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The Measurement of Stress

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1. A Brief History of Stress Research

Stress research was first formally established in the early- to mid-1900's, when physiologist Walter Cannon and endocrinologist Hans Selye independently began investigating stress physiology. Cannon described the typical range of physiological functioning, or set-points, as homeostasis and discovered that the body's 'fight-or-flight' system, supported by adrenaline (i.e., epinephrine), responds to a variety of threats to return the organism to homeostasis. Selye's work extended this knowledge by highlighting the role of the hypothalamic-pituitary-adrenocortical (HPA) axis as a critical mediator of the "General Adaptation Syndrome" of alarm, resistance, and exhaustion, explaining how the body develops new set-points under high demands. Today, the sympathetic-adrenal-medullary (SAM) and HPA axes are well outlined as the two major biologically-mediated stress pathways (e.g., Kemeny, 2003).

As stress research developed, investigators using lab-based acute stress paradigms identified the cardiovascular system as being intrinsically linked to stress pathways via the production of hormones. Because of its non-invasive measurement, assessing cardiovascular responses became a cornerstone of stress human study designs. As recognition of the interconnected relationships between the brain, stress pathways, and immune system grew (for a review, see Glaser & Kiecolt-Glaser, 2005), new subfields of study were established, such as psychoneuroendocrinology and psychoneuroimmunology. However, great variability in physiological stress responses across these systems was observed between people, suggesting that individual differences in how people experience stress contribute to physiological responses. These observations laid the foundation for the development of psychological stress theories, some of which are outlined below.

According to the Transactional Model of Stress and Coping, the degree to which a potential stressor results in a stress response is largely dependent upon whether the event is subjectively perceived, or appraised, as a threat or challenge (i.e., primary appraisal), as well as the ability to cope with the stressor successfully (i.e., secondary appraisal; Lazarus & Folkman, 1984). This cognitive appraisal process is described as iterative, with outcomes influenced both by action- and emotion-focused coping. The Conservation of Resources Theory (Hobfoll, 1989) describes stressors more specifically by whether actual or potential resources were lost or not gained after investment. Other theories further highlight the importance of protective resources in coping with stress. For example, the Stress Buffering Hypothesis (Cohen & Wills, 1985) posits that the amount and quality of one's perceived social support can buffer against the negative effects of stress. In the workplace context, the Demands-Resources Model (Bakker & Demerouti, 2007) describes how balance between demands and resources relate to strain and motivation, with personal resources like self-efficacy and optimism being key mediators in reducing job stress. Please other Stress section entries for more information psychological stress processes such as Stress and Reactions and Coping with Stress.

Although psychological stress theories have been invaluable to the field, the primary role of the stress responses to aid survival (i.e., physiological adaptation), became obscured. With the aim of re-rooting the concept of stress in the founding work of Cannon and Selye, the term 'allostasis' was coined to describe the variable and temporary adaptive physiological response to threat or challenge that supports return to baseline. The concept of allostasis incorporated psychological stress theories by embracing the new insight that the physiological stress response can be altered due to differences in perception and regulation. When allostatic processes are engaged too persistently over time, however, cumulative effects are theorized to lead to physiological dysregulation or allostatic load (McEwen, 2004). Although the complete aetiology

and when a well-regulated body shifts to dysregulated physiology remains unknown, non-communicable chronic health conditions appear to be the clinical manifestations of the body's attempt to adapt to its environment. Today, the field of stress research benefits from the complex and broad history that is essential to addressing stress-related issues and disorders using inter- and transdisciplinary approaches.

2. Defining Stress

To appropriately examine stress, it is imperative to first consider which type of stress we intend to measure. Stress methodology has historically encountered great variation in the definition and measurement of stress (Monroe, 2008). A lack of consensus on what stress is limits conclusions that can be drawn from research examining stress. Specificity in definitions, however, aids in the dissemination and translation of research findings both within and across scientific disciplines. A precise definition will guide the choice of an appropriate measure for the construct of interest, strengthen interpretation of results, and enhance the ability to aggregate findings, which depends on consistent, specific, and standardized terminology. In the absence of specific and consistent terminology, stress researchers are vulnerable to using inappropriate or unvalidated measures, as well as misinterpreting findings. As such, the field has begun to recognize the importance of defining and using terminology in stress research to clearly articulate the construct(s) measured (e.g., Cohen et al., 2019).

