Pillai, Roth, & Drake, 2014). In contrast, adults obtaining the recommended amount of 7 h of sleep on a regular basis are more likely to exhibit optimal health and well-being. We operationalize complex "sleep health" behavior in terms of a sufficient amount, regular timing, and adequate quality of sleep (Buysse, 2014), and expect that everyday stress responses will disrupt positive sleep behaviors.

4. Testing RRP dynamically

Although it is widely recognized that unhealthy lifestyles (e.g., persistent physical inactivity or sleep deficiency) have long-term health consequences, much less attention has been paid to the short-term influence of everyday stress on engagement in these activities. Given that the effects of stress on activity and sleep occur over short timescales (e.g., hours, days, or weeks; e.g., O'Connor, Conner, Jones, McMillan, & Ferguson, 2009; Payne, Jones, & Harris, 2010; Rutledge et al., 2009; Sonnentag & Jelden, 2009; Stucky et al., 2009), responses to everyday stress can create "moments-of-risk" that negatively impact immediate decisions (and perhaps longer term decisions as well) to engage in moderate-vigorous physical activity or sleep. Assisting individuals in coping with "high-risk" moments – or intervening "just-in-time" [JIT] upon momentary or daily stress that is likely to derail intended health behaviors – may be essential for long-term adherence to recommended health behaviors (e.g., Aspinwall & Taylor, 1997; Simkin & Gross, 1994).

Most existing tools to measure naturalistic stress provide broad, relatively static, person-level (i.e., stressed vs. non-stressed) categorizations that miss important temporal and contextual influences critical to shaping health behavior (i.e., patterns of stress and behavior within an individual over time, varying across settings and situations). Reliance on such tools particularly impedes the development of interventions that target specific mechanisms that convey the harmful effects of stress on health behaviors, and ultimately, on health outcomes. The within-person approach to studying everyday stressor responses addresses this limitation by collecting multiple assessments of stressors and responses as they naturally occur in daily life. Moreover, this conceptual and measurement approach is also useful for studying how physical activity and sleep vary within-person (across moments, between days,

etc.). That is, an individual who generally leads a sedentary lifestyle may also have days characterized by more or less sedentary moments; similarly, good or bad sleep behaviors may be characterized by the relative balance of many nights of sleep being good or bad. Overall, then, the emergent 'architecture' of health behaviors is built upon the individual 'bricks' that are instances of behavior (whether very transient or at the daily level); our approach attempts to leverage this dynamic and within-person thinking to better understand how predictors (in this case, indicators of the stress response) relate to within-person variation in health behaviors in everyday life.

Laboratory studies afford precise control over the onset and offset of a single objective event; this permits precise monitoring of the time course, but cannot determine how people respond to stressors from their natural environments (Warren et al., 2010). Stress-behavior relationships may also differ for specific components of the stress response. For example, the inability to "shut-off" a stress response (slow recovery) might interfere with sleep quality (particularly when experienced in the evening), whereas exhibiting a disproportionately strong response to a minor event (high reactivity) might predict disruption of physical activity. Another common limitation of laboratory methods is that they provide limited insight into how stress effects accumulate or 'pile-up' in everyday life. Laboratory paradigms typically measure how people respond to a *single* event; however, in real-life each exposure is typically not so circumscribed. Identification of the temporal features of how everyday stress responses impact sleep and decisions to be physically active thus requires an ecologically-valid, within-person approach that can capture stress response processes and behavioral enactment as they unfold naturally in time and in context.

Whereas exclusively between-person data and/or analytic approaches to studying stress may reveal relatively stable differences between individuals, the *within-person* approach to studying stress processes uses EOD and EMA methods to measure time-varying and contextual effects of stress on health and health behaviors. It also allows for stronger causal inference about the effects of stress on health behaviors by allowing individuals to serve as their own controls, thus ruling out the influence of between-person confounding variables (as each individual is compared to him or herself over time for analyses). For example, assume we were to measure features of interactions between spouses and motivation to exercise for a one week period. An observation that motivation to exercise diminishes at times immediately following a stressful interaction with one's spouse (i.e., within-person effect) could more readily and plausibly be attributed to that event (i.e., a time-varying within person factor) as opposed to a static between person factor such as a person's gender or socioeconomic status (i.e., if those variables remained invariant during that one week time period). Because health behavior decisions are in part contingent upon features of a person's immediate psychosocial environment and their internal psychological states, optimal interventions should not only target people *who* are at risk, but should be delivered at times *when* risk for making poor health behavior decisions is highest and most malleable (Jones, O'Connor, Conner, McMillan, & Ferguson, 2007). Reliance on *only* between-person measurements might misdirect intervention development efforts by failing to provide the temporal information required to assist stressed individuals (e.g., in coping with "high-risk" situations that threaten their adherence to exercise plans; Aspinwall & Taylor, 1997; Simkin & Gross, 1994). Only by using within-person methods is it possible to characterize the dynamics of how people respond to stressors, which can elucidate *specific* targets of the stress process exerting the most potent proximal influences on health behaviors. Moreover, these within-person methods typically also still provide the opportunity to characterize and examine between-person effects as well (often with great precision and high ecological validity).

