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Neural and Cardiovascular Pathways from Stigma to Suboptimal Health

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The Oxford Handbook of Stigma, Discrimination, and Health
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Abstract and Keywords

This chapter reviews research from the emerging field of social neuroscience to examine the underlying mechanisms that explain why stigma and discrimination lead to suboptimal health outcomes. The review is structured around three pathways through which stigmatization has negative effects on physical health, and it discusses neural and cardiovascular processes associated with (1) the stress that being a target of discrimination elicits, (2) impaired self-regulation of health behavior among targets of discrimination, and (3) how intergroup dynamics during interactions between health care provider and patient can result in suboptimal health care for stigmatized individuals. The insights offered by the neuroscience perspective provide crucial information on how to interrupt the downward stigma–health spiral and can inform policy to reduce the impact of stigma and discrimination on the physical health of its targets.

Keywords: cardiovascular mechanisms, doctor–patient interactions, neural mechanisms, self-control, social neuroscience, social exclusion, stress and coping, social pain, suboptimal health behavior

This book provides an overview of theory and research that help to explain why targets of stigma, whether due to an individual condition associated with one’s appearance or health or due to membership in a socially devalued group, have poorer health outcomes compared to nonstigmatized individuals. In this chapter, we add to these insights by reviewing research on stigma and discrimination from the emerging field of social neuroscience.

Since the early 2000s, researchers studying stigma, prejudice, and intergroup relations have started to incorporate neuroscientific measures in their work (for an overview, see Derks, Scheepers, & Ellemers, 2013). Partly, this was inspired by the realization that many of the more traditional research tools to examine the behavior of agents and targets of prejudice, such as self-reported attitudes and behavioral measures, would only yield

insight into the more overt and explicit psychological processes. However, in a research field in which many of the processes under investigation (e.g., prejudiced attitudes and strategies to cope with discrimination) are subject to social norms, desired self-views, and denial, and in which many processes unfold outside of people's awareness, additional measures were needed to better comprehend the underlying mechanisms that explain the pervasive nature of prejudice.

The introduction of neuroscience and psychophysiological measures to study cognitive and affective processes related to stereotyping and prejudice has yielded novel insights into the multilayered nature of stigma and prejudice, both from the perspective of the target and from the perspective of the perpetrator. By delineating different neural pathways, the aim of this chapter is to clarify the link between the experiences of stigmatized individuals (e.g., stereotypical expectations of others, being evaluated negatively, and social exclusion and rejection) and the suboptimal health outcomes among the stigmatized that are reported in other chapters of this handbook. (p. 242)

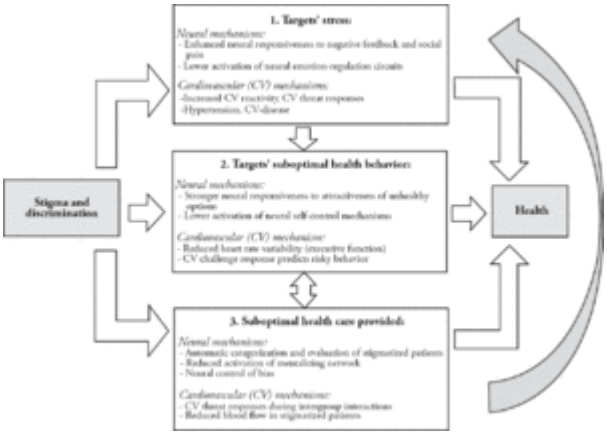

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Figure 13.1 The three psychophysiological pathways explaining the link between stigma and suboptimal health outcomes.

Drawing upon the model proposed by Major, Mendes, and Dovidio (2013), we structure our review around three pathways through which stigmatization has negative effects on physical health (Figure 13.1). First, we focus on the stress that being a target of discrimination elicits. We then discuss impaired self-regulation in the context of health behavior among targets of discrimination. Finally, we present studies revealing how intergroup dynamics during interactions between health care provider and patient can result in suboptimal health care for targets of discrimination/stigma. In our review of the neuroscientific processes underlying these three pathways, we address both central and peripheral parts of the nervous system and focus on studies examining brain activation (central nervous system), on the one hand, and cardiovascular (peripheral) responses, on the other hand.

The nervous system (Figure 13.2) consists of two different parts: the central nervous system (CNS) and the peripheral nervous system (PNS), of which the autonomic nervous system (ANS) is an important subdivision. The main function of the CNS, which consists of the brain and the spinal cord, is to integrate and process all information that comes from the body and the external world. Brain activity is most often examined using one of two techniques: electroencephalography (EEG), which is used to distinguish between

different stages in the way in which people process incoming information, and functional magnetic resonance imaging (fMRI), which is employed to identify the different brain structures (e.g., prefrontal cortex and amygdala) in which these processes occur.

The main function of the ANS (Figure 13.3) is to (mainly unconsciously) regulate the activity of bodily functions, such as heart rate, respiration, and digestion. The ANS consists of two different branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is the “action” system that prepares the body to deal with the demands of the (social) environment. For example, a classic and well-documented example of SNS activity is the “fight-or-flight” response, which results in a variety of bodily changes such as increased heart rate (HR), respiration rate, and skin conductance. By contrast, the PNS is the “recovery” system, which is mainly active when the organism is at rest. For example, the PNS plays a major role in digestion. Although the SNS and the PNS typically (p. 243) work in opposition of each other (when activity of one increases, activity of the other decreases), most of the time both SNS and PNS are to some extent active. In the current overview, we focus on *cardiovascular* (CV) measures, such as HR and blood pressure (BP), which are mainly under SNS control (i.e., when sympathetic activity increases, HR and BP typically also increase). We also review work on heart rate variability (HRV), which forms an index of PNS activity; specifically, when parasympathetic activity increases, HRV typically increases.

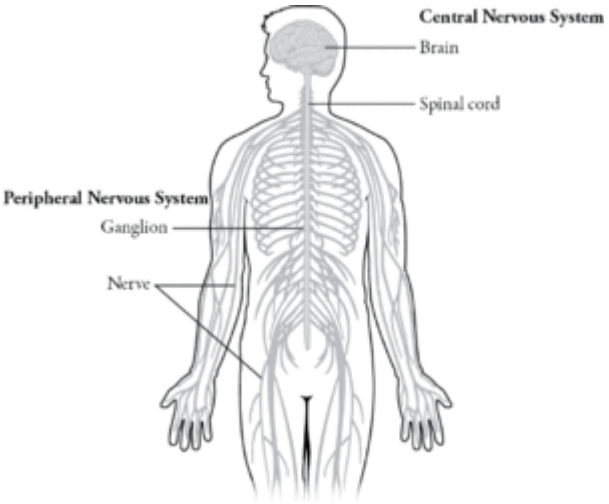

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Figure 13.2 Organization of the human nervous system.

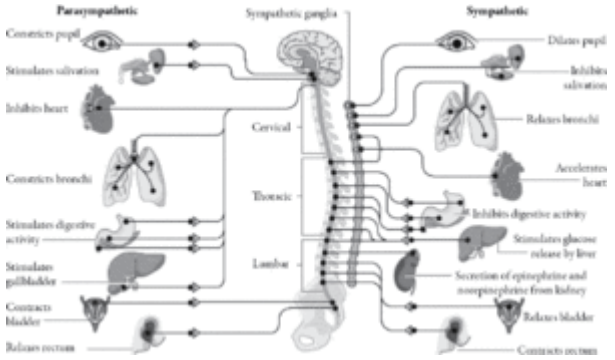

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Figure 13.3 The autonomous nervous system.

Before turning to a description of research on the neural pathways from discrimination to health, two important points must be made. First, the research described in this chapter is structured along a distinction between CNS activity (as measured by (p. 244) EEG/fMRI) and ANS activity (as measured by CV activity). It is important to note, however, that the two systems are strongly intertwined. For example, under stress, the "neural alarm system" (described in more detail later) that is part of the CNS triggers the SNS to regulate action to deal with the demand (Wager et al., 2009). The reverse effect also occurs: Particular types of heart activity (e.g., HRV) influence brain activity (Thayer & Lane, 2007).

A second issue concerns another layer of complexity, namely that in addition to CNS and ANS pathways, there are other important biological systems that are relevant for the relation between discrimination and health. The neuroendocrine system is a first one that comes to mind. However, because these influences are discussed in more detail in other chapters of this handbook (i.e., see Chapters 12 and 14), we do not discuss them here.

Pathway 1: Stigma Induces Stress and Maladaptive Coping

The first pathway by which stigma and discrimination lead to suboptimal health outcomes is through the stress that negative life experiences that accompany social devaluation can induce (see Chapters 5, 9, and 11 this volume). In the past 20 years, there has been a growing interest in conceptualizing stigma and discrimination as stressors with which people need to cope (for an overview, see Miller & Major, 2000). Individuals who have a stigmatizing condition, such as a physical deformity, or who suffer from collective stigma due to their race or gender have to face a higher quantity of daily stressors related to prejudice and discrimination. Moreover, although research on stigma and coping shows that stigmatized people may develop ways of coping with this threat, higher stress levels wear out the cardiovascular system and lead to higher morbidity and mortality among the stigmatized (Clark, Anderson, Clark, & Williams, 1999).

