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Physiological Mechanisms That Underlie the Effects of Interactional Unfairness on Deviant Behavior: The Role of Cortisol Activity

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Although experiencing unfairness is a primary source of stress, there are surprisingly few studies that have examined the physiological underpinnings of unfairness. Drawing from social self-preservation theory, we derive predictions regarding the effects of interactional unfairness on activity in the hypothalamic-pituitary-adrenocortical (HPA) axis, which is one of the body's primary hormonal systems for responding to stress. Using an experimental design with objective physiological measures, we found support for our hypothesis that interactional unfairness triggers the release of cortisol by the HPA axis. This cortisol activity in turn mediated the effects of interactional unfairness on deviant behavior. This indirect effect remained significant after controlling for established attitudinal and self-construal mediators of the justice–deviance relationship. We discuss the theoretical and practical implications of these findings for the occupational stress and organizational justice literatures.

Keywords: interactional justice, cortisol, stress, deviant behavior, identity

Managing employees' experiences of fairness is a critical undertaking in work organizations (Cropanzano, Bowen, & Gilliland, 2007). Employees place a high premium on justice because it is relevant for instrumental reasons (e.g., fairness ensures that rewards are controllable and predictable; Adams, 1965; Thibaut & Walker, 1975), relational reasons (e.g., fairness indicates that recipients are valued and respected; Lind & Tyler, 1988), and moral reasons (e.g., fair treatment is a moral imperative that everyone is owed; Folger, 2001). It is therefore not surprising that the fairness of work outcomes, procedures, and interpersonal treatment predict a variety of employee attitudes (e.g., job satisfaction, organizational commitment) and behaviors (e.g., task performance, citizenship behavior; Cohen-Charash & Spector, 2001; Colquitt, Conlon, Wesson, Porter, & Ng, 2001; Colquitt et al., 2013).

Looking beyond attitudes and behaviors, it has also been suggested that fairness is a key player in the stress process and has ramifications for employee health (Greenberg, 2004; Spector & Fox, 2005; Vermunt & Steensma, 2005; Zohar, 1995). According to this view, unfair treatment is a stressor (i.e., an event that threatens well-being and induces aversive emotional and physiological states; Hart & Cooper, 2001), which gives rise to various strains. Examples of strains that have been linked to unfairness include emotional exhaustion (Tepper, 2000), depression (Elovainio, Kivimäki, & Helkama, 2001), health complaints (de Boer, Bakker, Syroit, & Schaufeli, 2002), illness-related absences (Elovainio, Kivimäki, Vahtera, Virtanen, & Keltikangas-Järvinen, 2003), and insomnia (Greenberg, 2006). These and other health-related consequences of unfairness are summarized by Robbins, Ford, and Tetrick (2012).

In their review, Robbins et al. (2012) presented a model linking unfairness to employee health. Several mediating mechanisms for this relationship are proposed, such as perceived stress, negative emotional states, and unhealthy behaviors (e.g., alcohol consumption). They noted, however, that the strongest mechanisms through which unfairness impacts health are likely physiological ones, especially activity in the hypothalamic-pituitary-adrenocortical (HPA) axis. This axis is a hormonal response system that regulates the release of glucocorticoid (or cortisol), which mobilizes the body's energy resources in response to stress (Dickerson & Kemeny, 2004). Although it helps the body respond to stress and maintain homeostasis, prolonged exposure to elevated levels of cortisol is associated with a number of negative health effects (e.g., immune system dysregulation, development of heart disease; McEwen & Stellar, 1993). As noted by Miller, Chen, and Zhou (2007, p. 25), "cortisol is a critical biological intermediary; it is

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seen as a primary mechanism through which chronic stressors get inside the body to bring about disease." An understanding of the health-related effects of unfairness therefore necessitates an examination of physiological mechanisms, such as the release of cortisol by the HPA axis.

Despite the potentially important role of physiological mechanisms, few studies have explored the interplay of unfairness and physiological functioning. Robbins et al. (2012) identified only six studies that directly assessed health via biochemical tests or medical evaluations by trained health care workers. More common are studies that examined self-reported indicators of psychological health, such as perceived stress (17 studies), negative emotional states (23 studies), and burnout (27 studies). Unfortunately, self-ratings are a poor proxy for objective assessments of physiological health (Ganster & Perrewé, 2011; Semmer, Grebner, & Elfering, 2004), and they also contribute to common method variance when other variables are also self-reported (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). Thus, Robbins et al. (2012) concluded their review by saying that "research examining the impact of unfairness on objective measures of employee health is sorely lacking" (p. 250).

The aim our study was to examine the effects of interactional unfairness on cortisol activity and whether this activity contributes to deviant behavior. In addition to addressing the general need for research on unfairness-based physiological effects, our study makes several contributions. First, we examined physiological effects that are tied specifically to the HPA axis, which is believed to be the primary culprit for stress-related diseases (McEwen & Stellar, 1993; Miller et al., 2007). Previous research on the physiological effects of unfairness has focused on more distal health indicators, such as body mass index (Elovainio et al., 2006) and upper respiratory infections (Xie, Schaubroeck, & Lam, 2008), which do not directly reflect activity in the HPA axis. Cortisol also provides a more direct assessment of physiological responses to stress, more so than do skin conductivity and heart rate (Dickerson & Kemeny, 2004). These latter examples are more reflective of arousal, which can occur in the absence of stress (e.g., tasks that require careful attention but pose no threat to one's physical or social self can increase skin conductivity and heart rate). Thus, cortisol activity, which we examined in the current study, provides a "purer" gauge of the stress response.