5. A framework for our analytic approach

Our analytic approach involves coordinated analysis of data from multiple intensive longitudinal data sets that adopted similar within-person approaches to measuring stress responses in people's everyday lives. We will thus test a common set of hypotheses using data from independent studies that differ in their specific measures and also their sampling frequencies (EMA, EOD, or both), but that assess the same constructs (i.e., having indicators of some – and often all – of NA, PC, activity, and sleep). Given our primary interest in developing a self-reported assay of everyday stress, we focus on self-reports of stress. The specific approaches vary across the studies, including varied formats (e.g., Likert, visual analogue scale) to assess stressors/events and perceived stressfulness, different NA and PC items, and so forth. These contributing studies also vary widely in sample size (from about 100 to over 2000), in sample characteristics (e.g., on demographics such as age and gender, geographic region, health status), and the intensity and duration of intensive sampling (e.g., 5 to 20 samples daily for EMA studies over 2–14 days). Collectively leveraging this wide age range, patient and non-patient samples, working and non-working adults, geography, etc., for our coordinated analysis helps enhance the potential generalizability of our findings across measurement strategies, people, and settings.

Coordinated analyses of multiple data sets also strengthens conclusions through construct level replication, a hallmark of rigorous experimental work but relatively uncommon in naturalistic studies. This approach will permit us to address novel questions regarding the timescale over which the effects of everyday stressors operate to influence physical activity and sleep patterns, which will inform the optimal design of future data collection and intervention studies. By evaluating replicability across a variety of protocols and very diverse populations, coordinated analyses will also ensure that our finalized stress assay has widespread utility and applicability.

We employ several analytic approaches to study stress response components (RRP) and selected indicators (NA, PC). These approaches all recognize and take advantage of the hierarchical structure of data produced by within-person assessments. That is, because observations of responses to everyday stress are made multiples times for each individual, repeated measurements are nested within persons. In addition, some of our data sets include repeated measurements that reflect nesting across different time scales. Specifically, data from ecological momentary assessment (EMA) studies include repeated momentary observations made within a day, which are then repeated across multiple days. Thus, EMA data often have two times scales (within-day and across days) that must be considered in devising analytic approaches. In contrast, other studies (such as EOD designs) measure everyday stress across many days, but only a single time scale (i.e., across days but once a day).

Our overall approach is structured into three broad stages:

- Stage 1—Reliability and precision of stress response targets (RRP components across NA, PC indicators)
- Stage 2—Characterizing the time course of stress response indicators (NA, PC)
- Stage 3— Testing association between stress targets (each component [RRP] derived from each indicator [NA, PC]) and health behaviors

We utilize different but complementary analytic methods for

each stage, and attempt to leverage repeated assessments from daily life to extract both within-person and between-person information to better understand everyday stress responses. In Stage 1 we use approaches such as Generalizability Theory (G-Theory) that provide guidance for optimizing measurement reliability. Stage 1 results from G-theory analysis inform us about the number of items required to reliably measure NA and PC, as well as inform strategies for detecting reliable "momentary" change associated with RRP. In Stage 2 we use multilevel modeling (MLM) to examine the temporal properties of the stress response indicators (NA, PC) and of the systematic effects of stress response components (RRP). Stage 2 results provide "normative" information about the average magnitude of stressor reactivity, estimates for recovery, and a characterization of pile-up, including the temporal patterning of stressors (both within and across days). In Stage 3 we examine the most effective and efficient indicators for risk moments based on the associations of the stress response targets (RRP across NA and PCs) to specific health behaviors. Stage 3 will be used to optimize and test an ambulatory assay that can detect whether a particular individual has entered a "moment-of-risk" resulting from a reaction to stressor, an incomplete or prolonged recovery, and/or a concentrated bout of reaction-recovery cycles. We will then use this information to inform the design and testing of sophisticated within-person intervention approaches (e.g., JIT and ecological momentary interventions; see Heron & Smyth, 2010). Additionally, as aforementioned, the analytic approaches are performed simultaneously on multiple data sets in order to immediately establish replicability of results in diverse samples using diverse methods and to avoid 'chance' findings that may emerge in a single data set given the large number of analyses to be conducted (i.e., as a rigorous approach to manage type I error and establish replicability and generalizability). Each of these stages is described in additional detail below.

5.1. Stage 1—reliability and precision of indicators

Stage 1 will utilize approaches such as generalizability theory (G-theory) to determine the reliability of our operationalizations of NA and PC. For example, we can estimate the within-person reliability to determine the minimum number of items needed to reliability assess NA and PC.

Fig. 2 depicts the approach that G-theory takes to answer these questions. Assume each data point comes from a single individual measured across different time points. The goal of our stress assay will be to identify when NA (or PC) has significantly increased (e.g., from a baseline or resting state, or over what may be expected for a given context). For example, we may want to determine whether the increase in NA from 11th to the 12th occasion is sufficient to warrant serving as a trigger for intervention delivery (i.e., represents a stress response reactivity 'risk-moment'). G-theory offers a procedure to determine whether the difference between occasions is large relative to the variability ("error") in measurement within any given occasion (indicated by the vertical density curves).

