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*Hello, hello!**

*Comedian Deniz Göktas's opening word for his stand-up routine.

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Life flows on with all its force.

Human beings evolved to live in natural environments and in small communities held together by strong kinship ties. Today, we live in crowded cities, use social media, and are exposed to a continuous stream of economic, technological, and social change. Population growth, forced and voluntary migration, fragmented communities, economic inequality, and the exaggerations that run through both traditional and social media are steadily widening the distance between appearance and reality.

Humor is born and flourishes precisely in this distance.

Looking at it the other way around, we might say that societies with a highly developed sense of humor tend to have a considerable gap between appearance and reality. Political language claims possession of the truth. It speaks with sharp certainty and can be rigid as crystal. The language of humor, in contrast, is relaxed, expansive, flexible, and ambiguous. It also makes room for absurdity. Over time, absurdity has become one of the essential ingredients of a good joke.

What I mean is that a good joke requires the ordinary chain of cause and effect to disappear, if only for a moment, so that criticism and censorship can lose their grip. It would not be entirely wrong to place our own country rather high among the nations of the world when it comes to the distance between appearance and reality. Societies marked by strong state authority, feudal structures, and patriarchy are also remarkably fertile ground for humor, rather like a bubbling spring that never runs dry.

One important way of soothing intertwined mental and bodily unrest is to produce meaning. We create meaning through chains of thought, with words strung together one after another like pearls. Yet humor appears at the very moment we realize that something that seems meaningful may not, after all, be quite so meaningful.

As the distance between meaning and meaninglessness grows, the contrast between them becomes more pronounced and cracks begin to appear. Just as the widening gap between appearance and reality creates tension, so does the growing distance between meaning and meaninglessness. Humor is one of the most common and socially acceptable ways of releasing this tension and anger.

Humor prevents tension-laden fault lines from producing major ruptures. At the same time, it helps repair the cracks that have already formed.

When we talk about humor, we often find ourselves dealing with two related words: witticism and joke. Humor itself is a broad and inclusive concept, referring to a form of expression, an approach, or an art intended to evoke laughter and amusement. The Turkish word espri comes from the French esprit, meaning spirit, intellect, or wit, which in turn is related to the Latin spiritus, meaning "breath" or "spirit." Joke, on the other hand, refers to an act or utterance intended to amuse or make another person laugh without causing offense.

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The foundational psychoanalytic work on humor is Sigmund Freud's Jokes and Their Relation to the Unconscious (1). Freud writes that every dreamer is, in a sense, an insufferable joker, compelled to be so because he is in difficulty and the direct path is closed to him. He also links the humorous character of unconscious processes closely to the theory of jokes and the comic (1).

For anyone whose psychic organization is not at the psychotic level, drive tension can only be discharged through indirect routes. Humor is among the most sublimatory of these routes. Both aggressive and libidinal drives can find partial, and sufficiently healthy, satisfaction through humor (1,2).

Psychoanalysis holds that the ego carries the indelible imprint of the id. It would therefore be difficult for psychoanalysis not to see humor as evidence of the way the unconscious quietly governs speech and language. The mechanisms that give rise to laughter and smiling have much in common with the forms of psychic work that take place in dreams, including condensation, displacement, and representation (1,2).

Through condensation, we can say a great deal with very few words. Through displacement, repressed aggressive and sexual contents can return in another guise. Through representation, we can alter the structure of words, create double meanings, play with language, and produce absurdities or contradictions (1).

Although humor makes use of psychic processes similar to those found in dreams, there are also clear differences between the two. Humor, for example, is an exquisite form of play that seeks pleasure without resorting to the regression seen in dreams. In dreams, the search for pleasure proceeds through regression toward hallucinatory wish fulfillment in order to avoid unpleasure (1,2).

There is another important difference. Dreams are intensely personal, while humor is one of the most social of all psychic activities (1).

According to Freud, jokes can be produced through techniques based on words or through techniques based on thoughts. Jokes that rely on verbal techniques are generally described as innocent. Those based on thought, by contrast, are tendentious (1).

Innocent jokes involve two people. Both parties find the joke funny and smile or laugh. Tendentious jokes involve three people, almost as if they were recreating an Oedipal triangle. There is the person who makes the joke, the person who becomes its object, and the person who finds it funny. We might also describe them as the one who strikes, the one who is struck, and the one who laughs. The joke-teller laughs through the laughter of those who find the joke amusing (1).

Unlike innocent jokes, then, tendentious jokes have a social dimension (1).

I mentioned earlier that espri can also mean wit. The word wit is borrowed from the Arabic nukta(t), derived from the root n-k-t. It is also related to the Arabic verb nakata, meaning "to prod" or "to touch with something pointed."

The fact that espri also carries the meanings of spirit and breath, while wit evokes the image of prodding someone with something sharp, seems to hint at humor's relationship with aggressive and libidinal drives. This connection with two sources of drive tension may help explain why, throughout history, those who practiced humor as an art have so often found themselves in trouble with authority and, at times, with society itself.

According to legend, Hacivat and Karagöz, whose actual historical existence has never been conclusively established, nearly paid with their lives for their humorous exchanges. Aristophanes (446–386 BCE) mercilessly satirized the politicians, philosophers, and war policies of his time. Socrates (469–399 BCE), though not a humorist in the conventional sense, made masterful use of irony and probing questioning. Nasreddin Hodja, although there is no reliable historical evidence that he was punished for doing so, appears in countless anecdotes making fun of judges, imams, statesmen, and other powerful figures. Voltaire (1694–1778) was imprisoned in the Bastille for writings in which humor and irony served as weapons of political and religious criticism. Dario Fo (1926–2016) used humor

to target the church, the police, capitalism, and government.

Looking at the history of our own country, we might add Turhan Selçuk, Rıfat Ilgaz, Aziz Nesin, Refik Halit Karay, and Hüseyin Rahmi Gürpınar to the list. It would be easy to extend it much further by drawing on more recent examples from both Turkey and the rest of the world.

Humor, then, is not simply a means of entertainment. It is a "safety valve" against power, perhaps one as old as human history itself.

Directly prodding the state, the statesman, the powerful, the wealthy, or the judge with something sharp may have dangerous consequences. Humor makes it possible to criticize from a certain distance. It also offers a way for aggressive impulses to be discharged collectively.

Humor, and tendentious jokes in particular, does not confront dominant power directly. Instead, it makes power look ridiculous. In doing so, it turns authority and those who represent it into the objects of the joke. Authority is no longer merely criticized; it is ridiculed (3).

Those who hold power are much more accustomed to direct opposition, to someone simply saying, "I am against you." They know how to fend off this familiar form of rebellion and how to deal with it. But when humor moves beyond criticism and starts making fun of authority, those in power are often caught off guard.

Humor is rather like a child who simply speaks the truth, giving full meaning to the Turkish saying "çocuktan al haberi", which might roughly be rendered as "hear the truth from the mouth of a child." Authority then resembles an adult bewildered by a child who has unexpectedly said out loud what everyone else knows but does not say.

Perhaps the best response would be to ignore it. Yet when authority resorts to the methods it knows best, it ends up exposing itself.

The truth has now been acknowledged by the very authority at which it was directed.

The remark has hit its mark. The stone has fallen neatly into its proper place, and suddenly the whole picture is there for everyone to see.

The holes and cracks in the wall built by political language, a wall often lacking in truth, have been repaired by the language of humor. Humor has come to our aid at exactly the right moment and in exactly the way it was needed.

There is something about this that recalls psychoanalytic writings on how and when an interpretation should be made in psychoanalysis and psychoanalytic psychotherapy. Perhaps this is why one might say, "A psychotherapy session without a joke hardly counts as a psychotherapy session at all."

An interpretation, like humor, must touch the patient's truth. It may even need to pass through that truth.

And since we are speaking of telling the truth, I would like to voice one final truth: our joy can be stolen.

But truth, like everything else, has more than one face. Our joy may be stolen. What the thieves cannot steal is our capacity to take it back from them, or the possibility of experiencing it again with renewed intensity.

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Suicide attempts in Bipolar Disorder: The roles of interpersonal sensitivity and perceived cognitive failures

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SUMMARY

Objective: Suicidal behavior remains a major clinical concern in bipolar disorder even during euthymic periods. This study aimed to compare interpersonal sensitivity, perceived cognitive failures, and affective symptoms in euthymic patients with bipolar disorder with and without a history of suicide attempts, and to examine whether perceived cognitive failures mediate the association between interpersonal sensitivity and suicidal behavior.

Method: This cross-sectional study included 68 euthymic patients with bipolar disorder. Participants were divided according to the presence of a history of suicide attempts. All participants were evaluated with the Hamilton Anxiety Rating Scale (HAM-A), Hamilton Depression Rating Scale (HAM-D), Interpersonal Sensitivity Measure (IPSM), and Cognitive Failures Questionnaire (CFQ). Group comparisons, correlation analyses, and bootstrap mediation analyses were performed.

Results: Patients with a history of suicide attempts had higher HAM-D ($p=.008$), CFQ ($p=.005$), and IPSM total scores ($p=.033$). The number of lifetime suicide attempts was positively correlated with perceived cognitive failures ($r=.38$, $p<.01$) and interpersonal sensitivity ($r=.26$, $p<.05$). In the bias-corrected bootstrap mediation model controlling for medical comorbidity, perceived cognitive failures statistically mediated the association between interpersonal sensitivity and the number of suicide attempts ($\beta=.138$, $p=.019$), whereas anxiety and depressive symptoms did not.

Discussion: Euthymic patients with bipolar disorder and a history of suicide attempts showed greater interpersonal sensitivity and subjective cognitive difficulties. Perceived cognitive failures may represent an important factor involved in the association between interpersonal sensitivity and suicidal behavior. Assessing interpersonal and cognitive factors in addition to mood symptoms may improve the clinical evaluation of suicide risk.

Key Words: Bipolar disorder, interpersonal sensitivity, perceived cognitive failures, suicide.

INTRODUCTION

Bipolar disorder (BD), which is characterized by recurrent episodes of mania and depression, is a persistent and chronic psychiatric disorder (1). Notwithstanding sociodemographic characteristics, the prevalence of this psychiatric disorder exceeds 1% of the global population. BD is among the foremost causes of disability in adults, resulting in cognitive and functional impairment, as well as an escalation in suicide rates (2,3). BD has been identified as a psychiatric disorder with one of the highest suicide rates (4). Throughout the course of the disease, the elevated risk of suicide, which has been observed to manifest particularly during depressive episodes but also during the euthymic phase, constitutes a pivotal challenge in its clinical management. Research has demonstrated that individuals diagnosed with BD have lifetime suicide attempt rates ranging from 25 to 50 %. This risk is estimated to be approximately 20–30 times higher than that in the general population (5–7). Therefore, elucidating the clinical and psychosocial factors contributing to suicidal behavior in BD is of great importance for both risk assessment and the development of preventive interventions for patients with BD.

Interpersonal sensitivity is a personality trait characterized by an individual's heightened sensitivity to others' thoughts, feelings, and behaviors. This sensitivity is further characterized by expectations

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of negative evaluation or rejection and intense emotional responses to these situations (8). This trait encompasses components such as social anxiety, low self-esteem, and interpersonal dependency and is closely associated with various psychiatric disorders, particularly mood disorders (9). According to the extant literature, increased interpersonal sensitivity has been observed during both depressive and euthymic periods in patients with BD. This heightened sensitivity has been demonstrated to exert a detrimental effect on patients' social functioning and potentially contribute to an elevated suicide risk (8,10,11). Furthermore, research has indicated a correlation between interpersonal sensitivity, childhood trauma, and non-suicidal self-harm behaviors (12,13). These findings suggest that interpersonal sensitivity in BD may not merely be a concomitant trait but also a potentially critical factor for understanding and predicting suicidal behavior.

Cognitive failures are daily manifestations that include distraction, forgetfulness, lack of planning, and incorrect task performance. These failures reflect an individual's subjective perception of their cognitive functioning (14). These errors have been observed not only in elderly individuals or those with neurological diseases but also in various psychiatric conditions, including mood disorders (15). Cognitive impairments can be observed at all stages of BD (14). Contrary to the findings of certain studies (16,17), some studies have reported an association between cognitive impairments and heightened suicidal ideation or attempts in patients with BD (18,19). Another study revealed that favorable cognitive function is linked to a protective role against suicidal behavior in patients with BD (7). These findings suggest that cognitive impairments in BD may be an important component that affects not only functionality but also the risk of suicide. A meta-analysis of 41 studies underscored the necessity for additional research on the predictive role of cognitive functions in suicidal behavior in BD (20).

The extant literature suggests that both interpersonal sensitivity and cognitive difficulties may be associated with suicide risk in patients with BD. However, recent evidence also indicates that these associations are not uniform across studies. A recent systematic review and meta-analysis reported mixed findings regarding the relationship between cognitive functioning and suicidal ideation or suicide attempts in BD, with associations varying according to the cognitive domain assessed and the type of suicidal outcome examined (20). This inconsistency may partly reflect methodological differences, including the use of objective neuropsychological tests versus self-reported or perceived cognitive difficulties. Similarly, recent work has highlighted the clinical relevance of interpersonal sensitivity in mood disorders, but most studies have examined interpersonal sensitivity as an isolated psychosocial risk-related factor rather than in combination with cognitive complaints (8,13). Moreover, a recent scoping review of psychosocial factors associated with suicidality in BD emphasized that this literature often lacks explicit theory-driven models linking psychosocial factors to suicidal outcomes (21). In this context, the present study aimed to examine the potential statistical mediating role of perceived cognitive failures in the association between interpersonal sensitivity and suicide attempts in euthymic patients with BD. By evaluating interpersonal sensitivity, perceived cognitive failures, and suicidal behavior within a single model, this study seeks to provide an exploratory contribution to understanding psychosocial and perceived cognitive correlates of suicide risk in BD.

METHOD

Participants and Procedure

The study sample comprised 68 patients diagnosed with BD at the Necmettin Erbakan University Faculty of Medicine Psychiatry Clinic. The inclusion criteria for patients were as follows: having a diagnosis of BD according to the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5), being in the euthymic phase according to clinical evaluation and scales, and being aged between 18 and 60. The exclusion criteria for the study were as follows: Young Mania Rating Scale (YMRS) score >7, Hamilton Depression Rating Scale (HAM-D) score >7, presence of mental retardation, schizophrenia, or schizoaffective disorder, presence of serious uncontrolled physical illness or medication use that could affect cognitive function, history of head trauma and organic brain syn-

drome, and presence of alcohol and substance use disorders. All participants were assessed with the Young Mania Rating Scale and Hamilton Depression Rating Scale to confirm euthymia, and with the Hamilton Anxiety Rating Scale, Interpersonal Sensitivity Measure, and Cognitive Failures Questionnaire for clinical evaluation. Patients were divided into two groups based on the presence of suicide attempts: those with suicide attempts (SA) (n=23, 33.8%) and those without suicide attempts (NSA) (n=45, 66.2%). The presence and number of lifetime suicide attempts were determined through face-to-face clinical interviews with the patients and by reviewing detailed past medical records. For the group comparisons, suicide attempt was used as a dichotomous variable indicating the presence or absence of a lifetime suicide attempt. For the correlation and mediation analyses, the number of lifetime suicide attempts was used as a continuous outcome variable. Sociodemographic and psychometric variables were compared between the two groups. The sample size was calculated using G*Power 3.1.9.2 software (α=0.05, power=0.80, effect size=0.65). The analysis indicated that at least 64 participants should be included in this study.

Data Collection Tools

Sociodemographic data form: This form was designed to gather demographic information, including age, gender, education level, marital status, income level, and living status.

Young Mania Rating Scale (YMRS): This scale was developed by Young et al. to measure the severity of mania (22). The scale comprises 11 items, each with five stages. The interviewer utilizes a structured assessment tool to evaluate the severity of manic symptoms, focusing on the experiences of the previous week. The Turkish validity and reliability study yielded an internal consistency coefficient of 0.79 (23). The YMRS was used to ascertain that the BD patients included in the present study were in the remission phase.

Hamilton Depression Rating Scale (HAM-D): This scale is a widely used clinical tool for assessing the severity of depressive symptoms (24). This 17-item scale assesses depressive symptoms, including mood, sleep, appetite, guilt, libido, and loss of interest. Items are scored on a 0-4 or 0-2 scale. The scale's score range extends from 0 to 52, with higher scores denoting an elevated risk of depression. The evaluation can be conducted by an interviewer with psychopathology expertise. The adaptation of the scale to the Turkish population has demonstrated its high reliability and validity (25).

Hamilton Anxiety Rating Scale (HAM-A): This scale, which is a psychometric instrument that assesses the intensity of anxiety symptoms experienced by individuals, is a five-point Likert-type scale (26). The scale serves as an indicator of the severity of anxiety experienced by participants. Each item is assigned a score ranging from 0 to 4, with a maximum total score of 56, and no specific cutoff score has been established. The administration of this scale is best suited for interviewers with expertise in psychopathology, and it can be used for comparative studies. The Turkish adaptation of the scale has been found to have high validity and reliability (27).

Interpersonal Sensitivity Measure (IPSM): This scale was developed by Boyce and Parker (1989) to assess individuals' levels of sensitivity in interpersonal relationships (28). The scale was adapted into Turkish by Doğan and Sapmaz (2012) and subsequently reduced to 30 items. The Turkish version employs a five-point Likert scale (1 = "not at all appropriate," 5 = "definitely appropriate") and consists of three subscales: "Interpersonal Worry and Dependency" (IPSM-IWD), "Lack of Social Self-Confidence" (IPSM-LSSC), and "Non-Assertive Behaviors" (IPSM-NAB). Individuals who exhibit elevated scores on this scale demonstrate high interpersonal sensitivity. The internal consistency coefficient of the Turkish version of the scale was reported to be .88. The Cronbach's alpha values for the subscales were .90, .76, and .72, respectively (29).

Cognitive Failure Questionnaire (CFQ): The CFQ was developed by Broadbent et al. in 1982 (30). It is a 25-item self-report scale that assesses cognitive impairments in attention, memory, and motor function in daily life. The 4-point Likert-type

Turkish version developed by Yapıcı Eser et al. (2020) underwent a comprehensive examination of its validity and reliability. The instrument demonstrated high internal consistency reliability, as evidenced by a Cronbach's alpha coefficient of 0.91. In the adapted form for our country, the maximum score is 75, with higher scores indicating greater perceived cognitive impairment (31).

Statistical Analysis

All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) software (IBM, Statistics for Windows, Version 22) and Jamovi (v2.6.44) software for mediation analyses. The distribution characteristics of the data were evaluated using the Kolmogorov–Smirnov and Shapiro–Wilk tests. For continuous variables that exhibited a normal distribution, the independent samples t-test was employed, while the Mann–Whitney U test was utilized for non-normally distributed variables. The chi-square (χ^2) test was used to analyze categorical data. A significance level of $p<.05$ was established as the threshold for determining statistical differences between the groups. Spearman's correlation analysis was used to assess the relationships between variables These correlation analyses were considered exploratory, and no formal correction for multiple testing was applied. Therefore, the correlation findings were interpreted cautiously as hypothesis-generating associations. Furthermore, mediation analysis using a general linear model was conducted to assess the potential statistical mediating role of perceived cognitive failures in the association between interpersonal sensitivity and the number of suicide attempts. Anxiety and depressive symptoms were also included in the model, and medical comorbidity was entered as a covariate because it differed significantly between the suicide-attempt and non-attempt groups. To improve the robustness of the indirect-effect estimation in this modest clinical sample, the mediation analysis was repeated using 5000 bias-corrected bootstrap samples. Standardized β coefficients, standard errors (SE), 95% confidence intervals (CI), z-values, and p-values were reported. The indirect, direct, and total associations were evaluated separately, with the significance threshold set at $p<.05$.

RESULTS

Sociodemographic and Clinical Characteristics

The analysis revealed no statistically significant differences between participants with a history of suicide attempts (SA) and those without (NSA) in terms of demographic and clinical variables. These variables included age, gender, marital status, education level, income, place of residence, type of bipolar disorder, and tobacco use ($p>.05$). However, a notable finding was the significantly higher rate of medical comorbidity observed in the SA group (39.1% vs. 15.6%; $p=.03$). A detailed comparison of the sociodemographic characteristics of the groups is presented in Table 1.

Group Differences Among Psychometric Tests

A comparison of the scales used to assess the severity of psychiatric symptoms revealed that the SA group had significantly higher levels of depression ($p=.008$). Additionally, individuals in the SA group had significantly higher perceived cognitive failure scores ($p=.005$). The SA group had significantly higher interpersonal sensitivity total scores ($p=.033$), IPSM-IWD subscale scores ($p=.024$), and IPSM-LSSC subscale scores ($p=.033$) than the NSA group did. However, no significant difference was found between the groups in the IPSM-NAB subscale ($p=.515$). Table 2 presents a comparison of the psychometric test results.

Correlations Between Interpersonal Sensitivity, Perceived Cognitive Failures and Suicide Attempts

A positive relationship was found between the number of suicide attempts and CFQ scores ($r=.38$, $p<.01$) in the correlation analysis. Similarly, the IPSM total score was significantly correlated with the number of suicide attempts ($r=.26$, $p<.05$). A significant positive correlation was also found between the IPSM-LSSC and the number of suicide attempts ($r=.27$, $p<.05$). Furthermore, CFQ scores were positively correlated with the IPSM total scores ($r=.41$, $p<.01$), HAM-D scores ($r=.27$, $p<.05$), HAM-A scores ($r=.35$, $p<.01$), IPSM-IWD scores ($r=.34$, $p<.01$),

Table 1. Comparison of Sociodemographic and Clinical Characteristics Between Patients with and without Suicide Attempt (Mean ± SD).

