



The Pathway From Social Status to Physical Health: Taking a Closer Look at Stress as a Mediator

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Abstract

Stress is often invoked as a potential contributor to disparities in physical health as a function of social status. Although there is good reason to believe that stress exposure and stress responses may be an important pathway linking lower social status to poor health, direct evidence is lacking. We summarize the evidence for this pathway and limitations of that evidence, focusing particularly on how stress is conceptualized and measured. We argue that in addition to more direct tests of mediation, the measurement of the mediator—stress—could also be improved. We also propose that measuring theory-specific stress exposures may be more fruitful than assessing general stress exposures (e.g., life events, global perceived stress) by increasing theoretical clarity and predictive utility of stress in this context.

Keywords

socioeconomic status, social status, stress, physical health, health disparities

Reducing disparities in health due to social status, including individuals' socioeconomic positions, has been a primary objective of public policy in the United States for more than 30 years (https://www.healthy people.gov/2020/about/foundation-health-measures/ disparities). Decades of evidence support the association between social status (e.g., socioeconomic position, which includes both social rank and resources) and health; higher rates of disease and shorter life spans have been found among individuals of lower socioeconomic status (Adler, 2009; Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010; Chetty et al., 2016; Matthews & Gallo, 2011). This association is multiply determined and not simply due to poverty, poor health behaviors, or limited access to medical care (Braveman & Gottlieb, 2014; Lantz et al., 2001; Marmot & Sapolsky, 2014; Matthews, Gallo, & Taylor, 2010). For example, approximately 75% of the variance in health due to social status is left to be explained after inclusion of health behaviors (Pampel, Krueger, & Denney, 2010). Additionally, these disparities exist in both high- and low-income countries, as well as in countries with and without universal health care, and they are monotonic in nature, meaning that each reduction in social status is associated with increased risk for poor health (Adler, 2009). Further, this social-status-related health gradient can be more pronounced in high-income countries for chronic diseases thought to be influenced by psychological factors, such as coronary heart disease (Marmot & Sapolsky, 2014). All of these findings, along with the fact that subjective perceptions of social rank predict health at least as well as and independently of material resources (e.g., Adler et al., 1994; Cundiff & Matthews, 2017), have galvanized research on psychobiological factors, such as stress, that may help explain the link between social status and physical health.

Notably, stress has been conceptualized and measured in a number of different ways, and the utility of the term *stress* itself has been ardently debated (e.g., Cohen, Gianaros, & Manuck, 2016; Kagan, 2016). Recent integrative perspectives suggest that stress can be

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broadly defined as "a set of constructs representing stages in a process by which environmental demands that tax or exceed the adaptive capacity of an organism occasion psychological, behavioral, and biological responses that may place persons at risk for disease" (Cohen et al., 2016, p. 456). We adopt this definition of stress and note that stress can be further organized into stress exposure (i.e., stimuli or conditions that elicit stress responses) and stress responding (behavioral, affective, and biological changes elicited by stress exposure). For example, epidemiological research tends to measure stress exposure, primarily in the form of objective life events (e.g., divorce, job loss, death in the family), assuming that the same objective event is equally stressful for all individuals. Psychological research tends to focus on subjective perceptions of stress exposure (e.g., demands without adequate resources to cope, perceived threats) as well as affective and behavioral responses to stress. Closer to the disease process, biopsychosocial perspectives link objective and perceived stress exposures to changes in diseaserelevant biology, including stress responding (e.g., controlled exposure to stress in the laboratory and measurement of biological changes).

Stress as a Pathway Linking Social Status to Poor Physical Health: Current Evidence

Stress exposure and responses, particularly biological responses, are often invoked as important pathways linking lower social status to poorer physical health (e.g., Adler & Snibbe, 2003; Seeman, Merkin, Karlamangla, Koretz, & Seeman, 2014). Low social status is thought to contribute to disease, in part, because it entails more frequent and chronic exposure to stress (Cohen & Janicki-Deverts, 2012). Theoretically, this increased stress exposure affects health via psychological and biological responses that are more frequent, larger in magnitude (e.g., greater reactivity), or more prolonged (e.g., slower recovery), which in turn contribute to disease pathophysiology (Steptoe & Kivimaki, 2013).

