Research Square

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Nonlinear Association Between Cumulative Family Risk and Adolescent Aggressive Behavior: Moderation by Serotonergic Genes

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Abstract

Background

Cumulative family adversity is a well-established antecedent of adolescent aggressive behavior; however, it remains unclear whether this relationship follows a linear or nonlinear pattern and whether it varies across individuals. This study therefore aimed to examine how cumulative family adversity increased the risk of aggressive behavior and whether this association was moderated by serotonergic genes.

Methods

Participants were 1,088 adolescents followed from age 11 to 14 (M_{age} = 12.56 ± 0.34 years old at Time 1, 50.1% girls). Mother-reported family socioeconomic status, mother-reported negative parenting, adolescent-reported parent-child conflict, and teacher-reported adolescent aggressive behavior were assessed at Time 1 to Time 4, respectively. Adolescents' saliva samples were collected at Time 2. Hierarchical regression analyses were conducted to examine the relationship between cumulative family risk and adolescent aggressive behavior, as well as the moderation of serotonergic genes.

Results

The association between cumulative family risk and aggressive behavior showed a nonlinear, J-shaped pattern. Specifically, adolescents exhibited relatively low aggressive behavior at low levels of adversity, but their aggressive behavior escalated sharply once cumulative risk surpassed a critical threshold. Furthermore, the nonlinear relationship was observed only among adolescents carrying a greater number of low-serotonergic alleles, whereas no linear or nonlinear associations were found in their higher-serotonergic counterparts.

Conclusions

These findings indicate that adolescent aggressive behavior is shaped by genotype-specific, threshold-dependent stress responses. They also support prevention and intervention strategies that integrate both cumulative environmental risk and individual genetic vulnerability to improve effectiveness in reducing adolescent aggressive behavior.

Background

Aggressive behavior is the most common form of maladjustment among adolescents [1], which exerts long-term detrimental effects on both individual developmental outcomes and societal well-being [2, 3]. Cumulative family adversity, stemming from the co-occurrence or chronicity of multiple risk factors, has been consistently identified as a key antecedent of aggressive behavior [4, 5, 6]. Nevertheless, inconsistencies exist regarding the pattern of this relationship: some studies have revealed a linear dose-response association [7, 8], whereas others have indicated a nonlinear pattern, wherein the risk of aggressive behavior escalates or plateaus beyond a certain threshold of adversity [9].

One potential mechanism underlying these discrepancies is that adolescents may differ in their response patterns to risk exposure as a function of their genetic predispositions [10]. In particular, serotonergic genes have been repeatedly implicated as sources of individual variability in environmental sensitivity [11]. However, it remains unclear whether cumulative risk shapes adolescent aggressive behavior in linear or nonlinear patterns and whether this association varies depending on genetic susceptibility. Therefore, this study aimed to advance our understanding of how cumulative family adversity increases risk of aggressive behavior and whether this association is moderated by serotonergic genes.

Cumulative Family Risk and Aggressive Behavior

Family constitutes one of the most intimate microsystems in shaping youth development [11, 12, 13]. Exposure to adverse family environments, including poverty, negative parenting, and parent-child conflict, may promote risk-taking behaviors as adaptive strategies to enhance fitness [14], while simultaneously undermining emotion regulation and reinforcing hostile attributional biases [15, 16]; both pathways are theorized to ultimately converge on increased aggressive behavior.

Notably, family risk factors do not occur in isolation; adolescents exposed to one form of adversity are often confronted with multiple, co-occurring challenges [17, 18]. The cumulative risk hypothesis proposes that the sheer number of risk factors is a more robust predictor of adverse child outcomes than any single risk considered individually [19]. Despite accumulating evidence linking cumulative risk to maladaptive outcomes, there remains no consensus on whether the relationship follows a linear or a nonlinear pattern. Some studies have identified a linear dose-response association, such that every additional risk experienced is associated with an incrementally higher probability of maladaptive outcomes [8, 20]. In contrast, more recent research has suggested a nonlinear effect, typically manifesting in two complementary ways: the steeling (or threshold) effect [21] and the saturation effect [22].

The steeling effect suggests that low-level family adversity may be relatively benign or even beneficial, as it offers adolescents opportunities to expand their coping repertoire and master stressful experiences [21, 23]. However, cumulative, prolonged stress exposure accrues physiological costs from sustained adaptation, eliciting heightened defensive responses that, in turn, precipitate a sharp increase in aggressive behavior [24]. Alternatively, the saturation effect describes a pattern in which aggressive behavior accelerates up to a certain point but then plateaus—that is, the enhancing effect of family adversity progressively dissipates at high levels of stressor exposure. This pattern may stem from stress-attenuation processes, whereby emotional and physiological reactivity (e.g., HPA-axis responses) increases from minimal to moderate adversity but blunts under chronic, intense exposure, curtailing the deleterious effects of sustained stress-system activation [25]. Notably, these inconsistencies, rather than noise, imply a nuanced adversity–aggression link, possibly driven by biological or environmental vulnerability [10].