		Group			
		NSA (n=45)	SA (n=23)	t / Z / x^2	p
Age [a]		40.71 ± 12.35	43.91 ± 13.2	-0.988	.327
Years of Education (years) (median)		10.71 ± 4.48 (12)	10.13 ± 4.9 (12)	-0.353	.724
Gender	Female	24 (53.3%)	13 (56.5%)	0.062	.803
	Male	21 (46.7%)	10 (43.5%)		
Marital Status	Married	27 (60%)	14 (60.9%)	0.005	.945
	Single	18 (40%)	9 (39.1%)		
Income (TL) (median)		13791±18592 (0)	14934±24391 (0)	-0.485	.628
Occupational Status	Employed	19 (42.2%)	9 (39.1%)	4.034	.133
	Unemployed due to disorder	0 (0%)	2 (8.7%)		
	Unemployed due to other reason	26 (57.8%)	12 (52.2%)		
Living with	Alone	1 (2.2%)	0 (0%)	0.519	.471
	Family	44 (97.8%)	23 (100%)		
Place of Residency	Urban	44 (97.8%)	21 (91.3%)	1.512	.219
	Rural	1 (2.2%)	2 (8.7%)		
BMI [a]		29.11 ± 6.03	28.24 ± 4.24	0.615	.541
Bipolar Type	Type I	40 (88.9%)	20 (87%)	0.055	.815
	Type II	5 (11.1%)	3 (13%)		
Tobacco Use	Yes	16 (35.6%)	10 (43.5%)	0.405	.525
Seasonality	Yes	21 (46.7%)	10 (43.5%)	0.062	.803
Presence of Psychotic Symptoms in Episodes	Yes	19 (42.2%)	13 (59.1%)	1.685	.194
	Depression	14 (31.1%)	10 (43.5%)	1.125	.570
Predominant Polarity	Mania	23 (51.1%)	9 (39.1%)		
	Equal	8 (17.8)	4 (17.4%)		
Rapid Cycling	Yes	7 (15.6%)	1 (4.3%)	1.842	.175
History of ECT	Yes	4 (8.9%)	0 (0%)	2.172	.141
Medical Comorbidity	Yes	7 (15.6%)	9 (39.1%)	4.701	.030
Family History of Psychiatric Disorder	Yes	19 (42.2%)	65.2 (15%)	3.219	.073
Duration of Disorder (years) (median)		14.49 ± 9.8 (14)	13.89 ± 11.17 (10)	-0.377	.706
Age at onset of Disorder (median)		25.64 ± 10.61(22)	30.65 ± 11.98 (30)	-1.850	.064
Number of Suicide Attempts (median)			1.74 ± 0.75 (2)		

P≤0.05, Chi-square and Mann-Whitney U tests were carried out. [a]Independent Sample t-test was applied due to normally distributed data. SA: Suicide Attempt, NSA: Non-Suicide Attempt, BMI: Body Mass Index, ECT: Electro-Convulsive Therapy, TL: Turkish Liras, SD: standard deviation.

and IPSM-LSSC scores (r=.35, p<.01). The data related to the correlation analyses are presented in Table 3.

The Mediating Role of Perceived Cognitive Failures in the Relationship Between Interpersonal Sensitivity and Number of Suicide Attempts

In the mediation analysis, the number of suicide attempts was used as the outcome variable, and medical comorbidity was included as a covariate. The model was estimated using 5000 bias-corrected bootstrap samples. The indirect association between IPSM and the number of suicide attempts through CFQ was statistically significant (Estimate=0.00812, SE=0.00345, 95% BC bootstrap CI=0.00195–0.01837, β=0.1378, p=.019). IPSM was significantly associated with CFQ scores (Estimate=0.33050, SE=0.10196, β=0.3596, p=.001), and CFQ was significantly associated with the number of suicide attempts (Estimate=0.02456, SE=0.00717, β=0.3832, p<.001). In contrast, the indirect associations through HAM-A and HAM-D were not significant. The total association between IPSM and the number of suicide attempts was significant (Estimate=0.01595, SE=0.00687, β=0.2708, p=.020), whereas the direct association did not reach statistical significance (Estimate=0.00865,

Table 2. Comparison of Psychometric Tests of the Groups (Mean – SD).

	Group			
	NSA (n=45)	**SA (n=23)**	**t / Z**	**p**
HAM-A (median)	4.53 ± 5.22 (2)	4.22 ± 4.57 (3)	-0.334	.739
HAM-D (median)	1.27 ± 1.7 (0)	2.17 ± 1.37 (2)	-2.663	**.008**
YMRS (median)	0.18 ± 0.61 (0)	0.35 ± 0.78 (0)	-1.398	.162
CFQ (median)	15.73 ± 10.34 (15)	27.52 ± 18.36 (21)	-2.796	**.005**
IPSM Total [a]	68.29 ± 12.73	78.22 ± 19.34	-2.228	**.033**
IPSM-IWD [a]	34.31 ± 8.84	41.04 ± 15.25	-2.307	**.024**
IPSM-NAB [a]	21.71 ± 7.27	22.91 ± 6.95	-0.654	.515
IPSM-LSSC (median)	12.64 ± 4.3 (12)	14.7 ± 3.75 (14)	-2.137	**.033**

p ≤ 0.05, Chi-square and Mann-Whitney U tests were carried out. [a]Independent Sample t-test was applied due to normally distributed data. SA: Suicide Attempt, NSA: Non-Suicide Attempt, HAM-A: Hamilton Anxiety Scale, HAM-D: Hamilton Depression Scale, YMRS: Young Mania Rating Scale, CFQ: Cognitive Failure Questionnaire, IPSM: Interpersonal Sensitivity Measure, IPSM-IWD: Interpersonal Sensitivity Measure-Interpersonal Worry and Dependency, IPSM-LSSC: Interpersonal Sensitivity Measure-Lack of Social Self-Confidence, IPSM-NAB: Interpersonal Sensitivity Measure-Non-Assertive Behaviors, SD: standard deviation.

Table 3. Correlation Between Psychometric Tests and Sociodemographic Features.

(n=68)	Age	Years of Education	Duration of Disorder	Number of Suicide Attempts	CFQ	IPSM	HAM-A	HAM-D	IPSM-IWD	IPSM-LSSC	IPSM-NAB
Age	1										
Years of Education	-.37**	1									
Duration of Disorder	.52**	-.21	1								
Number of Suicide Attempts	.06	-.05	-.08	1							
CFQ	-.16	-.02	-.06	.38**	1						
IPSM	-.04	-.14	-.05	.26*	.41**	1					
HAM-A	-.16	-.08	-.12	.04	.35**	.18	1				
HAM-D	-.15	.05	-.07	.33**	.27*	.06	.47**	1			
IPSM-IWD	-.05	-.09	-.13	.23	.34**	.92**	.15	.08	1		
IPSM-LSSC	.05	.02	.01	.27*	.35**	.12	.18	.16	-.02	1	
IPSM-NAB	-.02	-.10	-.02	.12	.14	.67**	.10	-.10	.52**	-.22	1

*p<0.05, **p<0.01, Spearman's correlation test was carried out. HAM-A: Hamilton Anxiety Scale, HAM-D: Hamilton Depression Scale, CFQ: Cognitive Failure Questionnaire, IPSM: Interpersonal Sensitivity Measure, IPSM-IWD: Interpersonal Sensitivity Measure-Interpersonal Worry and Dependency, IPSM-LSSC: Interpersonal Sensitivity Measure -Lack of Social Self-Confidence, IPSM-NAB: Interpersonal Sensitivity Measure-Non-Assertive Behaviors.

SE=0.00645, β=0.1469, p=.180). Medical comorbidity did not show a significant direct or total association with the number of suicide attempts. These findings are consistent with a statistical mediating pathway in which higher interpersonal sensitivity is associated with more frequent perceived cognitive failures, which in turn are associated with a higher number of suicide attempts. The revised pathway diagram and the updated mediation results are presented in Figure 1 and Table 4.

DISCUSSION

Patients with euthymic BD and a history of suicide attempts had higher interpersonal sensitivity, perceived cognitive failure scores, and HAM-D scores than those without such a history. Although all participants met the predefined clinical and scale-based criteria for euthymia, the higher HAM-D scores in the SA group should not be interpreted as indicating an acute depressive episode; rather, they may reflect residual or subthreshold depressive vulnerability. Such residual depressive symptoms may still be clinically relevant for suicide risk in BD, even during euthymic periods. The number of suicide attempts was positively correlated with interpersonal sensitivity and perceived cognitive failures. The mediation analysis showed that perceived cognitive failures, but not anxiety or depressive symptoms, statistically mediated the association between interpersonal sensitivity and the number of suicide attempts after accounting for medical comorbidity. Consistent with the clinical relevance of residual depressive symptoms, HAM-D was significantly associated with the number of suicide attempts in the revised model; however, the indirect pathway from interpersonal sensitivity to the number of suicide attempts through HAM-D was not significant. Therefore, the main statistical indirect association in the present model appeared to be more closely related to perceived cognitive failures than to depressive symptoms. However, because of the cross-sectional design, this finding

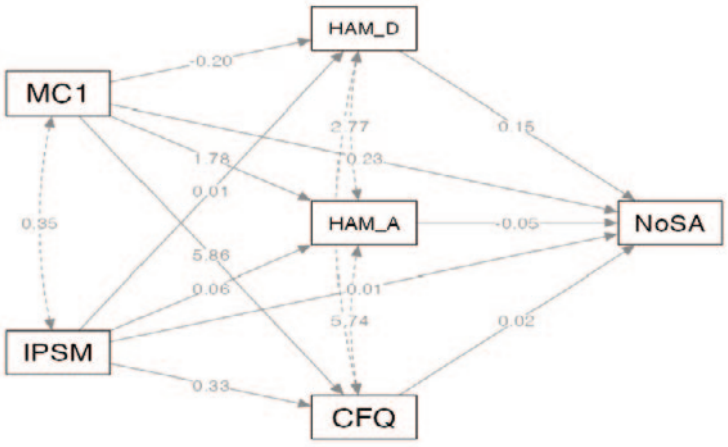

Figure 1. Path Diagram of the Mediation Model.

Standardized path coefficients are presented. The model tested the statistical mediating role of perceived cognitive failures in the association between interpersonal sensitivity and the number of suicide attempts, with medical comorbidity included as a covariate. IPSM: Interpersonal Sensitivity Measure; CFQ: Cognitive Failures Questionnaire; HAM_A: Hamilton Anxiety Rating Scale; HAM_D: Hamilton Depression Rating Scale; NoSA: Number of Suicide Attempts; MC1: presence of medical comorbidity.

Table 4. Indirect and Total Effects of Perceived Cognitive Failures, Anxiety, Depression, and Medical Comorbidity on the Relationship Between Interpersonal Sensitivity and Number of Suicide Attempts

Type	Effect	Estimate	SE	95% C.I.		□	z	p
				Lower	Upper			
Indirect	IPSM => CFQ => NoSA	0.00812	0.00345	0.00195	0.01837	0.1378	2.354	**.019**
	IPSM => HAM-A => NoSA	-0.00272	0.00213	-0.00817	-1.13e-4	-0.0463	-1.279	.201
	IPSM => HAM-D => NoSA	0.00191	0.00201	-8.77e-4	0.00814	0.0324	0.949	.343
	MC1 => CFQ => NoSA	0.14381	0.10199	-0.05180	0.62288	0.0658	1.410	.159
	MC1 => HAM-A => NoSA	-0.08516	0.07535	-0.32133	0.04432	-0.0390	-1.130	.258
	MC1 => HAM-D => NoSA	-0.02999	0.06956	-0.20736	0.09664	-0.0137	-0.431	.666
Component	IPSM => CFQ	0.33050	0.10196	0.00420	0.59340	0.3596	3.242	**.001**
	CFQ => NoSA	0.02456	0.00717	0.00540	0.04126	0.3832	3.425	**<.001**
	IPSM => HAM-A	0.05706	0.03696	6.86e-4	0.12424	0.1818	1.544	.123
	HAM-A => NoSA	-0.04774	0.02088	-0.09406	-0.00479	-0.2544	-2.287	**.022**
	IPSM => HAM-D	0.01290	0.01246	-0.01070	0.03450	0.1247	1.036	.300
	HAM-D => NoSA	0.14788	0.06257	0.02584	0.30905	0.2599	2.364	**.018**
	MC1 => CFQ	5.85643	3.78493	-3.80013	17.64228	0.1717	1.547	.122
	MC1 => HAM-A	1.78396	1.37204	-1.24245	5.65737	0.1531	1.300	.194
	MC1 => HAM-D	-0.20282	0.46245	-1.07157	0.70256	-0.0528	-0.439	.661
Direct	IPSM => NoSA	0.00865	0.00645	-0.00339	0.02186	0.1469	1.342	.180
	MC1 => NoSA	0.23379	0.22870	-0.19747	0.67988	0.1069	1.022	.307
Total	IPSM => NoSA	0.01595	0.00687	0.00139	0.03035	0.2708	2.322	**.020**
	MC1 => NoSA	0.26245	0.25495	-0.24571	0.82888	0.1201	1.029	.303

Note. Confidence intervals were computed using the bias-corrected bootstrap method with 5000 bootstrap samples. Betas are completely standardized effect sizes. HAM-A: Hamilton Anxiety Rating Scale; HAM-D: Hamilton Depression Rating Scale; CFQ: Cognitive Failures Questionnaire; IPSM: Interpersonal Sensitivity Measure; MC1: Medical comorbidity contrast indicator (1 = presence, 0 = absence); NoSA: Number of Suicide Attempts; C.I.: Confidence Interval; SE: Standard Error.

should be interpreted as an indirect statistical association rather than evidence of temporal or causal mediation. These findings suggest that suicide risk assessment in BD should address interpersonal and cognitive factors in addition to mood symptoms.

Interpersonal sensitivity has been associated with suicidal ideation and self-mutilative behaviors in non-clinical populations (32–34). Community-based studies have demonstrated a positive correlation between increased interpersonal sensitivity and the prevalence of self-harming behavior (12). Increased interpersonal sensitivity has also been reported in psychiatric disorders. Hidese et al. (2022) demonstrated positive correlations between general psychopathology and interpersonal sensitivity levels in schizophrenia, BD, and major depressive disorder (35). In a related study, Polskaya and colleagues (2023) reported an increase in interpersonal sensitivity among adolescent girls with suicidal tendencies and eating disorders (36). A more recent study underscored the significance of interpersonal sensitivity in patients with mood disorders (8). Research in this area has revealed heightened interpersonal sensitivity in patients with suicidal tendencies and treatment-resistant depression (37,38). Regardless of the type of mood disorder, interpersonal sensitivity has been found to be associated with atypical depression, childhood traumatic experiences, and increased impulsivity (9,39,40). However, some authors have suggested that BD is associated with higher interpersonal sensitivity among mood disorders (11,13). Furthermore, Kwon et al. (2024) proposed that heightened interpersonal sensitivity in patients with BD is associated with low self-esteem (13). The extant literature provides substantial evidence that interpersonal sensitivity is associated with suicidal thoughts, behaviors, and BD. However, there is a paucity of literature on interpersonal relationships in relation to suicidal behavior in patients with BD. In the present study, we sought to examine the relationship between interpersonal sensitivity and suicide attempts in patients with BD. The findings of the present study indicate that individuals with BD who have attempted suicide exhibit heightened levels of interpersonal sensitivity. Furthermore, these sensitivity levels were positively correlated with the number of suicide attempts. Most studies that have reported an increase in interpersonal sensitivity in patients diagnosed with bipolar disorder have been conducted on individuals experiencing depressive episodes. The present study found that interpersonal sensitivity persists even during euthymic periods. Components of interpersonal sensitivity, such as interpersonal worry/dependency and lack of social self-confidence, were also higher in patients with a history of suicide attempt. Recent network-based evidence indicates that interpersonal sensitivity is not a unitary construct but includes interrelated components such as interpersonal worry, dependency, and social self-confidence (8). This is consistent with our finding that IPSM total, IPSM-IWD, and IPSM-LSSC scores were higher in patients with a history of suicide attempt, whereas the non-assertive behavior subscale did not differ significantly between groups. Thus, the present findings suggest that the suicide-related relevance of interpersonal sensitivity in BD may be more

closely related to interpersonal worry/dependency and lack of social self-confidence than to non-assertive behaviors per se.

It has been established that cognitive impairments are more prevalent in all stages of BD than in healthy controls (14). However, contradictory results have been reported regarding the relationship between suicidal ideation or attempts and cognitive function in BD (20). While some researchers have reported that cognitive functions are not impaired in BD patients with suicidal thoughts or attempts compared to those without (16,17), others have suggested that cognitive functions are negatively affected in those with suicidal attempts (15,18,19). It has been posited that the variability in outcomes may be attributable to the assessment of disparate cognitive domains across different studies (20). Li et al. (2025) asserted that good cognitive function in patients with BD serves as a protective factor against suicidal thoughts (7). In another study with a relatively large sample size, non-suicidal self-harm behaviors in BD patients are also associated with cognitive dysfunction (41). A study evaluating subjective cognitive functions reached a similar conclusion. This study claimed that subjective cognitive functions were more impaired in patients with BD than in healthy individuals and that these impairments could even predict suicidal thoughts (14). In the present study, perceived cognitive failure scores were found to be elevated in patients who had attempted suicide, and these scores exhibited a robust positive correlation with the number of suicide attempts. Our findings align with studies emphasizing the clinical relevance of cognitive difficulties in self-harm-related outcomes in BD. However, recent evidence also suggests that the relationship between cognition and suicidality in BD is complex rather than linear. Le et al. (2024) reported mixed findings across cognitive domains in BD, indicating that cognitive functioning may relate differently to suicidal ideation and suicide attempts depending on the domain assessed (20). In addition, recent studies have shown that cognitive difficulties may be relevant to self-harm-related outcomes in BD, but these findings are not fully consistent across samples and assessment methods (41). Importantly, the CFQ used in the present study measures perceived cognitive failures in daily life rather than objective neuropsychological performance. Therefore, our findings should be interpreted as supporting the clinical relevance of perceived cognitive difficulties in relation to suicidal behavior, rather than as evidence that objective cognitive impairment directly contributes to suicide attempts. Furthermore, the positive association between perceived cognitive failures and interpersonal sensitivity suggests that these two domains may jointly characterize a subgroup of patients with greater suicide-related vulnerability.

A salient finding of this study is that perceived cognitive failures statistically mediated the association between interpersonal sensitivity and the number of suicide attempts in the mediation model, which included anxiety and depressive symptoms and controlled for medical comorbidity. This result suggests that when assessing suicide risk in BD, it may be useful to consider interpersonal sensitivity and perceived cognitive difficulties together rather than as entirely separate domains. Existing research has not sufficiently addressed how interpersonal sensitivity and perceived cognitive difficulties may be statistically linked to suicidal behavior within the same model. Prior research has largely treated these variables as independent risk-related factors (14,21). The main contribution of the present study is that it integrates two previously separate lines of research: the psychosocial literature on interpersonal sensitivity and the cognitive literature on perceived cognitive difficulties in BD. Rather than treating these variables as independent risk markers, the present model suggests that perceived cognitive failures may help characterize the statistical link between interpersonal sensitivity and the number of suicide attempts. Nevertheless, given the cross-sectional design and modest sample size, this interpretation should be regarded as exploratory and hypothesis-generating.

Individuals with high interpersonal sensitivity have been shown to experience heightened arousal, negative evaluation expectations, and perceptions of rejection in social interactions (8). This phenomenon may lead to an escalated state of vigilance, characterized by incessant monitoring of potential threats and an accentuated focus on emotionally laden information during cognitive processes such as attention and memory. Emotional overload, characterized by the excessive accumulation of distressing emotions, can lead to a marked depletion of cognitive resources. This, in turn, has

the potential to diminish an individual's sense of self-efficacy or the belief in one's ability to execute a task or achieve a goal. A decline in self-efficacy and impaired problem-solving capacity can reinforce feelings of helplessness and hopelessness in stressful life events, triggering suicidal tendencies. In this context, cognitive failures may be considered a perceived cognitive correlate that helps explain the association between interpersonal sensitivity and suicidal behavior. However, the present cross-sectional data cannot exactly determine whether interpersonal sensitivity precedes cognitive failures or whether these factors jointly reflect broader vulnerability processes related to suicide risk. These findings indicate that suicide risk assessment in patients with BD should not be limited to mood symptoms. The persistence of elevated interpersonal sensitivity and cognitive error frequency during the euthymic period underscores the necessity of incorporating these factors into the standard clinical assessment protocol. In individuals with high interpersonal sensitivity, perceptions of rejection, social withdrawal, and feelings of helplessness may be more pronounced, triggering mechanisms that increase the risk of suicide. Similarly, in patients who experience frequent cognitive failures, weaknesses in daily living skills, and problem-solving abilities may reduce their capacity to cope with stress, thereby increasing their susceptibility to suicidal behavior. Consequently, interpersonal and perceived cognitive domains may represent clinically relevant targets for future preventive intervention studies in individuals with BD, although their efficacy in reducing suicide risk should be tested in prospective interventional research. From a clinical perspective, patients with high interpersonal sensitivity and frequent perceived cognitive failures may benefit from more detailed assessment and psychosocial support focusing on interpersonal interpretation, coping with perceived rejection, problem-solving skills, cognitive organization strategies, and relapse/risk monitoring. These implications should be considered as hypothesis-generating clinical suggestions rather than evidence of established psychotherapeutic effects.

This study has some limitations. First, the modest sample size obtained from a single center limits the generalizability of the findings. In particular, the relatively small number of patients with a history of suicide attempt may reduce the statistical stability of the mediation model and increase the risk of overfitting. Although the mediation analysis was repeated using 5000 bias-corrected bootstrap samples and medical comorbidity was included as a covariate, the findings should still be regarded as exploratory and require replication in larger multicenter samples. The second limitation of this study is its cross-sectional design, which precludes conclusions about temporal ordering or causality. Therefore, the mediation findings should be interpreted as statistical indirect associations rather than evidence that interpersonal sensitivity causes cognitive failures or suicidal behavior. Another limitation is the retrospective assessment of lifetime suicide-attempt history. Although suicide attempts were evaluated using both face-to-face clinical interviews and detailed medical record review, recall bias and incomplete documentation cannot be entirely excluded. In addition, several clinically relevant variables, including medication type, psychotropic treatment characteristics, number of previous mood episodes, hospitalization history, illness severity indices, and comorbid anxiety disorders, were not included in the statistical analyses. These factors may influence both perceived cognitive difficulties and suicidal behavior in patients with BD. Furthermore, the correlation analyses were exploratory and were not corrected for multiple comparisons; therefore, these bivariate findings should be interpreted cautiously. Finally, cognitive failures were assessed using the CFQ, which is a self-report measure of perceived cognitive difficulties in daily life rather than an objective neuropsychological assessment. This may introduce response bias and limits the extent to which the findings can be generalized to objective cognitive impairment. Future studies with larger samples, longitudinal designs, more comprehensive clinical characterization, and both subjective and objective cognitive assessments should examine whether the observed associations remain significant after controlling for illness- and treatment-related variables. A notable strength of this study is its examination of the relationships between interpersonal sensitivity, perceived cognitive failures, and suicidal behavior in euthymic patients with BD within a unified model. The application of mediation analysis to statistically demonstrate the mediating role of perceived cognitive errors in the relationship between interpersonal sensitivity and suicidal behavior provides a unique contribution to the literature. Furthermore, the observation of

these relationships even during the euthymic period, when mood symptoms are minimal, suggests that risk factors are not limited to the acute period.

