Embodied Stereotype Threat:

Exploring brain and body mechanisms underlying performance impairments

Wendy Berry Mendes

Jeremy Jamieson

Harvard University

To appear in:

Stereotype Threat: Theory, Process, and Application.

M. Inzlicht and T. Schmader (Eds).

Correspondence can be directed to:

Wendy Berry Mendes

UC San Francisco

LPPI/Parnassus Avenue

San Francisco, CA 94143

Email: wendy.mendes@ucsf.edu

Abstract

In this chapter we explore brain and body mechanisms that link the experience of stereotype threat to changes in cognitive and behavioral performance. We begin by identifying a model of causal sequences of stereotype threat: 1) psychological states associated with stereotype threat, 2) neurobiological responses triggered by these psychological states, and 3) cognitive and behavioral outcomes that are influenced by the neurobiological states. We explore this theoretical path analysis throughout the chapter focusing on two broad psychological states often implicated in stereotype processes: stress arousal and vigilance. To explore stress arousal as an explanation for stereotype threat performance effects we highlight the biology underlying stress systems, stress typologies, and temporal trajectories of stress responses. We highlight how these neurobiological changes can influence cognitive and behavioral outcomes, and review existing stereotype threat research that explores these neurobiological responses. We then examine the broad category of vigilance in stereotype threat processes, and again highlight extant stereotype threat literature exploring neurobiological changes associated with vigilance. The intent of the chapter is to provide a neurobiological framework to assist stereotype threat researchers in identifying possible brain and body mechanisms that may be directly or indirectly implicated in performance changes engendered by stereotype threat.

Embodied Stereotype Threat:

Exploring brain and body mechanisms underlying performance impairments

Performance changes brought on by stereotype threat appear to be reliable and robust across many domains. Indeed so reliable are stereotype threat effects on performance that much of the current research on this topic focuses on *why* it happens rather than *if* or *when.* In the search for the answer (or answers) to how negative stereotypes influence performance changes, researchers have identified several candidate mechanisms. In an influential review paper, Schmader, Johns, and Forbes (2008) present a process model in which they implicate three mechanisms that may underlie impairments in working memory brought on by stereotype threat: stress arousal, vigilance, and self-regulation. In this chapter we capitalize on two of these mechanisms – stress arousal and vigilance – to explore how knowledge of negative stereotypes affects performance via brain and bodily mechanisms. That is, we delve under the skin to bring to light biological and neuroscience evidence that illuminates if, when, and how the body and brain responses can be viewed as direct or indirect causal effects on performance changes as a result of stereotype threat.

We explore these mechanisms by first describing what is known about how the underlying neurobiology is initiated by psychological states, which allows us to identify when we might expect a neurobiological response to be implicated in stereotype threat performance effects. We then examine the empirical evidence linking these brain- and bodily-responses to cognitive and behavioral outcomes, which sharpens our understanding of which types of tasks might be more susceptible to performance decrements and what the time course of the impairment might be. We then narrow our attention to stereotype threat research and review the

extant literature with a focus on interpreting these data given what we know about the underlying biology. We end with speculations on future directions and possible interventions, targeted at mind-body effects, to reduce performance impairments that follow from stereotype threat.

The overall model that we explore in this chapter in presented in Figure 1 and is referenced throughout. The figure presents three columns: psychological states, neurobiological responses, and performance outcomes. We present this figure as a theoretical path analysis that examines putative relationships between psychology and neurobiology and then between neurobiology and cognitive and physical outcomes. The arrows connecting the columns represent the amount of empirical data supporting the relationships with thicker arrows indicating a larger and more reliable body of work based on a qualitative review of the literature.

What might be most striking about Figure 1 is the number and strength of arrows connecting psychological states with neurobiological changes (i.e., left side of Figure 1) relative to the connection between the neurobiological changes and the performance outcomes (i.e., right side of Figure 1). Related to this, the theoretical path analysis suggests neurobiological responses as mediators linking psychological states of stereotype threat and performance outcomes, however, as we review the literature we will see that there is a paucity of studies that link neurobiological responses to performance outcomes and even fewer studies demonstrating neurobiological mediation. We discuss why this may be the case and suggest studies to explore possible direct and indirect neurobiological mechanisms involved in stereotype processes.

It also is important to note that in due to space constraints there are several potentially important factors not discussed in this chapter. For example, Figure 1 presents the stereotype path as initiating with psychological states which then triggers neurobiological responses. This assumes that psychology always precedes biology, and thus ignores the importance of individual

differences in the neurobiological milieu that may make one more likely to experience a psychological state. We touch on this point a bit when we discuss stereotype threat as a chronic stressor which may over time result in dysregulated HPA functioning, but the importance of individual differences in neurobiology and how that influences stereotype processes is worthy of its own chapter and is not explored in depth here.

Stress Arousal

Possibly the first thing that comes to mind when one thinks about stress arousal and academic performance is *test anxiety* – the idea that when facing an important test one's excessive worry over performance can instigate a cascade of bodily changes that can directly undermine performance. Stereotype threat theory suggests that this "arousal" or "anxiety" might be behind performance impairment (Ben-Zeev, Fein, & Inzlicht, 2005; O'Brien & Crandall, 2003), but can neurobiological responses be *directly* implicated in performance impairments? And if so, which ones? We attempt to answer these questions by examining the role of the brain and body in terms of how *stress* can be ignited by stereotype threat and then how the downstream brain and body responses may influence performance.

To explore how stress arousal might be implicated in stereotype threat effects we begin by reviewing the biology underlying stress responses and the psychological antecedents associated with "stress arousal." We then underscore two critical distinctions of stress arousal: 1) acute versus chronic, and 2) adaptive versus maladaptive. We highlight that not all stress response are created equal, and by drawing these distinctions we can derive more specific hypotheses regarding the effects of *stress arousal* as a potential explanation for performance impairments brought on by stereotype threat.

Stress systems

There are two primary stress systems in the body: the *sympathetic adrenal medullary* (SAM) and *hypothalamic pituitary adrenal cortical* (HPA) axes. At the risk of over-simplification, one can think of the SAM system as activating during fight-flight situations, whereas the HPA system is more conservative and requires more intense affective or physical states to disrupt its diurnal cycle. When the SAM system is activated, epinephrine is released from the adrenal medulla which contributes to several changes in the body such as increasing heart rate, dilating pupils, and inhibiting the gastrointestinal tract. HPA activation is initiated in the hypothalamus which releases corticotropin releasing hormone (CRH), triggering the anterior pituitary to release adrenocorticotropin hormone (ACTH), which then travels to the adrenal cortex and stimulates the adrenal cortex to release hormones, especially cortisol from the *zona fasciculata*.