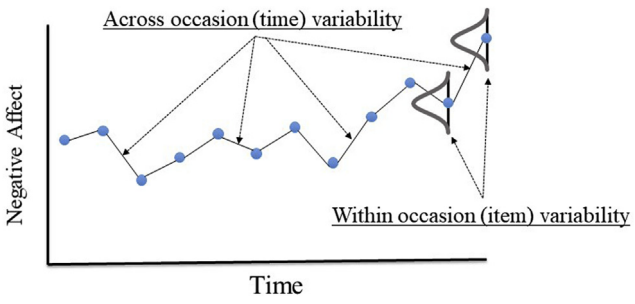

Fig. 2. Estimating reliable change over time.

5.2. Stage 2—characterizing the time course of stress response indicators

Fig. 3 depicts Stage 2 by illustrating the actual data from a sequence of repeated measurements of NA on 12 participants (left panel) from one of our datasets. As an initial step, we will use MLM to decompose the total variability in NA into between-person (i.e., across people) and within-person (i.e., across time) components. Essentially, this decomposition indicates that people exhibit relative differences from each other in their (average) level of NA (top right plot), as well as substantial variability (relative to their own average) across time (indicated by 'observation number' in the bottom right plot).

Once we have decomposed variability into within-person and between-person components, then we will hone in on the within-person variability to determine the time scale(s) over which it transpires. The primary focus of these analyses is to elucidate the time course of within-person variability in stress responses. Therefore, we will extend MLMs to further decompose within-person variability in NA (and PC) across different timescales: within-day and across days. The purpose of this approach is to determine "where most of the action is" in regards to NA and PC. Simply put, if people have a relatively stable mood within a day, but exhibit variability in mood from one day to the next, then an assessment and intervention strategy might be efficiently grounded in daily measurements and administered at the frequency of day. If, however, people exhibit substantial variability from one moment to another within the course of a single day, then that would imply a more time intensive approach that is responsive to rapid stress related fluctuations in NA and PC (e.g., JIT intervention elements, such as recommended techniques to reduce NA, that are tailored to moments of risks within individuals not merely to individuals at more general risk).

5.3. Stage 3—testing associations between stress response targets and health behaviors

Next we will evaluate the association of each stress response target (i.e., each RRP component across our NA and PC indicators) to each of our health behaviors (physical activity, sleep). In our secondary data analyses we are limited by the assessment strategies employed in the original studies. For health behaviors, both physical activity and sleep behaviors, the existing studies include a diverse assortment of subjective self-reports (EMA or daily diary) and wearable devices (e.g., actigraphy). In future work (e.g., replication in new sample) we will use both self-report items (optimized from our earlier work) and more objective assessments collected from empirically validated wearable devices. We will broadly operationalize the health behaviors in two different ways. First, we will examine if people are exhibiting daily behaviors consistent with meeting physician recommended levels (e.g., 150 min of MVPA per week, 7 h of sleep per night). Second, we will analyze clinically and empirically important components of each behavior in a more continuous fashion (i.e., do everyday stress responses disrupt healthy behaviors within persons). For activity, we will examine the frequency of moderate-vigorous physical activity and the duration of sedentary behavior. For sleep, we will examine the amount, timing, and quality of sleep.

A unique feature of our analytic strategy is that the goal is to relate operational definitions of each RRP component to physical activity and sleep *within-persons*. Typically, approaches to analyzing stress responses from EMA and diary data treat indicators such as

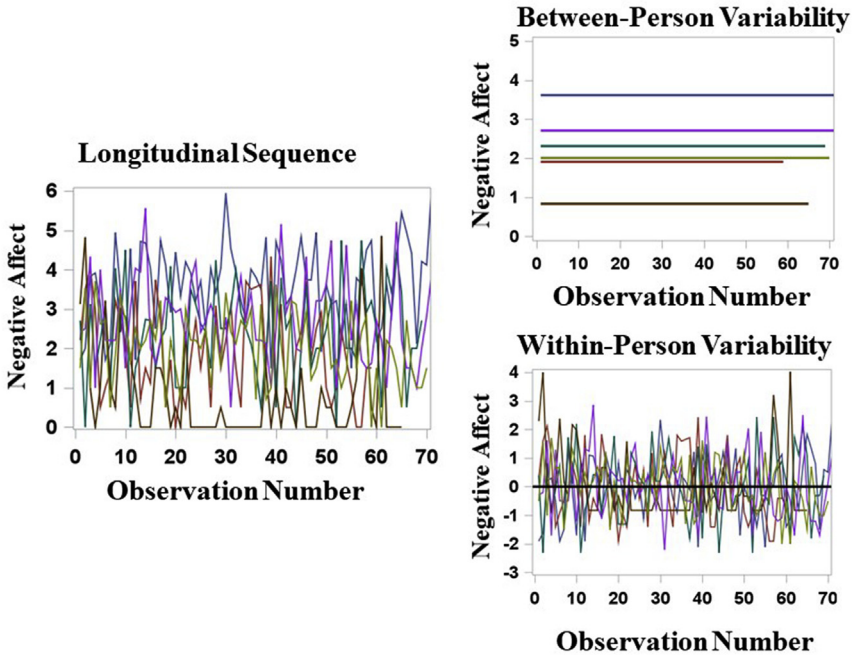

Fig. 3. Depiction of variability.