Research on physiological correlates of stigma helps to substantiate the direct link between stigma and stress. For example, it has been shown that people with lower socioeconomic status (SES), a condition that can be both a cause and a consequence of

discrimination and that can be experienced as a threat to identity (Johnson, Richeson, & Finkel, 2011), are more likely to develop physical and psychological illnesses and die prematurely (Adler et al., 1994). Importantly, health disparities between people with low versus high SES cannot be attributed completely to structural differences in material resources, illiteracy, or lower quality health care. In addition to the effects of objective indicators of SES, health outcomes can be predicted by *perceived* social standing. This means that two people with equally low objective SES can develop differential health outcomes depending on their *subjective* interpretation of their position on the social ladder (Marmot, 2004). Consider, for example, that a low SES student's subjective experience of stigma is higher when attending an elite university rather than a community college (Johnson et al., 2011). The more that people with low SES experience stigma because of their low social position, the more they show impaired stress regulation and recurrent biobehavioral stress responses that increase risk for ill health in later life (Gianaros et al., 2008).

Next, we present a review of research showing how the brain processes experienced stigma, followed by a discussion of how cardiovascular responses triggered by stress may lead to suboptimal health.

Neural Mechanisms Underlying the Stigma–Stress–Health Pathway

Emerging research examining how the brain processes experienced stigma not only uncovers how people process *single* stigmatizing events but also finds that *repetitive* experiences with stigmatization render the brain increasingly sensitive to detect and process cues that signal rejection and less able to regulate negative emotions. In addition to discussing this research, we review research that suggests that the brain processes social pain caused by stigma as if it were physical pain.

Brain Responses to Stereotype Threat

The first line of work we review finds that being primed with negative stereotypes about one's group leads people's brains to pay extra attention to negative feedback and failure. Stereotype threat is experienced by members of negatively stereotyped groups when they are in a situation in which they run the risk of confirming a negative stereotype about their group (Steele & Aronson, 1995). For example, stereotype threat is triggered when women perform a math test or when Black Americans take a test of their intellectual abilities after being reminded of the negative stereotypes concerning their group's performance in these domains (Schmader, Johns, & Forbes, 2008). The anxiety, self-doubt, and performance monitoring that are triggered by a negative stereotype lower people's working memory capacity and reduce the ability to perform optimally. As such, stereotype threat introduces an additional (p. 245) burden that is not experienced by nonstigmatized individuals, which over time may affect their health outcomes negatively (see Chapter 5, this volume).

Using different neuroscience techniques, researchers have revealed that stereotype threat induces people to scan their environment for negative feedback and regulate the negative emotions that are triggered by these negative stereotypes. For example, in an EEG study, Forbes and Leitner (2014) found that compared to women in a stereotype-neutral context, women experiencing stereotype threat while performing a math test paid more attention and recruited more working memory resources (increased interaction between the anterior cingulate cortex [ACC] and dorsolateral prefrontal cortex [dlPFC]) to process negative rather than positive feedback. This may have prevented them from using all their cognitive resources for optimal performance on the math task. In addition, in an fMRI study, Krendl, Richeson, Kelley, and Heatherton (2008) found that women performing a math test under stereotype threat not only showed reduced recruitment of regions associated with math performance but also enhanced activation of the ventral ACC, an area associated with the processing of affective information and emotion regulation. Combined, these studies suggest that being reminded of negative group-related stereotypes indeed adds stress, inducing people to regulate their emotions and be more vigilant for cues that signal that they may be confirming the negative group-related stereotype.

Brain Responses to Social Exclusion

A second line of work showing how stigma-induced stress can lower health outcomes focuses on the neural circuitries that are triggered when people experience social exclusion. Being excluded and ignored is one of the many negative life events with which individuals who are stigmatized or discriminated against are confronted (Link & Phelan, 2001). A large body of work shows that the social pain associated with social exclusion and rejection activates a neural alarm system that detects and triggers responses to environmental threats (Eisenberger & Lieberman, 2004). This system involves (but is not limited to) the dorsal anterior cingulate cortex (dACC) and anterior insula (AI).

For example, Eisenberger, Lieberman, and Williams (2003) tested participants in an fMRI scanner while they experienced social exclusion in a virtual ball-tossing game. Specifically, after being included for a couple of throws by two virtual throwers, participants no longer received any throws for the rest of the game. Results revealed that compared to included participants, excluded participants showed increased activation in the dACC and AI. Interestingly, these two brain regions have been associated with the processing of the affective (vs. sensory) component of physical pain, suggesting that social pain actually “hurts.” Similar results have been found for other types of social pain, such as negative social evaluations (Eisenberger, Inagaki, Muscatell, Haltom, & Leary, 2011) or disapproving faces (Burklund, Eisenberger, & Lieberman, 2007).

Differential activation of the neural alarm system is linked to objective health outcomes because it activates the SNS and the hypothalamic–pituitary–adrenal (HPA) axis. The SNS and HPA axis are involved in the regulation of the immune system’s inflammatory responses (for an overview, see Muscatell & Eisenberger, 2012) as well as the cardiovascular system (Brotman, Golden, & Wittstein, 2007). Inflammation is a defensive response triggered by the immune system in response to injury or illness. However, repeated or prolonged inflammation can contribute to the development of serious medical conditions such as cardiovascular disease, type II diabetes, asthma, arthritis, osteoporosis, and Alzheimer’s disease. In addition, activation of the SNS and HPA axis potentially harms the cardiovascular system because it increases heart rate and blood pressure, over time “wearing out” the arteries, as well as reducing insulin sensitivity and hemostasis (preventing and stopping bleeding) (Brotman et al., 2007). As such, repeated exposure to situations that activate the neural alarm system—for example, when being excluded repeatedly by others due to a stigmatizing condition—can damage one’s health.

Stigma also negatively impacts health through the partial overlap in neural circuitries triggered by social and physical pain: Experiences of the two types of pain may interact such that people who experience frequent social pain will be more sensitive to physical pain and vice versa. Indeed, Eisenberger, Jarcho, Lieberman, and Naliboff (2006) reported that individuals who experienced more social distress after having been experimentally excluded also rated pain stimuli as more unpleasant. Similarly, Black American patients reported more physical pain during a 4-week time period when they had experienced more racial discrimination in that time as well (Burgess et al., 2009). These results

suggest another way in which social rejection and exclusion experienced by stigmatized individuals may (p. 246) negatively impact their health: They will more likely experience pain as distressing and less bearable.

The research discussed so far has focused on discrete experiences with social rejection. However, being a member of a stigmatized group means that one experiences social rejection and isolation repeatedly. Neuroscientific research suggests that negative effects of social rejection accumulate so that previous experiences with rejection leave people even more sensitive for social (and possibly also physical) pain later in life and reduce their ability to regulate negative emotions effectively. One study found that 12th-grade students who had reported more social isolation in a 2-week diary study showed more activation in the dACC and AI while experiencing experimentally manipulated social exclusion 2 years later (Masten, Telzer, Fuligni, Lieberman, & Eisenberger, 2012). Similarly, individuals who are hypersensitive to social rejection, due to prolonged experiences with rejection by significant others or due to membership in a devalued group, show stronger activation of the dACC when confronted with disapproving faces (Burklund et al., 2007).

Applying these findings to individuals growing up with social rejection due to a stigmatizing condition or membership in a devalued group suggests that over time they may develop stronger sensitivity to cues that signal rejection and as such experience stronger negative health consequences due to social pain. Indeed, Gianaros and colleagues (2008) found that individuals growing up in low SES families develop an increased neural sensitivity to social threats. In an fMRI study, undergraduate students who grew up with parents they retrospectively perceived as having low social standing showed stronger amygdala activation to threatening faces compared to students who perceived their parents as having a higher social position. Stigma due to low SES may even affect how the brain develops: Gianaros et al. (2007) found that in addition to the effect of objective indicators of SES, people who subjectively perceive that they have low social standing—for example, because they have experienced discrimination due to their low SES—show reduced gray matter volume in the perigenual anterior cingulate (pACC). The pACC is a paralimbic region that is associated with adaptive emotional, neuroendocrine, and autonomic responses to environmental and psychological stressors. Although cross-sectional, this finding could mean that repeated exposure to the stress of low social standing during one’s life remodels the pACC in a similar way as has been previously documented for early childhood stressors and post-traumatic stress disorder (for examples, see, Cohen et al. [2006], who found that traumatic adverse life events in childhood were predictive of a smaller ACC and caudate nuclei, and Karl et al. [2006], who showed that post-traumatic stress disorder is associated with, among other regions, smaller ACC). Moreover, neuroanatomic changes of the pACC could predispose people with subjectively low SES to show maladaptive coping responses to psychological stressors, increasing their vulnerability to mental and physical illness later in life.

The study by Gianaros and colleagues (2008) may suggest that repeated exposure to social devaluation (i.e., low subjective SES) not only makes people more vigilant to detect threats (i.e., through a more vigilant neural alarm system) but also may reduce their ability to regulate negative emotions effectively. In addition, a relatively recent study found that individuals who reported low subjective childhood SES showed reduced activation of the right ventrolateral PFC (rVLPFC) when excluded in a virtual ball-tossing game (Yanagisawa et al., 2013). The rVLPFC is hypothesized to be important for self-control and the regulation and inhibition of social distress (Eisenberger et al., 2003). This finding therefore suggests that individuals who grew up in a family they perceived as having low social standing are less able to deal with social distress when others reject them.

