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Stigma Consciousness Modulates Cortisol Reactivity to Social Stress in Women

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Abstract

The aim of the current study was to examine whether stigma consciousness shapes cortisol responses to social stress among women in the lab. Undergraduate women ($N = 45$) completed background measures and then participated in a public speaking task, with assessments of cortisol prior to the stressor as well as 20- and 40-minutes post stressor onset. Results from multilevel models revealed that women higher in stigma consciousness evidenced blunted cortisol reactivity following social stress across the study session compared to women lower in stigma consciousness. This interaction was robust to adjustment for a number of covariates, including demographic (e.g., age), physiological (e.g., menstrual cycle) and psychological (e.g., depressive symptomatology) factors. Potential explanations for observed cortisol patterns are discussed, including hypo-reactivity of the hypothalamic-pituitary-adrenal (HPA) axis and elevated anticipatory stress. To conclude, implications for health disparities research are considered.

Keywords

stigma consciousness; women; HPA axis; cortisol reactivity

Intergroup interactions are fraught with potential difficulties (Stephan, 2014; Tajfel, 1982), especially in the presence of status discrepancy. Accordingly, researchers have framed interactions between members of different social groups via stress and coping models (Miller & Kaiser, 2001; Trawalter, Richeson, & Shelton, 2009). For members of lower status groups, threat of devaluation can loom large, taxing psychological and physiological resources (Branscombe, Ellemers, Spears, & Doosje, 1999; Derks, Inzlicht, & Kang, 2008; Steele, Spencer, & Aronson, 2002). Crucially, whether or not one expects to be devalued in intergroup interactions differs substantially from person to person, with *stigma consciousness* referring to the extent to which people expect to be discriminated against or stereotyped based on a devalued identity (Pinel, 1999). One way stigma consciousness may contribute to entrenched health disparities between members of higher and lower status

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groups is by shaping physiological responses to social stress (particularly resulting from intergroup interactions; Major, Mendes, & Dovidio, 2013). The aim of the current work was to examine the influence of stigma consciousness on one specific physiological system, the hypothalamic-pituitary-adrenal (HPA) axis.

Stigma Consciousness among Women

Because individuals derive self-worth from the social groups to which they belong (Tajfel & Turner, 1979), threat to these identities can be damaging for health and well-being (Pascoe & Richman, 2009). Consequences of such threat differ depending upon group status, with more deleterious effects for members of lower status groups (e.g., Scheepers & Ellemers, 2005; Schmitt, Branscombe, Kobrynowicz, & Owen, 2002). For example, social identity threat has been linked to increased psychological distress (e.g., Bombay, Matheson, & Anisman, 2014), elevated cardiovascular reactivity (e.g., Derks, Scheepers, Van Laar, & Ellemers, 2011; Sawyer, Major, Casad, Townsend, & Mendes, 2012) and depleted self-control (e.g., Inzlicht & Kang, 2010; Inzlicht, McKay, & Aronson, 2006). Consequently, members of lower status groups are often vigilant and sensitive to facets of the social environment that might convey devaluation (Kaiser, Vick, & Major, 2006; Murphy, Steele, & Gross, 2007; Steele et al., 2002).

The construct of stigma consciousness was developed in order to explain differences in the degree to which one is chronically self-conscious of a stigmatized social status (Brown & Pinel, 2003). While past research has examined stigma consciousness among members of diverse groups, including sexual minorities and racial minorities (e.g., Pinel, 1999), much of the research in this area has focused on women. Among women, stigma consciousness is associated with greater perceptions of discrimination at the personal and group level as well as acknowledgment of the ongoing prevalence of sexism in modern society (Pinel, 1999). Women higher in stigma consciousness are also more likely to attribute negative performance feedback from men to discrimination (Pinel, 2004). Women higher in stigma consciousness have also been shown to be more psychologically vulnerable to social identity threat (e.g., stereotype threat; Brown & Pinel, 2003). Stigma consciousness may be adaptive in that it affords the opportunity for accurate classification of negative treatment, but ongoing concerns over prejudice and discrimination may exacerbate recurring stress related to intergroup interactions for members of devalued groups (Miller, 2006).

