Routledge
Taylor & Francis Group

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The Biological Underpinnings of Peer Victimization: Understanding Why and How the Effects of Bullying Can Last a Lifetime

Recent research in the areas of neuroscience, neuroendocrinology, and genetics is reviewed providing convincing evidence for why and how the effects of bullying can last a lifetime. Specifically, the research reviewed herein indicates that (a) the brain experiences peer victimization in a similar way to physical pain, (b) peer victimization is robustly linked to dysregulation of the neuroendocrine response to stress, (c) certain genetic profiles place bullied children at greater risk for poorer sequelae, and (d) the experiences of peer victimization become biologically embedded in the physiology of the developing person, placing him or her at risk for life-long mental and physical health problems. These studies highlight the urgent need to prioritize the reduction of bullying.

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WITH EACH NEW MEDIA REPORT of a bullying-related youth suicide comes the growing appreciation of the need to reduce, if not eliminate, school bullying. It is clear

from these media reports, and more importantly from research, that bullying causes harm. Indeed, bullying is consistently associated with poorer physical and mental health as well as poorer academic achievement, both concurrently (see Card, Isaacs, & Hodges, 2007, for a review) and longitudinally (see McDougall & Vaillancourt, 2013, for a review). Despite the growing body of evidence over the past 3 decades pointing to the harmful impact of bullying on victims, some still view peer bullying as a normal part of growing up and as something that can help kids to toughen up and build character. In this article, we review recent research in the areas of neuroscience, neuroendocrinology, and genetics that provides compelling evidence for why bullying hurts so much and why, for many, the hurt persists well beyond the actual abuse.

Neuroscience

The enduring pain associated with bullying is well illustrated by a recent (Weldon, 2012) news story that made headlines around the world. Robin Tomlin, a married father of two who is dying of a liver disease, sought to remedy a longstanding issue that has caused him "prolonged grief." Tomlin was severely bullied in high school. The level of vitriol directed at him was made public when school officials allowed his 1970 high school yearbook picture to be published with the authorized typewritten descriptor "Fag." In recounting his feelings about the incident, Tomlin told one reporter that "I feel like, emotionally, they [his bullies] have been beating me with a stick for 42 years" (Weldon, 2012).

Tomlin's description is consistent with research by Chen, Williams, Fitness, and Newton (2008), who found that "individuals can relive and re-experience social pain more easily and more intensely than physical pain" (p. 789). *Social pain* is the term used to describe the feelings of pain that follow the experiences of peer rejection, ostracism, or loss. Social pain can also be used to describe the feelings associated with being bullied (Vaillancourt, Hymel, & McDougall, 2010), a humiliating experience that is

not easily, if ever, forgotten. In describing the social and emotional pain that he continued to experience long after the bullying had ceased, Tomlin's metaphor, "beating me with a stick," is consistent with how social pain is described by people around the world (Eisenberger, 2012). Comparing social pain to physical pain, however, is more than a metaphor, as recent neuroscience research has shown that social pain is, in fact, experienced in a similar way to physical pain, at least as far as the brain is concerned. Indeed, recent studies have shown that social pain and physical pain rely on similar neurobiological and neural substrates (see Eisenberger, 2012; Panksepp, 1998; Vaillancourt et al., 2010, for reviews) and, as a result, are experienced physiologically in a similar manner.

Neuroendocrinology

Researchers are just beginning to understand the physiology that underlies peer victimization and how such experiences get under the skin. For example, they have long conceptualized peer victimization as a stressful experience. However, it is only recently that research has demonstrated the biological mechanisms that verify this assumption, although the picture is more complicated than originally thought. Specifically, a growing number of studies over the past decade have converged to demonstrate that peer victimization is linked to dysregulation of the hypothalamic-pituitary-adrenal axis (HPA axis), the body's stress response system (e.g., Kliewer, 2006; Knack, Jensen-Campbell, & Baum, 2011; Ouellet-Morin et al., 2011; Rudolph, Troop-Gordon, & Granger, 2011; Vaillancourt et al., 2011; Vaillancourt et al., 2008; see Knack & Vaillancourt, 2012, for review). Simply put, when exposed to stressful events, the body typically reacts with the quick secretion of cortisol, the end product of the HPA axis, which essentially prepares the body for a flight-or-fight response (see Lupien et al., 2005; Wolf, 2003, for review). This rapid release of cortisol is followed by a steady decline and this curve characterizes an adaptive response to stress.

