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EMPIRICAL RESEARCH

Inhibitory Control Mediates the Associations Between Parenting Practices and Depressive Symptoms in Adolescents: The Moderating Role of Catechol-O-Methyltransferase (*COMT*) Gene

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Abstract
Ample evidence suggested that parental responsiveness, demandingness, and autonomy granting protect adolescents from depressive symptoms. However, what is less well understood is how parenting practices reduce the risk of depressive symptoms. This study tested the protective effects of parenting practices and inhibitory control on depressive symptoms, along with the mediating role of inhibitory control and the moderating role of the *COMT* gene in linking parenting practices to depressive symptoms. The study utilized cross-sectional data from a community sample of Chinese Han adolescents ($N = 943$, $M_{age} = 15.25$ years, $SD = 0.70$ years; 51.9% girls). Results showed that parental responsiveness and autonomy granting promoted higher inhibitory control, which in turn was associated with lower depressive symptoms. Further, the mediation effects were moderated by the *COMT* gene. For adolescents with ValVal homozygotes, both responsiveness and autonomy granting were related to higher levels of inhibitory control, which reduced risk for depressive symptoms, but the mediation effects were not observed among Met allele carriers. The mediating role of inhibitory control did not hold in the parental demandingness model. Findings support the cognitive theory that inhibitory control is a proximal factor linking parenting practices to depressive symptoms exclusively in ValVal homozygotes. These results also suggested that differentiating different dimensions of parenting practices may help to further clarify the processes by which parenting practices eventuate depressive symptoms.

Keywords Parenting practices; · Inhibitory control; · *COMT* gene; · Depressive symptoms

Introduction

Depression in adolescence is a major health problem, both for its potential societal burden and its risk for severe individual disability. As such, much work has been devoted to identifying the factors that prevent and attenuate depressive symptoms, with research consistently documenting the important role of parenting practices (e.g., McLeod et al., 2007). Recent research attention is appropriately shifting from identifying protective factors towards examining how positive parenting practices reduces the risk of their children's depressive symptoms. This study aimed to explore the underlying mediating process that links the effects of parenting practices to adolescent depressive symptoms. Besides, not all children respond to their parent's behavior in the same way, with some children benefit more than others from good quality rearing. The differences between children in response to parenting practices may depend on their genetic makeup. Given the role of dopamine in reward sensitivity, genes regulating dopaminergic function, such as the catechol-O-methyltransferase (*COMT*) gene, have been reported repeatedly as modifiers of reaction to environments (e.g., Blair et al., 2015; Sulik et al., 2015b; Zhang et al., 2018). Therefore, this study also investigated the role of the *COMT* gene in adolescents' sensitivity to parenting practices.

Parenting Practices and Adolescent Depressive Symptoms

Three core dimensions of the parents' behavior—responsiveness, demandingness, and autonomy granting—are often used to characterize parenting practices. *Responsiveness* (warmth)

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refers to the extent to which the parent responds to the child's needs in an accepting, supportive manner (Steinberg, 2020). *Demandingness* (control) can be defined as the degree to which the parent provides clear and consistent guidelines and maintains knowledge about children by maturity demands, supervision, and disciplinary efforts usually range from supervision and monitoring to power assertion and harsh discipline (Steinberg, 2020). These two broad parenting dimensions were from Baumrind's (1991) typology which has been widely adopted. Furthermore, Steinberg et al. (1992) expanded Baumrind's framework by adding autonomy granting as a distinct dimension reflecting some crucial parenting characteristics during adolescence. *Autonomy granting* reflects the extent to which the parent employs democratic discipline and encourages adolescents to express individuality within the family (Pinquart, 2017; Steinberg et al., 1992).

Parental responsiveness, demandingness, and autonomy granting play important roles in the development of depressive symptoms (McLeod et al., 2007; Pinquart, 2017; Yap et al., 2014). Responsive parenting practices has protective effects on the development of depressive symptoms (e.g., McLeod et al., 2007; Pinquart, 2017) because such parenting practices could help adolescents to develop a positive self-perception and prevent negative feelings (Wouters et al., 2018). Notably, different forms of parental demandingness may have differing effects on child depressive symptoms. Positive parental demandingness involving supervision and monitoring is associated with decreased levels of depressive symptoms (Ruybal & Crano, 2020; Yap et al., 2014). This is because by engaging in age-appropriate limit-setting and supervision, parents may be knowledgeable about their adolescents' whereabouts and then could offer support and help with reducing negative feelings (Fröjd et al., 2007). It is possible that parental monitoring and limit-setting may be perceived by children as strong indicators that their parents care for them and want to ensure their safety and wellbeing, which increases parent-child attachment security (Koehn & Kerns, 2018) and in turn decreases depression (Spruit et al., 2020). In contrast, negative demandingness, such as harsh discipline and over-controlling, is often identified with depressive symptoms (Lo et al., 2021). The present study chose to examine positive demandingness to identify protective factors of depressive symptoms. Autonomy granting would facilitate children's positive emotional functioning (Matte-Gagne et al., 2015) and help them develop stronger resilience in the face of stressful events (Soenens et al., 2017). As such, parents give more autonomy granting to their children, lowering the risk for developing depressive symptoms (Pinquart, 2017).

Moreover, the three aspects of parenting practices are relatively independent of each other and are differentially associated with adolescent adjustment. For example, a cross-cultural study revealed that autonomy granting and responsiveness were positively associated with adolescents' well-being, but the relation was not found for demandingness (Filus et al., 2019; Garthe et al., 2015). Besides, a meta-analysis found that the affective aspect was more strongly related to depressive symptoms than was parental control aspects (McLeod et al., 2007). To the extent that parental responsiveness, demandingness, and autonomy granting represent the affective, behavioral control, and psychological control aspects of parenting practices respectively, the present study examined such parenting practices separately.

Inhibitory Control as A Mediator

Protective parenting practices may reduce the risk of depressive symptoms in multiple pathways. In addition to examining the direct effect of protective parenting practices on depressive symptoms, there is an increasing interest to identify the more proximal mediators that might explain how parenting practices reduce the risk of depressive symptoms. Cognitive theories of psychopathology suggest that one potential proximal mediator linking distal environmental context with depressive symptoms is cognitive factors (Nolen-Hoeksema & Watkins, 2011). Inhibitory control, which reflects the ability to control dominant responses to override a strong internal predisposition or external stimulus (Diamond, 2013), is a particularly cognitive factor. According to the gateway mechanism of depressive symptoms, inhibitory control impaired by stress provides a loose gateway for depressive thought (De Raedt & Koster, 2010). In contrast, responsive, demanding, and autonomy-granting parenting practices promote the development of adolescent's inhibitory control (e.g., Lengua et al., 2007; Roskam et al., 2014), which may protect adolescents from negative thoughts and depressive symptoms.

