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Are some adolescents differentially susceptible to the influence of bullying on depression?

Priya A. Iyer, Angela Liegey Dougall, Lauri A. Jensen-Campbell *

University of Texas-Arlington, Department of Psychology, Arlington, Texas, United States

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This paper is dedicated to Dr. Andy Baum and Dr. Duane Burhmester. They provided significant contributions to both our lives and this project; you both may be gone too soon, but you will never be forgotten

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ABSTRACT

Not all children react to being bullied in the same way. Some children may be more predisposed to depression when bullied than are other children. Using a G × E approach, this study examined the influence of a genetic polymorphism in the serotonin transport gene (5-HTTLPR) on the victimization-depression link. The validity of the diathesis-stress versus environmental susceptibility hypothesis was tested. A total of 157 adolescents (M_{age} = 12.21) took part in this study. For adolescents with the S,S/S,L variants, victimization was positively related to depression. No relationship between victimization and depression was found for children with the L,L variant. Findings further suggest that the influence of 5HTTLPR on the association between victimization and depression more closely follow an environmental susceptibility model.

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1. Introduction

Although a certain degree of exposure to aggressive behaviors is likely to be normal for most children and adolescents, some children are the *repeated* target of abuse by their peers. This constant exposure to the negative aggressive actions (e.g., teasing, threats, rumors, physical harm) of one or more peers or classmates is often termed peer victimization or bullying (Andreou, 2001; Olweus, 2001). Estimates of the percentage of American children who report being victimized by their peers have ranged from 10% to 30% (Grills & Ollendick, 2002; Limber & Small, 2003; Nansel et al., 2001). This statistic is exceedingly important when one considers the potential for harm that peer victimization may cause. Being bullied by peers has been associated with a myriad of long-term psychological and physical problems (Arseneault, Bowes, & Shakoor, 2010; Cleary 2000; Knack, Jensen-Campbell, & Baum, 2011; Miller & Vaillancourt, 2007).

The current study examined whether being bullied predicted increases in depression and anxiety in early adolescence. Perhaps more importantly, this study focused on a potential genetic moderator to the victimization-depression association, namely the influence of the serotonin transport gene 5-HTTLPR polymorphism, which has been associated with physiological responsiveness to chronic stress (Moffitt, Caspi, & Rutter, 2005). We anticipated that young adolescent children with at least one S allele in 5-HTTLPR would be more susceptible to the negative influences of bullying on depression and anxiety than would adolescent children who were L,L homozygote (i.e., we anticipated a G × E interaction).

Although people can be bullied at any point in the lifespan, adolescence is a particularly important developmental period to study. As of 2009, adolescents were a part of the largest group in the world at 1.2 billion, accounting for approximately 18% of the world population (UNICEF, 2011). The sheer number of adolescents sheds light on the significance of studying this age group. Of greater consequence, early adolescence is associated with significant changes in biological, cognitive, and social functioning that are rivaled only by those changes seen in infancy (Larson & Richards, 1994). For example, early adolescent children undergo a number of major physiological changes including puberty, changes in the endocrine and immune systems, and rapid brain maturation (Golub et al., 2008; Walker, Walder, & Reynolds, 2001). Adolescents also undergo a number of changes in their social environment (Larson & Richards, 1994). Peers and friends begin to play an increasingly important socialization role (Hartup, 1996). As friendship intimacy increases during adolescence and supplements intimacy with parents, adolescents also begin to rely more on friends to satisfy needs and solve problems that arise (Furman & Buhrmester, 1985). Both longitudinal and cross-sectional studies have found that peer victimization also peaks during this time (Nansel et al., 2001; Nylund, Asparouhov, & Muthén, 2007).

* Corresponding author.
E-mail address: lcampbell@uta.edu (L.A. Jensen-Campbell).

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Additionally, the onset of mental illnesses, such as depression, anxiety disorders, and mood disorders, increases substantially during adolescence as a result of a greater vulnerability to stress that occurs as the adolescent goes through major physiological changes (e.g., Paus et al., 2008). Adolescent anxiety and depressive disorders show an approximate two to three fold increased risk for adulthood anxiety (Pine, Cohen, Gurley, Brook, & Ma, 1998). Depression is the second leading cause of disability adjusted life years (DALYs; i.e., productive years lost due to premature death or physical disability) starting at 15 years (Murray & Lopez, 1997; World Health Organization, 2011) and is often an early symptom of other physical health problems (e.g., Henningsen, Zimmermann, & Sattel, 2003; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999). Finally, depression is a major risk factor for suicide, which is the 3rd leading cause of child and adolescent mortality in the United States (Centers for Disease Control and Prevention (CDC), 2011).

Ironically, it is during this period, when adolescents are going through major physiological changes and are the most vulnerable to depression and anxiety symptoms that they are also most likely to become victims of peer abuse (Eisenberg & Aalsma, 2005). A meta-analysis conducted by Hawker and Boulton (2002) found that being bullied was most strongly related to depression, more so than any other component of psychological distress. Bullying was also associated with increased risk of suicidal ideation, suicide attempts, and suicide (Hay & Meldrum, 2010; Kaminski & Fang, 2009; Kim & Leventhal, 2008). For example, data collected by the Centers for Disease Control and Prevention as part of the Youth Risk Behavior Surveillance System revealed that there was a 73% increased likelihood that the adolescent would attempt suicide at least once if the adolescent was bullied compared to if the adolescent was not bullied (Centers for Disease Control and Prevention (CDC), 2009).