2.1 Terminology

Stress as a stand-alone word refers to the complex, interactive, and multilevel process of adaptation to the environment (Epel et al., 2018). When conceptualizing a stress-related construct or choosing a measure, the most imperative distinction is between the stressor and the response to the stressor (i.e., the stress response). A stressor, or stress exposure, refers to the event, situation, or environmental stimulus that elicits a psychological, biological, or behavioural response. The stress response entails a person's subjective appraisal of the stressor, as well as the psychological, behavioural, and physiological processes that occur as a result.

2.1.1 Stressors

Stressors can first be categorized according to temporal components; acute stressors are time-limited or episodic, with a relatively short and identifiable duration (e.g., minutes to hours). Conversely, chronic stressors (also called chronic difficulties or adversity) are long-term stressors that continually persist or recur frequently for six months or longer (Epel et al., 2018).

Acute stressors can be further identified by severity. Relatively minor acute stressors may include typical daily hassles, such as getting caught in traffic, or more intense, but less frequent, short-term exposures (e.g., public speaking, final exams). Severe acute stressors include exposure to life events that are substantially demanding or threatening and may contribute to a major disruption or upheaval. These events can be either negative experiences like a job loss, or positive, yet demanding, like a promotion. Life events that are particularly severe include traumatic events, which involve the added threat to physical and/or psychological safety.

Importantly, exposure to one stressor may increase the likelihood of more stressors occurring (Cohen et al., 2019), referred to as secondary stressors. For example, an acute, primary stressor like a car accident can initiate a cascade of additional, secondary stressors, such as being fired from work for missing a shift and ensuing financial hardship.

2.1.2 Stress Responses

When a potential stressor is perceived as challenging, threatening, and/or harmful, a stress response is mounted. This response includes the activation of a complex, multifaceted system response including cognitive, emotional, biological, and behavioural changes to aid the individual in meeting perceived current demands (Kemeny, 2003; McEwen, 1998). The temporal length of the response (e.g., minutes to years) and extent to which it negatively affects health largely depend on the type and severity of the stressor.

Short-term alterations in functioning (e.g., minutes to days) are referred to as acute stress responses. For example, acute biological stress responses include changes in the autonomic, neuroendocrine, metabolic, and immune systems that drive confrontation or avoidance of the stressor. Reactivity is an additional term in the subfield of stress physiology, which includes the entire process of short-term adaption, including response and recovery. A distinct characteristic of acute stress responses is that they have a baseline, an identifiable peak, and recovery (Epel et al., 2018).

Chronic stressors can result in long-term psychological, physiological, and behavioural responses referred to as chronic stress responses. Chronic heightened activation of the body's stress response systems increases allostatic load, or 'wear and tear' on the body. The resulting long-term changes in emotions, behaviours, cognitions and cognitive functioning, as well as physiological functioning, may then manifest as multi-system dysregulation (McEwen, 2004). The type of resulting systemic effects, as well as where, how, and when they occur, appear to depend on the nature of the stress responses and for how long stress systems are chronically activated.

Although acute and chronic stressors and stress responses are presented as conceptually distinct, acute stressors can initiate chronic stress responses even in the absence of an identifiable chronic stressor, in which the effects of the initiating stressor persist after the stressor is resolved. For example, this may be the case when individuals are exposed to traumatic events and develop post-traumatic stress disorder (PTSD).

3. Stress Measurement

3.1 Measuring Stressors Retrospectively

When measuring exposure to acute and chronic stressors, life event checklists are commonly used because they are typically simple, inexpensive, and easy to administer (Slavich, 2016). Checklists involve endorsing specific life experiences and may be specific to stressor classification. For example, some checklists focus on acute stressors, like major stressful life events, or may only assess potentially traumatic events. Other checklists focus on exposure to chronic stressors, and some combine acute life events and chronic experiences to capture a range of stressful life experiences. Checklists may ask participants to identify whether they have experienced a stressor at any point in their lifetime, over a specified amount of time (e.g., the past six or twelve months), or during specific developmental periods, such as childhood; for a review of validated tools for measuring childhood adversity, see Oh et al., 2018.

Despite the advantages of checklists, assumptions can influence whether and how the total number of stressful life events predict outcomes of interest. Many checklists are sum scored, assuming equal valence and severity across events. However, research has shown that all stressors are not equally impactful. For example, interpersonal stressors contribute to greater distress and negative outcomes than non-interpersonal events (Cohen et al., 2019). Hence,

additional features of some checklists that may be beneficial to include are the use of life domain categories and collecting data regarding duration of the stressor as well as perceived valence and severity.