Some initial evidence has provided support for the hypothesis that inhibitory control mediates the relation of parenting practices to depressive symptoms. On the one hand, positive parenting practices were associated with increased inhibitory control. Children with responsive and autonomy-encouraging parents may be more motivated to internalize parental directions, a process necessary for conceptualizing alternative nondominant responses (Grusec & Goodnow, 1994; Kochanska & Kim, 2014; Matte-Gagne et al., 2015). Parental demanding strategies involving monitoring and limit-setting provided a behavioral control and regulatory template, which may teach offspring to suppress a dominant response and to initiate a subdominant response (LeCuyer & Houck, 2006; Lengua et al., 2007). Furthermore, positive parenting practices involving responsiveness and demandingness may help children to achieve higher levels of inhibitory control by yielding a

general positive interactive context in which the child feels safe and comfortable, such a context fosters effortful control and promotes internalization of rules (Heikamp et al., 2013; Nordling et al., 2016). Similarly, evidence from neurobiological research suggested that positive parenting practices facilitate inhibitory control related brain regions. Adolescents experiencing more responsive parenting practices have been found in several studies to demonstrate reduced gray matter in the anterior cingulate cortex (ACC) (Rao et al., 2010; Whittle et al., 2014). The greater thinning in the ACC was associated with higher temperamental effortful control—the ability to inhibit a dominant response (Vijayakumar et al., 2014). On the other hand, individuals with high inhibitory control showed decreased depressive symptoms because inhibitory control promotes the regulation of emotional reactions to stress and attentional shifting to positive stimuli (Lengua, 2003).

Also consistent with the mediation hypothesis, recent longitudinal studies have frequently highlighted the mediating role of inhibitory control for understanding the relationship between parenting practices and psychopathology in general (e.g., Lengua et al., 2015; Lengua et al., 2020; Hentges et al., 2020) and suicide in particular (Connell et al., 2019). For example, the indirect effects of the family-focused intervention on adolescent psychopathological symptoms were observed, with treatment-related improvements in inhibitory control predicting reductions in internalizing and externalizing symptoms (Hentges et al., 2020). Therefore, it is reasonable to expect that higher levels of inhibitory control fostered by positive parenting practices may improve individuals' ability to disengage from negative thoughts and in turn reduces the risk of depressive symptoms.

Notably, there appears to be a bidirectional relationship between parenting practices and inhibitory control. Research has indicated that in addition to parenting practices predicting executive function or effortful control, the executive function or effortful control also predicted parenting practices in young children (e.g., Blair et al., 2014; Eisenberg et al., 2015). However, the strength and direction of associations between parenting practices and inhibitory control may depend on the developmental periods. Recent longitudinal studies have indicated that the bidirectional association was more robust in early ages, and diminished in older age (Sulik et al., 2015a; Tiberio et al., 2016). In particular, the null result of the child effect on parenting practices has been reported during early adolescence (Tiberio et al., 2016). The child effects on parenting practices may have already been established early in life, and therefore do not have additional effects later on parenting practices. Accordingly, due to the cross-sectional data obtained in the present study, this study preferred to test the mediating role of inhibitory in the association between parenting practices and depressive symptoms, rather than the reverse.

COMT Gene as A Potential Moderator

Although all children are impacted by parenting practices, some children benefit more than others from good quality rearing. Numerous genetic polymorphisms have been suggested as influencing sensitivity to the environment (e.g., Green et al., 2017; Van Assche et al., 2016; Zhang et al., 2016). Given the late-maturing prefrontal cortex (PFC) makes adolescents particularly sensitive to environmental influence (Andersen & Teicher, 2008), this study focused on the *COMT* gene which expressed abundantly in PFC. The *COMT* gene encodes the COMT enzyme, which plays a critical role in the metabolism of dopamine, norepinephrine, and epinephrine in the PFC (Gogos et al., 1998). This gene contains a common functional polymorphism ($Val^{158}Met$ or rs4680) characterized by valine to methionine substitution at codon 158 that results in a 40% reduction in COMT enzyme activity (Lachman et al., 1996).

The *COMT* $Val^{158}Met$ polymorphism has been found to moderate the associations between environments and a broad range of emotional and cognitive outcomes (e.g., Kurowski et al., 2017; Sulik et al., 2015b). However, mixed findings were obtained regarding both depressive symptoms and inhibitory control. Specifically, some studies have described *COMT* ValVal homozygotes as a "risk" genotype that confers greater vulnerability to depressive symptoms in the context of adversity (e.g., Drury et al., 2010), whereas other studies found individuals with Met allele (MetMet and ValMet) to be more at risk as a result of adversity (e.g., Åberg et al., 2011; Mandelli et al., 2007). With regard to inhibitory control, the majority of study has linked *COMT* ValVal homozygotes rather than Met allele with greater sensitivity to the environment, but the direction of the association between adversity and inhibitory control in ValVal homozygotes were mixed (Blair et al., 2015; Park et al., 2017; Zhang et al., 2018). Some studies showed that children carrying ValVal homozygotes exhibited faster growth of executive function in the presence of early adversity relative to children with Met allele (Blair et al., 2015), whereas others found that ValVal homozygotes linked with poor inhibitory control for children experiencing adversity (Park et al., 2017; Zhang et al., 2018). According to the late-maturing PFC theory, fMRI studies also suggested that the ValVal homozygotes may render adolescents particularly sensitive to environments by decreasing the connectivity between the PFC and the amygdala that involved in processing emotion (Drabant et al., 2006; Klucken et al., 2015).

Notably, above mentioned literature was adopted the recessive genetic model for Val, but a few studies adopted three distinct genotype groups (Quan et al., 2017) or the dominant model which compared MetMet homozygotes with Val allele carriers also obtained mixed results (Hosang et al., 2017; Kurowski et al., 2017; Nyman et al., 2011; Sulik et al., 2015b). Sulik et al. (2015b) showed that parenting practices were positively related to inhibitory control and internalizing problems for MetMet boys, whereas Hosang et al. (2017) and Kurowski et al. (2017) found Val allele conferring greater sensitivity to the environment, and a recent study regarding executive function obtained null results (Quan et al., 2017). Nevertheless, these mixed findings do not necessarily deny a moderating role of *COMT* gene; rather, they suggest that further study is warranted.

One possible reason behind the inconsistencies is that the effect of the *COMT* gene depends on basal dopamine level. More specifically, evidence has suggested an inverted U-shaped relationship between dopamine activity and PFC (a critical region relate to inhibitory control) function, with both dopaminergic hypofunction and hyperfunction, were related to poor PFC function (Goldman-Rakic et al., 2000). As such, decreased dopamine availability in ValVal homozygotes is associated with greater PFC function when basal dopamine levels are high, but with reduced PFC function when basal levels are low. In brief, the *COMT* gene determines where on the inverted U-shaped curve of PFC dopamine function an individual lies (Tunbridge et al., 2006), which in turn may be associated with the PFC-amygdala connectivity and depressive symptoms. There are marked developmental changes in the basal dopamine levels during puberty (Benes et al., 2000), which may result in a distinctive pattern of gene-environment interaction (G × E) during adolescence. However, previous studies were largely been limited to children or adults (e.g., Drury et al., 2010; Blair et al., 2015; Sulik et al., 2015b), little is known about the interaction between parenting practices and the *COMT* gene in adolescents. To address this gap, this study aimed to test the moderating role of the *COMT* gene in the associations among parenting practices, inhibitory control, and depressive symptoms.

