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Group identity, discrimination, and well-being: confluence of psychosocial and neurobiological factors

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In this paper we examine the variability in the associations between discrimination/stigma and vulnerability to poor health outcomes in light of psychosocial and neurobiological processes that might contribute to these relations. Depending on the features of the discrimination or stigma, different neurobiological stress reactions occur (i.e., cortisol reactivity vs. blunting). The effects of discrimination and stigma on well-being may be moderated by oxytocin, as this hormone influences processes related to the salience of the social category. Emerging areas that may further illuminate the links between discrimination and health outcomes involve the inflammatory immune system, as well as intergenerational transmission of severe or chronic stressors.

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Discrimination (acts against the individual due to a group membership) and stigma (negative social stereotypes associated with a group categorization) are powerful stressors that can contribute to a broad range of neurobiological alterations that lead to poor well-being [1]. The impact of discrimination on health has been repeatedly demonstrated in adults [2•], and also appears in relation to child and adolescent health [3]. Although neurobiological changes elicited by stressors often reflect adaptive responses to meet ongoing challenges, in the context of chronic stressors, these changes may have adverse effects [1,4]. Chronic stressful events occur at disproportionately high rates among disadvantaged or stigmatized groups, possibly leading to psychological disorders (e.g., depression, anxiety) and physical disturbances involving inflammatory-related processes (e.g., type 2 diabetes, heart disease).

Despite the considerable health impacts of stressors, not all groups who are targets of discrimination show the same profile of poorer outcomes and, under some conditions, enhanced well-being may be evident. Similarly, not all individuals within stigmatized groups are equally vulnerable or affected by the negative social attitudes and discrimination they encounter. We consider this variability in the relations between group experiences and vulnerability to poorer health outcomes and identify psychosocial and neurobiological processes that might mediate these relations.

Group identification

Social identity theory provides a framework that delineates the processes by which individuals identify with social groups, and how these memberships serve as a basis for positive self-worth and provide access to social resources that allow individuals to contend with life's challenges [5]. Social identities have the capacity not only to mobilize support networks, but also to shape appraisals of events, and to furnish individuals with coping strategies to manage adversity [5]. Individuals hold multiple social identities (e.g., religion, workplace, gender, diagnosed illness, ethnicity) that vary in importance across people and situations. Irrespective of the features that define the group, enhancing the strength of individuals' social identification, or the number of groups to which they feel a sense of belongingness, has been linked to positive mental [6,7] and physical [8] health outcomes. Indeed, developing and maintaining positive social identities may well serve as a 'social cure' by promoting individuals' psychological and biological capacity to deal with numerous illnesses [9].

Even as social identities form the basis for a social cure, there are conditions under which they may render group members more vulnerable to illness [10], and it seems that how individuals come to interpret identity is key in this regard [11]. Although identification with the group can be protective of well-being [12•,13], encountering discrimination can cause the identity to be less affirming [12•], and greater identification (particularly when the identity is salient and important) can sensitize individuals to the negative effects of discrimination [14•]. For example, among First Nations peoples in Canada, when identity pride was high, it buffered against the negative effects of perceived discrimination on depressive symptoms. However, greater identity centrality (importance) was associated with greater perceived discrimination, and exacerbated the relation between discrimination and

depression [15]. It would seem that when discrimination is encountered by group members who attribute positive value to the identity, it is less likely to elicit such negative impacts on well-being. By contrast, when identification entails the internalization of stigma, group members are more likely to anticipate continued devaluation and discrimination in other contexts and across time, rendering them more vulnerable to poor outcomes [11,16].

