Examining The Link Between Harsh Parenting And Non-Suicidal Self-Injury In Adolescence: The Role of The COMT Val158Met Polymorphism And Depressive Symptoms

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Abstract

Background: Previous studies have suggested negative parenting environments, especially harsh parenting, is a specific risk factor for non-suicidal self-injury (NSSI). However, the potential mechanism between harsh parenting and NSSI has not been explored. Based on the experiential avoidance model and empirical researches, we aimed to examine if depressive symptoms are a mediator between harsh parenting and NSSI. Moreover, the catechol-O-methyltransferase (COMT) Val158Met polymorphism related to depressive symptoms may also exert a moderating effect on NSSI, thus, the interaction between harsh parenting and COMT were also considered in our study.

Method: 373 junior high school students were recruited for the study by using a longitudinal design. Adolescents answered self-report questionnaires and provided Saliva samples for DNA genotyping.

Result: The results revealed that harsh parenting was positively associated with NSSI after 18 months, and this association was mediated by depressive symptoms. Moreover, the moderating role of COMT in the direct and indirect effect of harsh parenting on NSSI only among adolescents with two Val alleles. However, the relationship was not significant for Met carriers.

Conclusion: Genetic variations of COMT Val158Met may be a critical candidate in understanding the development of depression and NSSI. We conclude that the Val homozygotes of the COMT Val158Met polymorphism play a susceptible role both in depressive symptoms and NSSI.

Introduction

Non-suicidal self-injury (NSSI) refers to the deliberate, direct, and socially unacceptable destruction of one's own body tissue without conscious suicidal intent [1], has consistently been reported to be associated with a variety of emotional or borderline personality disorders and increased risks for suicide [2, 3]. The onset and prevalence of NSSI are especially common in adolescence. A recent meta-analysis showed a NSSI lifetime prevalence of 17.2%, 13.4% and 5.5% in adolescents, young adults, and adults respectively [4]. The high rate of NSSI behavior in Chinese adolescent is even up to 22.4%-29% [5, 6]. Clearly, adolescent NSSI is a serious public health concern worldwide [7], and it is important to identify the potential mechanisms and etiology of NSSI during this period.

Harsh parenting refers to a wide range of aversive parenting behaviors, includes physical (e.g., spanking, slapping, or hitting) and verbal punishment (e.g., yelling and cursing) at children who have done something wrong [8, 9]. According to Linehan [10], exposure to harsh parenting may influence the likelihood of engaging in NSSI behaviors. Victor et al. [11] also emphasized harsh parenting such as shouting, swearing, spanking, predicted increased odds of subsequent adolescent NSSI onset in a longitudinal design. Although previous research has investigated the linkage between harsh parenting and NSSI in Western countries [12, 13], research into this issue is still rare in China. As a Chinese proverb, "Beating and scolding is the emblem of love", in the traditional Chinese culture context, harsh parenting behaviors were generally considered as an indication of parental involvement, concern, and love,

therefore, harsh punishment is still adopted by approximately 50% parents in China [14–16]. Considering the cultural differences, it is necessary to further investigate the relationship between harsh parenting and NSSI in China.

Depressive symptoms have been frequently identified as a mediator of interpersonal risk factors and NSSI. On the one hand, NSSI is often considered to be an emotion-regulation strategy to decrease youth's emotional distress by distracting from intense emotion through the sight of blood, the sensation of pain, or a focus on the injury itself [1, 17]. Experiential avoidance model (EAM) proposed that NSSI is maintained by negative reinforcement in the form of escape from unwanted emotional experiences [18]. Consistent with the model, individuals who experience higher levels of anhedonia and depression, are more likely to report a history of NSSI [19, 20]. On the other hand, ample empirical studies have revealed the mediating role of depression between interpersonal stressors and NSSI among adolescents. Recent findings from a longitudinal study showed that depressive symptoms play a mediating role in the association between peer bullying and NSSI [21]. Meanwhile, Madjar et al. [22] also found that sense of loneliness in school could increase the risk of NSSI by increases in severity of depressive symptoms. Although previous studies have indicated correlations of harsh parenting and depressive symptoms with NSSI [20, 23, 24], no research has explicitly addressed the mechanisms underlying harsh parenting, depression, and NSSI, especially lack of evidence from longitudinal studies. To bridge the research gaps, we conducted a three-waves longitudinal study to evaluate the longitudinal associations among harsh parenting, depressive symptoms, and NSSI in a community sample of Chinese adolescents.

