Van Med J 33 (3):357 -365, 2026
DOI: 10.5505/vmj.2026.57442

Anhedonia and Somatic Symptoms in Adolescents: Beyond Depression and Anxiety

Oğuz Bilal Karakuş, İkranur Sezer, Kübra Kalaycı , Hayriye Rumeysa Erdem , Ayşe Tuğçe Uysal, Merve Menemencioğlu, Onur Burak Dursun

Department of Child and Adolescent Psychiatry, Trabzon Kanuni Training and Research Hospital, Trabzon, Türkiye

Abstract

Introduction: Anhedonia is increasingly recognized as a transdiagnostic construct associated with multiple dimensions of psychopathology. However, its relationship with somatic symptom burden in adolescents, independent of core internalizing symptoms such as depression and anxiety, remains unclear. This study aimed to examine the association between anhedonia and somatic symptoms in adolescents and to determine whether this relationship persists beyond depressive and anxiety symptom severity.

Materials and Methods: A total of 242 adolescents aged 12–18 years referred to a child and adolescent psychiatry outpatient clinic were included. Psychiatric diagnoses were established using a semi-structured clinical interview. Depression severity was assessed with a clinician-rated instrument, while anhedonia, anxiety symptoms, and somatic symptoms were evaluated using validated self-report measures. Hierarchical regression analyses were conducted in three steps: Model I included age and sex; Model II added depression and anxiety; and Model III included anhedonia.

Results: Clinically significant anhedonia was identified in 27.7% of participants. Adolescents with anhedonia exhibited higher somatic symptom severity ($p<0.001$). Somatic symptom scores were positively correlated with depression and anxiety severity and negatively correlated with hedonic capacity ($p<0.001$). In hierarchical regression analyses, female sex ($\beta=0.295$), depression ($\beta=0.410$), and anxiety ($\beta=0.370$) were significant predictors of somatic symptoms. In the final model, anhedonia remained an independent predictor ($\beta=-0.184$, $p=0.003$), explaining additional variance beyond core internalizing symptoms.

Conclusion: Anhedonia is independently associated with somatic symptom burden in adolescents. These findings suggest that somatic symptoms should be conceptualized not only as manifestations of internalizing psychopathology but also in relation to dysfunctions in reward–motivation systems and interoceptive processing.

Key words: Anhedonia; medically unexplained symptoms; adolescent; depression; anxiety.

Introduction

Somatic symptoms are multidimensional clinical phenomena encompassing bodily experiences such as pain, fatigue, gastrointestinal symptoms, and various autonomic manifestations (1). These symptoms are considered not only indicators of organic diseases but also developmentally shaped experiences emerging from the interaction of biological predispositions, psychological processes, and environmental factors (2). Although a substantial proportion of somatic symptoms during childhood and adolescence are transient and self-limiting, in a specific subgroup these symptoms tend to persist and become associated with clinically significant functional impairment (1,3). Epidemiological evidence indicates that somatic symptoms are observed in approximately 25–30% of children and adolescents (4,5). While these symptoms occur at similar rates across sexes during early childhood, they become more prevalent among girls during adolescence (3). A high and persistent somatic symptom burden has been associated with school absenteeism, impaired social functioning, increased healthcare utilization, and reduced quality of life (4,6). Furthermore, longitudinal studies suggest that somatic symptoms may constitute a risk indicator for the later development of depressive and anxiety disorders (6). Although somatic symptoms demonstrate strong associations with internalizing symptoms, particularly depression and anxiety, these relationships may not fully account for the entire spectrum of somatic symptom experiences (5). Accordingly, recent theoretical models propose that interoceptive processes involved in the perception and interpretation of bodily signals may play a central role in somatic symptom experience (7). Interoception refers to the process through which an organism perceives its internal physiological states and interprets these signals within a cognitive–emotional framework (7). Heightened bodily sensitivity or predictive

*__Corresponding Author:__ Oguz Bilal Karakus Department of Child and Adolescent Psychiatry, Trabzon Kanuni Training and Research Hospital, Post code: 61040 Trabzon, Turkey **E-mail:** bilal_k422@hotmail.com **Orcid:** Oğuz Bilal Karakuş 0000-0001-6623-7375, İkranur Sezer 0009-0003-2353-8911, Kübra Kalaycı 0009-0001-9262-3031, Hayriye Rumeysa Erdem 0009-0009-8382-3646, Ayşe Tuğçe Uysal 0009-0008-5805-282X, Merve Menemencioğlu 0009-0009-1881-2123, Onur Burak Dursun 0000-0003-2990-9851