Second, we examined the effects of interactional unfairness (i.e., unfair interpersonal treatment and explanations; Bies & Moag, 1986) as opposed to distributive unfairness (i.e., unfair work outcomes; Adams, 1965) and procedural unfairness (i.e., unfair procedures used to establish outcomes; Lind & Tyler, 1988). Although stress reactions are believed to be strongest in response to interactional unfairness (Greenberg, 2004), prior research has mostly examined distributive and procedural unfairness (e.g., Brotheridge, 2003; Vermunt & Steensma, 2003; Xie et al., 2008). This lack of attention is surprising given that interpersonal stressors produce high levels of cortisol activity (Almeida, 2005; Almeida, McGonagle, & King, 2009). Thus, current estimates of the effects of unfairness on physiological health may be underestimated, given that most of the evidence is based on less distressing types of unfairness. The idea that interactional unfairness is particularly stressful is consistent with social self-preservation theory (Dickerson & Kemeny, 2004), which proposes that HPA activity occurs in response to direct threats to one's social self. Although procedural and distributive justice have implications for one's social self (e.g., Johnson & Lord, 2010), interactional unfairness sends the clearest and most direct signal of social rejection and devaluation (Bies, 2001).

Third, we manipulated interactional unfairness, allowing us to draw conclusions about the causal effects of unfairness on physiological functioning. Nearly all previous unfairness research involving physiological measures has been correlational in nature (e.g., Elovainio et al., 2006; Ferrie et al., 2006; Xie et al., 2008). While informative, these studies cannot rule out alternative causal explanations (e.g., appraisals of unfairness may result from poor physiological health). In addition, manipulating unfairness helps bypass the limitations of having participants recall unfairness experiences *a posteriori* (e.g., memory biases; K. van den Bos, 2001). Given our interest in examining the acute effects of unfairness on stress, an experimental research design that captures justice and stress *in situ* is necessary.

A final contribution is that we also assessed a behavioral outcome of the unfairness–stress process. Although a growing body of evidence has suggested that cortisol activity elicits psychological strains (e.g., anxiety disorders; e.g., McEwen & Sapolsky, 1995; Soravia & de Quervain, 2012), it is unclear what implications cortisol activity has for organization-relevant behavior. To date, unfairness studies involving objective measures of physiological health have excluded behavioral consequences of experiencing stress, whereas studies involving subjective measures have been limited to withdrawal behaviors (e.g., de Boer et al., 2002; Elovainio et al., 2003). Because deviant behavior is a ubiquitous outcome of interactional unfairness (Cohen-Charash & Spector, 2001), we tested whether cortisol activity contributes to this effect. As Judge and Colquitt (2004, p. 395) noted, "unfair treatment can have a visceral—even physiological—effect on employees, thereby disrupting work attitudes and behaviors," and we provide an initial test of this idea.

Interactional Unfairness, Physiological Stress, and Deviant Behavior

Social self-preservation theory (Dickerson & Kemeny, 2004) provides an explanation for the effects of interactional unfairness on cortisol activity and deviant behavior. This theory posits that people have a social self-preservation system that is sensitive to potential threats to their social self. When threatened, the social self-preservation system guides physiological, psychological, and behavioral responses in order to cope with the threat, including HPA activation and the release of cortisol (Dickerson & Kemeny, 2004). Although it was traditionally believed that cortisol is released in response to all types of stressors, it has since been concluded that stressors that threaten the central goals of preserving one's physical and social selves are most responsible for triggering the release of cortisol (Dickerson & Kemeny, 2004; Sapolsky, 2004). Once released, cortisol mobilizes resources and coordinates activity in other physiological systems in order to respond to the short-term demands of threats (Sapolsky, Romero, & Munck, 2000).

To date, meta-analytical evidence (Dickerson & Kemeny, 2004) suggests that acute, evaluative social threats lead to the strongest cortisol activity. Especially potent social-evaluative threats are those that signal a lack of esteem, respect, and acceptance by

others. As mentioned earlier, one reason people care about justice is because it communicates information relevant to their relational concerns: Fairness signals that recipients are valued and respected, whereas unfairness signals the opposite (Blader & Tyler, 2005; Lind, 2001; Lind & Tyler, 1988). Of the different types of unfairness, interactional unfairness sends the clearest signal of social devaluation and rejection, as it entails social interactions characterized by a lack of respect and propriety (Bies, 2001; Bies & Moag, 1986). For this reason, Greenberg (2004) concluded that interactional unfairness is likely the most stressful form of unfairness. Based on social self-preservation theory, exposure to interactional unfairness should therefore trigger the release of cortisol.

> *Hypothesis 1:* Experiencing interactional unfairness results in higher cortisol levels than does experiencing interactional fairness.

The release of cortisol in response to social-evaluative threats mobilizes energy-dependent systems that produce behavioral outputs intended to cope with or reduce the threat (Dickerson & Kemeny, 2004). Evidence from the glucocorticoid physiology literature (O'Leary, 1990; Sapolsky et al., 2000) supports the notion that cortisol activity stimulates energy production and suppresses unneeded physiological activities in order to cope with threats. One way of preserving the social self is via displays of aggression toward the source of social-evaluative threats (Dickerson & Kemeny, 2004). Indeed, research findings have indicated that cortisol activity is linked to the expression of socially aggressive behaviors (e.g., Ranjit et al., 2009; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998). There are two explanations as to why this link exists. First, cortisol activity impacts information processing in a way that facilitates social aggression. For example, research findings have suggested that acute cortisol activity promotes impulsivity and risky decision making, and it lessens sensitivity to punishment (Putman, Antypa, Crysovergi, & van der Does, 2010; R. van den Bos, Harteveld, & Stoop, 2009; Wohl, Matheson, Young, & Anisman, 2008). When people are impulsive, willing to take risks, and blind to the possible harmful consequences that their behavior has on others, they are more likely to engage in deviant acts (Lynam, 2011; Spector, 2011).