The difference in the way of coding a genetic polymorphism may also result in mixed results, and different interpretations of the nature of gene-environment interactions (Aliev et al., 2014). As outline earlier, there is a lack of a clearly defined grouping method for heterozygous (i.e., ValMet) individuals (e.g., Blair et al., 2015; Sulik et al., 2015b). Most of studies have adopted the recessive model for Val by grouping ValMet individuals with MetMet individuals, primarily due to sample size limitations, but potentially obfuscating the complexity of the mode of genetic inheritance (Dick et al., 2015). Therefore, to avoid inappropriate genetic coding, this study first adopted the two dummy variables (i.e., ValVal vs. ValMet and MetMet vs. ValMet) to interpret the nature of gene by environment interaction as recommended (Aliev et al., 2014).

Notably, the inconsistencies and small effect size also raise the doubt on the robustness of the candidate gene approach (Dick et al., 2015; Duncan & Keller, 2011). In particular, a recent meta-analysis involving externalizing, internalizing, and cognitive outcomes, failed to find significant *COMT* by environment interactions (Cao et al., 2019). In this regard, researchers have suggested that candidate genetic studies should consider adopting more stringent thresholds for statistical significance and require replication (Roisman et al., 2012). As such, this study utilized the corrected *p*-value and the split-half validation to test the robustness of results, which may provide a strong test of replication due to the lack of difference in methods (Johnston et al., 2013).

Hypotheses

The present study proposed a moderated mediation model to extend previous studies by integrating environmental context, cognition, and genetic factors in understanding the etiology of depressive symptoms. This study aimed to explore how responsive, demanding, and autonomy granting parenting practices operate to influence depressive symptoms and why some individuals benefit more than others from good quality rearing utilizing cross-sectional data from a community sample of Chinese Han adolescents. Drawing from the cognitive theory of psychopathology, the present study expected that such parenting practices would be associated with better inhibitory control, which in turn, would be associated with decreased depressive symptoms. Moreover, this study expected that the *COMT* gene would moderate the strength of inhibitory control as a mediator linking parenting practices and depressive symptoms. As there are mixed findings regarding which genotype confers sensitivity to the environment, no specific hypotheses were postulated.

Methods

Participants

One thousand and eighty parent-child dyads, of predominantly Chinese Han ethnicity (96.9%), were recruited from 7 junior middle schools in the urban areas. Among the adolescent participants, 541 were girls (50.1%) and age ranged from 13 to 17 years ($M = 15.24$, $SD = 0.61$). Of the initial sample, 34 (3.1%) were excluded because of being Chinese minority ethnicity, 47 (4.4%) adolescents were had

Table 1 Characteristics of the study sample

Characteristics	Initial Sample ($N = 1080$)	Final Sample ($N = 943$)	Differences[a]
Adolescent ethnicity, %			
Chinese Han	96.9	100	—[b]
Chinese Minority	3.1	0	
Adolescent age, *M* (*SD*)	15.24 (0.61)	15.24 (0.61)	$t = 0.11$, $p = 0.91$
Adolescent gender, %			
Boys	49.9	48.1	$\chi^2 = 9.24$, $p = 0.003$
Girls	50.1	51.9	
COMT			
ValVal	56.6	56.5	$\chi^2 = 1.02$, $p = 0.60$
ValMet	36.3	36.1	
MetMet	7.1	7.4	
Responsiveness, *M* (*SD*)	−0.04 (0.67)	−0.03 (0.67)	$t = 1.88$, $p = 0.06$
Demandingness, *M* (*SD*)	−0.01 (0.61)	−0.01 (0.61)	$t = 1.42$, $p = 0.16$
Autonomy Granting, *M* (*SD*)	0.00 (0.53)	0.01 (0.52)	$t = 2.36$, $p = 0.02$
Inhibitory Control, *M* (*SD*)	1.76 (0.28)	1.76 (0.27)	$t = 1.84$, $p = 0.07$
Depressive Symptoms, *M* (*SD*)	7.24 (6.84)	7.16 (6.76)	$t = -1.27$, $p = 0.20$

[a]Differences between adolescents retaining in and those dropping out of the study

[b]To minimize potential population stratification bias, the final sample excluded all adolescent of Chinese Minority ethnicity. Thus, the χ^2 test for ethnicity difference was not conducted

incomplete data, and 69 (6.4%) parents refused to complete the questionnaire. The final sample comprised 943 individuals (51.9% girls; $M = 15.24$, $SD = 0.61$). Except for gender distribution and autonomy granting, no significant differences were found between adolescents retaining in and those dropping out of the study in terms of age, genotype, responsiveness, demandingness, inhibitory control, and depressive symptoms (see Table 1).

Procedure

This study was approved by the local ethics committee. Informed assent from adolescents and consent from their parents and school principals were obtained prior to data collection. Adolescents were asked to complete a measure on depressive symptoms, parenting practices and provide a saliva sample for DNA extraction. When adolescents finished the self-report questionnaires, they were given an envelope to take home. The adolescents' parents completed the questionnaires in the envelope regarding children's inhibitory control, and the adolescents took the envelope back to their headteachers the following day. To be specific, during a single class period in their classrooms, trained research assistants (not teachers or staff of their schools) provided detailed instructions for completing questionnaires and a demonstration on saliva collection. Saliva samples were checked one by one on location by research assistants. After data collection, each participant involved acquired a gift (about $1).

Measurements

Depressive symptoms

The Chinese version of Children's Depression Inventory (CDI; Chen et al., 2000; Kovacs, 1992) was used to assess adolescent depressive symptoms. The CDI consists of 27 items. For each item, the participants identified one of the three statements that best described themselves during the past two weeks (e.g., "I am sad occasionally", "I am sad many times", and "I am sad all the time"). Higher sum scores indicate more symptoms. In this study, the Cronbach's alpha was 0.88. The CDI-based single-factor construct of depressive symptoms showed a good fit to the data ($\chi^2 = 914.30$, $df = 324$, RMSEA = 0.04 [0.04, 0.05], CFI = 0.95, TLI = 0.95).

Parenting practices

Adolescents were asked to report their perceived parental *responsiveness* (i.e., acceptance/involvement, 9 items, Cronbach's alpha was 0.85, e.g., 'My parents spend time just talking with me'), *demandingness* (i.e., supervision/strictness, 8 items, Cronbach's alpha was 0.76, e.g., 'How much do your parents try to know where you go at night?'), and *autonomy granting* (reverse scored, 9 items, Cronbach's alpha was 0.68, e.g., 'My parents tell me that their ideas are correct and that I should not question them') via the Parenting Style Index (PSI, Steinberg et al., 1992).