Acute versus chronic stress arousal

The distinctions in the time course and intensity required to activate these systems are critical to both understanding how stereotype threat operates in people's daily lives as well as to how scientists approach studying stereotype threat processes in the lab. When considering how stress arousal may explain stereotype threat effects on performance in one's daily life it is useful to first draw the distinction between environmental triggers that are *acute* versus *chronic.* For example, an acute environmental trigger would be one that occurs with little pre-warning such as being a student who is called upon in a classroom (i.e., cold-calling). In this split second the SAM system could respond with a cascade of physiological changes that could impair (or enhance) cognitive performance, but implicating the full cascade of HPA axis stress responses with cortisol as the end product is not likely to be a candidate for understanding performance outcomes because of the time course of HPA activation. Thus, acute situations are more likely to

be mediated by changes in SAM, especially when they occur with little warning as in the case described here.

Stereotype threat as a chronic stressor would look very different. For example, consider a college engineering course that is well into the semester and comprises primarily male students, is taught by a male professor, and the classroom walls are lined with pictures of famous engineers, all of whom happen to be male. For a female engineering student in the course, especially one who is sensitive to these stereotype threat triggers, each class session might result in incrementally more "stress," which would accumulate over time. She might wake up the morning of the class feeling anxious, be preoccupied with thoughts about her performance on the way to class, and sit in class thinking that at any moment she will be called upon and be in jeopardy of being negatively evaluated by the professor and the other class members. This scenario describes a *chronic stressor* and to the extent that the environment was perceived as socially evaluative and *threatening* we would expect over-activation of the HPA axis. Evidence of *hyper-responsiveness* of the HPA would be indicated by higher waking cortisol the day of the engineering class, less habituation of HPA responses to the classroom, and slower recovery following the end of the class. Interestingly, if this environment repeated over years, rather than months, eventually the HPA responses would likely be dysregulated and possibly show *hyporesponsiveness*, or a flattened diurnal cycle. It is interesting to speculate that hyporesponsiveness may be associated with the psychological disengagement in stereotyped academic domains seen among stigmatized group members (Davies, Spencer, Quinn, & Gerhardstein, 2002).

From a chronic stress perspective we would anticipate that an overactive HPA response would influence low affinity receptors in the hippocampus and begin to impair memory (Figure

1). For individuals who perceive their environment as an unremitting source of stereotype threat (e.g., the female engineering student in our example above) they may develop an overactive HPA response which may impair both learning and recall of knowledge. Over time a hypo-responsive HPA might exert behavioral manifestations such as withdrawing from academic environments or an inability to rally effort associated with the domain that triggered the chronic stress.

The above section may lead the casual reader to infer that acute stress activates SAM and chronic stress activates HPA, but this would be a faulty conclusion. HPA activation most certainly can occur during acute stress, and indeed a large literature examining cortisol as an end product of acute stress states relies almost exclusively on cortisol as the primary measure of *stress* (see Dickerson & Kemeny, 2004, for a review). Our point here is that acute HPA activation as an explanation for performance impairments brought on by stereotype threat are probably more likely to occur during anticipated important, but isolated, events like a standardized test (e.g., SAT, GRE, MCAT), an important oral presentation, or a job interview.

Adaptive versus maladaptive stress responses

Independent of the acute versus chronic distinction of stress arousal, not all stress responses are created equal. Indeed, it is problematic to think of stress as a unidimensional construct that ranges from low to high with high stress interpreted as maladaptive. There are at least two problems with this conception. First this view of stress fails to acknowledge that some stress responses are benign and, indeed, part of the adaptive response required because reactivity mobilizes energy to cope with the task at hand. The second problem is that low levels of "stress arousal" may actually indicate withdrawal or disengagement from a task, which would manifest itself in low stress arousal but also poor performance. For both of these problems a detailed

understanding of stress system typologies allows us to understand both the psychological states that bring about stereotype threat, and also how neurobiological responses may contribute to performance impairments.

Challenge and threat theory. Several theories have differentiated adaptive stress from maladaptive stress (e.g., Collins & Frankenhaeuser, 1978; Dienstbier, 1989). Challenge and Threat theory (Blascovich & Mendes, 2010; Tomaka, et al., 1993), for example, integrates appraisals and psychophysiological theories and makes predictions regarding distinctions in cardiovascular reactivity resulting from appraisal processes. The basic tenets of this theory are that an individual's perceptions of how demanding a task is can be offset by their assessment of the personal and situational resources they have to meet the tasks demands (see Lazarus & Folkman, 1991). For example, imagine a student taking a final exam for an important class. That exam could vary on many dimensions such as its difficulty, its grading structure, and the number and types of questions. All of those features can be perceived of as how "demanding" the exam is, and different students will assess those demands differently, but simply how demanding the test is does not necessarily predict responses to taking the exam. Individuals also can assess their resources to complete the exam. How much did they study, do they have natural ability in this domain, do they have dispositional styles that make them more optimistic (and hence more likely to persevere on difficult questions), or were they allowed to bring in notes that have the information they need to answer some of the questions? All of these components would be considered resources. Challenge and threat theory maintains that responses to stressful situations are a combination of individuals' assessments of available resources relative to task demands, when resources are higher than demands individuals are more likely to experience *challenge*, whereas when demands exceed resources individuals experience *threat*.

Importantly, these psychological states of challenge and threat can be differentiated by the change in physiological responses that are concomitant with the experiences. Specifically, challenge states tend to be associated with greater SAM activation, and are characterized by increases in ventricular contractility, cardiac efficiency (i.e., greater cardiac output), and vasodilation in the arterioles, which provides greater blood flow to the brain and periphery. Similar to challenge states, threat states also are characterized by an increase in sympathetic activation, but in contrast to challenge, threat states consist of less efficient cardiac responses and vasoconstriction. These patterns may be critical in understanding stereotype threat processes because these physiological states can either facilitate (in challenge) or impair (in threat) performance.

Physiological striving. Like CV reactivity, neuroendocrine reactivity brought on by acute stress may also be differentiated into adaptive and maladaptive responses by examining changes in catabolic *and* anabolic hormones (Southwick, Vythilingam, & Charney, 2005; Epel, McEwen, & Ickovics, 1998). Cortisol, a catabolic hormone, has been shown to rise in response to psychological stressors that are perceived as effortful, threatening, or socially evaluative (Dickerson & Kemeny, 2004; Lovallo & Thomas, 2000). Less studied, but of growing interest, are anabolic hormones (ones that promote growth), which, among other things, can counter-regulate catabolic hormones. Anabolic hormones often indicate more adaptive coping with stressors and have been linked to physical and psychological thriving (Epel, et al., 1998). One anabolic hormone of particular interest—dehydroepiandrosterone (DHEA and DHEA(S))—is excreted by the *zona reticularis* of the adrenal cortex in response to adrenocorticotropic hormone (ACTH), and thus is often released during acute stress, presumably conferring protection from catabolic aspects of the stress response (Wolf, et al. 1997). DHEA(S) has many salutary effects,

achieved in part by the fact that it serves as a precursor to estrogen and androgens (Labrie, et al., 2000). In an experimental study, European Americans who were higher in implicit racial bias reported greater threat appraisals and showed *lower* DHEA(S) increases when socially evaluated by African American evaluators relative to European American evaluators, whereas cortisol changes did not differ as a function of implicit bias (Mendes, et al., 2007).

