

Metacognition in anorexia nervosa: Comparison of adolescents with anorexia nervosa and healthy controls in terms of anxiety, depression, and maternal mental symptoms

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Summary

Background: Metacognition is an emerging area of research in anorexia nervosa (AN). The primary aim of this study was to compare adolescents with AN and healthy controls on self-reported measures of metacognition, depression, and anxiety. Additionally, maternal mental symptoms were compared between the two groups. A secondary aim was to investigate the effects of self-reported depression and anxiety, as well as maternal mental symptoms, on metacognition.

Subjects and Methods: Twenty-seven adolescents with AN and 54 healthy controls completed the Metacognition Questionnaire for Children and Adolescents (MCQ-C), the Childhood Depression Inventory (CDI), and the Screen for Child Anxiety-Related Emotional Disorders (SCARED). Mothers completed the Symptom Assessment Questionnaire (SA-45).

Results: Multivariate analyses revealed that adolescents with AN exhibited higher scores on measures of dysfunctional metacognitive activity. These adolescents also had elevated levels of depression and anxiety, which were positively correlated with metacognitive dysfunction. Moreover, maternal mental symptoms were positively associated with the patients' metacognitive dysfunction.

Conclusions: Metacognitions may be central to understanding the cognitive disturbances in AN, and metacognitive treatment strategies could hold promise for the development of novel psychological interventions for AN. Further research is needed to explore the impact of depression and anxiety, along with maternal mental symptoms, on metacognition in individuals with AN.

Keywords: anorexia nervosa, metacognition, adolescent, depression, anxiety, maternal mental symptoms, path analysis

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INTRODUCTION

Metacognition is described as a high-level cognitive construct that enables individuals to notice, monitor, and examine their own thought processes. Similarly, it is an individual's knowledge of what they know, what they think, or their eyes that have turned to their own cognitive processes (Tosun & Iraq, 2008). The development of metacognitive processes begins between the ages of 3 and 5, coinciding with the development of theory of mind, and can continue throughout life. The development of metacognition accelerates in parallel with the development of cognitive processes, particularly during the school years. Metacognitive beliefs are responsible for the functional and dysfunctional control of the mind (Wells & Cartwright-Hatton, 2004). Metacognitions, which are thoughts about thinking, play a crucial role in the functional and harmonious operation of cognitive processes. Dysfunction in this system are thought to contribute significantly to the development and persistence of many psychopathologies (Tosun & Irak, 2008). Metacognitions

have been investigated in several disorders, such as anxiety (Wells & Carter, 2001; Fisher & Wells, 2008; Ünver et al., 2021), depression (Wells et al., 2009; Papageorgiou & Wells, 2003), addiction (Spada and Wells, 2009; Spada, Caselli & Wells, 2009), post-traumatic stress disorder (Roussis & Wells, 2006; Roussis & Wells, 2008), psychosis (Morrison & Wells, 2003), and attention deficit hyperactivity disorder (ADHD) (Ünver et al., 2022).

Recently, metacognitions have also been addressed in eating disorders. In a study comparing anorexia nervosa (AN) patients with a control group, individuals with AN scored higher than the other groups on dimensions of dysfunctional metacognitions such as uncontrollability and danger, need for control, cognitive confidence and cognitive awareness (Cooper et al., 2007). Another study found that patients diagnosed with AN had higher levels of anxiety, obsessive-compulsive symptoms and dysfunctional metacognitions compared to the control group (Palomba et al., 2017). In another study comparing patients diagnosed with AN, dieters and a healthy control group, Woolrich, Cooper and Turner (2008) showed that patients with

AN and dieters used dysfunctional metacognitions to a greater extent than the control group. Furthermore, metacognitions in AN share common features with metacognitions in anxiety disorders (Konstantellou et al., 2011; Cooper, 2005). Individuals with eating disorders often fail to cope with anxiety and self-regulation when faced with weight and body image related stimuli. Studies of people with eating and anxiety disorders have shown that these two groups have higher levels of rumination, uncertainty intolerance, positive worry beliefs, negative problem solving and cognitive avoidance compared to healthy controls (Woolrich, Cooper & Turner, 2008).