Coping Strategies

One important qualification to the work we reviewed so far is that there is also research suggesting that stigmatized individuals develop coping strategies to protect them against social rejection, thereby insulating them against the negative health consequences of stigma-related stress. For example, a study of Black students who were excluded by Whites in a virtual ball-tossing game (Masten, Telzer, & Eisenberger, 2011) found that some Black participants who believed that the other players in the game excluded them due to their race did not show the characteristic brain activation associated with social pain (e.g., dACC). Moreover, they showed stronger activity in regions that have been associated with successful regulation of threat responses (e.g., rostral anterior cingulated cortex [rACC]). This suggests that attributing social exclusion to discrimination may help the stigmatized cope successfully with negative treatment, reducing possible negative effects for their health.

Similarly, an fMRI study revealed that when psoriasis patients (psoriasis is a highly visible (p. 247) disfiguring medical skin condition) were presented with disgust-bearing faces, they showed less insula activation compared to control subjects without skin disease (Kleyn et al., 2009). At the same time, psoriasis patients were less able than control subjects to distinguish between different levels of disgust. Importantly, this effect was specific for disgust: There were no differences between patients and controls in brain and behavioral responses to fearful faces. The authors suggest that this could indicate that, over time, psoriasis patients may have developed a coping strategy that allows them to disregard expressions of social rejection that their condition may trigger in others. Thus, whereas research on social exclusion suggests that experiencing social exclusion triggers neural circuitry that are negatively related to health outcomes, individuals who can attribute social exclusion to an external cause may not experience social exclusion as a threat that triggers their neural alarm system (Crocker & Major, 1989).

It is important to note, however, that coping with stigma by attributing negative treatment to discrimination is only an option when the stigmatizing condition is visible to others. This suggest that whereas Black Americans and people with a visible medical condition may be able to protect their self-esteem by attributing negative outcomes to their stigmatizing condition, people with low SES or an invisible medical condition, although equally vulnerable to social rejection, will be less able to attribute this to external causes (Crocker, Voelkl, Testa, & Major, 1991; for a discussion of concealable stigma, see Chapter 15, this volume).

Cardiovascular Mechanisms Underlying the Stigma–Stress–Health Pathway

In addition to the neural mechanisms that are triggered by stigma-induced stress, CV responses to experienced discrimination or stigmatization form an important pathway leading to reduced health. Specifically, people who experience more stress—for example,

due to their stigmatized status—are more likely to suffer negative health consequence due to the detrimental effects of stress on the cardiovascular system (Steptoe & Kivimaki, 2013).

On the basis of the biopsychosocial model (Blascovich, 2008), it is possible to distinguish negative stress (threat) from positive stress (challenge) on the basis of specific patterns of CV responses. During demanding situations in which people experience stress, their SNS activates the heart muscle to pump faster (increased HR) and with more force (increased ventricular contractility [VC]). When people assess that they have the resources to deal with the demands of the situation, this leads to a CV response profile indicative of challenge, in which blood vessels dilate (lower total peripheral resistance [TPR]), which in combination with increased cardiac activity (HR and VC) leads to increased cardiac output (CO; the amount of blood pumped out by the heart). This challenge pattern is a benign cardiovascular state characterized by relatively stable blood pressure whereby blood can flow through the arterials to muscles, glands, and the brain. However, when people assess that they do not have the resources to deal with the demands of the situation (or in terms of neural processes, when stress triggers the neural alarm system), this leads to a CV response profile indicative of threat. During threat, blood vessels contract (increased TPR), providing less room for blood to flow (stable CO), despite the increased HR and VC, leading to a maladaptive cardiovascular state characterized by relatively high blood pressure.

Thus, under threat, the heart and the vasculature work in opposition, “wearing out” the arteries (Sapolsky, 1994). This causes injuries to coronary arteries and the buildup of arterial plaque, in turn leading to lack of oxygen in the heart muscle and ultimately heart attacks. Moreover, the buildup of plaque in other arteries (e.g., in the brain) may lead to other health issues, such as stroke and lower arterial elasticity. In addition, repeated increases of blood pressure lead to failure of hemodynamic regulatory processes and, in turn, to chronically high blood pressure (i.e., hypertension).

Meta-analytic evidence shows that stronger CV reactivity to laboratory stressors is indeed predictive of the development of cardiovascular disease (Chida & Steptoe, 2010). There is also evidence that poor CV *recovery* from stressors is predictive of the development of cardiovascular disease (Panaite, Salomon, Jin, & Rottenberg, 2015). Not surprisingly, the cardiovascular pathway from stigma to reduced health has received considerable research attention (for overviews, see Braveman, Egerter, & Williams, 2011; Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Clark et al., 1999; Couto, Goto, & Bastos, 2012; Pascoe & Richman, 2009; see also Chapters 11 and 14, this volume). This research has provided considerable evidence for a relation between (perceived) discrimination and a variety of CV outcome variables, ranging from blood pressure reactivity to the development of hypertension. In addition, this work has also identified important (p. 248) moderating variables of the relation between discrimination and CV outcome variables. These moderators are discussed next, beginning with the type of discrimination, followed by individual differences in coping ability.

Type of Discrimination

The relationship between discrimination and CV outcome variables is complex, and to understand this relationship and design interventions to attenuate it, it is important to draw distinctions between different forms of discrimination. In their review of the relationship between discrimination and CV outcomes, Brondolo and colleagues (2011) distinguished among three forms of discrimination: interpersonal discrimination (receiving, as an individual, discriminatory treatment from another individual), internalized discrimination (acceptance by minority group members of negative stereotypes and prejudice against their group), and institutionalized discrimination (policies and institutions that treat minority group members unequally).

Brondolo et al. (2011) found the strongest evidence for relations between interpersonal discrimination and blood pressure reactivity and between institutionalized discrimination and the development of hypertension. There was no clear relationship between internalized discrimination and CV outcome variables, which may be explained by the idea that for people who accept discrimination against their group, encountering instances of discrimination is no longer (extremely) threatening because this fits their expectancies and worldview (Townsend, Major, Sawyer, & Mendes, 2010). Finally, Brondolo et al. propose that although stress is the main process in the relationship between interpersonal discrimination and blood pressure, lifestyle factors (e.g., obesity) also play an important role in the relationship between institutionalized discrimination and hypertension. Thus, when designing specific interventions for the negative health outcomes resulting from experienced discrimination, it is important to consider the different processes through which different forms of discrimination negatively impact these health outcomes.

Research addressing the distinction between more “subtle” and more “blatant” forms of discrimination has indicated that blatant forms can sometimes be easier to deal with compared to subtle forms (Crocker & Major, 1989). In a study by Merritt, Bennett, Williams, Edwards, and Sollers (2006), Black participants were confronted with a description of blatant racism or with a description of a similar situation in which it was more ambiguous whether racism played a role. Interestingly, this latter, more ambiguous situation yielded stronger blood pressure increases (and slower recovery) compared to the situation in which racism was more blatant (see also Guyll, Matthews, & Bromberger, 2001). Similarly, in the context of gender discrimination, Salomon, Burgess, and Bosson (2015) showed that although hostile (i.e., more blatant) sexism did initially elicit stronger CV reactivity in women, benevolent (i.e., more subtle) sexism led to slower recovery to baseline levels. In other words, although the initial impact of hostile sexism was greater, the CV effects of benevolent sexism lasted longer.

Individual Differences in Coping Ability

Despite the fact that being the victim of discrimination can generally be viewed as a stressor, there are considerable individual differences in the level of stress caused by being such a victim, which reflect differences in the ability to cope with discrimination.

Here, we provide an overview of individual difference variables and dispositions that moderate the relation between perceived discrimination and CV responses. We discuss the influence of prior encounters with discrimination, trait anger, social support seeking, and social identity.

First, previous experiences with racial discrimination increase CV reactivity to new race-based stressors. Guyll et al. (2001) measured the extent to which Black American women attributed past negative treatment to discrimination. The participants delivered a speech on how they would respond to a situation in which they were accused of shoplifting (i.e., a possibly racist situation) while blood pressure was measured. Results indicated that participants who had encountered racism in their life more often showed stronger increases in blood pressure compared to participants who had encountered racism in their life less often. Guyll et al. concluded that Black persons who experience more discrimination are more vulnerable to developing hypertension and CV disease compared to Black persons who experience less discrimination.

A second moderator of the relationship between experienced discrimination and CV responses is whether people who encounter discrimination experience and express anger (see Chapter 19, this volume). Epidemiological studies have revealed that the relation between racial discrimination and higher resting state blood pressure is particularly strong for those high in trait anger (Clark, 2006a). (p. 249) In addition to the inclination to respond with anger to negative events, whether anger in response to racism is inhibited or expressed has been found to impact CV recovery (Dorr, Brosschot, Sollers, & Thayer, 2007). Whereas expressing one’s anger generally facilitates CV recovery, this did not happen for Black Americans who expressed their anger about the racist attitudes of a debating partner. Dorr et al. explained this by suggesting that expressing anger is the socially inappropriate way for Black Americans to respond. This means that for Black Americans, either expressing or inhibiting their anger in response to racist events delays CV recovery.