Received: 23.03.2026, Accepted: 16.06.2026

Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

processing errors within this system may lead to the misinterpretation of physiologically benign sensations as indicators of threat or illness. Moreover, alterations in interoceptive processing have been suggested to relate not only to anxiety and depressive symptoms but also to motivational systems (7). Within this context, anhedonia emerges as a noteworthy dimension for understanding motivational processes potentially associated with somatic symptoms. Anhedonia is characterized by diminished capacity to experience pleasure and reduced reward expectancy and is defined as a core psychopathological construct associated with functional alterations in reward–motivation systems (8). Alterations within mesolimbic dopaminergic circuits have been shown to influence not only emotional experience but also pain perception and the regulation of bodily sensations (9). The fact that pain and reward processing share partially overlapping neural substrates, including the anterior cingulate cortex, insula, and striatal networks, suggests a potential biological intersection between anhedonia and somatic symptoms (8). Anhedonia also represents a motivational dimension that is partially dissociable from negative affect. Whereas depressive and anxiety symptoms are typically characterized by increased distress, anhedonia involves reduced reward anticipation and diminished behavioral engagement, potentially exerting specific effects on energy levels, initiation of activity, and the subjective evaluation of bodily experiences (10). This distinction is important for questioning whether somatic symptoms merely reflect manifestations of negative affect. Indeed, both somatic symptoms and anhedonia have been reported as transdiagnostic dimensions observed across various psychiatric conditions (2,10). Adolescence provides a unique developmental context for evaluating this relationship. Neurodevelopmental research indicates that limbic and striatal systems associated with reward sensitivity mature relatively early, whereas prefrontal networks responsible for cognitive control and emotion regulation develop later (11). This developmental asymmetry may increase emotional and motivational reactivity while creating temporary vulnerabilities in the regulation and reinterpretation of bodily signals. Concurrent developmental changes in interoceptive processes and heightened sensitivity to social rewards may further facilitate the expression of psychological distress through bodily sensations (12). The observed increase in somatic symptom burden during adolescence and its strong associations with internalizing symptoms suggest that mechanisms related to motivational and emotional processes may become more clinically salient during this developmental period (5). Nevertheless, a substantial portion of the existing literature has been conducted in adult samples or populations with general medical conditions. Moreover, many studies examining the relationship between anhedonia and somatic symptoms have not adequately disentangled its specific contribution from depressive and anxiety symptoms. This methodological limitation complicates the clarification of the nature of the somatic symptom–anhedonia relationship, particularly in clinically referred adolescent samples. In this context, the present study aims to investigate the relationship between anhedonia levels and somatic symptom burden among adolescents referred to a child and adolescent psychiatry outpatient clinic and to determine whether this association persists independently of core depressive and anxiety symptoms.

Materials and Methods

Study design and participants: The study sample consisted of adolescents aged 12–18 years who were referred to the Child and Adolescent Psychiatry Outpatient Clinic of a training and research hospital between January 5 and March 5, 2026. Participants who met the age criteria and provided written informed consent from themselves and their parents/legal guardians were included in the study. Exclusion criteria comprised the presence of autism spectrum disorder, intellectual disability, psychotic disorders, bipolar disorder (manic episode), cognitive difficulties severe enough to prevent completion of the assessment tools, and any chronic medical illness that could contribute to somatic symptoms (e.g., epilepsy, asthma, diabetes mellitus, migraine). Sample size was calculated using the GPower 3.1.9.7 software. Considering the reference study (13), the effect size for multiple regression analyses was set at f=0.07 (α=0.05, power =95%), indicating that a minimum of 225 participants was required.

Data collection tools: A structured Sociodemographic Data Form developed by the researchers was used to collect information regarding age, sex, educational status, household income, school attendance, and psychiatric diagnoses. Psychiatric diagnoses and the presence of anhedonia were assessed using the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-5). This semi-structured diagnostic interview evaluates psychopathology in

Table 1: Comparison of Sociodemographic and Clinical Variables According to the Presence of Anhedonia (n=242)