With regard to peer victimization, several studies have now shown that bullied children tend to produce less cortisol than their nonbullied peers (e.g., Kliewer, 2006; Knack et al., 2011; Ouellet-Morin et al., 2011; Vaillancourt et al., 2008). This pattern of hyposecretion has been noted in relation to exposure to an acute social stressor (e.g., Knack et al., 2011; Ouellet-Morin et al., 2011), as well as in relation to daily average (diurnal) levels of cortisol (e.g., Hansen et al., 2006; Kliewer, 2006; Knack et al., 2011; Vaillancourt et al., 2008). Although this blunted pattern of cortisol production is opposite to what is typically observed in response to acute stress, it is consistent with studies examining cortisol levels in relation to extreme or prolonged stress. Indeed, this lowered production of cortisol in response to on-going stress has been well documented in research on child maltreatment (e.g., Bremner & Vermetten, 2001; Shea, Walsh, MacMillan, & Steiner, 2005). As well, among adults, holocaust victims (Yehuda et al., 1995) and repeated rape victims (Resnick, Yehuda, Pitman, & Foy, 1995) have also been shown to secrete lower levels of cortisol. What these survivors have in common is that their exposure to violence was severe, recurrent, and very likely associated with terror. Not surprising then, is the oft-replicated finding that individuals with posttraumatic stress disorder (PTSD) also tend to underproduce cortisol. For example, Morris, Compas, and Garber's (2012) meta-analytic review of 47 studies showed that the daily output of cortisol for individuals with PTSD was lower ($d = -.36$), compared to nontraumatized controls. PTSD can only be diagnosed if a person has experienced a traumatic event or events that "involve actual or threatened death or serious injury, or a threat to the physical integrity of oneself or others" (American Psychiatric Association, 2000, p. 463). Moreover, the individual's response to this threat must include "intense fear, helplessness, or horror" (p. 463). Using data from a nationally representative sample of Norwegian children, Idsoe, Dyregrov, and Idsoe (2012) reported that 28% of bullied boys and 40% of bullied girls in grades 8 and 9 scored in the clinical range for PTSD.

The findings on hypocortisolism in relation to extreme and prolonged stress are well-documented and extend to an atypical circadian cortisol pattern. For example, in a recent meta-analytical review, Miller, Chen, and Zhou (2007) showed that, whereas individuals typically show a pattern of higher morning levels of cortisol followed by lower afternoon levels of cortisol, chronic and severe stressors tend to be associated with the opposite pattern—lower morning levels of cortisol and higher afternoon levels of cortisol, a similar pattern that has been observed in response to physical threats to self. Thus, from a physiological perspective, peer victimization represents a relatively extreme and/or persistent stressor, which ultimately leads to uncharacteristic cortisol levels.

Moving one step closer to understanding the biological mechanisms underlying responses to peer victimization, it is important to consider recent research conducted by Ouellet-Morin and colleagues (2011). In this study, pairs of identical twins were assessed at 5 and 10 years of age, with one twin experiencing peer victimization at age 10 and the other not. Ouellet-Morin et al. found that peer victimization had a causal effect on how the body responded to stress, with victimized children displaying a blunted cortisol response to a psychosocial stress test, consistent with the research previously described. By examining discordant monozygotic twins, the effect could not be attributed to variations in either genetic makeup, family environment, or other concomitant factors, nor could they be attributed to the twins' perceptions of the degree of stress experienced during the task. Rather, the blunted cortisol reactivity was causally linked to the stress of peer victimization.