A final limitation of stressor checklists is recall bias. People can reliably recall major life events, especially within the past 10 years, however events perceived as less severe are not reliably recalled with checklists (Monroe, 2008). Because of the superior reliability and ability to provide rich data, semi-structured interview-based systems conducted by a trained interviewer may be more useful. However, administering interviews and analysing the resulting qualitative data requires significantly more time, effort, and likely more financial investment than checklists. As such, the investigator should weigh the advantages and disadvantages of each method when choosing a retrospective stressor exposure measure.

3.2 Measuring Stressors in Real Time

The induction of an acute stressor in the lab allows for relatively easy and thorough examination of multifaceted stress responses in a controlled environment. In-lab stressors can be standardized (e.g., Trier Social Stress Test, cold pressor test, cognitive tasks) or quasi-naturalistic (e.g., resolving a disagreement, discriminatory or social rejection manipulations). However, these acute lab stressors vary in their ability to activate features of the stress response; thus, investigators must consider their outcome of interest when choosing a lab-based stressor. Furthermore, while the wealth of stress response data collected in the lab can be extensive, the generalizability to real-world contexts may be limited due to the artificial setting and lack of access to external resources that are typically available to an individual.

The measurement of stressors in natural environments offers greater ecological validity and recall reliability than typical life event checklists or lab-based manipulations. Ecological momentary assessment (EMA), also known as experience sampling or diary methods, involves the repeated sampling of participants' experiences and behaviours in their natural environment. EMA can assess stressor exposure and responses as they occur or within close temporal proximity to the exposure (e.g., within minutes to hours), ensuring reasonably accurate recall and potentially greater generalizability to other real-world experiences. EMA studies commonly utilize smartphone apps or online surveys to target recent time periods (e.g., past 24 hours) or pre-defined experiences (e.g., argument with spouse), but may also include the continuous measurement of physiological data via wearable technologies (e.g., heart rate monitor, actigraphy; Shiffman et al., 2008). Although EMA has many advantages, investigators must consider the potential for high participant burden, access to necessary resources (e.g., time and personnel investment, adequate funding for compensation and equipment), and suitability of the technology. EMA investigators must also be strategic about which 'moments' to collect data (i.e., random intervals, fixed intervals, event-based), depending on the research question and expectations of construct variability across time and context.

3.3. Measuring Psychological and Behavioural Stress Responses

3.3.1 General Stress Perception Ratings

Known by the popular notion of 'feeling stressed', global perceptions of stress experiences are typically assessed by self-report measures. These surveys often capture the individual's general perceptions of unpredictability and uncontrollability within the past week or month. Other measures may assess perceptions of chronic stress in specific domains (e.g., work overload, social isolation) over longer periods of time.

Notably, these scales assume that respondents are aware of and able to report their degree of experienced distress. Self-reported measures have inherent limitations, including vulnerabilities to social desirability biases and relative comparisons as well as dependence on personality traits, current affective states, and cultural factors that influence an individual's awareness of and willingness to report perceived stress. These limitations can all potentially contribute to weaker relationships between perceived stress and expected outcomes of interest (Epel et al., 2018).

3.3.2 Context-Specific Stress Perception Ratings

Beyond general stress perceptions, responses to more specific stimuli or events are assessed across a wide variety of measures. For instance, clinical measures of PTSD symptoms in scales may capture emotional, cognitive, and behavioural reactions experienced within the past month regarding a certain potentially traumatic acute stressor. Context-specific chronic stress perceptions can generally be categorized by a role (e.g., caregiver), aspect of identity (e.g., discrimination), or life situation (e.g., socioeconomic and work circumstances). These perceptions may be considered as predictors as well as contextual moderators that alter stress responses and stress-related health outcomes.

3.3.3 Specific Psychological and Behavioural Processes

Psychological and behavioural responses to stressors are typically measured via self-report measures that assess a wide range of responses, which ultimately form an iterative and integrative process.[1] Using surveys, cognitive, emotional, and behavioural responses can be general (e.g., 'how do you typically respond?'), context-specific (e.g., within a romantic relationship), or stimulus-specific (e.g., in response to an event). Cognitive responses to stressors include the appraisal of a situation as a threat or challenge and perceived ability to control or cope with the situation, as well as perseverative cognitions (e.g., rumination). Affective responses may be measured by ratings of specific emotions (e.g., anger, sadness), positive and negative affect, and self-reported or behaviourally-coded motivational states, such as approach and avoidance. Additionally, a variety of questionnaires measure efforts to manage stress responses through behavioural coping (e.g., smoking, overeating, seeking support) or emotional coping and regulation strategies (e.g., cognitive reappraisal, acceptance). Please other Stress section entries for more information psychological stress processes such as Coping with Stress and Stress Management and Interventions.