Although links between social status, stress exposure and response, and physical health have intuitive appeal, empirical support is limited because analyses are piecemeal; researchers examine associations between social status and stress exposure or responding *or* between stress exposure and responding and physical health, rather than the full pathway of interest (social status \rightarrow stress exposure and response \rightarrow physical health). Thus, despite the fact that stress exposure and responding is commonly cited as a mechanism linking social status and health, this assumption is rarely tested (Matthews et al., 2010). Further, the few observational studies that

did test the full mediational path did not provide clear conclusions about stress exposure as a mediating pathway. For example, although several of these studies documented a reduction in the effect size of the relationship between social status and physical health after controlling for stress exposure (consistent with mediation), the majority found little or no evidence of stress exposure as a mediator, including a lack of differences in reported objective stress exposure or perceived stress as a function of social status (Matthews et al., 2010).

In addition to epidemiological and other observational research examining health outcomes, laboratory studies of biological responses to acute stress are also relevant. Biological responses to stress in the lab have been linked to death and disease onset (e.g., Chida & Steptoe, 2010), and these acute biological changes following stress exposure are thought to be an important driver of disease pathology. However, laboratory evidence often does not find social-status differences in biological reactivity to acute stress. For example, a recent review and meta-analysis found no reliable association between social status and cardiovascular reactivity to acute stressors in the laboratory (Boylan, Cundiff, & Matthews, 2018), and another large study found no reliable association between social status and cortisol reactivity to laboratory-based stressors (Lê-Scherban et al., 2018). There is evidence that inflammatory reactivity to stress exposure may differ according to people's social status, although there are very few studies to draw from (Derry et al., 2013; Muscatell et al., 2016). Notably, biological recovery following stress exposure has also been linked to disease risk (Chida & Steptoe, 2010). This may be a promising but underresearched biological pathway given that lower social status has been linked with delayed recovery in cardiovascular, inflammatory, and cortisol responses to stress (Boylan et al., 2018; Brydon, Edwards, Mohamed-Ali, & Steptoe, 2004; Derry et al., 2013; Lê-Scherban et al., 2018). Again, however, there are few relevant studies examining biological recovery in this context.

Taken together, current findings from observational and laboratory studies of social status and stress raise questions about whether stress exposure (objective events), perceived stress, or biological stress responses, especially reactivity, are a viable pathway linking social status and physical health. There are multiple potential reasons for the lack of compelling evidence. We focus on the possibility that the current evidence may be the result of methodological choices rather than a true null effect. We argue that stress exposure and biological responses to stress have previously been operationalized in ways that may not be particularly relevant for understanding the mechanisms linking social status and physical health. We suggest that refining measures of stress exposure in

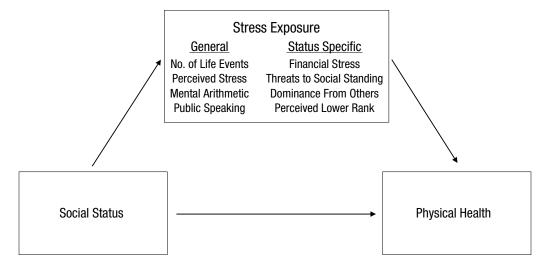


Fig. 1. The simple mediational model central to the research discussed in this article: stress exposure acts as a mediator to the effect of social status on physical health. The model shows that disparities in stress may partially account for disparities in health. The stress box provides examples of general operationalizations of stress typical of those reported in the current literature and examples of stressors specific to the context of social status, which we argue would be fruitful to explore. Stressors are thought to cause biological changes in the cardiovascular (e.g., blood pressure), neuroendocrine (e.g., cortisol), and inflammatory (e.g., circulating interleukin-6) systems during reactivity to and recovery from stress. These biological stress responses associated with stress exposure (not depicted) link stress to poor physical health.

both observational and laboratory research will more clearly elucidate potential links among social status, stress exposure and response, and health (see Fig. 1).