Not all individuals who experience harsh parenting and depression engage in NSSI, Maciejewski et al. [25] indicted that NSSI was influenced by both environment and genetic. Brodsky [26] proposed a diathesis-stress models for suicide and NSSI by combined the findings from the fields of biology, neurology, and genetics, which provides a specific framework to understand how gene–environment (G × E) interactions correlated with suicidal or NSSI behaviors directly. However, through this framework previous studies mostly considering the genetic factor of suicide, only a few studies have identified the genetic factors related to NSSI such as serotonin transporter gene (5-HTTLPR) and brain-derived neurotrophic factor (BDNF Val66Met) gene [27, 28]. Catechol-O-methyltransferase (COMT) located on chromosome 22q11.1-q11.2 [29], and plays an important role in regulating an individual's processing of emotion and cognition by inactivating catecholamine neurotransmitters. The activity of the COMT enzyme is influenced mainly by a functional single nucleotide polymorphism (rs4680; G to A) in the COMT coding region causes Val158Met aminoacid substitution in the corresponding protein, with Val allele exhibiting a 3- to 4-fold increase in enzyme activity compared to Met allele [30, 31]. Considering COMT Val158Met gene is associated with several traits related to NSSI behaviors, including emotion regulation [32, 33] and borderline personality disorders [34, 35] and suicidal behaviors [36, 37]. Thus, we speculate that the COMT Val158Met gene might moderate the relationship between harsh parenting and NSSI directly.

According to Brodsky [26], diathesis-stress model for suicide and NSSI also highlighted the complex interaction between environment and genetics factors could impact the expression of neurological and phenotypes (e.g., emotional dysregulation) that predispose to suicide and NSSI, thereby increasing the

diathesis for the propensity to react to stressors for NSSI. That means, Phenotypes bridge the gap between the distal risk genes and the elusive disease process [38]. Namely, phenotypes would play a meditator role in the relationship between G × E interactions and behavior outcomes. As we mentioned before, depression symptoms as a mediator may be useful for elucidating the role of biological mechanisms in the risk for NSSI. There are numerous genetic association studies implicating the COMT Val158Met polymorphism in the incidence of major depression disorder [38–40]. In the context of G × E, COMT Val158Met has been found to interact with environmental variations to predict outcomes related to depression symptoms. For example, a longitudinal study has shown that the Val-allele children were more likely to develop depression after exposure to high-risk nurturing environment [41]. Cao et al. [42] also found that adolescents with Val/Val genotype were more sensitive to depression under negative peer relationships. Based on the theoretical and empirical evidence, it is plausible to assume that the interaction between COMT Val158Met polymorphism and harsh parenting would lead to NSSI through depressive symptoms.

To examined the potential mechanisms of NSSI, the current study constructs a moderated mediation model (see Fig. 1). Specifically, we proposed the following hypotheses:(1) harsh parenting would be positively associated with NSSI among adolescent; (2) depressive symptoms would mediate the relation between harsh parenting and NSSI; (3) COMT rs4680 would moderates the link between harsh parenting and NSSI, and the link between harsh parenting and depressive symptoms in the mediation model of the relationship between harsh parenting and NSSI.

Method

Participants

Participants were recruited from four junior high schools in Guizhou province, China. We randomly contacted four public junior high schools and their principals' approval of the survey was obtained. Then, at each school, four classes from grades 7 were randomly selected. At baseline (T1), 536 adolescents (M age = 12.80 ± 0.84 years, 52.2% girls) were enrolled, and then participants completed two follow-up assessments which were undertaken at 6 and 12 months from baseline (T 2, n = 516, M age = 13.52 ± 0.84 years, 52.9% girls; T3, n = 373, M age = 14.66± 0.87 years, 54.2% girls). No significant differences were found in the variables of interest (i.e., harsh parenting, depressive symptoms and NSSI) and other demographic variables like age and gender between adolescents who participated in all assessments and those who did not.

Written informed consents were obtained from each participant and their parents and school principals at each data collection. Meanwhile, all participants were notified that their participation is completely voluntary and they have the right to withdraw at any time. After completing each survey, each participant received a gift for their participation. This study was approved by the Research Commission of authors' University and the principals of participating schools.

Measures

Harsh parenting

Harsh parenting was assessed using Chinese version of the Parent–Child Conflict Tactics Scale (CTSPC, [43, 44]. The questionnaire included 18 items and adolescents responded to the stem question “How do your parents react when you have done something wrong or they really don't like?” by rating how often (0 = “never” to 6 = “more than 20 times”) their parents acted according to each item. Psychological aggression was assessed by combining five items like “my parents called me things like, ‘stupid’ or ‘lazy’.”, and physical assault was assessed by combining eleven items like “spanked on bottom with bare hand.”. The items were averaged with higher scores indicating higher levels of harsh parenting during the past year. In the present study, Cronbach’s alphas coefficient for this scale was 0.83 at T1.

