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Original article

Depressive symptoms in adolescence: The role of perceived parental support, psychological control, and proactive control in interaction with 5-HTTLPR

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ABSTRACT

Background: Parenting dimensions are associated with depressive symptoms in adolescents. We investigated the role of perceived parenting dimensions and gene-environment interactions between these perceived parenting dimensions and five well-known variable number of tandem repeats (VNTRs): 5-HTTLPR, STin2, DAT1, DRD4, and MAO-A, in depressive symptoms.
Methods: From a non-clinical sample of 1111 Belgian adolescents (mean age: 13.79 years, SD = .94; 51% boys), 1103 adolescents consented for genetic research. Five VNTRs were analyzed using DNA from saliva samples. Perceived parenting dimensions (i.e., support, proactive control, psychological control, punishment, and harsh punishment) were examined using self-report scales completed by adolescents and their parents. Depressive symptoms were investigated using the CES-D self-report scale. Statistical analyses were performed in R using linear regression.
Results: Parental support, as perceived by the adolescent, was negatively associated with depressive symptoms (CES-D) and psychological control was positively associated with these symptoms. The only interaction effect withstanding correction for multiple testing was observed for 5-HTTLPR and the difference in proactive control as perceived by adolescents in comparison to parents. Short-allele carriers showed more depressive symptoms when there was a higher discrepancy in proactive control as perceived by adolescents versus parents.
Conclusions: Our results suggest that perceived parenting dimensions are associated with depressive symptoms, as measured by the CES-D. We only found modest evidence for 5-HTTLPR as a moderator in the association between the difference in perception of proactive control (adolescents vs. parents) and depressive symptoms.

1. Introduction

Adolescent depression is a problem worldwide with multiple implications in adulthood, for instance, an increased risk for depression and other psychopathology, more difficulties in education and poorer economic outcomes later in life [1–3]. Adolescents are involved in a complex and intense interplay with their environment, including relationships with parents [4]. Multiple family-related factors play a role in adolescent depression, such as parental depression as a risk and sustaining factor and parental hostility and rejection [5–8]. Multiple chronic and episodic stressors, such as physical or psychological abuse and family conflicts also influence depressive symptoms [9–11]. Parental emotional support, positive parental affect and an approving and validating attitude represent protective factors [12–14]. Additionally, the adolescents' genetic background and the interactions

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between genes and environment can influence their risk for depressive symptoms: one adolescent may be more prone to depressive symptoms than the other [15–18].

The present study contributes to research on gene-environment interactions (G × E) in parenting and adolescent depression by including positive and negative parenting factors simultaneously. Researchers typically consider three parenting dimensions: support, psychological control, and behavioral control [19]. We use a comprehensive five-dimensional model of parenting, by distinguishing three aspects of behavioral control: proactive control, punishment, and harsh punishment [20]. We focus on the dopamine and serotonin systems and selected five variable number tandem repeats (VNTRs) known to be involved in adolescent depression [21–26]. Due to the current concerns in G × E research regarding small sample sizes, lack of power, and publication biases, as reviewed by Duncan and Keller [27], we chose to analyze all five VNTRs and to report all the results in the Supplementary data.

5-HTTLPR and STin2 are two VNTRs located in the serotonin transporter gene (SLC6A4 at 17q12, in the promoter region and the second intron, respectively [28,29]) that possibly both affect gene expression [30] and are assumed to play an important role in the development of depression and depressive symptoms, as well as social anxiety [31–33]. The most common variants of the 5-HTTLPR are the 14R ("short" allele) and 16R ("long" allele) [28]. Results remain inconclusive, but the 14R is believed to play a role in environmental sensitivity [25,26,34–36]. The most frequent alleles described for STin2 are 9R, 10R and 12R [37]. It is unclear whether the 9R or 12R variant is associated with susceptibility for depressive symptoms [37,38].

The dopamine transporter gene *DAT1* (SLC6A3, at 5p15.3 [39]) and the dopamine receptor gene *DRD4* (11p15 [40]) contribute to the dopamine system. The number of DAT1 repeats ranges from 3 to 13, and 9R and 10R occur most often [39]. The 9R allele is associated with increased dopamine binding and signaling [41]. In DRD4, the variants with two, four or seven repeats are most common [42]. The 2R variant is associated with depression [43], the 7R allele with externalizing problems [44].