The Moderating Role of Serotonergic Genes

The hormesis model of psychosocial stress suggests that interindividual variability in biological or environmental vulnerabilities may modulate the hormetic threshold [10], yielding divergent linear or nonlinear responses to adversity. Consistent with this, recent neurobiological research indicated that developmental change of resting-state functional connectivity (rsFC) in the default mode network (DMN) moderated the association between adversity and internalizing problems. Specifically, declining DMN rsFC levels amplified the nonlinear pattern, whereas increasing connectivity attenuated—thereby flattened—the nonlinear association [10]. The DMN rsFC partly reflects the functioning of prefrontal inhibitory control over limbic circuits, suggesting that top-down regulation of stress-reactive networks constitutes a key biological vulnerability. Given this, the serotonergic genes, which are involved in regulating prefrontal inhibitory control over the amygdala-hypothalamus-periaqueductal gray network [26], may moderate adolescents' response patterns to family adversity.

Given the large number of serotonergic genes, identifying those that serve as candidate indices of environmental sensitivity is a critical task. Three polymorphisms—tryptophan hydroxylase 1 (*TPH1*) rs1800532, serotonin receptor 1A (*5-HTR1A*) rs6295, and serotonin receptor 2A (*5-HTR2A*) rs6311—were selected for two reasons: their well-established involvement in serotonergic function and their reliable interactions with environmental factors. Specifically, *TPH1* encodes tryptophan hydroxylase, the rate-limiting enzyme in serotonin synthesis, whereas *5-HTR1A* and *5-HTR2A* encode receptors regulating serotonergic neurotransmission (see Table 1). Functionally, the A allele of *TPH1*, the G allele of *5-HTR1A*, and the C allele of *5-HTR2A* have been shown to confer reduced serotonergic function [28, 30, 32, 33].

Table 1
The Coding Scheme of SNPs and Their Associations with Serotonergic Function

SNP	Association with Serotonergic Function	Coding
TPH1 rs1800532	Located on chromosome 11p15.3–p14, *TPH1* encodes tryptophan hydroxylase, the rate-limiting enzyme responsible for serotonin synthesis. Tryptophan is converted into 5-hydroxytryptophan, the precursor of serotonin, through the action of tryptophan hydroxylase [27]. Individuals carrying the A allele exhibit lower cerebrospinal fluid concentrations of 5-hydroxyindoleacetic acid (5-HIAA), a major serotonin metabolite, suggesting relatively lower serotonergic function [28].	CC = 2 AC = 1 AA = 0
5-HTR1A rs6295	Located on chromosome 5q12.3, *5-HTR1A* encodes the serotonin 1A receptor. Activation of the *5-HTR1A* autoreceptor inhibits the firing of serotonergic neurons in the raphe nuclei, thereby reducing serotonin synthesis and release [29]. The promoter polymorphism rs6295 (C–1019G) is a functional regulatory variant. The G allele disrupts the binding of the transcription factors DEAF-1 and Hes5, resulting in altered receptor expression and relatively lower serotonergic neurotransmission [30].	CC = 2 CG = 1 GG = 0
5-HTR2A rs6311	Located on chromosome 13q14–q21, *5-HTR2A* encodes the serotonin 2A receptor. Activation of the *5-HTR2A* receptor modulates serotonergic neurotransmission through cortical feedback mechanisms [31]. The promoter polymorphism rs6311 (A–1438G) is located upstream of the core promoter region and influences transcriptional activity. Consistent with previous multilocus serotonergic genetic profile studies, the C (the complementary base of G) allele was coded as the low-function serotonergic allele.	TT = 2 TC = 1 CC = 0

Notably, accumulating research has indicated that serotonergic genes could moderate the association between adversity and adolescent aggressive behavior [11, 34]. For instance, *5-HTTLPR* has been shown to interact with life stress in predicting aggressive behavior, such that short allele carriers exhibited greater increments in aggression following chronic stress relative to long allele homozygotes [35]. However, existing studies have primarily focused on linear moderating effects, while potential nonlinear patterns (e.g., threshold-dependent genetic effects) remain unclear. Furthermore, previous research has largely adopted a single-gene approach, but accumulating evidence suggests that *TPH1*, *5-HTR1A*, and *5-HTR2A* may operate jointly to modulate serotonergic function [36, 37, 38]. Therefore, this study created a multilocus genetic profile score (MGPS) integrating *TPH1*, *5-HTR1A*, and *5-HTR2A* as a genetic indicator to explore its moderating role in response patterns to adversity.