Second, cortisol activity contributes to aggression because it depletes the resources needed to control socially aggressive urges. When faced with social-evaluative threats, the HPA axis triggers the release of cortisol, which redirects energy resources for needed metabolic functions (Dickerson & Kemeny, 2004). The primary energy resource used to fuel these metabolic functions is glucose, which also fuels self-regulatory functions like aligning behavior with social norms and overriding impulses (Gailliot et al., 2007). Thus, cortisol activity draws from a finite pool of resources that, when depleted, reduce people's ability to exert subsequent self-control (Baumeister, Bratslavsky, Muraven, & Tice, 1998). In line with this idea, it has been shown that resource depletion makes it more difficult for people to inhibit deviant impulses (e.g., Barnes, Schaubroeck, Huth, & Ghumman, 2011; Christian & Ellis, 2011; Gino, Schweitzer, Mead, & Ariely, 2011).

According to social self-preservation theory (Dickerson & Kemeny, 2004), the cortisol activity triggered by interactional unfairness is expected to increase the likelihood of people subsequently engaging in deviant behavior. This logic is also consistent with the allostatic load model, which describes how the body responds to stressor-related challenges to its homeostatic systems (see Ganster & Rosen, 2013). This model posits that stress hormones like cortisol are part of the first response system that facilitates adaptation to stressors, which in turn influences secondary processes (e.g., cardiovascular) and tertiary outcomes (e.g., psychological and health consequences). For both self-preservation theory and the allostatic load model, cortisol is a key mediator linking stressors to outcomes. We therefore hypothesized the following:

> *Hypothesis 2:* Cortisol levels are positively related to subsequent deviant behavior.

> *Hypothesis 3:* Cortisol levels mediate the effect of interactional unfairness on deviant behavior.

There are at least two other mediation explanations for the effect of interactional unfairness on deviant behavior (Cohen-Charash & Spector, 2001; Colquitt et al., 2001). One explanation is that experiencing unfairness causes recipients to develop negative emotions and attitudes toward the unfair party (Judge, Scott, & Ilies, 2006). Indeed, a recent meta-analytic review by Colquitt and colleagues (2013) highlighted emotions and social exchange attitudes as primary mediating mechanisms of the effects of unfairness. When employees hold negative attitudes, it reduces their willingness to work in favor of the unfair party and often encourages retaliatory acts (Cohen-Charash & Spector, 2001). Deviant behavior also offers a cathartic means for dissatisfied recipients to "pay back" the unfairness they experienced, enabling them to restore equilibrium in their exchange relationships (Bennett & Robinson, 2003; Bies, 2001). Thus, dissatisfaction with the unfair party is another mediator of the link between unfairness and deviant behavior.

A second explanation is that experiencing interactional unfairness causes a shift in recipients' self-concepts away from the source of unfairness (Blader & Tyler, 2005; Lind, 2001). For example, Johnson and Lord (2010) found that exposure to unfair treatment reduced the activation of people's interdependent identities, or the extent to which they define themselves in terms of their connections to others. When recipients no longer feel a sense of belonging and duty to the unfair party, it makes it easier for them to act against the party's interests (Johnson & Saboe, 2011). A weak interdependent identity also reduces recipients' sensitivity to how their own behavior may adversely impact others (Yang, Johnson, Zhang, Spector, & Xu, 2013). Thus, like dissatisfaction, a weak interdependent identity is another established mediator of the effects of unfairness (Blader & Tyler, 2009; Johnson & Lord, 2010; Olkkonen & Lipponen, 2006).

Given that multiple mechanisms mediate the effect of unfairness on deviant behavior, it is important to verify that cortisol activity makes a unique contribution incremental to dissatisfaction and identity. We suspect that cortisol activity has unique effects on deviant behavior because the HPA axis is mostly distinct from the physiological systems that regulate affect and cognition, such as the limbic system and frontal cortex. To address this issue, we therefore controlled for dissatisfaction and identity when examining the mediating effects of cortisol activity.

> *Hypothesis 4:* The indirect effect of interactional unfairness on deviant behavior via cortisol levels remains significant after controlling for dissatisfaction and interdependent identity.

We tested our set of hypotheses using an experimental design in which participants were exposed to either interactional unfairness or fairness. We based our manipulation on previous research that manipulated justice (e.g., Johnson & Lord, 2010; Zapata-Phelan, Colquitt, Scott, & Livingston, 2009). Our decision to conduct an experiment was driven by the need to verify that unfairness has causal effects on acute physiological stress, which is not possible with studies using correlational designs (e.g., Elovainio et al., 2006; Xie et al., 2008). Our method is described below.

Method

Participants

We recruited participants enrolled in undergraduate courses from a large university in the southeastern United States. Based on a power analysis (Faul, Erdfelder, Buchner, & Lang, 2009), a sample size of 79 was needed to have sufficient power (.80) to detect a small effect ($R^2 = .09$; $r = .30$) with an α of .05, which led us to recruit 86 participants. Of these 86, two were excluded because they did not feel comfortable providing saliva samples for the cortisol measure, four were excluded because they reported a history of substance abuse (which influences physiological reactivity and can produce biased cortisol measurement; e.g., Young & Nolen-Hoeksema, 2001), six were excluded because the intra-assay coefficient of variation (i.e., an index of unreliability) was 20% or higher and the two duplicates of their saliva samples (from the same tube) had an absolute difference larger than .02 μg/dL (Granger et al., 2012), and six were excluded because they provided outlier scores on the manipulation check items (i.e., they rated their experience as fair yet were in the unfair condition).[1] Of the 68 in the final sample, 48 (70%) were female, 32 (47%) were currently employed, their average age was 21.0 years ($SD = 3.0$), and the majority were either Caucasian (53%) or African American (23%). Of those who were employed, the average number of hours worked per week was 24.3 ($SD = 8.8$). Chi-square tests and independent-sample t tests indicated that there were no significant differences between the 18 excluded participants and the final sample of 68 participants in terms of the aforementioned demographic characteristics.