Parenting practices were indexed as the average of standardized items. The three-factor construct of parenting practices showed only fair fit to the data ($\chi^2 = 1343.71$, $df = 292$, RMSEA = 0.062 [0.058, 0.065], CFI = 0.85, TLI = 0.83) with loadings ranging from 0.13 to 0.86 for the items. But the three-factor model showed a statistically improved fit over the one-factor model or the two-factor model with the responsiveness and autonomy granting loading on one factor in this sample according to the Satorra-Bentler chi-square difference test ($\Delta\chi^2$s ≥ 370.79, ps < 0.001). Therefore, the present study examined responsiveness, autonomy granting, and demandingness separately.

Inhibitory control

Inhibitory control was assessed using a subscale of ten items from the parent form of the BRIEF (The Behavior Rating Inventory of Executive Function, Gioia et al., 2002). These ten items asked about children's behavior in everyday situations to assess the inhibitory control of impulsive behaviors (e.g., "gets out of control more than friends."). Items were answered on a 3-point Likert scale ranging from 0 (never) to 2 (often). After reverse coded, the items were averaged to derive a mean score, with higher scores indicating the better ability of inhibitory control. In this study, the Cronbach's alpha of the subscale was 0.81.

Genotyping

Genomic DNA was extracted from participants' saliva samples. DNA extraction and genotyping were performed using the MALDI-TOF in the MassARRAY system (Sequenom Inc., San Diego, California, USA) according to the manufacturer's instructions. The *COMT* gene $Val^{158}Met$ polymorphism was amplified using the following primer sequences: forward ACGTTGGATGACCCAGCGGATGG TGGATTT, reverse ACGTTGGATGTTTTCCAGGTCT GACAACGG. Genotype calling was performed with MassARRAY RT software 3.0.0.4 and analyzed using the MassARRAY Typer software 3.4. The distribution of the three *COMT* genotypes ($N_{MetMet} = 70$, $N_{ValMet} = 340$, $N_{ValVal} = 533$) did not differ significantly from Hardy–Weinberg equilibrium ($\chi^2 = 2.34$, $p = 0.13$). Genotype was dummy coded first using the ValMet group as the reference, resulting in two variables representing the ValVal/ValMet contrast and the MetMet/ValMet contrast.

Statistical analyses

All statistical analyses were conducted using SPSS 23.0. First, the Pearson correlation coefficient (r) and t-test were used to examine associations between all study variables. Second, the mediation and moderated-mediation analyses were conducted using PROCESS macro for SPSS (Hayes, 2013), which provided a bootstrap estimate of the indirect effect between the independent and dependent variable, an estimated standard error, and 95% confidence intervals (CI). When confidence intervals for the indirect effect do not include zero, this indicates a significant indirect effect at the $p < 0.05$ level. Direct and indirect effects were tested using 5000 bootstrap samples. Adolescent's gender and age were included as control variables in all analyses considering the importance of puberty in the development of inhibitory control and depressive symptoms. The corresponding p level was corrected using the Benjamini and Hochberg (B-H) procedure in order to control Type I error (Benjamini and Hochberg, 1995). To avoid inappropriate genetic coding, the genotypes were encoded into two dummy variables (ValVal vs.ValMet and MetMet vs. ValMet) to interpret the genetic mode of *COMT*. Then, based on the results from two dummy variables, the additive, recessive, or dominant model was chosen to re-test the moderating role of *COMT*. Two additional analyses were conducted to test the correspondence of the G × E effect with the diathesis–stress (Monroe & Simons, 1991) and differential susceptibility (Belsky & Pluess, 2009) models. Specifically, both the Roisman et al. (2012) region of significance (RoS) and the Widaman et al. (2012) re-parameterization approaches were conducted to estimate the RoS and the crossover point and its 95% confidence interval (more details can be found in the Appendix). Furthermore, in order to provide further support for the robustness of results, the internal replication was conducted as previous studies did (Cao et al., 2018). Subsequently, the Comprehensive Meta-Analysis (CMA 2.0) program was utilized to transform the results of the two subsamples into the common metric of correlations and to combine effect sizes. Within this meta-analysis, the effect sizes of parenting practices-outcome associations across the two genotype groups (Met allele versus ValVal homozygotes) were computed and compared.

Results

Descriptive Statistics and Preliminary Analyses

Characteristics of the study sample, means, standard deviations, and correlations for main variables were presented in Table 1 and Table 2. All parenting practices showed significant negative correlations with depressive symptoms. Responsiveness and autonomy granting were positively, while demandingness was not, correlated with inhibitory control. Higher level of inhibitory control was associated with lower levels of depressive symptoms. *T*-test analyses revealed that parenting practices did not differ between *COMT* genotypes (ts ≤ 0.46, ps ≥ 0.65),

Table 2 Correlations for all study variables

Variable	1	2	3	4	5	6	7	8
1. Age	1							
2. Gender	0.001	1						
3. *COMT*	0.02	0.02	1					
4. Responsiveness	−0.003	−0.02	−0.01	1				
5. Demandingness	−0.02	-0.25^{***}	−0.01	0.29^{***}	1			
6. Autonomy Granting	−0.01	-0.12^{***}	0.02	0.39^{***}	0.003	1		
7. Inhibitory Control	0.05	-0.14^{***}	−0.01	0.11^{***}	0.02	0.15^{***}	1	
8. Depressive Symptoms	−0.02	0.04	0.01	-0.45^{***}	-0.12^{***}	-0.30^{***}	-0.13^{***}	1

$^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

indicating the absence of correlation between genes and environment (*r*GE).

Mediation Analyses

Both the relation between responsiveness and autonomy granting and depressive symptoms were mediated by inhibitory control (responsiveness: indirect effect = −0.05, *SE* = 0.03, 95% CI [−0.12, −0.01]; autonomy granting: indirect effect = −0.08, *SE* = 0.04, 95% CI [−0.16, −0.02]). Specifically, responsiveness ($b = 0.10$, $p = 0.001$) and autonomy granting ($b = 0.14$, $p = 0.001$) were positively associated with inhibitory control, which in turn was negatively associated with depressive symptoms (responsiveness: $b = -0.53$, $p = 0.01$; autonomy granting: $b = -0.56$, $p = 0.01$). When inhibitory control was included in the model, the direct effects of responsiveness ($b = -2.96$, $p < 0.001$) and autonomy granting ($b = -1.96$, $p < 0.001$) on depressive symptoms remained significant. These findings remain robust after B-H correction for multiple testing. In contrast, the indirect effect from demandingness to depressive symptoms via inhibitory control was not significant (indirect effect = 0.01, *SE* = 0.03, 95% CI [−0.04, 0.08]).

Moderated Mediation Analyses

The moderated mediation models for responsiveness and autonomy granting were first tested using the two-dummy coded genotypes. The results showed that the indirect effect of inhibitory control was stronger in ValVal carriers compared to ValMet carriers (responsiveness: effect = −0.08, *SE* = 0.05, 95%CI [−0.19, −0.001]; autonomy granting: effect = −0.10, *SE* = 0.06, 95%CI [−0.24, −0.01]). There was no difference in the indirect effect between MetMet and ValMet carriers (responsiveness: effect = −0.07, *SE* = 0.06, 95%CI [−0.21, 0.04]; autonomy granting: effect = −0.02, *SE* = 0.07, 95%CI [−0.17, 0.12]). In addition, the genotype was also dummy coded using the MetMet group as the reference, the difference in the indirect effect between MetMet and ValVal was not significant (responsiveness: effect = −0.01, *SE* = 0.06, 95%CI [−0.13, 0.10]; autonomy granting: effect = −0.08, *SE* = 0.08, 95%CI [−0.25, 0.05]). According to Aliev et al. (2014) recommendation, this pattern of genetic mode was corresponded to a recessive model for Val.