A variety of factors have been identified as influencing metacognition. It has been suggested that elevated levels of depression and anxiety can have a detrimental impact on metacognitive functioning. A study conducted with adolescents demonstrated that increasing levels of depression and anxiety negatively affected metacognitive skills (Ünver et al., 2022). Similarly, a study involving adolescents diagnosed with obsessive-compulsive disorder (OCD) found that metacognition was adversely impacted as co-occurring internalizing symptoms increased (Isaksen et al., 2024). Elevated levels of depression and anxiety are commonly observed in individuals diagnosed with anorexia nervosa (Ünver et al., 2020; Aquilina, Agius & Sharma, 2014), which suggests that these symptoms may also negatively affect metacognitive functioning in patients with AN.

Studies show that the frequency of psychopathology increases in children of parents with psychiatric disorders like anxiety and depression (Cooper et al., 2006; Goodman et al., 2011; Thevenet, Geoffray & Georgieff N, 2016). The danger-related cognitions of mothers with anxiety disorders may be transmitted to their children. At the same time, metacognitions, like cognitions, may be transmitted to children in a developmental manner, but the mechanisms by which this transmission occurs are not yet known (Esbjörn et al., 2016). In patients diagnosed with AN, the relationship between mother and adolescent is emphasised. The stress level of mothers of adolescents with AN increases and stress levels may cause maternal depression and anxiety symptoms. This could lead to adolescents with AN experience more family conflict (Sim et al., 2009; Starzomska & Tadeusiewicz, 2016). Maternal mental symptoms may influence the metacognition of adolescents diagnosed with AN as well as cognitions.

The primary aim of this study is to compare the metacognitions of adolescents diagnosed with AN with a

healthy control group. Another aim is to investigate the influence of the patients' depression and anxiety levels and their mothers' mental symptoms on the patients' metacognitions. Our hypotheses are that adolescents with AN will exhibit more dysfunctional metacognitions compared to the healthy control group, and that higher levels of depression and anxiety, along with maternal mental symptoms, will be associated with increased dysfunctional metacognitions in adolescents.

SUBJECTS AND METHODS

Study Design and Participants

This study was designed as an observational and cross-sectional study. It was conducted at the Child and Adolescent Psychiatry Outpatient Clinic of the Marmara University Medical School Pendik Training and Research Hospital. The patient group consisted of adolescents who met the diagnostic criteria for AN according to DSM-5 and who were followed up and treated in our outpatient clinic. Twenty-seven adolescents aged between 12 and 18 years who volunteered to participate in the study comprised the patient group. Exclusion criteria for the patient group included intellectual disability, autism spectrum disorder, psychotic disorder, neurological disorder (e.g. epilepsy), substance use, or history of head trauma. For the control group, 54 children and adolescents aged 12 to 18 years were recruited from a secondary school near the hospital who volunteered to take part in the study. Exclusion criteria for the control group included current psychiatric disorder or history of psychiatric disorder, mental retardation, epilepsy or other neurological disorder or traumatic brain injury, and chronic/severe medical illness. Ethical approval was obtained from the Marmara University School of Medicine Ethics Committee (06.10.2023.1031).

Patients were assessed using the Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version (K-SADS-PL). Mothers completed the Sociodemographic Data Form and the Symptom Assessment Questionnaire (SA-45). Adolescents completed the Childhood Depression Inventory (CDI), the Metacognition Questionnaire for Children and Adolescents (MCQ-C), and the Screen for Child Anxiety-Related Emotional Disorders (SCARED).

ASSESSMENT INSTRUMENTS

Sociodemographic form

This form was designed by the researchers. It records the age and gender of the participants and the educational level, age and psychiatric history of the mothers.

Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version (K-SADS-PL)

The K-SADS-PL is a semi-structured diagnostic interview developed by Kaufman et al. (1997). The validity and reliability study was conducted by Gökler et al. (2004).

Symptom Assessment Questionnaire (SA-45)

This scale consists of 45 questions and 9 symptom domains. The Turkish validity and reliability study was conducted by Epözdemir (2009). This scale allows individuals to rate their own mental symptoms on a 5-point scale. It includes subscales such as anxiety, depression, hostility, interpersonal sensitivity, obsessive-compulsive, paranoid ideation, phobic anxiety, psychoticism and somatisation.