Current Study

Family adversities tend to co-occur and cluster, with cumulative exposure demonstrating robust dose-response associations with youth aggressive behavior. However, gaps remain in understanding whether cumulative family risk shapes adolescent aggressive behavior in linear or nonlinear patterns, and whether this association varies depending on genetic susceptibility. Utilizing a multilocus genetic profile score (MGPS) approach and multi-informant measurement, this study sought to examine the relation between cumulative family risk and aggressive behavior, and whether this association was moderated by serotonergic genes in a community sample of Chinese adolescents. Drawing on theoretical considerations and empirical evidence, we hypothesized that there would be a nonlinear relationship between cumulative risk and aggressive behavior, and that

serotonergic genes would moderate this association. Nevertheless, no specific hypothesis regarding the gene–environment interaction pattern was proposed due to insufficient empirical evidence.

Methods

Participants

Data for the present study were derived from a longitudinal study of Chinese children and adolescents, with data collected annually across four waves from Grade 6 to Grade 9. At Grade 7, one thousand and ninety adolescents consented to provide a saliva sample for genotyping (1,088 valid readings). Among these adolescents, 545 were girls (50.1%). At Grade 6 (Wave 1), participants ranged in age from 11 to 14 years (M = 12.56, SD = 0.34). Nearly all participants (96.9%) were of Han Chinese ethnicity, the majority ethnic group in China. Participants with genetic data did not differ significantly from those who did not provide saliva samples in socioeconomic status (SES; t = -0.66, p = .51) or stressful life events (t = -0.21, p = .83), but they exhibited lower levels of aggressive behavior (Wave 1: t = 7.59, p < .001; Wave2: t = 6.06, p < .001; Wave 3: t = 3.57, p < .001; Wave4: t = 5.58, p < .001) and negative parenting (t = 2.63, p < .01), as well as higher levels of parent–adolescent conflict (t = -2.56, p < .05).

Design and Procedure

This study was approved by the local ethics committee (SDNU2024008). Informed assent from adolescents themselves and consent from their parents and school principals were obtained prior to data collection. Questionnaires for adolescents were group-administered by trained research assistants in students' classrooms during regular school hours. Meanwhile, the participating headteachers in each school were gathered together and administered a set of measures of behaviors of the students in their classes. The headteachers completed questionnaires assessing the adolescents' aggressive behavior from Wave 1 to Wave 4. The adolescents' parents were given an envelope containing questionnaires about their family socioeconomic status at Wave 1 and negative parenting at Wave 2, and the adolescents returned the envelope to their headteachers the following day. At Wave 3 (Grade 8), adolescents were asked to report their parent–adolescent conflict, stressful life events. Specifically, saliva samples were collected in the classrooms at Wave 2. Adolescents were given detailed instructions and a demonstration of the saliva collection procedure by the trained research assistants (not teachers or staff of their schools). Saliva samples were checked one by one on location, and new collection occurred for failed samples. After data collection, each adolescent involved acquired a gift (about $1).

Measures

Cumulative Family Risk

The present study constructed a cumulative family risk score comprising three indicators of family adversity: negative parenting, parent–adolescent conflict, and low SES. Consistent with prior studies [19, 39], each indicator was dichotomized. Participants scoring in the highest quartile on negative parenting and parent–adolescent conflict, or in the lowest quartile on SES, were coded as 1 (at risk), whereas all others were coded as 0 (no risk). The scores of all risk factors were summed to create a cumulative risk index as an indicator of the individual's exposure to family adversities, with values ranging from 0 to 3.

Negative parenting. Mothers were asked to report their negative parenting practices regarding *rejection* (4 items; e.g., "I yell at my child when I am angry") and *punishment* (7 items; e.g., "I believe physical punishment to be the best way of disciplining") using the Chinese version of Child Rearing Practices Report [40]. Each item was answered on a 5-point Likert scale ranging from 0 (not all at true) to 4 (almost always true). These negative dimensions were averaged to calculate *negative parenting.* The Cronbach's alpha was .70.

Parent-adolescent conflict. Adolescents' perceptions of conflict intensity with their parents were measured by the Issues Checklist [41]. Adolescents indicated whether the 16 specific topics (e.g., chores, cursing) were discussed or not with their parents within the past 2 weeks (using a binary scale, yes or no). Then, for each endorsed topic of discussion, adolescents reported the conflict intensity of the discussion of each topic, using a 5-point scale that varied from 1 (very calm) to 5 (very angry). To be consistent with previous research [42], conflict intensity was calculated by averaging adolescents' ratings on items rated 2 or greater. The Cronbach's alpha was .86.

Family socioeconomic status. SES was indicated by five indexes: household income per month, mother's and father's educational levels, and occupational prestige [43]. Educational level was coded as 1 = *junior high school or lower*, 2 = *senior high school graduate*, and 3 = *some college or more*, whereas occupational prestige was coded as 1 = *peasant or unemployed*, 2 = *blue-collar*, and 3 = *professional or semiprofessional*. Monthly income was divided into three grades with 1 = *less than ¥3000*, 2 = *between ¥3000 and ¥6000*, and 3 = *more than ¥6000*. Each indicator was standardized and then averaged to calculate a z-score with higher scores indicating higher SES. In our sample, parents' educational backgrounds ranged from primary school (1.2% for mothers and 0.5% for fathers) to a college or university degree (61.9% for mothers and 71.1% for fathers). Most of the parents worked in occupational status ranged from peasants or unemployed (6.8% for mothers and 3.5% for fathers), blue-collar (22.0% of the mothers and 23.7% of the fathers) to professional or semi-professional occupations (65.9% of the mothers and 71.5% of the fathers).