Table 3 Conditional indirect effects of responsiveness on depressive symptoms by *COMT*

Model	*Effect*	*SE*	95% CI
Responsiveness to Depressive symptoms via Inhibitory control			
ValVal	−0.08	0.04	[−0.19, −0.02]
Met	−0.02	0.03	[−0.09, 0.03]
Autonomy granting to Depressive symptoms via Inhibitory control			
ValVal	−0.12	0.05	[−0.23, −0.03]
Met	−0.02	0.03	[−0.09, 0.03]

Subsequently, the recessive model was adopted to test the moderated mediation model using a bootstrapping procedure for responsiveness and autonomy granting (see Table 3, Fig. 1, and Fig. 2). The overall tests of moderated mediation were significant for inhibitory control (responsiveness: effect = −0.07, *SE* = 0.05, 95%CI [−0.19, −0.003]; autonomy granting: effect = −0.09, *SE* = 0.06, 95%CI [−0.22, −0.01]). The results showed that *COMT* gene moderated the associations between parenting practices and inhibitory control (responsiveness: $b = 0.13$, $p = 0.047$; autonomy granting: $b = 0.16$, $p = 0.01$). These interactions remain robust after B-H correction for multiple testing. Specifically, the indirect effects from responsiveness to depressive symptoms via inhibitory control were significant for the ValVal homozygotes group but not for the Met allele group (see Table 3). In addition, *COMT* gene did not moderate the direct effect of responsiveness ($b = -0.32$, $p = 0.43$) and autonomy granting ($b = -0.01$, $p = 0.99$) on depressive symptoms.

To interpret the pattern of the G × E interaction (see details in the Appendix), the Widaman et al. (2012) competitive model-fitting approach was conducted using the

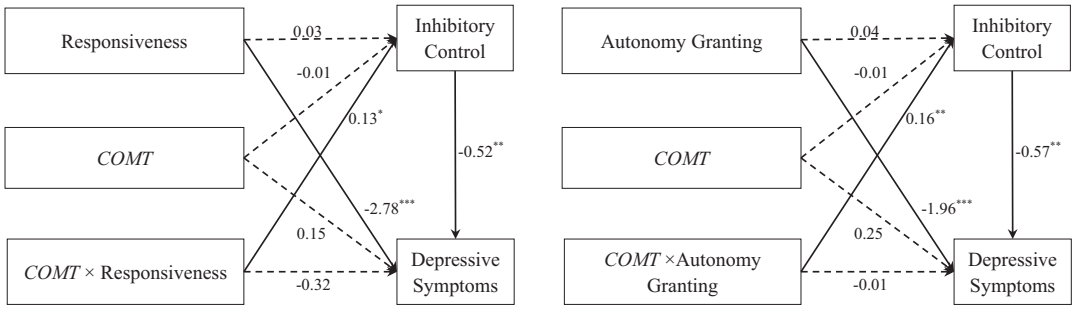

Fig. 1 The moderated-mediation model testing whether *COMT* moderates the direct and indirect effects among responsiveness, inhibitory control, and depressive symptoms. Age and gender not depicted here, were included as covariates. *Dash line*, non-significant paths; solid line, significant paths. Note: $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

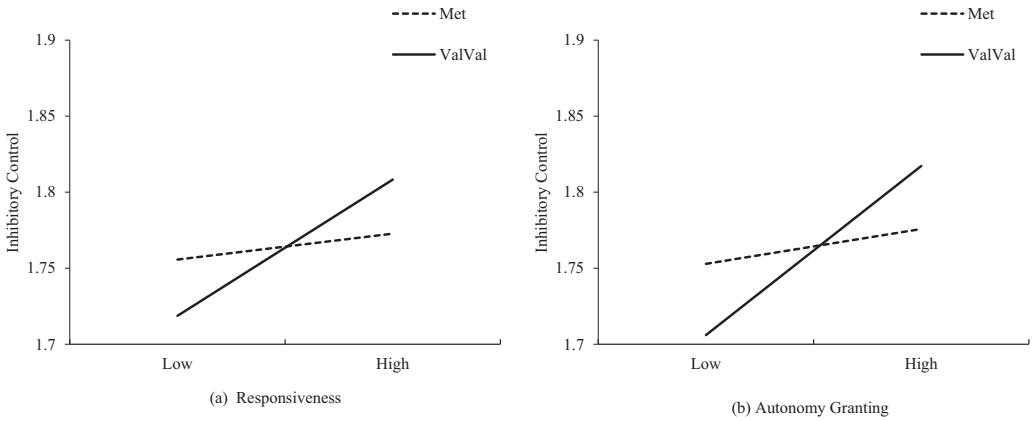

Fig. 2 Interaction between *COMT* and parenting practices associated with adolescent inhibitory control. Simple slopes predicting inhibitory control from responsiveness (**a**) and autonomy granting (**b**) in different genotype groups

parenting practices and the recessive genetic group (Met allele versus ValVal homozygotes), with a priori hypothesis of ValVal homozygotes being more sensitive. As shown in Table 4 and Table 5, the crossover point was 0.04 (95% CI [−0.94, 1.02]) and 0.08 (95% CI [−0.70, 0.86]) for responsiveness and autonomy granting respectively, which fell within the range of parenting practices (responsiveness ranged from −4.69 to 1.42 and autonomy granting ranged from −3.59 to 2.54). The estimated lower and upper bounds of regions of significance for autonomy granting were −1.07 and 1.47 respectively, which fell within the range of parenting practices. That is, adolescents carrying ValVal homozygotes would exhibit better inhibitory control than Met allele carriers when the standardized score of autonomy granting was higher than 1.47. However, adolescents with ValVal homozygotes would exhibit poor inhibitory control than Met allele carriers, if the standardized score of autonomy granting was lower than −1.07. But the lower (−4.85) and upper bounds (7.94) of regions of significance for responsiveness fell outside of its range. This result indicated that the difference in inhibitory control between ValVal homozygotes and Met allele carriers was not significant within the observation range of responsiveness although the slopes for the ValVal homozygotes differing (marginally) significantly from the slopes for the Met allele carriers.

