CHAPTER

14 Affective Reactions as Mediators of the Relationship Between Stigma and Health

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Abstract

This chapter provides an overview of how emotions can contribute to poorer health among stigmatized populations. First, it describes some of the primary affective responses that stigmatized individuals might experience, including externalizing emotions, uncertainty, and anxious affect. These affective responses can occur as a result of interacting with individuals who display subtle or overt signs of bias or perceiving a system as unfair, or they can occur from expectations based on prior experiences that shape perception. Second, this chapter reviews how these affective states may alter underlying biological processes to directly influence health. Finally, it examines indirect pathways whereby emotion processes potentiate health-damaging behaviors, such as poor eating habits, restless sleep, excessive alcohol and drug abuse, and risky behavior. Overall, research in this area suggests that affective experiences resulting from stigmatization can change biology and behavior in ways that can ultimately lead to poor health.

Key Words: Affect, Discrimination, Cardiovascular reactivity; Emotion; Health, Psychobiology, Neuroendocrinology, Race, Social neuroscience

Health disparities between advantaged and disadvantaged group members are pervasive and socially significant (see Chapter 2, this volume). For example, in the United States, individuals stigmatized based on racial categories, such as African Americans, are more likely than individuals not stigmatized by race, such as European Americans, to develop hypertension, cardiovascular disease, and lung cancer and to have more years of morbidity and higher mortality rates (Borrell, Kiefe, Diez-Roux, Williams, & Gordon-Larsen, 2013; Krieger, 2014; Paradies, 2006; see also Chapters 2, 9, and 11, this volume). Although health disparities based on stigmatizing characteristics such as race/ethnicity, sexual identity, and lower socioeconomic status are clear, what is less clear is why these health disparities exist and persist. In addition, although economic factors related to quality health care, living conditions, and environmental exposures contribute to health disparities, psychological factors related to the added burden of a stigmatized identity, perceived discrimination and unfair treatment, microaggressions (i.e., subtle, common statements that might be interpreted as demeaning to a member of a stigmatized group), vigilance for bias, and physiologic influence of anxious affect also can contribute to widening health disparities (Basáñez, Unger, Soto, Crano, & Baezconde-Garbanati, 2013; Borrell et al., 2013; Paradies et al., 2015; Williams, Yu, Jackson, & Anderson, 1997). This chapter focuses on these latter psychological factors and, specifically, how emotional reactivity triggered by experiences or perceptions of discrimination and unfair treatment based on individuals' stigmatized status can lead to poor health.

In Figure 14.1, we present a model of how affective experiences of stigmatized persons influence health outcomes. We organize our analysis at the level of the person (in this case, a stigmatized person) and how the perceived and experienced social environment influences the thoughts, feelings, and behaviors of the stigmatized person. We delve into

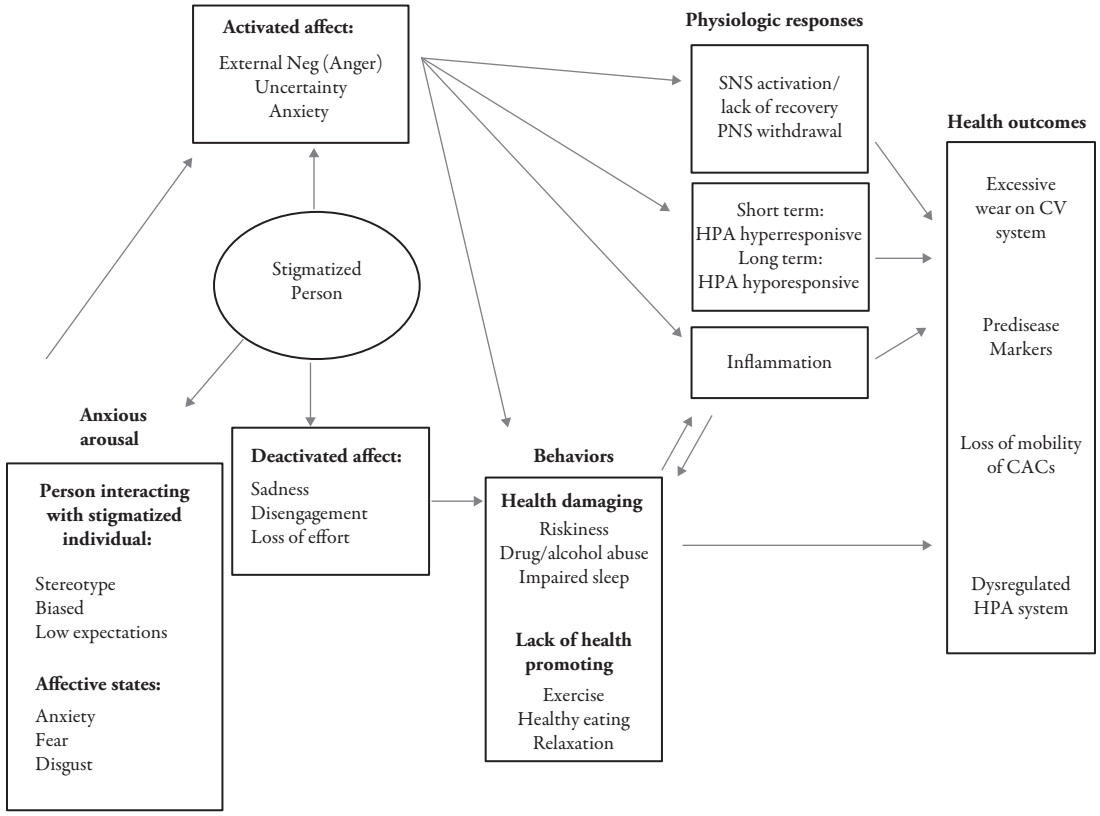

Figure 14.1 How affective experiences of stigmatized persons may influence health outcomes.

these thoughts and feelings by considering the *affective* and *neurobiological* responses of the stigmatized and relate these responses to physiological pathways implicated in health outcomes and behavioral pathways implicated in health behaviors. The model also addresses key concepts that complicate a straightforward explanation that stigma can "get under the skin" directly from the experience of stress or negative emotions. Instead, we underscore concepts such as timing, length, and persistence of aversive reactions to social environments experienced by stigmatized persons, how they make meaning from their social experiences, and developmental factors that may alter the stigma → affect → health pathway.

We use the words *affect* and *affective* as overarching terms to represent responses that are high-arousal, self-relevant, and "hot" (relative to "cold" cognitive) responses that individuals experience. Affect encompasses constructs such as *stress, emotion*, and *motivation* (cf., Gross, 2015). It is a general term that allows us to integrate research from health fields, which commonly use the term *stress* to indicate any short-term physiologic change; research from social and clinical psychology that more likely relies on specific "emotion" categories such as anger, sadness, and fear; and research from motivational perspectives that focus on mental states and behavioral responses with concepts such as avoidance, threat, and vigilance. Although one could draw clear differences among the concepts of stress, emotion, and motivation, there exists biological and conceptual overlap of these mental states.