Metacognition Questionnaire for Children and Adolescents (MCQ-C)

The MCQ-C was developed by Bacow et al. (2009) and consists of 24 items. The scale consists of 4 subscales: positive metaworry; negative metaworry; superstitious, punishment, and responsibility beliefs and cognitive monitoring. The Turkish validity and reliability study was conducted by Irak (2012). Each item is rated on a Likert scale ranging from '(1) strongly disagree' to '(4) strongly agree'. Scores range from 24 to 96, with higher scores indicating dysfunctional metacognitive activity. The appropriateness and validity of the MCQ-C for use in the AN population was assessed by examining the Cronbach's alpha coefficient on the total score of the scale, which was 0.814. In addition, Cronbach's alpha coefficient for the subscales, positive metaworry was 0.808, negative metaworry was 0.797, superstition, punishment

and responsibility beliefs subscale was 0.584 and cognitive monitoring was 0.693.

Childhood Depression Inventory (CDI)

The CDI can be administered to children and adolescents aged 6-17 years. It is based on the Beck Depression Scale and was developed by Kovacs (1992). The child chooses the response that best describes his or her feelings over the past two weeks. The scale consists of 27 items, each of which is scored as '0', '1' or '2' depending on the severity of the symptom. A higher score indicates more severe depression. The Turkish validity and reliability study was carried out by Öy (1991).

The Screen for Child Anxiety-Related Emotional Disorders (SCARED)

Developed by Birmaher et al. (1997), the Turkish validity and reliability study was conducted by Çakmakçı (2004). Each item is scored between 0-2 and the scale consists of 41 questions. An increase in the total score indicates a higher level of anxiety. Subscale scores can also be calculated for panic disorder/somatic complaints (13 items), generalised anxiety disorder (9 items), separation anxiety (8 items), social phobia (7 items) and school phobia (4 items). A total score of 25 or more indicates the presence of an anxiety disorder.

STATISTICAL ANALYSIS

Data were analysed using SPSS for IBM version 20.0. Descriptive statistical methods shown as mean \pm standard deviation (SD) were used to analyse the study data. The chi-square (χ^2) test or Fisher's exact test was used to compare numerical data; Student's t-test and Mann Whitney-U test were used to compare continuous variables. Pearson's correlation analysis was used for parametric variables. Predictive factors for metacognition in all group were evaluated using linear regression analysis. The main question addressed in the analyses was whether maternal mental symptoms affect the metacognition of adolescents via AN diagnosis, age, anxiety, and depressive symptoms. The path model for metacognition in adolescents was tested using the PROCESS macro for SPSS v2.16.3 (Hayes, 2013) based on ordinary least squares (OLS) regression. In this study, model 4 in Hayes's Process was used to test the mediating

effect of anxiety and depression of adolescents (see Fig. 1). Before modeling, age, presence of AN diagnosis, maternal mental symptoms, depression, and anxiety, which may influence the risk of metacognition in adolescents, were entered into a linear regression analysis to test for possible effects. While anxiety, depression, and maternal mental symptoms predicted adolescent metacognition, age and AN diagnosis did not. The model was built separately for both the mediating effects of anxiety and depression when testing models related to the direct and indirect effects of maternal mental symptoms on adolescent metacognition.

To ensure the validity and reliability of results in structural equation modeling (SEM), an adequate sample size

is critical. Established methodological guidelines suggest that sample size requirements depend on several factors including model complexity, the number of estimated parameters, and the estimation method employed. A commonly cited rule of thumb is a minimum of 10 cases per estimated parameter, though more conservative standards suggest 15–20 cases per parameter for increased model stability (Kline, 2023). In this study coefficients suggest a moderately complex model, well within the manageable scope of standard mediation frameworks (Hayes Model 4), supporting the appropriateness of standard SEM sample size guidelines.

Table 1. Comparison of groups in terms of Childhood Depression Inventory, SCARED, MCQ-C, and SA-45