3.4. Measuring Physiological Stress Responses

Physiological assessment can provide information about acute stress reactivity as well as long-term biological alterations. The primary physiological markers of stress responses involve indicators of activation in the SAM and HPA axes. Epinephrine and norepinephrine are released when the sympathetic arm of the SAM axis activates and the parasympathetic nervous system withdraws. Because these catecholamines are quickly mobilized and metabolized, they can only be reliably assessed via current levels in the blood. The HPA axis may be initiated simultaneously, but the measurable presence of its main biomarker, the hormone cortisol, is delayed by 10-15 minutes. Because cortisol has a longer half-life in the blood and can be

[1] Of note, responses to stressors do not always occur in a linear fashion; predictions about future potential stressors can produce cognitive and emotional responses that take place before the stressor occurs, known as anticipatory stress responses.

collected non-invasively via saliva, its measurement is generally more popular than catecholamines.

Activation of the SAM and HPA pathways also influence cardiovascular functioning, which can be estimated via blood pressure and heart rate. Additionally, heart rate variability is a proxy for the sympathetic and parasympathetic nervous systems' control over the heart. As hormones from these stress systems also regulate immune functioning, indicators of systemic inflammation can be employed as stress-related biomarkers as well. The utility of each biomarker within the context of acute and chronic stress is summarized in table 1.

Healthy acute stress responses can help individuals adapt to challenges and changing circumstances and restore homeostasis; however, acute stress responses that are deemed either hyper- or hypoactive for a given situation can signal a maladaptive stress response (McEwen, 1998). Additionally, persistent and excessive exposure to catecholamines and cortisol (e.g., through chronic or recurring stress responses) can lead to long-term, pervasive effects on cardiovascular, immune, and metabolic functioning. Biomarkers mentioned above may therefore be assessed during reactivity or at rest to indicate chronic physiological functioning in these systems. When measured collectively, indicators that exceed clinical norms may represent allostatic load. Examples of both clinically relevant and research-specific methods for physiologically assessing chronic stress are outlined in table 2.

Notably, many physiological markers have diurnal rhythms and control other bodily functions beyond the stress response. Prior to utilizing any of these biomarkers, it is therefore critical to gain an understanding of the biomarker's primary and secondary roles and be aware of methodological considerations when designing the study and drawing conclusions from the data. For example, a single sample of salivary cortisol to assess an acute or chronic stress response is relatively meaningless due to the strong diurnal rhythm driven by awakening time and other behavioural influences, such as exercise and food and beverage consumption. Thus, repeated sampling of salivary cortisol within consistent and controlled conditions is imperative.

4. Contextual Influences

An accurate understanding of an individual's experiences is impossible without considering the various contexts that shape that individual's stressor exposure, perceptions, and stress reactivity. The social ecological model[2] (Bronfenbrenner, 1977) explains development at multiple levels of environmental influence (i.e., individual, interpersonal, organizational, community, and public policy), and can be informative for understanding contextual influences in stress processes.

The individual level, which includes an individual's personal experiences, beliefs, personality, genetics, etc., is likely the most familiar to psychological researchers. At the interpersonal level, the size and quality of one's close social network, such as family, friends, and romantic relationships, can alter the likelihood of exposure to certain stressors as well as the perception and ability to cope with stress in adaptive and maladaptive ways. The organizations the individual is a part of, including workplaces, schools, and healthcare organizations, may additionally contribute to stressor exposure and response. For example, jobs may be dangerous,

[2] The social ecological model later evolved into the Bioecological model (Bronfenbrenner & Ceci, 1994), which included changes in contextual categorization, terminology, the addition of time, and greater focus on proximal processes. Here we have chosen to present the simpler model as an example of levels involved in contextual influences for the sake of clarity and brevity; however, we recommend conferring with the Bioecological model for the more complex and mature theoretical approach.

labour-intensive, or require conflict management. Furthermore, access to adequate healthcare and mental health resources as well as workplace policies surrounding sick leave and disability resources may affect the ability to cope with stressors. The community level entails the broader built environment, cultural values, and norms. Examples at the community level include crime rates, availability of safe public transportation, and societal norms surrounding substance abuse. Lastly, public policy may influence objective and subjective experiences of stress through its impact on laws and resources available at the local, state, and national levels, as well as public opinion and culture more broadly.