The chapter is organized such that first we review some of the most studied affective responses associated with stigmatization. Next, we discuss links between affective responses experienced by stigmatized persons and the short-term and long-term biological sequelae that may lead to poor physical health outcomes. We refer to these links as *direct paths* given that the processes and reactions triggered by stigmatized status presumably lead to direct alteration of biological responses implicated in health (Figure 14.1, solid lines). We consider multiple biological pathways that are implicated in emotion–health relationships to attempt to be comprehensive in our review and inspire more

cross-systems research in examining links between stigma and health. Finally, we examine *indirect paths* that identify how affective reactions can bring about behavior that can ultimately undermine health (Figure 14.1, dashed lines). Specifically, affective responses to stigmatization may affect health not only due to dysregulated biological pathways but also by changing the health behaviors in which people engage. These behaviors can range from active states such as greater risk-taking to passive states characterized by lack of interest in health-promoting behaviors such as exercise or healthy eating.

Affective Responses

Individuals with stigmatized identities live in a world in which they may perceive or experience treatment that is different from treatment experienced by nonstigmatized others. Much research, as highlighted in this book, focuses on how these experiences shape and influence stigmatized persons' lives (see Chapters 12 and 13, this volume). To understand the affective experiences of stigmatized individuals and how these responses might be linked to health, we focus on common affective responses to stigmatization.

Theory and research point to a core set of affective responses experienced by stigmatized individuals. We focus here on a subset of affective responses that are often implicated in health and behavioral pathways. To begin, we set the stage by discussing the psychological, physiologic, and behavioral responses of nonstigmatized group members in their interactions with members of stigmatized groups. We then focus on how these interactions may affect stigmatized group members, specifically (1) *externalizing negative affect*, which comprises negative emotions that are outwardly directed typically toward other people, such as anger and aggression, and can be engendered by overt bias, perceptions of unfairness, and discrimination; (2) *uncertainty or vigilance*, which is an activated, prolonged response associated with monitoring the social environment and is typically evoked by ambiguous situations; and (3) *anxious affect*, which refers to the transmission of one individual's anxious physiologic state to another person during an interaction. This limited focus, however, is not meant to suggest that emotions such as guilt, disgust, fear, and sadness are not also part of the stigmatized person's emotional life but, rather, reflects that the subset of emotions we focus on here are more often studied in relation to health outcomes.

Setting the Stage: Responses to Interacting with Stigmatized Individuals

To understand the affective consequences of stigmatization, we must first consider how members of nonstigmatized groups respond to interactions with stigmatized individuals. A considerable amount of evidence suggests that interacting with stigmatized individuals can create feelings of discomfort, threat, stress, fear, avoidance, and/or disgust for the nonstigmatized individuals, with the specific feelings depending on the nature of the stigma category (Blascovich, Mendes, Hunter, Lickel, & Kowai-Bell, 2001; Fiske, 2010; Inbar, Pizarro, Knobe, & Bloom, 2009; Mendes, Blascovich, Lickel, & Hunter, 2002; Olsson, Ebert, Banaji, & Phelps, 2005; Vanman, Paul, Ito, & Miller, 1997). For example, social and affective scientists have demonstrated that individuals show a range of physiological changes characteristic of negative affect, such as anxiety, stress, disgust, and fear, when interacting with stigmatized others (reviewed later). These physiological changes include increased blood pressure, less efficient cardiac responses, tightening of the vasculature, more corrugator activation, less anabolic hormones, greater amygdala activation, and impaired executive control (for a review, see Major, Mendes, & Dovidio, 2013).

Measuring reactions toward stigmatized group members is complicated by the reality that individuals who feel uncomfortable interacting with stigmatized individuals or hold biased beliefs about stigmatized group members may be unwilling to explicitly report these biases so as to avoid being labeled as prejudiced (Blascovich, Mendes, & Seery, 2002). Indeed, within the contemporary United States, endorsing explicit biased attitudes or engaging in blatantly biased behavior toward some stigmatized group members is largely condemned, although not all stigmatized groups are similarly "protected" from biased attitudes and behaviors. Although blatant discrimination might be easier to identify, biases can operate at a nonconscious level and be more difficult to verify. However, these biases can still be insidious and create strained, awkward, or unpleasant interpersonal encounters between stigmatized and nonstigmatized individuals (Mendes et al., 2002; Richeson & Shelton, 2007). Adding further complexity is that even individuals with no intent to be biased may still respond with biased behavior automatically, without awareness, toward stigmatized group members (Devine, 1989). Given that biases may not be reported because

individuals are either unwilling or unable to report their biases, researchers have circumvented these obstacles by relying on indicators that tend to be more automatic, reflexive, and/or below conscious awareness. For example, such biases can be captured via measures of physiologic change such as neural activation, neuroendocrine responses, or autonomic nervous system reactivity. Research relying on physiologic changes often reveals more negative affective responses when individuals interact with stigmatized compared to nonstigmatized others.

In contrast, explicit responses—reactions that are reflective and under deliberate control—often fail to show similar negative affective responses revealed with implicit measures. Indeed, explicit measures can show the opposite pattern, with nonstigmatized individuals showing *greater* positivity toward stigmatized than nonstigmatized others (Blascovich et al., 2002; Mendes & Koslov, 2013; Vanman et al., 1997). For example, when examining self-reported emotional states stemming from a social interaction with a racial minority, European American participants will often report feeling *more* positive emotions and greater liking of a racial minority partner compared to a same-race partner in the same type of interaction. This type of "overcorrection" might be a deliberate interpersonal strategy to either mask racial biases or suppress felt anxiety. Indeed, overcorrection is reduced when individuals are exposed to resource-depleting tasks such as stress inductions or cognitive load manipulations, suggesting that these overcorrection strategies are fragile and temporary (Mendes & Koslov, 2013).

If physiological responses suggest more negative responses toward stigmatized members, and self-reported responses suggest more positively valenced reactions, the natural question to ask is the following: What are the behaviors of nonstigmatized persons during interactions with stigmatized persons? Evidence suggests that nonstigmatized individuals interacting with stigmatized individuals often leak their biases through a variety of subtle behaviors that are difficult to regulate and consciously control. For example, European Americans interacting with African Americans show greater nonverbal displays of anxiety, tension, and discomfort and, in some cases, exaggerated (disingenuous) positivity (Mendes & Koslov, 2013; Richeson & Shelton, 2007).

In summary, members of nonstigmatized groups are likely to show a complex pattern of self-reported, implicit, physiologic, and behavioral responses during interactions with stigmatized individuals. Self-reported affect is often overly positive, whereas implicit, physiologic, and behavioral indicators are more consistent with experiences of negative affective states such as stress and anxiety. These divergent patterns across different levels of analysis are likely to influence stigmatized group members' affective states during interactions, which is where we turn our attention next.

Externalized Negative Affect

Now that we have considered how nonstigmatized individuals respond to interactions with members of stigmatized groups, our focus shifts to understanding the affective states that might occur for the stigmatized individuals involved in these interactions. When considering how individuals may respond to interactions characterized by overt or subtle forms of bias against them, an obvious possibility is that anger will ensue. Indeed, to the extent that unfair or biased treatment is labeled as discriminatory, anger is anticipated to be the modal response (Gibbons et al., 2010; Mendes, Major, McCoy, & Blascovich, 2008). This is not surprising when we consider the range of nonstigmatized persons' behavior (real or perceived) toward stigmatized individuals, which can include discounting, underestimating, ignoring, belittling, dominating, harassing, or disingenuous positivity.