	Anorexia Nervosa (n=27) mean±sd	Control (n=54) mean±sd	z/t value	p value
Childhood Depression Inventory	21.19±9.95	10.41±6.14	-4.548 ^b	<0.001
Panic disorder	0.84±0.57	0.41±0.32	-3.090 ^b	0.002
Generalized anxiety disorder	1.29±0.66	0.67±0.44	-3.805 ^b	<0.001
SCARED				
Seperation anxiety disorder	0.56±0.30	0.58±0.34	-0.263 ^b	0.793
Social phobia	1.03±0.61	0.80±0.37	-1.699 ^b	0.089
School phobia	0.78±0.62	0.58±0.34	-2.183 ^b	0.029
Total	0.91±0.45	0.57±0.26	-3.233 ^b	0.001
Positive metaworry	12.15±4.28	9.77±3.58	-2.456 ^b	0.014
Negative metaworry	19.15±3.86	15.20±4.32	3.960 ^a	<0.001
MCQ-C				
Superstition, punishment and responsibility	18.29±3.20	15.09±3.48	-3.832 ^b	<0.001
Cognitive monitoring	17.92±3.12	17.53±4.00	-0.320 ^b	0.749
Total	67.40±9.29	57.61±9.85	4.179 ^a	<0.001
Anxiety	11.60±5.42	7.55±2.61	-2.553 ^b	0.011
Depression	13.33±6.50	8.63±3.14	-2.600 ^b	0.009
Hostility	7.93±3.08	6.65±2.47	-1.871 ^b	0.061
Interpersonal sensitivity	13.20±5.63	7.76±2.73	-3.382 ^b	0.001
SA-45				
Obsessive-compulsive	12.53±4.94	9.74±3.91	-1.979 ^b	0.048
Paranoid ideation	7.93±3.08	6.65±2.47	-2.536 ^b	0.011
Phobic anxiety	6.27±2.38	6.73±2.83	-1.871 ^b	0.061
Psychoticism	9.40±2.92	6.78±2.55	-3.311 ^b	0.001
Somatization	11.73±5.71	8.54±3.45	-2.019 ^b	0.044
Total	103.26±37.13	71.61±17.14	-2.809	0.005

^a; Student t test, ^b; Mann Whitney U test, significant results are shown in bold.

MCQ-C; Metacognition Questionnaire for Children and Adolescents, **SCARED**; The Screen for Child Anxiety Related Emotional Disorders, **SA-45**; Symptom Assessment Questionnaire.

RESULTS

Sociodemographic Data

In total, 81 adolescents (27 in the AN group and 54 in the control group) were included in the study. The mean age in the AN group (15.60 ± 1.57 years) and the control group (15.70 ± 1.74 years) was similar ($t = -0.262, p = 0.794$). In total, 3.7% of the AN ($n = 1$) and control group ($n = 2$) were male ($\chi^2 = 0.000, p = 1.000$). There was a higher prevalence of psychiatric disorders among the mothers ($n=8, \% 29.6$) of the patients diagnosed with AN ($\chi^2 = 1,627, p = 0.001$).

Clinical Assessment

In total, 63% ($n = 17$) of the AN group had ≥ 1 comorbid diagnosis, with the following rates: major depressive disorder: 48.1%; generalized anxiety disorder: 48.1%; social phobia: 33.3%; ADHD: 25.9%; panic disorder: 22.2%; separation anxiety disorder: 3.7%; obsessive-compulsive

disorder: 3.7%; tic disorders: 3.7%; specific phobia: 3.7%. None of the adolescents smoked, drank alcohol, or used drugs.

Group Comparisons

CDI total score, SCARED total score and subscale scores, MCQ-C total score and subscale scores, and SA-45 total score and subscale scores were compared between the two groups. The AN group scored significantly higher than the control group in terms of CDI score, SCARED total score, SCARED panic disorder, generalized anxiety disorder, and school phobia subscale scores. Moreover, the difference between the AN and control groups was significant for the SA-45 subscale scores except for the hostility and phobic anxiety subscale scores and MCQ-C subscale scores except for the cognitive monitoring subscale score (Table 1). Table 2 shows the correlations between the study scale scores in the patient group. Positive correlations were found between the CDI total score, SCARED subscale and total scores, and MCQ-C

Table 2. Correlation analysis of MCQ-C with SA-45, Childhood Depression Inventory, and SCARED in patient group.