Procedure

People who were interested in participating were instructed to contact a trained research assistant for a screening process to determine their eligibility. Only those who were not currently taking immunosuppressive medications (e.g., cyclosporine) were eligible to participate, which is recommended for research involving cortisol (Kudielka, Gierens, Hellhammer, Wüst, & Schlotz, 2012). The research assistant scheduled individual appointments for all eligible participants and provided instructions regarding necessary preparations for the laboratory experiment. Consistent with best practices (Nicolson, 2008; Zoccola, Quas, & Yim, 2010), participants were instructed to refrain from the following behaviors (within specific time limits) before the scheduled appointment: dental work (3 days), flossing or brushing teeth (1 hr), smoking (1 hr), eating (1 hr), consuming caffeinated drinks (3 hr), and consuming alcoholic drinks (12 hr). All appointments were scheduled between 2 p.m. and 6 p.m. to control for circadian fluctuations in cortisol levels (Thorsteinsson, James, & Gregg, 1998; Zoccola et al., 2010). Participants received extra credit in exchange for their participation in the study.

Upon arriving at the laboratory at the time of their appointment, participants were randomly assigned into either a fair or unfair interactional justice condition. Random assignment was determined via computer, and conditions were masked until it was time to deliver the manipulation. A research assistant led participants to a small individual room housed in the laboratory where the experiment would take place. Participants read over the consent form while the experimenter briefed them on the procedure (e.g., how saliva samples would be collected). After providing informed consent, the participants completed a survey containing questions about their background, including gender, age, and work experience. When finished, participants notified the research assistant, who instructed them to relax while they waited for the experimenter. Approximately 20 min after they arrived at the lab, the experimenter entered the room and collected the first (baseline) sample of unstimulated saliva from participants by having them drool into a 50-ml conical polypropylene tube. Participants were instructed to provide as much saliva as possible within 3 min (a minimum of 10 ml was required for each sample). Once the saliva sample was collected, participants completed two word puzzles (adapted from Stajkovic, Locke, Blair, & Piccolo, 2009). The interactional justice manipulation (described below) was delivered at this time. After the experimenter scored the task and reported performance feedback to participants, the participants were given two surveys to complete. One survey included an open-ended question that asked for participants' evaluations of the experimenter, which they were told would be mailed to the university's Institutional Review Board. Completed evaluations were sealed in an envelope to ensure confidentiality. The second survey contained the manipulation check items and the relational identity measure. After participants completed both surveys (approximately 25–30 min after the justice manipulations), the experimenter collected the second (test) saliva sample. After doing so, the experimenter left participants alone in the room (it was during this time that participants had the opportunity to engage in deviant behavior). Participants were left alone for approximately 20 min to relax and watch a nature video. After this rest period, the experimenter returned and collected the third (recovery) saliva sample. When finished, participants exited the small room and were debriefed.

Interactional Justice Manipulation

Based on random assignment, participants were exposed to either interactional unfairness ($n = 35$) or fairness ($n = 33$). The same study procedures described above were followed for both conditions. The only difference between the fair and unfair conditions was whether three interactional justice criteria (i.e., respect,

[1] The six outlier participants rated their experience of interactional justice similar to that of participants in the fair condition, $t(37) = 1.94$, $p > .05$, and significantly higher than that of participants in the unfair condition, $t(39) = 4.40$, $p < .001$. None of the outlier participants engaged in any deviant acts.

propriety, and justification) were violated. The fair and unfair conditions are described below and summarized in Table 1.

In the *fair condition*, the experimenter showed patience and politeness when explaining the instructions for the task. For example, the experimenter faced the participant, maintained eye contact, and smiled when speaking. In addition, the experimenter asked if participants had any questions about the task, paid close attention to what participants said, and provided reasonable explanations to any questions. After the task was completed and graded, performance feedback was provided to participants in a respectful and encouraging manner. Specifically, participants were told, "I graded both of your puzzles. You got more than 60% of the total words correct, so you earned one extra credit point. Very nice!" When entering and exiting the room, the experimenter carefully and quietly opened and closed the door.

In the *unfair condition*, the experimenter was impatient and impolite when explaining the instructions for the task. For example, the experimenter said, "Please try to finish the puzzles quickly because I really do not want to be here all day." When handing the task materials to participants, the experimenter "accidentally" dropped them on the floor and left them there for participants to pick up. The experimenter also did not face participants or maintain eye contact when speaking to them. After providing instructions, participants were not prompted for questions, and any questions or issues that participants raised were ignored. Participants were also reproached for being slow on the task: "You're not done yet? What's taking so long? Hurry up!" When delivering the feedback, participants were told: "I graded both of your puzzles. You managed to get more than 60% of the total words correct, so be happy that you get one extra credit." When entering and exiting the room, the experimenter abruptly and loudly opened and closed the door.

Measures

Cortisol. Consistent with the psychosomatic medicine literature (Granger et al., 2012; Vining, McGinley, Maksvytis, & Ho, 1983), we measured salivary (vs. blood) cortisol for two reasons. First, salivary cortisol levels represent free (unbound) cortisol, which is highly correlated with free plasma cortisol levels in the blood stream. Second, collecting saliva is easier and less invasive than collecting blood, which requires trained medical personnel. Participants' salivary cortisol levels were measured by ELISA (Salimetrics, 2008) following kit instructions for three measurement times. All saliva samples were centrifuged at 1,500 rpm at 4°C, and the supernatants were aliquotted into Eppendorf tubes and frozen at −80°C until thawed for ELISA analysis. The calibration range of the ELISA assay we used was .012 to 3.0 μg/dL and has an analytical sensitivity of <.003 μg/dL. In addition, the intra-assay coefficient of variation reported by Salimetrics (2008) for this assay is 3.65% and was 4.20% in our analysis. Salimetrics reports an inter-assay coefficient of variation as 6.41%. Based on best practices, the first (baseline) sample was collected 20 min after participants arrived at the laboratory, the second (test) sample was collected 25–30 min after the justice manipulation, and the third (recovery) sample was collected after 20 min of relaxation (Dickerson & Kemeny, 2004; Zoccola et al., 2010). These delays in collecting saliva samples were observed because cortisol activity reaches peak levels 20–40 min after stimulus onset (Nicolson, 2008). There were no differences in cortisol across the fair and unfair conditions at baseline, $t(66) = 0.34$, *ns*, or recovery, $t(66) = 0.73$, *ns*. Baseline cortisol was included as a covariate in all analyses. To correct for positive skewness in the cortisol data, we performed a natural log transformation (e.g., Weekes et al., 2008).