Perceived cognitive failures significantly mediated the association between interpersonal sensitivity and suicidal behavior in euthymic patients with BD. These findings suggest that suicide risk in BD may be influenced not only by mood symptoms but also by interpersonal sensitivity and subjective cognitive difficulties. Assessing these domains may improve risk evaluation and help inform targeted preventive interventions. Further longitudinal studies with larger multicenter samples are needed.

Ethics Approval and Consent to Participate: The present study was conducted in accordance with the Declaration of Helsinki for investigations involving humans and was approved by the local ethics committee on human research (IRB Date/Number: 6.9.2024 / 2024.5137). Written informed consent was obtained from all patients prior to participation.

Consent for Publication: Not applicable

Data Availability Statement: Data supporting the findings are available from the corresponding author upon reasonable request.

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Transdiagnostic family-focused therapy for youth with recent-onset bipolar disorder and schizophrenia: A pilot study in Türkiye

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SUMMARY

Objective: Bipolar disorder and schizophrenia are severe disorders that often begin in adolescence and affect both youth and caregivers. Although interest in transdiagnostic psychosocial approaches has grown in recent years, few studies have examined the same structured family-based intervention across these diagnostic groups, particularly in non-Western settings. This single-arm pilot study evaluated the preliminary clinical and family-related outcomes of a brief, manual-based Family-Focused Therapy (FFT) program delivered in routine outpatient youth mental health care.

Method: Adolescents aged 15-19 years diagnosed with bipolar disorder (n=10) or schizophrenia (n=10), along with their caregivers, received a 9-session FFT program. Overall, 87% completed the intervention and were included in analyses. Primary outcomes included depressive symptoms, psychosocial functioning, and illness insight. Secondary outcomes included caregiver depressive symptoms, caregiver burden, and family functioning. Pre-post changes and exploratory group x time patterns were examined using within-group tests and repeated-measures models.

Results: Depressive symptom severity decreased and psychosocial functioning improved in both diagnostic groups, with substantial within-group standardized mean change effects. Illness insight also showed pre-post improvement. Caregiver burden decreased in both groups, whereas caregiver depressive symptoms decreased only among parents of adolescents with bipolar disorder. Family functioning changes differed across diagnostic groups and informants.

Discussion: A brief transdiagnostic FFT program was associated with pre-post improvements across multiple clinical and family-related domains. Although conclusions are limited by the single-arm design, findings provide preliminary evidence suggesting the potential clinical relevance of FFT in routine outpatient care and warrant larger controlled trials of transdiagnostic family interventions in diverse clinical contexts.

Key Words: Family-focused therapy, bipolar disorder, schizophrenia, transdiagnostic approach, early intervention, adolescents

INTRODUCTION

Bipolar disorder and schizophrenia are among the most severe psychiatric conditions, characterized by a chronic course and significant functional impairment. Although traditionally classified as distinct diagnostic entities, these disorders share substantial overlap in symptom expression, genetic vulnerability, and neurobiological mechanisms (1). This overlap is already evident during the prodromal stages of illness, when young people may present with subthreshold mood, anxiety, sleep, and psychotic-like symptoms together with emerging functional decline (2–5). Symptom co-occurrence frequently persists following diagnosis: anxiety and depressive symptoms commonly accompany psychotic disorders, while psychotic features may also emerge during mood episodes (6,7). This clinical overlap complicates early differentiation of illness trajectories and highlights the limitations of rigid categorical distinctions between mood and psychotic disorders (8).

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The overlap between bipolar disorder and schizophrenia is also reflected in the psychosocial challenges experienced by families. Caregivers of individuals with severe mental illness frequently report elevated stress, depressive symptoms, and subjective burden, particularly during the early stages of illness when uncertainty regarding diagnosis, prognosis, and treatment is high (9–11). A recent systematic review indicates that caregivers of individuals with bipolar disorder and schizophrenia spectrum disorders experience similarly high levels of burden, which are more closely associated with symptom severity and functional impairment than with diagnostic category (12). These findings suggest that caregiver support needs may be shaped more by shared clinical challenges than by specific diagnostic distinctions.

In parallel with these observations, there has been growing interest in transdiagnostic psychosocial interventions that target shared mechanisms across diagnostic categories. Recent studies indicate that such approaches are feasible and show encouraging preliminary outcomes across internalizing disorders, bipolar disorder, and the psychosis spectrum (13–17). Rather than focusing on diagnosis-specific symptom clusters, transdiagnostic interventions aim to address common processes such as mood dysregulation, psychosocial functioning, illness insight, and treatment adherence. This perspective may be particularly relevant in youth mental health, where early-stage clinical presentations are often mixed, comorbid, and diagnostically fluid (18–20).

Late adolescence represents a developmental period during which many young people remain highly dependent on their families for emotional, financial, and practical support. In the context of severe mental illness, families often become central to treatment engagement, symptom monitoring, and crisis management. Consequently, family members frequently assume multiple roles as caregivers, supporters, and collaborators in care during the early stages of illness. These developmental and contextual factors highlight the importance of involving families in psychosocial interventions for youth with bipolar disorder and schizophrenia (21,22). Although family interventions for bipolar disorder and schizophrenia have typically been examined within separate diagnostic literatures, systematic reviews consistently demonstrate benefits for symptom outcomes, relapse prevention, psychosocial functioning, and caregiver well-being across both conditions (23–25).

Family interventions aim to improve patient and caregiver outcomes by enhancing illness understanding, reducing family stress and caregiver burden, improving communication patterns, strengthening problem-solving skills, and supporting families' roles in illness management (26,27). Among these approaches, Family-Focused Therapy for Early-Onset Youth and Young Adults (FFT-EOY) is a structured, manualized family intervention that integrates psychoeducation, communication skills training, and problem-solving strategies within a vulnerability-stress framework (28). By targeting shared processes such as depressive symptoms, psychosocial functioning, illness insight, and family communication, FFT represents a promising candidate for transdiagnostic application in early-stage severe mental illness.

Despite increasing evidence for family-based psychosocial interventions for severe mental illness, most studies have been conducted in high-income Western settings, and evidence from low- and middle-income countries remains limited (22). Moreover, existing family-intervention studies have generally been developed and evaluated within diagnosis-specific frameworks, leaving uncertainty about whether the same structured family-based intervention can be applied across early-stage bipolar and psychotic disorders. In Türkiye, Ozerdem et al. applied Family-Focused Therapy (FFT) in a series of cases with established bipolar disorder and demonstrated that the intervention could be implemented with minimal modification in a cultural context characterized by strong family interconnectedness and high levels of family involvement in care, supporting the cultural feasibility of FFT in a non-Western setting (29). However, to date, FFT has not been examined as a transdiagnostic intervention for youth in the early stages of bipolar disorder and schizophrenia in the Turkish setting.

The present single-arm pilot study aimed to examine preliminary pre-post changes following par-

ticipation in a brief transdiagnostic FFT program among clinically stable youth (aged 15-19 years) with recent-onset bipolar disorder or schizophrenia and their caregivers. The study focused on youth depressive symptoms, psychosocial functioning, and illness insight, as well as caregiver depressive symptoms, caregiver burden, and family functioning assessed through both youth and caregiver reports. Based on the shared clinical and family-level targets of FFT, we anticipated favorable pre-post changes across these domains and explored whether patterns of change differed descriptively by diagnostic group. Given the exploratory pilot design, the study was intended to provide preliminary evidence regarding the feasibility and potential clinical relevance of applying a brief transdiagnostic FFT program in routine outpatient youth mental health care, and to inform future controlled and longitudinal trials.

METHOD

Study design and ethical considerations: This study was designed as a prospective, single-arm, pre-post exploratory pilot investigation examining changes following participation in Family-Focused Therapy (FFT). The study is reported in accordance with the Transparent Reporting of Evaluations with Nonrandomized Designs (TREND) guidelines for nonrandomized intervention studies (30). The study was conducted between April 2022 and September 2023 at Dokuz Eylül University. The study protocol was approved by Dokuz Eylul University Non-Interventional Research Ethics Committee, approval no. 2022/11-07, ClinicalTrials.gov registration no. NCT05809193. Registration occurred after enrollment began due to administrative procedures; no changes were made to the protocol or prespecified outcomes after registration.

Recruitment and eligibility: Eligibility criteria permitted inclusion of youth aged 15-21 years; however, all participants who enrolled during the recruitment period were between 15 and 19 years of age. Participants were diagnosed with bipolar disorder type I-II or schizophrenia according to DSM-5 criteria using age-appropriate structured diagnostic interviews: the Schedule for Affective Disorders and Schizophrenia for School-Age Children Present and Lifetime Version, DSM-5 Turkish Adaptation (K-SADS-PL-DSM-5-T) for participants younger than 18 years, and the Structured Clinical Interview for DSM-5 Disorders Clinician Version, Turkish Adaptation (SCID-5/CV) for participants aged 18 years or older (31,32). Recent-onset illness was defined as ≤24 months since symptom onset at enrollment. Inclusion criteria required:

1) Clinical stability, defined as the absence of an acute mood or psychotic episode and the ability to participate in outpatient psychotherapy (HDRS ≤ 21, YMRS ≤ 14);

2) Absence of neurodevelopmental disorder, intellectual disability, alcohol/substance abuse or dependence; and

3) Availability of at least one participating parent/caregiver who was literate and had no cognitive impairment or current severe psychiatric disorder that would interfere with participation.

Following the informational briefing, 12 adolescents with bipolar disorder (BD) and 11 adolescents with schizophrenia (SCZ), together with their parents, volunteered to participate. Of these, 10 adolescents in each diagnostic group and their parents completed the FFT program and were included in the final analyses. Participants who discontinued participation did so due to logistical reasons (e.g., scheduling/transportation) rather than clinical deterioration. Retention rate was calculated as the proportion of enrolled families who completed all post-treatment assessments and was used as a pragmatic indicator of feasibility.

Assessment: Informed consent was obtained from both patients and parents. Clinical follow-up and pharmacological treatment continued as usual and were not modified by the study protocol. Baseline pharmacological treatments were recorded during the baseline interview and summarized descriptively. Medication changes during the intervention period were not systematically monitored as a study variable and therefore were not included in the statistical analyses. Baseline and post-treatment clini-

cal assessments were conducted by two independent child and adolescent psychiatry residents who were not involved in the treatment process or the therapy sessions N.G.K./Z.E.A.

The adolescents' current symptoms and functioning were assessed using the Hamilton Depression Rating Scale (HDRS) (33), Young Mania Rating Scale (YMRS) (34), Scale for the Assessment of Positive Symptoms (SAPS) (35), Brief Negative Symptom Scale (BNSS) (36), Personal and Social Performance Scale (PSP) (37), and Scale of Unawareness of Mental Disorder (SUMD) (38). Adolescents completed the McMaster Family Assessment Device (39). Simultaneously, parents completed the Beck Depression Inventory (BDI) (40), Zarit Burden Interview (ZBI) (41) and McMaster Family Assessment Device. The BDI was used to assess caregiver depressive symptom severity and was not intended to establish a clinical diagnosis of parental depression. Assessments were conducted before and after the therapy sessions. If both parents attended therapy, they were asked to decide which parent would complete the questionnaires, and the same parent completed both baseline and post-treatment assessments.

Outcome Measures: Primary outcomes were depressive symptom severity assessed with the Hamilton Depression Rating Scale (HDRS), psychosocial functioning assessed with the Personal and Social Performance Scale (PSP), and illness insight assessed with the Scale of Unawareness of Mental Disorder (SUMD; lower scores indicate greater insight). Secondary outcomes included caregiver depressive symptoms and caregiver burden assessed with the Beck Depression Inventory (BDI) and the Zarit Burden Interview (ZBI), as well as family functioning assessed with the McMaster Family Assessment Device (FAD; adolescent and parent reports). Because the FAD includes multiple subdomains of family functioning, subscale analyses were considered exploratory. Additional clinician-rated symptom measures (manic, positive, and negative symptoms) and detailed family functioning results are reported in the Supplementary Material.

Therapy Sessions: FFT sessions were conducted following the manual "Family-Focused Therapy (FFT) for Youth with Mood and Psychotic Disorders: Clinicians' Treatment Manual (CHAMP Version, 2020) (28). In our study, for practical purposes, FFT was revised to 9 sessions, including 3 sessions each on psychoeducation, communication, and problem-solving skills. The revision received written approval from Prof. Dr. David Miklowitz.

The therapy was delivered as a combined program for youth diagnosed with bipolar disorder or schizophrenia, using the same manualized FFT protocol across diagnostic groups. Importantly, the psychoeducation module was delivered using a shared transdiagnostic framework emphasizing common mechanisms across mood and psychotic disorders (e.g., stress reactivity, sleep disruption, depressive symptoms, and relapse prevention). The intervention was delivered with the participating youth and at least one parent/caregiver. When available, both parents were allowed to attend sessions; however, siblings were not included as regular participants in the therapy program.

Sessions 1-3 consisted of a shared psychoeducation module delivered in a small multi-family format, with two families (i.e., two youth and their participating caregivers) attending each session. Families were grouped irrespective of diagnosis, such that each multi-family session could include any combination of BD and SCZ families. This module focused on goal setting, treatment overview, symptom recognition (including manic, depressive, positive, negative, and anxiety symptoms), the vulnerability-stress model, stress and coping strategies, sleep regulation, relaxation exercises, optimization of family support, relapse prevention planning, and medication adherence. This initial multi-family format was selected to enhance feasibility in routine outpatient care and to facilitate normalization and shared learning across families facing early-stage severe mental illness.

From session 4 onward, therapy was delivered in an individual family format. Sessions 4-6 focused on communication skills training, including expressing positive feelings, active listening, making positive requests for change, improving communication

Figure 1. Structure of the Family-Focused Therapy Sessions

clarity, and expressing negative emotions in a constructive manner. Sessions 7-9 were dedicated to problem-solving skills training, including introduction to the problem-solving framework, guided practice, consolidation of skills, and termination of treatment. A schematic overview of the session content is provided in Figure 1.

During the therapy process, an interactive approach was adopted to actively engage adolescents and their parents. To minimize variability in practice, the primary therapist remained the same throughout the entire process, with additional support provided by co-therapists when necessary. The therapy sessions occurred on a weekly basis with the primary therapist (E.S.) and co-therapists (B.A., G.K. or S.A.). The duration of each session was approximately 60-90 minutes. Sessions were supported with visual presentations, and participants were provided with session notes, reminder materials, and homework assignments as specified in the therapy manual. If the interval between sessions exceeded two weeks, the subsequent session began with a brief review to reinforce previously covered material and re-establish treatment continuity. Although the therapists did not have formal certification in family therapy, the primary therapist had clinical experience in child and adolescent psychiatry and family-based psychosocial care, and the therapy team reviewed the FFT manual and session materials before implementation. Formal supervision or independent fidelity assessments were not conducted; however, the intervention was delivered in accordance with the manualized FFT protocol to support treatment consistency.

Statistical analyses

Statistical analyses were conducted using SPSS (version 29.0). Baseline differences between diagnostic groups were examined using independent samples t-tests for continuous variables and Fisher's exact test for categorical variables. Within-group pre-post changes were examined using paired samples t-tests, and group × time patterns were tested using repeated-measures ANOVA. Given the small sample size and exploratory pilot design, statistical significance testing was considered descriptive rather than confirmatory, and interpretation emphasized the direction and magnitude of effects rather than p-values alone.

Given baseline differences in psychosocial functioning, post-intervention PSP scores were additionally analyzed using ANCOVA, with diagnostic group as the fixed factor and baseline PSP score entered as a covariate. Homogeneity of regression slopes was assessed by testing the baseline PSP × group interaction. Other baseline variables, including psychiatric comorbidity and pharmacological treatment, were summarized descriptively but were not entered as covariates because of the small sample size and the risk of model overfitting.

Within-group effect sizes were computed as Hedges' g for standardized mean change (gΔ), calculated as the mean within-participant change divided by the standard deviation of individual

change scores, with the small-sample correction applied (42). Effect sizes were coded such that positive values indicated improvement; therefore, for outcomes in which lower scores reflected better clinical status, change-score signs were reversed before calculating Hedges' g/\. Ninety-five percent confidence intervals for Hedges' g/\ were derived using bootstrap resampling with 1,000 resamples. No formal correction for multiple comparisons was applied; analyses involving multiple FAD subscales were considered exploratory and hypothesis-generating. All tests were two-tailed. Analyses were conducted as complete-case analyses including participants who completed post-treatment assessments, and no imputation was applied.

RESULTS

Sociodemographic Variables: Of 23 eligible families who enrolled in the study (BD=12, SCZ=11), 20 (87%) completed all post-treatment assessments (BD=10, SCZ=10). Discontinuations were due to scheduling or transportation difficulties rather than clinical worsening. No statistically significant baseline differences were observed between the BD and SCZ groups with respect to age, sex, years of education or parental education level. Mean illness duration was comparable between groups (BD: 13.5 months; SCZ: 12.6 months). Psychiatric comorbidity was common in both groups and was numerically more frequent in the SCZ group than in the BD group (70% vs. 30%), although this difference was not statistically significant. Patterns of baseline pharmacological treatment differed between groups. Mood stabilizer use was more frequent in the BD group than in the SCZ group, whereas antidepressant use was observed only in the SCZ group. These medication variables were summarized descriptively and were not included as covariates in inferential models because of the small sample size. Baseline characteristics are presented in Table 1.

Table 1: Sociodemographic characteristics and illness history of youth with BD and SCZ

	BD *(n=10)* Mean (SD)	SCZ *(n=10)* Mean (SD)	p
Age	16.50 (0.85)	16.45 (1.50)	0.93
Sex (F/M) (n)	6/4	5/5	1.0
Years of education	11.2 (0.79)	10.3 (1.25)	0.07
Mother education level	12.1 (3.6)	10.5 (4.4)	0.38
Father education level	9.4 (5.1)	11.7 (4.9)	0.32
Duration of illness (months)	13.5 (6.88)	12.6 (6.92)	0.77
Baseline medications (n, %)			
Antipsychotic	9 (90.0)	10 (100.0)	1.0
Mood stabilizers	6 (60.0)	0 (0.0)	0.011
Antidepressant	0 (0.0)	4 (40.0)	0.087
Anxiolytic	1 (10.0)	2 (20.0)	1.0
Psychiatric comorbidity (n, %)			
Total	3 (30.0)	7 (70.0)	0.179
Generalized anxiety disorder	1 (10.0)	3 (30.0)	
Social anxiety disorder	1 (10.0)	2 (20.0)	
Obsessive-compulsive disorder	1 (10.0)	2 (20.0)	

Note. Categorical variables were compared using Fisher s exact test.

Primary Outcomes: Depressive Symptoms (HDRS): Both diagnostic groups demonstrated significant reductions in depressive symptom severity from pre- to post-intervention (BD: $p = 0.003$; SCZ: $p = 0.001$) (Table 2). No significant group × time interaction was detected ($F(1,18) = 0.06$, $p = .81$, partial $\eta^2 = .003$), providing no evidence of differential pre-post change between diagnostic groups.

Psychosocial Functioning (PSP): At baseline, PSP scores were significantly higher in the BD group than in the SCZ group ($p = 0.002$). Improvements in psychosocial functioning were observed in both groups from pre- to post-intervention (both $p < 0.001$; Table 2), with large standardized mean change effect sizes. The mean absolute increase in PSP was 16.0 points in the BD group and 7.8 points in the SCZ group. A significant group × time interaction was detected ($F(1,18) = 14.22$, $p = 0.001$, partial $\eta^2 = .44$), suggesting an exploratory group × time pattern in PSP change.

Given the substantial baseline difference in functioning, post-treatment PSP scores were additionally examined using ANCOVA adjusting for baseline PSP. In this baseline-adjusted exploratory model, diagnostic group was associated with post-treatment PSP scores, $F(1,17) = 25.44$, $p < 0.001$. Adjusted mean PSP scores were higher in the BD group ($M = 66.31$, $SE = 1.56$) than in the SCZ group ($M = 53.79$, $SE = 1.56$), with an estimated adjusted mean difference of 12.51 points (95% CI [7.28, 17.74]).

Illness Insight (SUMD): Significant within-group pre-post improvements in illness insight were observed in both diagnostic groups (BD: $p = 0.014$; SCZ: $p = 0.004$) (Table 2). No significant group × time interaction was detected, providing no evidence of differential pre–post change between diagnostic groups ($F(1,18) = 1.067$, $p = .315$, partial $\eta^2 = .056$).