Stigma Consciousness and the HPA Axis

The HPA axis is one physiological system that is sensitive to environmental stressors, including social stressors (Dickerson & Kemeny, 2004). Frequent activation of this axis in response to stress may lead to systematic modifications in its operation (Miller, Chen, & Zhou, 2007). In turn, these changes in HPA axis functioning may be implicated in the development of chronic physical health problems (Lupien, McEwen, Gunnar, & Heim, 2009; Miller, Chen, & Parker, 2011). A number of scholars have thus included the HPA axis as a target system in the production of health disparities between members of dominant and devalued groups (e.g., Major et al., 2013; Myers, 2009).

To date, no studies of which we are aware have investigated the influence of stigma consciousness on HPA axis reactivity to social stress. One prior study (Tomiyama et al., 2014) examined associations between weight-based stigma consciousness and diurnal salivary cortisol profiles, including total daily secretion, awakening response and diurnal slope. The researchers also measured serum cortisol from fasting morning blood samples. Weight-based stigma consciousness was not associated with any of the salivary cortisol measures, but it was associated with elevated morning serum cortisol. This finding supports increased HPA axis activity for individuals higher in stigma consciousness, but it does not specifically address the issue of HPA axis stress reactivity, the focus of the current research.

The Current Study

The current study examined effects of stigma consciousness on HPA axis reactivity to social stress among women. As mentioned previously, women have been a focal group in past research on stigma consciousness (Pinel, 1999) and sexism has been shown to have pernicious effects on women's physical health (Molix, 2014). Furthermore, within-sex comparisons are important due to differences in patterns of HPA axis reactivity between sexes (Kudielka & Kirschbaum, 2005). Overall, our major aim was to examine whether stigma consciousness would be associated with a pattern of hyper- or hypo-reactivity of the HPA axis following social stress in women.

Method

Participants

Forty-five undergraduate women from a large private university in the Southern United States participated in the current study for partial course credit. Participants were 19.33 years of age on average ($SD = 1.24$) and identified as Caucasian (81%), multi-racial (7%), Asian American (5%), Latina (5%) and African American (2%). All potential participants were informed that the study involved an online survey followed by a laboratory session during which they would be asked to engage in a public speaking task. Women diagnosed with an endocrine disorder were not eligible to participate in the current study.

Procedure and Measures

At least 24 hours prior to participation in the laboratory session, participants were required to log onto a secure website (*Survey Monkey*) in order to complete a background survey. This survey included the Stigma Consciousness Questionnaire, which has demonstrated adequate internal consistency in past work with women, $\alpha = .72$ (Pinel, 1999), as well as in the present study, $\alpha = .76$. Depressive symptomatology was also assessed as a covariate via the widely-used Center for Epidemiologic Studies Depression Scale ($\alpha = .85$; Radloff, 1977). Upon completion of this survey, participants were reminded to refrain from consuming substances known to affect cortisol levels (e.g., alcohol, caffeine) the morning of scheduled sessions. All sessions were scheduled between 13:00 and 17:00 in order to control for the diurnal rhythm of cortisol, which reaches its waking nadir in the afternoon.

When participants arrived at the laboratory, they were greeted by a female research assistant and given 20 minutes to acclimate to the laboratory space. Participants also completed

demographic measures and reported information relevant to cortisol analyses, including age, body mass index (BMI; calculated from self-reported height and weight according to the standard formula), oral contraceptive usage, other prescription medication usage and smoking status. After 20 minutes had elapsed, the first saliva sample was collected. All saliva samples were collected via passive drool. Participants were asked to expectorate approximately 1mL of saliva through a small plastic straw into an IBL SaliCap collection device (Hamburg, Germany).