Internal Replication and Meta-Analysis

To test the robustness of these findings, an internal replication analysis was conducted by randomly splitting the total sample into two subsamples ($N1 = 476$; $N2 = 467$). None of the primary variables (i.e., gender, age, *COMT* genotypes, depressive symptoms, inhibitory control, responsiveness, demandingness, and autonomy-granting) were significant different between two subsamples ($|\chi^2|$ s < 0.81, ps > 0.05; $|t|$ s < 1.93, ps > 0.05). The moderated mediation model for responsiveness (effect = −0.15, SE = 0.11, 95%CI [−0.40, 0.00]) and autonomy-granting (effect = −0.21, SE = 0.14, 95%CI [−0.54, −0.01]) were replicated in subsample 1, but not in subsample 2 (responsiveness: effect = −0.03, SE = 0.04,

Table 4 Re-parameterized regression analyses for parental responsiveness and autonomy granting

Parameter	Responsiveness				Autonomy Granting			
	Differential Susceptibility		Diathesis-Stress		Differential Susceptibility		Diathesis-Stress	
	Strong: Model a	Weak: Model b	Strong: Model c	Weak: Model d	Strong: Model a	Weak: Model b	Strong: Model c	Weak: Model d
B_0	1.48 (0.22)***	1.48 (0.22)***	1.49 (0.22)***	1.52 (0.22)***	1.47 (0.22)***	1.46 (0.22)***	1.49 (0.22)***	1.56 (0.22)***
C	0.03 (0.41)	0.04 (0.50)	1.42 (—)^{b}	1.42 (—)^{b}	0.07 (0.32)	0.08 (0.40)	2.54 (—)^{b}	2.54 (—)^{b}
95% *CI* of C	[−0.77, 0.83]	[−0.94, 1.02]	–	–	[−0.56, 0.70]	[−0.70, 0.86]	–	–
B_1	0.04 (0.01)***	0.04 (0.01)***	0.02 (0.01)**	0.03 (0.01)***	0.06 (0.01)***	0.06 (0.01)***	0.02 (0.01)*	0.04 (0.01)***
B_2	0.00 (–)^{b}	0.01 (0.01)	0.00 (–)^{b}	0.02 (0.01)*	0.00 (–)^{b}	0.01 (0.01)	0.00 (–)^{b}	0.03 (0.01)***
B_3	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)	0.02 (0.01)
B_4	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***	−0.07 (0.02)***
R^2	0.036	0.036	0.029	0.033	0.045	0.046	0.028	0.041
F (df)	8.67 (4, 938)***	7.00 (5, 937)***	9.44 (3, 939)***	8.12 (4, 938)***	11.08 (4938)***	9.00 (5937)***	9.07(3939)***	9.94(4938)***
F vs. a (df)	–	0.38 (1, 937)	6.19 (1, 938)*	–	–	0.71 (1937)	16.67 (1938)***	–
F vs. b (df)	0.38 (1, 937)	–	3.28 (2, 937)*	2.50 (1937)	0.71 (1937)	–	8.69 (2937)***	5.10 (1937)*
AIC	208.00	209.63	212.21	210.14	198.69	199.98	213.30	203.09
BIC	237.10	243.57	236.45	239.24	227.79	233.92	237.55	232.19

CI confidential interval, *F* vs. a refers to *F* tests of the difference in R^2 for a given model versus the strong differential susceptibility model, the superscript *b* refers to parameter constrained to a certain value, and its *SE* is not applicable, so is listed as—

$^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

Table 5 Regions of significance (RoS) for statistically significant ($p < 0.05$) COMT by parenting practices variable interactions

Parenting practices	RoS of X		Range of X	PoI	PA	X^2 *or* ZX^2	Crossover Point
	Lower bound	Upper bound					
Responsiveness	−4.85	7.94	[−4.69, 1.42]	0.08	0.48	*ns*	0.04
Autonomy Granting	−1.07	1.47	[−3.59, 2.54]	0.31	0.47	*ns*	0.08

PoI the proportion of interaction, *PA* the proportion affected, X^2 *or* ZX^2 represents whether X^2 or ZX^2, or the set of both nonlinear terms together was statistically significant in the equation $Y = B_0 + B_1X + B_2Z + B_3XZ + B_4X^2 + B_5ZX^2 + B_6\text{Gender} + B_7\text{Age}$; *X* and *Z* represent parenting practices and the *COMT* gene, respectively; ns, not significant; Crossover denotes the value of *X* (parenting practices, standardized to $M = 0$, $SD = 1$) at which the regression lines intersected

95%CI [−0.13, 0.05]; autonomy granting: effect = −0.04, $SE = 0.05$, 95%CI [−0.16, 0.04]).

The meta-analysis revealed that the combined effect size for the associations between parenting practices and inhibitory control amounted to $r = 0.18$ (responsiveness: $p < 0.001$, 95% CI = 0.10, 0.26) and $r = 0.22$ (autonomy granting: $p < 0.001$, 95% CI = 0.13, 0.30) for carriers of ValVal homozygotes in a homogeneous set (Q ($df = 1$) ≤ 1.97, $p > 0.05$, $I^2 = 49.24\%$), respectively. The combined effect sizes for Met allele carriers were $r = 0.04$ ($p = 0.47$, 95% CI = −0.06, 0.13) and $r = 0.03$ ($p = 0.60$, 95% CI = −0.07, 0.12) in a homogeneous set (Q ($df = 1$)s ≤ 0.99, ps ≥ 0.05, I^2s ≤ 0.00%), respectively. The difference between two genotype groups was significant (responsiveness: $Q_{\text{contrast}} = 3.51$, $p = 0.06$; autonomy granting: $Q_{\text{contrast}} = 8.49$, $p = 0.004$), supporting the results that carriers of the ValVal homozygotes were more sensitive to parenting practices. The combined effect size for the associations between inhibitory control and depressive symptoms amounted to $r = -0.09$ ($p < 0.01$, 95% CI = −0.15, −0.02) in a homogeneous set (Q ($df = 1$) = 1.15, $p = 0.28$, $I^2 = 12.88\%$).

Discussion

A broad range of positive parenting practices, such as responsiveness, demandingness, and autonomy granting, has been identified as an important protective predictor for adolescent depressive symptoms. However, it remains unclear how such parenting practices operate to influence depressive symptoms and why some individuals benefit more than others from good quality rearing. To address these gaps, this study adopted a moderated meditation model to examine the effect of parenting practices on adolescent depressive symptoms, as mediated by inhibitory and moderated by the *COMT* gene.

The present study showed that the effects of parental responsiveness and autonomy granting, but not demandingness, on adolescent depressive symptoms were mediated by inhibitory control. Additionally, the associations among such parenting practices, inhibitory control, and depressive symptoms were moderated by the *COMT* gene. Adolescents with ValVal homozygotes were more reactive to the protective effects of positive parenting practices compared to adolescents carrying Met allele. The present study sheds light on the potential for the cognitive theory of psychopathology in understanding the relation between parenting practices and depressive symptoms and provided evidence that the *COMT* gene could regulate the degree to which a child is influenced by environment.