Depressive symptoms

Adolescent depressive symptoms were assessed using the Centre for Epidemiological Studies Depression Scale for Children (CES-DC [45]). The scale has been successfully applied to children and adolescents in China [46]. The scale includes 20 items, such as “I do not think I can concentrate on my work.” All responses range from 1 (never) to 4 (always). The items were averaged with higher scores indicating higher levels of depressive symptoms. In the present study, Cronbach’s alphas coefficient for this scale was 0.84 at T1 and 0.87 at T2.

Non-suicidal self-injury

Non-suicidal self-injury was measured using a shortened and modified version of the Deliberate Self-Harm Inventory (DSHI), which was developed and validated by Gratz [47]. The scale includes 9 items, such as “I pluck out my hair deliberately”, “Get yourself electrocuted deliberately without life-threatening”. Each of the items was rated on a 4-point scale from 0 (never) to 3 (more than 5 times), reflecting participants’ frequency of self-injury behavior over the specified time periods (e.g., lifetime, since the last assessment). Scores were calculated by averaging all the responses, with higher scores indicating higher levels of NSSI. In the present study, Cronbach’s alphas coefficient for this scale was 0.83 at T1 and 0.88 at T3.

Genotyping

Genomic DNA was extracted from the children’s buccal mucosa on a cotton swab using a Tissue DNA Kit (BioTeke Corporation, Beijing, China) at T3. The single nucleotide polymorphism (SNP) genotyping was performed using the MassARRAY system (Sequenom Inc., San Diego, California, USA) by means of matrix assisted laser desorption ionization time of flight mass spectrometry method (MALDI-TOF). The COMT rs4680 polymorphism was amplified by polymerase chain reaction (PCR) with forward primer (ACGTTGGATGTAGGTGTCAATGGCCTCCAG) and reverse primer (ACGTTGGATGTCATGGGTGACACCAAGGAG).

Data analysis

First, a preliminary analysis was conducted to compute the means, standard deviations, and correlations among the main variables. Second, the χ2 test was used to tested whether the distributions of COMT rs4680 genotype fit of The Hardy–Weinberg equilibrium. Third, the mediated moderation model displayed in Figure 1 was examined. Given that adolescents' age and gender would account for the individual differences relating to the main variables [20, 48], we controlled these demographic variables in our statistical analyses. Therefore, T1 harsh parenting as the independent variable, T3 NSSI as the outcome variable, T2 depressive symptoms as the mediator, and COMT Val158Met as the moderator, were entered into the mediated moderation model. Age and gender were entered as covariates. Depressive symptoms and NSSI at T1 were also controlled in all subsequent analyses. For the purpose of minimizing multicollinearity, we standardized all the predictors.

SPSS software version 19.0 was used to perform the preliminary and χ2 test and PROCESS macro software (model 8) in SPSS [49] was used to test the moderated mediation displayed in Figure1. Specifically, in PROCESS, Model 8 was applied for testing the mediated moderation displayed in Figure 1. According to the Model 8 [50], the moderated mediating effect was computed using a bias-corrected bootstrapping with 5,000 samples; a 95% confidence interval (CI) that does not include zero indicated a significant effect. If the mediated moderation was significant, slope tests were conducted afterward. According to Aiken et al. [51] two values of harsh parenting, including low (one standard deviation below the mean) and high (one standard deviation above the mean) levels, were defined to acquire detailed information.

Results

Descriptive statistics and correlations

The allele distribution of COMT Val158Met polymorphisms was 202 Val/Val individuals (94 male, 108 female) and 148 Val/Met (66 male, 82 female) and 23 Met/Met individuals (12 male, 11 female), representing distributions previously observed in the Asian sample. No deviations from the Hardy–Weinberg equilibrium were detected for the genotypes ($\chi^2(2) = 0.15$, $p = 0.93$). Based on previous studies [42], we coded COMT genotypes as a two-level, Met carrier model (Val/Val = 1, Met/ Val, Met/ Met = 0).

Table 1 depicts the descriptive statistics and correlations for all study variables. There were no significant associations between COMT genotypes and harsh parenting, indicating the absence of correlation between genes and environment. In addition, the results of the bivariate correlations showed that harsh parenting was positively correlated with NSSI at Time1 and Time 3, respectively, with depressive symptoms at Time1 and Time 2, respectively.

Table 1
Means, Standard Deviations, and Correlations Among All Variables.

