



Does cortisol moderate the environmental association between peer victimization and depression symptoms? A genetically informed twin study

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ABSTRACT

Many youths who are victimized by peers suffer from depression symptoms. However, not all bullying victims show depression symptoms and individuals' biological sensitivity may play an important moderating role in this regard. In line with this notion, peer victimization has been associated with increased depressive symptoms in youth with higher basal cortisol secretion. It is unclear, however, whether this moderating effect of cortisol really concerns the *environmental* effect of peer victimization on depression. Indeed, genetic factors can also influence individuals' environmental experiences, including peer victimization, and part of these genetic factors may be those associated with depression. Using a genetically informed design based on 159 monozygotic and 120 dizygotic twin pairs (52% girls) assessed at age 14 years, this study examined whether cortisol secretion moderates the environmental or the genetic association between peer victimization and depression symptoms. Salivary cortisol at awakening was obtained with buccal swabs during four school week days. Peer victimization and depression were assessed via self-reports. Cholesky modeling revealed that peer victimization was associated with depression symptoms via both genetic and environmental pathways. Moreover, the *environmental* association between peer victimization and depression symptoms steadily increased with increasing levels of morning cortisol. The *genetic* association between peer victimization and depression symptoms also varied, albeit less, as a function of individuals' cortisol secretion. These findings support the hypothesis that peer victimization increases internalizing psychopathology mainly in youth with heightened biological reactivity to environmental conditions.

1. Introduction

Peer victimization, defined as the use of power and aggression to cause distress or control another person, is an important problem in many schools with severe risks for the victims (Craig et al., 2009; Oriol et al., 2017). Among the immediate sequelae are internalizing problems, notably depression symptoms (Reijntjes et al., 2010). Still, not all victims experience depression symptoms. Indeed, a meta-analysis showed an average longitudinal effect size of only 0.18 (range from 0.00 to 0.44) from peer victimization to internalizing symptoms (Reijntjes et al., 2010). This modest average effect and large variability might hide moderating factors, however, that enhance or buffer the

negative effect of peer victimization. One important moderating factor may be individuals' biological sensitivity to context. According to the Biological Sensitivity to Context (BSC) hypothesis (Ellis et al., 2011), some individuals are more sensitive to environmental influences—good or bad—than others. This biological sensitivity is believed to be partly rooted in genetically influenced physiological reactivity.

1.1. The moderating role of cortisol

Hormonal pathways, notably cortisol secretion, play a critical role in physiological reactivity to environmental experiences (Fries et al., 2005). Cortisol levels are typically higher at awakening and gradually

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decrease thereafter (Stone et al., 2001). There are, however, considerable inter-individual differences in diurnal cortisol secretion as well as in cortisol reactivity to acute stress, with potentially important mental health implications (Fries et al., 2005; Miller et al., 2007). Whereas lower cortisol levels are often linked with externalizing behaviors in adolescents (Alink et al., 2008), higher levels are typically associated with internalizing symptoms, including depression (Halligan et al., 2007). Moreover, elevated morning cortisol and cortisol reactivity to social stress predict increases in adolescents' depression symptoms over time (Goodyer et al., 2000; Susman et al., 1997). Individual differences in cortisol secretion may thus explain why only some victims show depression symptoms.

Several studies suggest that cortisol secretion moderates the association between stress exposure and mental health (Hagan et al., 2014; Kuhlman et al., 2017; Rudolph et al., 2011, 2010). Although these studies included a variety of stressors, cortisol measures and outcomes, findings generally indicate a stronger link between social stressors and internalizing problems in individuals with higher cortisol secretion. Cortisol as a moderator of the link between peer victimization and depression symptoms was tested in only one study (Rudolph et al., 2011). Results showed that peer victimization was associated with increased depressive symptoms in youths with high basal cortisol secretion as measured prior to a social stress task. It has been argued that pre-test cortisol levels may indicate an individual's baseline sensitivity to potential stress, both at the psychological and neurophysiological levels (Balodis et al., 2010). This finding thus seemingly supports the notion that a high biological sensitivity can exacerbate environmental influences on depressive symptoms. It is unclear, however, whether this moderating effect of cortisol really concerns the *environmental* association peer victimization and depression, as implicitly assumed by the BSC hypothesis. This question is important, as many social experiences partly arise as a consequence of individuals' genetically influenced characteristics (Jaffee and Price, 2007). Evidence from genetically informed research such as twin studies suggests that heritable factors explain over half of inter-individual differences in peer victimization (e.g., Brendgen et al., 2011). Moreover, genetic factors that influence peer rejection and victimization also influence depression symptoms (Bowes et al., 2013; Brendgen et al., 2009). Importantly, this *genetic* association between peer-related stress and depression symptoms reflects a gene-environment correlation (rGE), which is considered to indicate the effect of individuals' genetically driven characteristics (e.g., depressive behavior) on their environmental experiences (e.g., peer victimization), not the other way around (Lau and Eley, 2008). This rGE is in line with non-genetically informed studies showing that depression symptoms predict increased peer victimization (Reijntjes et al., 2010). A finding that differential cortisol secretion moderates the *genetic* association between peer victimization and depression symptoms would thus indicate moderation of the likelihood that individuals who are genetically vulnerable for depression become victims of bullying. However, such a finding would not support the BSC hypothesis, which postulates that physiological reactivity should moderate environmental effects on individuals. Only a moderating effect of cortisol on the *environmental* association between peer victimization and depression symptoms would be consistent with this hypothesis. Examining this issue may inform conceptual models and interventions to prevent depressive symptomatology in bullying victims.