Genetics

Related research also shows that exposure to peer victimization influences DNA methylation. DNA methylation is an epigenetic mechanism that maintains gene activity or changes gene expression by activating or silencing the gene, resulting in the development of phenotypes that

are time-dependent and are not determined by the DNA sequence at that locus. Epigenetic alterations seem to function as a "biological mechanism for translating environmental signals into organismal molecular events" (Bick et al., 2012, p. 1418). Scientists are particularly interested in DNA methylation because it is implicated, among other things, in the emergence of neuropsychiatric disorders (see Tsankova, Renthal, Kumar, & Nestler, 2007, for review). In a recent study by Ouellett-Morin and colleagues (2012), increased DNA methylation of the serotonin transporter (*SERT*) gene between ages 5 and 10 was found for those twins who had been bullied but not for those who had not been bullied. Children with higher *SERT* DNA methylation also showed a blunted cortisol response to stress. Animal studies clearly support a pathway from early life stress to changes in DNA methylation (e.g., Weaver et al., 2004; see Meaney, 2010, for review), a finding that has also been demonstrated in children who have experienced early adversity (see Bick et al., 2012, for review), including child maltreatment (Beach, Brody, Todorov, Gunter, & Philibert, 2010; McGowan et al., 2009), and can now be extended to children who have been bullied by their peers.

Other evidence supporting the idea that victimization can alter biology comes from research by Shalev et al. (2012) examining another biomarker of stress—telomere length. Telomere refers to the repetitive nucleotide sequence (TTAGGG) at the end of chromosomes that promotes "chromosomal stability and also regulates the cells' cellular replicative lifespan" (Kiecolt-Glaser et al., 2011, p. 16). Telomere erosion has been linked to normal processes such as aging, but it has also been associated with health behaviors such as smoking and obesity, as well as diseases such as cancer, diabetes, and cardiovascular problems (Kiecolt-Glaser et al., 2011). That is, smoking, obesity, and disease, as well as aging, have all been associated with shorter telomere length. Importantly, shorter telomere length has also been linked to psychological stress (e.g., Epel et al., 2004) and mortality (e.g., Kimura et al., 2008; van der Harst et al., 2010; Willeit et al., 2010). In a recent longitudinal study of children who were tested at age 5 and 10,

Shalev et al. found that exposure to violence, including peer victimization, was associated with significant telomere erosion. That is, children who were exposed to violence showed greater reduction of telomere length at age 10 than did those who were not exposed to violence. This finding replicates other studies linking exposure to childhood adversity, including child abuse, to shortened telomere length (Kiecolt-Glaser et al., 2011; Tyrka et al., 2010).

Taken together, the emerging studies reviewed herein show quite clearly that peer victimization experiences are hurtful and can alter normal biological functioning. Although the physiological data show that victimization does get under the skin, it does not explain why the visible impact of victimization differs across bullied youth. Why is it that some victimized children show negative long-term effects, but others do not?

Gene × Environment Interactions

To date, research addressing heterogeneity in outcomes associated with peer victimization have focused on characteristics of the individual victim or his or her immediate social environment that serve as moderators and mediators of risk. For example, Rueger, Malecki, and Demaray's (2011) research indicates that girls are more affected by peer victimization in the long-run than boys. Sugimura and Rudolph (2012) found that child variables, such as temperament or gender, moderated the relationship between peer victimization and aggression and depressive symptoms. As one example, peer victimization predicted depression among girls who were high on negative emotionality. Bonanno and Hymel (2010) found that suicidal ideation was higher among victimized youths who perceived their situation as socially hopeless and those who perceived themselves to have less social support. Even though these studies are important in trying to identify factors that can be targeted and manipulated in helping victimized children to minimize the negative effects of peer harassment, they implicitly focus responsibility on the victim who becomes the agent of change. We contend that biological processes, many of which are less

directly under the control of the individual or his/her immediate social environment, may also be implicated and worthy of consideration.

Again, recent research on biological processes provides some initial insights into the mechanisms that may be operating. Particularly noteworthy here is research on the polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR). The 5-HTTLPR polymorphism affects the rate of gene transcription with the short allele being less effective than the long allele (Karg, Burmeister, Shedden, & Sen, 2011). An allele is an alternative form of the same gene. Caspi et al. (2003) reported that among severely maltreated children, those who had a short-short allele for 5-HTTLPR, were far more likely to be depressed in adulthood than those with a short-long or long-long allele. Moreover, those who had a long-long allele were at no greater risk for depression than those who were not maltreated in childhood. A recent meta-analysis (Karg et al., 2011) documents the accumulated evidence of the moderating role of the serotonin transporter gene in the relationship between stress and depression.