Within-group standardized mean change effect

Table 2: Primary outcomes (HDRS, PSP, SUMD)

Outcome	Group	Baseline Mean (SD)	Post-therapy Mean (SD)	p (within group)	Hedges g (95% CI)
HDRS	BD (n=10)	12.7 (8.49)	2.7 (4.08)	0.003	1.14 [0.56, 1.68]
	SCZ (n=10)	13.4 (6.52)	4.2 (3.05)	0.001	1.34 [0.79, 1.93]
PSP	BD (n=10)	56.0 (9.19)	72.0 (4.83)	<0.001	2.58 [1.98, 3.15]
	SCZ (n=10)	40.3 (10.01)	48.1 (10.35)	<0.001	1.84 [1.27, 2.39]
SUMD	BD (n=10)	6.2 (3.29)	3.4 (0.70)	0.014	0.89 [0.36, 1.42]
	SCZ (n=10)	8.6 (4.14)	4.3 (1.38)	0.004	1.10 [0.51, 1.66]

Note. Baseline PSP scores differed significantly between groups, with higher functioning in the BD group. Effect sizes are reported as Hedges g for standardized mean change (g $\underline{\Delta}$), calculated using the standard deviation of individual change scores. Ninety-five percent confidence intervals were derived using bootstrap resampling of the mean change. The direction of change is indicated by the pre- and post-treatment means.

sizes were large across primary outcomes, and the 95% confidence intervals indicated improvement (Table 2).

Secondary Outcomes: Caregiver Depression and Burden: Among parents of adolescents with BD, depressive symptoms assessed with the Beck Depression Inventory showed a significant pre-post decrease ($p = 0.009$), whereas no significant change was observed among parents of adolescents with SCZ ($p = 0.22$). Baseline BDI scores among parents of adolescents with SCZ showed substantial variability, indicating heterogeneity in depressive symptom severity within this small subgroup. Caregiver burden, assessed with the Zarit Burden Interview, decreased significantly in both diagnostic groups (BD parents: $p = 0.004$; SCZ parents: $p = 0.022$) (Table 3). No significant group × time interaction was observed for caregiver depressive symptoms or caregiver burden (both $p > .05$). Overall, caregiver burden showed pre-post reductions in both diagnostic groups, whereas caregiver depressive symptoms showed a statistically significant pre-post reduction only among parents of adolescents with BD.

Family Functioning (FAD): Family functioning results are presented descriptively due to multiple subdomain analyses. At baseline, no significant differences were observed between the BD and SCZ groups in family functioning domains. However, patterns of change differed notably between adolescent and parent reports across diagnostic groups.

Among adolescents with BD, significant within-group pre-post changes indicating improvement were observed in the problem-solving ($p = 0.012$) and general functioning ($p = 0.016$) subdomains of the Family Assessment Device. In contrast, adolescents with SCZ showed significant within-group changes across multiple family functioning domains, including problem-solving ($p = 0.032$), communication ($p = 0.011$), roles ($p = 0.025$), affective responsiveness ($p = 0.021$), behavior control ($p = 0.009$), and general functioning ($p = 0.003$). Between-group comparisons revealed a significant group × time interaction only for the behavior control subdomain ($F(1,18) = 5.26$, $p = 0.03$, partial $\eta^2 = 0.226$). Adolescent-reported outcomes are summarized in Supplementary Table 2s.

Parent-reported family functioning showed a different pattern. Among parents of adolescents with BD, significant within-group improvements were found in problem-solving ($p = 0.013$), communication ($p = 0.023$), and general functioning ($p = 0.015$). In contrast, parents of adolescents with SCZ demonstrated a significant improvement only in the behavior control subdomain from pre- to post-intervention ($p = 0.002$). Parent-reported outcomes are presented in Supplementary Table 3s.

Across informants, problem-solving and general functioning showed significant within-group changes in the BD group, whereas behavior control was the only subdomain showing significant change in both adolescent and parent reports in the SCZ group.

DISCUSSION

The present single-arm pilot study examined pre-post changes following a brief, transdiagnostic Family-Focused Therapy (FFT) program in youth with recent-onset bipolar disorder (BD) or schizophrenia (SCZ) and their caregivers in a Turkish outpatient setting. Overall, the findings

Table 3. Caregiver outcomes (BDI, ZBI)

Outcome	Group	Baseline Mean (SD)	Post-therapy Mean (SD)	p (within group)	Hedges g (95% CI)
BDI	BD-P (n=10)	14.7 (9.07)	8.6 (4.20)	0.009	0.95 [0.41, 1.50]
	SCZ-P (n=10)	16.6 (14.03)	13.7 (11.75)	0.22	0.38 [-0.13, 1.00]
ZBI	BD-P (n=10)	29.3 (13.35)	23.8 (14.44)	0.004	1.10 [0.62, 1.67]
	SCZ-P (n=10)	36.1 (18.03)	27.5 (16.71)	0.022	0.80 [0.27, 1.37]

Note. Effect sizes represent standardized mean change (Hedges g $\underline{\Delta}$) calculated using the standard deviation of individual change scores; 95% confidence intervals were derived using bootstrap resampling of the mean change. Effect sizes are reported such that positive values indicate improvement.

suggest preliminary improvements across several youth and caregiver outcomes and support the practicality of delivering a 9-session FFT program within routine care, although controlled trials are needed to determine efficacy and durability of effects.

Depressive Symptoms, Psychosocial Functioning, and Illness Insight

Pre-post reductions in depressive symptoms were observed in both the BD and SCZ groups following participation in the FFT program. These findings are consistent with prior research demonstrating associations between family-based interventions and improvements in depressive symptoms in bipolar disorder (43,44). In bipolar disorder, depressive symptoms, particularly subsyndromal depression, are a major determinant of functional impairment and suicide risk. Persistent depressive symptoms have been identified as one of the strongest predictors of poor functional outcomes in BD, even during periods of symptomatic stability (45).

Beyond bipolar disorder, depression is also highly prevalent in schizophrenia and may occur throughout the course of the illness, including during the early stages and following a first psychotic episode (46,47). However, evidence regarding the impact of psychosocial interventions on depressive symptoms in schizophrenia remains limited (48). In clinical practice, treatment approaches for schizophrenia often prioritize positive symptoms, while depressive symptoms may be underrecognized or misattributed to negative symptomatology. This diagnostic overlap may complicate both assessment and intervention (48). Early psychosocial interventions that incorporate family involvement may therefore play an important role in addressing depressive symptomatology alongside core illness features. In this context, structured psychosocial interventions that address depressive symptoms within the family environment may be particularly relevant for supporting functional recovery across the mood-psychosis spectrum.

In parallel, improvements were observed in both psychosocial functioning and illness insight across diagnostic groups. Illness insight is recognized as a key factor influencing treatment adherence and long-term outcomes; however, its development is shaped by complex cognitive, emotional, and social processes (49). The specific mechanisms underlying these changes remain unclear and warrant further investigation in controlled longitudinal studies.

The larger functional gains observed in the BD group should be interpreted in the context of their higher baseline functioning, and baseline differences may have partially contributed to the observed group × time interaction. Although post-treatment PSP scores were additionally examined using ANCOVA adjusting for baseline PSP, the small sample size limits conclusions regarding differential functional change between diagnostic groups. In addition, the higher psychiatric comorbidity rate in the SCZ group and baseline differences in pharmacological treatment may have contributed to clinical heterogeneity and influenced pre-post change patterns. Future trials may benefit from larger samples that allow stratification or adjustment for baseline functioning, comorbidity, and medication-related variables using analytic approaches such as mixed-effects models.

Caregiver Depression and Burden

Baseline levels of caregiver depressive symptoms and burden were numerically higher in the SCZ-parent group, although between-group differences were not statistically significant. Pre-post reductions in caregiver burden were observed in both groups; however, caregiver depressive symptoms showed a statistically significant decrease only among parents of adolescents with BD. This pattern may suggest that caregiver burden and caregiver depressive symptoms do not change uniformly following a brief family-focused intervention. In particular, the absence of a statistically significant change in BDI scores among SCZ parents should be interpreted cautiously, given the substantial variability in baseline BDI scores and the use of a self-report symptom measure rather than a structured diagnostic interview.

Previous research indicates that improvements in patient symptoms do not necessarily translate into

improved caregiver well-being, particularly in psychotic disorders characterized by persistent functional impairments (50). Parental mental health may represent an important and clinically relevant moderator of psychosocial intervention outcomes and therefore warrants greater attention in early intervention frameworks.

These findings highlight the potential importance of routinely considering caregiver depressive symptoms within early family-based intervention pathways. In families affected by schizophrenia, future studies could examine whether augmenting FFT with brief caregiver-focused modules (e.g., mood monitoring, stress management, or referral pathways) improves the sustainability of family-level gains in early intervention settings. Overall, these findings provide preliminary support for the clinical relevance of family-based interventions during the early stages of severe mental illness.

Family Functioning

Family functioning showed descriptively different patterns of pre-post change across diagnostic groups and informants, highlighting distinct patterns of perceived change within families. Adolescents with SCZ reported pre-post improvements across multiple family functioning domains, whereas adolescents with BD reported more limited changes. Parent reports revealed a different pattern, with broader improvements observed among parents of adolescents with BD. These findings are consistent with previous studies suggesting that adolescents and caregivers may perceive changes in family dynamics differently and that families affected by psychotic disorders may require longer follow-up periods to recognize meaningful changes (51,52). This informant-specific pattern highlights the value of multi-informant assessment in family-based interventions and suggests that adolescents and caregivers may attend to different aspects of family functioning during early intervention.

Several limitations should be considered when interpreting the findings of this study. The sample size was small, reflecting the pilot and exploratory nature of the investigation, which limited statistical power and generalizability and increased uncertainty around effect estimates. Participation was voluntary, and some eligible families declined participation, potentially introducing selection bias. The absence of a control or waitlist comparison group also precludes causal inference regarding observed changes. Therefore, pre-post improvements cannot be attributed specifically to FFT, and alternative explanations such as natural clinical recovery, regression to the mean, nonspecific therapeutic factors, or concurrent clinical care cannot be excluded. The inclusion of clinically stable youth with recent-onset illness may also have influenced the observed pre-post changes, as newly diagnosed youth and their families may differ from more chronic samples in psychosocial support needs, family involvement, treatment engagement, and natural recovery trajectories.

Baseline differences between diagnostic groups may also have influenced the observed patterns of change. The BD and SCZ groups differed in psychosocial functioning at baseline, psychiatric comorbidity was numerically more frequent in the SCZ group, and baseline pharmacological treatments differed between groups. Although baseline PSP was adjusted for in an ANCOVA analysis, the small sample size did not permit broader multivariable adjustment for comorbidity or medication-related variables. In addition, medication changes during the intervention period were not systematically monitored, limiting the ability to disentangle pharmacological and psychosocial influences.

Caregiver depressive symptoms were assessed using a self-report measure rather than structured diagnostic interviews; therefore, BDI scores should be interpreted as indicators of depressive symptom severity rather than diagnoses of parental depression. The substantial variability in baseline BDI scores among parents of adolescents with SCZ further limits conclusions regarding caregiver depressive symptom change in this subgroup. Finally, therapists delivering the intervention were not blinded, formal fidelity ratings or independent supervision procedures were not conducted, and the absence of follow-up assessments limits conclusions regarding the durability of observed changes.

This pilot study provides preliminary findings regarding the potential relevance of Family-Focused Therapy for youth with recent-onset bipolar disorder and schizophrenia using a transdiagnostic approach. To our knowledge, this is among the first pilot studies to apply the same structured family-focused intervention to early-stage bipolar disorder and schizophrenia in youth while simultaneously examining patient, family, and caregiver outcomes using a multi-informant design.

The findings highlight the potential value of early family-based psychosocial interventions for addressing both core clinical symptoms and family-level processes during the early stages of severe mental illness. Although diagnosis-specific conclusions cannot be drawn from this pilot study, the observed patterns are consistent with the relevance of targeting shared mechanisms across the mood-psychosis spectrum, including depressive symptomatology, psychosocial functioning, illness insight, family communication, and caregiver burden. These findings provide preliminary support for the potential role of transdiagnostic, family-based interventions as scalable components of early intervention strategies in severe mental illness.

Future research should prioritize larger, controlled, and longitudinal studies with systematic medication monitoring to evaluate the durability of observed changes, clarify causal effects, and identify moderators and mediators of treatment response. Such studies should also examine which youth and caregiver characteristics predict response to transdiagnostic family-based treatment.

In particular, the differential patterns observed in caregiver burden and caregiver depressive symptoms raise the possibility that caregiver mental health may represent a distinct and clinically relevant treatment target. Incorporating targeted support for caregivers, especially those experiencing depressive symptoms, may further strengthen the clinical utility and sustainability of early family-based interventions for both bipolar disorder and schizophrenia.

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethics approval: The study protocol was approved by the Dokuz Eylul University Non-Interventional Research Ethics Committee; approval no. 2022/11-07.

Trial registration: ClinicalTrials.gov identifier: ClinicalTrials.gov identifier: NCT05809193.

Informed Consent: All participants and their parents/legal guardians were informed about the study, and written informed consent was obtained.

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Data Availability: The data can be made available upon reasonable request by contacting the corresponding author.

AI Use Statement: AI-based tools were used only for language editing. No AI tools were used for data analysis, interpretation, or generation of scientific content.

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Visualizing mental health: A study on schizophrenia awareness and graphic design evolution

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SUMMARY

Objective: Social stigma toward schizophrenia is often perpetuated by inaccurate media portrayals. This study investigates how evidence-based psychoeducation influences the attitudes of graphic design students—future visual communicators—and how this knowledge shifts their creative output.

Method: Eighty graphic design students in Turkey participated in a 60-minute structured psychoeducation session led by psychiatrists. Participants created awareness posters before and after the intervention. Changes were measured using the Social Distance Scale (SDS), Survey on Knowledge about Schizophrenia, and Belief toward Mental Illness Scale (BMI). Qualitative shifts in visual metaphors and storytelling were also analyzed.

Results: Post-intervention, both SDS and BMI scores decreased significantly ($p < 0.05$), indicating reduced stigma. While 85% of participants consistently supported professional medical help, the perception of schizophrenia as a purely social issue dropped from 23% to 20%. Critically, while technical design elements (color, typography) remained statistically constant, there was a significant evolution in narrative language, characterized by nuanced metaphors and sophisticated conceptual framing.

Discussion: Psychoeducation effectively improves theoretical knowledge and corrects misconceptions, though altering deeply rooted behavioral social distance remains a challenge. The intervention primarily influenced "conceptual depth" and "empathy-driven storytelling" rather than aesthetic choices, suggesting that education refines how designers think about a subject. Integrating psychoeducation into design curricula fosters less-stigmatizing representations of mental illness. This interdisciplinary approach is vital for refining the visual metaphors used by communicators to reshape public perception.

Key Words: Schizophrenia; Social Stigma; Graphic Design; Psychoeducation

INTRODUCTION

Historically, prejudice against individuals with mental health conditions has been more severe than that directed at other social groups, leading to profound humiliation and social rejection (1,2). This stigmatization effectively bifurcates society into a "normal" majority and a "stigmatized" minority, with the latter often perceived through a lens of fear as either dangerous or inherently weak (3). The structural mechanism of this phenomenon is built upon three primary pillars: cognitive, affective, and behavioral (4). Within this framework, negative stereotypes fuel affective prejudice, manifesting as visceral emotional reactions such as anxiety or resentment toward those with psychiatric disorders (5). Ultimately, these internal biases culminate in behavioral discrimination, resulting in systemic barriers within the labor market and healthcare systems (6)

Linguistic labeling and the perception of personal responsibility significantly exacerbate the stigmatization of mental health, often reducing individuals to their diagnoses through "othering" language (7). Unlike physical illnesses, societal assumptions often incorrectly place personal fault on the individual, further justifying discriminatory attitudes (3). These perceptions frequently lead to unfair characterizations of patients as dangerous or

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untrustworthy, resulting in a diminished social identity and systematic community exclusion (8). Comparative research consistently identifies schizophrenia as one of the most heavily stigmatized disorders due to inaccurate public associations with violence (9). Ultimately, this profound social isolation is clinically detrimental, as it is directly linked to increased depressive symptoms, cognitive impairment, and a substantial decline in overall quality of life (10)

At the core of these negative societal attitudes is a profound lack of mental health literacy and persistent misconceptions regarding psychiatric treatments. When society lacks accurate information, unfounded stereotypes—such as the inherent "danger" of individuals with schizophrenia—become deeply entrenched in the collective psyche (11). However, evidence suggests that prejudice is not immutable; individuals who participate in structured and accurate educational programs regarding the nature of schizophrenia often begin to re-evaluate their biases and adopt more inclusive perspectives (12).

Addressing stigma among younger populations is particularly critical, given the severe long-term effects of untreated mental illness. Current strategies for stigma reduction typically utilize two main pillars: education and social contact. While direct social contact has proven more effective for university-aged students, educational interventions often yield superior results among school-age children (1). Recent meta-analytic evidence indicates that psychoeducation-based programs significantly reduce internalized stigma in the short term, although the long-term sustainability of these effects remains a challenge (13). Current perspectives suggest that traditional neurobiological models may inadvertently reinforce stigma through connotations of biological otherness, whereas computational frameworks like predictive processing offer a more normalizing approach by bridging the gap between biology and lived experience (14). This study also emphasizes the critical importance of psychoeducation in reducing stigma and improving mental health literacy.

This study aims to evaluate the impact of a structured psychoeducation intervention on the conceptual and visual narrative strategies of graphic design students regarding stigma reduction for schizophrenia. Adopting a pre-test/post-test quasi-experimental design within the framework of action research and qualitative document analysis, the research seeks to investigate how clinical knowledge influences the transformation of psychiatric concepts into visual metaphors. As an action research model, this study provides a systematic, practice-based approach to addressing educational challenges and enhancing the pedagogical process in design studios. Furthermore, by analyzing the structural and qualitative evolution of poster designs, the study aims to foster social responsibility awareness and ethical representation skills, thereby strengthening the intersection between mental health advocacy and graphic design education. Given the critical role of media in shaping public perception, this study also emphasizes the importance of accurate visual representation in mitigating the stigmatizing effects often perpetuated by mainstream media regarding schizophrenia.

METHOD

Study Design and Intervention: The research was structured as a pre-test/post-test quasi-experimental study to evaluate the impact of a 60-minute structured psychoeducation session on graphic design students students who voluntarily participated in the study, and the intervention was developed by two psychiatrists. The intervention consisted of a 60-minute structured psychoeducation session. It covered the etiology, symptoms, diagnosis, treatment, and rehabilitation of schizophrenia. In the first phase (pre-intervention), participants comp-leted quantitative scales and developed initial poster designs based on their baseline knowledge of schizophrenia. Following the educational intervention, the same quantitative scales were re-administered, and a second set of posters was produced by the students to assess the evolution in their conceptual framing. This dual-layered approach allowed for the comparative analysis of both numerical data from the scales and the qualitative shift in visual narratives—specifically focusing on the use of original metaphors, visual storytelling, and the representation of schizophrenia subtypes and treatment processes

Assessment Tools: The participants were evaluated using the Social Distance Scale (SDS), which was originally developed in Turkish by Arkar et al. (15) to measure behavioral rejection, and the Belief toward Mental Illness Scale (BMI), originally developed by Hirai and Clum (16) to assess cognitive stereotypes, for which the Turkish validity and reliability studies have been established (17).

Additionally, a Survey on Knowledge about Schizophrenia was developed to measure knowledge levels regarding the disorder. This survey utilized a vignette followed by 5-point Likert-type questions, including such as: "Which of the following best describes Mr. Ahmet's condition?" "Mr. Ahmet's condition stems from social problems he is experiencing (unemployment, poverty, family problems, etc.)." "Mr. Ahmet's condition stems from a weak personality structure." "Mr. Ahmet's condition is a state of extreme sadness." "Mr. Ahmet's condition is based on genetic factors." "Mr. Ahmet's condition has a biological basis; it stems from changes in the brain's biochemistry." "Which of the following should someone with similar complaints do first to overcome this condition?"

Structural and Qualitative Evaluation of Graphic Design Products: This study employs a multi-layered analytical model to evaluate graphic design products, distinguishing between technical proficiency and conceptual narrative depth. In the initial phase, designs are analyzed based on formal elements—such as color palette, typography, and visual hierarchy—in accordance with statistical and visual grammar rules. The second phase involves a qualitative examination of narrative language, focusing on original metaphors and visual storytelling strategies to define the conceptual framing of the subject matter such as pathology-focused representation (e.g., chaos, fragmentation, danger imagery), recovery-oriented representation (e.g., healing, integration, support), stigmatizing metaphors (e.g., isolation, fear-based imagery), empathy-driven narratives (e.g., humanization, social inclusion).The final stage subjects the communicative impact to a systematic evaluation regarding ethical boundaries and potential stigmatization risks. To enhance reliability, two independent evaluators with expertise in psychiatry and visual communication reviewed the posters. Discrepancies were resolved through consensus.

Ethical Approval and Study Setting; Ethical approval for this study was obtained from the Kocaeli University Faculty of Medicine Ethics Committee. The research was conducted between November 2024 and December 2024 through collaboration between the Kocaeli University Faculty of Medicine and the Faculty of Fine Arts. The study sample consisted of volunteers enrolled in the Department of Graphic Design at Kocaeli University Faculty of Fine Arts, who responded positively to a call for poster design.

Statistical Analysis

Data were analyzed using SPSS version 23.0. Descriptive statistics were expressed as frequency, percentage, mean, and standard deviation. As the skewness and kurtosis values of the numerical variables ranged between ±1.5, the data were assumed to follow a normal distribution. The Social Distance Scale (SDS) was defined as the primary outcome, while the Belief toward Mental Illness Scale (BMI) was considered a secondary outcome. The Paired Samples t-test was employed to compare the pre- and post-test scores of the continuous variables within the same group to evaluate the impact of the intervention. Additionally, an Independent Samples t-test was used to compare continuous variables between independent groups, where applicable. Statistical significance was set at $p < 0.05$. Data were analyzed using paired-samples t-tests to compare pre-intervention and post-intervention scores. To control for the risk of Type I error inflation due to multiple testing, p-values were adjusted using the Holm-Bonferroni method. Additionally, Cohen's d was calculated for each comparison to assess the effect size and practical significance of the findings. To evaluate the changes in categorical responses (Agree, Undecided, Disagree) between pre-test and post-test assessments, the Stuart-Maxwell test was employed to assess marginal homogeneity.