A third factor moderating the relationship between experienced discrimination and CV responses is the inclination of the target to seek social support (Clark, 2006b). This notion fits with the idea that being embedded in a good social network, and receiving social support when experiencing adversity, is a chief predictor of a variety of health outcomes. Clark found that Black women who delivered a speech on a neutral topic showed higher blood pressure reactivity to the degree that they perceived they had experienced discrimination in the past (see also Guyll et al., 2001). However, this relationship was attenuated for participants high in support seeking.

In addition to support seeking, Cooper, Thayer, and Waldstein (2014) showed the positive effects of prayer for CV recovery after racism-related stress. Black women reported the extent to which they dealt with racism by means of praying and then recalled and relived an incident in which they were the victims of racism. Coping by means of prayer led to less CV reactivity and quicker recovery after recalling an incident involving racial

discrimination. Together, the studies reviewed here provide evidence that support has an important stress attenuating function for targets of discrimination.

Finally, there is evidence that social identification—that is, the extent to which people identify with their group—makes people more vulnerable to experience CV responses indicative of threat due to discrimination. Eliezer, Major, and Mendes (2010) examined the interaction between gender identification and the perceived pervasiveness of gender discrimination on CV threat responses. Female participants who varied in how central gender was in their self-concept read that sexism was either rare or prevalent and then gave a speech relaying this information to another person. Women who read that gender discrimination is pervasive (vs. rare) showed more threat CV reactivity irrespective of level of identification. However, women low in gender identification recovered from threat associated with pervasive sex discrimination more quickly than did women high in gender identification. Thus, although high group identification among members of stigmatized groups is necessary to instigate collective action and to establish social change, in the short term, this may be at the expense of one’s health.

Summary of Research Findings: Pathway 1

The research on the neural and cardiovascular processes that are triggered when people experience stress due to their stigmatized status reveals some underlying mechanisms by which the added stress of stigma can negatively impact health. First, the research reviewed reveals the brain circuits that enable people to detect and process single experiences with discrimination and social rejection, suggesting that experienced stigma is processed as a physically painful event. Moreover, experienced stigma triggers maladaptive cardiovascular responses that over time may result in negative health outcomes such as cardiovascular disease, diabetes, asthma, and Alzheimer’s disease. Although there is work suggesting that the stigmatized may have developed coping strategies that allow them to be less affected by acute experiences of stigma (e.g., attributing rejection to discrimination and searching for social support), a growing body of research also suggests that life-long experiences with being stigmatized may take a toll (Sapolsky, 1994). This is because stigmatizing conditions may over time change how people process and respond to their social world, inducing people to scan their environment for signals of social rejection and exacerbating cardiovascular reactivity to stigma- (and even non-stigma-)related stressors. Ultimately, experiencing stigma and discrimination can even remodel the brain, predisposing stigmatized individuals to show maladaptive coping responses to psychological stressors and thereby increasing their vulnerability to mental and physical illness later in life.

Pathway 2: Stigma Encourages Suboptimal Health Behavior

Apart from the direct negative effects that stigma-induced stress can have on the health of stigmatized individuals, the suboptimal health decisions members of disadvantaged groups may make because of their experience of stigma (see Chapter 19, this volume) represent an indirect pathway to health. (p. 250) The stress of social devaluation can lead people to escape or avoid stigma-related stress through coping strategies that directly damage their health, such as smoking, overeating, using drugs and alcohol, and behaving in a risky manner (Pascoe & Richman, 2009). In addition, coping with the stress of stigmatization can indirectly lead to deleterious health consequences through the depletion of self-regulatory ability that it causes (Inzlicht & Kang, 2010).

It is clear that self-control is a crucial factor in maintaining good health. In order to refrain from eating unhealthy foods and smoking, to control one's alcohol intake, exercise on a regular basis, adhere to medical regimes, and refrain from unsafe sex, people need to effortfully inhibit their immediate desire and replace it with behavior that is in line with more abstract health promotion goals. Here, we review research measuring neural and cardiovascular processes to shed light on how stigmatization leaves people less able to override their immediate desires and instead implement behavior that better serves their health.

Neural Processes Explaining Suboptimal Health Behavior Among the Stigmatized

In order to explain the deleterious effects of stigma on health-related behaviors, we first present research showing neural evidence for self-control failure in the moment in which people experience stress due to stigma. Then we present evidence that the negative effects of stigma on self-control failure also spillover to when they have left the threatening situation.

Stigma Reduces Self-Control in the Moment

Evidence for neural mechanisms that help explain self-control failure due to stigma can be found in research that examines how brain processes that are associated with executive control are affected by stress and coping. In the previous section, we discussed research that suggests that acute stigma-induced stress may, on the one hand, increase vigilance for detecting threats (Eisenberger et al., 2003) but, on the other hand, reduce the ability to self-regulate in the moment (Yanagisawa et al., 2013). When examining how stigma reduces self-control in the health domain, we find a comparable pattern, with stigma not only increasing the attractiveness of unhealthy options but also reducing the ability to self-regulate one's behavior.

Maier, Makwana, and Hare (2015) found that acute stress reduced people's ability to control their eating behavior, leading them to choose tasty food over healthy food. Neuroimaging results revealed that this effect was due to two processes. First, acute stress made unhealthy food more attractive. The amygdala and ventral striatum of participants under stress, compared to participants who did not experience stress, were more responsive to tasty rather than untasty food choices. Furthermore, people under acute stress showed a greater connectivity between the amygdala and ventral striatum, on the one hand, and the ventromedial prefrontal cortex (vmPFC), on the other hand. This suggests that stress not only makes the brain respond more strongly to tasty versus less tasty foods but also increases the impact that the taste of food has on the decision-making process. Second, the more participants reported being stressed, the less connectivity there was between the vmPFC and the dlPFC during food choices that required the most self-control (e.g., choosing healthy but less tasty food over tasty but unhealthy food). This finding is consistent with earlier work that implicated the dlPFC in self-control when overcoming food temptations (Hare, Camerer, & Rangel, 2009). Applying these results to the stress that members of devalued groups experience suggests that they may be less able to control their health behavior while under stress because unhealthy options become more appealing and brain mechanisms that serve to apply self-control are activated less.

Stigma Spills over to Nonthreatening Situations

Apart from the effects that stigma may have on self-control during acute stigma-related stress, research has begun to look beyond the stressful situations that members of devalued groups experience to examine what happens when people leave these threatening environments (Inzlicht & Kang, 2010). Many common coping strategies to deal with stigma-induced stress, such as attempting to ignore social rejection, downregulating negative emotions, or maintaining academic motivation regardless of negative stereotypes, require effort and self-control (Inzlicht & Gutsell, 2007; Inzlicht & Kang, 2010; Richeson & Shelton, 2007). As a result, coping with the daily stress of stigma can leave people mentally fatigued and with reduced motivation and ability to apply effortful self-control in other life domains, such as their health. Mental fatigue can induce a shift in priorities from "have-to" goals that require mentally effortful control (e.g., maintaining a diet and exercising) to "want-to" goals (p. 251) that lead to immediate gratification (e.g., relaxing, smoking, and consuming sugar; Inzlicht, Schmeichel, & Macrae, 2014), which results in suboptimal health behavior. For example, after performing a math test under stereotype threat, women showed more aggression and ate more ice cream, suggesting that stereotype threat spilled over to when women left the threatening situation, leaving them with reduced ability to apply self-control (Inzlicht & Kang, 2010).

By examining neural indicators of self-control, neuroscience research can shed light on why self-control is reduced. In most studies, the ability to apply self-control is examined by measuring EEG responses while participants perform a test measuring their executive control. Here, executive control refers to their ability to override prepotent responses—

for example, in a Stroop task in which participants have to override their automatic response of reading a presented word (“blue”) and instead name the color in which the word is presented (e.g., red). Importantly, performance on this type of task has been shown to be predictive of health-related self-regulation—for example, consumption of fatty food, medication adherence, and even early mortality (Bogg & Roberts, 2004).

One of the main mechanisms by which people are able to control their performance on tasks requiring response inhibition is the ACC-based *conflict detection system*. This system, which also responds to social exclusion as discussed in the previous section (Eisenberger et al., 2003), detects situations in which desired and actual outcomes diverge and cognitive control is needed (e.g., detecting the need to restrain oneself when offered another beer). This system is then thought to trigger a second regulatory system, based in the PFC (for a review, see Botvinick, Braver, Barch, Carter, & Cohen, 2001). The ACC-based conflict monitoring system is typically studied by measuring an event-related brain potential related to response monitoring that is amplified when participants commit an error in an executive control task (the error-related negativity [ERN]; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN has been shown to originate from the dACC (Van Veen & Carter, 2002).

Event-related potential (ERP) research using this methodology suggests that stigma-induced stress may lead to reduced self-control in the health domain when people have left stressful situations because it *deregulates* the conflict-detection system that people rely on to apply self-control. In fact, when people perform a self-control task when they are anxious or experience negative affect, at first this may actually enhance their conflict monitoring and performance. For example, Wiswede, Münte, and Rüsseler (2009) found that when women received derogatory (compared to encouraging) feedback while they were performing a response inhibition task, they displayed larger ERNs, suggesting that they were more strongly monitoring their performance. Similarly, Forbes, Schmader, and Allen (2008) found that minority students who value academics show stronger performance monitoring (larger ERNs) when they perform a task under threat of the negative stereotype concerning the lower intelligence of ethnic minorities. These findings suggest that stigma-related stress may actually enhance self-control at first because it may enhance people’s motivation to perform well, which results in stronger performance monitoring and initial higher performance.