Deviant behavior. Four opportunities existed for participants to engage in deviant behaviors while working alone in the room following the justice manipulation and the second (test) measure of cortisol. Multiple opportunities were created because deviant behavior is a low base rate phenomenon; thus, the likelihood of participants engaging in a deviant act when presented with a single opportunity is low. Three opportunities involved theft, which is an established outcome of unfairness (e.g., Greenberg, 1993; Johnson & Lord, 2010). Before participants arrived, the lab was arranged such that 12 pens and four USB memory drives were placed within reach of participants' workstation. Whether participants took any pens or memory drives (coded as 1 = Yes and 0 = No) served as indicators of deviant behavior in the first two opportunities. The third theft opportunity involved lottery tickets. Participants were told that in addition to earning extra credit, they would also receive tickets that gave them a chance to win a prize drawing. The experimenter determined the number of tickets that participants earned (all participants earned between three and five tickets, with no difference between the fair and unfair conditions). The roll of tickets was left in the room with participants, who were told it was their responsibility to take the correct number of tickets. Whether par-

Table 1
Experimental Manipulation of Interactional Justice

Criterion	Relevant literature	Experimental manipulation
Respect	Bies & Moag (1986)	In the fair condition, the experimenter interacted with participants in a patient and polite manner (e.g., used eye contact while talking to participants, lightly closed or opened the door when entering and leaving); in the unfair condition, the experimenter interacted with participants in an impatient and impolite manner (e.g., avoided eye contact, roughly opened and closed the door).
Propriety	Bies & Moag (1986)	In the fair condition, the experimenter did not make any improper comments; in the unfair condition, the experimenter made such comments (e.g., "Please try to finish the puzzle quickly because I really do not want to be here all day," and "You're not done yet? What's taking so long? Hurry up!").
Justification	Greenberg (1993)	In the fair condition, the experimenter responded to all questions from participants with reasonable explanations (e.g., demonstrated how to complete the tasks, explained how much longer the experiment would last) and verified their understanding; in the unfair condition, the experimenter ignored all questions and concerns raised by participants.

ticipants took more tickets than instructed (coded as 1 = Yes and 0 = No) was an indicator of deviant behavior. The final opportunity to engage in deviant behavior involved disobeying instructions from the experimenter. Specifically, participants were asked to not move any chess pieces from an ongoing game on a nearby table within reach of the workstation. Whether participants moved any chess pieces (coded as 1 = Yes and 0 = No) was the final indicator of deviant behavior. We created an overall deviant behavior score by aggregating participants' scores on each of the four indicators. In total, seven participants (or 11%), all of whom were in the unfair condition, engaged in at least one deviant act.

Manipulation checks. To assess the efficacy of the manipulation, we used Colquitt's (2001) nine-item measure of interpersonal and informational justice ($\alpha = .94$). Example items include "The experimenter treated me with respect" and "The experimenter's explanations regarding the procedures were reasonable." Participants responded to the items using a 6-point Likert scale ranging from 1 (*Strongly Disagree*) to 6 (*Strongly Agree*). Participants also completed Colquitt's four-item measure of distributive justice ($\alpha = .93$) and six-item measure of procedural justice ($\alpha = .74$). Example items are "The number of extra credit points I got reflected the effort I have put into the tasks" (distributive justice) and "The rules used to determine my extra credit were applied consistently" (procedural justice). These additional items were included so we could verify that the manipulation affected interactional justice specifically and not the other types of justice.

Control variables. *Dissatisfaction with the experimenter* was measured using an open-ended question on a survey that was purportedly being conducted by the university's Institutional Review Board. The question read "How do you feel about the experimenter of the study?" Responses were independently coded by the first and second authors (where 1 = dissatisfied, 0 = neutral, and −1 = satisfied), who were blind to the experimental condition when coding responses. Initial inter-rater reliability was .96 (there were discrepancies in four cases, which the authors resolved via discussion). *Interdependent identity* was measured using the five-item relational subscale ($\alpha = .78$) of the Levels of Self-Concept Scale (Johnson, Selenta, & Lord, 2006), which has been used previously to capture state-based identity levels (e.g., Johnson, Chang, & Rosen, 2010; Johnson & Lord, 2010). An example item is "It is important to me that I uphold my commitments to significant people in my life." Participants also reported on whether they were currently taking steroid-based medication or oral contraceptives (1 = Yes and 0 = No), which impact cortisol reactivity. Controlling for steroid-based medication and oral contraceptives partials out confounding variance without reducing the generalizability of the findings (Granger et al., 2012; Kudielka et al., 2012).

Results

Manipulation Check

We assessed the efficacy of our manipulation by regressing participants' interactional justice scores on the interactional justice condition (coded as 1 = unfair and −1 = fair). Justice condition was a significant predictor of interactional fairness scores ($\beta = -.49$, $R^2 = .24$, $p < .01$) such that participants in the unfair condition rated interactional fairness lower than did those in the fair condition. We also regressed the distributive fairness and procedural fairness scores on justice condition. However, neither distributive fairness ($\beta = -.14$, $R^2 = .02$, *ns*) nor procedural fairness ($\beta = -.18$, $R^2 = .03$, *ns*) was significant. These results indicate that our manipulation successfully influenced interactional justice but not distributive or procedural justice.