In sum, across various physiological systems responses have been identified that differentiate stress states. Typically the more *adaptive* stress response is associated with more approach-oriented motivation and challenge appraisals compared to the maladaptive stress response. These critical outcomes notwithstanding, our question regarding how these distinctions in stress arousal aid us in explaining performance impairments brought on by stereotype threat are most useful when we examine how these stress typologies impair (or facilitate) cognitive and physical performance.

Stress typologies influence cognition

The distinction of adaptive and maladaptive stress allow for a more nuanced understanding of how stress influences performance. For example, in Dienstbier's (1989) review of physiologically "tough" patterns, he questioned the commonly-held belief that "arousal" would be related to cognitive or behavioral performance in a curvilinear relation (similar to the Yerkes-Dodson principle). Numerous studies show strong linear relations, with no evidence of curvilinear effects, between SAM activation and cognitive and physical performance. Most typically, greater catecholamine increases from baseline are associated with better math performance among students (Dienstbier, 1989; Jamieson, Mendes, Blackstock, & Schmader, 2010) and physical performance also yields a similar finding: greater increases in catecholamines

are associated with better technical competence among military paratroopers training (Ursin, Baade, & Levine, 1978).

Profiles associated with challenge and threat responses have also been associated with different performance outcomes. Recall that challenge tends to be associated with greater sympathetic increases. In a number of experiments individuals who experienced "challenge" performed better at cognitive tasks, such as word-finding and pattern recognition tasks (e.g., Blascovich, Mendes, Hunter, & Salomon, 1999), which is consistent with the linear relationship between sympathetic nervous system activation and performance. In one recent study in which participants were randomly assigned to experience challenge *or* threat states, those in the challenge condition provided more accurate answers in an anchor-and-adjustment decision-making task, which is a task that has been linked to conscious control (Kassam, Koslov, & Mendes, 2009). Importantly, the CV responses differentiating challenge from threat *mediated* the relationship between the psychological state and the decision making outcome, implicating bodily changes brought on by challenge to be associated with improved decision-making outcomes.

In contrast, there is evidence for the inverted U-relation (Yerkes-Dodson) when considering HPA activation—specifically cortisol responses—on performance. For example, memory is improved when there are small increases in cortisol, but is impaired at higher levels of cortisol (Lovallo & Thomas, 2000). The inverted-U relation may be explained by different receptors in the hippocampus, which has high-affinity and low-affinity receptors for cortisol. At low levels of cortisol production, high-affinity receptors are activated, which can improve memory. However, at higher levels of cortisol production or when cortisol is chronically activated, the low-affinity receptors are activated, which can impair memory (Reul & de Kloet,

1985). Taken together we expect that during active tasks, SAM activation more often has a linear relationship with cognitive and behavioral performance measures whereas HPA activation shows an inverted-U between arousal and performance.

Stereotype threat studies

These biological processes provide us a framework to understanding when and how stereotype threat might influence performance, especially given the previous distinction between acute and chronic stress and adaptive versus maladaptive stress responses. For example, an adaptive response to acute stress would be characterized by a strong sympathetic response in which we would expect improved cognitive and physical performance, especially on tasks that benefit from effort and perseverance and less relevant for tasks that are retrieval based (Figure 1). Therefore, the prediction would be that if a negative stereotype is activated and this resulted in a shift to greater perceived demands relative to resources then we would expect to observe a psychological threat state. However, if resources are already high or are increased then the negative stereotype might not result in impaired performance. Thus, this theory might be useful to understand when primed negative stereotypes do not impair performance or assist in developing interventions to combat existing negative stereotypes.

Since the first conceptualization of stereotype threat, arousal/anxiety has been hypothesized to be part of the process through which performance is impaired (Steele & Aronson, 1995). However, there is little work that actually measures biological responses during performance situations associated with stereotype threat, although several research traditions implicate stress without measuring it, e.g., misattribution of arousal paradigms (Ben-Zeev, et al, 2005). We reviewed the literature to identify published studies that directly measured physiological responses associated with “stress” (Table 1. We also examine if there is any

evidence that the physiological response *mediates* the link between the psychological state of stereotype threat and performance outcomes.

One of the first studies to provide evidence that stress arousal was associated with the experience of stereotype threat examined blood pressure changes during two 5 minute blocks of the remote associates task (RAT) in which European- and African-Americans participants were randomly assigned to either a "tests are racially biased" condition or a "tests are unbiased" condition (Blascovich, Spencer, Quinn, & Steele, 2001). African-American participants assigned to the biased test condition (the stereotype threat manipulation) exhibited greater mean arterial blood pressure relative to the other three conditions. Importantly, however, race by condition did not show a robust interaction until the second block of the task. This finding is not surprising given the temporal trajectories of adaptive and maladaptive stress responses – sluggish habituation for maladaptive stress, but quick habituation for adaptive stress responses. There was no evidence, however, that the physiological response *mediated* performance effects: The authors reported controlling for performance, and the performance covariate did not reduce the effect of race or condition on blood pressure reactivity. Though not a formal test of mediation it does suggest that blood pressure changes could not be directly linked – at least not in a linear sense – to performance decrements.

More recently, Vick, Seery, Blascovich and Weisbuch (2008) examined physiological responses associated with challenge and threat among men and women who were assigned to either a "gender-biased" or "gender-fair" math task. The authors observed a sex by condition interaction for cardiac output and total peripheral resistance. Examining the mean responses from this study it appears that when a math test was described as gender-fair women exhibited the adaptive, challenge, profile more so than men. In contrast when the test was described as gender-

biased women exhibited threat profiles relative to men. What is notable about these findings is that the interaction between sex and condition seems to be largely primarily by the *challenge* response of male participants in the "gender-biased" condition, a pattern consistent Walton and Cohen's (2003) stereotype lift meta-analysis that suggest dominant groups perform better under "biased" test conditions. However, there were no reports of performance differences so it is not possible to determine if the physiological responses explained performance decrements or if there were any performance decrements observed as a function of stereotype threat.

We have highlighted these papers that have explicitly tested physiological reactivity associated with stress in standard stereotype threat studies with a more complete list presented in Table 1. Though the studies we review showed some support that the state of stereotype threat results in more maladaptive stress (or threat) as we have defined here, none of them reported evidence that the changes in physiological reactivity could explain performance decrements, even though as we outlined earlier there is evidence to hypothesize the physiological reactivity may be part of the causal link to performance decrements. Indeed, in our search of the literature we could not find any published papers in which stress arousal, measuring with a neurobiological response, even partially mediated the link between stereotype threat manipulations and cognitive performance (in the next section we examine vigilance and attentional mediators in which physiological responses have shown mediation). We believe there are at least four reasons why this may be the case: 1) timing of physiology relative to performance; 2) types of tasks employed; 3) stress arousal measures; and 4) measurement issues.