Research confirms the idea that members of stigmatized groups may respond to biased interactions with anger. For example, in studies comparing inter- and intraracial dyads, African Americans receiving negative social feedback from European American confederates (e.g., "I wouldn't be interested in becoming friends") showed greater sympathetic nervous system reactions, more attributions of racial bias, and more observable anger behavior (raised voice) compared to African Americans receiving the same type of negative feedback from African American confederates (Jamieson, Koslov, Nock, & Mendes, 2013; Mendes et al., 2008). This constellation of physiologic, behavioral, and attributional responses indicates an approach-oriented affective state most consistent with anger (Carver & Harmon-Jones, 2009; Herrald & Tomaka, 2002).

Attributional ambiguity theory offers a useful framework to identify when individuals would be more likely to experience anger versus a more internalized negative response, such as shame or sadness (Crocker & Major, 1989). Attributional ambiguity is the idea that stigmatized individuals live in an environment in which there are extra attributional explanations for positive and

negative outcomes that they experience. When individuals perceive unfair treatment such as racial bias, the cause for negative evaluation, rejection, or unfair treatment can be internal—the stigmatized person had a personal flaw—or external—the person who rejected them was biased against their social group. External attributions, such as bias, trigger externalizing responses like blame and anger. Anger experiences might trigger a host of physiological changes that over time create more accumulated physical damage. However, anger reactions are far from universal in terms of the short-term physiologic change (Kassam & Mendes, 2013) and long-term health consequence (Kubzansky & Ichiro, 2000), and they may also differ by cultural context (Kitayama et al., 2015). For example, anger expression in East Asian cultures often functions as a dominance display, whereas in Western cultures anger typically functions as a way to vent frustration. These different functions appear to affect long-term health outcomes, such that East Asians who report more dispositional anger tend to have biological indicators suggesting better health (e.g., lower cholesterol, lower blood pressure, and lower immune C-reactive protein) compared to Westerners who report more dispositional anger (Kitayama et al., 2015).

Uncertainty

Moving beyond anger, another common affective response to intergroup interactions is uncertainty. Uncertainty is an aversive state engendered by unknown or unknowable factors (Gao & Gudykunst, 1990; van den Bos, 2001). Feelings of uncertainty may be frequent among the stigmatized because nonstigmatized individuals often show subtle biases or mixed-valenced responses toward them (as discussed previously), making it difficult to interpret their meaning. Resolving uncertainty requires greater monitoring of and alertness to the interaction partners' behavior and environment, and this excessive monitoring, or *vigilance* (Mendes, Gray, Mendoza-Denton, Major, & Epel, 2007; Scherer, Zentner, & Stern, 2004), can create a prolonged state of negative affective responses. A seminal study in this area examined the cognitions and behavior of stigmatized compared to nonstigmatized individuals (Frable, Blackstone, & Scherbaum, 1990). Results showed that stigmatized individuals were more vigilant of their physical environment and consequently remembered more details of the laboratory experience compared to nonstigmatized individuals. These data suggest that stigmatized individuals may be especially vigilant or "mindful" when in new or uncertain situations perhaps in an effort to monitor the physical and social environment for possible threats (Kaiser, Vick, & Major, 2006).

For African Americans, a heightened attention to and vigilance for detecting subtle forms of racial bias have been well documented in a variety of interaction contexts, including getting-acquainted interactions (Mendes et al., 2008), physician–patient encounters (Penner et al., 2010), and teacher–student interactions (Okonofua & Eberhardt, 2015). Being vigilant during interracial interactions with European Americans has pragmatic utility for African Americans because subtle expressions of bias may be the tip of the iceberg, representing a small part of a larger profile of discriminatory behaviors that directly impact the quality of African Americans' lives, including the health care they receive, whether they are hired for a job, and the punishments they are given in school (Okonofua & Eberhardt, 2015).

Complementing and extending these studies with African Americans, a growing body of research in social psychology and social neuroscience suggests that individuals who are lower in social standing, such as those who are from lower socioeconomic status (SES) families or who perceive themselves as lower in social status, may also feel greater anxiety and uncertainty and hence are more vigilant of others during social interactions. For example, Kraus and Keltner (2009) found that lower SES more than higher SES individuals exert more effort and are more engaged during social interactions with strangers, which can lead to better accuracy at reading the emotions of others (Kraus, Côté, & Keltner, 2010). Neuroimaging studies have found that lower status individuals tend to engage brain regions involved in "mentalizing," or thinking about the thoughts and feelings of others, to a greater degree than do their higher status counterparts (Muscatell et al., 2012). Perhaps due to this heightened attentional focus on others, stigmatized individuals also may show biases in the way that they interpret ambiguous social situations. For example, low SES adolescents make more negative, hostile attributions regarding ambiguous social situations, which are related to greater cardiovascular reactivity (Chen, Matthews, & Zhou, 2007; Dodge & Somberg, 1987). Taken together, this work suggests that for some stigmatized persons, the social world can be fraught with uncertainty and require constant monitoring of others and their environment.

The previous examples refer to situations in which stigmatized people do not know how their interaction partners feel about them—situations characterized by attributional ambiguity. Another possible condition occurs when mixed messages are communicated—when interaction partners provide what seems to be positive treatment, but the positive treatment seems to be disingenuous or there are other signals communicated that are counter to the surface-level positive treatment. For example, nonstigmatized individuals may want to cover or suppress the biases they hold toward stigmatized group members, as mentioned previously, and one possible outcome is that they *overcorrect* or act overly positive toward stigmatized individuals so as to not appear biased. When stigmatized individuals perceive a lack of authenticity or detect a discrepancy between explicit and implicit feelings, being the target of overly positive treatment can lead to negative affective responses among stigmatized individuals. For example, Crocker, Voelkl, Testa, and Major (1991) found that African American participants' self-esteem *decreased* after positive evaluations from European American partners.

This question was extended to examine affective responses and physiologic reactivity to positive feedback in a study that randomly assigned African American and European American participants to receive positive social feedback from either an African American or a European American same-sex (confederate) partner (Mendes et al., 2008). Participants then engaged in a time-pressured, cooperative task. For all race–partner combinations except one, positive social feedback resulted in an adaptive physiological response—specifically, increased cardiac output and decreased peripheral resistance. This is an unsurprising finding and consistent with the idea that positive social feedback can engender healthy, salubrious responses to those who receive it. However, for African Americans, positive feedback from a European American partner was associated with maladaptive reactivity—decreased cardiac output and increased vascular resistance—suggesting a response that was more malignant. Moreover, a similar pattern was observed in behavior and performance outcomes. Behavior during the cooperative task was coded for *vigilance*—operationalized as how often the participant looked away from the computer task and toward the partner. African American participants who received positive feedback from a European American partner monitored their partner more and showed greater vigilance during the task compared to all other race combination pairings. Not surprisingly, given this distracted behavior, performance was also lower for African Americans who received positive feedback from a European American partner compared to the other dyads.