How Are We Currently Measuring Stress?

Observational studies examining stress as a mediator of social status and physical health typically assess a frequency or count of stress exposures (e.g., number of stressful events experienced during the past year, such as divorce, physical attack or assault, death of a child) or general perceptions of stress (e.g., how often participants felt tense or psychologically stressed in preceding days or months). They often fail to capture perceived severity and duration of the stressors experienced, which may be an important feature of how stress exposure and response differ across social status (e.g., Almeida, Neupert, Banks, & Serido, 2005; Grzywacz, Almeida, Neupert, & Ettner, 2004). Observational studies, particularly large epidemiological studies, often cannot effectively capture important responses to stress exposures (e.g., Did the individual perceive the event as stressful or threatening, controllable or uncontrollable? Was there an affective change associated with the stressor? Were there biological changes associated with the stressor?). Additionally, the exposures assessed are often not specific to the context of social status (e.g., financial stress) but rather are general measures of stress based on historical norms of epidemiological stress measurement (e.g., life-events checklists). Further, many studies operationalize stress as dysregulation in static biological measures (e.g., elevated circulating inflammatory markers), which problematically conflates stress exposure with biological correlates of stress. This approach falsely assumes that because stress can produce dysregulation in biological markers, dysregulation in biological markers reflects only stress. While stress may sufficiently produce dysregulation in biological markers, it is unlikely to be the only sufficient cause of dysregulation (e.g., stress may influence circulating inflammation, but so does body fat).

There are likewise issues with standardized laboratory manipulations of stress in the context of socialstatus-related health disparities. Most often, cognitive stressors (e.g., mental arithmetic) are employed, although social stressors, such as public speaking, are also sometimes used, as well as a combination of both types of stressors (Boylan et al., 2018). Although such manipulations represent well-accepted laboratory stress protocols, they are designed to reflect general stressors and are not specific to the context of social status, which limits what we may learn from them. For example, it may be problematic to assume that responses to a serial-subtraction task adequately capture hypothesized differences in stress exposure or subsequent stress response as a function of social status. Additionally, familiarity and engagement with the tasks can influence stress responses (Wright & Kirby, 2001), and such tasks may not be equally familiar and challenging across levels of social status.

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Moving Forward

Many questions remain about whether disparities in stress exposure and response contribute to disparities in health. As mentioned above, very few studies have explicitly tested mediational models, which significantly limits the evidence base for understanding whether and to what extent stress may play a role in social-status-related health disparities. Additionally, the limited evidence that is available seems to suggest that we may be measuring stress exposure and responses in several ways that do not usefully explain the link between social status and health.

We argue that in addition to more direct empirical tests of mediation, the measurement of the mediator (i.e., stress) could also be improved. Research would benefit from more complete assessments of selfreported stress exposure (e.g., severity, controllability, domain; Epel et al., 2018; Gallo et al., 2013) and from exploration of novel measurement and manipulation of laboratory stress exposure in order to identify stressors that may be viable mediators. We propose that measuring stress exposures specific to the independent variable of interest—social status—may be more fruitful than assessment of general stress exposures (e.g., life events, global perceived stress) and may increase theoretical clarity and predictive utility of stress in this context (e.g., Almeida et al., 2005; see Fig. 1). We are not arguing for one particular theoretical model but rather calling for a more consistent reliance on theoretical models in empirical research addressing stress in the context of social-status-related health disparities.

Along these lines, researchers may want to consider measuring and manipulating experiences that mimic real-world stress exposures known or thought to disproportionately affect individuals of lower social status, thus potentially explaining disparities. For example, individuals of lower social status may be exposed to the following types of objective or perceived stress more frequently or severely compared with higher-status individuals: financial stress; stress associated with actual or perceived social subordination; threats to social status such as being devalued, rejected, or discriminated against; lack of control over one's environment; and being exposed to more dominance and control from others (Almeida et al., 2005; Cundiff & Smith, 2017; Gallo, Smith, & Cox, 2006; Smith, Cundiff, & Uchino, 2012). Therefore, researchers interested in social-statusrelated health disparities may find benefit in measuring and manipulating stressors with these characteristics. Additionally, each individual stress exposure or reaction is likely to account for only a small portion of the increased risk associated with lower social status, and thus utilizing multiple measures of context-specific stress exposure and reactivity may increase predictive utility (Adler, Bush, & Pantell, 2012).