NA and PC as dependent variables, treat self-reported stress as a predictor variable, and apply MLM to estimate the effects of stress on NA and PC. For example, emotional reactivity has been operationalized by the within-person regression coefficient relating NA to concurrent (or lagged) reports of everyday stressors (e.g., Sliwinski & Scott, 2014). This approach allows characterization of between-person differences in reactivity, which is useful for answering *who* questions, such as, "are highly reactive people more likely to develop health problems than less reactive people?" (e.g., Piazza et al., 2013). This conventional approach, however, does not readily accommodate examination of within-person variation in reactivity across time, which is essential for answering *when* questions such as "are people more likely to have a bad night's sleep following days on which they exhibited a high degree of reactivity compared to days on which their reactivity was low?"

Our strategy for answering *when* questions like the one above is as follows. First, we will calculate, for each person's time series, changes in NA and PC that reflect each of the RRP operationalizations. Thus, it is possible for an individual to have a different recovery score, for example, on each day of the study, or even multiple recovery scores on a given day (for EMA designs). Second, these RRP quantities will serve as time-varying predictor variables for relating to health behaviors, which will serve as the outcome variables for these analyses. For example, we will examine if an indicator of reactivity (e.g., a large increase in NA subsequent to a reported stressor in everyday life) is predictive of poorer sleep that night. We will use MLM to estimate these effects and explore interactions among the various RRP indices (although there are technical challenges, given the non-independence of the indices). Our overarching hypothesis is that within-person (across time) variability RRP targets will predict subsequent sleep and physical activity.

There are several non-trivial challenges to this approach. First, we will need to address the fact that the RRP targets may (and likely will) relate to health behaviors across different timescales. For example, overnight sleep varies across a daily timescale whereas physical activity levels can vary widely both within and across days. This creates some complexities regarding how to aggregate across multiple within-day measurements for predicting overnight sleep, for example. We can address this challenge by, for example, exploring multiple approaches to aggregations, such as using the average, the peak or the last reactivity score of the day for predicting overnight sleep.

Second, because we are essentially using various types of change scores in NA and PC as predictors, reliability becomes an especially important limiting factor. Indeed, the viability of this approach will depend upon results from Stage 1 analysis that examine the within-person reliability of both stress response indicators. That is, some of our RRP targets reflect a change score in NA or PC from one assessment to the next—if such changes cannot be reliability measured, then neither can we produce reliable estimates of RRPs. We will use results from Stage 1 to optimize reliability of NA and PC, and use estimates of within-person reliabilities for each indicator to assist in interpretation of results from the different studies (i.e., we would give more emphasis to results from studies that more reliably measure NA and PC).

A third challenge is the "curse of multiplicity" that will arise from examining multiple operational definitions for each indicator of RRP across the two indicators. This will lead to a large number of analyses, thus raising concerns about type I error and generalizability. To minimize the possibility of capitalizing on a spurious result, we will conduct identical analyses across multiple data sets to directly evaluate the reproducibility and generalizability of any observed associations. That is, we will identify reliable patterns of associations that exist across multiple data sets, rather than take as valid any single result from a specific study.

6. Applying a stress response assay to just-in-time intervention innovations

Once we have completed our analytic goals, we will have developed an efficient and optimized assay (i.e., measurement tool) for the precise, within-person assessment of everyday stress. Such an assay would hold great potential for the development of highly tailored and effective interventions. There is a growing awareness of the need for better approaches to personalized/precision

medicine (e.g., Lutz et al., 2010). In addition to the overarching focus on tailoring treatment based on biological features (e.g., genetics), there is a need for methods that tailor treatment to phenotypic aspects (e.g., psychological, behavioral, social) of an individual; this work provides one such innovative approach to achieving this goal. Moreover, precision medicine can be thought of as going beyond tailoring treatment to stable attributes – but to include tailoring in a dynamic sense and in context; getting the right treatment to the right person at the right time. We propose to develop and test – using the data derived from the early activities and guided by the SOBC experimental medicine methods – an ambulatory stress assay that will provide the conceptual and practical basis for such an approach. In the short-term, we see this as a necessary step towards the development and dissemination of JIT interventions based on self-reported indicators of stress responses (e.g., Smyth & Heron, 2016); over time, such data will be useful in the development of additional, more sophisticated, approaches to adaptive intervention design and administration.

Our basic premise is that intervention can leverage the time-varying nature of the optimized ambulatory stress assay to implement JIT intervention components in daily life (e.g., delivered at a pre-specified moment in which treatment is tailored to a person's specific need in the moment; Chueh & Barnett, 1997; Smyth & Heron, 2016). The purpose of this aspect of our work is to show 'proof of concept' for such interventions. Specifically, that we can intervene on the stress response targets identified in our initial stages as described above (Phase 1), and refined in a subsequent assay development phase, and demonstrate that they are malleable and impact physical activity and sleep behaviors. Leveraging the unique strengths of our ambulatory assay, we will develop and implement a JIT intervention.