Aggressive Behavior

Aggressive behavior was assessed using a subscale of 15 items (e.g., “cruelty, bullying, or meanness to others”) from the Child Behavior Checklist-Teacher Report Form (CBCL-TRF) [44]. Teachers rated each adolescent’s aggressive behavior on a 3-point Likert scale ranging from 0 (not true) to 2 (very true or often true). Aggressive behavior was calculated by averaging the scores across the items, with higher scores indicating higher levels of aggressive behavior. The Cronbach’s alphas were .84, .84, .85, and .85 across the four waves.

Stressful Life Events (SLE)

The Adolescent Self-Rating Life Events Checklist (ASLEC) was used to assess stressful life events experienced by adolescents during the past six months [45]. To correspond to the three risk indicators examined in the present study, three equivalent SLE indicators were selected for replication validation: family with financial hardship, frequent quarrels with parents, and parental unemployment. Each event was coded as 0 (did not happen) or 1 (happened). The three indicators were summed to create a cumulative family-related stressful life event score ranging from 0 to 3, with higher scores indicating exposure to a greater number of family-related stressful life events. The Cronbach’s alpha was .88.

Genotyping

Genomic DNA was extracted from participants’ saliva samples. DNA extraction and genotyping of the three SNPs were performed using the MALDI-TOF in the MassARRAY system (Sequenom Inc., San Diego, California, USA) according to the manufacturer’s instructions. The amplification primer sequences for the three polymorphisms are shown in Table 2. Genotype calling was performed with MassARRAY RT software 3.0.0.4 and analyzed using the MassARRAY Typer software 3.4. Genotype data were unavailable for two participants owing to genotyping failure. One-quarter of the samples were randomly selected for duplicate genotyping to check for errors. The error rate was lower than 1%. The distributions of the three genotypes were consistent with Hardy-Weinberg equilibrium. Minor allele frequencies in our sample were above 5%. No significant associations were found among the three genotypes (χ^2s ≤ 2.93, *df* = 4, *p*s ≥ .59).

Table 2
Genotype Distributions and Hardy-Weinberg Equilibrium Tests

SNP	Amplification sequence primer	Minor allele frequency	Hardy-Weinberg equilibrium		Coding		
			χ^2	*p*	0	1	2
TPH1 rs1800532	Forward CGTCCTGTGGCTGGTTACTT Reverse ACGCTGCAGTGCTTAACAT	46.5%	0.28	0.60	AA = 231	AC = 550	CC = 307
5-HTR1A rs6295	Forward ACGTTGGATGTTGTTTTCTTCATTGGGCCG Reverse ACGTTGGATGGCTTGACATCATTGGCTGAC	26.3%	3.26	0.07	GG = 87	CG = 399	CC = 602
5-HTR2A rs6311	Forward AGAGAAACTGGAGAAATGGC Reverse CCACTCTGGACACAAACACT	47.7%	0.85	0.36	CC = 290	CT = 558	TT = 240

Based on past research [46, 47], we considered alleles associated with lower serotonergic functioning to confer sensitivity. We fitted three regression models to test whether linear or nonlinear (e.g., dominant/recessive) coding of candidate genes and summing of the genes was most appropriate for creating MGPS [47]. The results indicated that the disaggregated model fit the data well, and the change in R^2 was small and nonsignificant, supporting the use of a linear, additive polygenic index across the three genetic markers. Accordingly, each candidate gene included in the MGPS was scored 0, 1, or 2 for the number of alleles associated with relatively higher serotonergic functioning. Lower MGPS reflected a greater number of low-serotonergic alleles. The distribution of the scores was as follows: 0 (*N* = 2), 1 (*N* = 38), 2 (*N* = 151), 3 (*N* = 311), 4 (*N* = 339), 5 (*N* = 206), and 6 (*N* = 41). Due to the small number of participants scoring 0, they were combined with those scoring 1 for subsequent analysis [48].

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 associations between cumulative family risk and aggressive behavior were examined using hierarchical regression models: a linear model and a nonlinear model including a quadratic term to test for accelerating or diminishing effects. Third, the moderating role of the serotonergic MGPS was tested. Simple slope analyses were performed to examine the patterns of significant interactions between MGPS and the linear and quadratic terms. Fourth, replication analysis was conducted using stressful life events in family context. Gender, ethnicity and the mean level of aggressive behavior across Wave 1-Wave 3 were included as covariates in all models. Continuous independent variables were standardized before analysis to reduce multicollinearity.