RESULTS

The demographic and psychiatric characteristics of the participants are shown in Table 1. Among the

Table 1 Sociodemographics and psychiatric characteristics

Variables	n	%
Gender		
Female	67	83.7
Male	13	16.3
Academic Year		
1st Year	16	20.0
2nd Year	20	25.0
3rd Year	16	20.0
4th Year	28	35.0
Living Arrangement		
Roommate	8	10.0
Family	21	26.2
State Dormitory (KYK)	29	36.3
Private Dormitory	11	13.7
Living Alone	6	7.5
Other	5	6.3
Previous Mental Health Project Experience		
No	53	67.1
Yes	26	32.9
History of Psychiatric Complaints		
No	46	57.5
Yes	34	42.5
Received Psychiatric Help		
No	50	62.5
Yes	30	37.5
Perceived Benefit (n=30)		
No benefit at all	5	16.7
Mild benefit	7	23.3
Moderate benefit	9	30.0
Significant benefit	7	23.3
High benefit	2	6.7
Professional Consulted for Complaints		
Psychologist	21	26.3
Psychiatrist	17	21.3
Counseling (PDR)	6	7.5
Family Physician	2	2.5
Current Psychiatric Diagnosis		
None	55	68.7
Present	25	31.3
Type of Diagnosis (n=25)		
Anxiety Disorder	14	38.89
Depression	10	27.78
Eating Disorder	3	8.33
Obsessive-Compulsive Disorder (OCD)	3	8.33
Personality Disorder	2	5.56
Developmental Disorders	2	5.56
Bipolar Affective Disorder	2	5.56
Family History of Psychiatric Disorders		
None	63	78.7
Present	17	21.3
Family Diagnosis (n=24)		
Depression	10	41.67
Anxiety Disorder	5	20.83
Bipolar Affective Disorder	3	12.5
Psychotic Disorder	2	8.33
Obsessive-Compulsive Disorder (OCD)	2	8.33
Personality Disorder	1	4.17
Developmental Disorders	1	4.17

participants, 83.7% were female and 16.3% were male. The largest groups of participants were fourth-year (35%) and second-year (25 %) students. Regarding living arrangements, most (36.3%) resided in state dormitories (KYK), while 26.3% lived with their families. Additionally, 32.9% of the participants had previous experience in mental health projects, whereas 67.1% had no such experience.

While 42.5% of the participants reported having experienced psychiatric complaints in the past, 62.5% had not received professional psychiatric help. Among the 30 participants who sought help, 23.3% reported a high level of benefit, 23.3% reported a moderate benefit, and 23.3% reported a mild benefit. For mental health concerns, the most frequently consulted professionals were psychologists (26.3%) and psychiatrists (21.3%). Furthermore, 31.3% of the participants had a psychiatric diagnosis. Among those diagnosed, the most common disorders were anxiety disorders (56%) and depression (40%). Finally, 21.3% of the participants reported a family history of psychiatric illness.

Participants' perspectives on psychiatric disorders are presented in Table 2. Regarding their level of knowledge about psychiatric disorders, 38.8% of the participants reported a moderate level, 28.8% reported a mild level, and 18.8% stated that they were highly knowledgeable. While the internet was identified as the most significant source of information (71.3%), social media ranked as the second most common source (45%). When recommending interventions for someone with a psychiatric disorder, 65% of participants suggested referral to a psychiatrist/psychologist, 41.3% recommended psychotherapy, and 27.5% suggested art-therapy.

The analysis of the pre- and post-test scores revealed a statistically significant decrease in both the Social Distance Scale ($p = 0.013$) and the Belief toward Mental Illness Scale (BMI) ($p= 0.002$). The reduction in the mean scores for the Social Distance Scale indicates that the intervention effectively diminished participants' desire for social distance from individuals with mental health conditions. Similarly, the significant decline in BMI scores suggests a positive shift in participants' beliefs and a reduction in stigmatizing attitudes following the study. (Table 3)

Table 2 Participants' perspectives, knowledge levels, and information sources regarding psychiatric disorders

Variables	n	%
What would you recommend to someone experiencing psychological or psychiatric problems?		
Referral to a Psychiatrist/Psychologist	52	65.0
Psychotherapy	33	41.3
Art therapy	22	27.5
Concentration and relaxation exercises	15	18.8
Practicing meditation, yoga, or religious chanting	14	17.5
Using psychopharmacological medications	7	8.8
Becoming more religious	3	3.8
Engaging in treatments suitable for religious beliefs	3	3.8
Trying natural products	1	1.3
Electroconvulsive therapy (ECT)	1	1.3
What is your level of knowledge of psychiatric disorders?		
No knowledge	8	10.0
Mild knowledge	23	28.8
Moderate knowledge	31	38.8
Substantial knowledge	15	18.8
Very knowledgeable	3	3.8
What is your source of information on psychiatric disorders?		
Internet	57	71.3
Social Media	36	45.0
Social Circle	28	35.0
Books	24	30.0
Conferences	7	8.8
Academic Environment	4	5.0
Other	8	10.0

Table 3. Comparison of Pre-test and Post-test Total Scores for Social Distance Scale and Belief toward Mental Illness Scale (BMI)

Scale	Pre-test (Mean–SD)	Post-test (Mean–SD)	T	p-value	Adj. p (Holm)	Cohen□s d
Social Distance Scale (SDS)	80.57–19.92	72.61–21.73	2.549	0.013	0.026	0.29
Belief toward Mental Illness Scale (BMI)	74.45–15.30	66.12–14.85	3.245	0.002	0.004	0.36

Paired samples t-test

SDS: Social Distance Scale; BMI: Belief toward Mental Illness Scale. Adjusted p-values were calculated using the Holm-Bonferroni method. Cohen□sd interpretation: 0.2=small, 0.5=medium, 0.8=large.

When comparing participants who showed a decrease in social distance scores with those who did not, no statistically significant differences were observed regarding gender, level of psychiatric knowledge, history of mental illness, family history of psychiatric disorders, living arrangement, or prior participation in projects ($p > 0.05$)

The findings in Table 4 indicate that the participants' perspectives on the causes of Mr. Ahmet's condition underwent notable changes from the pre-test to the post-test. The Stuart-Maxwell test revealed no statistically significant changes in the distribution of participants' beliefs regarding etiological factors following the intervention ($p > 0.05$), although a trend was observed for the "weak personality" item ($\chi^2 = 5.12$, $p = 0.077$)

egorizing recurring themes and metaphors, the study found that pre-intervention designs were predominantly characterized by pathology-focused and stigmatizing representations. In contrast, post-intervention posters demonstrated a clear transition toward recovery-oriented and empathy-driven narratives. While the technical execution remained stable, the frequency of human-centered visual storytelling increased, indicating that psychoeducation effectively deepened the conceptual framing of the students' work.

Beyond the visual narratives, the etiological beliefs and social-context framings also showed measurable changes following the intervention. Although some percentage shifts in the descriptive data appeared modest, the overall trend consistently

Table 4. Participants' opinions on the etiology of Mr. Ahmet's condition (pre-test vs. post-test)

Etiological Factors	Assessment	Agree (%)	Undecided (%)	Disagree (%)	Stuart-Maxwell (χ^2)	p-value
Social Problems (unemployment, poverty, etc.)	Pre-test/Post-test	23.0/20.0	31.0/30.0	27.0/30.0	0.84	0.657
Weak Personality Structure	Pre-test/Post-test	16.0/25.0	28.0/22.0	37.0/33.0	5.12	0.077
State of Extreme Sadness	Pre-test/Post-test	20.0/21.0	36.0/27.0	25.0/31.0	2.58	0.275
Genetic Factors	Pre-test/Post-test	26.0/31.0	30.0/25.0	25.0/34.0	2.34	0.310
Biological Basis (brain biochemistry)	Pre-test/Post-test	34.0/34.0	28.0/25.0	19.0/21.0	0.45	0.798

Stuart-Maxwell

As illustrated in Table 5, the rates of participants identifying the case as a mental health disorder and their inclination to recommend professional help as the primary treatment option remained consistently high in both the pre-test and post-test; no significant change was observed in these fundamental attitudes throughout the study.

The comparative analysis of posters produced by graphic design students revealed that core design components—such as color palette, typography, and visual layout—remained largely consistent across both phases. However, a structured semi-quantitative content analysis identified a significant shift at the conceptual and narrative levels. By cat-

pointed toward a less stigmatizing and more biologically informed understanding of schizophrenia. These findings underscore that while technical competencies in design education are essential, psychoeducation plays a critical role in fostering the narrative and ethical representation skills necessary for communicating complex mental health issues. This evolution in conceptual depth, rather than mere aesthetic modification, highlights the intervention's success in shifting student perspectives from clinical stereotypes to more nuanced, human-centered portrayals.

Table 5: Participants' identification of the condition and recommended first action (pre-test vs. post-test)

Variables	**Pre-test (%)**	**Post-test (%)**
Identification of Mr. Ahmet's condition as a **mental health disorder**	95.1	93.8
Recommendation of a **mental health professional or medical doctor** as the first step	86.4	86.3

DISCUSSION

This study examined the attitudes and social distance tendencies of students from the Department of Graphic Design at Kocaeli University, Faculty of Fine Arts, toward schizophrenia and evaluated the effects of a short-term psychoeducational intervention on these attitudes. The findings indicate a significant decrease in participants' social distance and stigmatizing beliefs toward individuals diagnosed with schizophrenia following the training. This suggests that the stigma surrounding mental illness is not a fixed construct but rather one that can be modified through appropriate educational interventions (5).

The observed decrease in SDS scores suggests that the intervention may have had a favorable influence on participants' immediate knowledge and behavioral tendencies, a finding consistent with literature highlighting the effectiveness of psychoeducation in reducing social distance among young adults (18). However, while even a single-session training produced a measurable shift in the narrative and conceptual depth of the students' poster designs, these results should be interpreted with caution (13). Although the intervention led to a discernible transformation in visual storytelling, it remains unclear whether these changes reflect a lasting reduction in personal stigma or a temporary response to the educational content. Consequently, while the study underscores the potential value of implementing such programs in university settings, further research is needed to determine the durability of these effects and whether such conceptual shifts in design translate into sustained attitudinal changes over time.

Participants' tendency to attribute schizophrenia primarily to biological and early developmental factors suggests that the illness was not predominantly perceived as a personal flaw. The relatively low endorsement of explanations based on personality weaknesses further indicates a reduction in accusatory attitudes. However, previous research emphasizes that biological explanations do not necessarily eliminate stigma and may, in some cases, reinforce perceptions of chronicity or dangerousness (19).

The data further show that participants most frequently explained Ahmet Bey's condition in terms of changes in brain biochemistry (34%). In the post-test, agreement with the items "Genetic factors" and "Weak personality structure" increased. Greater endorsement of genetic factors may reflect improved knowledge regarding the etiopathogenesis of schizophrenia. Nevertheless, the increased attribution to personality traits was unexpected. This finding suggests that certain misconceptions may persist despite the intervention and underscores the importance of continuous and structured educational efforts.

Another striking finding of this study is that even after the intervention, the participants continued to harbor reservations about establishing close social relationships with individuals diagnosed with schizophrenia. The persistent high rates of reluctance to marry someone with schizophrenia or to rent a house for such individuals demonstrate that social distance intensifies as the level of intimacy in the social relationship increases. This indicates that stigma is not merely a product of a lack of information but is deeply intertwined with complex emotional and cultural factors (20).

The fact that a vast majority of participants indicated that individuals with schizophrenia should consult a mental health professional signifies a positive attitude toward professional help-seeking behavior. The prominence of recommendations for referral to psychologists and psychiatrists reflects the collective perception of schizophrenia as a medical condition requiring specialized expertise. Furthermore, the relatively high emphasis placed on art therapy can be interpreted as a unique finding of this study, likely influenced by the fact that it was conducted within a Faculty of Fine Arts, where students were more attuned to creative therapeutic modalities.

Although not reaching statistical significance, the observation that participants with previous experience seeking psychiatric help had lower social distance scores aligns with the literature emphasizing the 'contact hypothesis'—the idea that direct or indirect contact with individuals with mental illness reduces stigma (21). Finally, the finding that partic-

ipants primarily rely on the Internet and social media for information regarding mental disorders highlights the urgent need for structured, evidence-based mental health education within university curricula to counteract the potential misinformation found online (22).

While this study provides valuable insights, its findings are limited by the absence of a randomized control group, the brief nature of the single-session intervention which resulted in non-significant shifts in etiological beliefs, and the limited methodological detail regarding the standardized criteria used for the qualitative poster analysis , including category development and inter-rater reliability, suggesting that future longitudinal programs are necessary to achieve a more profound reduction in stigma. Also, the use of a convenience sample from a single university restricts generalizability. Finally, the use of self-report measures may introduce response bias, including social desirability effects. Future studies should employ controlled, longitudinal designs with more diverse samples and more standardized qualitative procedures.

This exploratory study indicates that while psychoeducation-based interventions may reduce social distance among university students, the persistence of stigma in high-intimacy contexts highlights the complexity of such attitudes. These preliminary findings suggest that integrating informative and contact-based approaches could be beneficial, though the results should be interpreted cautiously due to the study's design limitations. For arts education, developing sustained, experience-based programs remains a potential pathway for fostering more inclusive perspectives and reducing long-term stigma.

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Postural instability in children with specific learning disorder: A neurodevelopmental perspective

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SUMMARY

Objective: This study examined whether postural instability in children with Specific Learning Disorder (SLD) is associated with cognitive-attentional processes or broader motor involvement.

Method: Seventy children aged 7–11 years participated in the study, including 41 children with SLD and 29 typically developing controls. Postural stability was assessed using the NeuroCom Balance Master and the Modified Clinical Test of Sensory Interaction on Balance (mCTSIB) across four conditions: hard surface eyes open (HSEO), hard surface eyes closed (HSEC), foam surface eyes open (FSEO), and foam surface eyes closed (FSEC). Attention was measured using the Swanson, Nolan, and Pelham Scale–IV (SNAP-IV), and cognitive performance was estimated using the Vocabulary and Block Design subtests of the Wechsler Intelligence Scale for Children–Revised (WISC-R). Parent-reported motor coordination difficulties were assessed using the Developmental Coordination Disorder Questionnaire–2007 (DCDQ'07). Group differences were examined using independent samples tests, followed by ANCOVA models adjusting for attentional symptoms, verbal ability, and parent-reported motor coordination difficulties. Hierarchical multiple regression analyses were conducted to identify predictors of postural performance.

Results: Children with SLD demonstrated significantly greater postural sway across all balance conditions (all $p < .001$). Group differences remained significant after adjusting for attentional symptoms and verbal ability. Additional ANCOVA models including DCDQ'07 scores also indicated persistent group differences in postural control parameters. Hierarchical regression analyses showed that SLD diagnosis was significantly associated with postural instability across conditions, whereas BMI was associated only in the HSEO condition.

Discussion: Postural control difficulties were more frequently observed in children with SLD compared with typically developing peers, even after adjusting for attentional symptoms, verbal abilities, and parent-reported motor coordination difficulties. However, given the cross-sectional design and potential confounding factors, including ADHD comorbidity and socioeconomic differences, these findings should be interpreted cautiously. Overall, the results suggest that postural instability may be related to broader neurodevelopmental mechanisms involving sensorimotor integration.

Key Words: specific learning disorder, postural instability, attention-deficit/hyperactivity disorder

INTRODUCTION

Specific Learning Disorder (SLD) is defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) as a neurodevelopmental condition characterized by persistent difficulties in learning and using academic skills such as reading, writing, or mathematics, despite adequate intelligence, instruction, and cultural opportunity (1). In clinical practice, children often present with mixed learning difficulties across reading, spelling, and arithmetic, reflecting the heterogeneous nature of SLD (2). Comorbidity is highly prevalent, with approximately 60% of individuals with dyslexia meeting the criteria for at least one neuropsychiatric disorder. Among these, ADHD is the most common comorbidity, whereas findings regarding associations with anxiety and mood disorders remain inconsistent across studies (3). These observations suggest that learning disorders extend beyond isolated linguistic deficits and may

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involve broader impairments in attention, executive functioning, and sensorimotor coordination (4).

Postural stability—the ability to maintain the body's center of mass within the base of support—depends on the efficient integration of visual, vestibular, and proprioceptive inputs within cerebellar and cortical motor networks (5). Disruptions in these systems can lead to balance instability and inefficient motor responses. Children with neurodevelopmental disorders, including SLD frequently display subtle motor coordination and balance difficulties (6,7). Several theories propose that cerebellar and sensorimotor connectivity abnormalities contribute to these impairments. Specifically, cerebellar dysfunction may hinder the automatic coordination of sensory and motor information, reducing the ability to compensate for postural disturbances and affecting the automatization of reading-related skills (6, 8, 9,10). Such deficits can manifest as difficulties in balance, ocular fixation, or fine motor tasks (11). The cerebellum therefore plays a central role linking motor and cognitive domains, and postural instability may represent a behavioral marker of broader cerebellar inefficiency in SLD (12).

In addition to neurobiological mechanisms, attention and body composition can influence postural control. Children with SLD frequently present with attentional deficits, and those with combined motor and attention problems show greater instability (13). Likewise, higher body-mass index (BMI) has been associated with increased body sway and delayed postural adjustments, suggesting that biomechanical factors may exacerbate balance difficulties (14). These interactions highlight the importance of considering attentional and physical variables when evaluating postural performance in SLD.

Although postural instability has been reported in specific learning disorders, findings remain inconsistent. Some studies have demonstrated significant increases in body sway or center-of-pressure displacement, whereas others have found no differences compared to typically developing peers. Previous research has investigated postural control in dyslexic children using diverse approaches, including large-scale cohorts, task-based paradigms, and intervention studies (15,16,18). However, few studies have examined the influence of covariates such as attention and intelligence on postural performance. The present study therefore aimed to compare static and dynamic postural control under two visual conditions (eyes open and eyes closed) in children with and without SLD, while controlling for attentional and intellectual factors to determine whether postural instability represents a secondary consequence of cognitive deficits or an independent marker of motor involvement.

METHOD

Participants

The study included 70 children aged 7–11 years, comprising 41 with SLD and 29 typically developing controls. To form the SLD group (SLDG), all files of children (n = 790) registered with a diagnosis of SLD in the hospital registry system were reviewed with the permission of hospital management. Among these, 529 children aged 7–11 years were screened, and 138 children who had been followed up in the child psychiatry clinic between 2018 and 2019 were included in the preliminary pool. Sixty-four children whose contact information was up to date and who met DSM-5 criteria for SLD were successfully reached by telephone. After the children and their parents were informed about the study, 43 children agreed to participate and were scheduled for assessments at the child psychiatry and physical therapy and rehabilitation clinics. Two children who did not attend their appointments were excluded, resulting in a final sample of 41 participants.

For the control group (CG), 40 typically developing children were initially recruited from local primary schools in the same geographical area and comparable socioeconomic backgrounds. However, 11 children did not attend the scheduled evaluation sessions due to logistical reasons (e.g., scheduling conflicts or family availability) and were therefore excluded, resulting in a final sample of 29 participants. Group matching was used to ensure compa-

rable mean age and sex distribution between the SLD and control groups. Control participants were free of learning problems, as confirmed by parent and teacher reports and a brief clinical interview.

Exclusion criteria for both groups included neurological or chronic medical conditions, physical disability, intellectual disability, autism spectrum disorder, psychotic disorder, adoption, or institutionalization. All participants were native Turkish speakers and attended regular educational settings representative of the urban school-age population served by the clinic. The SLDG comprised 15 girls (36.6%) and 26 boys (63.4%), while the control group included 10 girls (34.5%) and 19 boys (65.5%).

The study was approved by the Clinical Research Ethics Committee of the Marmara University School of Medicine (Decision No: 09.2018.205) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the parents, and verbal assent was obtained from all participating children prior to enrollment.

Procedure

The parents of the children were contacted by telephone and scheduled for appointments at the child psychiatry clinic and the physical therapy and rehabilitation clinic. A child and adolescent psychiatrist conducted mental status examinations of 41 children in the SLD group and 29 children in the CG, as well as their parents, using the K-SADS-PL. The diagnosis of SLD was confirmed according to DSM-5 criteria through a structured clinical interview conducted by the same psychiatrist, together with a review of prior educational and clinical records when available.

During the psychiatric evaluation, which lasted approximately 45 minutes, parents completed a sociodemographic data form, the SNAP-IV, and the Developmental Coordination Disorder Questionnaire (DCDQ'07), a parent-report screening instrument designed to identify children at risk for developmental coordination difficulties. As the DCDQ'07 is a screening measure rather than a diagnostic tool, the results were interpreted in conjunction with the clinical evaluation. To estimate the children's intellectual level, the Vocabulary and Block Design subtests of the Wechsler Intelligence Scale for Children–Revised (WISC-R) were administered.

Following the psychiatric assessment, each child underwent postural control and balance testing at the physical therapy and rehabilitation clinic. These assessments were conducted by a physical therapy specialist and lasted approximately 30 minutes. Body mass index (BMI) was also measured at the same clinic while the children were wearing light indoor clothing and no shoes.

All evaluations were conducted individually in a quiet clinical setting and followed the same standardized order for all participants to ensure procedural consistency. Each session was completed within approximately 75 minutes in total. Both the children and their parents were informed about the procedures and instructed on how to proceed.

Materials

Sociodemographic Questionnaire

Parents completed a questionnaire providing information on the child's age and sex, parental education level, number of family members, and family income.

Wechsler Intelligence Scale for Children–Revised (WISC-R): In the present study, the WISC-R was administered to both groups to estimate full-scale IQ scores (19). The Turkish adaptation of the WISC-R was developed by Savaşır and Şahin (20). The WISC-R comprises two main sections—verbal and performance—each including six subtests assessing distinct aspects of intelligence. In this study, only the Vocabulary and Block Design subtests were administered. The Block Design subtest consists of 11 items that assess visual discrimination, organization, visuomotor coordination, and processing speed. The Vocabulary subtest measures the ability to define and describe words and concepts, reflecting verbal reasoning and long-term

memory. Estimated IQ scores were calculated based on the formula derived from the Vocabulary and Block Design subtests (21).

