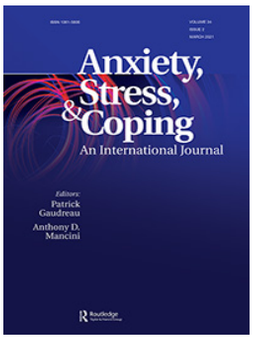

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BRIEF REPORT

Stress overload in the spread of coronavirus

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ABSTRACT

Background and Objectives: The incidence of novel coronavirus infection across the globe has been uneven, hitting some population subgroups harder than others. Media coverage has proffered explanations for this differential vulnerability, but psychosocial risk factors have been largely ignored. In contrast, multiple theories, medical and psychological, point to one psychosocial factor – stress – as important to the etiology of disease. They also agree that pathogenic stress arises from the particular circumstance in which adaptational demands overwhelm a person's resources, creating "stress overload" that deregulates normal functioning and increases susceptibility to illness. Assessment of stress overload is proposed as essential to understanding viral spread in the current pandemic.

Methods: Studies are reviewed explicating (1) stress overload theories and relevant empirical evidence, (2) construction of a stress overload measure and related validity evidence.

Results: Findings support the role of stress overload in illness and the accuracy of the measure in predicting illness.

Conclusion: It is concluded that assessment of stress overload may help to explain the observed coronavirus disparities, and to identify populations at risk for imminent infection. The 10-item Stress Overload Scale-Short is offered as a potentially useful tool for researchers and clinicians working to map, and stem, the proliferation of coronavirus.

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COVID-19; stress overload; coronavirus etiology; stress measure; SOS-S

The spread of the novel coronavirus across the globe has been uneven, revealing disparities in infection rates. This has sparked questions in the popular media about why some people appear more vulnerable than others (e.g., Netburn, 2020; Resnik, 2020; Slaughter, 2020). In attempts to answer this "infection enigma" (Resnik, 2020), a variety of possible risk factors have been suggested: Being male (Resnik, 2020), older, poor (Noppert, 2020), African American (Law, 2020), or having a chronic illness (Netburn, 2020), genetic predisposition (Resnik, 2020), or compromised immune system (Slaughter, 2020).

The possibility of psychosocial risk factors, however, has been largely ignored in the media. With diligent search, articles on coronavirus anxiety (Heisz, 2020) and stress (Cardona, 2020) can be found. These argue that such factors are not simply byproducts of pandemic warnings or comorbidities associated with infection, but actual etiological agents that deregulate normal functioning and thereby increase the risk for viral infection (Heisz, 2020).

Stress theories of illness

In fact, there is a considerable history of theories underscoring the role of stress in illness, and offering explanations for why it affects people differently. The seminal medical theory (Selye, 1956) stated that

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any adaptational demand is distressing, but most often the body rallies resources to cope with these demands and return to homeostasis. It is only when one's resources are exhausted that these demands can cause cellular damage or even death. This idea was updated (McEwen, 2000) to focus on the allostatic rather than homeostatic system, but retained the idea that excessive demand load can cause the body's response to become dysregulated and pathogenic. Psychological theories of stress emphasize perception over physiology. The seminal theory of this type (Lazarus & Folkman , 1984) stated that if demands are appraised to be within one's coping abilities, they are seen as a challenge – perhaps annoying but not destructive. It is only when demands are appraised to exceed coping resources that they assume the dimension of a threat, resulting in physical and mental dysfunction. Another theory (Hobfoll, 1989) echoes this idea, positing that the expenditure of one's resources to deal with demands is always upsetting. But when expenditures become excessive, this instigates a downward spiral of perceived loss and inadequacy (to meet future demands) that renders a person susceptible to illness.

A common theme may be discerned among these diverse theories. In the words of one prominent stress researcher, "They all share … a process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease" (Cohen et al., 1995). In short, the pathogenic form of stress arises from the particular circumstance in which demands outweigh resources. This has important implications for understanding the role of stress in the spread of the coronavirus.

First, the theories agree that not every stress experience results in illness. Some stress feelings are transitory, and dissipate when demands are met and normalcy regained. It is only the persistent state of being overwhelmed by demands that increases susceptibility to illness. The term "stress overload" was coined (Lunney, 2006) to describe this state and differentiate it from the other fleeting and benign forms of stress. Second, this in turn implies that not everyone exposed to stressors will get sick. Individuals differ in the number and intensity of demands they must face, as well as the extent and type of resources they bring to such demands. It is only that fraction of the population who are facing demands that exceed their resources who are at increased risk for illness, according to theory.