Variables		ANH- (n=175), M±SD, n(%)	ANH+ (n=67) , M±SD, n(%)	Statistics	p	Effect Size
Age (years)		15.19 ±1.79	15.81 ± 1.34	t=-2.953	0.004	Cohen's d=0.374
Gender (m:f)		67:108	15:52	X^2=5.466	0.019	Cramer's V=0.150
Family Income Level	Low	23 (13.1)	8 (11.9)	X^2=5.647	0.130	Cramer's V=0.153
	Lower- Middle	54 (30.9)	30 (44.8)			
	Middle -High	62 (35.4)	22 (32.8)			
	High	36 (20.6)	7 (10.4)			
Family Structure (nuclear family)		145 (82.9)	50 (74.6)	X^2=2.097	0.148	Cramer's V=0.093
Psychiatric Diagnoses	MDD	31 (17.8)	55 (82.1)	X^2=87.071	<0.001	Cramer's V=0.601
	Anxiety Disorders	55 (31.6)	15 (22.4)	X^2=1.996	0.158	Cramer's V=0.091
	ADHD	48 (27.6)	12 (17.9)	X^2=2.422	0.120	Cramer's V=0.100
	OCD	17 (9.8)	6 (9.0)	X^2=0.037	0.847	Cramer's V=0.012
Current psychotropic medication use		72 (41.1)	30 (44.8)	X^2=0.262	0.609	Cramer's V=0.033
CSI-Total		18.94 ± 14.82	38.75 ± 17.48	t=-8.842	<0.001	Cohen's d=1.270
DARS-Total		46.75 ± 9.17	25.00 ± 8.45	t=16.867	<0.001	Cohen's d= 2.423
Core Anxiety		21.98 ±11.82	28.03 ± 13.69	t=-3.408	<0.001	Cohen's d=.490
Core Depression		27.05 ± 9.31	49.82 ± 12.61	t=-13.442	<0.001	Cohen's d=2.204

M: Mean, **SD:** Standard Deviation, ANH+ = Presence of Anhedonia; ANH− = Absence of Anhedonia ,m: Male, **f:** Female, **MDD:** Major Depressive Disorder, **ADHD:** Attention Deficit Hyperactivity Disorder, **OCD:** Obsessive Compulsive Disorder, **t:** Independent Sample's t Test, **X^2:** Chi-square

children and adolescents according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria (14). The Turkish version has established validity and reliability (15). For subgroup comparisons, participants were classified as ANH+ if clinically significant anhedonia was identified during the K-SADS-5 interview based on DSM-5 depressive symptom assessments. Participants without clinically significant anhedonia were classified as ANH−. This categorical grouping was used only for descriptive subgroup comparisons, whereas the primary analyses examining associations with somatic symptoms were conducted using continuous anhedonia severity scores. Depressive symptom severity was evaluated using the Children's Depression Rating Scale–Revised (CDRS-R), a clinician-administered instrument consisting of 17 items designed to assess depressive symptoms in children and adolescents (16). The Turkish version has demonstrated validity and reliability in adolescent populations (17). The Cronbach's alpha coefficient for the CDRS-R in the current sample was 0.920. Anhedonia severity was measured with the Dimensional Anhedonia Rating Scale (DARS), a 17-item self-report instrument developed to assess anhedonia (18). Each item is rated on a 5-point Likert scale ranging from 0 to 4, with lower scores indicating greater severity of anhedonia. The Turkish version has recently been validated and shown to be reliable (19). In the present sample, the Cronbach's alpha coefficient for the DARS was 0.915. DARS total scores were used as continuous measures of anhedonia severity. Anxiety symptoms were assessed using the Revised Child Anxiety and Depression Scale (RCADS), a 47-item self-report instrument designed to evaluate anxiety and depression symptoms in children and adolescents based on DSM-IV criteria (20). The Turkish version has established validity and reliability (21). The Cronbach's alpha coefficient for the RCADS in the present sample was 0.894. Somatic symptom severity was evaluated using the Children's Somatization Inventory–24 (CSI-24), a 24-item self-report instrument developed to assess the frequency and severity of somatic symptoms in children and adolescents (22). The Turkish version has demonstrated adequate validity and reliability (23). The Cronbach's alpha coefficient for the CSI-24 in the current sample was 0.903.

Statistical analysis: Prior to analysis, the dataset was examined for missing values and outliers. Distributional properties of continuous variables were assessed using the Kolmogorov–Smirnov test. Independent samples t-tests were used for comparisons of continuous variables between two groups. Categorical variables were compared using chi-square tests. Associations between variables were evaluated using Pearson correlation analysis depending on distributional properties. Hierarchical regression analyses were conducted to examine predictors of somatic symptom severity. In Model I, age and sex were included. In Model II, depression and anxiety severity were added. In Model III, anhedonia was included to evaluate its independent contribution to somatic symptom severity. To minimize the shared item effect, depression severity was calculated after removing the anhedonia and somatic symptom items from the CDRS-R (core depression). Similarly, anxiety severity was computed after excluding depression, obsessive–compulsive, and somatic-related items from the RCADS (core anxiety). The specific items removed for the computation of core depression and core anxiety scores are presented in Supplementary Table 1. All analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 27.0, and the level of statistical significance was set at $p<0.05$.

Ethical approval: Ethical approval for this study was obtained from the Trabzon University Faculty of Medicine Non-Interventional Scientific Research Ethics Committee (Date: January 5, 2026; Approval No: 2026/26047). Written informed consent was obtained from all participants and their legal guardians prior to participation. The study was conducted in accordance with the Declaration of Helsinki.