Importantly, all levels are interconnected and dynamically influence each other. For example, consequences of low minimum wage requirements include limited social mobility and increased poverty. These community-level stressors can then increase an individual's stress perception and risk of stressor exposure through financial strain and systemic discrimination and racism. From ecological and complexity sciences frameworks, these relationships are not merely additive but non-linear and emergent, spanning across multiple levels to inform a larger whole. Although it is not feasible to assess every possible predictor or moderator in stress research, it is necessary to consider the various contexts that influence outcomes at levels beyond the individual.

5. Applied Implications of Stress Measurement

The appropriate assessment of stressors, stress responses, and related outcomes can hold broad, important implications outside of basic research. For example, exposure to chronic stressors, as well as those that are acute but severe, can precipitate and exacerbate chronic mental and physical illnesses, particularly when coupled with maladaptive stress responses and unhealthy coping efforts (Alessi & Bennett, 2020; Cohen et al., 2019). Beyond individual outcomes, long-term effects include negative social outcomes (e.g., worsened family functioning, criminality, job loss) as well as significant economic costs, such as declines in productivity, higher rates of disability, and healthcare spending. Stress measurement can aid in mitigating these negative impacts by identifying at-risk individuals, assessing changes due to interventions, and promoting positive organizational and public policy changes across everyday settings. For example, settings like healthcare, the workplace, and schools provide an opportunity to reduce significant sources of stress, improve access to resources, and foster adaptive coping when stressors arise. Notably, such policy changes necessitate political and organizational investment in patient, employee, and student well-being to achieve long-term economic savings in the face of perceived short-term financial barriers.

For instance, currently only integrative healthcare systems and practices broadly examine stress-related risk factors as part of screening for exposure and holistic treatment of health conditions. Although screening for psychological distress, specific disorders, and important social determinants of health (e.g., food insecurity, family or partner violence) are commonly advocated for across multiple care specialties, limited financial and healthcare resources are significant barriers to the routine use of these screeners in most practices. Nonetheless, coordinated assessment of stress-related risk factors across treatment settings can serve to more efficiently detect at-risk populations and improve health-related outcomes and healthcare costs.

In the workplace, employees' stress is predictive of worker achievement, productivity, absenteeism, and presenteeism, as well as their overall health (Salvagioni et al., 2017). To

enhance work-related outcomes and employee health, organizations can utilize stress measures to identify workplace stressors and understand how workplace policies and interventions affect perceived stress related to work (e.g., burnout) as well as other life domains. In doing so, organizations can use stress measurement to foster higher productivity and reduce the risk of employee burnout and turnover.

Measuring stress in education, especially in K-12 schools, provides an opportunity to identify children most in need of additional support and resources. It is also crucial that the resources needed to reduce stressor exposure are available and accessible, making effective coordination of community and government level resources integral. Further, educational settings are in a unique position to provide early interventions to help children and adolescents develop better socioemotional and stress management skills, such as mindfulness, adaptive coping strategies, and healthy behaviours that can empower individuals and maximize resiliency.

6. Conclusion

The scientific study of stress has a broad history spanning multiple disciplines, enabling researchers to study the multifaceted effects of stressors and responses on health and human behaviour. Because of the increasingly inter- and trans-disciplinary nature of stress research, careful attention to precise and informed decision-making surrounding constructs of interest and the use of appropriate measures and methodology is integral to rigorous stress measurement and the theoretical and empirical advancement of the field. In addition, although it is not feasible to include every aspect of stress measurement in a study, the measurement of relevant contextual influences will provide greater predictive validity than a single stress measure. As stress is a complex, dynamic, and iterative process, consideration of researchers' findings within the larger whole should also be of focus as the field moves forward.

A relatively young field, stress research has already contributed greatly to our understanding of how lived experiences culminate in long-term effects on the individual, immediate networks, and society. The wide-ranging effects of stress highlight the importance of continued stress measurement to identify significant sources of stress, develop effective interventions to reduce stressor exposure, promote healthy stress responses, and efficiently redirect resources to those most in need.