Why did positive feedback from European American partners lead to negative affective and physiological responses from African American participants? One possibility is that African American participants did not trust the positive feedback given by European American partners, which triggered greater vigilance possibly in an attempt to determine the authenticity of the feedback. To explore whether distrust of majority group members was a critical factor, Major, Sawyer, et al. (2013) developed a measure to tap the extent to which stigmatized group members are *suspicious* of nonstigmatized group members' motives to act in an egalitarian manner. Across three studies, Latinos who scored high on this suspicion measure reacted to positive feedback from European American peers with increased reported stress, heightened uncertainty, decreased self-esteem, and cardiovascular reactivity consistent with threat responses (Major et al., 2016).

A recent study attempted to directly test the role of trust in interracial interactions using a pharmacological manipulation assumed to be directly related to trust—oxytocin. In a placebo-controlled, double-blind study, African American participants who received a placebo intranasal spray and received positive social feedback from European American confederates showed similar affective responses as those described previously—greater threat reactivity and more vigilance. In contrast, African American participants who received oxytocin and the same positive feedback showed adaptive physiological responses and were significantly more trusting of their European American partners in a monetary trust game (Park, Flores, Woolley, & Mendes, 2017). Results from this study point to the potentially critical role of trust in interracial interactions and suggest the possibility that for some stigmatized individuals, positive feedback may be perceived as less trustworthy and therefore associated with a cascade of negative affective and physiological responses.

In summary, interactions between nonstigmatized and stigmatized individuals are likely to be fraught with uncertainty, which can engender vigilance and greater attention to others on the part of members of stigmatized groups. Nonstigmatized individuals may also give off mixed messages

regarding their true feelings in such interactions, which may hamper trust and lead to greater suspicion of motives for their stigmatized interaction partners. The accompanying physiologic activation stemming from these affective states may have health consequences over time.

Anxious Affect via Physiologic Influence

Another pathway by which affective responses to stigmatization may influence health is via *physiologic influence* (lower left hand corner of Figure 14.1). The idea here is that if stigmatized individuals are more likely to be vigilant for detecting bias, and nonstigmatized interaction partners are likely to display anxious responses, then the combination of these factors may place stigmatized targets into a prime position to "catch" the anxious affect of their partner. To the extent that daily life provides multiple incidences of these subtle transmissions, it might lead to cumulative wear and tear on the body over time for members of stigmatized groups.

One way to measure transmission of anxious affect is with physiologic influence—the extent to which individuals' physiological responses change as a function of their partner's physiological reactions. Prior research has shown that in interpersonal encounters, observing or interacting with others experiencing activated emotions can engender physiological changes in the observer (Buchanan, Bagley, Stansfield, & Preston, 2012; Butler et al., 2003; Levenson & Gottman, 1983; Soto & Levenson, 2009). Although previous work has focused on physiological influence among close others, such as romantic relationships or mother and child, recent work has shown that newly acquainted dyads mutually influence each other and can "catch" the affective states of their partner (West, Page-Gould, Koslov, Major, & Mendes, in press).

Recent research also highlights the potential usefulness of physiologic influence in understanding stigma–health relationships. Among strangers during a competitive interaction, men who were randomly assigned to a high-status position were more likely to have physiologic influence on their lower status partners than the reverse. In other words, lower status men were more likely to "catch" the affective responses of higher status males, as evidenced by mirroring the physiologic changes of their partner in a time-lag design (rather than lower status men driving the physiologic response of higher status men). When higher status partners showed an increase (or decrease) in sympathetic nervous system responses, lower status partners showed the same physiological change in the next time unit (i.e., 30 seconds later; Kraus & Mendes, 2014). Reversing the model—using lower status partners' responses to predict changes in higher status partners' reactivity—showed no physiologic influence from lower to higher status members. Physiologic influence in itself is not necessarily a maladaptive response—infants show the same physiologic changes as their mothers (Waters, West, & Mendes, 2014). However, to the extent that lower status and stigmatized individuals are "catching" the anxious arousal of individuals interacting with them, this might lead to a greater cumulative toll on the wear and tear of their physiologic systems.

Interracial dyads (specifically, European American and African American dyads) show a similar pattern of physiologic influence as lower and higher status male dyads (West et al., in press). When African American participants were paired with anxious European American partners—defined as partners who showed greater cortisol increases or more observable signs of avoidance or who self-reported more discomfort—the European American partner's physiology was more predictive of the African American participant's physiologic changes than the African American partner's physiology was predictive of the European American participant's physiologic changes. That is, there was more physiologic influence between high anxious European American individuals and their African American partners than there was between low anxious European Americans and their African American partners. Anxiety did not moderate the other dyadic combination—anxious African Americans did not have physiologic influence on European American partners, nor did anxious European Americans have an influence on same-race partners. This suggests that not all anxiety is "caught"; rather, intergroup anxiety seems to be especially contagious for African Americans interacting with highly anxious European Americans. Further supporting these findings, African American participants who were higher in race rejection sensitivity—that is, who were anxious and expected to be rejected due to their race (see Chapter 20, this volume)—were more likely to show physiologic influence from their European American partners. These findings suggest a possible pathway through which stigmatized individuals might experience more activation of their biological systems during social interactions with people who are uncomfortable or anxious during their interactions with them.

Direct Paths from Affective Responses to Physiologic Changes

In this section, we delve more deeply into the neurobiological changes that can follow from the three activated affective responses identified previously: externalized negative affect stemming from overt or subtle negative treatment, uncertainty or vigilance from ambiguous situations, and anxious affect that can be experienced in anticipation or "caught" from an anxious interaction partner (Figure 14.1, solid lines). Recent theorizing on the biology underlying the relationship between negative affective responses and physiologic changes suggests several candidate biologic pathways that might be linked to long-term health outcomes for members of stigmatized groups. We consider these pathways and highlight some of the complexity associated with straightforward interpretations that specific affective states trigger unhealthy physiologic responses leading to poor health.

It is important to note that there is no simple one-to-one mapping of an affective state and a physiologic response. To generate plausible pathways from affective states to health outcomes via physiologic pathways, multiple physiologic responses should be considered. In addition, the *context* and *temporal trajectory* (which we describe later) can shed light on how these responses might affect health (Kassam, Koslov, & Mendes, 2009; Muhtadie, Koslov, Akinola, & Mendes, 2015; Obradovic, Bush, Stamperdahl, Adler, & Boyce, 2010). In the following sections, we briefly review the most commonly studied biologic systems: neural activation, neuroendocrine and immune responses, biomarkers (cellular alterations), and the autonomic nervous system. For each of these systems, we focus on how stigma might influence short- and long-term changes and how these changes might affect health outcomes.

Neural Responses

In recent decades, the burgeoning field of social neuroscience and increased use of brain imaging technologies in social and health psychology studies have begun to shed light on how neural responses to social interactions may be implicated in linking stigmatization and health. One important line of inquiry suggests that heightened vigilance, involving thinking about or anticipating others' thoughts or actions, might affect the health of stigmatized individuals through neural pathways. In particular, greater activation of mentalizing-related neural circuits (circuits associated with thinking about or representing others' minds) may start a cascade of physiological stress responses that, over time, could lead to allostatic load (i.e., alterations in the set points of physiologic systems) and poorer health (McEwen, 1998a). For example, one key mentalizing-related brain region, the dorsomedial prefrontal cortex (dmPFC), has dense anatomical projections to the amygdala and other brainstem neural structures that are critical for initiating activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal cortical (HPA) axis (Robinson, Charney, Overstreet, Vytal, & Grillon, 2012). As such, greater dmPFC-related attention to others may lead to the activation of other neural regions and subsequent physiological changes that can lead to bodily "wear and tear" over time (Muscatell et al., 2015, 2016). Thus, vigilance can activate dmPFC responses, initiating a cascade of peripheral physiologic changes intimately tied to allostatic load.