2. Study objectives

We used a genetically informative design based on Monozygotic (MZ) and Dizygotic (DZ) twin pairs, whose peer victimization, salivary cortisol, and depression symptoms were assessed at age 14 years. This developmental period is of interest, as depression symptoms often increase during early adolescence (Dekker et al., 2007). Hypotheses were tested using cortisol measured in a naturalistic setting (i.e., at home). We focused on morning salivary cortisol, because (a) it may be

considered to represent a “pre-challenge” measure of the HPA axis activity during the day, (b) it is partly influenced by genetic factors (Ouellet-Morin et al., 2016) contributing to stability and (c) elevated morning cortisol has been linked to elevated depression symptoms (Halligan et al., 2007), and it thus may yield results similar to those found by Rudolph et al. (2011). We expected that the association between cortisol and depression symptoms would at least partly be explained by common genetic influences. Moreover, common genetic factors should at least partly explain the correlation between peer victimization and depression symptoms, indicating rGE. However, the association between peer victimization and depression symptoms should also be partly environmentally-driven and this environmentally-driven association should be moderated by cortisol secretion levels. These hypotheses were tested controlling for several potential confounders linked to peer victimization, depression symptoms or cortisol, i.e., family adversity, harsh parenting, birth weight, pubertal status, Body Mass Index (BMI), and individuals' aggressive behavior and biological sex (e.g., Cole et al., 2014; Jessop and Turner-Cobb, 2008; Kiess et al., 1995; Rudolph et al., 2010; Wüst et al., 2005). Morning cortisol was also corrected for potential effects of awakening and sampling times, sleep duration and quality, medications, menstruation for girls, and current and persistent health conditions.

3. Material and methods

3.1. Sample

Study participants were part of a population-based sample of 448 MZ and same-sex DZ twin pairs from the greater Montreal area who were recruited at birth between November 1995 and July 1998. Twins were first seen at 5 months of age and then prospectively assessed for a variety of child and family characteristics. Ninety-five percent of parents lived together, 44% of the twins were the firstborn children, 66% of mothers and 60% of fathers were between 25 and 34 years old, 17% of mothers and 14% of fathers had not finished high school, 28% of mothers and 27% of fathers held a university degree, 83% of the parents were employed, 10% of the families received social welfare or unemployment insurance, and 30% of families had an annual income of < \$30,000. Most families were of European descent (87%), 3% were of African descent, 3% were of Asian descent, and 1% were Native North Americans. Zygosity was assessed with 8–10 highly polymorphic genetic markers. Twins were diagnosed as Monozygotic when concordant for every genetic marker. When genetic material was insufficient, zygosity was determined based on physical resemblance at ages 18 months and 9 years (Spitz et al., 1996). The comparison of both methods in a subsample of 237 same-sex pairs revealed a 94% correspondence rate. The present study includes data collected in grade 8 (mean age = 14.07 years, SD = 0.30), when 279 twin pairs participated. Analyses for the present paper were performed on the 203 twin pairs (MZ males = 54, MZ females = 66, DZ males = 42, DZ females = 41) with valid data on cortisol, of whom 78% had collected saliva at awakening on each of the four collection days, 17% on three days, 4% on two days and 2% on one day. Participants with and those without valid cortisol data did not differ on any measure except with respect to aggression (see description of control variables below), with the former being less aggressive than the latter. The final study sample did not differ from those who dropped out or who were excluded due to invalid cortisol data regarding maternal or paternal education level, paternal age at the twins' birth or child temperament at age 5 months. However, families retained in the study had higher annual revenues and mothers were older at the twins' birth. They were also less likely to be single-parent households and more likely to be Caucasian.

3.2. Procedure

Letters explaining the objectives of the study were sent to the

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