Our intervention considers both the stress response components (RRP) and their indicators (NA, PCs). We use as a starting point that each of the stress response targets (i.e., across all RRP components and indicators) equally matter for predicting physical activity and sleep behaviors. Yet we recognize that results from Phase 1 may suggest a more refined set of components and/or indicators for physical activity and/or sleep behaviors (e.g., strongest predictors, differential relationships to outcomes, particularly "toxic" combinations of predictors, time-varying contexts that moderate relationships, etc.). As this information emerges from Phase 1, we will adapt our approach to developing the intervention accordingly, although the overall structure and approach will be consistent. Specifically, we aim to develop and test a JIT intervention to demonstrate a proof of concept causal chain; namely, that the stress targets are malleable in response to JIT intervention and are related to improvements in health behaviors.

In brief, the conceptual logic of our JIT approach is to match an intervention component to the risk moment, using the RRP component identified as negatively impacting health behavior. For example, if our initial work identifies high reactivity in PC as a predictor of poor sleep (e.g., longer sleep latency), we would identify intervention components that should (based on empirical evidence, clinical judgment, and/or theory) be effective at ameliorating that specific risk component (in this example, a high PC reaction). These intervention components could be tailored to each unique risk target (i.e., each RRP as indexed by NA and PC), further tailored by the health behavior outcome desired to enhance (e.g., reducing sedentary time versus enhancing sleep quality), and even adjusted based on individual difference factors. There are a wide range of intervention components that can be adapted to form effective "micro-interventions" for use in our JIT intervention. These include - but are certainly not limited to - forms of relaxation training (breathing, muscle relaxation, imagery), meditative exercises, simple cognitive approaches (e.g., reframing, reappraisal), promoting positive states/pleasant activities, and self-regulatory approaches (e.g., goal setting, action planning), among many others.

Of course, we appreciate the many challenges of such (or related) intervention approaches; for example, determining the optimal level of each stress response target for triggering an intervention, specifying the precise content of intervention elements to be administered (and how they may be matched to eliciting target, current context, person factors, or emergent combinations thereof), adaptation of the intervention over time, and so forth.

7. Summary and conclusions

The SOBC network applies the experimental medicine approach to behavior change by identifying targets for intervention to produce healthy behaviors. The overarching goal of our project addresses this aim by developing an efficient, ecologically valid, within-person approach to measuring and intervening on the deleterious effects of everyday stress on meeting medically recommended levels of two health behaviors: physical activity and sleep patterns. The conceptual and analytic approach described in this paper will make it possible to characterize the dynamics of how people respond to stressors, which can elucidate *specific* features of stress responses (i.e., reactivity, recovery and pile-up) that exert the most potent proximal influences of health behaviors. We argue that such responses shape health behavior decisions and enactment "in the moment." To this end we are developing, refining, and validating a stress response assay (through coordinated analyses across multiple independent studies) that assesses these malleable components of the stress responses that potentially drive health behavior decisions and enactment as they unfold, in real-time and in individuals' natural environments. By measuring these specific components of the everyday stress responses as they unfold in real-time, we hope to identify targets for innovative ambulatory interventions designed to engage these targets, with the goal of breaking (or attenuating) the link between everyday stress responses and unhealthy behaviors in daily life.

The approach outlined above describes our initial approach toward elucidating the within-person structure of RRP on our selected stress response indicators (NA and PCs) in daily life. In addition, we plan to apply a similar approach to investigate the temporal patterning of our focal health behaviors—physical activity and sleep. Connecting these two lines of analyses will provide useful information for devising our stress assay. A major strength of our approach is that it will permit identification of "moments-of-risk", which are high-stress situations that threaten an individual's capacity for making healthy choices (e.g., watching TV vs. being active) or that put them in a psychological state (e.g., evening rumination) that disrupts their sleep. Our assay will provide information about *when* stress interventions will be most effective for mitigating the deleterious and immediate effects of daily stress. We emphasized the utility of this approach to support just-in-time interventions, but our assay will also allow/permit characterization of risk profiles for individuals that could identify *who* is most affected by daily stress and the type of intervention that might be most effective for them. Thus, our assay will provide targets for real-time interventions in people's daily lives (at either the within-day/moment or day level; e.g., ecological momentary interventions), and also precision interventions - such as training at-risk individuals to cope with specific response components of stressful situations that place them at risk (e.g., high reactivity, slow recovery, frequent pile-up).

Funding

This study was supported by the National Institutes of Health

Science of Behavior Change Common Fund Program through an award administered by the National Institutes of Aging (UH2-AG052167). Additional information on this project, including ongoing updates on results and technical details, can be found at the project page hosted on the Open Science Framework (https://osf.io/njpbj/) as they become available.