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. Age (T1)	12.81	0.87	1							
2. Gender[a]	0.44	0.50	0.12*	1						
3. COMT Val158Met	0.46	0.50	0.01	-0.01	1					
4. Harsh parenting (T1)	0.55	0.60	0.06	-0.004	0.03	1				
5. Depressive Symptoms (T1)	1.99	0.47	0.06	-0.12*	-0.03	0.39**	1			
6. NSSI (T1)	0.12	0.26	-0.07	0.03	-0.02	0.31**	0.30**	1		
7.. Depressive Symptoms (T2)	2.14	0.50	0.01	-0.19*	0.05	0.30**	0.58**	0.28**	1	
8. NSSI (T3)	0.25	0.36	-0.05	-0.01	0.02	0.17**	0.23**	0.39**	0.34**	1

Notes: T1: Time 1; T2: Time 2; T3: Time 3. [a] Female = 0, Male = 1. NSSI represent non-suicidal self-injury. * p < .05. ** p < .01.

Mediated moderation analysis

Table 2 shows a significant longitudinally moderated mediation model for harsh parenting and NSSI relationships. Specifically, as shown in Fig. 2, harsh parenting at T1 was positively associated with NSSI at T3 (β = 0.24, p < 0.001), and was also could significantly positively affect NSSI at T3 through depressive symptoms at T2 (β = 0.19, p < 0.05; β = 0.38, p < 0.001), whereas COMT was not associated with depressive symptoms at T2 (β = 0.07, p > 0.05) and NSSI at T3 (β = -0.01, p > 0.05). Furthermore, COMT significantly moderated the impacts of harsh parenting at T1 on NSSI at T3 (β = -0.23, p < 0.05) and depressive symptoms at T2 (β = -0.22, p < 0.05). The above results showed that the interaction between harsh parenting and COMT would predict adolescent future NSSI level directly, and also partially indirectly affect NSSI through depressive symptoms as a mediating variable.

Table 2
Testing the moderated mediation model in adolescent.

Variable	Depressive Symptoms (T2)			NSSI (T3)		
	β	*SE*	*95% CI*	*β*	*SE*	*95% CI*
Harsh parenting (T1)	0.19*	0.08	[0.03, 0.35]	0.24***	0.09	[0.06, 0.42]
COMT	0.07	0.10	[-0.12, 0.26]	-0.01	0.11	[-0.23, 0.20]
Harsh parenting (T1) × COMT	-0.22*	0.10	[-0.42, -0.01]	-0.23*	0.12	[-0.46, -0.004]
Depressive Symptoms (T2)				0.38***	0.07	[0.24, 0.51]
Age	-0.02	0.06	[-0.13, 0.09]	-0.10	0.06	[-0.23, 0.02]
Gender	0.29*	0.10	[0.09, 0.48]	-0.19	0.11	[-0.41, 0.04]
Depressive Symptoms (T1)	0.50***	0.06	[0.39, 0.61]	0.03	0.07	[-0.11, 0.17]
NSSI (T1)	-0.10*	0.05	[-0.20, -0.004]	0.01	0.05	[-0.10,0.11]
R^2	0.32			0.19		
F	18.30***			7.90***		
Notes:* p < .05. ** p < .01. ** p < .001.						

To facilitate description, a simple slope analysis was conducted to identify the interaction effect between harsh parenting at T1 and COMT on NSSI at T3. As shown in Fig.3, harsh parenting at T1 positively predicted NSSI at T3 only among individuals with Val/Val genotypes (b = 0.30, t = 3.23, p = 0.001, 95% CI = 0.12 to 0.48) compared to Met carriers (b = -0.04, t = -0.68, p = 0.57, 95% CI = -0.19 to 0.11). Meanwhile, to examine the interaction effect between harsh parenting at T1 and COMT on depressive symptoms at T2, we used the same procedures employed for characterizing the interaction for the prediction of NSSI at T3. As shown in Fig. 4, harsh parenting at T1 positively predicted depressive symptoms only among individuals with Val/Val genotypes (b = 0.18, t = 2.22, p = 0.03, 95% CI = 0.02 to 0.34) compared to Met carriers (b = -0.05, t = -0.68, p = 0.50, 95% CI = -0.18 to 0.09). Overall, the results indicated that the moderating effect of COMT only significant for Val/Val individuals while not for Met carriers.

Discussion

Guided by the theorical model from Brodsky [26], the goal of current study was to understand how COMT Val158Met polymorphism interact with harsh parenting to predict NSSI among adolescence. Using a three-wave longitudinal sample of Chinese adolescents, the results indicated that adolescence exposed high level harsh parenting was associated with more NSSI eighteen months later, and depression symptoms partially mediated the association between harsh parenting and NSSI. Moreover, our study found that COMT Val158Met polymorphism moderates the link between harsh parenting and NSSI, and

the link between harsh parenting and depression in the mediation model of the relationship between harsh parenting and NSSI. Notably, the current study expands on previous work to emphasize the NSSI etiology from the perspective of genetic and environment.