As expected, parental responsiveness, demandingness, and autonomy granting were associated with decreased levels of depressive symptoms. These findings were consistent with previous studies (e.g., Pinquart, 2017; Yap et al., 2014), and provided further evidence that positive parenting practices protected adolescents from the risk of depressive symptoms. More importantly, consistent with the cognitive theories of psychopathology (Nolen-Hoeksema & Watkins, 2011), this study suggested a potential pathway, in which responsiveness and autonomy granting reduce depressive symptoms through the improvement of inhibitory control. Adolescents perceiving their parents as providing more responsive parenting practices exhibited better inhibitory control, and in turn, decreased risk of depressive symptoms. As outlined earlier, adolescents in a responsive and autonomy-encouraging family context may be more motivated to internalize parental directions, a process necessary for conceptualizing alternative nondominant responses (Kochanska & Kim, 2014; Matte-Gagne et al., 2015). Besides, responsive parenting practices facilitate superior brain functioning in ways that are likely to promote inhibitory control. For instance, adolescents with responsive parents have thinner ACC (Rao et al., 2010; Whittle et al., 2014), which in turn associated with a better ability to inhibit a dominant response (Vijayakumar et al., 2014). Improvement in inhibitory control is critical for success in inhibiting negative beliefs or attentional bias, thereby decreasing depressive symptoms (Connell et al., 2019).

It is noteworthy that the mediating effect of inhibitory control did not hold for the demandingness model. It is possible that parental demandingness or control has a less strong effect on inhibitory control in adolescence, a period

when individual demands for autonomy significantly increase (Lionetti et al., 2019; McElhaney et al., 2009). Besides, previous research revealed that the effects of responsive parenting practices on inhibitory control and depressive symptoms were stronger than parental demanding strategies involving limit-setting and monitoring (Beaver et al., 2007; Garthe et al., 2015). Overall, these results suggested that control and affective aspects of parenting practices are likely to involve—in somewhat different psychosocial mechanisms—in the etiology of depressive symptoms. Thus, differentiating different dimensions of parenting practices may help to further clarify the processes by which parenting practices eventuate adolescent depressive symptoms.

Through the moderated-mediation analyses, this study found that inhibitory control mediated the relation between parental responsiveness, autonomy granting, and adolescent depressive symptoms only for ValVal homozygotes. This result was consistent with previous research (Blair et al., 2015; Park et al., 2017; Zhang et al., 2018), children carrying ValVal homozygotes exhibited better inhibitory control in the presence of good quality of rearing relative to children with Met allele. Such findings that the susceptibility to parenting influence depended on the *COMT* is well in line with the evidence from fMRI studies indicating that this polymorphism impacts the neural circuit that has been implicated in sensitivity to environment. The ValVal homozygotes of *COMT* were reported to be associated with decreased connectivity between the PFC and the amygdala (Drabant et al., 2006; Klucken et al., 2015). It has been hypothesized that weakened functional connectivity in corticolimbic circuits may render adolescents especially vulnerable to the influence of environment (Andersen & Teicher, 2008). Similarly, results from prior studies indicated that the ValVal homozygotes confer greater sensitivity to stress, which in turn increases susceptibility to impaired executive functioning in contexts of high adversity (Zhang et al., 2018). As outlined earlier, the relation of dopamine to executive functioning approximates an inverted U-shape function. Thus, the intermediate levels of dopamine resulting from the coordination of a certain *COMT* genotype and basal dopamine levels, appear to be optimal for inhibitory control performance. When dopamine levels peak during puberty (Benes et al., 2000), decreased catecholamine availability conferred by the ValVal homozygotes would result in optimal levels of dopamine, which enhances sensitivity to environments (Andersen & Teicher, 2008). Since the basal level of dopamine was not assessed in this study, future studies should contain direct measures of dopamine levels of relevant neural circuitry to replicate the effect of the *COMT* gene. Such an approach may move us away from simplistic notions of sensitive alleles, recognizing which allele conferring environmental sensitivity depends in part on the background on which the gene exerts its effect. Moreover, most of the adolescents in this study may have entered puberty and pubertal changes become more prevalent and stronger in effect, which might be altering other aspects related to inhibitory control in addition to the basal levels of dopamine. Thereby, future studies should consider the puberty stage in examining the G × E effects. In addition, the G × E term predicted inhibitory control directly, rather than depressive symptoms. This finding suggested that inhibitory control may be an endophenotype closer along the causal chain to the gene, then the strength of the G × E effects was stronger for inhibitory control than for the distal outcomes (i.e., depressive symptoms in the present study). It may also offer evidence for the mediated pleiotropic effect, the phenomenon in which individual genetic variants affect more than one phenotypic trait (Solovieff et al., 2013). The pleiotropic effect reflects the overlap in genetic influence between multiple traits, which may provide implications for the development of transdiagnostic models of psychopathology.

Due to the accumulating doubt about the robustness of G × E studies, this study made every reasonable effort, including a theoretically grounded conceptual model, more stringent thresholds for statistical significance, large sample size, and internal replication, to yield reliable results. Although the replication efforts failed in subsample 2, the meta-analytically derived *COMT* × parenting practices interactions on inhibitory control and the relation between inhibitory control and depressive symptoms remained statistically significant and the effect sizes were similar in magnitude obtained in the full sample. Internal replication attempts strengthen the reliability of the current findings. However, the limiting statistical power and inflated estimates of true replicability by this split-half validation analysis may also increase the risk of false positives. Clearly, there is a particular need for external replication in independent samples to validate the observed moderated mediation model.

This study has several strengths. First, the relatively large sample size provided sufficient statistical power in examining the moderated mediation model. Second, a more rigorous *p*-value and internal replication strengthen the robustness of the current findings. Third, the utilizing of multiple informants avoided inflating the common method variance.

It is also important to consider the limitations of this study. First, due to the cross-sectional design, the results did not provide information that allowed drawing causal conclusions. Despite the theoretical considerations and prospective studies provided substantial support for this association, future research with longitudinal design will be needed to fully explore the moderated mediation model. Second, these effect sizes in this study although significant were small in magnitude, which is often observed in

candidate gene studies. Recent studies have sought to make more progress by exploring collective contributions of multiple loci at once, such as polygenic risk score profiling and gene-gene interactions, but also hampered by small genetic effects (e.g., Cao et al., 2018; Davies et al., 2019). Thereby, finding the "missing heritability" of complex psychopathology to extend current results is still an important future direction. Third, the lack of analyses to estimate the ancestral information of this sample for adjusting the population stratification is also a limitation, which may confound the results of genetic association studies. Also limiting the present work is that the inhibitory control assessed by the mother-reported questionnaire can be prone to a number of biases, such as reporting biases and recall biases.

Conclusion

Responsive, demanding, and autonomy granting parenting practices often protect adolescents from the risk of depressive symptoms. Yet, it is important to note that such parenting practices may operate to reduce depressive symptoms through certain proximal cognitive factors. Besides, adolescents may be disproportionately susceptible to the beneficial effects of positive parenting practices. The current study tested the mediating role of inhibitory control in the links between parenting practices and depressive symptoms, with *COMT* gene as a moderator using cross-sectional data. The findings indicated that responsiveness and autonomy granting fostered better inhibitory control, which in turn reduced the risk of depressive symptoms. The results also demonstrated that adolescents residing in similar responsive and autonomy-supportive families exhibit differential paths of adaptation. The *COMT* gene moderates the mediational pathway from responsiveness and autonomy granting to inhibitory control, such that adolescents with ValVal homozygotes may be more benefitted from responsive and autonomy-supportive parenting practices than Met allele carriers, and consequently had greater inhibitory control. The study of cognitive (i.e., inhibitory control) and biological (i.e., *COMT* gene) factors provides a valuable multifactorial model for understanding the complex mechanisms by which positive parenting practices reduce the risk of depressive symptoms. Furthermore, these findings have implications for adolescent depressive symptoms interventions by highlighting the protective effects of responsive parenting practices, as well as the importance of considering individual differences in genetic susceptibility.