	MCQ-C				
	PMW	NMW	SPR	CM	Total
Childhood Depression Inventory	0.301**	0.678***	0.671***	0.110	0.665***
Panic disorder	0.657***	0.595***	0.564***	0.084	0.585***
Generalized anxiety disorder	0.714***	0.607***	0.518***	0.221*	0.681***
SCARED					
Seperation anxiety disorder	0.336**	0.328***	0.206	0.242*	0.335**
Social phobia	0.465***	0.376***	0.308**	0.219	0.434***
School phobia	0.393***	0.251**	0.266*	-0.062	0.263**
Total	0.714***	0.633***	0.537***	0.191	0.652***
Anxiety	0,316**	0,340**	0,393**	0,021	0,388**
Depression	0,295*	0,280*	0,452***	0,056	0,372**
Hostility	0,278*	0,095	0,244*	-0,063	0,187
Interpersonal sensitivity	0.374**	0.342**	0.389**	0.053	0.420***
SA-45					
Obsessive-compulsive	0.289*	0.288*	0.378**	0.052	0.368**
Paranoid ideation	0,429***	0,351**	0,329**	0,108	0,463***
Phobic anxiety	0.278*	0.095	0.244*	-0.063	0.187
Psychoticism	0,233	0,206	0,175	0,256*	0,303*
Somatization	0,368**	0,247*	0,203	-0,053	0,262*
Total	0.445***	0.367**	0.434***	0.064	0.472***

Pearson correlation test, *r* coefficient is presented in the table. *, $p < 0.05$, **, $p < 0.01$, ***, $p < 0.001$.

MCQ-C; Metacognition Questionnaire for Children and Adolescents, **PMW**; Positive Metaworry, **NMW**; Negative Metaworry, **SPR**; Superstition, Punishment and Responsibility, **CM**; Cognitive Monitoring, **SCARED**; The Screen for Child Anxiety related Emotional Disorders, **SA-45**; Symptom Assessment Questionnaire.

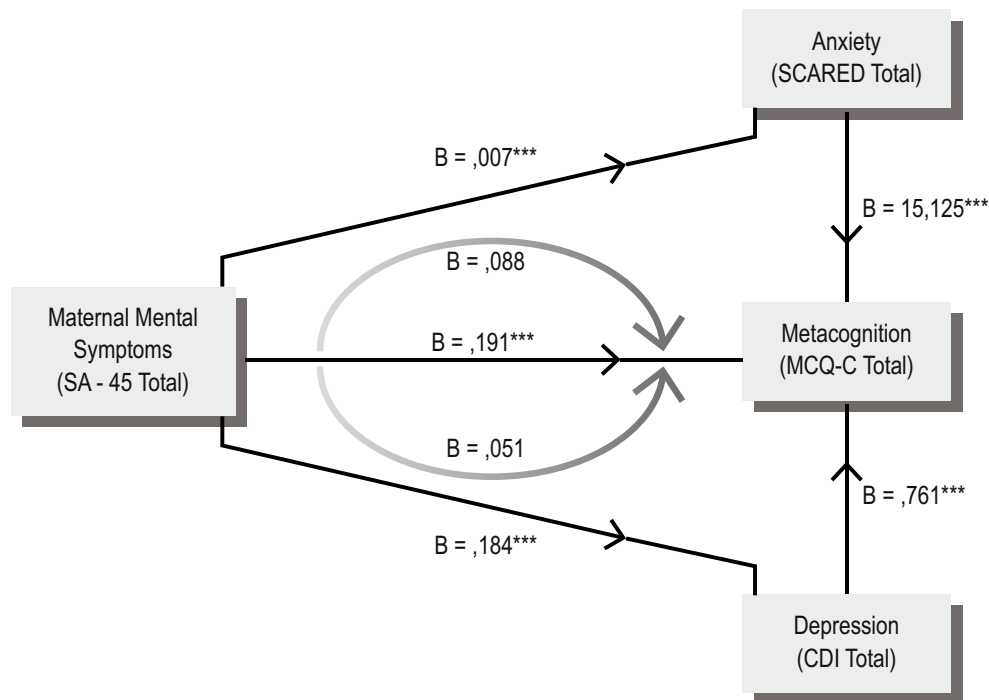


Figure 1. Diagram of a hypothetical mediation model.

Note. Standardized path coefficients are presented in bold with the mediational paths. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ (Andrew Hayes's mediation model 4).

Path model analysis revealed that the direct effect of maternal mental symptoms on metacognitions was insignificant ($B=0.088$ for the model in which anxiety as a mediator, $B=0.051$ for the model in which depression as a mediator). A significant indirect effect of maternal mental symptoms on metacognition through anxiety ($B= 0.104$) and depressive symptoms ($B= 0.140$). The indirect effect of maternal mental symptoms was significant on metacognitions through total anxiety and depressive symptoms ($B= 0.191$, $p < 0.001$).

subscale and total scores. Similarly, positive correlations were found between the SA-45 subscale and total scores and MCQ-C subscale and total scores.

anxiety and depressive symptoms ($B= 0.191$, $p < 0.001$). The structural model is shown in Figure 1.