However, when members of stigmatized groups show enhanced attempts to apply self-control while still in the threatening situation, these attempts to apply self-control have been found to wane over time and reduce the ability or willingness to apply self-control on subsequent tasks. For example, Luu, Collins, and Tucker (2000) showed that although negative affect enhanced neural error monitoring in the first 200 trials of a response inhibition task, these enhanced levels of error monitoring then declined to levels similar to those of individuals who did not experience negative affect. Consequently, reductions in error monitoring, as examined with the ERN, can also explain reductions in the ability to control behavior later on (the spillover effect). Inzlicht and Gutsell (2007) found that when people deal with negative emotions by effortfully suppressing them, this lowers

their ability to apply executive control (as measured with the ERN) in a subsequent task. In their study, participants were asked to suppress their emotions while watching an emotional movie, after which they had to perform a response inhibition task. Compared to a control condition in which no emotion suppression was required, participants who had effortfully suppressed their emotions while watching the movie were less able to inhibit prepotent but incorrect responses in the subsequent task. Moreover, their neural responses revealed that this was due to the fact that their neural system for detecting errors (as measured with ERNs) no longer responded when they committed an error in the task.

This result was replicated by Wang, Yang, and Wang (2014), who additionally showed that a (p. 252) different type of coping with negative emotions did not result in reduced self-control. That is, participants who were induced to *reappraise* their emotions by adopting a neutral attitude to the movie and thinking about it analytically showed similar ERNs as those of control participants when they made an error. Combined, these results suggest that when individuals deal with negative emotions due to stigmatization by suppressing them, this may leave them less able to apply self-control in other life domains. However, dealing with stigma by reappraising the stress it causes might allow for successful coping that does not harm one's ability to show self-control in the health domain.

Finally, direct evidence for the role of disabled conflict monitoring as a result of dealing with *stigma*-induced stress was reported by Inzlicht and Kang (2010), who found that women who had first performed a math test under stereotype threat indeed showed lower performance on a subsequent executive control measure (indicating that their self-control was impaired). However, the ERP results revealed that stereotype-related stress induced women to show amplified ACC activation so that all types of trials were flagged as relevant and worthy of attention, even those that did not require self-control. This suggests that stereotype threat spills over to situations beyond the stressful situation because it disrupts the ACC performance-monitoring system, thereby impairing self-control.

Cardiovascular Processes Explaining Suboptimal Health Behavior Among the Stigmatized

Although there is less research on cardiovascular processes related to suboptimal health behavior, it has been identified that CV responses play at least two roles in (failed) self-regulation following stigmatization. The first concerns the relation between discrimination, reduced HRV, and impaired self-regulation; the second concerns the relation between discrimination and CV processes that stimulate risk-taking.

Discrimination Impacts Heart Rate Variability

HRV is indexed by the variation in the time interval between heartbeats. High HRV, either at rest (as an individual difference variable) or in response to specific demanding events, is thought to index executive function, effective coping with stress, and high self-

regulatory capacity (Butler, Wilhelm, & Gross, 2006; Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004; Thayer & Lane, 2007). Several studies suggest that discrimination may reduce people's ability to self-regulate by reducing HRV.

HRV is an index of vagal (parasympathetic) neural activity. According to the neurovisceral integration model (Thayer & Lane, 2007), activation of the vagus nerve inhibits the influence of the prefrontal cortex on the subcortical brain structures (e.g., the amygdala) that are involved in emotion and motivation. As a consequence, the downregulation of (negative) affective states is marked by higher levels of HRV (Butler et al., 2006). Although most research in this field is correlational, the hypothesis is that HRV has a causal role in enhancing coping and self-regulation. Indeed, there is evidence that manipulating HRV by means of physical training increases cognitive functioning (Hansen et al., 2004).

High resting-state HRV functions as a resource to navigate through all types of daily stressors (Thayer & Lane, 2007). More precisely, individuals with high resting-state HRV perform better on tasks measuring their executive function and working memory, and they show greater behavioral flexibility when they need to perform under stress. Given its role in self-regulation more generally, it is not surprising that HRV has an important role in regulating health-related behaviors and the ability to inhibit behavior that leads to poorer health outcomes. For example, alcoholics who showed greater HRV reactivity in response to alcohol cues displayed more effective coping in the form of less rumination, less negative affect, and a stronger resistance to drink compared to alcoholics who did not show increased HRV in response to alcohol cues (Ingjaldsson, Laberg, & Thayer, 2003). More generally, given that low HRV indexes poor coping potential, it may not be surprising that reduced HRV is a predictor of cardiovascular disease and mortality and plays a role in a diversity of psychopathologies, such as the development of anxiety disorder (Thayer & Lane, 2007).

Evidence from a study of Black women suggests that HRV decreases as a direct response to experiencing discrimination (Wagner, Lampert, Tennen, & Feinn, 2013). Black women who reported higher levels of experienced discrimination in their lives showed stronger reductions in HRV in response to a racial stressor (being accused of shoplifting) compared to Black women who had experienced less discrimination in their lives. Similarly, a study by Akinola and Mendes (2013) revealed that experimentally induced experiences with low social standing reduce HRV. The (p. 253) implication of this work is that people who experience threat due to stigma or low social standing may also be less able to regulate their behavior in the health domain.

Discrimination Triggers Risky Behavior

In response to discrimination, anger-induced CV challenge profiles can lead to suboptimal health outcomes among the stigmatized by stimulating risk-taking. Jamieson, Koslov, Nock, and Mendes (2013) conducted an experiment on social rejection by same-race versus cross-race others. Being rejected by someone from a different race yielded CV reactivity in line with challenge for both White and Black participants. This fits previous

work showing that challenge, as an approach tendency, relates to anger after group-based rejection (Mendes, Major, McCoy, & Blascovich, 2008). However, Jamieson et al. also found that these anger-related approach tendencies in turn led to increased risk-taking in a card game. Given that risk-taking is a primary predictor of negative health behaviors, what might at first seem a functional and benign response to race-based rejection (challenge) might indirectly also generate negative health outcomes through an increased tendency to take risks. Given that Black individuals are more likely to experience cross-race rejection compared to White individuals, this study suggests that Blacks' repeated exposure to racial discrimination may induce them to take more risks concerning their health, eventually leading to lower health outcomes.

Summary of Research Findings: Pathway 2

In this section, we discussed work that reveals why the stigmatized are less able to apply self-control in the health domain. Coping efforts directed at regulating stigma-induced stress have the negative side effect that they tax people's ability and motivation to apply self-control in other life domains. The work presented previously shows that this occurs because when people are coping with stress, this simultaneously increases the attractiveness of unhealthy options while it reduces the activation of neural circuitry that allows them to detect situations in which they need to apply self-control and to regulate their behavior accordingly. Moreover, stress due to stigma also reduces variability in heart rate, which is hypothesized to be crucial for effective coping and self-regulation. Finally, although some coping strategies seem to tax self-control less than others (i.e., reappraisal vs. emotion suppression), even when targets of stigma believe they are able to cope with discrimination (leading to a CV challenge response), this may leave them vulnerable to risky behavioral choices later on.

Pathway 3: The Stigmatized Receive Suboptimal Health Care

The final pathway discussed in this chapter by which stigma can reduce health outcomes is through the reduced quality of health care that stigmatized individuals receive (see Chapter 10, this volume). Research suggests that interactions between health care providers and patients belonging to stigmatized groups are characterized by intergroup bias and intergroup anxiety (for an overview, see Major et al., 2013). Doctors can have explicit (consciously held) or implicit (unconsciously held) biases toward members of stigmatized groups, leading them to treat members of these groups less warm and friendly and even make different medical decisions—for example, when a doctor needs to decide whether a patient should receive expensive elective surgery (Smedley, Stith, & Nelson, 2003) or pain treatment. Interactions between health care providers and patients with a stigmatizing condition are more likely to be uncomfortable and stressful for both

parties, taxing the executive resources of both the doctor (possibly leading to suboptimal medical decisions) and the patient (reducing his or her ability to understand medical information; Burgess, Warren, Phelan, Dovidio, & van Ryn, 2010; Richeson & Trawalter, 2005).

In this part of this chapter, we present work that helps explain why members of stigmatized groups receive lower quality health care by uncovering neural and cardiovascular mechanisms that are activated when health care providers interact with patients who have a stigmatizing condition (e.g., overweight patients) or who belong to socially devalued groups (e.g., ethnic minorities).

Neural Mechanisms Explaining Why Members of Stigmatized Groups May Receive Low-Quality Health Care

Research on the neural mechanisms underlying social categorization, empathy, and the suppression of prejudiced responses toward stigmatized individuals helps to identify why members of stigmatized groups may receive differential treatment from health care providers.