Swanson, Nolan, and Pelham Scale–IV (SNAP-IV): The Swanson, Nolan, and Pelham Scale–IV (SNAP-IV), developed as part of the Multimodal Treatment Study for Attention-Deficit/Hyperactivity Disorder (MTA), was completed by parents (22). The first 18 items assess ADHD symptoms based on DSM-IV criteria—nine for inattention and nine for hyperactivity/impulsivity—rated on a 4-point Likert scale from 0 (not at all) to 3 (very much). Total scores, inattention subscale scores, and hyperactivity/impulsivity subscale scores were calculated and labeled as SNAP-Total, SNAP-I, and SNAP-H/I, respectively.

Developmental Coordination Questionnaire (DCDQ'07): Motor coordination was assessed using the parent-report Developmental Coordination Questionnaire (DCDQ'07) (23). The DCDQ'07 is a 15-item, 5-point Likert-type screening tool for children aged 5–15 years, evaluating motor performance across three domains: Control During Movement, Fine Motor/Handwriting, and General Coordination. Lower scores indicate greater motor difficulties. The revised version has demonstrated good internal consistency (Cronbach's $\alpha > .85$) and satisfactory sensitivity and specificity. The Turkish version has been shown to have acceptable validity and reliability in pediatric samples (24). As a screening instrument, DCDQ'07 results were interpreted alongside comprehensive clinical evaluation and additional assessment measures.

Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-PL): Parents and children were interviewed using the Turkish version of the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime Version (K-SADS-PL-T) to identify current and lifetime psychiatric diagnoses. The K-SADS-PL is a semi-structured diagnostic interview developed by Kaufman et al. (1997) and adapted to Turkish by Gökler et al. (25,26). It assesses present and past psychopathology in children and adolescents through interviews with both the child and parent, followed by a clinician's best-estimate diagnosis. Diagnoses not covered by the K-SADS-PL-T were determined through a clinical interview based on DSM-5 diagnostic criteria.

NeuroCom Balance Master

Postural balance was assessed using the NeuroCom Balance Master® system with the Modified Clinical Test of Sensory Interaction on Balance (mCTSIB). This method has been shown to be reliable for evaluating postural control in healthy children aged 9–11 years (27, 28).

Each child's postural sway was evaluated under four experimental conditions:

1. Hard surface, eyes open (HSEO)
2. Hard surface, eyes closed (HSEC)
3. Foam surface, eyes open (FSEO)
4. Foam surface, eyes closed (FSEC)

The hard surface condition involved standing directly on the force plate, while in the foam surface condition, a 30 × 30 cm foam pad was placed on the plate to reduce somatosensory input. Participants stood upright during each condition while the system recorded postural oscillation velocity (°/s). Each condition was repeated three times, and the mean oscillation rate was used for analysis (29). Postural sway velocity (°/s) was automatically calculated by the system, with higher values indicating greater body sway and poorer postural stability. This parameter reflects the participant's ability to integrate visual, vestibular, and somatosensory inputs to maintain balance under varying sensory conditions.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 22.0 (IBM Corp., Armonk, NY, USA). The adequacy of the sample size was verified through an a priori power analysis using G*Power. Assuming a medium effect size ($d = 0.5$), $\alpha = 0.05$, and desired power of 0.72, a total sample of 70 participants was determined to

Table 1. Sociodemographic characteristics of the SLD and control groups

Variable	SLDG (n = 41) Mean ± SD	CG (n = 29) Mean ± SD	Test / p
Age (years)	9.41 ± 0.92	9.70 ± 1.09	Z = −0.870, p = .420
Maternal age (years)	35.80 ± 5.41	35.45 ± 3.44	t = 0.312, p = .756
Paternal age (years)	41.39 ± 7.36	39.10 ± 3.45	Z = −0.717, p = .473
Monthly per capita income (TL/month)	642.68 ± 479.11	1306.90 ± 1262.56	Z = −3.684, p < .001
Variable	N (%)	N (%)	
Maternal education level			
Primary school graduate	30 (73.2%)	16 (55.2%)	
High school graduate	8 (19.5%)	4 (13.8%)	
University graduate	3 (7.3%)	9 (31.0%)	x† = 6.735, p = .034
Paternal education level			
Primary school graduate	31 (75.6%)	11 (37.9%)	
High school graduate	9 (22.0%)	10 (34.5%)	
University graduate	1 (2.4%)	8 (27.6%)	x† = 13.356, p = .001

Note. Data are presented as mean ± standard deviation (SD) or n (%). Independent samples *t* tests were used for normally distributed continuous variables, whereas Mann–Whitney U tests were used for non-normally distributed variables. Z denotes the Mann–Whitney U test statistic; x† denotes the chi-square test statistic. Bold values indicate statistically significant differences ($p < .05$).

be sufficient.

The normality of continuous variables was examined using the Kolmogorov–Smirnov test. Depending on the distribution, between-group comparisons were performed using Student's t-test or the Mann–Whitney U test. The chi-square (χ^2) test was used for categorical variables.

Bivariate Pearson correlation analyses were conducted to examine the relationships among postural control parameters (HSEO, HSEC, FSEO, FSEC), WISC-R subtests (Vocabulary and Block Design), and SNAP-IV subscales (SNAP-I, SNAP-H/I, SNAP-Total). Partial correlation analyses were subsequently performed to evaluate the associations between postural control parameters and WISC-R scores while controlling for SNAP-I scores. To further examine group differences while controlling for potential confounders, two separate analyses of covariance (ANCOVA) were conducted with group (dyslexia vs. control) as the independent variable and postural control scores as the dependent variables. In Model 1, SNAP-I and the WISC-R Vocabulary subtest score were included as covariates. In Model 2, the DCDQ'07 total score was entered as a covariate to evaluate whether group differences in postural control remained after accounting for developmental coordination difficulties.

Finally, multiple linear regression analyses were conducted separately for each postural control parameter (HSEO, HSEC, FSEO, FSEC) to identify predictors of balance performance. Body mass index (BMI), SLD group status (SLD vs. control), and SNAP-I scores were entered as predictors in the models. Preliminary analyses confirmed that the assumptions of normality, linearity, multicollinearity, and homoscedasticity were not violated. All statistical tests were two-tailed, and $p < 0.05$ was considered statistically significant.

RESULTS

Demographics and Clinical Characteristics

Mean age in the SLDG was 9.41 ± 0.92 years versus 9.70 ± 1.09 years in the CG; the difference was not significant (Z = –0.870, p = .420). The proportion of girls was similar between groups (36.6% vs. 34.5%; $\chi^2 = 0.033$, p = .856). Mean maternal and paternal ages did not differ significantly (maternal: t = 0.312, p = .756; paternal: Z = –0.717, p=.473). Monthly per capita income was significantly lower in the SLDG (642.68 ± 479.11 TL) than in the CG (1306.90 ± 1262.56 TL; Z=–3.684, p<.001). Fathers in the CG had higher education levels (χ^2 =13.356, p=.001), whereas maternal education also differed significantly between groups (χ^2= 6.735, p=.034). Detailed sociodemographic and physical characteristics are presented in Tables 1 and 2. Table 2 presents the anthropometric characteristics of participants. Mean BMI was significantly higher in girls in the SLDG than in those in the control group (t=2.140, p=.044), while boys in the CG were significantly taller and had higher height percentiles (t=–2.583, p=.013; Z=–1.999, p =.046).

In the SLDG (n = 41), 34 participants (82.9%) had attention-deficit hyperactivity disorder (ADHD), 19 (46.3%) had an anxiety disorder, 17 (41.5%) had

Table 2. Sex-specific anthropometric characteristics of the SLD and control groups

Variable	SLDG (Mean ± SD)	CG (Mean ± SD)	Test / p
Boys			
Height (cm)	134.11 ± 9.17	140.78 ± 7.63	t = -2.583, p = .013
Height percentile	43.10 ± 29.55	63.67 ± 36.87	t = -2.077, p = .044
Weight (kg)	34.69 ± 9.46	37.98 ± 7.88	t = -1.233, p = .224
Weight percentile	57.29 ± 33.44	68.86 ± 30.31	t = -1.192, p = .240
BMI (kg/m†)	19.10 ± 3.52	19.03 ± 2.80	t = 0.078, p = .938
Girls			
Height (cm)	135.33 ± 8.13	140.22 ± 11.50	t = -1.220, p = .235
Height percentile	49.82 ± 30.51	51.69 ± 34.37	Z = -0.030, p = .976
Weight (kg)	37.60 ± 11.07	33.97 ± 5.63	Z = -0.628, p = .530
Weight percentile	70.34 ± 26.59	48.58 ± 29.19	t = 1.872, p = .075
BMI (kg/m†)	20.21 ± 3.99	17.21 ± 1.57	t = 2.140, p = .044

Note. Data are presented as mean – standard deviation (SD). Independent samples *t* tests were used for normally distributed variables, whereas Mann□Whitney U tests (reported as Z statistics) were used for non-normally distributed variables. BMI = body mass index.

developmental coordination disorder (DCD), 7 (17.1%) had oppositional defiant disorder (ODD), 3 (7.3%) had a tic disorder, and 1 (2.4%) had depressive disorder, whereas in the CG (n = 29), 5 participants (17.1%) had ADHD, 9 (31.0%) had an anxiety disorder, and 1 (3.4%) had depressive disorder. ADHD ($\chi^2 = 22.52$, $p < .001$), ODD ($\chi^2 = 5.48$, $p = .02$), and DCD ($\chi^2 = 15.88$, $p < .001$) were significantly more frequent in the SLD group, whereas anxiety, tic, and depressive disorders did not differ significantly between groups (all $p > .05$). Overall, 36 participants in the SLD group (87.8%) and 4 participants in the control group (13.8%) had at least one comorbid psychiatric diagnosis ($\chi^2 = 37.99$, $p < .001$).

The control group obtained significantly higher scores on the WISC-R Vocabulary and Block Design subtests and on the estimated IQ. The SLD group had significantly higher scores on the SNAP-I, SNAP-H/I, and SNAP-Total subscales (all $p < .01$). The SLD group also demonstrated significantly lower DCDQ'07 total scores compared with the control group ($p < .001$), suggesting greater developmental coordination difficulties in the SLD group. Postural control performance differed significantly between groups across all conditions of the mCTSIB. The SLD group demonstrated higher sway velocities (poorer stability) in both hard-surface (HSEO, HSEC) and foam-surface (FSEO, FSEC) conditions (all $p < .001$). Corresponding effect sizes (Cohen's $d = 0.70–1.63$) indicated large between-group differences. Higher sway velocity scores on the mCTSIB indicate greater postural instability and reduced sensory integration efficiency. Descriptive and comparative results are summarized in Table 3.

Table 3. Comparison of cognitive, attentional, motor coordination, and postural control measures between the SLD and control groups

Variable	SLDG (Mean ± SD)	CG (Mean ± SD)	Statistical analysis	Cohen□s d
WISC-R Vocabulary subtest (raw score)	6.75 ± 2.37	11.44 ± 2.52	t = -7.897, p < .001	1.93
WISC-R Block Design subtest (raw score)	10.50 ± 2.66	14.02 ± 3.17	Z = -4.200, p < .001	1.19
WISC-R Verbal score	77.92 ± 16.15	109.86 ± 16.90	t = -7.993, p < .001	1.89
WISC-R Performance score	102.56 ± 17.99	126.27 ± 20.21	Z = -4.263, p < .001	1.23
Estimated IQ	89.66 ± 12.43	120.86 ± 17.73	Z = -5.972, p < .001	2.03
SNAP-I	13.43 ± 4.78	5.62 ± 5.67	Z = -5.135, p = .001	1.46
SNAP-H/I	9.78 ± 6.09	5.89 ± 4.56	Z = -2.748, p = .006	0.70
SNAP-Total	23.21 ± 9.25	11.51 ± 8.22	t = 5.453, p < .001	1.32
DCD-Q7	49.60 ± 12.56	64.75 ± 8.70	t = -5.606, p < .001	1.36
HSEO	0.75 ± 0.30	0.51 ± 0.12	Z = -4.086, p < .001	1.06
HSEC	0.96 ± 0.52	0.62 ± 0.15	Z = -3.457, p = .001	0.81
FSEO	1.58 ± 0.52	0.88 ± 0.24	Z = -5.572, p < .001	1.63
FSEC	2.07 ± 0.62	1.34 ± 0.25	Z = -4.772, p < .001	1.41

Note. WISC-R=Wechsler Intelligence Scale for Children-Revised; SNAP-IV = Swanson, Nolan, and Pelham Rating Scale□IV; SNAP-I =Inattention subscale; SNAP-H/I = Hyperactivity/Impulsivity subscale; SNAP-Total = Total score; DCDQ□07≠Developmental Coordination Disorder Questionnaire-2007; HSEO = Hard surface-eyes open; HSEC=Hard surface-eyes closed; FSEO=Foam surface-eyes open; FSEC = Foam surface-eyes closed. Independent samples t tests were used for normally distributed continuous variables, whereas Mann-Whitney U tests (reported as Z values) were used for non-normally distributed variables. (0.20 = small, 0.50 = medium, 0.80 = large).

Table 4. Pearson correlation coefficients (r) between postural control parameters and cognitive, attentional, and motor measures

Measure	WISC-R Vocabulary subtest score	WISC-R Block Design subtest score	SNAP-I	SNAP-H/I	SNAP-Total	DCD-Q
HSEO	r = -.318, p = .008	r = -.153, p = .211	r = .300, p = .012	r = .267, p = .026	r = .330, p = .005	*r = -0.304 p = 0.010*
HSEC	r = -.337, p = .005	r = -.223, p = .066	r = .298, p = .012	r = .110, p = .364	r = .243, p = .043	*r = -0.252 p = 0.036*
FSEO	r = -.457, p < .001	r = -.308, p = .010	r = .452, p < .001	r = .295, p = .013	r = .438, p < .001	*r = -0.364 p = 0.002*
FSEC	r = -.460, p < .001	r = -.253, p = .036	r = .257, p = .032	r = .179, p = .139	r = .255, p = .033	*r = -0.309 p = 0.009*

Note. ADHD = Attention-Deficit/Hyperactivity Disorder; HSEO = Hard Surface-Eyes Open; HSEC = Hard Surface-Eyes Closed; FSEO = Foam Surface-Eyes Open; FSEC = Foam Surface-Eyes Closed; SNAP-I = Swanson, Nolan, and Pelham Scale Inattention Subscale; SNAP-H/I = Swanson, Nolan, and Pelham Scale-Hyperactivity/Impulsivity Subscale; SNAP-Total = Swanson, Nolan, and Pelham Scale-Total Score. DCDQ 07 Developmental Coordination Disorder Questionnaire-2007. Values represent Pearson correlation coefficients (r). p < .05 indicates statistical significance.

Correlations Between Postural Control, Cognitive Performance, Attention, and Motor Coordination

Pearson correlation analyses (Table 4) were performed for the combined sample (N = 70). Poorer postural stability (higher sway velocity) was significantly associated with lower WISC-R Vocabulary subtest scores (r = –.46 to –.32, p < .01) and, to a lesser extent, with lower Block Design scores (r = –.31 to –.25, p < .05). In addition, higher sway velocity was positively correlated with SNAP-I and SNAP-Total scores (r = .24 to .45, p < .05), indicating that greater attentional difficulties were associated with poorer postural control. Postural sway parameters were also negatively correlated with DCDQ'07 scores (r = –.36 to –.25, p < .05), suggesting that children with greater developmental coordination difficulties exhibited poorer balance performance. When inattention (SNAP-I) was controlled for, the associations between postural control and Block Design scores became non-significant (all p > .10), whereas the negative association with Vocabulary scores remained significant under the FSEO and FSEC conditions (r = –.301, p = .013; r = –.396, p = .001). Correlation analyses were conducted for the combined sample to capture the full variability across participants and maximize statistical power.

ANCOVA Results: Postural Control After Adjustment for Attentional, Cognitive, and Motor Factors

To further examine whether group differences in postural control remained after adjusting for potential confounders, two separate analyses of covariance (ANCOVA) were conducted.

In the first model, SNAP-I and WISC-R Vocabulary subtest scores were included as covariates. After adjusting for these variables, the SLD group exhibited significantly greater postural sway than the control group in the HSEO ($F(1, 68) = 7.81$, $p = .007$, $\eta p^2 = .11$), FSEO ($F(1, 68) = 15.22$, $p < .001$, $\eta p^2 = .19$), and FSEC ($F(1, 68) = 16.17$, $p < .001$, $\eta p^2 = .20$) conditions. The difference in HSEC did not reach statistical significance ($F(1, 68) = 2.08$, $p = .155$, $\eta p^2 = .03$). In the second model, DCDQ'07 total scores were included as a covariate to account for parent-reported motor coordination difficulties, and the SLD group remained significantly different from the control group across all postural control parameters, including HSEO ($F(1, 67) = 8.99$, $p = .004$, $\eta p^2 = .12$), HSEC ($F(1, 67) = 7.22$, $p = .009$, $\eta p^2 = .10$), FSEO ($F(1, 67) = 28.91$, $p < .001$, $\eta p^2 = .30$), and FSEC ($F(1, 67) = 22.82$, $p < .001$, $\eta p^2 = .25$).

All ANCOVA assumptions were satisfied, including homogeneity of regression slopes (non-significant group × covariate interactions) and equality of variances as verified by Levene's test. These findings indicate that group differences in postural control remained significant after adjusting for attentional symptoms, verbal abilities, and parent-reported motor coordination difficulties. Importantly, the persistence of these differences after statistical adjustment suggests that postural control deficits in children with SLD may be associated with broader neurodevelopmental mechanisms related to sensorimotor integration; however, these findings should be interpreted cautiously given the presence of overlapping neurodevelopmental and psychiatric comorbidities.

Hierarchical Multiple Regression Analyses

Hierarchical regression results are presented in

Table 5. Hierarchical multiple regression analyses predicting postural control

Independent Variable	HSEO (Beta)	p	HSEC (Beta)	p	FSEO (Beta)	p	FSEC (Beta)	p
Constant	-	< .001	-	< .001	-	< .001	-	< .001
BMI	-0.277	.012	-0.135	.243	0.066	.496	0.073	.482
Diagnosis of SLD	0.471	< .001	0.324	.026	0.580	< .001	-0.662	< .001
SNAP-I	0.048	.719	0.141	.328	0.112	.356	-0.175	.176
Model summary	R^2 = .282	F(3,65)=8.489, p<.001	R^2 = .177	F(3,65)=4.659, p=.005	R^2= .417	F(3,65)=15.498, p<.001	R^2= .344	F(3,65)=11.344, p<.001
Adjusted R^2	.249		.136		.390		.315	

Note. Abbreviations: HSEO = Hard surface-eyes open; HSEC = Hard surface-eyes closed; FSEO = Foam surface-eyes open; FSEC = Foam surface-eyes closed; Beta = standardized regression coefficient; BMI = Body Mass Index; SNAP-I = Swanson, Nolan, and Pelham Scale-Inattention Subscale. Predictors were entered hierarchically into the regression models (Step 1: BMI; Step 2: SLD diagnosis [SLD = 1, control = 0]; Step 3: SNAP-I).

Table 5. Separate models were computed for each postural condition (HSEO, HSEC, FSEO, FSEC). Body mass index (BMI) was entered at Step 1, SLD diagnosis at Step 2 (dummy-coded: SLD = 1, control group = 0), and inattention (SNAP-I) at Step 3. All regression models were statistically significant: HSEO (R^2 = .28, F(3, 65) = 8.49, p < .001), HSEC (R^2 = .18, F(3, 65) = 4.66, p = .005), FSEO (R^2 = .42, F(3, 65) = 15.50, p < .001), and FSEC (R^2 = .34, F(3, 65) = 11.34, p < .001). Multicollinearity diagnostics indicated acceptable levels of collinearity among predictors (VIF < 2.0; tolerance > .50). In the HSEO model, both BMI (β = –.28, p = .012) and SLD diagnosis (β = –.47, p < .001) were significant predictors of postural performance. In the FSEO and FSEC models, only SLD diagnosis remained a significant predictor (β = –.58 and β = –.66, respectively; both p < .001). SNAP-I did not significantly predict postural control performance in any of the models (all p > .10).

Overall, the models explained between 18% and 42% of the variance in postural control measures. The consistent predictive effect of SLD diagnosis across postural conditions highlights its clinical relevance as a determinant of balance performance.

Post-hoc power analyses conducted using G*Power 3.1 for between-group comparisons, correlational analyses, and regression models indicated achieved statistical power (1–β) ranging from .88 to .99 for large effects, confirming adequate sensitivity to detect meaningful group differences. Taken together, these findings suggest that SLD diagnosis represents the most consistent predictor of postural control performance across different sensory conditions.

DISCUSSION

The present study compared postural stability between children with SLD and typically developing controls and further examined factors associated with postural control. In addition to cognitive and attentional measures, developmental coordination difficulties were also considered in the analyses. In our sample, postural control parameters were associated with several neurocognitive factors, including verbal abilities (Vocabulary subtest of the WISC-R), visuospatial abilities (Block Design subtest), attention scores, and motor coordination (DCDQ'07 scores). When attentional symptoms were controlled for, the association between visuospatial abilities and postural control was no longer significant, whereas the relationship between verbal abilities and postural control persisted under foam surface conditions. Importantly, group differences in postural stability remained observable after adjusting for attentional symptoms, verbal abilities, and developmental coordination scores; however, these findings should be interpreted within the context of a clinically complex and comorbid sample.

From a broader neurodevelopmental perspective, motor coordination and sensorimotor integration difficulties are increasingly recognized as shared features across multiple neurodevelopmental conditions. Contemporary models such as the ESSENCE framework emphasize that cognitive, behavioral, and motor difficulties frequently co-occur across disorders rather than representing entirely distinct diagnostic entities (Gillberg, 2010).

A large population-based cohort study of approximately 13,000 children aged 10–11 years who were screened for SLD reported that potentially affected

children showed weaker motor skills, even after accounting for literacy levels. More recent evidence also supports the presence of motor difficulties in children with dyslexia. A recent meta-analysis reported that children and adolescents with dyslexia show significantly poorer performance across multiple motor tasks compared with typically developing peers, highlighting the close relationship between motor and academic functions (31). Similarly, McPhillips and Sheehy (2004) observed that children with poor reading skills exhibited impairments in static and dynamic balance tasks, although these were not significantly associated with a diagnosis of SLD (32). Given these findings, researchers have increasingly examined postural stability in children with SLD to better understand the interplay between cognitive and motor functions.