Structural equation modelling

This study investigates the mediating role of anxiety and depression in the relationship between maternal mental symptoms and metacognition among all participants. The model, designed to assess the impact of SA-45 on MCQ-C, SCARED, and CDI, was tested through path analysis. Path model analysis revealed that the direct effect of maternal mental symptoms on metacognitions was insignificant ($B=0.088$ for the model in which anxiety as a mediator, $B=0.051$ for the model in which depression as a mediator). The results revealed a significant indirect effect of maternal mental symptoms on metacognition through anxiety ($B= 0.104$) and depressive symptoms ($B= 0.140$). The indirect effect of maternal mental symptoms was significant on metacognitions through total

DISCUSSION

The study revealed that adolescents diagnosed with AN exhibited significantly greater metacognitive dysfunction compared to healthy controls. Moreover, the mothers of adolescents with AN reported elevated levels of mental symptoms. Increases in adolescents' levels of depression and anxiety were positively associated with greater metacognitive dysfunction. Similarly, higher levels of maternal mental symptoms were linked to increased metacognitive dysfunction in their children. However, maternal symptoms did not have a direct effect on adolescents' metacognitive functioning; instead, their impact was indirect, operating through the exacerbation of depression and anxiety in the adolescents.

It is well established that comorbid pathologies in psychiatric disorders have a negative impact on

functionality (Zimmerman et al., 2018). The cumulative impact of multiple comorbidities in AN represents a significant challenge both clinically and theoretically. The high prevalence of comorbidity in our patient group is likely to have contributed to the deterioration in metacognitive functioning. As patients' levels of depression and anxiety increased, so did their metacognitive dysfunction. However, dysfunctional metacognition may also have contributed to the comorbidity of anxiety or depression symptoms. Furthermore, the results suggest a bidirectional relationship, where dysfunctional metacognitions may exacerbate anxiety and depression symptoms and vice versa. The presence of similar dysfunctional metacognitions in AN and anxiety disorders may suggest a reciprocal causal relationship (Konstantellou et al., 2011; Cooper, 2005). Furthermore, metacognitions in eating disorders may be transdiagnostic and may partly explain the temporal migration between diagnoses and the degree of comorbidity with eating disorders (Vann et al., 2014).

Further, individuals with AN frequently present with co-occurring psychiatric conditions such as OCD, and ADHD. When these comorbidities are present simultaneously, their effects may not simply be additive but rather synergistic, creating a more complex clinical picture. For example, the rigid cognitive patterns seen in OCD may amplify the perfectionistic and compulsive traits often observed in AN, while attentional difficulties associated with ADHD could further impair an individual's ability to engage in therapy. Such overlapping symptomatology may also have unique implications for metacognitive functioning, potentially disrupting cognitive monitoring, superstitious, punishment, and responsibility beliefs. As a result, individuals with multiple comorbidities may experience more persistent symptoms, poorer treatment outcomes, and greater cognitive-affective dysregulation than those with AN alone. Future research should aim to disentangle these complex interactions to inform more tailored, transdiagnostic intervention strategies (Marucci et al., 2018).

Studies have demonstrated that parental psychopathology can significantly influence the psychological symptoms exhibited by their children (Cooper et al., 2006; Schlüter-Müller, 2020). Such psychopathology can disrupt the parent-child relationship by promoting dysfunctional metacognitions, a dynamic that may be particularly pronounced in mothers due to their typically closer emotional bond with the child. Meta-analytic findings support this, revealing a stronger link between maternal and child internalising problems compared to paternal influences (Connell & Goodman, 2002). These are examples of the direct pathways through which maternal psychopathology can affect adolescents' metacognitive development. Indirect effects also play a critical role,