Results

Descriptive Statistics

Means, standard deviations, and correlations among main variables were presented in Table 3. There were no significant gene-environment correlations between the MGPS and cumulative family risk, indicating the absence of correlation between gene and environment. Cumulative family risk was positively correlated with aggressive behavior. The correlation between MGPS and aggressive behavior was not significant. The *t*-test analyses revealed that cumulative family risk (t = -0.04, p = .97) and MGPS (t = -0.37, p = .71) did not differ by gender, while boys exhibited higher levels of aggressive behavior than girls (Wave 1: t = 8.17, p < .001; Wave 2: t = 8.28, p < .001; Wave 3: t = 5.13, p < .001; Wave 4: t = 7.70, p < .001).

Table 3
Means, Standard Deviations, and Correlations Among Study Variables

Variable	**1**	**2**	**3**	**4**	**5**	**6**	**7**
1 MGPS	1						
2 Cumulative Family Risk	0.04	1					
3 Cumulative Stressful Life Events	-0.03	0.30***	1				
4 Aggressive Behavior (Wave 1)	0.04	0.03	0.02	1			
5 Aggressive Behavior (Wave 2)	0.02	0.04	-0.01	0.28***	1		
6 Aggressive Behavior (Wave 3)	0.04	0.07*	0.06	0.35***	0.60***	1	
7 Aggressive Behavior (Wave 4)	-0.01	0.11***	0.09**	0.31***	0.57***	0.63***	1
M	3.59	0.76	0.50	0.11	0.11	0.12	0.11
SD	1.16	0.79	0.66	0.19	0.18	0.20	0.19
Note. * p < .05, ** p < .01, *** p < .001.							

Linear and Nonlinear Effects of Cumulative Family Risk on Aggressive Behavior

To examine how cumulative family risk confers risk for aggressive behavior, a linear model and a nonlinear model were tested. As shown in Table 4, the linear term was significant in the linear model. After the quadratic term was added, the quadratic term was significant. Moreover, the nonlinear model explained more variance in aggressive behavior than the linear model. Specifically, the significant positive quadratic effect indicated a J-shaped association between cumulative family risk and adolescent aggressive behavior, with aggressive behavior increasing at an accelerating rate as cumulative family risk increased (see Fig. 1). Based on prior research, we also compared the predictive capacity of a single risk factor with that of cumulative family risk [49]. First, each family risk factor was entered separately into the regression model (negative parenting: β = 0.04, t = 1.47, p = .14; parent-adolescent conflict: β = 0.06, t = 2.79, p < .005; SES: β = 0.02, t = 0.70, p = .49). Then, each risk factor was entered together with a cumulative risk index that excluded this particular factor (negative parenting: β = 0.04, t = 1.48, p = .14; parent-adolescent conflict: β = 0.07, t = 2.80, p < .005; SES: β = 0.02, t = 0.65, p = .52). By comparing changes in the regression coefficient of each risk factor across the models, we were able to determine the extent to which its association with aggressive behavior was attributable to the risk factor itself versus to other co-occurring risk factors. After controlling for the total number of other risk factors present, only parent–adolescent conflict remained a significant individual predictor; however, none of the individual risk factors had a stronger predictive effect on aggressive behavior than cumulative family risk.

Table 4
The Moderating Effect of MGPS in the Relationship Between Cumulative Risk and Adolescents' Aggressive Behavior

			Step 1		Step 2		Step 3		Step 4		Step 5	
	R^2	ΔR^2	β	*t*	β	*t*	β	*t*	β	*t*	β	*t*
Gender	0.38	0.38***	-0.08	-2.94**	-0.08	-3.01**	-0.08	-3.06**	-0.08	-3.05**	-0.08	-2.98**
Ethnicity			-0.03	-1.28	-0.03	-1.28	-0.03	-1.25	-0.03	-1.28	-0.04	-1.53
Agg			0.59	22.53***	0.59	22.41***	0.59	22.38***	0.59	22.49***	0.58	22.26***
CR	0.39	0.01**			0.08	2.98**	0.04	1.19	0.04	1.14	0.04	1.17
CR^2	0.39	0.004*					0.08	2.53*	0.09	2.61**	0.09	2.95**
MGPS	0.39	0.003*							-0.05	-2.02*	-0.003	-0.08
CR×MGPS	0.40	0.005*									-0.01	-0.32
CR^2×MGPS											-0.08	-2.18*

Note. Agg = mean aggressive behavior across Wave 1–Wave 3, CR = cumulative risk, CR^2 = the quadratic term of cumulative risk, MGPS = multilocus genetic profile score.

* $p < .05$, ** $p < .01$, *** $p < .001$.

The Moderating Role of MGPS

Results of the moderation analysis are presented in Table 4. The main effects of the quadratic term of cumulative family risk and the MGPS were significant. The interaction between MGPS and the quadratic, rather than the linear, term of cumulative family risk was significant. Simple-slope analyses (see Fig. 1) showed that, among adolescents with lower MGPS, the quadratic term of cumulative family risk significantly positively predicted aggressive behavior ($b = 0.02$, $t = 4.58$, $p < .001$), indicating a J-shaped association in which aggressive behavior increased at an accelerating rate as cumulative family risk increased. Among adolescents with higher MGPS, the quadratic term of cumulative risk was not significant ($b = 0.001$, $t = 0.14$, $p = .89$).