Studies using center-of-pressure (CoP) measures have shown that postural sway along the anteroposterior and lateral axes is significantly greater in children with SLD (8,33). Consistent with these findings, previous studies have suggested that postural instability in dyslexia may reflect broader sensorimotor integration difficulties rather than deficits restricted solely to academic skills (34). Furthermore, all children, regardless of diagnosis, demonstrated improved postural stability when the distance to the visual fixation point was reduced (35). Legrand et al. (2012) reported that both SLD and control groups exhibited weaker postural control during a reading task, but this effect was significantly more pronounced in children with SLD (36). Similarly, tasks requiring attention and cognitive engagement, such as reading or visual fixation, have been shown to negatively affect postural control in SLD (17). The use of dual-task paradigms in SLD is based on the notion that such conditions minimize conscious compensatory strategies, thereby revealing subtle deficits in motor automaticity that are not evident under single-task conditions.

Consistent with these observations, children with SLD in the present study demonstrated greater postural impairments than those in the control group. Even after controlling for attentional processes, the relationship between verbal abilities and postural control remained significant under foam-surface conditions. Verbal abilities have previously been linked to motor difficulties (32), suggesting that attentional demands involved in reading and motion perception may influence CoP oscillations in both SLD and control groups. Consistent with previous findings, the current study showed that children with SLD performed worse than controls on most postural control parameters, even when attention and verbal ability scores were included as covariates (37). Importantly, these group differences remained significant after additionally adjusting for parent-reported motor coordination difficulties as assessed by DCDQ'07 scores, suggesting that the observed postural instability in the SLD group may not be entirely explained by attentional, cognitive, or motor coordination differences alone, although the contribution of overlapping neurodevelopmental and psychiatric comorbidities cannot be ruled out. Taken together, these findings suggest that balance impairments observed in children with SLD may reflect broader neurodevelopmental mechanisms rather than merely secondary consequences of attentional, cognitive, or motor coordination difficulties. It should also be noted that the DCDQ'07 is a parent-report screening instrument and does not provide a formal clinical diagnosis of developmental coordination disorder. Therefore, the present findings reflect adjustment for parent-reported motor coordination difficulties rather than confirmed DCD.

Importantly, the SLD group in the present study represents a clinically referred population with high rates of comorbid conditions, including ADHD and developmental coordination difficulties. Although statistical adjustments were applied, it is not possible to fully disentangle the specific contribution of SLD from overlapping neurodevelopmental and psychiatric features. In addition, socioeconomic differences between groups, including parental education and income levels, may have broader developmental implications, such as differences in access to structured activities and environmental stimulation, and residual confounding related to these factors cannot be excluded. Furthermore, although attentional symptoms were statistically controlled using SNAP-I scores, this approach reflects dimensional symptom severity rather than categorical diagnosis and therefore does not fully capture the broader clinical impact of

comorbid ADHD, which was more prevalent in the SLD group. Accordingly, the findings should be interpreted cautiously and are more reflective of a clinically complex SLD sample rather than a disorder-specific effect.

A notable finding of this study was that BMI and SLD diagnosis were associated with postural stability in the HSEO condition, explaining approximately one-third of the variance in the model. While BMI was significant only in this specific condition, SLD diagnosis remained a significant statistical predictor of postural performance across postural conditions, particularly on the foam surface, where it explained up to 50% of the variance. Previous studies have reported that body weight and height influence postural control in children and that BMI may be a stronger predictor of balance when visual information is available (14). In line with these findings, BMI affected postural control under hard-surface, eyes-open conditions but lost significance in more challenging conditions (e.g., foam surface or eyes closed). These findings suggest that postural imbalance in SLD may involve complex multisensory processes requiring increased integration of sensory inputs to maintain stability. According to the cerebellar deficit hypothesis proposed by Nicolson, Fawcett, and Dean (2001), motor coordination and balance difficulties in dyslexia may be related to broader alterations in cerebellar-mediated sensorimotor integration rather than deficits limited to reading processes (38). These mechanisms warrant further investigation in larger and longitudinal studies.

From a clinical perspective, these findings suggest that motor and sensorimotor characteristics may represent part of the broader neurodevelopmental profile of children with SLD rather than merely secondary consequences of academic or attentional difficulties. Assessing motor and balance functions may therefore provide additional insight into the neurodevelopmental characteristics of children with SLD and contribute to more comprehensive clinical evaluations. In addition, the findings highlight the potential importance of screening for motor coordination difficulties, such as developmental coordination disorder, in children presenting with learning problems. Early identification of motor and balance difficulties may inform multidisciplinary assessment and support the development of targeted motor or sensorimotor interventions.

The present study has several limitations. First, the sample size was relatively small, which may limit the generalizability of the findings. Future studies with larger and more diverse samples are needed to confirm these results. Second, attentional symptoms were assessed using parental reports (SNAP-IV) rather than objective behavioral attention measures. Including performance-based attention assessments in future research may help to further clarify the relationship between attention and postural control. Third, visual and auditory factors that may influence postural control were not systematically evaluated. Sensory impairments can affect balance performance and may contribute to postural instability. In addition, postural control was not examined during reading or other dual-task conditions, which might provide additional insight into the interaction between cognitive load and postural regulation in children with SLD. Another limitation is that socioeconomic differences were observed between the groups, including parental education and monthly income levels. These factors may influence children's access to organized physical activities or sports participation, which could potentially affect balance performance. Information regarding participation in structured physical activity was not available in the present dataset and therefore could not be included in the analyses. Despite these limitations, the study also has several strengths. Postural control was assessed using an objective balance assessment system (NeuroCom Balance Master), and standardized measures of intelligence, attention, and motor coordination (DCDQ'07) were included, allowing several potential confounding factors to be considered in the analyses.

Given the substantial diagnostic overlap among neurodevelopmental disorders, postural impairment may not have diagnostic specificity for SLD and may instead reflect broader neurodevelopmental dysfunction. The present findings suggest that the relationship between SLD and balance is influenced by cognitive and attentional processes, including verbal abilities and attention. However, even after statistically controlling for these factors, balance difficulties remained more frequently

observed in children with SLD compared with typically developing peers within a clinically referred and comorbid sample. These findings support the view that postural instability may represent a transdiagnostic marker of sensorimotor integration difficulties rather than a disorder-specific feature, highlighting the potential value of broader cognitive and motor assessments in clinical evaluation (34).

Ethics Declarations

Conflict of Interest: The authors declare that they have no conflicts of interest, financial or otherwise, related to the material presented in this study.

Ethical Approval: The study was approved by the Clinical Research Ethics Committee of the Marmara University School of Medicine (Decision No: 09.2018.205) and was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from the parents, and verbal assent was obtained from all participating children prior to enrollment.

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Mentalization and Resilience: The mediating role of problem-solving

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SUMMARY

Objective: This study examined whether perceived problem-solving ability mediates the association between mentalization and resilience in non-clinical adults.

Method: A cross-sectional correlational design was used. The sample comprised 422 adults (222 women, 200 men; age range 18–55, M = 34.44, SD = 10.70). Data were collected with the Mentalization Scale (MentS), the Problem-Solving Inventory (PSI), and the Resilience Scale for Adults (RSA). The mediation model was tested with PROCESS Macro v5.0 (Model 4) using 5,000 bootstrap samples, and a sensitivity analysis controlling for age, gender, marital status, and income was also conducted. Higher PSI scores indicate lower perceived problem-solving ability.

Results: Mentalization was positively correlated with resilience ($r = .27$, $p < .001$) and negatively with PSI scores ($r = -.25$, $p < .001$), indicating that individuals with stronger mentalization reported better perceived problem-solving ability. PSI scores were negatively associated with resilience ($r = -.39$, $p < .001$). The total association between mentalization and resilience was significant ($B = 0.54$, $p < .001$). When problem-solving ability was added, the direct association remained significant but decreased ($B = 0.37$, $p < .001$), and the indirect association was significant ($B = 0.17$, 95% CI [0.08, 0.29]). The model explained 18% of the variance ($R^2 = .18$), and the pattern remained stable after demographic adjustment ($R^2 = .197$).

Discussion: Perceived problem-solving ability partially mediates the association between mentalization and resilience within a cross-sectional statistical model. Intervention programs aimed at strengthening resilience may benefit from addressing both mentalization and problem-solving.

Key Words: Mentalization, problem-solving, resilience, mediation analysis.

INTRODUCTION

Resilience refers to the ability to sustain adaptive functioning during prolonged stress, adverse life conditions, or traumatic experiences (1). This concept cannot be explained solely by the absence of risk factors; rather, it is associated with the effective use of protective resources within the individual and their surroundings. Friborg et al. (2) demonstrated that resilience is a multidimensional construct comprising six dimensions: 'perception of self', 'perception of future', 'structured style', 'social competence', 'family cohesion', and 'social resources'. As summarized in Friborg et al. (1), longitudinal studies have shown that resilient individuals tend to have an internal locus of control, optimism, and empathy, and are also able to use family and social support networks more effectively. Therefore, identifying the individual and environmental factors associated with resilience is important for preventive mental health interventions.

Mentalization is defined as "the mental process by which an individual implicitly and explicitly interprets the actions of himself and others as meaningful on the basis of intentional mental states such as personal desires, needs, feelings, beliefs, and reasons" (3, p.21). Although related to concepts like social cognition, empathy, and theory of mind, mentalization is a comprehensive process that, unlike these, encompasses both self-reflection and the understanding of others' mental states (4). Mentalization capacity has been reported to be positively linked to secure attachment patterns and

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negatively rela-ted to attachment anxiety and avoidance (4). This concept, long studied in clinical settings, has recently begun to be considered a protective factor in non-clinical populations as well (5).

The link between mentalization and resilience has been consistently confirmed across studies with diverse samples. Kealy et al. (6) found that, among 1,065 men reporting mental health issues, higher reflective functioning correlated with greater resilience and lower psychological distress. In studies conducted with healthcare workers, significant correlations between mentalization and resilience were reported (7,8), and mentalization has been implicated in resilience through its role in coping and stress regulation (9). Schwarzer et al. (5) demonstrated that mentalization acts as a mediator between stress levels and coping behavior among non-clinical adults; in a longitudinal study, they also found that mentalization predicted well-being 12 months later (10). Szegő and Szabó (11) and Maerz et al. (12) also reported that mentalization is linked to resilience. Taken together, these findings indicate that mentalization is associated with resilience; however, the mechanisms through which this relationship operates have not been sufficiently explored.

Problem-solving ability is an individual's skill to handle stressful situations using cognitive and behavioral strategies (13). Research supports a link between problem-solving and resilience. Among university students, resilience was positively associated with problem-solving (14-16). Another study (17) experimentally demonstrated that training to develop problem-solving skills significantly increased resilience among nursing students. These findings highlight that problem-solving skills serve as a cognitive resource associated with resilience.

The theoretical link between mentalization and problem-solving can be understood through the lens of metacognitive processes. Fonagy and Bateman (18) have argued that mentalization, as a higher-order cognitive function, enables individuals to reorganize their mental resources in stressful situations. Suominen (19) has discussed how metacognitive capacities support the development of resilience and that impairments in these capacities increase the risk of stress-related disorders. At the empirical level, Channon et al. (20) reported that individuals with mentalization difficulties also exhibit lower social problem-solving performance. This finding suggests that mentalization capacity may be related to processes that support problem-solving. A person's ability to accurately interpret their own and others' mental states may confer a cognitive advantage during the stages of problem identification and solution generation.

The literature summarized above shows that although the relationships between mentalization and resilience, as well as between problem-solving and resilience, have been demonstrated separately, research integrating these three variables into a single model remains limited. Specifically, studies directly examining the relationship between mentalization and problem-solving in non-clinical adults are scarce. Taken together, these findings suggest that problem-solving ability may represent a pathway through which mentalization is associated with resilience. This study investigates whether perceived problem-solving ability mediates the relationship between mentalization and resilience. The hypotheses are: (1) Mentalization is expected to predict lower PSI scores, indicating better perceived problem-solving ability. (2) Lower PSI scores are expected to predict higher resilience. (3) Perceived problem-solving ability is expected to mediate the association between mentalization and resilience.

METHOD

Research Design

This research was structured as a cross-sectional, correlational study to explore the mediating role of perceived problem-solving in the association between mentalization and resilience.

Population and Sample

A total of 422 volunteer adults, recruited through online convenience sampling, participated in the study. Participants were eligible if they were 18

years of age or older (with no upper age limit applied), volunteered to take part, provided informed consent, and completed all study measures. Respondents younger than 18, as well as duplicate submissions and cases showing careless or implausible response patterns, were excluded during data preparation. Participants were recruited from the general adult community through online platforms rather than from any clinical or psychiatric treatment setting; on this basis the sample was characterized as non-clinical. Because no standardized psychiatric screening instrument or structured diagnostic interview was administered, clinical status was not formally verified, and the term "non-clinical" is used here to denote a community-based, non-treatment-seeking sample.

Data Collection Tools

Sociodemographic Information Form: This form, prepared by the researcher, contains questions about participants' gender, age, education level, monthly income, marital status, and relationship status.

The Mentalization Scale (MentS): Developed by Dimitrijević et al. (4) to evaluate individuals' mentalization capacity. Törenli-Kaya et al. (21) conducted the Turkish adaptation of the scale. The Turkish version includes 25 items and uses a 5-point Likert scale. It comprises three subscales: 'self-related mentalization' (MentS-S), 'other-related mentalization' (MentS-O), and 'motivation to mentalize' (MentS-M). In the adaptation study, Cronbach's alpha coefficients were reported as .84 for the entire scale and .78, .80, and .79 for the subscales, respectively.

The Problem-Solving Inventory (PSI): Heppner and Petersen (13) developed the inventory to assess how individuals perceive their own problem-solving abilities. The Turkish adaptation was carried out by Şahin et al. (22). The inventory includes 35 items and employs a 6-point Likert scale. It consists of three subscales: 'problem-solving confidence', 'approach-avoidance style', and 'personal control'. Three items are not included in the scoring as filler items; therefore, the number of scored items is 32. Higher scores on the scale reflect a more negative perception of problem-solving ability. In the Turkish adaptation study, the Cronbach's alpha for the inventory's total score was reported as .88.

The Resilience Scale for Adults (RSA): It was developed by Friborg et al. (1-2) to assess protective resources in adults. The Turkish adaptation was conducted by Basım and Çetin (23). The scale includes 33 items and is administered using the semantic differential format. It comprises six subscales: 'perception of self', 'perception of future', 'structured style', 'social competence', 'family cohesion', and 'social resources' (2,23). Higher scores reflect stronger resilience. In the Turkish adaptation study, subscale internal consistency coefficients ranged from .66 to .81.

Data Collection Process

Ethical approval was granted on May 31, 2024, under Decision No. 63103. Data were gathered online through Google Forms from June to November 2024. Before completing the questionnaires, participants were briefed on the study's purpose, the principle of voluntariness, and data confidentiality, and they provided informed consent. Because respondents could not advance to the next section without completing the current one, all items functioned as required fields, which accounts for the absence of missing data on the study measures.

Data Analysis

Data were analyzed using IBM SPSS Statistics 27. Prior to the main analyses, the dataset was screened for duplicate entries and for careless or implausible response patterns (e.g., uniform responding across all items), as well as for missing values and multivariate outliers. Multivariate outliers were examined using Mahalanobis distance. Relationship status and relationship duration variables contained structurally missing values, as these questions were applicable only to participants who reported being in a romantic relationship; therefore, these variables were not included in the mediation analyses.

Table 1. Demographic Characteristics of the Participants

		n	%
Gender	Female	222	52.6
	Male	200	47.4
Age	18-25	109	25.8
	26-40	195	46.2
	41-55	118	28.0
Monthly Income	Low	118	28.0
	Middle	228	54.0
	High	76	18.0
Education Status	High School Graduate	81	19.2
	Undergraduate/Bachelor's degree	226	53.6
	Master's / PhD degree	115	27.3
Marital Status	Unmarried	253	60.0
	Married	169	40.0
Relationship Status*	In a relationship	189	44.8
	No relationship	64	15.2
Relationship Duration**	0-12 Months	20	4.7
	1- 3 Years	31	7.3
	3-5 Years	101	23.9
	Over 5 years	37	8.8
Total		422	100.0

* Relationship status was assessed among unmarried participants (n = 253); percentages are based on the total sample. ** Relationship duration was assessed among participants currently in a relationship (n = 189); percentages are based on the total sample.

Descriptive statistics, including means, standard deviations, skewness, and kurtosis values, were calculated for all study variables. Internal consistency was evaluated using Cronbach's alpha coefficients. Pearson correlation analysis was conducted to examine bivariate associations among the variables.

A post hoc power analysis and a complementary sensitivity analysis were conducted based on the adjusted regression model to evaluate the adequacy of the sample size for detecting the observed effects.

The mediation model was tested using PROCESS Macro v5.0, Model 4, with 5,000 bootstrap samples. The indirect effect was considered statistically significant when the 95% bootstrap confidence interval did not include zero. The mediation model was first estimated without covariates and subsequently re-estimated after controlling for age, gender, marital status, and income. This adjusted model was used to examine whether the indirect association between mentalization and resilience through perceived problem-solving ability remained significant after accounting for demographic covariates.

RESULTS

Sample Characteristics

The final analytic sample comprised 422 participants, and no missing data were observed for the variables included in the main analyses; therefore, no imputation procedure was applied. Among the participants, 222 (52.6%) were female and 200 (47.4%) were male. The mean age was 34.44 (SD = 10.70), with an age range of 18–55. By age group, 109 participants (25.8%) were aged 18–25, 195 (46.2%) were aged 26–40, and 118 (28.0%) were aged 41–55. In terms of educational level, 81 participants (19.2%) had a high school diploma, 226 participants (53.6%) had a bachelor's or associate's degree, and 115 participants (27.3%) held a master's or doctoral degree. By monthly income level, 118 participants (28.0%) were in the low-income group, 228 (54.0%) in the middle-income group, and 76 (18.0%) in the high-income group. In terms of marital status, 253 participants (60.0%) were unmarried, and 169 participants (40.0%) were married. The demographic characteristics of the participants are presented in Table 1.

The maximum Mahalanobis distance value was 12.88, which was below the critical chi-square value of 13.82 at df = 2 and $p < .001$, indicating that no influential multivariate outliers were detected.

A post hoc power analysis based on the adjusted model (six predictors; $R^2 = .197$, $f^2 = .245$) indicat-

Table 2. Descriptive Statistics and Reliability Coefficients

	n	Min	Max	M	SD	Kurtosis	Skewness	(a)
MentS	422	52	114	92.34	11.79	0.46	-0.89	0.73
MentS-M	422	12	40	29.51	5.42	1.31	-1.06	0.62
MentS-O	422	11	45	33.14	5.67	1.78	-1.15	0.61
MentS-S	422	11	39	29.69	5.31	1.61	-1.18	0.61
PSI	422	41	152	93.76	21.93	-0.42	0.08	0.92
Problem-solving confidence	422	12	55	30.69	9.30	-0.60	0.22	0.89
Approach-avoidance style	422	18	78	47.88	11.90	-0.29	-0.01	0.86
Personal control	422	5	26	15.20	4.22	-0.35	0.15	0.67
RSA	422	41	165	111.04	23.76	0.36	-0.55	0.92
Perception of self	422	6	30	21.10	5.11	-0.11	-0.46	0.74
Perception of future	422	4	20	13.14	4.20	-0.73	-0.32	0.79
Structured style	422	4	20	12.62	4.24	-0.49	-0.38	0.70
Social competence	422	6	30	20.27	5.59	-0.31	-0.43	0.76
Family cohesion	422	6	30	19.30	5.36	-0.07	-0.33	0.76
Social resources	422	7	35	24.60	6.32	-0.10	-0.63	0.79

MentS: The Mentalization Scale; MentS-M: Motivation to mentalize; MentS-O: Other-related mentalization; MentS-S: Self-related mentalization; PSI: The Problem-Solving Inventory; RSA: The Resilience Scale for Adults

Table 3. Pearson Correlation Coefficients Among Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1-MentS	1														
2-MentS-M	.72**	1													
3-MentS-O	.78**	.40**	1												
4-MentS-S	.65**	.15**	.27**	1											
5-PSI	-.25**	-.18**	-.19**	-.15**	1										
6-Problem-solving confidence	-.19**	-.17**	-.15**	-.09	.87**	1									
7-Approach-avoidance style	-.22**	-.14**	-.19**	-.15**	.91**	.62**	1								
8-Personal control	-.22**	-.18**	-.14**	-.15**	.72**	.56**	.54**	1							
9-RSA	.27**	.18**	.22**	.17**	-.39**	-.35**	-.35**	-.27**	1						
10-Perception of self	.22**	.17**	.15**	.17**	-.36**	-.30**	-.33**	-.26**	.78**	1					
11-Perception of future	.18**	.12*	.13**	.14**	-.32**	-.28**	-.28**	-.22**	.75**	.72**	1				
12-Structured style	.17**	.07	.13**	.17**	-.26**	-.25**	-.22**	-.16**	.73**	.54**	.60**	1			
13-Social competence	.20**	.18**	.15**	.09	-.31**	-.31**	-.27**	-.20**	.78**	.53**	.44**	.50**	1		
14-Family cohesion	.19**	.13**	.17**	.11*	-.25**	-.20**	-.23**	-.23**	.71**	.38**	.39**	.36**	.40**	1	
15-Social resources	.25**	.16**	.25**	.12*	-.30**	-.26**	-.29**	-.19**	.84**	.49**	.46**	.48**	.66**	.65**	1

*p < .05, **p < .01. Test used: Pearson correlation.
MentS: The Mentalization Scale; MentS-M: Motivation to mentalize; MentS-O: Other-related mentalization; MentS-S: Self-related mentalization; PSI: The Problem-Solving Inventory; RSA: The Resilience Scale for Adults

ed that the achieved power was approximately 1.00. A complementary sensitivity analysis (α=.05, power = .80, N = 422, six predictors) showed that the minimum detectable effect was f^2 = .033, indicating that the sample was adequately powered to detect even small effects.