The first critical factor is the timing of the "stress" response and the performance change. Temporal activation of stress responses differ between adaptive and maladaptive stress responses. If a stereotype threat manipulation occurs and this activates the HPA responses it

might take as long as 10 minutes or more for increasing levels of cortisol to have an effect on neural regions. In contrast, an adaptive stress response characterized by strong sympathetic activation might show its greatest effects on performance very early in a task and might dissipate after only a minute or two of a test. In other words, timing of the physiological response and performance outcome must be considered in terms of their temporal activation and shut off. Another factor is the type of task being performed. As we outlined, memory and information retrieval are more likely to be influenced by chronic stress and "threat" responses, whereas execution, effort and perseverance may be impaired as a result of blunted sympathetic activation.

The third factor is the stress arousal measure. Examining physiological responses that only present one component of the stress response (e.g., cortisol as opposed to cortisol and counter-regulatory hormones) or measures that represent combined influences of different physiological systems (heart rate that is dually innervated by sympathetic and parasympathetic branches) is likely to obscure relationships between physiology and performance. Finally, a typical mediational analysis assumes linear relationships between a mediator (in this case, physiology) and an outcome (performance). But this analytic approach might be misguided for two reasons. First, the relationship between the neurobiological response and the performance variable may not be linear but rather curvilinear so higher order trends should always be tested. More problematic is that neurobiological responses, like the ones we reviewed here, may be released in pulsatile patterns, which would obscure linear relations and render standard GLM techniques inappropriate.

To overcome the last obstacle, experiments could be devised that constrain the physiological response in ways that allow for more precise identification of the physiological responses as the mechanism of action. For example, a dexamethasone suppression test (DST),

which involves an oral administration of a steroid that suppresses ACTH and hence cortisol production, could be used to examine if HPA activation can partly explain performance decrements associated with stereotype threat. In this type of study participants would be assigned to either a placebo or DST and also to a stereotype threat versus control condition then complete a task with memory or retrieval components. The prediction would be that the placebo group exposed to a stereotype threat manipulation would have poorer performance than the placebo non-stereotype threatened condition, which is the standard stereotype threat effect. However, if the DST participants exposed to stereotype threat conditions do not show performance decrements relative to the DST participants in the control condition then cortisol could be directly implicated the causal process of stereotype threat performance decrements. This type of paradigm allows one to sidestep the difficulties of relating responses that may not share a one-to-one dosage level relationship even though the neurobiological response is the mechanism of action.

Strategies to combat maladaptive stress arousal

Several successful interventions have been developed to counteract performance impairments believed to be linked to stereotype threat (see Cohen, Purdie-Vaughs, & Garcia, this volume). If stress arousal is directly (or even indirectly) responsible for performance impairments associated with stereotype threat, what can the literature on stress and emotion regulation teach us about combating stereotype threat effects on performance? One potentially useful strategy capitalizes on reappraising “anxiety” or “arousal” (Jamieson, et al., 2010; Johns, Schmader, & Martens, 2005; Schmader, Forbes, Zhang, & Mendes, 2009). This approach emphasizes the idea that even though arousal is multi-dimensional, the precise assessment of

one's internal states can be ambiguous, which allows for flexibility in terms of labeling one's stress state.

The effectiveness of reappraising arousal was examined among a group of college students preparing to take the GRE (Jamieson, et al., 2010). In this study, participants were randomly assigned to either a reappraisal condition or a control condition. The reappraisal condition informed participants about challenge states, that is they were told that the arousal they were feeling before they took the GRE actually was signaling that their body was preparing for action and would be associated with better performance. The control condition was not provided this reappraisal strategy. Participants provided saliva samples at baseline (prior to the manipulation) and immediately before taking the GRE math and verbal sections that were assayed for salivary alpha amylase (sAA), a proxy for catecholamine levels. Results showed that participants in the reappraisal condition had a greater increase in sAA and performed better on the GRE-math section than participants in the control condition. Correlations between sAA levels and GRE-math performance showed the expected positive relationship – the greater the increase in sAA from baseline to the math task was associated with better math performance. Furthermore, the effects of reappraisal were evident when participants took the actual GRE. Between 1 and 3 months later, participants returned to the lab after they had taken the actual GRE and brought in their score reports. Participants who had been in the reappraisal condition had obtained higher GRE-math scores than those in the control condition. Why the reappraisal manipulation had such long lasting effects is unclear. It may be because participants remembered the reappraisal manipulation on the day of the actual exam or possibly those in the reappraisal condition were emboldened from the lab study and studied harder than those in the control condition.

This study highlights several important points from this chapter. First, consistent with the adaptive stress profile the greater the sympathetic activation the better the performance with no indications of a curvilinear pattern between "arousal" and performance. Second, the effects were not observed with the GRE verbal section. This may be due to the types of questions from the math compared to verbal sections. The math section requires active execution, which like physical exercise, is enhanced at higher levels of sympathetic activation. Verbal problems are often recall or comprehension questions which do not benefit from sympathetic activation in the same way. Finally, the study demonstrates the importance of labeling one's physical state, which may influence subsequent reactivity thus suggesting flexibility of stress responses. Importantly, the reappraisal strategy employed was not one that tried to dampen or minimize the reactivity, but rather accentuate the "arousal" component as a beneficial state. We believe that exploiting stress and emotion regulation techniques to modify and enhance physiological responses associated with stress may prove to be a useful intervention for stereotype threat research.

Vigilance

The experience of stereotype threat requires stigmatized individuals to reconcile environmental cues associated with their stigmatized status while simultaneously marshalling the cognitive resources necessary to perform well on tasks. Thus, another possible mechanism of stereotype threat performance decrements is vigilance – the process of excessively monitoring the environment for threat cues at the same time attending to the task at hand. This perspective suggests that the experience of stereotype threat occupies or diverts attentional resources, thereby debilitating performance on higher-order tasks, which may be responsible for performance decrements (e.g. Schmader, et al., 2008). There are several brain and bodily responses associated with vigilance that may shed light on this possible mechanism. Here we review responses from

measures obtained from parasympathetic reactivity, electroencephalogram (EEG) and, more specifically, event-related potentials (ERP), and functional magnetic resonance imaging (fMRI). For each of these measures we describe the links between vigilance and the neurobiological response to develop specific hypotheses of how the neurobiological response would be associated with performance. Then, as before, we then review the extant stereotype threat literature that has adopted these measures and how successful (or not) they have been.

Parasympathetic reactivity

In the first half of this chapter we reviewed stress arousal as a mechanism of stereotype threat effects and we focused on activation of the sympathetic nervous system (SNS) ignoring a large part of the autonomic nervous system: the parasympathetic system (PNS). Given that these two systems can operate independently (Berntson, Cacioppo, & Quigley, 1993) our distinction was not merely didactic but rather dictated by the role that the SNS serves in stress, and the PNS serves in attention or vigilance.