Tests of the Hypotheses

Descriptive statistics and correlations among the focal variables are reported in Table 2. We used hierarchical regression to test all hypotheses and ran supplementary bootstrapping analyses for Hypotheses 3 and 4 (Preacher & Hayes, 2008). For Hypothesis 1, we regressed cortisol on control variables (steroid and contraceptive use, and baseline cortisol) in Step 1, followed by interactional unfairness in Step 2. In support of our prediction, interactional unfairness had a significant, positive effect on cortisol level ($\beta = .33$, $\Delta R^2 = .10$, $p < .05$), and the full model with all variables was significant, $F(4, 63) = 8.02$, $R^2 = .25$, $p < .01$. For Hypothesis 2, we regressed deviant behavior on the control variables (steroid and contraceptive use, and baseline cortisol) in Step 1, followed by cortisol in Step 2. In support of our prediction, cortisol had a significant, positive relation with deviant behavior ($\beta = .50$, $\Delta R^2 = .25$, $p < .01$), and the full model was significant, $F(4, 63) = 9.98$, $R^2 = .31$, $p < .01$.

For Hypothesis 3, which predicted that cortisol mediates the effects of interactional unfairness on deviant behavior, we re-

Table 2
Descriptive Statistics and Correlations Among the Focal Variables

Variable	Overall		Unfair condition		Fair condition						
	Mean	*SD*	Mean	*SD*	Mean	*SD*	1	2	3	4	5
Independent variable											
1. Interactional unfairness	0.02	1.00	1.00	0.00	−1.00	0.00	—				
Dependent variables											
2. Cortisol	0.14	0.08	0.16	0.09	0.11	0.05	.31	—			
3. Deviant behavior	0.13	0.31	0.25	0.38	0.00	0.00	.30	.50	—		
Control variables											
4. Dissatisfaction	−0.33	0.71	0.17	0.85	−0.87	0.41	.43	.09	.13	—	
5. Interdependent identity	4.61	0.42	4.56	0.46	4.67	0.39	−.18	.17	–.12	−.09	—

Note. $N = 68$. Interactional unfairness was coded as 1 = unfair ($n = 35$) and −1 = fair ($n = 33$). Unit of cortisol levels was ug/dL. Correlations with absolute values ≥.21 are statistically significant at $p < .05$.

gressed deviant behavior on interactional unfairness in Step 1, followed by cortisol and control variables (steroid and contraceptive use, and baseline cortisol) in Step 2. As shown in Table 3, the significant effect of interactional unfairness in Step 1 ($\beta = .30$, $p < .05$) became nonsignificant in Step 2 ($\beta = .16$, *ns*) after cortisol was added to the model. The result from a Sobel (1982) test revealed that the decrease in the regression weight for interactional justice from Step 1 to Step 2 was significant ($z = 2.06$, $p < .05$). Thus, interactional unfairness has a significant indirect effect on deviant behavior via cortisol. In order to estimate the size of this indirect effect, we used bootstrapping (Preacher & Hayes, 2008). As shown in Table 4 (Model 1), the indirect effect of interactional unfairness on deviant behavior via cortisol was .066, and its 95% confidence interval did not include zero (lower limit = .004; upper limit = .181). The total effect of interactional unfairness on deviant behavior was also significant (total effect = .131), but the direct effect of interactional unfairness was not (direct effect = .065). Thus, Hypothesis 3 was supported.

For Hypothesis 4, we reran the hierarchical regression and bootstrapping analyses for testing mediation but this time included dissatisfaction and interdependent identity as control variables. As shown in Table 3, cortisol remained significant ($\beta = .44$, $p < .05$) even after these variables were entered in the regression model at Step 3. The full model with all variables was significant, $F(6, 61) = 6.24$, $R^2 = .33$, $p < .05$. When we ran the bootstrapping analysis with dissatisfaction and identity as control variables, the indirect effect of cortisol was .061, and its 95% confidence interval did not include zero (lower limit = .005; upper limit = .170). Hence, Hypothesis 4 received support because the indirect effect of interactional unfairness on deviant behavior through cortisol remained significant after controlling for dissatisfaction and identity.[2]

Discussion

Experiencing unfairness is a stressful occurrence, especially during interpersonal interactions (Greenberg, 2004). Interactional unfairness is a social-evaluative threat because it signals low social esteem and acceptance to recipients (Bies, 2001). When faced with such threats, social self-preservation theory (Dickerson & Kemeny, 2004) posits that the HPA axis is activated, which releases cortisol in response. The release of cortisol is beneficial because it mobilizes the body's energy resources in order to cope with social-evaluative threats (Miller et al., 2007; Semmer et al., 2004). There are, however, downsides to cortisol activity, because it promotes impulsive and risky behaviors and it siphons away energy resources that would otherwise be used to regulate and control behavior (Montoya, Terburg, Bos, & van Honk, 2012). Thus, one consequence of cortisol activity is that it increases the likelihood of exhibiting deviant behavior. In fact, we found that cortisol activity mediated the effect of interactional unfairness on deviant behavior, which occurred incremental to established attitudinal (dissatisfaction) and self-construal (interdependent identity) effects. Below we discuss the implications of these findings.

Table 3
Effects of Interactional Unfairness on Deviant Behavior

Predictor	Deviant behavior		
	Step 1	Step 2	Step 3
Independent variable			
Interactional unfairness	.30*	.16	.14
Mediator			
Cortisol		.51*	.44*
Covariates			
Baseline cortisol		.12	.11
Steroids		−.05	−.03
Contraceptives		−.10	−.10
Dissatisfaction			.12
Interdependent identity			−.15
ΔF		8.46*	1.00
ΔR^2		.20	.04
Model F	6.94*	8.78*	6.24*
Model R^2	.09	.29	.33

Note. $N = 68$. Interactional unfairness was coded as 1 = unfair and −1 = fair. Standardized regression coefficients (βs) are reported in the table.
* $p < .05$.