After the first saliva sample had been collected, participants began the social stress task, which was composed of the speech paradigm from the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). Participants were informed that they would have 10 minutes to prepare a 5-minute speech on why they would make a good candidate for a desirable job, which would subsequently be delivered in front of two trained evaluators as well as videotaped. In order to vary cues related to social identity threat, half of the participants were randomly assigned to speak about their qualifications for a job in the domains of science, technology or engineering in front of two male evaluators and half were assigned to speak about a job in the domains of English, arts or communications in front of a male and female evaluator. All evaluators were trained to express a neutral but evaluative demeanor, both verbally and non-verbally, throughout the course of the speech.

After participants delivered their speeches, they provided a second saliva sample (20-minutes post stressor onset) and completed a measure of state-level stress (State-Trait Anxiety Inventory, $\alpha = .87$; Spielberger, Gorsuch, & Lushene, 1983). Finally, 40-minutes post stressor onset, participants provided a final saliva sample. All saliva samples were frozen at -20°C during each session and immediately transferred to storage at -80°C upon session completion. Samples were shipped in single batch to Brandeis University where cortisol assays were performed in duplicate via commercially available enzyme-linked immunosorbent assay (ELISA). Intra- and inter-assay coefficients of variance were both acceptable (5.64% and 9.98%, respectively).

Analyses

One participant's saliva samples generated cortisol values greater than detectable range (i.e., $> 100 \text{ nmol/L}$) at all three time points and therefore her cortisol data were missing for the following analyses. As salivary cortisol typically demonstrates a positively skewed distribution (Schultheiss, Schiepe, & Rawolle, 2012), consistent with prior researchers we utilized a natural log transformation for cortisol values at each time point. Results with logged cortisol values are presented in the following analyses. Data were analyzed via multilevel models using the lme4 package (Bates, Maechler, Bolker, & Walker, 2015) available within R (R Core Team, 2013). In order to calculate the intra-class correlation coefficient (ICC), we began by estimating an unconditional model with only a random intercept. Next, we added a fixed effect of time at Level 1 to examine the overall trend in cortisol across the study session. To address the primary aim of the study, we created multilevel models with cortisol as the dependent variable and time as the independent variable at Level 1 while at Level 2 we entered stigma consciousness predicting both the Level 1 intercept and the Level 1 slope. Finally, to examine the robustness of the effects of

stigma consciousness on cortisol, we added a number of covariates to the previous model in a fully adjusted model which is shown below.

Level 1

$$\text{Cortisol}_{ti} = \pi_{0i} + \pi_{1i}^*(\text{Time}_{ti}) + e_{ti}$$

Level 2

$$\pi_{0i} = \beta_{00} + \beta_{01}^*(\text{Age}_i) + \beta_{02}^*(\text{BMI}_i) + \beta_{03}^*(\text{Oral Contraceptives}_i) + \beta_{04}^*(\text{Smoking}_i) + \beta_{05}^*(\text{Menstrual Cycle}_i) + \beta_{06}^*(\text{Medication Usage}_i) + \beta_{07}^*(\text{Depressive Symptomatology}_i) + \beta_{08}^*(\text{Stigma Consciousness}_i) + u_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}^*(\text{Age}_i) + \beta_{12}^*(\text{BMI}_i) + \beta_{13}^*(\text{Oral Contraceptives}_i) + \beta_{14}^*(\text{Smoking}_i) + \beta_{15}^*(\text{Menstrual Cycle}_i) + \beta_{16}^*(\text{Medication Usage}_i) + \beta_{17}^*(\text{Depressive Symptomatology}_i) + \beta_{18}^*(\text{Stigma Consciousness}_i) + u_{1i}$$

Results

Adaptation of the speech task designed to vary identity threat cues (i.e., panel composition and job categories) was not significantly associated with cortisol values at any of the three time points. Moreover, speech task version did not have significant main effects or interactions in any of the following models and including speech task version did not change the significance or pattern of other variables in the final model (shown in analyses presented in the online supplemental material). Therefore, speech task version is not considered further here. Means and standard deviations as well as bivariate correlations among each of the key variables are displayed in Table 1. Stigma consciousness was not correlated with state-level stress for women in the current study, $r(45) = -.04$, $p = .79$.