Sensitivity Analyses

To test the robustness of these findings, an alternative cumulative stressful life events score was derived from the corresponding ASLEC items (i.e., family with financial hardship, frequent quarrels with parents, and parental unemployment). As shown in Table 5, the linear effect of cumulative stressful life events was significant. Compared with the linear term, the quadratic term of cumulative stressful life events explained significantly more variance in aggressive behavior. Moreover, the interaction between the quadratic term of cumulative stressful life events and MGPS was significant. Simple-slope analyses (see Fig. 1) also showed a significant positive effect between the quadratic term of cumulative stressful life events and aggressive behavior among adolescents with lower MGPS ($b = 0.02$, $t = 4.78$, $p < .001$), but not among adolescents with higher MGPS ($b = -0.003$, $t = -1.08$, $p = .28$).

Table 5
The Moderating Effect of MGPS in the Relationship Between Cumulative Stressful Life Events and Adolescents' Aggressive Behavior

			Step 1		Step 2		Step 3		Step 4		Step 5	
	R^2	ΔR^2	β	*t*	β	*t*	β	*t*	β	*t*	β	*t*
Gender	0.34	0.34^{***}	-0.09	-3.20^{**}	-0.09	-3.39^{**}	-0.09	-3.30^{**}	-0.09	-3.30^{**}	-0.09	-3.25^{**}
Ethnicity			-0.05	-1.78	-0.05	-1.87	-0.05	-1.83	-0.05	-1.86	-0.06	-2.21^{*}
Agg			0.56	20.93^{***}	0.56	21.05^{***}	0.56	21.10^{***}	0.56	21.11^{***}	0.56	21.36^{***}
SLE	0.36	0.01^{***}			0.10	3.72^{***}	0.04	1.22	0.04	1.18	0.04	1.20
SLE^2	0.36	0.003^{*}					0.08	2.17^{*}	0.08	2.18^{*}	0.08	2.20^{*}
MGPS	0.36	0.001							-0.02	-0.94	0.05	1.47
SLE×MGPS	0.37	0.01^{***}									0.04	1.24
SLE^2×MGPS											-0.16	-3.97^{***}

Note. Agg = mean aggressive behavior across Wave 1–Wave 3, SLE = stressful life events, SLE^2 = the quadratic term of stressful life events, MGPS = multilocus genetic profile score.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Discussion

Cumulative family adversity shows robust dose-response associations with youth mental health, especially aggressive behavior [6]. However, a gap remains in understanding whether cumulative risk influences adolescent aggressive behavior in linear or nonlinear patterns, and whether this association varies across individuals. To address these gaps, this study examined the relationship between cumulative family risk and aggressive behavior, as moderated by serotonergic genes, in a community sample of Chinese adolescents. Overall, this study found a nonlinear (J-shaped) association between cumulative family risk and aggressive behavior: adolescents exhibited relatively low levels of aggression under low adversity, but their aggressive behavior escalated sharply once cumulative risk surpassed a critical threshold. Furthermore, the nonlinear relationship emerged only among adolescents with more low-serotonergic alleles, whereas no significant linear or nonlinear associations were found in their higher-serotonergic counterparts.

Consistent with prior studies [8, 9], the accumulation of multiple family adversities is associated with an elevated risk of aggressive behavior. Notably, we observed a J-shaped association between cumulative family adversity and aggressive behavior, supporting the "steeling" (or threshold) effect, whereby the risk of aggressive behavior increased sharply once individuals cross a critical risk threshold. These findings may be interpreted from two perspectives. First, informed by the steeling-effect models, exposure to low-level family adversity provides adolescents with opportunities to develop, enact, and expand their repertoire of coping strategies and master stressful experiences [21, 23]. Consequently, low-level family-adversity exposure can be relatively benign and even confer developmental benefits: adolescents acquire adaptive capacities to navigate adverse experiences rather than engaging in aggressive behavior. Second, as posited by sensitization models, cumulative, prolonged exposure to adversity can elicit heightened defensive responses (e.g., flight-oriented reactions) in the face of threatening stimuli. Accordingly, when accumulated stress reaches moderate-to-high levels, it may overwhelm youths' regulatory capacities, which in turn drives the disproportionate increase in aggressive behavior observed in the present study.

Furthermore, allostatic-load theory [50] suggests that the J-shaped association reflects a shift from adaptive functioning under mild adversity to accumulating physiological costs beyond a critical risk threshold. Although allostatic responses are adaptive in the short-term, their repeated or prolonged activation can impose substantial long-term physiological costs [24]. Specifically, under low-to-moderate levels of stressor exposure, the load associated with ongoing allostatic accommodation is likely negligible. In contrast, under chronic or recurrent stress, the costs of sustaining allostatic accommodation may outweigh its adaptive benefits [24, 51], resulting in cumulative physiological strain that ultimately increases adolescents' propensity for aggressive responses [52].