MAO-A is an X-linked gene associated with degradation of monoamines (e.g., serotonin and dopamine) [45]. The 3-repeat and 4-repeat variants are most prevalent. The 3R allele ("low activity") is associated with psychopathology and is also involved in G × E interactions [46].

Most G × E research on adolescents examined extreme environments such as childhood trauma and maltreatment, sexual abuse or threatening life events [25,47,48]. More common factors such as conflicts in the family and peer victimization as well as positive parenting have been investigated in interaction with multiple polymorphisms related to serotonin, dopamine, and oxytocin [21,22,49–54]. We look at perceived parenting as represented by five dimensions [20]: perceived support, proactive control (i.e., consistent rule setting), psychological control (i.e., intrusive type of control, parents manipulate thoughts, emotions and feelings of the adolescent), punishment, and harsh punishment (i.e., physical punishment), in interaction with the above mentioned VNTRs. We focus primarily on parenting dimensions as perceived by adolescents. However, previous research shows the importance of differences across informants in the perception of parenting [55,56]. Hence, for each parenting dimension, we compute the differences between the perception of adolescents and parents as a measure of mismatch in perception of parenting and explore their association as an environmental variable with depressive symptoms in G × E. Because genes can influence susceptibility to the environment [21], they may also influence some of adolescents' emotional response to discordances in the perception of parenting.

To our knowledge, there is only limited research that simultaneously addresses positive and negative perceived parenting dimensions from both the adolescent and parent perspectives. Additionally, we are not aware of studies using difference in perception scores between parents and adolescents in a G × E context. Given the contradictory findings in the literature and the lack of replication, no specific genotypes or alleles were designated as risk alleles prior to analysis. This approach again is different from most previous research in the field of parenting and adolescent depression.

Our aim is to investigate how the five perceived parenting dimensions [20] are associated with adolescent depressive symptoms. First, we focus on the main effect of perception of the adolescent as an environmental factor and hypothesize perceived support and perceived psychological control to be associated most strongly with adolescent depression. Additionally, we hypothesize that polymorphisms of the serotonin and dopamine systems may interact with the parenting dimensions as perceived by the adolescent. We also extended current research using the differences in perception of parenting between adolescents and parents in interaction with the five selected polymorphisms.

2. Method

2.1. Sample and measures

We use data from the Strategies project (acronym for Studying Transactions in Adolescence: Testing Genes in Interaction with Environments). A sample of 1111 adolescents (mean age: 13.79 years, SD = .94, 51% boys) was recruited from nine schools situated in Flanders, Belgium. From this sample, a total of 1103 adolescents (99%) agreed to participate in the genetic part of the study. If two siblings from the same family participated, one sibling per family was selected randomly, which led to a final sample of n = 1045. Ancestry was assessed using the grandparents' country of birth (88.8% European, 6.5% non-European Mediterranean, 2.2% non-European, non-Mediterranean ancestry, and 2.5% missing).

G*Power 3 and ML-DEs [57,58], two statistical programs to conduct power analysis, had shown that a sample size of 800 has more than 80% power for detecting small environmental effects, genetic effects, and G × E interactions, each accounting for only 1% of the variance with α = .05. Because we had no prior information available on the 5-factor model for parenting [20], we could only estimate power using the proportion of explained variance. Based on these estimates, the present study was sufficiently powered. The study protocol was approved by the biomedical Internal Review Board of the researchers' university. Parents and adolescents gave active written consent for the use of psychological data and DNA sampling, separately.

All adolescents and their parents (response rate: 72%; n = 797) were asked to complete self-report questionnaires concerning parenting dimensions. Only adolescents reported on depressive symptoms.

Established self-report questionnaires with strong psychometric characteristics were used. For depressive symptoms, we used the Center for Epidemiologic Studies Depression Scale (CES-D Scale; [59]), which assesses depressive symptoms over the last week. Although a score of 16 or higher can be considered a "possible case" of clinical depression, we preferred to interpret the scale continuously. We did not dichotomize our sample using this cut-off to avoid drawing preliminary clinical conclusions from this non-clinical sample. Perceived parenting dimensions were assessed using the four subscales of the Leuven Adolescent Perceived Parenting Scale (LAPPS; [60]), the four subscales of the shortened