Table 4
Partitioning the Total Effect of Interactional Unfairness on Deviant Behavior Into Direct Effects and Indirect Effects

Model and effect type	Effect size	95% Confidence interval	
		Lower limit	Upper limit
Model 1			
Total effect	.131*		
Direct effect (unfairness)	.065		
Indirect effect (cortisol)	.066*	.004	.181
Model 2[a]			
Total effect	.134†		
Direct effect (unfairness)	.065		
Indirect effect (cortisol)	.061*	.005	.170

Note. $N = 68$. Use of steroid-based medication, use of oral contraceptives, and baseline cortisol were included as control variables in both models.
[a] Controlling for dissatisfaction and interdependent identity.
† $p < .10$. * $p < .05$.

Theoretical and Practical Implications

To date, the majority of evidence in support of social self-preservation theory involves threats in performance domains (Dickerson & Kemeny, 2004). For example, elevated cortisol activity is observed when people give speeches or perform arithmetic tasks in the presence of others. However, as Dickerson and Kemeny (2004, p. 382) noted, "Many social-evaluative experiences do not occur within a performance context but, instead, involve negative interpersonal evaluations and/or rejection within ongoing social interactions." Our findings verify that interpersonal interactions characterized as unfair are capable of inciting activity in the HPA axis, which is consistent with the view that interpersonal stressors (e.g., conflict, verbal aggression) are potent sources of stress and produce high levels of cortisol activity (Almeida et al., 2009). Thus, the tenets of social self-preservation theory are

[2] Note that results from the bootstrapping analyses were rounded to three decimal places (instead of two) to better ascertain the size of focal effects and their statistical significance (see prior practices; e.g., Preacher & Hayes, 2004).

not restricted solely to performance contexts. For example, in work settings, interpersonal phenomena like low-quality leader–member exchange and team conflict may also elicit cortisol activity. It would be informative to compare the relative effects of performance versus interpersonal stressors on cortisol activity.

Our findings also have implications for the occupational stress literature, which highlights the central role of HPA activity when responding to work stressors (Ganster & Rosen, 2013; Kudielka & Kern, 2004; Semmer et al., 2004). Although cortisol activity has been linked to a variety of psychological and behavioral reactions such as anxiety disorders and risky decision making (e.g., Granger et al., 2012; Putman et al., 2010; Sapolsky, 2004; Soravia & de Quervain, 2012), it has yet to be demonstrated whether cortisol activity functions as a physiological pathway between work-relevant stressors and outcomes such as task performance and deviant behavior. Most studies involving objective measures of physiological health have neglected to examine behavioral consequences of experiencing stress. Our study addressed this deficiency by explicitly examining cortisol activity as an intermediate process between interactional unfairness and deviant behavior. While physiological health is an important outcome in and of itself, attention must also be paid to its downstream behavioral consequences.

Our findings also extend current justice theory and research. Recent person-centric approaches (e.g., Guo, Rupp, Weiss, & Trougakos, 2011) stress the importance of understanding how unfairness is experienced *in situ*. Although justice was traditionally examined as a between-person phenomenon, such an approach is misaligned with the actual experience of unfair events as they unfold (Guo et al., 2011). Additionally, person-centric approaches also stress the importance of considering the cognitive, affective, and bodily consequences of justice. To date, there has been considerable work delineating the cognitive consequences of unfairness, such as restoring equity in perceived input–outcome ratios (Adams, 1965), processing counterfactuals (Folger & Cropanzano, 2001), and forming fairness heuristics (Lind, 2001). There is also a growing literature on the affective consequences of unfairness (e.g., Weiss, Suckow, & Cropanzano, 1999) and strategies for coping with them (e.g., Barclay & Skarlicki, 2009). What is lacking, though, is an understanding of the bodily consequences of unfairness. Our merging of the social self-preservation and justice literatures highlights the HPA axis and specifically cortisol activity as a key physiological mechanism. Another such mechanism may be the sympathomedullary (SAM) system, which regulates fight-or-flight responses via the release of adrenaline (Chatterton, Vogelsong, Ellman, & Hudgens, 1996; Sapolsky, 2004). Activity in this system may be responsible for the observed relations of unfairness with changes in blood pressure (e.g., Xie et al., 2008). Our empirical findings lend credence to the person-centric perspective that the physiological ramifications of unfairness must be better integrated with the affective and cognitive ones.

A final theoretical implication of our findings is that some of the effects of unfairness are automatic (i.e., they occur outside awareness and control). This is possible because fairness is intertwined with basic needs and motives (e.g., Cropanzano, Byrne, Bobocel, & Rupp, 2001; Folger, 2001), which are typically regulated at subconscious levels (e.g., Johnson & Saboe, 2011; Strack & Deutsch, 2004). Thus, it has been suggested that some of the processes triggered by unfairness unfold outside people's awareness and control (Johnson & Lord, 2010; Johnson & Steinman, 2009; Skarlicki & Rupp, 2010), which would also include the physiological effects observed in this study. Similar to our recommendation that researchers must incorporate physiological mechanisms and measures into justice studies, researchers should also incorporate automatic effects and implicit measures as well. Failure to do so underestimates the total effect of unfairness. A variety of implicit measures exist that can be used to capture possible automatic effects of justice on people's affect and cognition (see the review by Uhlmann et al., 2012).

An implication of our findings for practice is that organizations must be cognizant of the physiological effects of unfairness and design interventions accordingly. Although there are interventions that help employees cope with the cognitive (e.g., Gilliland et al., 2001) and affective (e.g., Barclay & Skarlicki, 2009) consequences of unfairness, they do not directly assess physiological repercussions. What is needed, then, are stress-based interventions that help employees cope with the depleted resources owing to experiencing unfairness. For example, stress management programs centered around relaxation may help speed up the recovery process (van der Klink, Blonk, Schene, & van Dijk, 2001), and those that enhance self-efficacy may help employees accrue additional resources and counteract the effects of depletion (e.g., Jimmieson, 2000). Given the significant indirect effects we observed, any intervention that mitigates the physiological consequences of unfairness would also lessen the likelihood of deviant behavior. Interventions that restore physiological functioning and allostasis may also be necessary before affective and cognitive interventions can be effectively administered.