Are stress theories accurate?

Substantial evidence for a stress-illness link has accumulated, yielding a plethora of review articles (Glaser & Kiecolt-Glaser, 2009; Gouin, 2011; Guidi et al., 2020; Hänsel et al., 2010; Hussain, 2010; Juster et al., 2016; Koh, 2018; Marketon & Glaser, 2008; Pedersen et al., 2011; Reiche et al., 2005). Many of these acknowledge stress theories as foundational to this body of research (Hussain, 2010; Juster et al., 2016; Koh, 2018; Marketon & Glaser, 2008; Reiche et al., 2005), and in accordance with theory, differentiate pathogenic stress from its other forms (Gouin, 2011; Hussain, 2010; Marketon & Glaser, 2008). Although not termed stress overload, pathogenic stress is defined similarly, either as allostatic load (excessive demands that exceed coping capacity; Guidi et al., 2020) or as chronic stress (persistent demands that do not relent with coping efforts; Hänsel et al., 2010).

In regard to physical illness, this form of stress has been shown to increase susceptibility to infectious diseases (Gouin, 2011; Pedersen et al., 2011), facilitate the development and progression of cancer (Marketon & Glaser, 2008), allow the expression of latent viruses such as herpes and Epstein–Barr, and interfere with the healing of wounds (Gouin, 2011; Marketon & Glaser, 2008). A dismaying portent for the current pandemic is that it has also been found to trigger adverse reactions to vaccines (Gouin, 2011; Marketon & Glaser, 2008). In regard to psychiatric illness, such stress has been implicated in the onset of schizophrenia (Savransky et al., 2018) and major affective disorders (Cattaneo & Riva, 2016). Even this inventory is not exhaustive, for the full number of illnesses directly or indirectly related to stress is not yet known (Juster et al., 2016).

Beyond supporting stress theories, this research has augmented them in specifying the mechanisms by which stress deregulates normal functioning. The activation of the hypothalamic–pituitary–adrenal (HPA) axis produces stress hormones that block immune responses, lowering counts of T-cells, B-cells,

and NK-cells (Glaser & Kiecolt-Glaser, 2009; Hussain, 2010). In contrast, inflammatory responses become uncontrolled; the overproduction of cytokines increasing susceptibility to viral infection (Cohen et al., 2012; Hänsel et al., 2010) – and raising questions about the source of the cytokine storm seen in some coronavirus patients. Functional and structural changes to the mitochondria occur, extending even to DNA and gene expression, thereby opening multiple pathways to disease (Juster et al., 2016). Disruptions in glucocorticoid signaling not only permit the onset and progression of physical pathologies (Cohen et al., 2012), but impact the systems and genes relevant to mood disorders (Cattaneo & Riva, 2016). There are also behavioral changes, an increase in maladaptive responses to stress (such as smoking and drinking alcohol) and a decrease in adaptive ones (such as sleep and exercise), that impact health (Gouin, 2011). The mechanisms linking stress to pathology are thus multifold, and so complexly intertwined that they are still not fully understood (Hussain, 2010).

Without exception, research reviews agree that stress is a major etiological factor in illness, and provide evidence to support theoretical premises. By demonstrating links to a breadth of disorders, they show the effects of stress to be general, and not tied to any one organ (Juster et al., 2016). By focusing on excessive and unrelenting demands, they recognize that not every form of stress is pathogenic. In fact, acute stress is reported to *enhance* immune functioning (Gouin, 2011; Hussain, 2010). They acknowledge individual differences in stress susceptibility, with some people evidencing greater sensitivity to demands than others (Juster et al., 2016). Moreover, they tie these disparities to differential resources, in showing that adequate social support and economic reserves can protect immunological function (Hänsel et al., 2010).

Stress overload in the pandemic

There is little doubt that the coronavirus pandemic has imposed extraordinary adaptational demands upon people around the world. Beyond the new salience of death, there are a multitude of daily adjustments that must be made – more frequent hand-washing, maintaining distance from others, seeking once-staple household items, and so on. But not all pandemic demands are shared. Some people also experience job loss, perilous living conditions, or the death of a family member. In short, there are individual differences in demand-load arising from even this universal crisis.