However, the nonlinear pattern observed in the present study diverges from prior research supporting a risk-saturation model, whereby the association between adversity and adjustment outcomes attenuates progressively at higher levels of adversity [22]. In light of the hormesis model, stress and maladjustment outcomes are hypothesized to follow a cubic relationship: low-to-moderate stress is associated with fewer problems, while risk-saturation effects manifest at extreme stress levels [10]. The lower cumulative risk levels observed in the community sample may also have limited our ability to reach the sufficiently high adversity thresholds at which saturation effects would emerge.

Notably, the J-shaped association varied across individuals with different serotonergic genotypes, such that individuals carrying a greater number of low-serotonergic alleles may be more vulnerable to aggressive behavior when exposed to cumulative adversity. These findings suggest that interindividual differences in the magnitude of nonlinear effects of cumulative risk may arise from variability in biological vulnerabilities. This interaction pattern is consistent with the late-maturing prefrontal cortex (PFC) theory. According to this theory, the late-maturing PFC may provide insufficient top-down control over adversity-activated amygdala-related networks, rendering adolescents particularly vulnerable to stress [53]. Further, lower serotonergic activity has been shown to weaken prefrontal inhibitory control over the amygdala-hypothalamus-periaqueductal gray network [26]. Consequently, a greater number of low-serotonergic alleles may increase adolescents' vulnerability to cumulative family risk by reducing the PFC's capacity to regulate adversity-related activity in amygdala networks.

Moreover, adolescents with higher MGPS exhibited no linear or nonlinear association between cumulative risk and aggressive behavior. This finding suggests that enhanced serotonergic functioning may serve as a protective factor, mitigating the effects of cumulative adversities. Such a buffering effect protects individuals against the detrimental consequences of cumulative risk by strengthening hypothalamic-pituitary-adrenal (HPA) axis functioning. Specifically, pharmacological activation of the serotonergic system via acute administration of a selective serotonin reuptake inhibitor [54] or serotonin agonist [55] elevates plasma adrenocorticotropic hormone and corticosterone levels. These changes may, in turn, modulate the HPA stress axis response and/or feedback mechanisms that are essential for maintaining homeostasis in the context of repeated stress exposure [56].

Although some prior work has shown divergent cumulative-risk patterns (i.e., linear versus nonlinear associations) across varying biological and environmental vulnerabilities [10, 57]—for example, cumulative risk shows linear links to delinquency for boys and nonlinear links for girls [58]—the present study yielded no evidence for such vulnerability-associated heterogeneity. An alternative explanation for the absence of linear or nonlinear effects among higher-MGPS adolescents is that cumulative-risk severity may be insufficient to reach the threshold required to observe damaging effects in this community sample. As a community cohort, our sample exhibited relatively low family adversity, and few participants experienced extremely severe cumulative risk. From a hormesis perspective, less-vulnerable individuals are presumed to exhibit higher adversity-response thresholds, such that linear and nonlinear associations become evident only under extremely high levels of adversity [59]. That is, the buffering effect of high serotonergic polygenic propensity remains largely unchallenged within this low-to-moderate adversity range. However, higher-MGPS adolescents may also show heightened aggressive behavior under severe, chronic family adversity once stress surpasses their protective threshold. Future studies incorporating a wider range of cumulative adversities are required to examine this threshold-based interpretation.

The present findings have several implications. First, the impact of cumulative adversities was stronger than that of isolated single risks, underscoring the need for multidimensional cumulative-risk assessment in future work to better understand the etiology of adolescent aggressive behavior. Second, the J-shaped threshold pattern observed in this study challenges the oversimplified assumption that adversity increases psychopathological risk in a uniform manner. Consistent with hormesis and stress-steeling theories, low-to-moderate levels of family adversity may be beneficial and, in some contexts, promote resilience. In addition, this study extends prior models by showing that the form and magnitude of responses to adversity vary across individuals, reflecting differences in genetic and biological vulnerability. Practically, these findings inform individualized, resilience-oriented prevention and intervention strategies for youth. For adolescents exposed to low-to-moderate levels of family adversity, interventions should strengthen coping capacity and adaptive functioning to foster resilience. In contrast, for youth with biological vulnerability and elevated cumulative adversity, targeted interventions are needed to mitigate exacerbated behavioral risks. Integrating cumulative risk evaluation with individual susceptibility differences into program design can improve both the precision and overall effectiveness of adolescent mental health interventions.