In line with Linehan [10], our findings have identified harsh parenting as a significant interpersonal risk factors for NSSI in Chinese adolescence. This suggests that when designing intervention programs to reduce the incidents of NSSI, special attention should be given to adolescence who experience a longer period of physical or verbal punishment from their parents. More importantly, we confirmed that the depressive symptoms play a mediator role of the association between harsh parenting and NSSI. Harsh parenting as a negative parenting way was associated with more reported depressive symptoms [52, 53], which facilitates individuals to engage in NSSI to cope with negative feelings [1]. It supports the Experiential avoidance model that individuals who have intense reactions to emotional stimuli and difficulty regulating emotions may desire to escape from aversive experiences via NSSI. To our knowledge, this is one of the first studies to investigate this longitudinally mediation mechanism underlying the relationship between harsh parenting and NSSI.

When it comes to consider the genetics moderating role in the associations among harsh parenting, depressive symptoms, and NSSI, our findings are contributed to extant research into the negative parenting-NSSI linkage. We found two potential pathways that COMT play a moderating role in the relationship between harsh parenting and NSSI. One of the paths was consistent with our hypothesis that COMT moderated the relation between harsh parenting and NSSI directly. Specially, at highly harsh parenting environments, individuals with two Val alleles reported more NSSI eighteen months later compare to the Met carriers. Previous studies have demonstrated that COMT Val/Val genotype was associated with more persistent dopamine degradation and less synaptic dopamine which might increase flexibility but decrease stability of neural network activation states, hence rendering adolescents susceptible to negative environments [54]. For example, Kwon et al. [55] indicated that compared to the Met carriers genotype, individuals with Val/Val genotype showed higher suicidal ideation when they exposed to negative environment such as child abuse. Given NSSI and suicidal ideation were largely driven by overlapping genetic factors [25], it is possible that COMT Val158Met polymorphism may be one of the genetic factors to interpret both NSSI and suicidal ideation.

The other path was that COMT moderated the relation between harsh parenting and depressive symptoms. Same with the direct path, individuals with Val/Val genotype were exhibited more depressive symptoms when exposed to high harsh parenting, while less depressive symptoms when experiencing low harsh parenting. The result is consistent with prior findings that individuals with two Val alleles may be more susceptible to negative parenting environmental influences and developing depression [39, 41]. Although the definitive mechanisms of the Val/Val genotype being more sensitive to the environment remain an open question, combined with recent empirical studies we speculate that the neurobiological mechanisms may underlie these two interactive effects. Recently, a longitudinal twin study found that exposure to negative parenting would trigger adolescent's depressive symptoms by increasing the connectivity of the amygdala with the ventrolateral prefrontal cortex and this neurobehavioral association

is heritable during adolescence [56]. Furthermore, high-expressed COMT genotype (Val) is associated with increased amygdala activity [57, 58]. Thus, negative parenting and COMT may increase the vulnerable to depressive symptoms through their synergic effects on amygdala circuitry. Taken together, the significant interaction between harsh parenting and COMT Val158Met polymorphism on both NSSI and depressive phenotype provides some empirical evidence for Brodsky's diathesis-stress model of suicide and NSSI [26].

Several limitations need to be considered in our study. First, the current study used adolescent self-report to collect data which could be subjected to bias. So, in the future, studies should attempt to collect data from the children's parents or other caregivers. Second, although our study is the first to suggest the moderating role of COMT Val158Met polymorphism in NSSI, the effect of only a single genetic variant on NSSI was examined in this study, future work should expand the focus on other candidate genes or polygenic risk scores. Third, some studies find that Asian and Caucasian and Mexican samples are differ in their COMT Val158Met polymorphism distribution [59], thus, the results reported herein should interpreted carefully in terms of generalization to the overall adolescent population because of the ethnicity and region of our sample (i.e., Chinese Han).

Despite these limitations, the current study has some implications for educational and clinical practitioners. First, harsh parenting always be a familial risk factor for NSSI. Although harsh parenting is still acceptable in Chinese parents, it contributes to later increases in NSSI. In order to decrease its use, some prevention intervention ways can be used such as changing parents' favorable attitudes toward harsh parenting, teaching parents some skills in emotion coaching, and planning some Positive Parenting Programs [60–62]. Second, it was also found that harsh parenting could increase adolescence's NSSI through increasing their depressive symptoms, that provide insights into the future development of interventions for NSSI. Specifically, interventions that aim at decreasing the depression level caused by harsh parenting may help reduce children's NSSI. Emotion regulation has been identified as an effective intervention strategy for depression and NSSI [63]. Third, when working with adolescent with histories of maltreatment who have also depression, clinicians should take care to screen for self-injury thoughts and behaviors. Forth, individual differences in the susceptibility to harsh parenting environment are at least partially genetic influenced during adolescence. Thus, recognizing susceptible individuals are needed to inform clinical practice and intervention. Additionally, our study first identified the association between NSSI and COMT Val158Met polymorphism, this association was also confirmed in high-risk behaviors such as suicide. It is possible that both suicide and NSSI may share similar biological underpinnings, which suggests that future intervention of NSSI can consider combined some suicide prevention program.