Results from multilevel models are shown in Table 2. In the unconditional model, the ICC was .58, suggesting that over half of the variance in cortisol levels could be attributed to between-subject factors at Level 2. In the second model, including only a fixed effect of time, we found that on average cortisol levels did increase significantly across the study session, $\pi_{1i} = .08$, $t(87) = 2.09$, $p = .04$, consistent with past work on the TSST as a social stressor capable of eliciting an HPA axis response (Dickerson & Kemeny, 2004). The next model included an effect of stigma consciousness at Level 2 on both the intercept and slope of time predicting cortisol. Stigma consciousness was not significantly associated with the intercept (i.e., cortisol levels at the initial time point), $\beta_{01} = .16$, $t(42) = 1.59$, $p = .12$, but did significantly interact with time, $\beta_{11} = -.14$, $t(42) = -2.29$, $p = .03$. This represents a shallower slope among women relatively higher in stigma consciousness. The pattern of this interaction is illustrated in Figure 1, with mean raw cortisol scores at each time point plotted separately for women above and below the median value of stigma consciousness. As displayed in Figure 1, for women relatively lower in stigma consciousness, cortisol levels increased steadily across the study session. However, for women relatively higher in stigma consciousness, cortisol levels remained relatively flat across the study session.

Finally, a fully adjusted model was computed to test the robustness of the effects of stigma consciousness. In this model, including all covariates, the interaction between stigma consciousness and time remained statistically significant, $\beta_{18} = -.12$, $t(35) = -2.04$, $p < .05$.

There was also some evidence of a marginally significant association between stigma consciousness and the cortisol intercept in the fully adjusted model, $\beta_{08} = .17$, $t(35) = 1.77$, $p = .09$. This could indicate that women higher in stigma consciousness showed increased cortisol levels prior to the social stressor. However, this effect was only marginally significant and was not consistent across models. There was a significant association between depressive symptomatology and increased initial cortisol levels, $\beta_{07} = .59$, $t(35) = 2.49$, $p = .02$, as well as an interaction between depressive symptomatology and time, $\beta_{17} = -.42$, $t(35) = -2.97$, $p = .005$, that mirrored the interaction between stigma consciousness and time. Importantly, both interactions were significant in the fully adjusted model, suggesting independent effects on cortisol levels (i.e., the effects of stigma consciousness did not appear to be explained by depressive symptomatology).

Discussion

The current research revealed differences in HPA axis reactivity for women relatively higher and lower in stigma consciousness. Specifically, across statistical models, stigma consciousness was associated with blunted cortisol reactivity to social stress among women. Traditionally, researchers interested in social stigma and the HPA axis have speculated that stress resulting from stigmatized group membership may lead to hyper-reactivity. Although counter to these speculations, hypo-reactivity of the HPA axis following social stress among women higher in stigma consciousness is consistent with findings from work on other constructs related to social identity threat, including perceived discrimination (Weinstein et al., 2013) and exposure to structural stigma (Hatzenbuehler & McLaughlin, 2014), as well as other forms of stress and adversity (e.g., Carpenter et al., 2011; Elzinga et al., 2008; Lovallo et al., 2012).

Why stigma consciousness might lead to hypo-reactivity of the HPA axis following social stress remains an open question. One possible explanation is that vulnerability to social stress may lead to an up-regulation of the sympathetic-adrenal-medullary (SAM) axis and a complementary down-regulation of the HPA axis (Andrews, Ali, & Pruessner, 2013). Although we did not examine this hypothesis directly, some work has shown increased cardiovascular activation following social identity threat (e.g., Derks et al., 2011; Sawyer et al., 2012). Future research on this hypothesis may benefit from assessments of salivary α -amylase, a relatively non-invasive neuroendocrine marker of SAM activation. Another potential explanation for hypo-reactivity of the HPA axis is that social stressors evaluated as identity-threatening may be processed differently from other stressors at a neurological level. For example, in one study (Masten, Telzer, & Eisenberger, 2011), black American participants who attributed social exclusion to discrimination showed increased activity in the rostral anterior cingulate cortex (rACC), a region that is involved in the regulation of threat responses and provides inhibitory feedback to the HPA axis.