Limitations

In this section we discuss three limitations with the current study. First, caution should be exercised when generalizing our results to the workplace because our sample ($N = 68$) was relatively small and because we examined interpersonal exchanges between undergraduate students and an experimenter as opposed to employees and their supervisors. While our sample size was restricted due to resource and time constraints associated with collecting salivary cortisol, a retrospective power analysis (Faul et al., 2009) indicated that we had sufficient power ($>.70$) to detect effects. However, the exact magnitudes of the regression and bootstrapping estimates may not generalize (Cohen, Cohen, West, & Aiken, 2003). The extent to which results generalize is also constrained by the context of our experiment. On the one hand, participants in this study were likely less engaged in the task and less attached to the experimenter compared to employees with respect to their job and supervisor, and thus the observed effects may be stronger in the workplace. On the other hand, the greater engagement of employees may lessen the likelihood of retaliation out of fear of the consequences that deviant behavior causes in enduring work situations and relationships. Overall, we see the present study as a promising first look at the effects of unfairness on cortisol activity, but further research in different contexts and using different methods is warranted.

Second, although our experiment permitted us to draw conclusions about causal effects of unfairness on cortisol and deviant behavior, we did not directly manipulate cortisol to see what impact doing so has on deviant behavior. Stone-Romero and

Rosopa (2008) recommended that a follow-up experiment be conducted that verifies the causal effect of the mediator on the outcome. While we did not do so in this case, previous experimental findings have suggested that manipulating cortisol levels directly (e.g., via oral capsules) induces risky decision making and behaviors (e.g., Putman et al., 2010; R. van den Bos et al., 2009). Thus, we are optimistic that cortisol activity did indeed play a mediating role in the present study.

Third, although we demonstrated that cortisol activity mediated the effects of interactional unfairness on deviant behavior incremental to dissatisfaction with the experimenter and interdependent identity, there are other candidate variables that may serve as mediating mechanisms. For example, negative affective states (Spector & Fox, 2005) and disruptions in memory recall (Porath & Erez, 2007) can elicit deviant behavior in response to unfair or rude interpersonal treatment. Based on our data, then, we cannot conclude that cortisol activity has effects that are incremental to these other mechanisms. However, Dickerson and Kemeny (2004) concluded that cortisol activity operates independently from negative emotions, which suggests that the effects of cortisol on deviant behavior are unlikely to be explained by negative emotion.

Directions for Future Research and Conclusion

We highlight three important directions for future research. First, research on the physiological processes triggered by interactional unfairness should include other physiological measures in addition to cortisol activity. For example, levels of salivary alpha-amylase (another neuroendocrine biomarker) can be used to indicate responses of both the HPA system and the SAM system—the first physiological response system of stress processes (e.g., Chatterton et al., 1996). Consistent with the call for multiple indicators in examining the physiological basis of social aggression (Montoya et al., 2012), including multiple physiological indicators in future research may further the understanding of the complex physiological responses underlying the relation between interactional unfairness and deviant behavior. For example, profiles of cortisol and alpha-amylase activity may account for more variance in deviance than either one alone.

Second, consistent with the person-centric approach to organizational justice (Guo et al., 2011), future research should examine both short-term and long-term effects of unfairness on cortisol activity by using event-contingent or interval-contingent experience sampling approaches (Reis & Gable, 2000). Such research would build upon the effects of unfairness on acute cortisol activity and short-term deviant behavior observed in this study. It is possible that unfairness recipients' acute and chronic cortisol activity may have differential effects on patterns of short-term versus long-term deviant behaviors. Using experience sampling approaches that document physiological reactions to unfairness outside of the lab may provide more accurate estimates of effect sizes. As Dickerson and Kemeny (2004, p. 382) noted, a "social evaluative threat in an experimental setting is not nearly as distressing as it is in real life, where negative evaluations can have profound, long-lasting consequences." Another advantage of such experience sampling approaches is the ability to track changing patterns of cortisol reactivity and recovery during chronic exposure to unfairness. As noted by Ganster and Rosen (2013) in their review, differences in recovery rates may account for significant variance in physiological and psychological health outcomes.

Lastly, it would be informative to examine the effects of multiple types of unfairness on physiological responses. Albeit limited, evidence to date has suggested that the effects of different types of unfairness may have different physiological underpinnings. In support of this idea, it has been found that fairness and unfairness are processed in unique brain regions (Tabibnia, Sapute, & Lieberman, 2008), as are distributive fairness information and procedural fairness information (Dulebohn, Conlon, Sarinopoulos, Davison, & McNamara, 2009). Perhaps procedural unfairness has stronger effects on physiological systems that regulate long-term responses to stress, given the enduring nature of entity-based procedures. Distributive and interactional unfairness, in contrast, are tied more directly to specific events, which may translate into stronger effects on short-term systems that regulate acute stress. Examining multiple types of unfairness simultaneously would also enable researchers to better gauge their relative effects on physiological functioning.

In conclusion, our experimental study provides initial evidence that interactional unfairness affects cortisol activity within the HPA axis. This finding is noteworthy because HPA activity is the primary contributor to stress-related diseases (McEwen & Stellar, 1993; Miller et al., 2007). It is also noteworthy because unfairness had a significant indirect effect on deviant behavior through cortisol activity. Our study underscores the importance of examining the physiological processes that underlie interactional unfairness and the downstream behavioral consequences of those processes. We hope our study helps spark additional research on this topic, which is sorely needed (e.g., Greenberg, 2004; Robbins et al., 2012).

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