Despite these meaningful implications, several limitations should be considered. First, this study included only a limited set of family risk indicators within the family context, which constrains a comprehensive test of the hormesis model [60]. Because adolescent development unfolds across multiple interconnected ecological contexts, future research should determine whether the nonlinear cumulative risk pattern observed here generalizes to other environmental domains. Second, consistent with a common methodological limitation in cumulative risk research, the present study focused primarily on the quantity of adverse family exposures. However, not all adverse environmental types, severities, or developmental timings may elicit the observed nonlinear effects [21]. Accordingly, future research should move beyond cumulative scores by incorporating dimensions of severity, chronicity, and developmental timing to more precisely elucidate how cumulative adversity confers risk for aggressive behavior. Third, although a substantial body of research indicates that susceptibility to environmental stress varies across development [10, 61], far less is known about whether the cumulative risk pattern also changes over different developmental periods. Future studies should explore whether gene–environment threshold effects vary across developmental stages.

Conclusions

Prior research on cumulative environmental risk has largely assumed linear dose–response effects on adolescent aggressive behavior. This study extends the literature by examining whether cumulative family risk relates to aggressive behavior in a linear versus nonlinear manner, and whether this association is moderated by serotonergic genes. Overall, results revealed a J-shaped relationship between cumulative family risk and aggressive behavior. Moreover, the magnitude of these nonlinear effects varied as a function of serotonergic genes susceptibility. Adolescents with lower MGPS showed a pronounced J-shaped association, whereas no significant linear or nonlinear relationships were observed among adolescents with higher MGPS. Interpreting these findings within the hormesis model and the late-maturing PFC framework, lower serotonergic genotypes may weaken prefrontal inhibitory control over the amygdala-related network, thereby conferring greater vulnerability to stress. Together, these findings suggest that adolescent aggressive behavior is shaped by genotype-specific, threshold-dependent stress responses. They also support prevention and intervention

strategies that integrate both cumulative environmental risk and individual genetic vulnerability to improve effectiveness in reducing adolescent aggressive behavior.

Abbreviations

5-HTR1A
Serotonin receptor 1A
5-HTR2A
Serotonin receptor 2A
ASLEC
The Adolescent Self-Rating Life Events Checklist
CBCL-TRF
Child Behavior Checklist-Teacher Report Form
DMN
Default mode network
DNA
Deoxyribonucleic acid
HPA
Hypothalamic-pituitary-adrenal
MGPS
Multilocus genetic profile score
PFC
Prefrontal cortex
rsFC
Resting-state functional connectivity
SES
Socioeconomic status
SLE
Stressful Life Events
SNP
Single nucleotide polymorphism
TPH1
Tryptophan hydroxylase 1

Declarations

Ethics approval and consent to participate

This study was approved by Shandong Normal University Ethical Committee (Reference Number: SDNU2024008). All procedures performed in the study were in accordance with the ethical standards of the 1964 Helsinki Declaration and its later amendments. Informed consent was obtained from all participants and their parents and school principals.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Authors' information

Author Notes

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Author Contribution

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

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Data Availability

The datasets generated and/or analyzed during the current study are not publicly available due national policy restrictions governing the sharing of genetic information but are available from the corresponding author on reasonable request.

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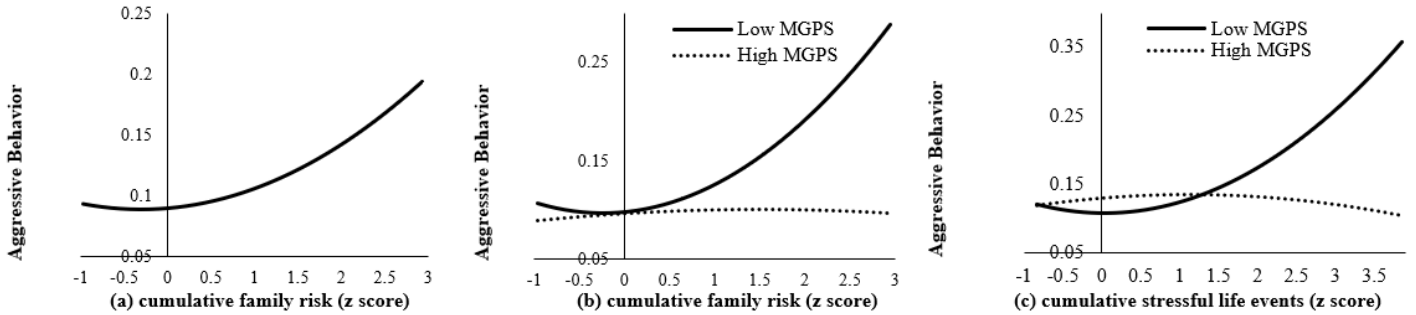
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Figures

The Nonlinear Association Between Cumulative Family Risk and Adolescents' Aggressive Behavior



Note. Panel (a) shows the main effect of cumulative family risk. Panel (b) shows the interaction between MGPS and cumulative family risk in predicting adolescent aggressive behavior; panel (c) shows the interaction between MGPS and cumulative stressful life events in predicting adolescent aggressive behavior.

Figure 1

See image above for figure legend