Neural Correlates of Social Categorization

The first stage in which health care providers may distinguish between patients with and those (p. 254) without a stigmatizing condition is the moment they first meet patients. Research on neural mechanisms underlying person perception reveals that social categorization, the process by which we decide whether someone belongs to our in-group or not, occurs within the first 100s of milliseconds of perception. For example, social categorization based on race is already visible in event-related brain potentials that occur approximately 120 msec after a face is presented (for a review, see Ito & Bartholow, 2009). Similarly, early forms of social categorization have been found in ERPs to other stigmatized groups, such as obese versus normal-weight individuals (Schupp & Renner, 2011), women with versus those without headscarves (Van Nunspeet, Ellemers, Derks, & Nieuwenhuis, 2014), and pictures of homosexual versus heterosexual couples (Dickter, Forestell, & Mulder, 2015).

In parallel, fMRI studies show that the brain responds differently to faces that belong to one’s in-group rather than to an out-group. On the one hand, in-group faces trigger greater activation of the fusiform face area, which is associated with individuation and superior memory of faces (Golby, Gabrieli, Chiao, & Eberhardt, 2001). In addition, in-group faces more strongly activate the striatum (Van Bavel, Packer, & Cunningham, 2008), which is associated with the processing of rewards. Similarly, ERP studies show that in-group faces spontaneously trigger deeper levels of attention (Ito & Bartholow, 2009), especially among individuals who strongly identify with their group (Derks, Stedehouder, & Ito, 2015).

On the other hand, racial out-group members and people belonging to stigmatized groups trigger stronger activation of the amygdala (Harris & Fiske, 2009; Phelps et al., 2000; Wheeler & Fiske, 2005). The amygdala is thought to play a role in the processing of arousing events, such as threatening stimuli, and this amygdala response is therefore interpreted as reflecting an immediate threat response to out-group members (Amodio, 2014). However, this effect of race is reduced for familiar faces (Phelps et al., 2000) or when people are asked to individuate faces (Wheeler & Fiske, 2005).

Combined, this work suggests that patients belonging to stigmatized groups may be perceived differently by health care providers already in the early stages of perception, receiving less individuating attention and eliciting more threat-related processing instead. Importantly, both the reduced individuation processes and the increased amygdala activation are predictive of implicit in-group bias (Derks et al., 2015; Phelps et al., 2000). However, these effects may be attenuated if health care providers make an effort to individuate their patients or over time when patients become more familiar to their doctors.

Neural Processes Underlying Empathy

Another reason why stigmatized individuals receive lower quality health care is that health care providers may feel less empathic toward stigmatized individuals. For example, White physicians have been found to rate pain experienced by Black patients as less intense than pain experienced by White patients (Burgess et al., 2014). As a result, they are less inclined to prescribe pain treatment to Black than to White patients. One of the neural mechanisms that may explain this result is the brain’s response to the pain of others, which is stronger when perceiving people belonging to one’s own rather than a different social category.

Perceiving someone else performing a motor task or experiencing pain triggers neural activation that is similar to processing one’s own experiences. This is called “neural resonance,” and it allows one to understand what someone else is experiencing by sharing the experience at a neural level (Decety, 2011). Neural resonance is found in motor areas when we watch someone else perform a motor task, but it is also found when we watch someone else experience pain. In this case, brain areas associated with the emotional and sensory experiences of pain are activated, including the insula, medial cingulate cortex, periaqueductal gray, and thalamus. Moreover, activation in these areas is related to self-reported empathy for the targets and also helping behavior (Hein, Silani, Preuschoff, Batson, & Singer, 2010).

Studies of neural resonance to the pain of others have revealed that people resonate less with the pain of people who are less similar to them or who belong to an out-group (Hein et al., 2010). Similarly, people demonstrated less activation of areas associated with pain processing when watching obese rather than normal-weight people experiencing pain (Azevedo, Macaluso, Viola, Sanim, & Aglioti, 2014). Decety, Echols, and Correll (2010) showed that neural resonance to perceiving the pain of HIV patients is a function of the attributions of responsibility made for this stigmatizing condition. That is, when

participants thought the patient contracted HIV due to a blood transfusion, neural resonance to pain in terms of activation of the right AI and periaqueductal gray was (p. 255) even greater than that to a nonstigmatized control target. However, when the patient was thought to have contracted HIV through intravenous drug use, neural resonance (i.e., activation in the right AI and anterior midcingulate cortex) was smaller compared to that to the nonstigmatized control target. Gutsell and Inzlicht (2012) found that people showed less neural resonance for sadness expressed by people belonging to ethnic out-groups rather than to their own ethnic group and that this effect was even larger for people who scored higher on prejudice.

Finally, some stigmatizing conditions may even lead health care providers to dehumanize their patients, perceiving them as objects rather than people and denying them of mental states. In two fMRI studies, Harris and Fiske (2009) measured participants' medial prefrontal cortex (mPFC) activation while the participants viewed pictures of objects and people belonging to different social groups. The mPFC is a brain region that is associated with social cognition and mentalizing. Harris and Fiske found that the mPFC was not only activated less to objects than to people but also activated less in response to one specific group of people, namely people belonging to groups that elicit disgust, such as homeless people and drug addicts. Patients belonging to stigmatized groups that are associated with low competence and low warmth, such as poor people and welfare recipients, but also people who are blamed for their illness (e.g., patients with HIV due to intravenous drug use), may be dehumanized by their health care providers. This means that they are viewed as less human, leading health care providers to show less compassion toward these patients, deny them of human experiences such as pain and sadness, and, as a result, provide suboptimal health care. However, Harris and Fiske also revealed an important moderator of the dehumanization effect: Encouraging participants to individuate the people they were shown increased activation in the mPFC even for homeless people and drug addicts—people who were dehumanized in a control condition. Given that health care providers often need to infer individuating information from their patients, the dehumanization effect may be reduced in doctor–patient interactions.

Neural Processes Associated with the Control of Prejudiced Responses

Although many of the neural processes that lead to prejudiced responses toward stigmatized patients are triggered spontaneously, this does not preclude health care providers from monitoring their behavior in order to limit expression of prejudice. Indeed, research shows that many people who interact with members of stigmatized groups are motivated, either for internal or for external reasons, to suppress their biases and behave in unprejudiced ways. Moreover, during approximately the past decade, neuroscience research has uncovered neural mechanisms by which people are able to regulate their behavior so that automatic stereotypes and biases do not seep through (for an overview, see Amodio, 2014). This research points to a neural network that allows people to regulate their responses to members of stigmatized and devalued groups. This network consists of the ACC and the PFC, and it is similar to the previously discussed network by which people regulate their health behavior (e.g., overcome their temptation for

unhealthy food). As theorized by conflict monitoring theory (Botvinick et al., 2001), activation in the ACC serves to detect situations in which conflicts arise between automatic responses (i.e., bias) and the goal to behave in an unprejudiced way. ACC activation then triggers activation in dorsolateral regions of the PFC, which serves to implement goal-directed behavior aimed at responding without prejudice.

In parallel to research on the neural mechanisms of self-control in relation to health behavior, many studies on the conflict detection mechanisms of prejudice regulation have examined the error-related negativity as an indicator of ACC activation. In a typical study, participants perform a task that measures their implicit associations between stimuli related to social groups (e.g., Blacks vs. Whites) and specific stereotypes (e.g., pictures of guns vs. tools) or evaluations (positive vs. negative words) while brain activity is measured by EEG. Several studies have found that people who show stronger conflict detection are better able to suppress their automatic stereotypes and evaluations of stigmatized groups and show unprejudiced behavior. For example, Amodio and colleagues (2004) had participants perform a task measuring implicit associations between Black targets and guns. They found that participants who showed larger ERNs (indicating stronger dACC activation) on trials that were associated with race bias (i.e., mistakenly classifying a tool as a gun when it was preceded by a Black face) were better able to control prejudiced responses.

An important moderator of this effect seems to be whether people are motivated to respond without prejudice for *internal* reasons (because of their personal values) and/or for *external* reasons (because (p. 256) they want to avoid disapproval from others). People who are motivated to respond in an unprejudiced way for internal reasons only (i.e., people with high internal but low external motivation) are best able to monitor their behavior and detect prejudiced responses (Amodio, 2014). Correspondingly, Van Nunspeet and colleagues (2014) found that reminding people of their moral values regarding equal treatment of people from different ethnicities increased conflict detection (as measured with the ERN) in a task measuring implicit negative bias toward Muslim women.

Interestingly, Amodio, Kubota, Harmon-Jones, and Devine (2006) revealed that people driven by external motivation may also succeed in suppressing their prejudiced behavior; however, they do so in a less efficient way compared to people driven by internal cues. Amodio et al. also found that because they *preconsciously* detect errors with the dACC (as measured with the ERN), people who are *internally* motivated are able to suppress their prejudice regardless of whether or not they are monitored. By contrast, externally motivated people suppress their bias only when they are worried about social disapproval and do so by *consciously* detecting erroneous responses, as indicated with enhanced rostral ACC activation when they fail (measured by error-related positivity [Pe], which follows upon the ERN). This suggests that health care providers who are internally motivated to behave in an unbiased way toward stigmatized patients or who are reminded of their morality are more likely to succeed in inhibiting automatic bias in their

interactions with stigmatized patients because they monitor their behavior with preconscious rather than conscious neural mechanisms and do not rely on external cues.