often mediated through parenting behaviours and the broader emotional climate of the home. For instance, maternal depression is associated with less responsive and engaged parenting, which may hinder the development of adaptive metacognitive skills in children. Similarly, maternal anxiety can contribute to a chronically tense home environment, further complicating the adolescent's ability to cultivate coherent and flexible metacognitive strategies. Maternal mental health symptoms also affect the quality of parenting. Mothers experiencing depression or anxiety may exhibit less emotional availability, reduced sensitivity, or inconsistent discipline. These disruptions can impair the development of secure attachment, which is foundational for reflective functioning and the internalization of metacognitive strategies. Chronic maternal psychological distress can contribute to increased family conflict, lower cohesion, and greater household instability. Such an environment may create cognitive and emotional overload for adolescents, reducing opportunities for the co-regulation and reflective dialogue necessary for the development of metacognitive skills. Children and adolescents learn metacognitive strategies partly by observing others. A mother who struggles with emotional regulation or distorted thinking patterns may inadvertently model maladaptive cognitive processing styles, which adolescents may internalize. This vicarious learning can lead to dysfunctional metacognitive patterns, such as rumination or impaired mentalization (Goodman et al., 2011; Thevenet, Geoffroy, & Georgieff, 2016).

Mothers of adolescents diagnosed with AN often experience heightened stress levels, contributing to increased family conflict (Sim et al., 2009). In our study, mothers in the patient group reported significantly higher levels of mental symptoms compared to controls. Notably, while maternal mental symptoms did not have a direct impact on adolescent metacognitions, they indirectly influenced metacognition by exacerbating depression and anxiety in adolescents. These indirect pathways could involve changes in the home environment, social interactions, or parenting styles due to maternal mental symptoms, all of which may influence the adolescent's metacognitive development (Baron & Kenny, 1986). While the transmission of cognitions from mothers to children has been recognized developmentally, the specific mechanisms underlying this transmission remain unclear (Esbjörn et al., 2016). Maternal metacognition was not assessed in our study, highlighting the need for future research to more comprehensively explore the relationship between maternal and child metacognition.

The results of this study showed that adolescents with AN have dysfunctional metacognitions compared to healthy controls. This is consistent with previous

research on metacognition in adults with AN (Vann et al., 2014; Olstad et al., 2014). It has also been found that high metacognition scores are associated with higher levels of eating disorder symptoms (Olstad et al., 2014). The higher metacognitive dysfunction in the clinical group may highlight maladaptive metacognition as an underlying psychopathological process, although longitudinal or experimental studies are needed for further clarification.

No significant difference was found between the AN and control groups in terms of the 'cognitive monitoring' scores, a subscale specified in the metacognition. Similar findings were reported in a study comparing metacognition in pediatric OCD patients and healthy controls, where mild OCD patients showed cognitive monitoring subscale levels similar to healthy controls, increasing with disease severity (Isaksen et al., 2024). In our study, AN patients were not classified by severity of illness, but were included during ongoing treatment, which may explain the similarity in cognitive monitoring subscale levels between patient and control groups. In another study conducted with adolescents diagnosed with anxiety disorder, the cognitive monitoring subscale level was found to be higher in the control group, which was explained that cognitive monitoring subscale level does not always result in the development of psychopathology and that the monitoring of the young people in the control group is not as occupied with internal stimuli as the young people in the patient group (Bacow et al., 2009).

Given the crucial role of metacognition in the functional and harmonious functioning of cognitive processes, it is conceivable that people with AN may misattribute importance to body weight and shape, while failing to anticipate the consequences of low body weight. In addition, impaired cognitive functions due to biochemical disorders in people with AN may further impair metacognition. Furthermore, recent literature suggests that individuals with AN may exhibit dysfunctional metacognition and reduced gray matter volumes in prefrontal brain regions associated with cognitive processes. Dysfunctional metacognitions may be linked to atypical functioning in regions such as the anterior cingulate cortex, medial prefrontal cortex mPFC, and insular cortex. Functional and structural alterations in these regions may contribute to the rigid cognitive patterns, and poor insight often observed in individuals with AN. However, it remains unclear whether these neurobiological differences represent reversible state-dependent changes associated with malnutrition and acute illness or more permanent trait-like markers predisposing individuals to anorexia nervosa. Longitudinal studies are still needed to disentangle whether the observed alterations in brain structure and function—particularly in regions such as the anterior

cingulate cortex, medial prefrontal cortex, and insula—normalize with sustained recovery, or if they reflect enduring vulnerabilities that may contribute to relapse. This ambiguity underscores the importance of incorporating both neurodevelopmental perspectives and recovery-focused research when examining the neurobiological basis of metacognitive dysfunction in AN (Olivia et al., 2020).