Moreover, people vary in the resources they bring to these new demands. In regard to the physiological reserves emphasized by Selye (1956) and McEwen (2000), some may be hardy, and others already taxed. This corresponds with the differential vulnerability observed in the elderly, chronically ill, and immunocompromised. In regard to more tangible resources, some may be blessed with material comfort or social support, while others may be ill-equipped to deal with the economic and emotional repercussions of the pandemic. This, too, maps onto observed demographic disparities, and intimates that the underprivileged experience the pandemic as a more personal threat (Lazarus & Folkman, 1984) or debilitating loss (Hobfoll, 1989) than others.

But it is the people disadvantaged in both regards – facing greater pandemic demands with fewer resources – who are most likely to be pushed into the state of stress overload. It is that stress overload, in turn, which leads to dysregulated functioning and increased susceptibility to the coronavirus. Assessment of stress overload would therefore seem crucial to understanding the uneven spread of the virus, and predicting which populations are vulnerable to future infection.

Stress measurement

Perhaps one reason that stress has not been more widely examined as an etiological factor in the current pandemic is that stress assessment is problematic. Stress measures are, for the most part, divorced from theory (Lazarus, 1990) and psychometrically unsound (Dohrenwend, 2006). One approach has been to index recent stressful events in a person's life, whether major (Holmes & Rahe, 1967) or daily (Kanner et al., 1981). However, from the perspective of theory, such scales are only assessing one aspect of stress overload, demands, and ignoring the other, resources.

Enumerating a person's demands without subtracting their compensatory reserves yields a score that likely overestimates their true stress level. Another approach has been to assess a person's stress-resistance (Bartone, 2007). Such scales, theoretically speaking, are flawed in the opposite direction. By inventorying resources without considering the drain of demands, they yield scores that likely underestimate true stress. A third approach has been to directly assess stress symptoms (e.g., feeling nervous and stressed; Cohen et al., 1983). But theories are clear that acute, transitory feelings of stress do not necessarily signal an impending state of overload. By failing to distinguish benign from pathogenic symptoms, these scales can also yield inaccurate scores.

The Stress Overload Scales were constructed for the express purpose of assessing the pathogenic form of stress described in theories. A large pool of items that reflected thoughts and feelings of being overwhelmed were collected, and then subjected to a sequence of empirical tests. Early in this series, factor analytic investigations revealed that these items formed two clusters, one relating to demand load (e.g., felt like things kept piling up) and the other relating to exhausted resources (e.g., felt like just giving up). Because these factors matched the twin pillars of stress overload, one criterion for item retention was that it had to be a strong marker for one of the factors. Other criteria were that the item had to demonstrate psychometric strength (good reliability, construct and criterion validity), and comprehensibility across the demographically variegated community samples. At the end of five years, only 24 items survived to form the full SOS (Amirkhan, 2012); and because this measure was still seen as unwieldy by some researchers, the best 10 of these items were selected to form the SOS-Short (Amirkhan, 2018).

To complete the SOS-S, respondents report the extent to which they experienced each of the 10 thoughts or feelings in the prior week by means of 5-point response scales anchored at 1 (*Not at All*) and 5 (*A Lot*). Completion time is typically less than 5 min. The items form two subscales, Event Load (EL) which reflects demands, and Personal Vulnerability (PV) which reflects depleted resources. The subscale scores may be summed into a single total, with higher values indicating a greater likelihood of stress overload. Or they may be divided at the mean into high and low ranges, and then crossed to form a four-category diagnostic grid (see Appendix). These categories differentiate respondents according to their risk for stress overload: Low Stress indicates people who report few demands and adequate resources, who are at least risk; Challenged, a term borrowed from Lazarus and Folkman (1984), designates people facing many demands but with adequate resources, who are at low risk; Fragile, a term coined by Amirkhan (2012), describes people with depleted resources but few impinging demands, who are also at low risk; and High Stress identifies people who are facing many demands with inadequate resources and, according to theory, are at greatest risk.

Are stress overload measures accurate?

To date, the SOS and its derivative SOS-S are the only measures specific to stress overload. Perhaps because of their exhaustive empirical derivation, or perhaps because of their correspondence with stress theory, they have proven better predictors of pathology than popular stress measures (see Amirkhan, 2012, 2018). Total scores from the SOS measures have yielded stronger correlations with indicators of illness than demand-focused measures such as the Social Readjustment Rating Scale (Holmes & Rahe, 1967) and the Daily Hassles measure (Kanner et al., 1981). They have also been better predictors than resource-focused measures of Hardiness (Bartone, 2007) and Mastery (Pearlin et al., 1981). Finally, they have outperformed symptom-focused measures such as the Perceived Stress Scale (Cohen et al., 1983). The illness indicators used in these comparisons included inventories of self-reported symptoms, both somatic (e.g., nausea, coughing) and behavioral (e.g., forgetfulness, moodiness).