References

Åkerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L., & Kecklund, G. (2002). Sleep disturbances, work stress and work hours: A cross-sectional study. *Journal of Psychosomatic Research, 53*(3), 741–748. https://doi.org/10.1016/S0022-3999(02)00333-1.

Almeida, D. M. (2005). Resilience and vulnerability to daily stressors assessed via diary methods. *Current Directions in Psychological Science, 14*(2), 64–68. https://doi.org/10.1111/j.0963-7214.2005.00336.x.

Almeida, D. M., Piazza, J. R., Stawski, R. S., & Klein, L. C. (2010). The speedometer of life: Stress, health and aging. In K. W. Schaie, & S. L. Willis (Eds.), *Handbook of the psychology of aging* (pp. 191–206). Amsterdam; Boston: Academic Press.

Anton, S., & Miller, P. (2005). Do negative emotions predict alcohol consumption, saturated fat intake, and physical activity in older adults? *Behavior Modification, 29*(4), 677–688. PMID: 15911688.

Aspinwall, L. G., & Taylor, S. E. (1997). A stitch in time: Self-regulation and proactive coping. *Psychological Bulletin, 121*(3), 417–436. https://doi.org/10.1037//0033-2909.121.3.417.

Barnes, D. E., & Yaffe, K. (2011). The projected effect of risk factor reduction on Alzheimer's disease prevalence. *Lancet Neurology, 10*(9), 819–828. https://doi.org/10.1016/S1474-4422(11)70072-2.

Bergeman, C. S., & Deboeck, P. R. (2014). Trait stress resistance and dynamic stress dissipation on health and well-being: The reservoir model. For a special issue of research in human development: The promise and challenges of integrating multiple time scales in adult developmental inquiry. Denis Gerstorf, Christiane Hoppmann, and Nilam Ram (Eds.). *Research in Human Development, 11*(2), 108–125.

Bose, M., Oliván, B., & Laferrère, B. (2009). Stress and obesity: The role of the hypothalamic–pituitary–adrenal axis in metabolic disease. *Current Opinion in Endocrinology Diabetes and Obesity, 16*(5), 340–346. https://doi.org/10.1097/MED.0b013e32832fa137.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

Brummett, B., Babyak, M., Siegler, I. C., Vitaliano, P. P., Ballard, E. L., Gwyther, L. P., et al. (2006). Associations among perceptions of social support, negative affect, and quality of sleep in caregivers and noncaregivers. *Health Psychology, 25*(2), 220–225. PMID: 16569114.

Buman, M. P., Winkler, E. A. H., Kurka, J. M., Hekler, E. B., Baldwin, C. M., Owen, N., et al. (2014). Reallocating time to sleep, sedentary behaviors, or active behaviors: Associations with cardiovascular disease risk biomarkers, NHANES 2005-2006. *American Journal of Epidemiology, 179*(3), 323–334. https://doi.org/10.1093/aje/kwt292.

Buxton, O. M., & Marcelli, E. (2010). Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Social Science & Medicine, 71*(5), 1027–1036.

Buysse, D. J. (2014). Sleep health: Can we define it? Does it matter? *Sleep, 37*(1), 9–17. http://doi.org/10.5665/sleep.3298.

Centers for Disease Control and Prevention. (2008). Prevalence of self-reported physically active adults–United States, 2007. *MMWR Morbidity and Mortality Weekly Report, 57*(48), 1297–1300.

Charles, S. T., & Almeida, D. M. (2006). Daily reports of symptoms and negative affect: Not all symptoms are the same. *Psychology and Health, 21*(1), 1–17. https://doi.org/10.1080/14768320500129239.

Charles, S. T., Piazza, J. R., Sliwinski, M., Mogle, J., & Almeida, D. M. (2013). The wear-and-tear of daily stressors on mental health. *Psychological Science, 24*(5), 733–741. https://doi.org/10.1177/0956797612462222.

Chueh, H., & Barnett, G. O. (1997). "Just-in-time" clinical information. *Academic Medicine, 72*(6), 512–517. https://doi.org/10.1097/00001888-199706000-00016.

Clancy, F., Prestwich, A., Caperon, L., & O'Connor, D. (2016). Perseverative cognition and health behaviors: A systematic review and meta-analysis. *Frontiers in Human NeuroScience, 10*, 1–12. https://doi.org/10.3389/fnhum.2016.00534.

Farmer, M. E., Locke, B. Z., Mościcki, E. K., Dannenberg, A. L., Larson, D. B., & Radloff, L. S. (1998). Physical activity and depressive symptoms: The NHANES I epidemiologic follow-up study. *American Journal of Epidemiology, 128*(6), 1340–1351. PMID: 3264110.

Heron, K. E., & Smyth, J. M. (2010). Ecological momentary interventions: Incorporating mobile technology into psychosocial and health behaviour treatments. *British Journal of Health Psychology, 15*(Pt 1), 1–39. https://doi.org/10.1348/135910709X466063.

Jerstad, S. J., Boutelle, K. N., Ness, K. K., & Stice, E. (2010). Prospective reciprocal relations between physical activity and depression in female adolescents. *Journal of Consulting and Clinical Psychology, 78*(2), 268–272. PMCID: PMC2847789.