Results

Data from a total of 242 participants were analyzed. Of these, 160 were female and 82 were male. Most participants were attending school (86.5%, n=205). Clinically significant anhedonia was identified in 27.7% of participants (n=67; ANH+), whereas 72.3% (n=175; ANH–) did not exhibit anhedonia. The most common psychiatric diagnosis was major depressive disorder (MDD) (35.5%), followed by anxiety disorders (28.9%) and attention-deficit/hyperactivity disorder (ADHD) (24.8%). Comparisons according to anhedonia status are presented in Table 1. The ANH+ group was significantly older ($p=0.004$) and had a higher proportion of females ($p=0.019$). No significant differences were found between groups in terms of family structure or household income ($p>0.05$). Additionally, rates of current psychotropic medication use did not differ significantly between the ANH+ and ANH− groups ($p>0.05$). Both anxiety and depression severity were significantly higher in the ANH+ group (p *values*<0.001). Additionally, the ANH+ group had significantly higher CSI-Total scores ($p<0.001$). Associations between CSI-Total scores and study variables are presented in Table 2.

Table 2: Correlations Between CSI-Total Scores and Study Variables (n = 242)

Variables[a]	CSI-Total
Age (years)	$r=0.118$, $p=0.066$
Gender*	$r=0.305$, $p<0.001$
School absenteeism (days)**	$r=0.150$, $p=0.031$
DARS-Total	$r=-0.444$, $p<0.001$
Core Anxiety	$r=0.530$, $p<0.001$
Core Depression	$r=0.542$, $p<0.001$

***** Gender coded as 0 = male, 1 = female; point-biserial correlation coefficient was used. ****** Calculated based on 208 participants who were attending school. [a.] Pearson correlation analysis.

No significant relationship was observed between age and CSI-Total scores. However, female sex ($p<0.001$) and school absenteeism ($p=0.031$) were positively associated with higher CSI-Total scores. CSI-Total scores showed significant positive correlations with anxiety and depression severity (p *values*<0.001), and a significant negative correlation with DARS-Total scores ($p<0.001$). Subgroup analyses according to diagnostic categories revealed that this pattern was consistent across diagnoses. Among participants diagnosed with MDD (n=86), CSI-Total scores showed a moderate negative correlation with DARS-Total scores ($r=-.385$). Similar moderate negative correlations were observed in participants with anxiety disorders (n=70, $r=-0.471$) and ADHD (n=60, $r=-0.308$). Results of the hierarchical regression analysis conducted to identify predictors of somatic symptom burden (CSI-Total scores) are presented in Table 3. Female sex was associated with increased somatic symptom severity ($\beta=0.295$, $p<0.001$). After controlling for age and sex, the addition of anxiety and depression severity to the model showed that both anxiety severity ($\beta=0.370$, $p<0.001$) and depression severity ($\beta=0.410$, $p<0.001$) were independently associated with increased somatic symptom severity. In the final model, after controlling for age, sex, anxiety severity, and depression severity, lower DARS-Total scores (indicating greater anhedonia severity) were

Table 3: Hierarchical Regression Analysis Predicting Total Somatic Symptom Severity (CSI-Total) (n = 242)

Independent Variables	Model I			Model II			Model III		
	β	t	p	β	t	p	β	t	p
Age	0.083	1.338	0.182	0.002	0.047	0.962	-0.005	-0.102	0.918
Gender (female, ref:male)	0.295	4.779	<0.001	0.107	2.093	0.037	0.096	1.902	0.058
Core-Anxiety	-	-	-	0.370	7.024	<0.001	0.383	7.335	<0.001
Core-Depression	-	-	-	0.410	7.989	<0.001	0.291	4.534	<0.001
DARS-Total	-	-	-	-	-	-	-0.184	-2.975	0.003

***** Model Summary: Model I: R^2 =0.100, Adjusted R^2 = 0.093, $F_{(2,239)}$ = 13.283, p<0.001 Model II: R^2 = 0.452, Adjusted R^2 = 0.443, $F_{(4,237)}$ = 48.826, p<0.001 Model III: R^2 = 0.472, Adjusted R^2 = 0.460, $F_{(5,236)}$ = 42.125, p<0.001 Dependent Variable: CSI-Toplam, β: Standardized Regression Coefficient

significantly associated with increased somatic symptom severity (β=–0.184, p=0.003). Additional sensitivity analyses including current psychotropic medication use as a covariate demonstrated that the association between DARS-Total scores and somatic symptom severity remained significant (β=–0.198, p=0.002), whereas psychotropic medication use itself was not independently associated with somatic symptom severity (p=0.360). Multicollinearity diagnostics indicated no significant multicollinearity among the predictors included in the regression model (tolerance=0.591–0.951; VIF=1.052–1.695).