For more information, see these open access resources on stress measurement:

- Stress Measurement Network, maintained by University of California, San Francisco: https://www.stressmeasurement.org/measurement-toolbox
- Healthcare Toolbox, maintained by Children's Hospital of Philadelphia: https://www.healthcaretoolbox.org/
- The PhenX Toolkit, maintained by RTI International: https://www.phenxtoolkit.org/
- Health Measures, maintained by Northwestern University: https://www.healthmeasures.net/
- NIH Public Health Emergency and Disaster Research Response: https://dr2.nlm.nih.gov/

Table 1. A summary of acute and chronic stress-related biomarkers for research

	Acute		Chronic	
	Changes	Design	Changes	Design
Neuroendocrine				
Cortisol (Salivary)	↑ during stressor; ↓ during recovery*	Multiple samples; every 10-20 minutes up to 2 hours post-baseline	Alterations in diurnal rhythm; hyper- or hypoactive	Multiple samples across each day for multiple days
Epinephrine/ Norepinephrine (Blood)	↑ during stressor; ↓ during recovery	Multiple samples via IV; every 2-5 minutes up to 45-60 minutes post-baseline	N/A	N/A
Salivary alpha amylase	↑ during stressor; ↓ during recovery	Multiple samples; every 2-5 minutes up to 45-60 minutes post-baseline	N/A	N/A
Cardiovascular				
Systolic & Diastolic Blood Pressure	↑ during stressor; ↓ during recovery	Multiple samples; every 2 minutes during stressor and 5 minutes during baseline and recovery	Elevated; clinical cut-off indicate dysregulated functioning or increased disease risk	Resting; typically 3 measures for systolic and diastolic blood pressure collected and averaged separately or combined using a formula (e.g., mean arterial pressure)
Heart Rate	↑ during stressor; ↓ during recovery	Collected continuously; multiple samples identified within each period	Elevated; clinical cut off indicating abnormal or increased disease risk	Resting; typically 3 measures collected and averaged
Heart Rate Variability	↓ during stressor; ↑during recovery	Collected continuously; multiple samples identified within each period	Decreased; no current clinical cut off	Resting; respiration collected concurrently** or paced-breathing task utilized
Immune				
Proinflammatory Cytokines	↑ during stressor; recovery typically too slow to capture in lab	Typically 3 blood samples collected: baseline, 45 min and 120 minutes post stressor	Comparatively higher; clinical cut-off not yet known	Blood, saliva, or blood spot; single sample, typically while fasting

Anti-inflammatory Cytokines	↓ during stressor; recovery typically too slow to capture in lab	Typically 3 blood samples collected: at baseline and 45 and 120 minutes post-stressor	Comparatively lower; clinical cut-off not yet known	Blood, saliva, or blood spot; single sample, typically while fasting
C-Reactive Protein	No reliable acute changes	N/A	Elevated; clinical cut-offs indicate dysregulated functioning or increased disease risk	Blood, saliva, or blood spot; single sample, typically while fasting
Glucocorticoid Sensitivity in immune cells	Limited evidence in humans following acute stress induction	Typically 2 blood samples collected: at baseline and 60 minutes post-stressor	Comparatively lower; clinical cut-off not yet known	Blood; single sample, typically while fasting

Note. IV = intravenous catheter. *Salivary cortisol provides an estimate of biologically-available cortisol in the blood as opposed to blood samples which additionally measure protein-bound cortisol. Of note, cortisol reactivity to acute stressors may not follow this pattern if the individual does not perceive the stressor during the manipulation or timing in regards to diurnal rhythm or if the hypothalamic-pituitary-adrenal (HPA) axis is non-responsive to the lab manipulation due to chronic stress. **Heart rate and its variability, especially measures of high frequency, can be influenced by respiration rate; therefore, concurrently measuring both is recommended as best practice.

Table 2. A summary of common biomarkers of chronic stress dysregulation

Biomarkers		System
Clinical (Allostatic Load)	Urinary cortisol	Neuroendocrine
	Urinary epinephrine & norepinephrine	Neuroendocrine
	Serum dehydroepiandrosterone sulfate (DHEA-S)	Neuroendocrine
	Blood pressure (systolic & diastolic)	Cardiovascular
	Cholesterol, HDL & total	Cardiovascular
	Waist-to-hip ratio	Metabolic
	Body mass index	Metabolic
	Glycated hemoglobin (HbA1c)	Metabolic
	C-reactive protein	Inflammation
Research Only	Hair cortisol	Neuroendocrine
	Telomere length & telomerase Activity	Cellular aging
	Gene expression (e.g., CTRA)	Epigenetic alterations
	Herpesvirus reactivation	Cell-mediated immunity
	Mitochondria function	Intracellular allostatic load

Note. HDL= high-density lipoproteins; CTRA = conserved transcriptional response to adversity

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