Perhaps more ominous is the fact that the association between victimization and depression seems to hold steady over time. Depression and anxiety is also much higher for those individuals that continue to be bullied over time compared to those who are only bullied for a more limited period (Menesini, Modena, & Tani, 2009). Finally, this association between bullying and depression appears to be bi-directional in nature. Victimized children are more likely to become depressed, but depressed children are also more likely to be victimized because they exhibit distress that is often rewarding for the bullies, creating a viscous cycle of bullying and depression for these children (e.g., Arseneault et al., 2010; Egan & Perry, 1998).

Not all adolescents succumb to depression and anxiety when bullied. One possibility for this variability in adjustment outcomes is that individual characteristics of the child interact with environmental influences to produce outcomes. That is, some children may be constitutionally more at risk for developing depression and anxiety problems when exposed to bullying from peers than are other children. The notion that the developmental outcomes of a particular individual involve the interaction between hereditary and environmental factors is widely accepted (Caspi & Moffit, 2006; Plomin, Owen, & McGuffin, 1994). Recently, behavior geneticists have begun to examine the interaction between specific genotypes and environmental factors in relation to outcomes (Moffitt et al., 2005). Indeed, one specific functional polymorphism, namely a functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene has been associated with depression and anxiety in adults and children who have experienced adverse environmental influences.

5-HTTLPR codes for the serotonin transport protein 5-HTT that is responsible for the reuptake of extracellular serotonin back into the presynaptic neuron (Bah et al., 2007). The gene that codes for 5-HTT (SLC6A4) is found on the 17q12 chromosome (Bah et al., 2007; Holmes & Hariri, 2003; Holmes, Yang, Murphy, & Crawley, 2002). Humans have a 44-base pair insertion/deletion polymorphism found in the promoter region of the 5-HTT gene which is further up from the coding region of SLC6A4 (the serotonin transport linked polymorphic region, 5-HTTLPR) (Heinz et al., 2000; Lesch et al., 1996). This gene can be comprised of a long variant (L) and a short variant (S) (Heinz et al., 2000; Lesch et al. 1996). Those individuals who carry the L variant have 16 repeats within the gene while those who carry the S- variant have 14 repeats within the gene (Bah et al., 2007). About 30% of the human population carries the two L variant genes, 50% one S and one L, and finally, 20% of the human population carries two S variants of the gene (Caspi et al., 2003).

The S variant is the less active allele; this short allele has been shown to decrease the transcriptional efficiency of the 5-HTT gene promotor (Bah et al., 2007; Lesch et al., 1996). Individuals who have either one or two copies of the short (S) allele are known to have fewer serotonin transporters than those persons with an L,L genotype (Heinz et al., 2000; Holmes & Hariri, 2003; Lesch et al., 1996). In addition, individuals with at least one S allele show greater amygdala activation to fearful stimuli (Hariri et al., 2002) and are at greater risk for developing depression, anxiety-related traits, and psychiatric disorders (Caspi et al., 2003; Hairiri et al., 2005; Lesch et al., 1996; Melke et al., 2001; Sen et al., 2004; Serretti, Kato, De Ronchi, & Kinjoshita, 2006).

Some of the first evidence for 5-HTTLPR influencing the onset of depression after stressful life events was reported by Caspi et al. (2003) as part of a longitudinal study. The polymorphism of the 5-HTTLPR gene moderated the link between stressful life events and depression. Specifically, those individuals who had one or two variants of the S-allele were found to exhibit more depressive symptoms, were more often diagnosed with depression, and had more suicidal thoughts as a result of stressful events than those individuals containing the two L alleles. At least fifteen independent studies have found a gene-environment interaction between the 5-HTTLPR polymorphism and environmental adversities that have been associated with depression (e.g., Caspi et al., 2003; Hairiri et al., 2005; Lesch et al., 1996; Serretti et al., 2006).

Several recent studies have begun to examine the influence of 5-HTTLPR on the bullying-depression association. For example, Benjet (2010) found that 5-HTTLPR moderated the influence of relational victimization on depression in a sample of 78 adolescent girls. The polymorphism of 5-HTTLPR alone did not significantly predict depression suggesting that some adolescents may be more susceptible to depression but only when bullied. Similarly, Sugden et al. (2010) found that victimized adolescents with the S,S genotype had more emotional problems than victimized adolescents with the L,L genotype. Again, they did not find a main effect for 5-HTTLPR.

One potential problem with most of these G × E interaction explanations is that they have been framed exclusively in terms of risk. In other words, researchers have suggested that that individuals who carry at least one S allele may be more vulnerable to expressing specific negative developmental outcomes (e.g. depression) only if they additionally face environmental adversity such as being bullied (Monroe & Simons, 1991). Interestingly, research has not only found that individuals with at least one S allele report having more maladaptive problems than the L,L homozygote if they experience adverse environments (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Kochanska, Kim, Barry, & Philbert, 2001), but it also appears that these individuals with at least one S allele often appear to do better in more optimal environments than people with the L,L genotype (For examples, see Benjet, 2010, Fig. 1; Caspi et al., 2003, Fig. 1). In other words, the G × E interaction appears to cross within the possible range of scores suggesting a dis-