The PNS is most often measured with high-frequency heart rate variability (HRV), which is presumed to measure the activity of the vagus nerve, a cranial nerves originating in the medulla, which innervates a number of organs including the heart. HRV changes appear to be sensitive to a variety of psychological states, but are not particularly specific. However, accumulating evidence suggests that decreases in HRV during active tasks are associated with greater attentional control or effort (Croizet, et al., 2004; Porges, 2007). Indeed, cognitive psychophysiologists infer decreases in HRV as an index of attention or mental effort (Tattersall & Hockey, 1995). In the anchoring-adjustment study we described earlier (Kassam, et al., 2009) the strongest physiological predictor of performance was HRV changes: the greater the

withdrawal of the vagal brake during the decision making task, the better the performance (Kassam, et al., 2009).

Just as few stereotype threat studies have examined the physiological underpinnings of arousal stress processes, there is also a dearth of research on the biological mechanisms underlying vigilance. In one study of stereotype threat, Croizet, et al. (2004) examined changes in HRV during a stereotype threat paradigm, relying on the interpretation that decreases in HRV would index mental effort. They found that participants assigned to a stereotype threat prime had greater decreases in HRV and poorer performance than those in the control condition and that HRV changes mediated the relationship from the condition to the performance effects. This work provides some evidence that changes in parasympathetic activity may mediate the relationship between stereotype threat and performance; however, HRV decreases may index processes other than vigilance and performance monitoring, such as conscious control (Kassam, et al., 2009), anxiety or depression (Porges, 2007), or pessimism (Oveis, et al., 2009) to name a few. Although there may be great promise with exploiting HRV changes as a possible mechanism underlying stereotype threat some caution is warranted. It might be difficult to determine if vigilance induced decreases in HRV will facilitate or impair performance. In the Croizet article, for example, the argument was that stereotype threat induced vigilance which directed attention away from the task, however if attentional control could be marshaled toward the task and away from the environmental triggers of the stereotype threat then one might expect HRV decreases to be associated with performance enhancements.

Electroencephalogram (EEG)

Other non-invasive techniques can be used to measure vigilance and attentional processes in stereotype threat. For example, researchers have used evoked electroencephalogram (EEG)

signals, which measure electrical activity along the scalp via a network of sensors. Of specific interest for stereotype threat researchers interested in vigilance processes are event-related potentials (ERPs) – the average of a short epoch of EEG waveform data directly following an event (e.g., a response, stimulus onset, etc.). The high temporal resolution of ERP signals allows researchers to study the impact of psychological states on individuals' allocation of attention not just at conscious levels, but also at early processing stages. Thus, EEG methods help researchers determine how psychological states, like stereotype threat, impact low-level processes that are difficult to measure with standard behavioral methods.

To study vigilance processes, researchers have examined ERP signals measured at electrodes located in the medial-frontal area of the scalp. Broadly, medial-frontal ERP waves index vigilance and attention, especially when an error has been made or some other anxiety-provoking event has occurred. A specific type of medial-frontal ERP signal that stereotype threat researchers have focused on is the error-related negativity (ERN) component. The ERN signal is a negative-going deflection in the ERP waveform that is most pronounced at the fronto-central region on the midline of the scalp 30–180 ms after an error has been made (Yeung, Botvinick, & Cohen, 2004), and ERN amplitudes are larger after performance errors than after correct responses. The magnitude of ERN responses are used to index vigilance (i.e. error detection) and performance monitoring processes (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), as well as affective responses such as defensive motivation (Hajcak & Foti, 2008). Thus, ERN methods are not only useful for studying the cognitive effects of threat, but may also provide insight into participants' affective responses to stereotype threat (e.g. anxiety and/or motivation) because the ERN signal originates

in the ACC (Holroyd & Coles, 2002; Ullsperger & von Cramon, 2003), an area thought to underlie emotion regulation (e.g., Bush, Luu, & Posner, 2000).

Along similar lines, other social neuroscience research has also found evidence that stereotype threat leads participants to monitor their performance for mistakes. More specifically, Forbes et al. (2008) measured ERPs to explore early stage motivational processes in performance monitoring and also examined the moderating effect of domain identification. For minority participants who valued academics, the experience of stereotype threat led to an increased ERN response, which suggests that these participants were vigilant for performance related stimuli and were more efficient in responding to them. This increase in vigilance is indicative of the increased motivation to perform well under threat in domain identified targets (Jamieson & Harkins, 2009). However, Forbes and colleagues observed a very different pattern for minority participants who did not value academic success. Specially, rather than devoting attentional resources to performance monitoring, devaluing academics negatively predicted ERN amplitude under threat. This finding suggests that stigmatized individuals who no longer care about performance in stereotyped domains are not vigilant for potential errors, and instead disengage during performance.

EEG methods have also been used to study self-regulation and spillover processes under stereotype threat. Like the aforementioned work by Forbes et al. (2008), work by Inzlicht and Kang (in press) suggests that the experience of threat leads to hyper-vigilance, thereby debilitating self-regulation. In that work, threatened females exhibited higher amplitude medial-frontal ERP waves in response to Stroop errors (naming the incorrect color), as well as during correct high conflict (“blue” printed in red ink) and correct low conflict Stroop trials (“blue” printed in blue ink). On the other hand, males did not exhibit this increased ERP amplitude for

low conflict trials. Threatened females increased monitoring of every type of trial, even on low conflict trials not requiring vigilance, indicating that female participants under threat may lose self-regulatory capacity.

Functional Magnetic Resonance Imaging (fMRI)

Although EEG methods provide high temporal resolution, they are less able to localize an effect in the brain. However, within the past 20 years, advances in neuroimaging methods have allowed researchers to spatially localize processes related to attention and vigilance non-invasively. To study the brain regions underlying psychological states, researchers often measure blood oxygenation level dependent (BOLD) signals obtained via fMRI. The logic behind BOLD signals is that changes in brain activity lead to changes in blood flow to active brain regions in response to a thought, action, and/or psychological experience. BOLD signals provide a measure of neural activation which can be measured online while participants are experiencing the psychological state of interest. Thus, fMRI techniques can help inform researchers as to the brain regions underlying attention allocation and vigilance processes under conditions of stereotype threat.

The few stereotype threat studies that have used fMRI methods have observed activation in the ACC (e.g., Krendl, Richeson, Kelley, & Heatherton, 2008; Wraga et al., 2007), a finding that is consistent with research that has found stereotype threat increases the magnitude of ERN signals originating in the ACC (e.g., Forbes, et al., 2008). A multitude of fMRI research suggests that the ACC, specifically the ventral ACC, is activated by the experience of physical pain (e.g., Rainville, Duncan, Price, Carrier, & Bushnell, 1997), emotional distress (e.g., Eisenberger, Lieberman, & Williams, 2003), or as touched on earlier when individuals regulate emotional responses (e.g., Bush et al., 2000). Particularly relevant for stereotype threat researchers is the

acute sensitivity of ventral ACC regions to social feedback, especially negative feedback (Eisenberger et al., 2003; Somerville, Heatherton, & Kelley, 2006). That is, when threatened by the salience of stereotype-related cues stigmatized individuals may respond with increased activity in the ventral ACC.