Discussion

The findings of the present study conducted in a clinical adolescent sample support that anhedonia is significantly associated with the severity of somatic symptoms and that this association persists independently of depression and anxiety symptom severity. These results suggest that the burden of somatic symptoms may not merely reflect general internalizing psychopathology, but that reward and motivational processes may also contribute to the clinical presentation. Subgroup analyses by diagnostic categories demonstrated that the association between anhedonia and somatic symptom burden was similarly maintained among adolescents diagnosed with major depressive disorder (MDD), anxiety disorders, and attention-deficit/hyperactivity disorder (ADHD). This finding indicates that the observed relationship is not solely attributable to the diagnostic composition of the sample, but rather may reflect a broader mechanism observable across diverse psychopathological patterns. This pattern may support conceptualizations of anhedonia as a transdiagnostic dimension associated not only with affective distress but also with alterations in bodily and motivational processing across different psychiatric conditions (8,10). Previous studies conducted in depressive samples support this pattern of association between somatic symptoms and anhedonia (13). These studies suggest that anhedonia may play a specific role in understanding somatic symptomatology. In the present study, the persistence of the contribution of anhedonia to somatic symptoms even after controlling for depression and anxiety severity suggests that this relationship reflects a more specific psychological dimension. Group comparisons further supported this pattern. Adolescents with anhedonia exhibited significantly higher levels of both internalizing symptom severity (including anxiety and depressive symptoms) and total somatic symptom burden. This finding is consistent with literature reporting greater somatic symptom severity among individuals with anhedonia (13). Additionally, correlation findings demonstrating that somatic symptoms increase as hedonic capacity decreases suggest that anhedonia may be useful in identifying clinical subgroups characterized by prominent bodily symptoms. This pattern indicates that anhedonia is not limited to emotional processes but may also be closely related to bodily symptomatology (13,24). The observed association between school absenteeism and somatic symptom severity also highlights the functional impact of bodily symptoms during adolescence. Persistent somatic complaints may interfere with school attendance, academic engagement, peer relationships, and participation in daily activities, thereby contributing to broader psychosocial impairment, consistent with previous findings in pediatric functional somatic symptom research (2,3). From a clinical perspective, these findings suggest that somatic symptoms in

adolescents should be evaluated not only in terms of symptom severity but also with regard to their effects on daily functioning and educational continuity. The strong associations observed between anxiety and depression severity and somatic symptoms represent expected findings consistent with previous adolescent studies (4,5). However, the present results are noteworthy in demonstrating that anhedonia provides an additional and independent contribution despite the strong influence of internalizing symptoms. Although the additional variance explained by anhedonia was relatively modest, even small incremental contributions may be clinically meaningful in multifactorial phenomena such as somatic symptoms, particularly when they reflect transdiagnostic processes that are not fully captured by conventional internalizing symptom measures. This finding highlights the multidimensional nature of somatic symptoms and suggests that they cannot be explained solely within the framework of negative affectivity. This interpretation is also consistent with emerging theoretical models suggesting that disruptions in reward responsiveness and motivational engagement may contribute to the perception and amplification of bodily symptoms (8,25). The finding that female sex was associated with increased somatic symptom severity is also consistent with previous developmental psychopathology research demonstrating higher rates of functional somatic symptoms among adolescent girls and stronger associations with internalizing psychopathology during adolescence (2,4,5). Multiple biological and psychosocial factors may contribute to this association, including developmental differences in emotional processing, stress sensitivity, and tendencies toward internalizing symptom expression during adolescence (2,5). Sociocultural factors influencing emotional expression and help-seeking behaviors may also contribute to the greater reporting of bodily symptoms among adolescent girls (2,3). Taken together, these perspectives suggest that the relationship between anhedonia and somatic symptoms may best be conceptualized within an integrative framework involving reward dysfunction, altered motivational processing, and disturbances in interoceptive signaling. When the potential mechanisms underlying the association between anhedonia and somatic symptoms are examined at the neurobiological level, significant overlaps are observed between reward processing and pain regulation systems. Brain structures within the mesolimbic–prefrontal circuitry, including the anterior cingulate cortex, insula, and ventral striatum (particularly the nucleus accumbens), play central roles both in the affective component of pain and other somatic symptoms and in reward anticipation and hedonic experience (25). Functional alterations within these regions have been shown to affect not only reward processing but also the perception and regulation of bodily sensations (9). This overlap suggests that increasing anhedonia may lead to bodily sensations being experienced as more intense, more distressing, and less tolerable. Indeed, pleasurable stimuli such as pleasant odors, visual stimuli, music, or monetary rewards have been shown to inhibit pain perception (25). Dopaminergic and opioidergic systems also play critical roles in explaining this relationship. Dysfunction in the mesolimbic dopaminergic system reduces motivational processes, whereas alterations in the opioidergic system affect both reward experience and pain modulation (26,27). This indicates that anhedonia may represent a sensitivity dimension influencing not only emotional experience but also central processing of bodily sensations. The present findings may be particularly relevant during adolescence, a developmental period characterized by ongoing maturation of reward-related, emotional, and interoceptive neural systems (12). The developmental imbalance between relatively early-maturing limbic regions and later-developing prefrontal regulatory systems may increase vulnerability to both motivational dysfunction and altered interpretation of bodily sensations during this period (11). Interoceptive processing constitutes another important mechanism. Disruptions in the perception of bodily signals and their interpretation within cognitive-emotional contexts may lead to physiologically benign sensations being appraised as threatening or indicative of illness (28). It has been proposed that interactions between motivational system dysfunctions and altered interoceptive processing may influence how bodily sensations are perceived and interpreted, thereby contributing to the subjective experience of somatic symptoms (28,29). This framework may help explain why adolescents with more severe anhedonic symptoms may experience bodily sensations as more distressing or threatening. The "sickness behavior" model also provides a useful theoretical framework for explaining this pattern. This concept refers to a biobehavioral response pattern characterized by fatigue, increased pain sensitivity, motivational loss, and anhedonia, emerging in association with inflammatory processes and the release of proinflammatory cytokines