Abbreviations

NSSI: Non-Suicidal Self-Injury; COMT: Catechol-O-methyltransferase

Declarations

Ethics approval and consent to participate: The study was approved by the Research Commission of Beijing Normal University. Participants and their primary caregivers gave written informed consent for the assessment.

Consent for publication: Written informed consent for publication was obtained from all participants.

Availability of data and materials: The datasets analysed in the current study are available from the corresponding author on reasonable request.

Competing Interests: We declare that there are no conflicts of interest with respect to the authorship or the publication of this article.

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Figures

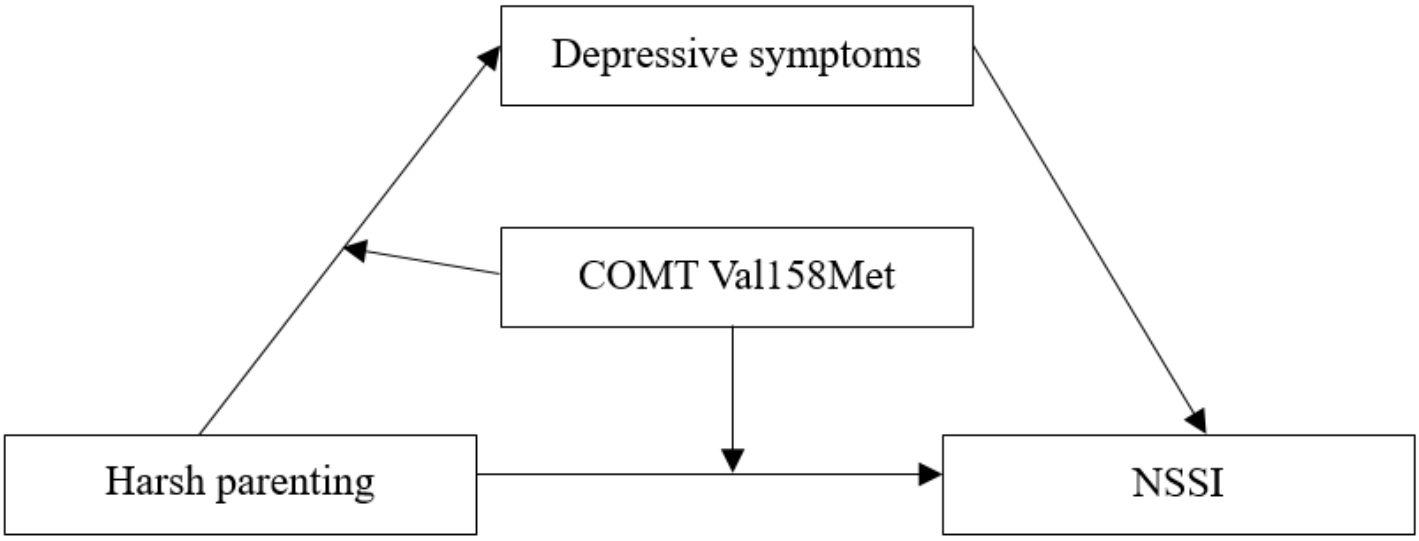

Figure 1

The proposed model of the association between harsh parenting, depressive symptoms, NSSI, and COMT Val158Met.