As described previously, situations of uncertainty can engender vigilance to make sense of what is happening. In contrast, situations in which bias is overt can engender attributions to discrimination and externalizing negative affect such as anger. Attributing overt negative social experiences to discrimination has been shown to have protective effects in terms of neural responses, at least in the short term. In one of few studies to investigate this issue, Masten, Telzer, and Eisenberger (2011) exposed African American participants to an episode of social rejection by two European American confederates who excluded the participant from an online ball-tossing game (known as cyberball). Following the rejection experience, participants were asked to make judgments regarding why they were excluded from the game. Interestingly, participants who attributed their rejection to discrimination (i.e., "They rejected me because of my race") showed lower levels of activity in the dorsal anterior cingulate cortex (dACC) compared to those who made internal attributions for their rejection. Given that dACC activity has been implicated in the processing of pain, fear, and other negative affective states (Eisenberger, Lieberman, & Williams, 2003), these results are consistent with behavioral work suggesting that making external attributions during negative social experiences may serve a self-esteem protective function for members of stigmatized groups, at least in the short term (Crocker & Major, 1989). Presumably, the external attribution shifted the negative experience from self-blame to other-blame, which lessens experiences of shame, fear, and pain typically associated with social rejection and

leads instead to perceptions of unfairness resulting in more anger. Much more work is needed to fully understand the neural effects of feeling stigmatized and how these responses may be associated with health outcomes.

Neuroendocrine System

The neuroendocrine system most commonly implicated in affective-health links is the HPA axis. The HPA axis is typically measured with its end-product, cortisol, and the underlying biological pathway starts with signals received at the hypothalamus that trigger the release of corticotrophin-releasing hormone (CRH). CRH then triggers the pituitary to release adrenocorticotropic hormone (ACTH), which stimulates the adrenals to release hormones, including cortisol. Cortisol is commonly examined by stress researchers who compare cortisol levels during a resting state to cortisol levels following exposure to an affectively charged event.

Relevant to stigma–health questions, research suggests that passive tasks (e.g., noise exposure or watching films) may not reliably increase cortisol responses, but tasks that are active and include elements of uncontrollability and/or social evaluation reliably lead to increases in cortisol (Dickerson & Kemeny, 2004). Although cortisol is commonly conceived of as a "stress hormone," it is important to note that cortisol increases do not invariantly relate to negative affect. Instead, cortisol increases can occur during sustained mental effort and active tasks that lead to approach behavior (or challenge; Dienstbier, 1989; Koslov, Mendes, Patjas, & Pizzagalli, 2011; Lovallo & Thomas, 2000). Also, the context most often studied among researchers interested in cortisol reactivity is a standardized stressor called the Trier Social Stress Test (TSST), which requires participants to give a speech and complete a difficult mental arithmetic task in the presence of two stoic evaluators. This context is a mix of social evaluation, uncontrollability, and one that requires cognitive effort and mental demand. This creates a bit of a puzzle for stigma–health researchers because an increase in cortisol might indicate an experience of uncontrollability, social evaluation, and negative affect—elements that are consistent with stress—but it might also indicate some benign state such as intense and prolonged mental effort. Furthermore, a lack of cortisol increase may indicate disengagement with the social context, which could also be harmful for health over time if this leads to social isolation and learned helplessness. Thus, like all "biomarkers" (biological responses that presumably relate to health outcomes), the context is critical to interpreting whether the response might be "adaptive" or "maladaptive."

In addition to examining reactivity to an affectively charged situation, HPA functioning can also be quantified over the course of a day. Cortisol level follows a diurnal cycle in which it peaks at wake and then declines throughout the day until it reaches its waking nadir in the evening hours. HPA daily functioning can provide insight into how the affective states of individuals influence neuroendocrine functioning, and it represents a plausible biological pathway that affects health (for a review, see Prather, 2016). For example, in depressed patient samples, exaggerated HPA axis functioning is indicated by higher cortisol observed in the evening (Stetler & Miller, 2011), whereas patients with post-traumatic stress disorder (PTSD) show alterations in daily cortisol marked by flattened diurnal cortisol rhythm including low levels of cortisol in the evening (Daskalakis, Lehrner, & Yehuda, 2013). In the context of stress, timing and severity of stressors play a role in driving deviations in daily cortisol rhythms. Data from a meta-analytic review of 107 studies demonstrated that cortisol initially rises in response to the beginning of a chronic stressor but then decreases as that stressor persists (Miller, Chen, & Zhou, 2007). This suggests that acute and novel experiences of discrimination might result in high levels of daily cortisol initially, but if feelings of stigmatization are chronic and pervasive, HPA activation might show more blunted diurnal cortisol responses (Adam et al., 2015; Fuller-Rowell, Doan, & Eccles, 2012).

An illustrative study in this area examined cortisol levels throughout the day in a sample of African Americans and European Americans. Fuller-Rowell et al. (2012) found that African Americans had lower cortisol awakening response (defined as the initial cortisol increase 30 minutes after waking) compared to European Americans, consistent with a less healthy profile among stigmatized group members. However, when examining responses across the entire day and the effects of perceived discrimination, a very different picture emerged. European Americans who reported more discrimination had flatter (unhealthier) diurnal cycles, consistent with the idea that discrimination might alter cortisol levels throughout the day in an unhealthy way. However, African Americans who reported more discrimination showed a healthier diurnal cycle—a robust cortisol awakening response followed by a steep decline throughout the day (Fuller-Rowell

et al., 2012). Thus, more labeling of discrimination among African Americans was associated with healthier neuroendocrine profiles.

The study by Fuller-Rowell et al. (2012) was cross-sectional—perceived discrimination was measured at the same time as daily cortisol responses. Interestingly, a different pattern of diurnal cortisol was observed with a longitudinal data set in which perceptions of discrimination were measured during adolescence and diurnal cortisol was obtained in early adulthood (early 30s). In this study by Adam et al. (2015), higher cumulative discrimination in adolescence predicted flatter cortisol slopes in early adulthood for both African Americans and European Americans, but importantly, among African Americans only, experiencing discrimination during adolescence predicted lower cortisol wakening responses as a young adult. Replicating the Fuller-Rowell et al. study, cross-sectional analyses revealed that for African Americans, experiencing discrimination as an adult was associated with larger cortisol awakening responses as an adult. We highlight these findings to demonstrate the importance of considering the chronicity and timing of stigmatization measures and how length of experiences of stigmatization might produce different effects on biological systems.