Extending this research to peer bullying, Benjet, Thompson, and Gotlib (2010) found that, among girls who endured relational victimization by peers, those with a short-short allele were far more likely to be depressed, as compared to those with a long-long allele. Similarly, in a longitudinal study of children from age 5 to age 12, Sugden et al. (2010) found that children with the short-short genotype for 5-HTTLPR were at far greater risk for developing emotional problems if frequently bullied, an effect that persisted even when controlling for previctimization emotional problems. These studies demonstrate that genes matter and interact with environmental triggers in determining the long-term impact of peer victimization. As in the case of 5-HTTLPR, biology can be a protective (long-long allele) or risk (short-short allele) factor.

Implications

Recognizing the biological implications and consequences of peer victimization serves to underscore the urgent need to eliminate bullying among children and youth. The emerging literature reviewed herein clearly indicates that the experience of peer victimization has immediate and lasting effects on biological functioning, even to the extent of creating ghosts in the genome (i.e., changes in methylation) that have important implications for future mental and physical health, as well life longevity (e.g., Kimura et al., 2008; van der Harst et al., 2010; Willeit et al., 2010). We offer two final examples in making our point; the first involving a well-documented link between peer victimization and poorer academic achievement (e.g., Nakamoto, & Schwartz, 2010 meta-analysis), and the second connecting peer victimization to poorer physical health (e.g., Gini & Pozzoli, 2009 meta-analysis).

Extending research on stress and human memory, which has demonstrated the deleterious effects of cortisol on brain structure and functioning, Vaillancourt et al. (2011) have shown a similar relationship with respect to peer victimization. Specifically, Vaillancourt and colleagues found direct predictive links from peer victimization and HPA dyregulation to poorer memory over the course of 4 months. However, they also found that peer victimization predicted depression (6 months later), which in turn predicted HPA dysregulation (6 months later), and this dysregulation of the HPA axis was associated with future memory impairment (4 months later) in the areas of the brain that have been shown to be especially sensitive to the effects of high circulating glucocorticoids (i.e., cortisol). In particular, deficits were noted in prefrontal executive functions and medial temporal lobe memory functions. Vaillancourt et al. suggested that perhaps the academic issues seen among bullied children are mediated by poorer memory functioning.

As the final example, Knack et al. (2011) found that peer victimization was associated with poorer health and a flattened cortisol awakening response (CAR). This altered atypical CAR was linked to health issues, suggesting that the relationship between peer victimization and poor physical health was mediated by differences in neuroendocrine functioning. This finding is interesting in that the early adversity literature suggests that the "epigenetic structure of genes

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underlying critical *stress response* and the *immune systems* are especially vulnerable to environmental signalling" (Bick et al., 2012, p. 1423; italics added).

In summation, it is clear from the extant literature that peer victimization is associated with significant concurrent and longitudinal problems in the area of physical and mental health, as well as in the academic domain. What has often been neglected in this area of research is a more comprehensive understanding of why these sequelae exist. Consistent with Bick et al.'s (2012) thesis and extended to peer victimization, we suggest that the experience of being bullied by peers becomes biologically embedded in the physiology of the developing person, which in turn modifies his or her health and, perhaps, learning trajectory. The evidence presented herein dispels any myth that the experience of peer victimization builds anything remotely positive or strong within individuals. On the contrary, the accumulating evidence clearly demonstrates that peer victimization erodes functioning at all levels, perhaps most important at the level of altering individual physiology.

It is not clear (as yet) if the biological scars brought on by peer victimization can be reversed—putting people back on to a healthier trajectory, although there is evidence suggesting hope. For example, two recent longitudinal studies have demonstrated positive neuroplastic changes to the amygdala (a brain structure that plays an important role in detecting stressful stimuli and initiating a response to cope) among individuals who participated in a mindfulness-based stress-reduction intervention (Hölzel et al., 2010; Hölzel et al., 2011). Within the area of DNA methylation changes induced by early adversity, there is also the suggestion that the effects may be reversed through behavioral or pharmacological interventions (Szyf & Bick, 2013; Vialou, Feng, Robison, & Nestler, 2013); granted, at this point in time, the mechanism remains nascent. The promise of this future work notwithstanding, we believe that a much simpler answer would be to fight the root cause directly, by having researchers and practitioners prioritize the reduction of bullying.

Acknowledgements

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