Metacognitions are known to influence both the development and maintenance of psychopathology (Tosun & Irak, 2008), potentially affecting treatment adherence and clinical outcomes. Clinical improvements in patients with AN, correlating with improved biological parameters and weight recovery, highlight the interplay between biological and cognitive functions in shaping metacognition (Ünver et al., 2024). Although biological parameters were not assessed in our study, their consideration remains critical to understanding changes in metacognition.

Nevertheless, existing literature indicates that metacognitive training is a promising adjunctive intervention for individuals with eating disorders, including adolescents and adults with AN. For adolescents, an online therapist-delivered intervention targeting thinking styles received positive feedback, demonstrated high treatment retention, and resulted in reduced perfectionism compared to wait-list controls (Balzan et al., 2023). In contrast, the adult study showed preliminary results, emphasizing the need for further research to validate the effectiveness of this treatment for AN (Lawson et al., 2022). Consequently, metacognitive dysfunction may play a critical role in the pathophysiology of AN, suggesting that metacognitive therapy could be a valuable treatment option for adolescents with AN.

LIMITATIONS

Our study has several limitations. These include a small sample size with unequal groups, enrolment of AN patients during ongoing psychiatric treatment, lack of maternal metacognitive assessments, and failure to assess fathers and siblings living with the patients. The prevalence of comorbid conditions among adolescents with AN was relatively high, with a comorbidity rate of 63%. Approximately half of these patients involved comorbid depression and anxiety. The analyses indicated that depression and anxiety scores significantly influenced metacognitive functioning, and these variables were included as predictors in the model. Although previous studies have demonstrated that metacognition is negatively affected in conditions such as ADHD (Ünver et al., 2022) and OCD (Isaksen et al., 2024), these comorbidities were less prevalent in our

sample. Specifically, one-fourth of the patients had comorbid ADHD, while those with OCD represented a smaller subgroup. As such, the findings did not sufficiently account for the potential confounding effects of comorbid conditions like ADHD and OCD, which may independently impair metacognitive functioning. Moreover, ADHD-like symptoms can also emerge in patients with AN due to underlying medical conditions (Marucci et al., 2018), which may have further influenced the outcomes.

Additionally, the maternal mental symptoms were evaluated exclusively through self-report scales, which limits the validity and depth of these findings. Self-report measures introduce several potential biases in clinical populations. Self-report bias refers to the discrepancy between the self-reported and actual values of a given measure, which can distort descriptive statistics and undermine causal inferences (Bauhoff, 2023). The lack of structured clinical interviews with mothers limits our ability to differentiate between bias in the results obtained from self-report measures. The cross-sectional design employed prevents causal inferences regarding the relationships among maternal mental symptoms, adolescents' anxiety and depression, and metacognitive dysfunction. To enhance methodological rigor, future studies should prioritize multi-informant approaches, integrating data from adolescents, fathers or co-parents, teachers, and clinical observers. Additionally, the use of objective or observational measures, such as structured clinical interviews, and behavioral assessments would provide a more comprehensive and nuanced understanding of maternal mental health and its impact on adolescent outcomes. In sum, while the current reliance on maternal self-report was a pragmatic choice within the constraints of the study design, we strongly recommend that subsequent research incorporate diverse data sources to mitigate bias and improve the reliability and validity of findings related to maternal mental health and adolescent metacognitive functioning.

Lastly, the other important limitation of the present study concerns the use of structural equation modeling with a relatively small sample size. Given the complexity of the mediation model tested, the statistical power may have been insufficient to detect subtle effects or to ensure the stability of parameter estimates. Therefore, the SEM

findings should be interpreted with caution and regarded as preliminary. Future studies with larger and more diverse samples are needed to validate these results and further explore the hypothesized relationships.

CONCLUSIONS

This study provides preliminary evidence of dysfunctional metacognitions in adolescents diagnosed with AN and highlights the importance of assessing comorbid depressive and anxiety symptoms in these patients, as well as their mothers' mental symptoms. The relatively small sample size significantly limits the statistical power and generalizability of the study's findings. Future research with longitudinal or experimental designs should include larger, more representative cohorts to strengthen external validity. Further, future studies would benefit from integrating comprehensive clinical assessments or structured diagnostic interviews for mothers, thereby enhancing the reliability and interpretability of maternal influence data.

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