Immune System

Although historically the immune system was thought to operate "from the neck down" with no input from the central nervous system, the field of psychoneuroimmunology has established that immune system activity is in fact sensitive to psychosocial and affective inputs. The most commonly studied component of immune system activation in the context of stigma and affective science research is the inflammatory response and inflammation. Inflammation is the primary response of the immune system, and it forms the body's "first line of defense" against injury or infection. The inflammatory response is orchestrated by proteins called pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-α, and systemic inflammation is also often measured in levels of C-reactive protein (CRP). Interestingly, systemic inflammation is implicated in a number of chronic diseases (e.g., cardiovascular disease, arthritis, and diabetes) and psychiatric disorders (e.g., major depressive disorder and PTSD), and it has been shown to increase in response to both acute and chronic stressors. Thus, inflammation is a strong candidate biological system for linking affective responses to stigmatization and health.

Some research suggests that members of stigmatized groups exhibit heightened levels of inflammation that might be associated with affective states. Lewis, Aiello, Leurgans, Kelly, and Barnes (2010) found that older African American adults who reported higher levels of everyday discrimination also had higher basal levels of CRP. However, results may be nuanced and further influenced by factors such as gender and racial identification. For example, other investigators reported associations between self-reported discrimination and CRP only among African American women and not African American men, and only at more moderate amounts of perceived discrimination (Cunningham et al., 2012), whereas yet others have found associations between discrimination and inflammation in youth, but only among those adolescents with low levels of positive racial identity (Brody, Yu, Miller, & Chen, 2015). Taken together, these results suggest that although African Americans may be at risk for heightened levels of inflammation due to their social and affective experiences, these relationships are complex and are likely moderated by other demographic and psychosocial factors.

With regard to affect-induced inflammatory reactivity, a few studies have found that members of lower status or stigmatized groups tend to show stronger inflammatory responses than nonstigmatized group members to acute stress. For example, African American women had greater increases in levels of IL-6 in response to an evaluative speech task compared to White women (Christian, Glaser, Porte, & Iams, 2013), and lower SES individuals have been shown in numerous studies to exhibit exaggerated inflammatory responses to various negative affect-inducing experiences such as negative social feedback (Brydon, Edwards, Mohamed-Ali, & Steptoe, 2004; Derry et al., 2013; Muscatell et al., 2016) compared to higher SES individuals. Research on nonstigmatized populations suggests that the affective states of shame and anxiety are especially likely to be associated with increases in levels of inflammation (Carroll et al., 2011; Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009; Moons & Shields, 2015), with more mixed results when anger is the dominant emotion experienced (Carroll et al., 2011; Moons & Shields, 2015). Thus, whether or not an individual experiences threat and anxiety or anger in response to stigmatization experiences may play a critical role in predicting inflammatory reactivity. Empirical research is necessary to test this prediction and to increase understanding of the links between stigma, affect, and inflammation.

Pre-disease Biomarkers: Circulating Angiogenic Cells

Although the biological measures reviewed thus far provide evidence linking affective states experienced by stigmatized individuals with health outcomes, these biological changes are rather distal to underlying disease processes. *Biomarkers* are biological changes that are not simply concomitant with affective and health outcomes but, rather, are directly implicated in the disease pathway. Pre-disease biomarkers have the added value that changes in these biological responses precede frank disease. One pre-disease biomarker that is receiving attention is that of circulating angiogenic cells (CACs; previously referred to as endothelial progenitor cells). CACs are a prime example of a pre-disease biomarker given that they are sensitive enough to reflect subtle changes in health status among young and midlife individuals with early stage endothelial dysfunction (a precursor to atherosclerosis) or metabolic syndrome but who do not yet have frank atherosclerotic plaques (Chen, Yiu, & Tse, 2011). In other words, they can help identify individuals who are at risk for atherosclerosis and other cardiovascular events but who have not yet fully developed such disease states, making such individuals prime targets for intervention.

CAC function can be examined in vitro, which reflects the capacity of CACs to migrate toward sites of tissue damage, where they promote repair via paracrine effects (e.g., further secretion of growth factors) (Urbich et al., 2005). CAC migration is decreased in patients with coronary artery disease (Vasa et al., 2001), atherosclerosis (Ohtsuka et al., 2013), and diabetes (Thum et al., 2007). In animals, delivering CACs or CAC-conditioned media to sites of ischemic vascular injury can regenerate damaged tissue (Kalka et al., 2000; O'Loughlin et al., 2013), further confirming their critical role in contributing to disease and dysfunction.

In a study by Aschbacher et al. (2016), CACs were examined among African Americans who completed a lab study in which they interacted with a same-sex European American stranger. Measures of trait anxiety and affective responses stemming from the social interaction, including threat (increased demands relative to resources) and disengagement (withdrawal from the situation), were also obtained. A month later, African American participants completed a blood draw, and the blood samples were then used in cell culture studies to test the mobility of the cells. Results showed that self-reported threat states resulting from a social interaction with a European American stranger were associated with lower CAC migration (i.e., poorer cell mobility), which is associated with pre-disease states. That is, African American participants who perceived more threat and reported feeling more disengaged during a social interaction with a European American stranger had indications, at the cellular level, that they were on an early path to develop heart disease. Although this work was based on a small, cross-sectional sample, was correlational, and did not offer a comparison same-race interaction or other control group, these data represent a potentially novel pathway linking stigma and disease, given that CAC function is directly implicated in the pathway to heart disease.

Autonomic Nervous System

The physiologic system perhaps most commonly studied in the context of stigma and affect research is the autonomic nervous system (ANS). The ANS functions, in part, to mobilize oxygenated blood from the heart to peripheral sites such as arms, hands, legs, feet, and the brain. The ANS, broadly representing the overlapping sympathetic, parasympathetic, cardiovascular, and enteric systems, is closely tied to affective states. This relationship is not surprising given that emotion/acute stress and ANS changes share similar temporal features. Both are short-lived experiences that typically last a few seconds to several minutes. This can be contrasted with neural activation, which is often measured in milliseconds, or neuroendocrine and immunological changes, which are measured over hours or days. In addition, affective responses are perceived as being "felt" in the body, and folk language implicates bodily changes in affective processes. Feeling sick to one's stomach when experiencing disgust, a racing heart when walking down a dark and deserted street, or hot and sweaty palms when filled with fury seem to effortlessly couple the affective state and the bodily change. Given this apparent natural coupling, it is not surprising that a large literature has amassed examining the relation between affective experiences and ANS changes.

The ANS is often implicated in stress–disease models. Dysregulation of these systems can be indicated by either hyper-elevation or hyporesponsiveness when individuals are at rest or in response to a physical or psychological task. The underlying assumption is that dysregulated levels of ANS responses may indicate disease risk, pre-disease indicators, or symptoms of underlying disease. In health research, the most commonly studied

responses include blood pressure levels, heart rate, and skin conductance. However, the prevalence of these measures in research is, in part, due to the ease with which they can be collected rather than due to their utility in predicting health outcomes and disease processes. Less commonly studied responses, such as changes in blood flow, cardiac output, or digestive changes, have also been linked to health outcomes (Jefferson et al., 2010), but they are considerably more expensive and time-consuming to collect.

Sympathetic nervous system (SNS) activation, typically thought of as the "fight-or-flight" response, can be triggered from a variety of affective states, including many of the negative affective states identified previously, such as acute stress, anger, threat, and anxiety. SNS responses can also increase in response to more positive affective states, including excitement, challenge, and interest (Kreibig, 2010; Mendes, 2016). Thus, simply knowing whether there was an increase in SNS responses provides little information regarding the affective experience or mental state of the person, nor is there clear evidence that SNS activation is necessarily harmful for health. Indeed, a characteristic marker of aging is a gradual decline in the ability to mount a strong SNS response (Mendes, 2010).