An alternative explanation for the cortisol patterns observed in the current study is that women higher in stigma consciousness experienced an elevation in anticipatory stress going into the lab session (Engert et al., 2013). Rather than hypo-reactivity of the HPA axis, these women may have demonstrated an earlier cortisol response that reached an upper threshold and then maintained throughout the social stress task. Such an explanation would be

consistent with past work (e.g., Turan et al., 2015) demonstrating sensitization to social stress (i.e., women higher in stigma consciousness may be sensitized to potentially stressful social interactions). While this is plausible, there was only marginally significant evidence for elevated initial cortisol levels among women higher in stigma consciousness in the fully adjusted model. Furthermore, there was some indication from the correlation matrix and Figure 1 that cortisol levels may have reversed by the end of the study, with higher levels at the final time point for women lower in stigma consciousness compared to women higher in stigma consciousness. Nonetheless, future research should seek to clarify whether stigma consciousness is indeed associated with hypo-reactivity of the HPA axis or rather heightened anticipatory stress.

Limitations and Conclusion

The current report describes the results of a relatively small laboratory study investigating an important and under-examined topic, the effects of stigma consciousness on physiological reactivity to social stress. We hope that this work stimulates greater research on this topic, yet the results of the current study should be understood in the context of the relatively small sample. This study capitalized on a repeated-measures design and the analytic strategy maintained statistical power by estimating unique intercepts and slopes for each individual participant. The relative homogeneity of the current sample is another potential limitation. We recruited college-age women, most of whom identified as Caucasian and all of whom attended a university with a relatively affluent student body in the Southern United States. These women's experiences of sexism may be limited compared to a more racially and socioeconomically diverse sample. We also adapted a well-used and validated social stress paradigm, the TSST, in order to attempt to vary cues to social identity threat. These variations were not significantly associated with outcomes in the models tested here but future research may employ other paradigms. In adapting the TSST, the mental arithmetic task was removed because it involves a negatively stereotyped domain for women. Including the mental arithmetic task may amplify the effects of stigma consciousness on cortisol reactivity but it is also possible that resultant stereotype threat could cause all women to respond physiologically in a similar manner. This remains an open question for future research.

Overall, this work contributes to a growing number of studies on social stigma, and social stress research more broadly, that are pointing toward an important role for hypo-reactivity of the HPA axis. Stigma consciousness could plausibly contribute to entrenched disparities in health for women and perhaps members of other devalued groups via effects on cortisol reactivity to social stress. Of course, the current study is preliminary and further research should be conducted in order to confirm and extend these findings. Pathways from social stress to impaired health and well-being are intricate and complex. The current work represents one attempt to understand a specific pathway through dysregulation of the HPA axis.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Mean untransformed cortisol levels and standard errors across the study session for women higher and lower in stigma consciousness. *Note.* Time points are prior to social stressor, 20-minutes and 40-minutes post stressor onset. A median split was used to group women as higher or lower in stigma consciousness.