Research using fMRI techniques has suggested that the experience of stereotype threat decreases participants' recruitment of attention resources. In some recent research, Krendl and colleagues (2008) observed that women not subject to stereotype threat exhibited activation in prefrontal and parietal areas, indicative of the recruitment of attentional resources, during math performance. However, when women were subject to stereotype threat, they exhibited less prefrontal and parietal activity, and instead demonstrated increased activity in the ventral stream of the ACC, which is involved in action monitoring and correction processes (Gehring & Knight, 2000) as well as emotional regulation (Somerville, et al., 2006). This finding was corroborated and extended in additional work by Wraga, and colleagues (2007), which found that stereotype threat increased activation in the ventral ACC, and that this activation predicted threatened participants' performance decrements on a mental rotation task. Thus, previous research provides some initial evidence that the experience of stereotype threat shifts how women utilize attentional resources. When not subject to threat women recruited resources from prefrontal regions associated with attention, but when threatened, females exhibited greater activation in monitoring, correction, and emotion regulation areas. Therefore, rather than focusing on task performance, threat caused women to recruit additional systems associated with performance monitoring and emotion regulation, potentially decreasing the amount of cognitive resources available for task performance (e.g. Beilock et al., 2007; Johns, Inzlicht, & Schmader, 2009).

Summary

In this chapter we reviewed various literatures that would further our understanding of stereotype threat processes. As many of the mechanisms that are believed to underlie stereotype threat processes have neurobiological concomitants we explored the process of stereotype threat from two angles. First we examined how different psychological and affective states associated with stereotype threat were linked to various neurobiological changes. The second angle examined how these various neurobiological changes might be associated with performance changes. We summarized this approach early in the chapter in Figure 1, but also highlighted that there are stronger links between the psychological states and the neurobiological changes than links between the neurobiological changes and the performance outcomes. Though we do believe that neurobiology may prove to be a useful candidate mechanism for stereotype threat, there is clearly much that is unknown regarding how biological changes influence performance, and multiple pathways that complicate these links. This review is meant to be both humbling in terms of how much is yet unknown about links between biology and performance, but also, we hope, inspiring as researchers continue to search for explanations of stereotype threat effects. Our intent in this chapter was to offer possible avenues for researchers to explore biological mechanisms, but also words of caution in that not all stress responses are created equal, and that many neurobiological responses have yet to show reliable influences on cognition. There is certainly more work to be done but by exploring brain and body mechanisms associated with stereotype threat we believe that the puzzle of how negative stereotypes influence performance can be revealed.

References

Beilock, S.L., Rydell, R.J., & McConnell, A.R. (2007). Stereotype threat and working memory: Mechanisms, alleviation, and spill over. *Journal of Experimental Psychology: General, 136(2),* 256-276.

Ben-Zeev, T., Fein, S., & Inzlicht, M. (2005). Stereotype threat and arousal. Journal of Experimental Social Psychology, 41, 174-181.

Berntson, G.G., Cacioppo, J.T., & Quigley, K.S. (1993). Cardiac psychophysiology and autonomic space in humans: Empirical perspectives and conceptual implications. *Psychological Bulletin, 114,* 296-322.

Blascovich, J. & Mendes, W. B. (2010). Social psychophysiology and embodiment. In: S. T. Fiske & Gilbert, D. T. (Eds.). *The Handbook of Social Psychology, 5th Edition.* New York: Wiley.

Blascovich, J., Mendes, W. B., Hunter, S. B., & Salomon, K., (1999). Social "facilitation" as challenge and threat. *Journal of Personality and Social Psychology, 77,* 68-77.

Blascovich, J., Spencer, S., Quinn, D., & Steele, C. (2001). African-Americans and high blood pressure: The role of stereotype threat. *Psychological Science, 12,* 225-229.

Bush, G., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4(6),* 215-222.

Collins, A. & Frankenhaeuser, M. (1978). Stress responses in male and female engineering students. *Journal of Human Stress, 4,* 43-48.

Croizet, J., Despres, G., Gauzin, M., Huguet, P., Levens, J., & Meot, A. (2004). Stereotype threat undermines intellectual performance by triggering a disruptive mental load. *Personality and Social Psychology Bulletin, 30,* 721-731.

Davies, P. G., Spencer, S. J., Quinn, D. M., & Gerhardstein, R. (2002). Consuming images: How television commercials that elicit stereotype threat can restrain women academically and professionally. *Personality and Social Psychology Bulletin, 28*(12), 1615-1628.

Derks, B., Inzlicht, M., & Kang, S. (2008). The neuroscience of stigma and stereotype threat. *Group Processes and Intergroup Relations,* 11, 163-181.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130* (3), 355-391.

Dienstbier, R. A. (1989). Arousal and physiological toughness: Implications for mental and physical health. *Psychological Review, 96,* 84-100.

Eisenberger, N.I., Lieberman, M.D., & Williams, K.D. (2003). Does rejection hurt? An fmri study of social exclusion. *Science, 302,* 290-292.

Epel, E., McEwen, B., & Ickovics, J. (1998). Embodying psychological thriving: Physical thriving in response to stress. *Journal of Social Issues, 54,* 301–322.

Forbes, C., Schmader, T., & Allen, J.J.B. (2008). Error monitoring in an intellectually threatening environment. *Social Cognitive Affective Neuroscience, 3,* 253-261.

Gehring, W.J., Goss, B., Coles, M.G.H., Meyer, D.E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4,* 385-390.

Gehring, W.J., & Knight, R.T. (2000). Prefrontal – cingulate interactions in action monitoring. *Nature Neuroscience, 3,* 516-520.

Hajcak, G., & Foti, D. (2008). Errors are aversive: Defensive motivation and the error-related negativity. *Psychological Science, 19,* 103-108.

Holroyd, C.B, & Coles, M.G.H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109,* 679-709.

Inzlicht, M., & Kang, S.K. (in press). Stereotype threat spillover: How coping with threats to social identity affects aggression, eating , decision-making, and attention. *Journal of Personality and Social Psychology.*

Jamieson, J., & Harkins, S. (2009). The effect of stereotype threat on the solving of quantitative GRE problems: A mere effort interpretation. *Personality and Social Psychology Bulletin. 35*(10), 1301-1314.

Jamieson, J., Mendes, W. B., Blackstock, E. & Schmader, T. (2010). Turning the knots in your stomach into bows: Reappraising arousal improves performance on the GRE. *Journal of Experimental Social Psychology, 46,* 208-212.

Johns, M.J., Inzlicht, M., & Schmader, T. (2008). Stereotype threat and executive resource depletion: Examining the influence of emotion regulation. *Journal of Experimental Psychology: 137(4),* 691-705.

Johns, M., Schmader, T., and Martens, A. (2005). Knowing is half the battle: Teaching stereotype threat as a means of improving women's math performance. *Psychological Science, 16*(3), 175-179.