A flaw in such validity tests is that items on stress measures can be redundant of items on symptom inventories, artificially inflating the covariance between measures. An SOS item regarding feelings of inadequacy, for example, could well have parallels on a mental health checklist. However, there is evidence to dispel concerns that the correlations between the SOS and illness indices are spurious. First, such correlations remained significant, and nearly unchanged in magnitude, even

when the measures were administered at separate times, with gaps of one week to one month (Amirkhan, 2018; Amirkhan et al., 2015). This makes the possibility of response bleeding from measure to measure unlikely. Second, the relationships were significant even after negative affectivity and social desirability dispositions were statistically controlled (Amirkhan, 2012, 2018). This eliminates two of the most pernicious response biases that plague stress measure validity tests. Third, the SOS was found to correlate with objective criteria of illness assessed through dissimilar methods (Amirkhan et al., 2015), some of which were self-reported (journal entries of practitioner visits and missed work days) and some not (assays showing abnormal cortisol levels). This minimizes the likelihood of fixed response-set effects.

In regard to the validity of categorical SOS scores, theory predicts that those in the High Stress category (reporting both high Event Load and high Personal Vulnerability) should be at greatest risk for illness. Establishing thresholds to differentiate low- from high-scorers on each of the EL and PV subscales was therefore crucial. The decision was made empirically: After testing several cut-off points (scale mid-points, medians, modes), only the means consistently produced the pattern predicted by theory. Using normative means to divide samples, people in the High Stress category were found to differ significantly from those in Fragile, Challenged, and Low Stress categories in terms of the number of reported illnesses, symptoms, doctor visits and sick days (Amirkhan, 2012, 2018; Amirkhan et al., 2015). This may be seen in Figure 1, which shows the combined somatic + behavioral symptoms reported by each of the diagnostic groups, both at the time of SOS testing and one week later (data from Amirkhan, 2018).

Stress overload measurement in the pandemic

The SOS measures, and particularly the SOS-S because of its low respondent burden, could prove useful tools in understanding differential vulnerabilities to the coronavirus, and in modeling its likely spread across population groups. For health researchers, total scores will likely be most useful. These could be used to compare the mean stress overload levels of different demographics and thereby identify those at greatest risk for infection. Unlike laboratory tests, the SOS tests could be quickly administered to broad swaths of the population, since there would be no testing restrictions nor histological samples required. Moreover, SOS testing would be a priori, allowing researchers to identify where infections are likely to occur, whereas laboratory tests are a posteriori and can only help map prior infection. Not that SOS tests would replace laboratory tests, but they could be a useful precursor by suggesting where the latter should be directed for the greatest payoff. Past research with SOS measures has already identified several high-risk population pockets including childhood trauma survivors (Amirkhan & Marckwordt, 2017), first-year college students (Amirkhan & Kofman, 2018), minority and undocumented youth (Amirkhan & Velasco, 2019).

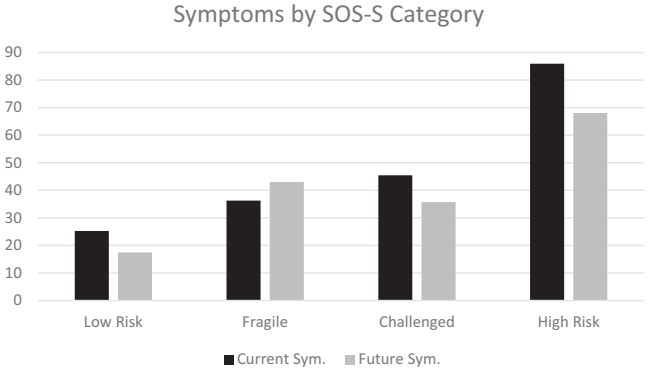

Figure 1. Illness symptoms reported by diagnostic category, at time of SOS-S and one-week later.

Categorical scoring will likely be more useful for practitioners. By determining the risk of infection for a given individual, it could serve a valuable triage function. Broad-based population mapping is valuable from a public health perspective, but not everyone in a high-risk group is equally likely to experience stress overload. Nurses working in Wuhan, China, for example, exhibited high SOS-S totals as a group, but among them, those who were single parents and/or working extended hours were particularly at risk (Mo et al., 2020). If mental-health services become overburdened during the pandemic, or if medical interventions are developed, practitioners could use categorical scores to determine priority for treatment. This is a unique feature of the SOS measures, since other scales do not provide cut-offs for differentiating high- from low-stress individuals.