Johansson, L., Guo, X., Waern, M., Ostling, S., Gustafson, D., Bengtsson, C., et al. (2010). Midlife psychological stress and risk of dementia: A 35-year longitudinal population study. *Brain, 133*(Pt 8), 2217–2224. https://doi.org/10.1093/brain/awq116.

Jones, F., O'Connor, D. B., Conner, M., McMillan, B., & Ferguson, E. (2007). Impact of daily mood, work hours, and iso-strain variables on self-reported health behaviors. *Journal of Applied Psychology, 92*(6), 1731–1740. https://doi.org/10.1037/0021-9010.92.6.1731.

Kalmbach, D. A., Pillai, V., Roth, T., & Drake, C. L. (2014). The interplay between daily affect and sleep: A 2-week study of young women. *Journal of Sleep Research, 23*(6), 636–645. https://doi.org/10.1111/jsr.12190.

Kim, E. J., & Dimsdale, J. E. (2007). The effect of psychosocial stress on sleep: A review of polysomnographic evidence. *Behavioral Sleep Medicine, 5*(4), 256–278. https://doi.org/10.1080/15402000701557383.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Spring Publishing Company.

Lutz, R. S., Stults-Kolehmainen, M. A., & Bartholomew, J. B. (2010). Exercise caution when stressed: Stages of change and the stress–exercise participation relationship. *Psychology of Sport and Exercise, 11*(6), 560–567. https://doi.org/10.1016/j.psychsport.2010.06.005.

Luyster, F. S., Strollo, P. J., Zee, P. C., & Walsh, J. K. (2012). Sleep: A health imperative. *Sleep, 35*(6), 727–734. https://doi.org/10.5665/sleep.1846.

Matthews, C. E., George, S. M., Moore, S. C., Bowles, H. R., Blair, A., Park, Y., et al. (2012). Amount of time spent in sedentary behaviors and cause-specific mortality in US adults. *American Journal of Clinical Nutrition, 95*(2), 437–445. https://doi.org/10.3945/ajcn.111.019620.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133*(1), 25–45. https://doi.org/10.1037/0033-2909.133.1.25.

Miller, A. C., Gordon, R. M., Daniele, R. J., & Diller, L. (1992). Stress, appraisal, and coping in mothers of disabled and nondisabled children. *Journal of Pediatric Psychology, 17*(5), 587–605. https://doi.org/10.1093/jpepsy/17.5.587.

Moberly, N. J., & Watkins, E. R. (2008). Ruminative self-focus and negative affect: An experience sampling study. *Journal of Abnormal Psychology, 117*(2), 314–323. PMCID: PMC2672047.

Mouchacca, J., Abbott, G. R., & Ball, K. (2013). Associations between psychological stress, eating, physical activity, sedentary behaviours and body weight among women: A longitudinal study. *BMC Public Health, 13*(1), 828. https://doi.org/10.1186/1471-2458-13-828.

Mroczek, D. K., Stawski, R. S., Turiano, N. A., Chan, W., Almeida, D. M., Neupert, S. D., et al. (2015). Emotional reactivity and mortality: Longitudinal findings from the VA normative aging study. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 70*, 398–406. https://doi.org/10.1093/geronb/gbt107.

Murray, G., Allen, N. B., & Trinder, J. (2002 Nov). Mood and the circadian system: Investigation of a circadian component in positive affect. *Chronobiology International, 19*(6), 1151–1169. PMID: 12511032.

National Heart, Lung, and Blood Institute. (2011). *2011 National Institutes of Health sleep disorders research plan. (DHHS Publication No. 11-7820)*. Washington, DC: U.S. Government Printing Office.

Ng, D. M., & Jeffery, R. W. (2003). Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychology, 22*(6), 638–642. https://doi.org/10.1037/0278-6133.33.6.638.

O'Connor, D. B., Conner, M., Jones, F., McMillan, B., & Ferguson, E. (2009). Exploring the benefits of conscientiousness: An investigation of the role of daily stressors and health behaviors. *Annals of Behavioral Medicine, 37*(2), 184–196. https://doi.org/10.1007/s12160-009-9087-6.

Payne, N., Jones, F., & Harris, P. R. (2010). A daily diary investigation of the impact of work stress on exercise intention realisation: Can planning overcome the disruptive influence of work? *Psychology & Health, 25*(1), 111–129. https://doi.org/10.1080/08870440903337622.

Penedo, F. J., & Dahn, J. R. (2005). Exercise and well-being: A review of mental and physical health benefits associated with physical activity. *Current Opinion in Psychiatry, 18*(2), 189–193.

Piazza, J. R., Charles, S. T., Sliwinski, M., Mogle, J., & Almeida, D. M. (2013). Affective reactivity to daily stressors and long-term risk of reporting a chronic physical health condition. *Annals of Behavioral Medicine, 45*, 110–120. https://doi.org/10.1007/s12160-012-9423-0.