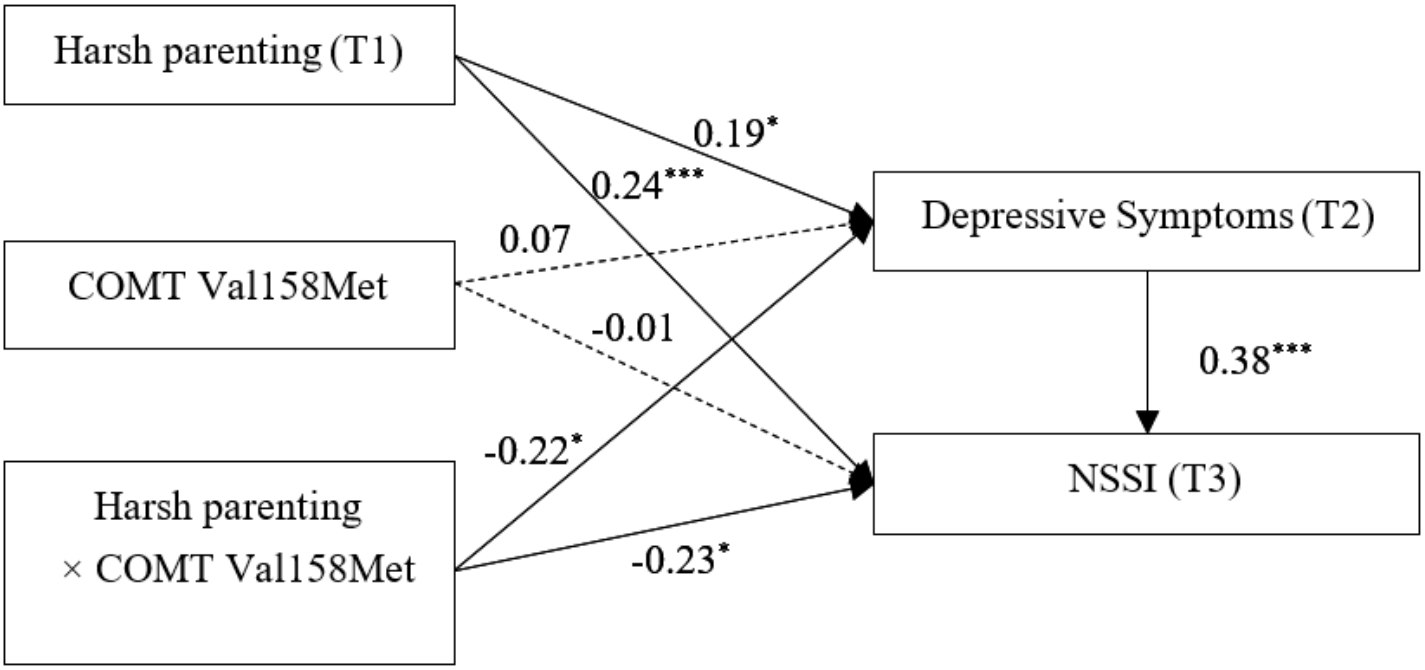

Figure 2

The moderated mediation model among adolescent T1 to T3. Note. The dotted line is not significant; *p < 0.05, **p < 0.01, ***p < 0.001.