Josephs, R.A., Newman, M.L., Brown, R.P., & Beer, J.M. (2003). Status, testosterone, and human intellectual performance: Stereotype threat as status concern. *Psychological Science, 14,* 158-163.

Kassam, K., Koslov, K., & Mendes, W. B. (2009). Decisions under distress: Stress profiles influence anchoring and adjustment. *Psychological Science, 20,* 1394-1399.

Krendl, A.C., Richeson, J.A., Kelley, W.M., & Heatherton, T.F. (2008). The negative consequences of threat: An fmri investigation of the neural mechanisms underlying women's underperformance in math. *Psychological Science,* 19, 168-175.

Labrie, F., Bélanger, A., Luu-The, V., Labrie, C., Simard, J., & Lin, S.-X. (2000). DHEA, the precursor of androgens and estrogens in peripheral tissues in the human: intracrinology, in: Kalimi, M. et Regelson, W., eds: *(DHEA): Biochemical Physiological and Clinical Aspects,* (pp. 191-213). New York: Walter De Gruyter Inc.

Lazarus, R., & Folkman, S. (1991). The concept of coping. *Stress and coping: An anthology (3rd ed.)* New York: Columbia University Press.

Lovallo, W. R., & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In J.T. Cacioppo, L. G. Tassinary & G. G. Berntson (Eds.), *Handbook of psychophysiology (2nd Ed.),* (pp. 342-367). New York: Cambridge University Press.

Matheson, K., & Cole, B. (2004). Coping with a threathened group identity: Psychological and neuroendocrine responses. *Journal of Experimental Social Psychology, 40,* 777-786.

Mendes, W. B., Blascovich, J., Hunter, S., Lickel, B., & Jost, J. (2007). Threatened by the unexpected: Challenge and threat during inter-ethnic interactions. *Journal of Personality and Social Psychology, 92,* 698-716.

Murphy, M. C., Steele, C. M. and Gross, J. J. (2007). Signaling threat how situational cues affect women in math, science, and engineering settings. *Psychological Science, 18,* 879-888.

Nieuwenhuis, S., Ridderinkhof, K.R., Blom, J., Band, G.P.H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology, 38,* 752-760.

O'Brien, L. T. & Crandall, C. S. (2003). Stereotype threat and arousal: Effects on women's math performance. *Personality and Social Psychology Bulletin, 29*(6), 782-789.

Osborne, J.W. (2007). Linking stereotype threat and anxiety. *Educational Psychology, 27(1),* 135-154.

Oveis, C., Cohen, A. B., Gruber, J., Shiota, M. N., Haidt, J., & Kelter, D. (2009). Resting respiratory sinus arrhythmia is associated with tonic positive emotionality. *Emotion, 9,* 265-270.

Porges, S.W. (2007). The polyvagal perspective. *Biological Psyhology, 74(2),* 116-143.

Rainville, P., Duncan, G.H., Price, D.D., Carrier, B., & Bushnell, M.C. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science, 277(5328),* 968-971.

Reul, J. & de Kloet, E. (1985). Two receptor systems for corticosterone in rat brain: microdistribution and differential occupation. *Endocrinology, 117,* 2505-2511.

Schmader, T., Forbes, C.E., Zhang, S., & Mendes, W.B. (2009). A meta-cognitive perspective on cognitive deficits experienced in intellectually threatening environments. *Personality and Social Psychology Bulletin, 35,* 584-596. Schmader, T., Johns, M., & Forbes, C. (2008). An integrated process model of stereotype threat effects on performance. Psychological Review, 115, 336-356.

Somerville, L.H., Heatherton, T.F., & Kelley, W.M. (2006). Disambiguating anterior cingulate cortex function: Differential response to experiences of expectancy violation and social rejection. *Nature Neuroscience, 9,* 1007-1008.

Southwick, S.M., Vythilingam, M., & Charney, D.S. (2005). The psychobiology of depression and resilience to stress: Implications for prevention and treatment. *Annual Review of Clinical Psychology, 1,* 255-291.

Steele, C.M., & Aronson, J. (1995). Stereotype threat and the intellectual test performance of African-Americans. *Journal of Personality and Social Psychology, 69,* 797-811.

Tattersall, A.J., & Hockey, G.R.J. (1995). Level of operator control and changes in heart rate variability during simulated flight maintenance. *Human Factos, 37(4),* 682-698.

Tomaka, J., Blascovich, J., & Kelsey, R. M. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65,* 248-260.

Ullsperger, M., & von Cramon, D.Y. (2003). Error monitoring using external feedback: Specific roles of the habenular complex, the reward system, and the cingulate motor area revealed by functional magnetic resonance imaging. *Journal of Neuroscience, 23,* 4308-4314.

Ursin, H., Baade, E., & Levine, S. (1978). *Psychobiology of stress: A study of coping men.* New York: Academic Press.

Vick, S.B., Seery, M.D., Blascovich, J., & Weisbuch, M. (2008). The effect of gender stereotype activation on challenge and threat motivational states. *Journal of Experimental Social Psychology, 44,* 624-630. Walton, G.M., & Cohen, G.L. (2003). Stereotype lift. *Journal of Experimental Social Psychology, 39,* 456-467.

Wolf, O.T., Neumann, O., Hellhammer, D.H., Geiben, A.C., Strasburger, C.J., Dressendörfer, R.A., et al. (1997). Effects of a two week physiological dehydroepiandrosterone substitution on cognitive performance and well-being in healthy elderly women and men. *Journal of Clinical Endocrinology and Metabolism, 82*, 2363–2367.

Wraga, M., Helt, M., Jacobs, E., & Sullivan, K. (2007). Neural basis of stereotype-induced shifts in women's mental rotation performance. *Social Cognitive and Affective Neuroscience, 2,* 12-19.

Yeung, N., Botvinick, M.M., & Cohen, J.D. (2004). The neural basis of error-detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111,* 931-959.

Table 1. Summary of empirical papers exploring neurobiological consequences of stereotype threat

Author(s)	*Year*	*Target Group*	*Neurobiological Measure*	*Performance Measure*	*Mediation Found*
Blascovich, Spencer, Quinn, & Steele	2001	African-Americans	Mean arterial blood pressure	Remote associates test	Not reported
Croizet, Despres, Gauzin, Huguet, Leyens, & Meot	2004	Academic major	HRV	Raven progressive matrices test	Yes
Derks, Inzlicht, & Kang	2008	Women	EEG	Stroop & automatic face evaluation	Yes
Forbes, Schmader, & Allen	2008	Latinos/ African Americans	EEG	Flanker task	Not reported
Inzlicht & Kang	In press	Women	EEG	Stroop	Yes
Josephs, Newman, Brown, & Beer	2003	Women	Testosterone	Quantitative GRE	Not reported
Krendl, Richeson, Kelley, & Heatherton	2008	Women	fMRI	Mixed math: arithmetic & modular arithmetic	Not reported
Matheson & Cole	2004	College identity	Cortisol	None reported	
Murphy, Steele, & Gross	2007	Women	Sympathetic activation	Recall test	Not reported
Osborne	2007	Women	Sympathetic activation/blood pressure	Quantitative GRE	Not reported
Vick, Seery, Weisbuch, & Blascovich	2008	Women	Cardiovascular reactivity	Quantitative GRE: just comparison problems	Not reported
Wraga, Helt, Jacobs, & Sullivan	2006	Women	fMRI	Mental rotation task	Yes

Note. HRV = heart rate variability; EEG = electroencephalogram; fMRI = functional magnetic resonance imaging

Figure Captions

Figure 1. The left side of the figure depicts relationships between psychological states and processes associated with neurobiological changes. The right side of the figure indicates relationships between the neurobiological changes and cognitive or physical outcomes. The thickness of the arrow represents the greater quality and quantity of data supporting the link. All arrows represent positive associations unless indicated with a minus (-) sign.