We also acknowledge that although our primary focus is on social-status-related health disparities that track a social gradient (see the first paragraph of the introduction), stress mediation is not at the heart of all theories regarding social status and health. For example, reactivity to stress can also be conceptualized as a stable individual difference that moderates associations between stress exposure and disease risk. In this framework, individuals who are "high reactors" and who also live in an environment with high exposure to stressors (e.g., low social status) may be most at risk for biological dysregulation and eventual disease (e.g., Lynch, Everson, Kaplan, Salonen, & Salonen, 1998). Results from a series of studies in a well-developed animal model also suggest multiple potential moderating effects in humans (Kaplan, Chen, & Manuck, 2009; Kaplan & Manuck, 1999). For example, the association between relative social status (i.e., rank in the local community) and stress exposure and responding may differ according to accessibility of resources and absolute social status (e.g., income, neighborhood socioeconomic status), at least for men (Kaplan & Manuck, 1999). Associations between income inequality and health also necessitate different conceptual frameworks as these frameworks are intended to explain why the health of all people may suffer with greater income inequality as opposed to why those lower in the social hierarchy suffer worse health because of their relative position in the hierarchy. If investigators want to examine whether stress plays a role in the inequality-health link, they may measure stress exposures or responses that could explain the "worse health for everyone" argument associated with inequality (e.g., everyone exposed to more violence, reduced social cohesion; Pickett & Wilkinson, 2015) as opposed to stress exposures or responses that could explain the worse health associated with being lower in social status (e.g., more financial stress).

Leveraging the Laboratory

Well-controlled laboratory studies have been underutilized by researchers who aim to understand social-status-related health disparities. Such studies would allow researchers to test assumptions about social status, stress exposure, and biobehavioral responses to stressors in ways that are not possible in large epidemiological and observational work. Transient manipulations of social status are far from a perfect corollary to absolute social status in the real world, but they are useful. For example, experimental designs that manipulate (perceived) social status can help answer questions about whether the psychological experience of being socially subordinate in and of itself is stressful (e.g., Cundiff, Smith, Baron, & Uchino, 2016; Pieritz, Süssenbach, Rief,

& Euteneuer, 2016). Further, designs that manipulate social status and subsequently expose participants to stress can answer questions about whether lower social status potentiates reactivity to stressors and, if so, what kind of stressors (e.g., financial stress vs. social conflict; e.g., Cundiff et al., 2016; Mendelson, Thurston, & Kubzansky, 2008). For example, some theories suggest that threats to collective or affiliative goals (but not individualistic or agentic goals) may be perceived as more stressful and evoke larger stress responses in low-status individuals (e.g., Laurin, Engstrom, & Alic, 2019).

Experimental work may be most useful if manipulations of social status are as consistent as possible with components of status that are predictive of health at the population level. For example, social status at the population level is most commonly assessed as a combination of income, education, occupation, and perceptions of one's relative standing. Because absolute education, occupation, and income typically cannot be manipulated, researchers may try to manipulate what they believe is the "active ingredient" in these absolute measures. For example, indicators of social status are moderately correlated, but each is independently associated with physical health, which has been interpreted as support for the idea that social stratification (e.g., rank) is partially responsible for disparities between social status and health (e.g., Daly, Boyce, & Wood, 2015). If this is how investigators are conceptualizing the problem, then creating a perceived hierarchy in the lab is theoretically consistent. Manipulating perceptions of one's standing relative to others on these indices may be a good option (e.g., being made to feel that you have much less money than someone else), and manipulations of actual—rather than perceived—relative rank are also quite possible (e.g., being asked to interact with someone who is relatively more educated than you are).