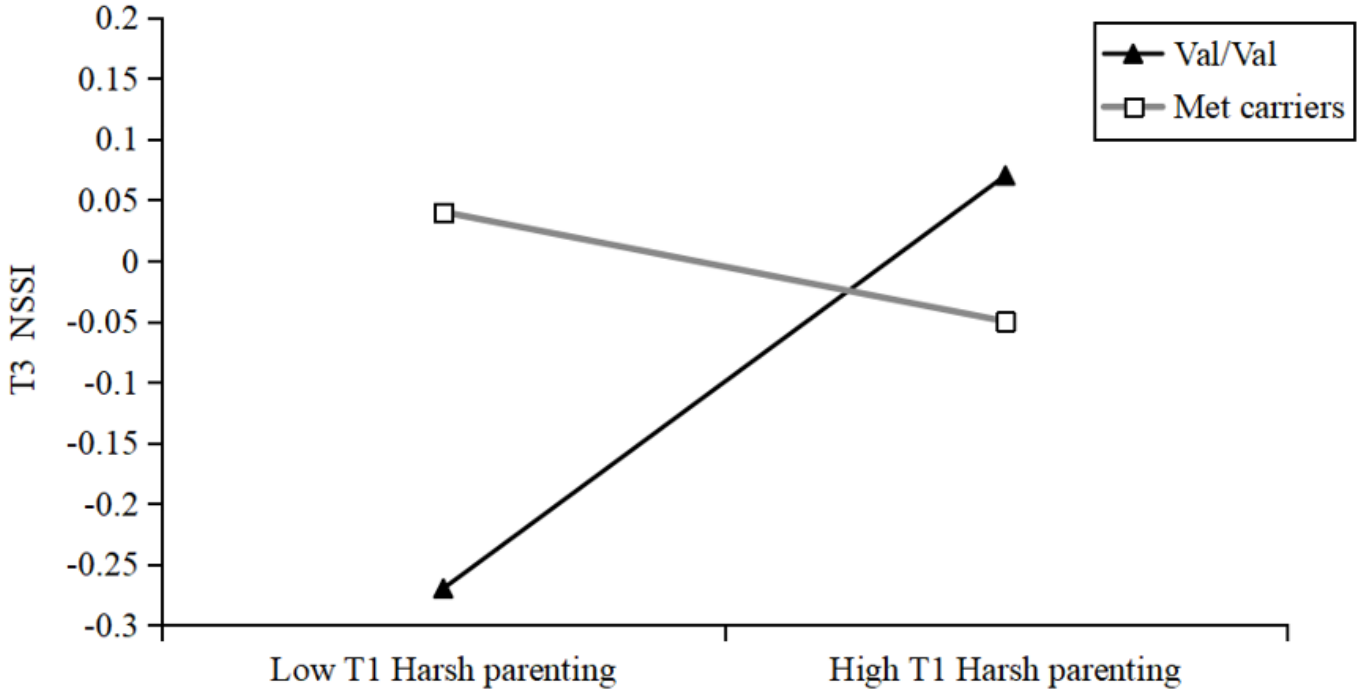

Figure 3

Illustrating the moderating effect of COMT genotype on levels of T1 harsh parenting for ratings of T3 NSSI.

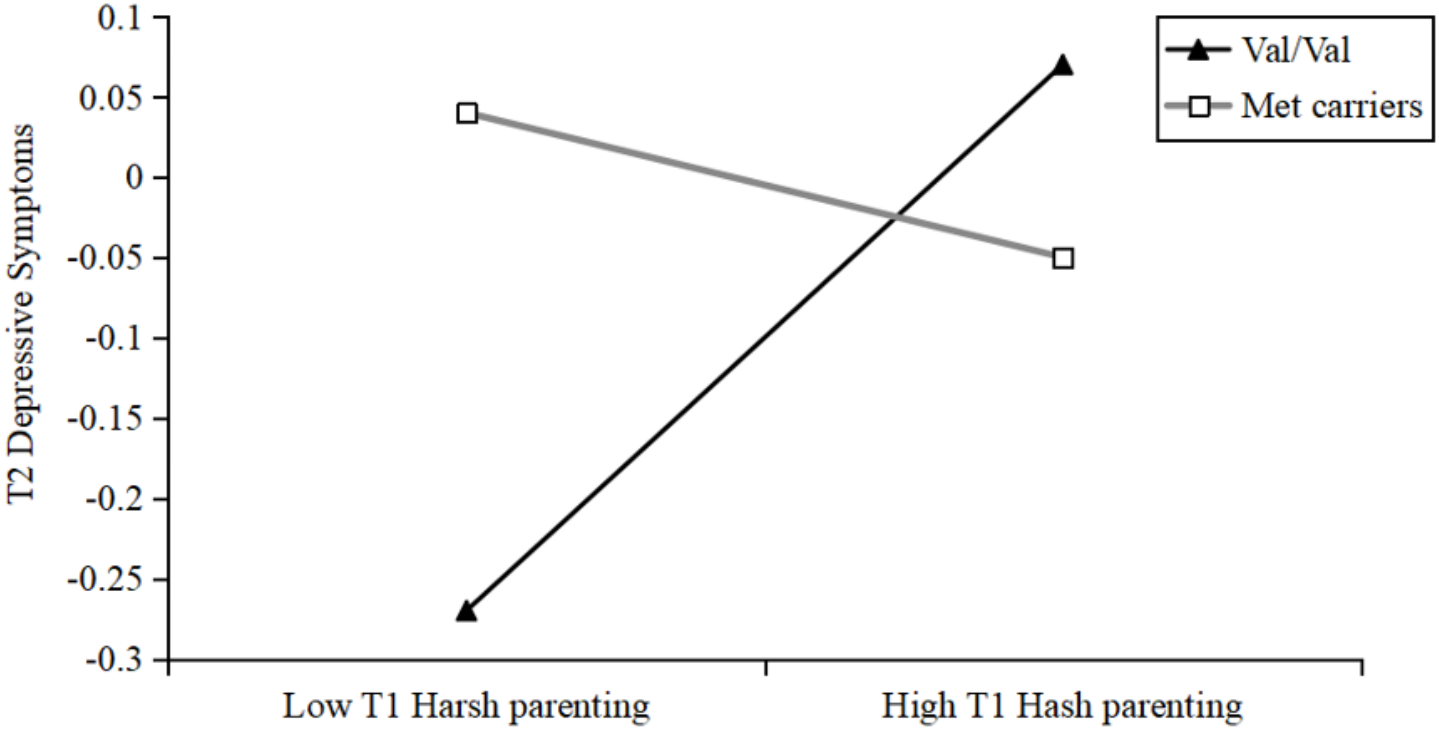

Figure 4

Illustrating the moderating effect of COMT genotype on levels of T1 harsh parenting for ratings of T2 depressive symptoms relationships.