Once people have detected a situation in which they need to control their prejudice, in a second step they need to implement their goal to respond without prejudice. fMRI studies show that this goal implementation stage is driven by activation of the dlPFC, which is found particularly in task trials that require participants to override their stereotypical associations with race and gender (for an example, see Knutson, Mah, Manly, & Grafman, 2007).

Although the research described up to this point reveals that, given the right motivation, health care providers may be able to control their prejudiced responses, there is also research that suggests that this control process takes up regulatory resources (Richeson et al., 2003). This is important because reduced executive resources due to cognitively taxing intergroup interactions may limit the ability of health care providers to do their job as effectively as they would with nonstigmatized patients. Just as stigma-induced stress can lower the ability to apply self-control among members of stigmatized groups, interactions with out-group members can tax executive resources, especially among individuals who score relatively high on prejudice.

Interracial interactions can be threatening and uncomfortable due to concerns about being perceived as prejudiced (Richeson & Shelton, 2007). In an fMRI study, Richeson and colleagues (2003) revealed why this is the case. They found that White participants who scored higher on racial bias showed stronger activation in the ACC and dlPFC when viewing Black compared to White faces, suggesting that people with stronger racial bias need to more strongly recruit their cognitive control mechanisms in order to respond in an unbiased way. Importantly, however, participants who showed stronger activation of ACC and dlPFC in response to Black faces also showed reduced executive function afterwards. If we translate this to an interaction between a health care provider who meets a patient from another social group, this could mean that—to the degree that the health care provider is prejudiced—cognitive resources are depleted sooner when meeting a stigmatized versus a nonstigmatized patient so that less mental resources are left for effective job performance (e.g., interpreting health complaints and deciding on treatment).

Cardiovascular Processes Explaining Why Members of Stigmatized Groups May Receive Low-Quality Health Care

Research using unobtrusive, online, and continuous CV markers of challenge and threat (Blascovich, 2008) has indicated that intergroup interactions are typically threatening but has also provided clues about how to turn these threats into challenges (Blascovich, Mendes, & Seery, 2002). This is important to know for improving the quality of the interactions between health care providers (e.g., majority group) and patients (e.g.,

minority group) and has implications for effectiveness of medical treatments, as outlined next.

Threat During Intergroup Interactions

There is a rich literature on the physiological signs of threat in intergroup interactions. For example, Littleford, O'Dougherty Wright, and Sayoc-Parial (2005) found that for Whites, (p. 257) interactions with Black persons elicited stronger increases in blood pressure compared to interactions with White persons. Similarly, research in the tradition of the biopsychosocial model (Blascovich, 2008) has provided strong evidence that whereas neutral *intra*group interactions elicit a CV response pattern indicative of challenge (high CO, low TPR), neutral *inter*group interactions typically elicit a CV response pattern indicative of threat (high TPR, low CO). These CV signs of threat have been demonstrated for both majority and minority group interactants (Blascovich, Mendes, Hunter, Lickel, & Kowai-Bell, 2001; Page-Gould, Mendes, & Major, 2010; for meta-analytic evidence, see Blascovich et al., 2002).

Intergroup interactions are even threatening in cooperative settings (Blascovich et al., 2001), and they elicit threat in minority group members when they are treated positively by a majority group member (Mendes et al., 2008). This latter effect was explained in terms of the ambiguity that minority group members face when receiving positive feedback from majority group members; for example, they may wonder whether the feedback was motivated by genuine intentions or resulted from political correctness concerns. In combination, these findings are important in health care settings in which majority health care providers are expected to work fully in the interest of a minority patient, in that positive intentions may still result in a maladaptive CV profile in the minority patient.

Intergroup Threat Lowers Blood Flow

How does intergroup threat negatively impact health outcomes? At the behavioral level, negative health outcomes can be explained as stemming from intergroup misunderstandings resulting from threat. However, there is also a more direct physiological way in which the intergroup dynamics in health care settings can undermine health.

Blascovich (2013) described how threat during intergroup interactions between patient and health care provider can have a direct negative impact on the patient's health outcomes by decreasing blood flow. Blood flow affects the success of a range of medical treatments by affecting the delivery of medication and the removal of wastes. Optimizing blood flow during medical treatment can thus minimize the amount of medication that must be administered for effective treatment and also minimize the side effects of medication. Because intergroup health care interactions increase CV threat, they may also lead to suboptimal blood flow in stigmatized patients, thus leading to suboptimal health outcomes.

Moderators of Intergroup Threat

Several factors have been shown to attenuate threat during intergroup interactions. For example, previous experiences with intergroup contact moderated CV threat responses during intergroup interactions (Blascovich et al., 2001; Page-Gould et al., 2010): Majority group members who had more (positive) previous intergroup contact with minority group members responded with less threat during intergroup encounters. Similar results have been found for majority group members who are more appreciative of intergroup differences (i.e., those with a multicultural orientation; Scheepers, Saguy, Dovidio, & Gaertner, 2014). These findings suggest possible interventions to improve intergroup interactions in health care settings, such as internships in ethnically diverse neighborhoods or training to raise awareness of intergroup differences. These interventions should make interactions less threatening and, in turn, medical treatments more effective.

A further factor that may turn the threat of intergroup interactions into "challenge" is focusing on the morality of equal treatment. Although physicians' Hippocratic oath requires that all people should have equal rights to receive the medical treatment they need, this equality can be framed in different ways, namely as an "ought" or as an "ideal." Research by Does, Derks, Ellemers, and Scheepers (2012) indicates that when ethnic majority group members view intergroup equality as an ideal (vs. an ought), this elicits a cardiovascular response pattern indicative of challenge (vs. threat). Thus, a focus on morality may not only improve the self-regulation of prejudice among health care providers (Van Nunspeet et al., 2014) but also make their interactions with out-group patients less stressful and even positively engaging.

Summary of Research Findings: Pathway 3

Intergroup interactions, including those in health care settings, are often experienced as awkward and uncomfortable by the members of both parties involved. In this section, we reviewed neuroscience research on the processes that play a role during these interactions. Brain research has illustrated how social categorization occurs quickly and unconsciously and also how this sows the seeds for prejudiced responses. In addition, brain research suggests dampened levels of empathy in health care providers when treating patients from an out-group **(p. 258)** versus an in-group. Automatic and often unintended stereotypes and prejudice among health care providers in combination with a lack of empathy can negatively influence the health outcomes among the stigmatized. In addition, the stress of awkward interethnic interactions can have a further reinforcing negative influence on these outcomes, for example, because they hinder optimal blood flow in patients. However, the neuroscience research on stereotypes, prejudice, and interethnic interactions has also provided insights in what can be done to cut these pathways and to prevent poorer health outcomes among the stigmatized. Specifically, making health care providers focus on the moral aspects of their self-image, making them recognize and appreciate intergroup differences, and stimulating them to individuate their patients may be among the most fruitful strategies in this context.

What Do We Learn from Neural and Cardiovascular Approaches?

In this chapter, we have focused on three pathways by which stigma can reduce health outcomes and presented evidence for neural and cardiovascular mechanisms that may underlie these effects. One of the benefits of examining the neural and cardiovascular processes described here (and other physiological processes that have been described in other chapters of this volume) is that it allows us to get a more tangible grasp of the complicated and diverse mechanisms that link the psychological experience of stigma to reduced physical health outcomes. Whereas research on behavioral outcomes of stigma goes a long way in establishing the detrimental effects of negative stereotypes and threatening intergroup settings on performance, self-control, well-being, and ultimately health, psychophysiological research allows us to pinpoint the upstream (i.e., neural) and downstream (e.g., hormonal and cardiovascular) processes that lead to these responses.

For example, the neuroscience research reviewed here shows that stigma has its effects already very early in perception, transforming the way targets (e.g., patients) and agents of prejudice (e.g., doctors) preconsciously view and interpret their social world. In addition, because of their proximity to negative health outcomes such as cardiovascular disease, the cardiovascular responses reviewed here form a more direct link between neural mechanisms triggered by stigma and bad health, suggesting how experiences with stigma may wear out the cardiovascular system. Not only does this research help uncover some of the physical mechanisms that are responsible for the effects of stigma but also it illuminates that repeated exposure to stigma renders these mechanisms increasingly sensitive, leading to increasingly divergent life experiences of those with and those without a stigmatizing condition.

Particularly thought-provoking in this respect are results revealing differences in brain structure among people with *subjective* low (vs. high) social standing (Gianaros et al, 2008) and more extreme CV reactivity to race-related stressors among racial minorities who experience mistreatment due to race on a daily basis (Guyll et al., 2001). This work clearly suggests that being the target of prejudice goes beyond the occasional stressful experience, as it renders stigmatized individuals more sensitive to cues that signal social rejection and leaves them with less self-regulatory capacities to cope and behave in a way that benefits their health. Furthermore, as suggested by Blascovich (2013), stigma-induced stress during medical interactions may reduce the effectiveness of medical treatment because of impairments in blood flow among patients who respond to medical interactions and treatments with higher cardiovascular threat responses.