Authors' Contributions Y.C. conceived of the study and participated in the interpretation of the data, performed the statistical analysis, and drafted the manuscript; G.C. conceived of the study, participated in its design and coordination, and helped to drafted the manuscript; L.J. participated in the design; W.Z. helped to draft the manuscript and participated in the interpretation of the data. All authors read and approved the final manuscript.

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Data Sharing and Declaration This manuscript's data will not be deposited.

Compliance with Ethical Standards

Conflicts of Interest The authors declare no competing interests.

Ethical Approval This study was approved by the Ethics Committee of Shandong Normal University. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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Appendix

Notably, literature on *COMT* by environment interactions has largely focused on negative environment and to a lesser extend to positive environment, such as positive parenting practices in this study. These studies have adopted the diathesis-stress model which argues individuals with vulnerable genes would suffer from negative environment compared to others (Monroe & Simons, 1991). But the differential susceptibility hypothesis holds that genes could be plasticity rather than vulnerability (Belsky & Pluess, 2009). That is individuals with plasticity alleles not only suffer from negative environment but also benefit from positive environment. Therefore, the present study tested two competing hypotheses about G×E interaction: the diathesis-stress hypothesis and the differential susceptibility hypothesis.

The Widaman et al. (2012)'s approach, which involves a re-parameterization of standard multiple linear equations by centering parenting practices at the crossover point on parenting practices (see Eq. 1):

$$Y = \begin{cases} GROUP = 1,\ Y = B0 + B1(X1 - C) + B3X3 + B4X4 + E \\ GROUP = 0,\ Y = B0 + B2(X1 - C) + B3X3 + B4X4 + E \end{cases} \quad (1)$$

where Y is inhibitory control, GROUP refers to allelic groups (0 = MetMet or ValMet and 1 = ValVal), *X1* refers to responsiveness or autonomy granting (standardized; ranged from −4.69 to 1.42 and −3.59 to 2.54, respectively), *X3* and *X4* refer to age and gender respectively, and E is the error term. *B0* is the intercept, *B1* is the slope for parenting practices for risk/plasticity alleles, *B2* is the slope for parenting practices for non-risk/non-plasticity alleles, *B3* and *B4* are slopes for age and gender respectively, *C* is the crossover point where the slopes for allelic groups cross. If *C* and its 95% confidence interval (CI) falls within the range of parenting practices, the G × E effect conforms to the disordinal form, supporting the differential susceptibility model. Otherwise, if *C* and 95% CI fall at or over the maximum of parenting practices, the G × E interaction is ordinal, supporting the diathesis-stress model (Widaman et al., 2012).

These two models can be further subdivided into "strong" and "weak" models. Strong models assume that "non-risk/non-plasticity alleles" carriers are not susceptible to environments (i.e., constraining $B2 = 0$), whereas weak versions assume that these individuals are to a lesser extent susceptible to environments than others with "risk/plasticity alleles" (i.e., relaxing $B2 = 0$). The present study tested all four models, comparing them on the basis of variance accounted (i.e., R^2), Akaike, and Bayesian information criteria (AIC and BIC). Models explaining more variance, hence better representing the data are favored. A model that is more parsimonious is preferred if two models explain a comparable amount of variance. Furthermore, smaller values of AIC and BIC indicate better model fit and are particularly useful when comparing non-nested models.

As shown in Table 4, in the model of responsiveness, the point estimate of the crossover point in model b, $C = 0.04$ ($SE = 0.50$), and its 95 % confidence intervals [−0.94, 1.02], fell within the range of parenting practices (ranged from −4.69 to 1.42), thus supporting the differential-susceptibility model, rather than the diathesis-stress model. Furthermore, constraining the slope of Met allele group to 0 (i.e., $B2 = 0$) as strong differential-susceptibility, there was no significant changes in the model fits ($\Delta R^2 = 0.00$, $F = 0.38$, $p = 0.54$), thus supporting the strong differential-susceptibility model. Moreover, the strong differential-susceptibility model (Model a) explained the largest variance and had the lowest AIC values among the four alternative models. Although the BIC value was smaller in Model c, considering the biased tendency of BIC favoring a simpler model and small value difference, the strong differential-susceptibility was the optimal model.

Similarly, in the model of autonomy granting, the point estimate of the crossover point in model b, $C = 0.08$ ($SE = 0.40$), and its 95 % confidence intervals [−0.70, 0.86], fell within the range of parenting practices (ranged from −3.59 to 2.54), thus supporting the differential-susceptibility model, rather than the diathesis-stress model. Furthermore, constraining the slope of Met group to 0 (i.e., $B2 = 0$) as strong differential-susceptibility, there was no significant changes in the model fits ($\Delta R^2 = 0.001$, $F = 0.71$, $p = 0.40$), thus supporting the strong differential-susceptibility model. Moreover, the strong differential-susceptibility model (Model a) explained the largest variance and had the lowest AIC and BIC values among the four alternative models. Therefore, the strong differential-susceptibility was the optimal model.

Besides, the region of significance (RoS) analysis was conducted (Roisman et al., 2012). First, the lower and upper bound, where the association between gene and inhibitory control is significant was estimated. Second, the crossover points at which the regression lines intersect are desired to be near 0. Third, the proportion of interaction (PoI) which is represented on the right side of the crossover point should be near 0.50. Fourth, the proportion affected (PA) which represents the proportion of the population that is differentially affected by the moderator should be near 50 % and greater than 16 %. Finally, the X^2 or ZX^2 term should be not significant to avoid the nonlinear diathesis-stress phenomenon.

As shown in Table 5, the estimated lower and upper bound of RoS for responsiveness fell outside of its range, which indicated that the difference in inhibitory control between ValVal and Met carriers was not significant within the observation range of responsiveness. This result indicated that the G × E effect for responsiveness is very small in magnitude. In the autonomy granting model, the lower and upper bound of RoS were −1.07 and 1.47. That is, adolescents carrying ValVal homozygotes would exhibit better inhibitory control than Met carriers when the standardized score of autonomy granting was higher than 1.47. However, adolescents with ValVal homozygotes would exhibit poor inhibitory control than Met carriers, if the standardized score of autonomy granting was lower than −1.07. The crossover point (0.08) was near 0 for parenting practices that have been standardized, PoI (0.31) was near 0.50, and PA (47%) was greater than 16 % (Roisman et al., 2012). These statistical indexes provided support for the differential susceptibility model, which argued that individuals with certain alleles would be more affected than others—both for better and for worse—by their rearing experiences (Belsky & Pluess, 2009).

Table 4 and Table 5

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