Table 1

Descriptive Statistics and Inter-correlations among Key Variables

Measure	M/n	SD/%	Range	1	2	3	4	5	6	7	8	9	10	11
1. Age	19.33	1.24	18–24	--										
2. BMI	21.99	4.77	16.64–46.98	.21	--									
3. Oral Contraceptives	29	63%	--	.16	-.22	--								
4. Smoking	3	7%	--	.29 [†]	-.09	.01	--							
5. Menstrual Cycle	16.96	11.74	0–38	-.05	.26 [†]	-.09	.10	--						
6. Medication Usage	12	26%	--	-.08	.02	.13	.04	.01	--					
7. Depressive Symptomatology	10.78	6.70	1–26	-.27 [†]	.18	.06	.06	.32 [*]	.06	--				
8. Stigma Consciousness	2.74	.78	1.1–4.5	-.03	-.24	.06	.15	-.12	.09	.09	--			
9. Cortisol (Time 1)	2.30	.49	1.31–3.15	-.19	.12	.01	-.25	.18	.17	.39 ^{**}	.22	--		
10. Cortisol (Time 2)	2.37	.52	1.29–3.63	.07	.20	.03	-.16	.21	.21	.25 [†]	.09	.80 ^{***}	--	
11. Cortisol (Time 3)	2.47	.67	.49–3.78	.24 [*]	.20	.14	-.22	.07	.19	-.20	-.17	.40 ^{**}	.64 ^{***}	--

Note. $N = 45$, $n = 44$ for correlations with cortisol values. BMI = Body mass index. Cortisol values were log-transformed. Dummy codes were assigned to oral contraceptives (0 = *no oral contraceptive usage*, 1 = *oral contraceptive usage*), smoking (0 = *nonsmoker*, 1 = *smoker*) and medication usage (0 = *no other prescription medication usage*, 1 = *other prescription medication usage*). Range represents observed range.

[†] $p < .10$,

^{*} $p < .05$,

^{**} $p < .01$,

^{***} $p < .001$.

Table 2
Results from Multilevel Models Predicting Cortisol Levels across Study Session

	b	SE	t	p		b	SE	t	p
Unconditional Model					Fully Adjusted Model				
<i>Intercept</i>	2.38	.07	32.78	< .001	Level 1				
Time Only Model					<i>Intercept</i>	2.21	.17	13.30	< .001
<i>Intercept</i>	2.30	.08	27.88	< .001	<i>Time</i>	-.001	.10	-.01	.99
<i>Time</i>	.08	.04	2.09	.04	Level 2				
Stigma Consciousness Model					<i>Age</i>	.011	.068	.17	.87
Level 1					<i>BMI</i>	.004	.018	.25	.80
<i>Intercept</i>	2.29	.08	30.32	< .001	<i>Oral Contraceptives</i>	-.007	.154	-.05	.96
<i>Time</i>	.08	.05	1.76	.09	<i>Smoking</i>	-.57	.31	-1.88	.07
Level 2					<i>Menstrual Cycle</i>	.005	.007	.80	.43
<i>Stigma Consciousness</i>	.16	.10	1.59	.12	<i>Medication Usage</i>	.13	.17	.81	.43
<i>Stigma Consciousness*Time</i>	-.14	.06	-2.29	.03	<i>Depressive Symptomatology</i>	.59	.24	2.49	.02
					<i>Stigma Consciousness</i>	.17	.10	1.77	.09
					<i>Age*Time</i>	.07	.04	1.69	.10
					<i>BMI*Time</i>	.004	.010	.39	.70
					<i>Oral Contraceptives*Time</i>	.064	.091	.70	.49
					<i>Smoking*Time</i>	-.08	.18	-.42	.68
					<i>Menstrual Cycle*Time</i>	.001	.004	.33	.74
					<i>Medication Usage*Time</i>	.10	.10	1.04	.30
					<i>Depressive Symptomatology*Time</i>	-.42	.14	-2.97	.005
					<i>Stigma Consciousness*Time</i>	-.12	.06	-2.04	< .05

Note. $n = 44$. BMI = Body mass index. Cortisol values were log-transformed. Dummy codes were assigned to oral contraceptives (0 = *no oral contraceptive usage*, 1 = *oral contraceptive usage*), smoking (0 = *nonsmoker*, 1 = *smoker*) and medication usage (0 = *no other prescription medication usage*, 1 = *other prescription medication usage*). Age, BMI, depressive symptomatology and stigma consciousness were grand mean-centered in the fully adjusted model. For the unconditional model, ICC = .58.