Rosengren, A., Hawken, S., Ounpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., et al., INTERHEART investigators. (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART study): Case-control study. *Lancet, 364*(9438), 953–962. https://doi.org/10.1016/S0140-6736(04)17019-0.

Rutledge, T., Stucky, E., Dollarhide, A., Shively, M., Jain, S., Wolfson, T., et al. (2009). A real-time assessment of work stress in physicians and nurses. *Health Psychology, 28*(2), 194–200. https://doi.org/10.1037/a0013145.

Schilling, O. K., & Diehl, M. (2014). Reactivity to stressor pile-up in adulthood: Effects on daily negative and positive affect. *Psychology and Aging, 29*(1), 72–83. https://doi.org/10.1037/a0035500.

Scott, S. B., Ram, N., Smyth, J. S., Almeida, D. M., & Sliwinski, M. J. (2017). Age differences in negative emotional responses to daily stressors depend on time since event. *Developmental Psychology, 53*, 177–190. PMC5198895.

Scott, S. B., Sliwinski, M. J., & Blanchard-Fields, F. (2013). Age differences in emotional responses to daily stress: The role of timing, severity, and global perceived stress. *Psychology and Aging, 28*(4), 1076–1087. https://doi.org/10.1037/a0034000.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601–630. https://doi.org/10.1037/0033-2909.130.4.601.

Seigel, K., Broman, J. E., & Hetta, J. (2002). Behavioral activation or inhibition during emotional stress-implications for exercise habits and emotional problems among young females. *Nordic Journal of Psychiatry, 56*(6), 441–446. https://doi.org/10.1080/08039480260389361.

Simkin, L. R., & Gross, A. M. (1994). Assessment of coping with high-risk situations for exercise relapse among healthy women. *Health Psychology, 13*(3), 274–277. https://doi.org/10.1037/0278-6133.13.3.274.

Sliwinski, M. J., Almeida, D. M., Smyth, J., & Stawski, R. S. (2009). Intraindividual change and variability in daily stress processes: Findings from two measurement-burst diary studies. *Psychology and Aging, 24*(4), 828–840. https://doi.org/10.1037/a0017925.

Sliwinski, M. J., & Scott, S. B. (2014). Physical and environmental constraints on emotional well-being in older adults. In P. Verhaeghen, & C. Hertzog (Eds.), *The Oxford handbook of emotion, social cognition, and everyday problem-solving during adulthood* (pp. 128–141). New York: Oxford University Press.

Smyth, J., & Heron, K. (2016). Is providing mobile interventions "just-in-time" helpful? An experimental proof of concept study of just-in-time intervention for stress management. *Proceedings of the IEEE Wireless Health (WH) Conference*, 89–95. https://doi.org/10.1109/WH.2016.7764561.

Smyth, J., Ockenfels, M., Porter, L., Kirschbaum, C., Hellhammer, D., & Stone, A. (1998). Stressors and mood measured on a momentary basis are associated with salivary cortisol secretion. *Psychoneuroendocrinology, 23*, 353–370.

Smyth, J., Zawadzki, M., & Gerin, W. (2013). Stress and disease: A structural and functional analysis. *Social and Personality Psychology Compass, 7*(4), 217–227. https://doi.org/10.1111/spc3.12020.

Sonnentag, S., & Jelden, S. (2009). Job stressors and the pursuit of sport activities: A day-level perspective. *Journal of Occupational Health Psychology, 14*(2), 165–181. https://doi.org/10.1037/a0014953.

Spira, A. P., Chen-Edinboro, L. P., Wu, M. N., & Yaffe, K. (2014). Impact of sleep on the risk of cognitive decline and dementia. *Current Opinion in Psychiatry, 27*, 478–483. https://doi.org/10.1097/YCO.0000000000000106.

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher, & J. Reason (Eds.), *Handbook of life stress, cognition and health* (pp. 629–649). New York: John Wiley & Sons.

Stone, A. A., Smyth, J. M., Pickering, T., & Schwartz, J. (1996). Daily mood variability: Form of diurnal patterns and determinants of diurnal patterns. *Journal of Applied Social Psychology, 26*(14), 1286–1305.

Stucky, E. R., Dresselhaus, T. R., Dollarhide, A., Shively, M., Maynard, G., Jain, S., et al. (2009). Intern to attending: Assessing stress among physicians. *Academic Medicine, 84*(2), 251–257.

Stults-Kolehmainen, M. A., & Sinha, R. (2014). The effects of stress on physical activity and exercise. *Sports Medicine, 44*(1), 81–121. https://doi.org/10.1007/s40279-013-0090-5.

Troiano, R. P., Berrigan, D., Dodd, K. W., Mâsse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. *Medicine and Science in Sports and Exercise, 40*(1), 181–188. https://doi.org/10.1249/mss.0b013e31815a51b3.

Warren, T. Y., Barry, V., Hooker, S. P., Sui, X., Church, T. S., & Blair, S. N. (2010). Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise, 42*(5), 879–885. https://doi.org/10.1249/MSS.0b013e3181c3aa7e.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063–1070. PMID: 3397865.

Zautra, A. J. (2005). *Emotions, stress, and health*. New York: Oxford University Press.