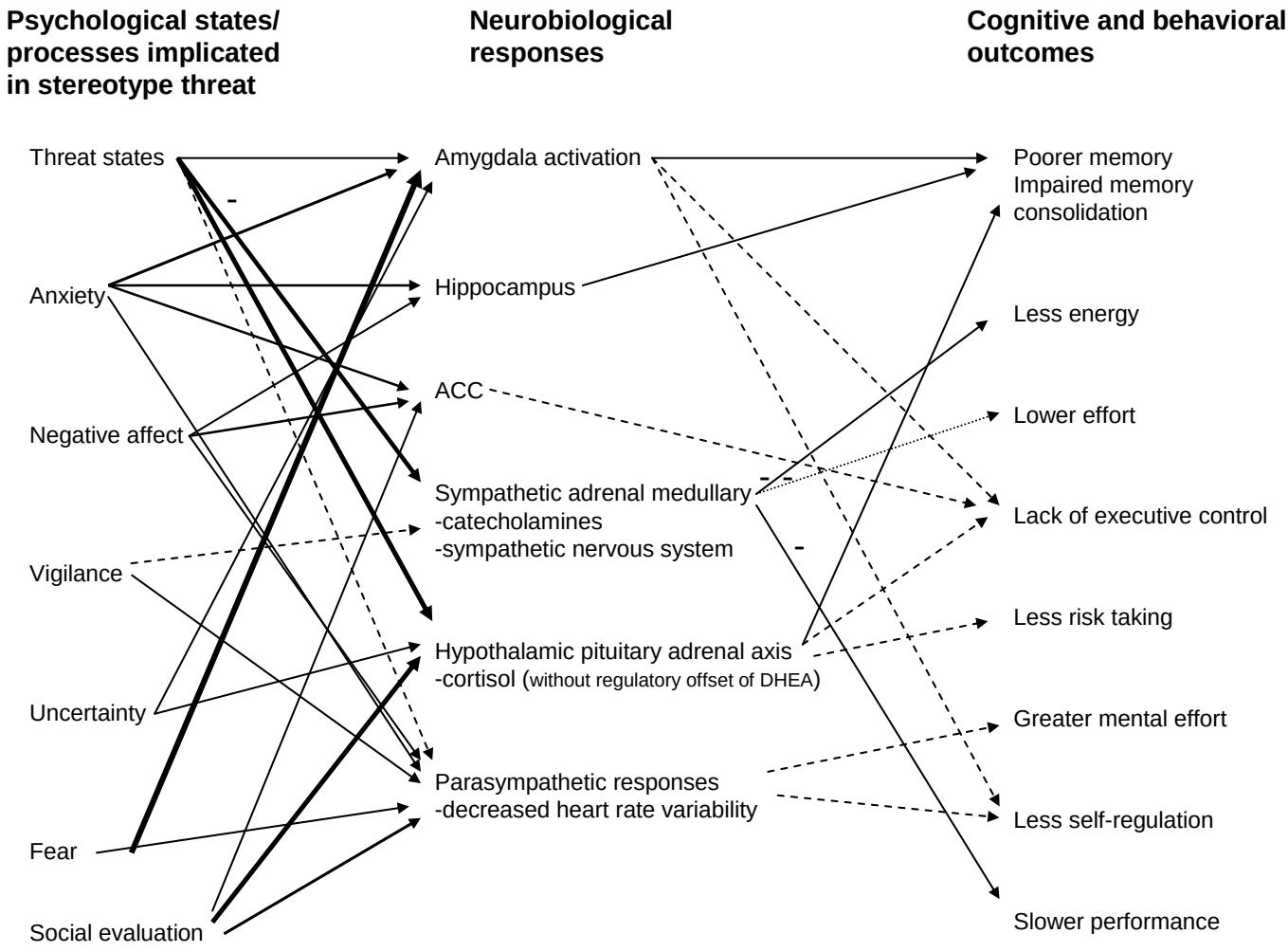
Psychological states/ processes implicated in stereotype threat
Neurobiological responses
Cognitive and behavioral outcomes
Threat states
Anxiety
Negative affect
Vigilance
Uncertainty
Fear
Social evaluation
Amygdala activation
Hippocampus
ACC
Sympathetic adrenal medullary
-catecholamines
-sympathetic nervous system
Hypothalamic pituitary adrenal axis
-cortisol (without regulatory offset of DHEA)
Parasympathetic responses
-decreased heart rate variability
Poorer memory
Impaired memory consolidation
Less energy
Lower effort
Lack of executive control
Less risk taking
Greater mental effort
Less self-regulation
Slower performance

Figure 1

Policy Box

Can exercise stop stereotype threat effects?

In this chapter we highlight brain and bodily changes associated with the experience of stereotype threat and how those changes might affect cognitive performance. Which leads to the question can changing bodily states alter cognitive performance? A growing research area in psychology and neuroscience is "embodied cognition," which examines how bodily responses can influence cognitive processes. For example, if you held a pencil in your mouth with your teeth (rather than your lips) this will activate the smiling muscles and without even realizing it you may find the morning comic strip funnier than you typically do. The idea behind this effect is that the smiling muscles are sending information to your brain that you are happy and hence the comics seem funnier. These same processes can be observed with *adaptive* stress profiles and cognition. Adaptive stress profiles are associated with increased sympathetic activation and increased blood flow to the brain and body, which can increase cognitive performance. These profiles can be brought on by psychological stress, but also with aerobic exercise. Acute effects of exercise and, of course, long lasting effects of conditioning, may buffer impairments in cognition by maintaining increased blood flow to the brain and body. Indeed, in a recent longitudinal medical study, individuals with greater cardiac output (a cardiac index of oxygenated blood pumped from the heart) had lower risk of cognitive declines in older age and reduced risk of Alzheimer's disease. In addition to the large body of evidence showing exercise is beneficial for physical and mental health, there is also reason to believe exercise can be beneficial for cognitive performance. Policy makers should be mindful that physical education in schools may have direct links to performance in the classroom.