Conclusion

Multiple theories and extensive research agree that stress increases susceptibility to infection, but stipulate that not every stress experience is pathogenic. It is only when the demands of adaptation overwhelm a person's resistive resources that stress overload occurs, which increases susceptibility to illness. This model corresponds well to the current pandemic, an experience that has imposed new demands and resource shortages on people around the world. Moreover, it offers an explanation for why some people are more likely to become infected than others. It was argued that stress overload assessment would be a valuable adjunct to histological testing, allowing researchers and practitioners to identify those at greatest risk for future infection. For this reason, the SOS-S is made available here (see Appendix) for use in the ongoing battle against further spread of the coronavirus.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Appendix. The Stress Overload Scale-Short

QUESTIONNAIRE # _____ **TODAY'S DATE** _____

S O S-S

(A Brief Measure of Day-to-Day Feelings)

Dr. James H. Amirkhan
Psychology Department
California State University Long Beach

INSTRUCTIONS:

Below, you will find 10 questions about your feelings during the past week. Please answer every question, even though some might sound similar. Each question names a common feeling, and has five answer boxes. You are to check the box that best describes how much you felt that particular feeling in the last week. Please be as honest as possible. There are no right or wrong answers, and your answers will be kept confidential.

IN THE PAST WEEK, have you felt:

1...inadequate?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
2...swamped by your responsibilities?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
3...that the odds were against you?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
4...that there wasn't enough time to get to everything?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
5...like nothing was going right?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
6...like you were rushed?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
7...like there was no escape?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
8...like things kept piling up?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
9...like just giving up?	☐	☐	☐	☐	☐
	Not At All				**A Lot**
10...like you were carrying a heavy load?	☐	☐	☐	☐	☐
	Not At All				**A Lot**

Thank You!

S O S-S
Scoring Instructions

STEP 1: Assign a numerical score to each response. A "Not At All" response is scored as a "1", while an "A Lot" response is scored as a "5". A response between these extremes is assigned a score according to its position; that is, a "2", "3", or "4".

STEP 2: Enter the numerical scores in the appropriate column below:

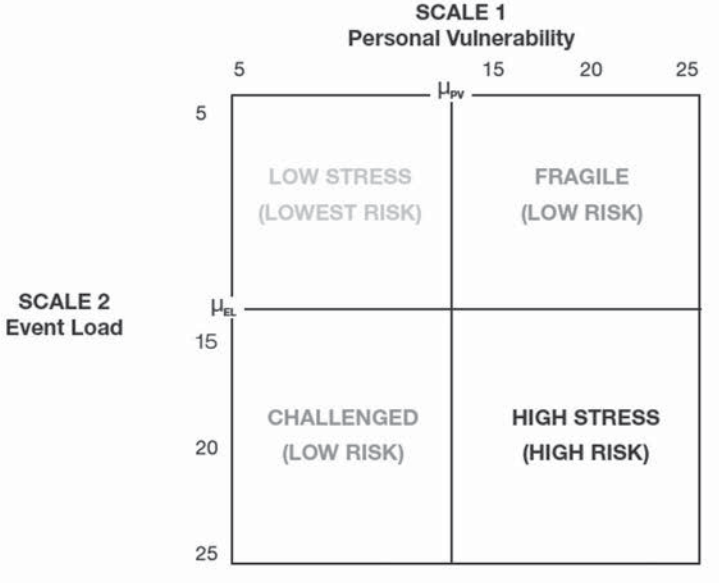
SCALE 1 *(Odd-Numbered Items)*		SCALE 2 *(Even-Numbered Items)*	
Item	**Score**	**Item**	**Score**
#1	_____	#2	_____
#3	_____	#4	_____
#5	_____	#6	_____
#7	_____	#8	_____
#9	_____	#10	_____

STEP 3: Add the scores in each column:

TOTAL 1 = _____
(Personal Vulnerability Scale)

TOTAL 2 =_____
(Event Load Scale)

STEP 4 For **CONTINUOUS SCORING:** Add Total 1 and Total 2.
For **CATEGORICAL SCORING:** Use these totals to locate the appropriate diagnostic quadrant for the respondent on the grid below:



NOTE: In normative samples (combined n = 657), μ_{PV} = 9.15 and μ_{EL} = 12.29. However, means will vary with the population under study, and should be reset accordingly.