Similarly, researchers interested in answering questions about social-status-related health disparities using laboratory manipulations should also manipulate stress exposures that are specific to social status. For example, interpersonal experiences of being devalued or discriminated against or participating in a task that mimics the experience of having to make difficult decisions about what to spend on a limited income (e.g., the spent task; http://playspent.org/html/) may be more useful than serial-subtraction or mirror-tracing tasks. Although some stress-reactivity paradigms (e.g., the Trier social stress test) incorporate status-relevant elements of stress (e.g., threats to perceived competence, uncontrollability), there is room for new reactivity paradigms that tap more specifically into theory-relevant stressors that may contribute to disease.

Other types of laboratory paradigms are also useful in testing theoretically informed hypotheses. For example, individuals' schemas, goals, and motivations might contribute to differences in stress exposure and responding as a function of social status (American Psychological Association Working Group on Stress and Health Disparities, 2017). These evaluative judgments and goal systems that influence behavior and biology (including stress responding) outside of conscious awareness can be assessed using implicit mechanisms (e.g., Ewart, Elder, & Smyth, 2014). Such designs might reveal status-related effects on stress physiology that differ from results using more common methodologies such as exclusive reliance on self-reports (which are more affected by self-presentation and social-desirability biases).

Finally, a complementary approach to testing causal assumptions through laboratory experiments is the use of ambulatory approaches to assess these same statusrelated stressors and their biological correlates in daily life (e.g., Chiang et al., 2015; Ewart et al., 2014; Gallo, Bogart, Vranceanu, & Matthews, 2005; Matthews et al., 2000). Such studies of daily life that can link stress exposure and responses to physical health across individuals of differing social status allow researchers to look at within-persons processes similar to those in the laboratory but with real-world, rather than contrived, stressors. Assumptions of laboratory manipulations can also be examined in these studies of daily life. For example, future research could examine whether individuals of lower social status are more likely to have day-to-day social interactions in which they perceive themselves to be lower in status and whether these repeated exposures (if they exist) contribute to differences in biology in real life.

Conclusions

Epidemiological findings clearly document the presence of health disparities among individuals of differing social status. However, whether these disparities in health are due in part to disparities in stress is less clear. There is a strong need for future work to explicitly test this proposed mediation model. Further, on the basis of the limited evidence that is available, researchers seem to be measuring stress exposure and responses in a number of ways that do not mediate the relationship between social status and health. We suggest that the measurement and manipulation of stress exposures could be improved by clearer use of theoretical models specific to the independent variable of interest. Measuring and manipulating social-status-specific rather than general stressor exposures and testing underlying assumptions in more controlled laboratory studies may improve our understanding of stress processes that link lower social status to poorer physical health (see Fig. 1). Contextualizing stress assessment to social-status-related

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health disparities across disciplines and research designs will also promote more consistency among theory, epidemiological research, observational research, and experimental research in this area. Such consistency may accelerate progress regarding whether, how, and what types of stress contribute to these disparities and would allow for stronger causal inference about the connections along the complex causal path from social status to stress to poor health.

Recommended Reading

- Almeida, D. M., Neupert, S. D., Banks, S. R., & Serido, J. (2005). (See References). An example of the nuances of stress measurement in daily life and links to physical health, including the assessment of status-specific stressors.
- Boylan, J. M., Cundiff, J. M., & Matthews, K. A. (2018). (See References). A review and meta-analysis examining whether cardiovascular stress responses differ by individual differences in socioeconomic status.
- Cundiff, J. M., Smith, T. W., Baron, C. E., & Uchino, B. N. (2016). (See References). A representative empirical article examining a laboratory manipulation of perceived status, status-specific stressors, and biological responses.
- Epel, E. S., Crosswell, A. D., Mayer, S. E., Prather, A. A., Slavich, G. M., Puterman, E., & Mendes, W. B. (2018). (See References). A broad review of issues in the definition and measurement of stress that also provides an organizing framework for the measurement of stress across disciplines.
- Matthews, K. A., Gallo, L. C., & Taylor, S. E. (2010). (See References). A review article that provides additional context and detail regarding the assessment of stress in observational research.

Transparency

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