(30).Although this response is considered an adaptive process promoting rest and recovery, similar behavioral patterns and inflammation-related alterations have also been reported in certain clinical conditions associated with depression and anxiety. Therefore, the relationship between somatic symptoms and anhedonic features may be shaped not only through negative affect but also through shared biobehavioral mechanisms (24,30). Taken together, these findings may support perspectives suggesting that somatic symptoms can emerge through the interaction of affective distress, motivational dysfunction, and altered bodily signal processing rather than through a single psychopathological mechanism (24,28,29). Although biological markers were not assessed in the present study, the observed clinical pattern appears consistent with this theoretical model.

Study limitations: Several limitations should be considered when interpreting the findings. First, the cross-sectional design precludes definitive conclusions regarding the directionality and causal nature of the observed associations. Second, the single-center clinical sample limits the generalizability of the findings to community-based populations. Referral bias may also have contributed to the overrepresentation of adolescents with more severe symptom profiles in this clinically referred sample. In addition, a substantial proportion of the sample consisted of adolescents diagnosed with depressive and anxiety disorders. Although depression and anxiety severity were statistically controlled in the regression analyses, the observed associations may partially reflect characteristics specific to clinically referred adolescents with prominent internalizing psychopathology. Furthermore, female participants were overrepresented in the sample, which may have influenced the observed symptom patterns and may limit the generalizability of the findings across sexes. Third, although clinician-administered interviews such as the K-SADS-5 and CDRS-R were used, anxiety, anhedonia, and somatic symptoms were assessed through self-report scales. This may lead to expectancy bias and social desirability effects among participants. Fourth, somatic symptoms were evaluated as a single composite symptom cluster; the specific contributions of pain, gastrointestinal, cardiorespiratory, and autonomic symptoms were not examined separately. Finally, the absence of biological markers, experimental assessments specific to somatic symptoms, and neuroimaging data prevented direct testing of the proposed neurobiological mechanisms. Future longitudinal and multicenter studies employing multimethod assessments are needed to clarify the causal nature of these relationships and to elucidate the underlying mechanisms in greater detail.

Conclusion

This study demonstrates that anhedonia is significantly associated with somatic symptoms in a clinical adolescent sample and that this relationship persists independently of depression and anxiety symptoms. The findings suggest that somatic symptoms should not be considered merely as reflections of general internalizing severity, but rather should also be evaluated within a more specific psychopathological framework involving reward and motivational systems and bodily signal processing mechanisms.

Ethical approval: Ethical approval for this study was obtained from the Trabzon University Faculty of Medicine Non-Interventional Scientific Research Ethics Committee (Date: January 5, 2026; Approval No: 2026/26047).

Conflict of interest: The authors declare no conflicts of interest

Financial disclosure: No financial support has been received for this study.

Author contributions: Concept (OBK, IS), Design (OBK, KK, HRE), Supervision (OBK, OBD), Materials (OBK, IS, KK, HRE, MM), Data collection (OBK, IS, KK, HRE, ATU, MM), Analysis (OBK, HRE, ATU, OBD), Literature Review (OBK, IS, ATU, MM), Writing (OBK), Critical Review (OBK, KK, OBD). All authors have read and approved the final version of the manuscript.

Data availability statement: The datasets generated and/or analyzed during the current study are not publicly available because they contain information that could compromise participant privacy but are available from the corresponding author on reasonable request.