Related to SNS is the parasympathetic nervous system (PNS), typically thought of as the "rest and digest" system. Although lay belief assumes SNS and PNS are reciprocal, these systems can operate relatively independently (Berntson, Cacioppo, & Quigley, 1991). The most commonly assessed measure of PNS is heart rate variability (HRV), which estimates the influence of the cardiac vagus nerve in modulating heart rate. At a physical health level, lower levels of cardiac vagal tone have prospectively predicted weight gain and have been linked to increased prevalence of cardiac infarctions and greater morbidity and mortality (Thayer & Lane, 2007). In general, lower levels of HRV are associated with being older, heavier, more sedentary, and a greater likelihood of mental and physical illness. Changes in HRV (HRV reactivity) can occur when the cardiac vagal nerve withdraws, resulting in less variability and hence lower HRV. Changes in HRV also occur when the cardiac vagus nerve activates and modulates heart period, which increases HRV. The current literature shows both beneficial and detrimental effects of decreased HRV reactivity: Greater HRV decreases have been linked to greater attentional focus on mental effort but also to negative emotions such as anger, anxiety, sadness, and acute stress responses.

Temporal Trajectories of Acute Autonomic Nervous System Reactivity Responses

In addition to considering the overall magnitude of a specific measure of ANS activation in a given study, it is also important to consider the temporal trajectory that indicator is following in response to an acute affective state. Indeed, the acute reactivity approach to understanding health outcomes makes assumptions regarding how affective experiences (e.g., stress and emotion) bring about acute changes (reactivity) in biological systems, such as ANS responses, which might accumulate over time to create excessive wear and tear on biological health (McEwen, 1998a). Scholars who use this approach typically expose participants to standardized tasks such as watching videos, giving evaluated speeches, or engaging in social interactions that activate physiologic changes, and then they interpret the profile of the resulting activation as maladaptive or harmful to health. In the simplest case, the "reactivity hypothesis" examines physiologic changes from a resting state to an activated state, with the assumption that the greater the activation, the more harmful the physiological response would be if experienced repeatedly. For example, in a study in which women described being unjustly accused of shoplifting, African American women who reported experiencing past discrimination had greater diastolic blood pressure compared to African American women who reported little experiences of prior discrimination in their life (Guyll, Matthews, & Bromberger, 2001). The authors of this work interpreted the findings as showing that "discrimination may act as a stressor that adversely affects cardiovascular health and that the effect may be mediated by pathogenic events associated with physiologic reactivity" (p. 322). Although the general reactivity hypothesis is intriguing, it likely cannot yield the full story on how stigma affects health. For example, in the study by Guyll et al., European American women who completed the same "discrimination" task showed larger blood pressure reactivity compared to African American women. Thus, reactivity might be part of the pathway from affective experiences to health outcomes, but the simple interpretation of "more is worse" is not sufficient.

Intensity of reactivity provides a snapshot of how individuals respond, but examining a more dynamic profile over time may provide a more comprehensive understanding. Figure 14.2 presents

four different trajectory profiles of reactivity (cf. McEwen, 1998b). A maladaptive (or unhealthy) response in anticipatory reactions would be characterized by a heightened response prior to the onset of an event. As depicted in Figure 14.2, anticipatory responses might create more wear and tear on the system because of the lengthened reactivity that precedes an event. For stigmatized individuals, this could be a function of having negative expectations for a social interaction, test, or job interview, which might be reflected in increased vigilance or anxiety, as described previously. These negative expectations might be especially harmful when transitioning to new environments. Intervention studies that target minority students at the beginning of their university career show that belongingness interventions can ultimately improve academic performance (Walton & Cohen, 2007). Psychologists who use these interventions note the importance of introducing them early in the semester, which presumably would reduce anticipatory anxious affect and vigilance for cues of bias before they were able to fully take hold (Yeager & Walton, 2011).

Unhealthy physiologic responses can also be characterized by the lack of recovery once a stressor is over. As depicted in the top right corner of Figure 14.2, whereas "healthy" reactivity is characterized by a return to baseline levels once a stressor is over, an unhealthy response would show a continued elevation in reactivity once the stressor has ended. Rumination in particular has been implicated in poor post-stress recovery (Nolen-Hoeksema, 2000). Individuals with stigmatized identities might be particularly prone to rumination because they are often left with ambiguous and uncertain reactions following an interaction (Hatzenbuehler, Nolen-Hoeksema, & Dovidio, 2009; Kaiser & Miller, 2001). To the extent that unfair treatment and discrimination create greater feelings of uncertainty, stigmatized individuals might show elevated responses following an event.

When responding to a novel event, a typical adaptive physiologic response would include an initial strong activation that coordinates metabolic systems to contend with the task at hand but also fairly quick habituation (Figure 14.2, bottom left), which has been labeled a "physiologically tough" response (Dienstbier, 1989). In contrast, a lack of habituation during a stressor (Figure 14.2, bottom left) might reflect an inflexibility of the system to

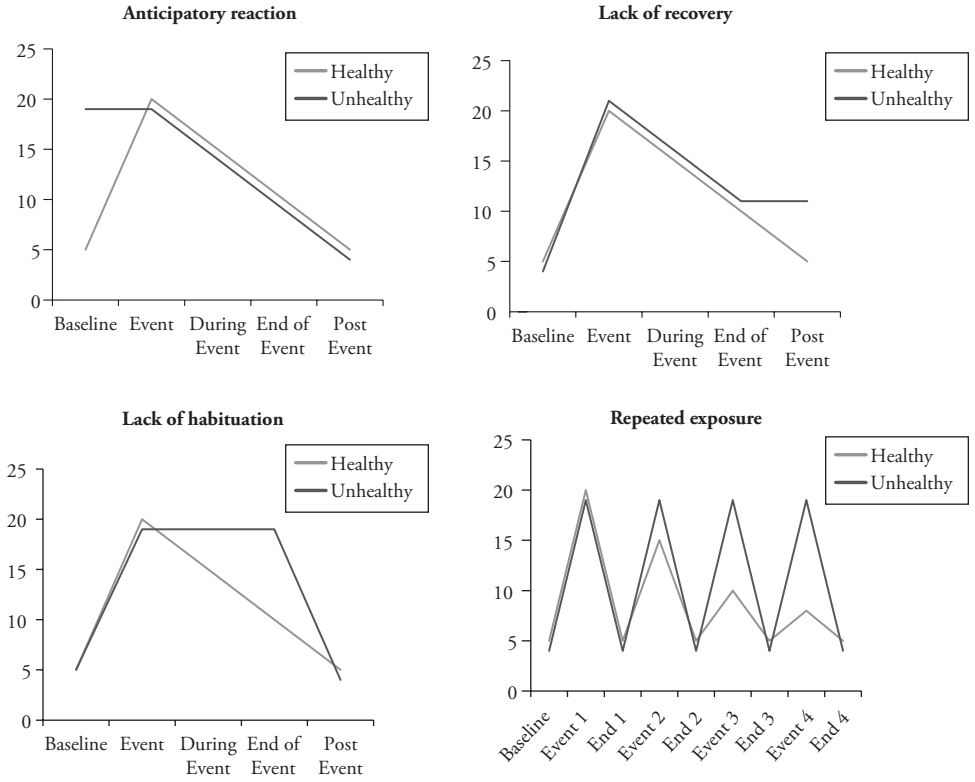

Figure 14.2 Conceptual differences in acute sympathetic nervous system activation.

quickly adapt, which may also ultimately create excessive wear and tear. To the extent that stigmatized individuals are hypervigilant during a task, this might result in a lack of habituation.