Neurobiological processes

The differential effects of discrimination on well-being can be elucidated through consideration of underlying neurobiological processes. The most well studied biological stress responses are the hormonal changes that accompany hypothalamic–pituitary–adrenal (HPA) functioning. When an event is appraised as a threat, a sequence of hormonal and neurotransmitter changes occur, culminating in the secretion of cortisol from the adrenal gland, which finds its way to the brain where it serves to turn off HPA activity, essentially acting as a negative feedback loop. Elevated cortisol levels can have many adaptive functions, such as stimulating the release of energy resources (e.g., contributing to the metabolism of carbohydrates and fats), and limiting excessive activity of other systems (e.g., immune activation) that might otherwise have detrimental effects [4]. Paradoxically, with chronic stressors or traumatic events that lead to posttraumatic stress disorder, cortisol functioning may be blunted relative to that of non-stressed individuals [17]. Although this down-regulation limits the damaging effects of persistent HPA functioning, the HPA system remains ready to be activated in a normal or exaggerated manner in response to subsequent meaningful stressors or reminders of previous stressful events [18,19•].

Consistent with the blunted cortisol variations following traumatic or chronic stressors, Blacks and Latinos displayed lower levels of cortisol, and flattening of normal diurnal cortisol variations, including a reduction of the typical early morning cortisol rise [20,21]. Importantly, when cross-race rejection (Whites rejecting Blacks or vice versa) was compared with same-race rejection, the former was accompanied by elevated cardiac output, increased emotional responses such as anger, cognitive changes comprising increased attentional bias and greater risk-taking behavior, but by diminished cortisol levels, possibly being related to chronic discriminatory experiences [22].

Despite multiple adaptive neurobiological responses, when stressor conditions are sufficiently severe, particularly if they are unpredictable, uncontrollable, and occur on a chronic intermittent basis, neurobiological systems may become overly taxed (allostatic overload), or secondary negative effects might evolve owing to excessive neuronal activation. Under these conditions, they become damaging or ineffective in dealing with further challenges, leading to adverse health outcomes [22]. The tipping threshold shifting from functional to dysfunctional processes has not been studied sufficiently, particularly from a longitudinal perspective. It has been suggested that the accumulation of experiences emanating from complex challenges (e.g., sustained social disturbances, early life abuse or neglect, social conflict, and poverty) may lead to what McEwen and Wingfield [23] referred to as 'type 2′ allostatic overload that requires system changes in social structures to attenuate the stressor environment, rather than individual treatment interventions. Indeed, the strain experienced as a result of the multiple challenges faced by some groups, or group members, lends itself to the evolution of health disturbances [20].

The effects of discrimination might also depend on the nature of the stigma associated with the group. Unlike the diminished cortisol apparent following exposure to racial/ethnic discrimination, experiences associated with being obese were accompanied by elevated cortisol levels [24,25], along with poorer well-being [16]. This raises the question as to whether the different outcomes are linked to stressor severity, or whether other features of the stigmatized identity are relevant. For example, some identities are construed as reflecting personal failings, and inclusion in the stigmatized social category is perceived to rest within individuals' behavior (i.e., is in their control to change or to have avoided in the first place) or are due to personal 'weaknesses' (e.g., obesity, HIV/AIDS status; depression/mental illness). Internalizing the stigma associated with these types of identities may result in efforts to conceal the identity, interpersonal distancing (particularly from other members of the same category), and a lower likelihood of seeking support or treatment [11]. Thus, unlike group memberships wherein individuals share a sense of common fate (i.e., 'being in this together'), these types of social identities are more isolating, and may involve neurobiological processes that undermine well-being.

Isolating social identities and oxytocin

Diverse neural circuits may be engaged in relation to different stressors [26], and social challenges such as discrimination and stigmatization may operate in unique ways. It is likely that the hormone oxytocin plays a moderating role in the relation between social stressors and well-being. The administration of oxytocin (intranasally) promotes enhanced empathy, generosity, trust, helping behavior [27•], and altruism biases that favor social causes [28]. Given these prosocial effects, the view was expressed that, among women, oxytocin is important in promoting tend-and-befriend characteristics [29•], whereas in males, oxytocin instigates a tend-and-defend response style [30]. Such patterns are evident at an interpersonal, as well as an intergroup level. For example, oxytocin increased lying to benefit one's ingroup [31], and defensive aggressive behaviors toward competing outgroup members [30]. Oxytocin might serve to enhance