Use of AI tools: AI tools (ChatGPT-5, OpenAI) were used solely for language editing and grammatical refinement of the manuscript. All AI-assisted suggestions were carefully reviewed, verified, and edited by the authors, who take full responsibility for the final content of the manuscript.

Acknowledgments: The authors would like to thank all the adolescents and their parents who participated in this study. The authors also thank Dr Onur Saraç for his valuable contributions to the study.

References

1. Lu J, Han Y, Liu X, Li W, Zhou X. Association between somatic symptoms and depression and anxiety in adolescents: a cross-sectional school-based study. BMJ Open. 2025 Sep 3;15(9):e103860. doi:10.1136/bmjopen-2025-103860.
2. Campo JV. Annual research review: functional somatic symptoms and associated anxiety and depression--developmental psychopathology in pediatric practice. J Child Psychol Psychiatry. 2012 May;53(5):575–92. doi:10.1111/j.1469-7610.2012.02535.x.
3. Rask CU, Olsen EM, Elberling H, Christensen MF, Ornbøl E, Fink P, et al. Functional somatic symptoms and associated impairment in 5-7-year-old children: the Copenhagen Child Cohort 2000. Eur J Epidemiol. 2009;24(10):625–34. doi:10.1007/s10654-009-9366-3.
4. Jungmann SM, Wagner L, Klein M, Kaurin A. Functional Somatic Symptoms and Emotion Regulation in Children and Adolescents. Clin Psychol Eur. 2022 Jun 30;4(2):e4299. doi:10.32872/cpe.4299.
5. Münker L, Rimvall MK, Frostholm L, Ørnbøl E, Wellnitz KB, Jeppesen P, et al. Exploring the course of functional somatic symptoms (FSS) from pre- to late adolescence and associated internalizing psychopathology - an observational cohort-study. BMC Psychiatry. 2024 Jul 8;24(1):495. doi:10.1186/s12888-024-05937-3.
6. Bohman H, Låftman SB, Cleland N, Lundberg M, Päären A, Jonsson U. Somatic symptoms in adolescence as a predictor of severe mental illness in adulthood: a long-term community-based follow-up study. Child Adolesc Psychiatry Ment Health. 2018;12:42. doi:10.1186/s13034-018-0245-0.
7. Zhou X, Ren F, Lui SSY, Chan RCK. Interoception, somatic symptoms, and somatization tendency in Chinese individuals with subsyndromal depression: A follow-up study. PsyCh J. 2024 Aug;13(4):616–24. doi:10.1002/pchj.739.
8. Rizvi SJ, Gandhi W, Salomons T. Reward processing as a common diathesis for chronic pain and depression. Neurosci Biobehav Rev. 2021 Aug;127:749–60. doi:10.1016/j.neubiorev.2021.04.033.
9. Navratilova E, Morimura K, Xie JY, Atcherley CW, Ossipov MH, Porreca F. Positive emotions and brain reward circuits in chronic pain. J Comp Neurol. 2016 Jun 1;524(8):1646–52. doi:10.1002/cne.23968.
10. Treadway MT, Zald DH. Reconsidering anhedonia in depression: Lessons from translational neuroscience. Neurosci Biobehav Rev. 2011 Jan 1;35(3):537–55. doi:10.1016/j.neubiorev.2010.06.006.
11. Casey BJ, Jones RM, Hare TA. The adolescent brain. Ann N Y Acad Sci. 2008 Mar;1124:111–26. doi:10.1196/annals.1440.010.
12. Murphy J, Brewer R, Catmur C, Bird G. Interoception and psychopathology: A developmental neuroscience perspective. Dev Cogn Neurosci. 2017 Feb;23:45–56. doi:10.1016/j.dcn.2016.12.006.
13. Pérez-Morenilla A, Salazar A, Failde I, González-Sáiz F, Micó JA. Factors related to painful somatic symptoms in patients with depression: The role of gender and anhedonia. Psychiatry Res Commun. 2025 Mar1;5(1):100204. doi:10.1016/j.psycom.2025.100204.
14. Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P, et al. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. J Am Acad Child Adolesc Psychiatry. 1997 Jul;36(7):980–8. doi:10.1097/00004583-199707000-00021.
15. Fatih Ü, Ferhunde Ö, Füsun ÇETİN Ç, S. Ebru ÇK, Devrim A, Dilşad FÖ, Halime TÇ, Dilek Ü, Kevser T, Cihan A, Bilge Merve K, Büşra Sultan A, Funda K, Emine T, Remzi K, Başak K, Burak K, Yusuf K, Abdülbaki A. Okul çağı çocukları için duygulanım bozuklukları ve şizofreni görüşme çizelgesi- şimdi ve yaşam boyu şekli-DSM-5-Kasım 2016- Türkçe uyarlamasının geçerlilik ve güvenirliği. Türk Psikiyatri Dergisi 2019;30(1):42-50. doi:10.5080/u23408.
16. Poznanski EO, Mokros HB. Children's Depression Rating Scale–Revised (CDRS-R). Los Angeles: Western Psychological Services; 1996.
17. Alşen Güney S, Baykara HB, İnal Emiroğlu N; Çocuklar için Depresyon Değerlendirme Ölçeği Revize Formunun Türk popülasyonundaki ergenlerde psikometrik özellikleri. Anatolian Journal of Psychiatry 2018; 19(Special issue.1):41-48. doi:10.5455/apd.292666.