Examining neural and cardiovascular mechanisms related to stigma also allows us to determine in more detail the effect and effectiveness of possible interventions to improve outcomes for members of stigmatized groups. For example, based on behavioral research only, stimulating health care providers to regulate their prejudiced responses toward

stigmatized patients by increasing their *external* motivation to behave in an unprejudiced way may seem like a good idea. This can be done, for example, by telling them that their medical decisions or the treatment evaluations reported by patients will be analyzed with regard to group differences (e.g., racial, ethnic, gender, or SES groups). However, by uncovering the neural and cardiovascular mechanisms that are triggered by such interventions, we are able to see that this approach may be suboptimal.

First, the efficient neural conflict-detection system that people can draw upon to control their prejudiced responses is triggered by internal but not external motivation (Amodio et al., 2006). Whereas internally motivated doctors will monitor their responses preconsciously, regardless of outside cues, externally motivated doctors use an rACC-based neural circuit that is only triggered when monitoring one's behavior is rewarded. In addition, urging health care providers that they *should* control their bias triggers cardiovascular threat responses that (p. 259) may erode their motivation to work toward equal treatment, possibly resulting in even more awkward intergroup interactions (Does et al., 2012). Instead, triggering health care providers' *internal* motivation to behave without bias—for example, by speaking to their moral ideals to treat people equally (Does et al., 2012; Van Nunspeet et al., 2014)—is more likely to enhance activation of the neural conflict-detection system that preconsciously regulates biased responses, resulting in cardiovascular challenge responses that may improve the quality of the doctor–patient interaction. As such, uncovering the neural and cardiovascular mechanisms that are responsible for the effects of interventions aimed at improving the health outcomes of stigmatized individuals allows for a more thorough evaluation of their drawbacks and benefits.

The research reviewed here highlights the impact of experiencing pervasive stigma by revealing how pervasive experiences with discrimination actually change the way in which individuals experience their environment in terms of how their brain is wired, how they process their environment, and how their cardiovascular system subsequently responds. As a consequence, we should not count only on the perpetrators of prejudice to reduce their negative treatment in order to improve health outcomes among members of stigmatized groups. Research efforts should also be directed at finding ways by which members of stigmatized groups can change their brain's responses to social rejection. Possibly, the increasingly popular interventions based on mindfulness meditation may prove useful because they have been found to change the way people perceive and experience stigma and stereotype threat (Weger, Hooper, Meier, & Hopthrow, 2012). For example, it has been demonstrated that trait mindfulness—the ability to pay attention to present thoughts and emotions from a nonjudgmental and compassionate stance (Kabat-Zinn, 1994)—is related to increased sensitivity to *internal* stimuli and executive function (as measured with EEG) but reduced emotional responsiveness to *external* emotional stimuli (Teper & Inzlicht, 2013). Given that both executive control (e.g., not eating unhealthy food) and responsiveness to external cues (rejection and negative feedback) are affected by stigma, mindfulness could prove to be a useful strategy to change the

nervous system’s response to discrimination. Indeed, recent research has found evidence that trait mindfulness predicts reduced impact of experiencing racist events on anxious arousal among Black Americans (Graham, West, & Roemer, 2013).

Although we presented the three pathways linking stigma to poor health separately, it is important to note that we think of them as connected and mutually reinforcing. That is, the more stress stigmatized individuals experience due to their stigmatized status (Pathway 1), the more likely they are to suffer from reduced ability to control their behavior in the health domain (Pathway 2), increasing the chances that they will show behaviors that are detrimental to health, such as overeating, foregoing physical exercise, and reduced adherence to medical regimes. Moreover, although Pathways 2 and 3 may seem unrelated at first because Pathway 2 focuses on stigmatized patients and Pathway 3 concentrates on prejudice in health care providers, the detrimental health behaviors triggered in the second path are likely to reinforce the processes in the third path. That is, when health care providers have even slightly more negative expectations of the ability of patients with a stigmatizing condition to adhere to medical regimes, processes triggered in their patients in Pathway 2 are likely to reinforce these beliefs even more. Moreover, because health care providers are less likely to empathize with the hardships of members of stigmatized groups (Pathway 3), they are also less likely to be aware of the effects of stigmatization on self-regulation of health (Pathway 2). This may lead them to attribute failure to adhere to medical advice to internal rather than external causes, resulting in a lower likelihood that they will provide stigmatized patients with elective procedures and specialized health care. Finally, the more negative doctor–patient interactions are, and the more suboptimal the treatment that stigmatized patients receive (Pathway 3), the more likely it is that this will trigger stereotype threat and feelings of exclusion and rejection among stigmatized patients (Pathway 1). Stigma-induced stress elicited within the treatment setting may further enhance health disparities because it lowers patients’ cognitive ability to process treatment instructions, reduces treatment compliance, and impairs patients’ communication skills (e.g., reduced fluency and self-disclosure). In addition, threat induced by suboptimal patient–doctor interactions can even lead patients to discount threatening health feedback, skip medical appointments, or even fail to seek medical advice altogether (Burgess et al., 2010). As such, we speculate that the pathways we discussed here separately combine into a *perpetuum mobile* that results in a downward spiral in the health outcomes of people suffering from stigma.

(p. 260) Limitations and Directions for Future Research

In this chapter, we reviewed research to identify underlying neural and physiological mechanisms that link stigma and discrimination to suboptimal health outcomes. We believe that the overview provided in this chapter shows that the social neuroscience approach can provide valuable insight into the three pathways from stigma to health disparities and can inform debates about how these pathways can be blocked most effectively. However, here, we note a number of limitations in the research discussed and present possible future directions that would allow us to capitalize on the promise of social neuroscience methods in the study of stigma and health even more.

A first issue we encountered while compiling this review is that although we were able to find studies that are applicable to the three pathways explaining the stigma–health relationship, many of the studies reviewed here do not focus on stigma per se. For example, although the work on the neural circuits underlying the experience of social rejection is clearly relevant to the current discussion, of the research reviewed, only a handful of studies actually focused directly on discrimination and low social standing as an exclusory experience (Gianaros et al., 2007, 2008; Kleyn et al., 2009; Masten et al., 2011; Yanagisawa et al., 2013). A similar argument can be made for the studies reviewed about reduced self-regulatory ability following different coping strategies (e.g., emotion suppression vs. reappraisal; Inzlicht & Gutsell, 2007; Wang et al., 2014). This is important because the results from studies focusing directly on stigma suggest that single experiences with social rejection may be different from chronically experiencing social rejection due to a stigmatizing condition. It is therefore unclear to what degree we should draw conclusions about the negative health effects of stigma-induced stress based on research among nonstigmatized participants.

In the same way, for the third pathway, which focuses on the suboptimal health care that the stigmatized receive, none of the research reviewed was conducted in health care contexts. This means that although we know much about neural and cardiovascular processes that play a part in how members of the nonstigmatized majority may view, empathize, and interact with individuals with stigmatizing conditions, no research to date has specifically measured these neural and cardiovascular mechanisms in a health care setting. On the one hand, this may seem trivial because health care providers are people like everyone else and are therefore likely to fall prey to similar biases and intergroup processes as found in the studies reviewed here. On the other hand, one could also argue that interactions between stigmatized patients and health care providers are less likely to trigger some of the processes responsible for suboptimal health care that were described in this chapter. This is because several variables that have been found to moderate the neural and cardiovascular processes leading to negative outcomes in intergroup interactions could be applicable to interactions between a stigmatized patient and a nonstigmatized health care provider. Although the research reviewed for Pathway 3

provides a solid base for arguing which neural and cardiovascular processes may account for suboptimal health care received by stigmatized patients, future research should test the degree to which these mechanisms operate among health care providers and within doctor–patient interactions.

A final concern of ours is that although the work reported here focuses on a diverse set of stigmatizing conditions, there is an overrepresentation of work that examines prejudice and discrimination based on group-based stigma, particularly race. For example, most of the cross-sectional and epidemiological evidence for correlations between perceptions of discrimination or rejection, coping styles, and neural and cardiovascular outcomes that were reviewed came from studies that examined these in the context of either racial discrimination perceived by Black Americans or perceptions of low social standing among people growing up in low SES families. Although other stigmatizing conditions (e.g., gender, weight, and sexual preference) have been examined in experimental studies that test for differences in neural and cardiovascular reactivity, to our knowledge, cross-sectional and epidemiological research focusing on the stress induced by other stigmatizing conditions is currently lacking. This is unfortunate because cross-sectional research among other devalued groups (e.g., women and sexual minorities) that directly relates their psychological experiences to concrete health outcomes is the most powerful evidence that the *psychological* experience of being marginalized explains variance in the physical health outcomes of stigmatized individuals over and above *objective* discrepancies in resources afforded to members of stigmatized versus nonstigmatized groups. Future research should fill this lacuna, which could possibly lead to an even stronger case (p. 261) for the high impact of stigma and discrimination on the health of the stigmatized.

Conclusion

We believe that the social neuroscience perspective holds great promise for the study of the detrimental effects of stigma and discrimination on physical health. In this chapter, we presented an interconnected model that describes how stigma is processed in the brains of both the targets and the agents of prejudice and discrimination, how this results in suboptimal health outcomes via malignant cardiovascular responses, and how these processes are self-perpetuating. We think that the insights offered by the neuroscience perspective provide crucial information on how to interrupt the downward stigma–health spiral, and we hope that the research reviewed here can be used to inform policy and interventions to reduce the impact of stigma and discrimination on the physical health of its targets.

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