Finally, repeated exposures to stress are typically characterized by an initial large response followed by smaller responses. For example, the first day of school, a job interview, or a first date all bring about a large initial physiologic response. However, repeated exposure to the same predictable environment is associated with an increasingly lower response. For stigmatized individuals, if the environment continues to feel unpredictable and uncertain, the same environment might continue to require a strong metabolic response, and habituation to repeated events may not occur or may take longer to show the same decline. In one study in which women completed social evaluation stressors (delivering speeches and completing difficult math tasks in the presence of stoic evaluators) on successive days, overweight and weight-conscious women were more likely to have repeated high levels of cortisol reactivity on successive days completing the TSST compared to non-overweight women or those who were less weight conscious, even though the two groups showed similar initial stress responses to the task (Epel et al., 2000). This suggests that feelings of stigmatization might result in repeated exposure to high levels of anxious arousal or general stress.

Indirect Pathways from Affect to Health: Potentiating Unhealthy Behaviors

Much of stigma–health research has attempted to answer the question of how stigma influences health outcomes by adopting the approach described previously—examining how perceptions of discrimination or negative social interactions trigger affective states associated with acute or chronic maladaptive physiological responses that lead to biological changes underlying physical health problems. This *direct* pathway approach is supported by animal research showing that exposure to stressors such as confined spaces, impoverished social environments, and predator environments (compared to neutral or enhanced environments) relates directly to biological alterations, disease, and mortality (McEwen, 1998b). However, support for direct pathways is limited by the impossibility of random assignment to social environments, the complexity of human social life, and the fact that affective states to disease processes might not be solely or even primarily an effect of a direct pathway. Instead (or at least in addition), poor health due to stigmatized status might be influenced indirectly via behaviors triggered by affective states (Figure 14.1, dashed lines). In this line of reasoning, affective responses trigger behavioral responses that can be health damaging both acutely and over time (see Chapter 11, this volume).

From a general stress perspective, Jackson, Knight, and Rafferty (2010) speculated that racial disparities in physical health compared to mental health could be related to how individuals cope with stress. They found that among European Americans, stress exposure combined with unhealthy behavior (excessive drinking, smoking, and poor eating habits) resulted in greater depression. However, among African Americans, stress exposure was associated with depression when it was *not paired* with unhealthy behavior. These authors speculated that among African Americans, engaging in unhealthy behaviors was a coping mechanism in response to discrimination that might protect mental health but increase exposure to physical health problems because of health-damaging behavior.

Another line of work in this domain explores the relationship between stigmatization, anger, and health behavior. In general, anger is considered an "approach-oriented" affective state (Carver & Harmon-Jones, 2009). This approach orientation, although it has some positive aspects such as motivating collective action, is also related to risk-taking. For example, individuals who were dispositionally angry or were induced to feel anger perceived less risk in their environment (Lerner & Keltner, 2001). In a longitudinal study of minority adolescents, perceptions of discrimination predicted substance use over time, and this effect was mediated by the experience of anger (Gibbons et al., 2012; see Chapter 19, this volume).

Experimental evidence that discrimination could lead to greater risk-taking via anger was provided in a study that examined risk-taking responses after in-group versus out-group social rejection (Jamieson et al., 2013). As noted previously, out-group social rejection (being rejected by a different race partner) is more likely than in-group rejection to be attributed to discrimination and more likely to increase feelings of anger. In this study, White and Black participants interacted with two individuals in a computer "chat room." The two individuals were represented as being from either the same racial group or a different racial group as that of the participant. All participants then received negative

interpersonal feedback and were rejected by the online partners. Consistent with other studies, being rejected by out-group partners was associated with more observed anger reactions, greater SNS activation, and lower cortisol responses compared to rejection from in-group partners. Immediately after the rejection, participants completed a risk-taking task (i.e., the Columbia Card Sort Task; Figner, Mackinlay, Wilkening, & Weber, 2009), which provided estimates of overall risk-taking and also the extent to which individuals were sensitive to reward information versus loss information. Out-group rejection (compared to in-group rejection) was associated with more self-reported anger, greater risk-taking, and more reward sensitivity. Reward sensitivity provides an indication of the extent to which individuals are riskier when rewards are higher and has been linked to greater likelihood of addiction and gambling (Reuter et al., 2005; Volkow et al., 2010). These data suggest that when discrimination engenders more anger, risk-taking behaviors, such as risky driving, unsafe sex, drug use, and gambling, might increase.

Although informative, anger is only one possible affective response that is triggered by stigmatization. As described previously, stigmatized individuals may also show more attentional vigilance for social and environmental threats. This exerted mental effort during social interactions might leave fewer cognitive resources or "mental bandwidth" for other tasks (for a similar argument regarding stereotype threat, see Schmader, Johns, & Forbes, 2008). Evidence for a pathway from greater attentional vigilance to poorer executive control derives from work in social neuroscience that suggests that effortful social cognition, such as trying to decode what others are thinking and feeling, engages both mentalizing-related neural systems and "executive control" neural systems (Meyer, Spunt, Berkman, Taylor, & Lieberman, 2012). Because they are likely engaging these executive control regions during social interactions with nonstigmatized others, stigmatized individuals may deplete their cognitive resources and thus be less able to engage executive control networks during future situations (Murphy, Richeson, Shelton, Rheinschmidt, & Bergsieker, 2012). This could lead to poorer emotion regulation, greater likelihood of consuming palatable but unhealthy food, heightened tendency to use alcohol and drugs and to gamble, and so on (Heatherton, Herman, & Polivy, 1991; see Chapters 11, 19, and 27, this volume). Evidence for this possibility is currently limited, however, and it will thus be important for future work to examine if the greater tendency of stigmatized individuals to focus on social interactions affects their neural activation and behavior during subsequent self-control tasks or situations that require executive control.

Conclusion

In this chapter, we reviewed theories and empirical data on affective responses associated with stigmatization as a way to provide a useful framework to understand why stigmatized individuals might have poorer physical health. We emphasized how affective states generated by biased interactions, including anger, uncertainty, and anxiety via physiologic influence, can alter neural activity and HPA, immune, and ANS activation; influence pre-disease biomarkers; and lead to changes in behavior. Although an important body of evidence is beginning to accumulate examining these processes, much more work is needed in this area to further explicate how stigmatization influences affective states and the biological and behavioral consequences of such experiences. Our hope is that future work in this area will look across the range of affective experiences, the multiple neurobiological systems that are implicated, and the multitude of health-compromising behaviors that might be triggered so that we can establish a more comprehensive understanding of how stigma might influence health via affective processes.

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