18. Rizvi SJ, Quilty LC, Sproule BA, Cyriac A, Michael Bagby R, Kennedy SH. Development and validation of the Dimensional Anhedonia Rating Scale (DARS) in a community sample and individuals with major depression. Psychiatry Res. 2015 Sep 30;229(1–2):109–19. doi:10.1016/j.psychres.2015.07.062.
19. Karakus OB, Doganer MZ, Turhan İY, Cesur TD, Cakir EO, Yuksel AS, et al. Dimensional Anhedonia Rating Scale (DARS) Turkish Version: Examination of Psychometric Properties in Depressed and Ultra-High Risk/Psychotic Adolescents. Psychiatr Q. 2025 Jun 28. doi:10.1007/s11126-025-10177-z.
20. Chorpita BF, Yim L, Moffitt C, Umemoto LA, Francis SE. Assessment of symptoms of DSM-IV anxiety and depression in children: a revised child anxiety and depression scale. Behav Res Ther. 2000 Aug 1;38(8):835–55. doi:10.1016/S0005-7967(99)00130-8.
21. Gormez V, Kilincaslan A, Ebesutani C, Orengul AC, Kaya I, Ceri V, et al. Psychometric Properties of the Parent Version of the Revised Child Anxiety and Depression Scale in a Clinical Sample of Turkish Children and Adolescents. Child Psychiatry Hum Dev. 2017 Dec;48(6):922–33. doi:10.1007/s10578-017-0716-1.
22. Walker LS, Beck JE, Garber J, Lambert W. Children's Somatization Inventory: psychometric properties of the revised form (CSI-24). J Pediatr Psychol. 2009 May;34(4):430–40. doi:10.1093/jpepsy/jsn093.
23. Kadıoğlu H, Sişman FN, Ergün A. Reliability and Validity of the Turkish Version of Children's Somatization Inventory. Asian Nurs Res. 2012 Mar;6(1):9–12. doi:10.1016/j.anr.2012.02.004.
24. Heitmann H, Andlauer TFM, Korn T, Mühlau M, Henningsen P, Hemmer B, et al. Fatigue, depression, and pain in multiple sclerosis: How neuroinflammation translates into dysfunctional reward processing and anhedonic symptoms. Mult Scler. 2022 Jun;28(7):1020–7. doi:10.1177/135245852097227.
25. Nees F, Becker S. Psychological Processes in Chronic Pain: Influences of Reward and Fear Learning as Key Mechanisms - Behavioral Evidence, Neural Circuits, and Maladaptive Changes. Neuroscience. 2018 Sep1;387:72–84. doi:10.1016/j.neuroscience.2017.08.051.
26. Garland EL, Trøstheim M, Eikemo M, Ernst G, Leknes S. Anhedonia in chronic pain and prescription opioid misuse. Psychol Med. 2020 Sep;50(12):1977–88. doi:10.1017/S0033291719002010.
27. Liu X, Wang N, Gu L, Guo J, Wang J, Luo F. Reward Processing under Chronic Pain from the Perspective of "Liking" and "Wanting": A Narrative Review. Pain Res Manag. 2019 Apr 21;2019:6760121. doi:10.1155/2019/6760121.
28. Dunn BD, Stefanovitch I, Evans D, Oliver C, Hawkins A, Dalgleish T. Can you feel the beat? Interoceptive awareness is an interactive function of anxiety- and depression-specific symptom dimensions. Behav Res Ther. 2010 Nov;48(11):1133–8. doi:10.1016/j.brat.2010.07.006.
29. Saltafossi M, Heck D, Kluger DS, Varga S. Common threads: Altered interoceptive processes across affective and anxiety disorders. J Affect Disord. 2025 Jan 15;369:24454. doi:10.1016/j.jad.2024.09.135.
30. Dantzer R. Cytokine-induced sickness behavior: where do we stand? Brain Behav Immun. 2001 Mar;15(1):7–24. doi:10.1006/brbi.2000.0613.