Descriptive Statistics and Reliability

Descriptive statistics, normality values, and internal consistency coefficients for the scales are presented in Table 2. High scores on the PSI indicate a deficiency in perceived problem-solving ability; therefore, reverse-scoring must be taken into account when interpreting PSI scores (13).

As presented in Table 2, the mean total score on the MentS was 92.34 (SD=11.79), the mean total score for the PSI was 93.76 (SD=21.93), and the mean total score for the RSA was 111.04 (SD= 23.76). Skewness and kurtosis across all scales were within the ±2 range, suggesting the data adhere to a normal distribution (24). Regarding internal consistency, the Cronbach's alpha coefficient for the MentS total score was .73, and the subscale coefficients were .62 for MentS-M, .61 for MentS-O, and .61 for MentS-S. For the PSI, the Cronbach's alpha coefficient for the total score was .92, with subscale values of .89 for problem-solving confidence, .86 for approach-avoidance style, and .67 for personal control. For the RSA, the Cronbach's alpha coefficient for the total score was .92, and the subscale values were .74 for perception of self, .79 for perception of future, .70 for structured style, .76 for social competence, .76 for family cohesion, and .79 for social resources.

Correlations Among Variables

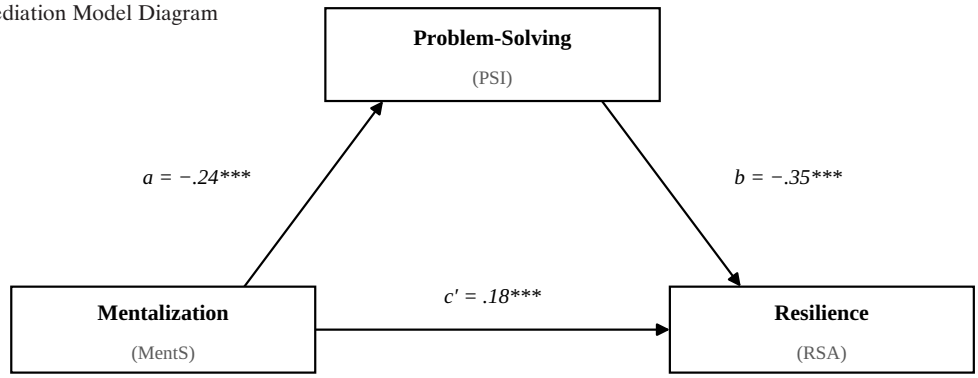
The Pearson correlation coefficients between the three main scales and their subscales are shown in Table 3. When interpreted in light of the reverse scoring of the PSI (high PSI score = low perceived

Table 4. Mediation Analysis Results: Unadjusted and Adjusted Models

Effect	Unadjusted model				Adjusted model			
	B (SE)	t	p	95% CI	B (SE)	t	p	95% CI
MentS -> PSI (Path a)	-0.46 (0.09)	-5.19	< .001	[-0.63, -0.28]	-0.44 (0.09)	-5.02	< .001	[-0.61, -0.27]
PSI -> RSA (Path b)	-0.42 (0.05)	-8.70	< .001	[-0.52, -0.33]	-0.38 (0.05)	-7.56	< .001	[-0.48, -0.28]
Total effect: MentS -> RSA (Path c)	0.54 (0.09)	5.66	< .001	[0.35, 0.72]	0.53 (0.09)	5.55	< .001	[0.34, 0.71]
Direct effect: MentS -> RSA (Path c')	0.37 (0.09)	3.99	< .001	[0.19, 0.55]	0.36 (0.09)	3.94	< .001	[0.18, 0.54]
Indirect effect: MentS -> PSI -> RSA (a x b)	0.17 (0.05)	-	-	[0.08, 0.29]	0.165 (0.049)	-	-	[0.081, 0.274]

Analysis was conducted using PROCESS Macro v5.0 (25) , Model 4. N = 422. MentS = Mentalization Scale; PSI = Problem- Solving Inventory; RSA = Resilience Scale for Adults.
The adjusted model controlled for age, gender, marital status, and income. Unadjusted model: R=.43, R^2 =.18, F (2, 419) = 47.10, p< .001. Adjusted outcome model: R = .444, R^2 =.197, F (6, 415) = 16.98, p < .001. Higher PSI scores indicate lower perceived problem-solving ability. All coefficients are unstandardized (B). Indirect effects are unstandardized bootstrap estimates based on 5,000 samples; statistical significance was evaluated using 95% bootstrap confidence intervals. Covariate coefficients are not displayed to preserve table readability.

Figure 1. Mediation Model Diagram



Covariates: age, gender, marital status, income

Indirect effect (*a* × *b*): *B* = .165, BootSE = .049, 95% CI [.081, .274]

Total effect (*c*): β = .26***

***$p < .001$

MentS = Mentalization Scale; PSI = Problem-Solving Inventory; RSA = Resilience Scale for Adults. Lower PSI scores indicate better perceived problem-solving ability. Path coefficients are standardized beta coefficients (β) from the adjusted mediation model controlling for age, gender, marital status, and income. The indirect effect is reported as an unstandardized bootstrap estimate. Arrows indicate statistical associations rather than causal effects.

problem-solving ability), the relationships among the variables are summarized below.

A significant negative correlation was observed between the total mentalization score and the total PSI score (r= -.25, p< .001). This finding indicates that participants with higher mentalization perceive themselves as more competent problem solvers. All three subdimensions of mentalization showed significant negative correlations with the total PSI score (MentS-M: r= -.18; MentS-O: r=-.19; MentS-S: r= -.15; all p < .01).

A significant positive correlation was identified between the total MentS score and the total RSA score (r= .27, p< .001). All mentalization subdimensions showed significant positive correlations with the total RSA score (MentS-M: r= .18; MentS-O: r= .22; MentS-S: r= .17; all p< .001).

A significant negative correlation was observed between the total PSI score and the total RSA score (r= -.39, p< .001). Given the PSI's reverse-scoring structure, this result indicates that participants who perceive themselves as skilled at solving problems exhibit high levels of resilience. All three PSI subscales showed significant negative correlations with the RSA total score (problem-solving confidence: r = -.35; approach-avoidance style: r = -.35; personal control: r= -.27; all p< .001).

Two exceptions stand out at the sub-dimension level: The correlations between MentS-S and the problem-solving confidence dimension of PSI (r= -.09, p= .054) and between MentS-S and the social competence dimension of RSA (r= .09, p= .071) were not statistically significant.

Mediational Analysis

The role of perceived problem-solving ability (mediator) in the association between mentalization (predictor) and resilience (outcome) was examined using PROCESS Macro v5.0, Model 4. The indirect effect was tested with 5,000 bootstrap samples. The results are presented in Table 4 and Figure 1.

The model was statistically significant (R= .43, R^2 = .18, F(2, 419)=47.10, p< .001). These results indicate that mentalization and perceived problem-solving ability together explain 18% of the variance in resilience.

Path a: Mentalization significantly and negatively predicted PSI scores, indicating better perceived

problem-solving ability (B= -0.46, SE= 0.09, β= -.25, t= -5.19, p< .001, 95% CI [-0.63, -0.28]). This finding indicates that higher mentalization is associated with lower PSI scores, reflecting better perceived problem-solving ability.

Path b: PSI scores, indicating better perceived problem-solving ability, significantly and negatively predicted resilience (B= -0.42, SE= 0.05, β= -.39, t = -8.70, p< .001, 95% CI [-0.52, -0.33]). Given the PSI's reverse scoring, participants who view themselves as more competent problem solvers also report higher levels of resilience.

Total effect (path c): The total effect of mentalization on resilience was positive and significant (B= 0.54, SE= 0.09, β= .27, t= 5.66, p< .001, 95% CI [0.35, 0.72]).

Direct effect (path c'): When perceived problem-solving ability was included in the model, the direct effect of mentalization on resilience decreased but remained significant (B= 0.37, SE= 0.09, β= .18, t = 3.99, p< .001, 95% CI [0.19, 0.55]).

Indirect effect (a×b): The bootstrap analysis showed that the indirect effect was statistically significant in the unadjusted model, B= 0.17, SE= 0.05, 95% CI [0.08, 0.29]. As shown in Table 4, the indirect effect also remained significant in the adjusted model controlling for age, gender, marital status, and income, B= 0.165, BootSE= 0.049, 95% CI [0.081, 0.274]. Because both confidence intervals did not include zero, the indirect association between mentalization and resilience through perceived problem-solving ability was statistically significant. The reduction of the total effect (β = .27) to the direct effect (β= .18), while the direct effect remained significant, is consistent with a partial mediation pattern.

DISCUSSION

This study explored how perceived problem-solving ability mediates the link between mentalization and resilience in non-clinical adults. Mediation analysis indicated that perceived problem-solving ability partially mediated this relationship (indirect effect B = 0.17, 95% CI [0.08, 0.29]). After the mediating variable was added to the model, the direct effect of mentalization on resilience remained significant (β = .18, p < .001) but was smaller than the total effect (β = .27). This finding indicates that mentalization predicts resilience both directly and indirectly through perceived problem-solving ability.

In the first phase of the model, mentalization level significantly predicted PSI scores, such that higher mentalization was associated with better perceived problem-solving ability (β=-.25, p < .001). Because high PSI scores reflect lower perceived ability, this negative relationship indicates that individuals with higher mentalization capacity tend to assess their problem-solving abilities as more competent. This finding aligns with previous research demonstrating a link between mentalization and social problem-solving performance. Channon et al. (20) showed that mentalization difficulties negatively affect social problem-solving performance in individuals with Asperger syndrome. In a non-clinical sample, Cerciello et al. (26) found that mentalization predicts hyperactivation of dominance behavior system, suggesting that mentalization plays a role in regulating interpersonal processes that may be relevant for problem-solving. From a theoretical perspective, mentalization supports problem identification, the generation of alternative solutions, and the evaluation of outcomes by enabling individuals to comprehend their own and others' mental states (18).

In the second stage of the model, perceived problem-solving ability was a significant predictor of resilience (β = −.39, p < .001). The direction of this relationship indicates that individuals who evaluate their problem-solving abilities more positively also possess higher levels of resilience. This finding aligns with prior research indicating that constructive problem-solving approaches are positively correlated with resilience, whereas negative approaches are negatively correlated with it among university students (14); that problem-solving skills and resilience are significantly correlated (15); and that training in problem-solving skills significantly increased resilience scores (17). Overall, these findings support the view that problem-solving ability is an important cognitive resource associated with resilience.

It should be noted that the model accounted for a modest proportion of the variance in resilience (18%), indicating that a substantial part remains unexplained. The persistence of a significant direct effect of mentalization on resilience after accounting for the mediating variable ($c' = .18$, $p < .001$) suggests that mechanisms beyond perceived problem-solving may also operate between mentalization and resilience. The literature suggests that the link between mentalization and resilience can also be explained by processes such as emotion regulation, attachment security, and epistemic confidence. Schwarzer et al. (5) demonstrated that mentalization mediates between stress and coping in a community sample. In a more recent study, Schwarzer et al. (10) found that mentalization predicts well-being and emotion regulation. Similarly, Maerz et al. (12) demonstrated, using a structural equation model, that mentalization predicts depressive symptoms and life satisfaction through resilience. Kealy et al. (6), on the other hand, found that reflective functioning is associated with resilience and personal development initiative in men. Suominen (19) has argued that metacognitive capacities support the development of resilience, and that deficits in these abilities increase the risk of stress-related disorders; this view aligns with the idea that mentalization can contribute to resilience beyond problem-solving. Other individual-difference factors not assessed in the present study, including self-efficacy, social support, depressive and anxiety symptoms, and personality traits, may also contribute to resilience and warrant examination in future research. Taken together, these findings suggest that while perceived problem-solving is one of the multiple pathways linking mentalization to resilience, it is not the only one.

To the best of our knowledge, this is the first study to examine mentalization, perceived problem-solving, and resilience together within a mediational model framework in a non-clinical adult sample. The connection between mentalization and problem-solving has previously been examined primarily in clinical samples with different measurement tools (20). This study, however, shows this relationship in the general population using standard self-report scales. From an applied perspective, the findings suggest that integrating mentalization-focused approaches with problem-solving skills training in intervention programs aimed at strengthening resilience may be beneficial. Adding problem-solving modules to mentalization-based therapy (MBT) programs or enriching problem-solving skills training with mentalization components may contribute to more effective interventions. Because this study employed a cross-sectional, non-intervention design, these clinical suggestions should be regarded as preliminary and hypothesis-generating, and require confirmation through intervention-based research.

Limitations

Several limitations of this study should be acknowledged. First, because the study used a cross-sectional correlational design, it is not possible to establish causal relationships between variables. The path coefficients in the mediation model show statistical associations; however, establishing temporal precedence and causality requires longitudinal or experimental research. Second, because all data were collected via self-report scales, there is a potential concern regarding common method variance. In particular, the PSI assesses how someone perceives their own problem-solving ability rather than their actual performance; therefore, the results are limited to perceived skill level. In addition, although the sample was described as non-clinical, no standardized psychiatric screening instrument or diagnostic interview was administered. Therefore, the term “non-clinical” should be interpreted as referring to a community-based convenience sample rather than to a sample formally screened for psychiatric disorders. Future studies should include structured screening tools to more clearly distinguish clinical and non-clinical participants. Third, the relatively low reliability coefficients of the MentS subscales ($\alpha = .61$–$.62$) should be noted; while the total score reliability ($\alpha = .73$) is acceptable, analyses at the subscale level may be affected by this limitation. In addition, the correlation matrix included multiple subscale-level comparisons that were not corrected for the number of tests, which increases the risk of Type I error; these subscale-level associations should therefore be interpreted as exploratory. The primary mediation analysis, however, was based on total scores and is not affected by this concern. Lastly, relying on convenience sampling restricts the generalizability of the findings.

CONCLUSION

This study showed that mentalization capacity predicts resilience in non-clinical adults both directly and through perceived problem-solving ability. This partial mediation finding suggests that perceived problem-solving ability is one of the pathways linking mentalization to resilience. Future research is recommended to test this model using longitudinal designs, to include additional mediating variables such as emotion regulation and attachment security, and to test it across different sample groups, including clinical samples and different age groups.

Declarations

AI-assisted language editing was used to improve grammar and clarity. The author reviewed and edited all AI-assisted outputs and assumes full responsibility for the manuscript's content.

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Linkage to psychiatric care after the special needs report for children and its predictors: A retrospective study of 2363 cases

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SUMMARY

Objective: The Special Needs Report for Children (SNRC; Cocuklar Icin Ozel Gereksinim Raporu, COZGER), introduced in Turkey in 2019, restructured pediatric disability evaluation. Whether it facilitates linkage to child mental health services remains underexamined. This study evaluated the characteristics of children receiving SNRC, post-report psychiatric linkage to care, and its predictors.

Method: Records of 2363 children and adolescents who received SNRC at a single tertiary center between February 20, 2019 and December 31, 2025 were retrospectively reviewed. Pre-report follow-up was defined as ≥2 non-SNRC outpatient visits during the 12 months preceding report issuance, and post-report linkage as ≥1 non-SNRC psychiatric visit after report issuance. Predictors were examined using binary logistic regression.

Results: Of 2363 cases, 60.9% were male and the mean age at first presentation was 8.4 ± 4.7 years. Overall, 59.4% had no psychiatric diagnosis, with neurological, endocrine, and genetic comorbidities prominent in this group. Among 960 patients with a psychiatric diagnosis, the most frequent were intellectual disability, language/speech disorders, specific learning disorder, and autism spectrum disorder. Post-report linkage occurred in 35.1% of the full sample and 46.5% of the psychiatric subgroup. Positive predictors were psychiatric comorbidity count (OR=3.77; 95% CI: 2.63–5.40), number of reports (OR=2.84; 2.48–3.26), prior follow-up (OR=2.58; 2.17–3.07), autism spectrum disorder (OR=1.84; 1.33–2.53), and male sex (OR=1.20; 1.01–1.43); out-of-province residence was the only negative predictor (OR=0.43; 0.35–0.53).

Discussion: The modest post-report linkage rate suggests that the SNRC process functions only partially as a gateway to ongoing care. These findings support reconceptualizing SNRC not merely as a reporting procedure but as a structural opportunity for linkage to child mental health services.

Key Words: Special needs report for children, child and adolescent mental health, linkage to care, neurodevelopmental disorders, healthcare utilization

INTRODUCTION

Disability is the inability to participate fully in activities of daily living and society because of limitations in physical, cognitive, sensory, or psychiatric functioning and is recognized as a global public health concern (1-2). In childhood, disability encompasses a broad range of diagnoses, including physical illnesses, neurodevelopmental disorders (such as intellectual disability, autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), and specific learning disorder (SLD)), and sensory impairments (3-4). Early identification of these conditions and the development of appropriate intervention plans are critical to maximizing the child's developmental potential (5-6).

For many years, children with disabilities in Türkiye were assessed using the same medical board report as adults. In response to criticism that this system lacked child-specific developmental assessment criteria, the Regulation on Special Needs Assessment for Children, which entered into force on 20 February 2019, established the Special Needs Report for Children (SNRC) system (7). The SNRC differs from the previous medical board report in three fundamental respects: (a) it replaces

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the adult concept of a disability percentage with seven levels of special need appropriate to the child's developmental context (Special Needs / Mild Special Needs / Moderate Special Needs / Advanced Special Needs / Very Severe Special Needs / Significant Special Needs / Special Condition Requirement); (b) needs are coded separately by domain (cognitive development, language and speech, psychiatry, social-emotional development, etc.); and (c) diagnostic criteria are based on the DSM-5 classification (7-10). However, under the regulation's coding system, some DSM-5 diagnoses, most notably ADHD, are not listed as primary special-needs diagnoses and are generally documented as comorbid diagnoses. This structural feature must be considered when interpreting diagnostic distributions and follow-up patterns in the SNRC population.

Studies from Türkiye examining the sociodemographic and clinical profiles of SNRC applicants have shown that most applicants are boys and that intellectual disability, ASD, and SLD are the most common diagnoses (11-15). The higher prevalence of neurodevelopmental disorders among boys is consistent with the literature (16-17). Regional sociodemographic differences, rates of consanguineous marriage, and access to health care have also been reported to influence the profile of SNRC applicants (18-19).

Whether the SNRC process offers a window of opportunity for linkage to child mental health services beyond the issuance of a report has not been adequately investigated (36-37). Within this conceptual framework, the SNRC process could facilitate linkage through three mechanisms: (a) the child undergoes a structured psychiatric assessment; (b) the family receives structured information about the diagnosis, prognosis, and treatment options; and (c) the reporting process creates a formal point of contact between the family and the outpatient clinic. The extent to which these mechanisms operate in practice, specifically the proportion of patients linked to outpatient care after the SNRC and the factors associated with this linkage, has not been systematically documented. The available literature has focused largely on describing applicant profiles, and very few studies have examined patterns of linkage to psychiatric services after the SNRC. Dropout rates from child and adolescent mental health services range from 30% to 75%, and treatment adherence is influenced by multidimensional factors, including geographic access, socioeconomic status, parental attitudes, and clinical characteristics (20-23). Nonadherence to medication is also common in neurodevelopmental disorders (24-25), but how this issue should be addressed within the SNRC context remains unclear.

The growing migrant and refugee population in Türkiye raises additional determinants of health care access, including language barriers, insurance coverage, differences in legal status, and cultural distance (26). Given the distinct sociocultural dynamics of different regions, regional studies are expected to contribute to national policy development (27-28).

Against this background, the present study aimed to document the sociodemographic and clinical characteristics of children and adolescents issued an SNRC at the Child and Adolescent Psychiatry outpatient clinic of Bolu Abant İzzet Baysal University Faculty of Medicine, their patterns of linkage to psychiatric services after report issuance, and the sociodemographic, clinical, and disorder-specific factors associated with those patterns. The single-center design provided a homogeneous dataset because the same clinical team and consistent assessment procedures were used. The province's urban and rural catchment areas and inward migration also provided a suitable setting in which to examine geographic access and migrant health variables together.

METHODS

Study Design and Sample

This study was a retrospective chart review with a longitudinal component. Because clinical contact patterns before and after report issuance were retrospectively reconstructed over time for the same patient, the design differed from a conventional cross-sectional study that evaluates data at a single point. However, because data were collected through a single retrospective review covering the

study period, the study did not have a cohort design. The electronic and paper records of all children and adolescents aged 0-18 years who were issued an SNRC at the Child and Adolescent Psychiatry outpatient clinic of Bolu Abant İzzet Baysal University Faculty of Medicine between 20 February 2019, when the SNRC regulation entered into force, and 31 December 2025 were systematically reviewed. A total of 2363 cases were identified.

The inclusion criteria were (a) having been assessed and issued an SNRC at our outpatient clinic during the study period and (b) having undergone a psychiatric assessment according to DSM-5 criteria, with the presence or absence of a diagnosis documented in the record. The exclusion criteria were (a) presentation solely for a medical board report without a psychiatric assessment and (b) insufficient data in the medical record.

Patients presenting from outside the province were included to reflect the center's actual referral population and patterns of service access; place of residence was also analyzed as an independent variable.

SNRC Assessment System and Clinical Process

The SNRC is an assessment system established by the regulation dated 20 February 2019. It is based on the DSM-5 classification and emphasizes the child's development as a whole (7). The regulation operationalized special needs as the additional need for health, education, rehabilitation, and other services, beyond those required by individuals without physical or developmental functional limitations, to enable the child to participate equally in social life. It defined seven levels: Special Needs, Mild Special Needs, Moderate Special Needs, Advanced Special Needs, Very Severe Special Needs, Significant Special Needs, and Special Condition Requirement. SNRC assessments are coded separately by domain. The psychiatry domain (category E) covers intellectual disability, ASD, anxiety disorders, obsessive-compulsive spectrum disorders, post-traumatic stress disorder, psychosis, and mood disorders. ADHD is rarely recorded as a primary special-needs diagnosis under the regulation's coding system and is more often documented as a comorbid diagnosis. This structural feature defined the framework for the diagnostic classification used in the present study (see below). We did not analyze the levels of intellectual disability specified in the regulation (Special Needs / Mild / Moderate / Advanced / Very Severe / Significant / Special Condition Requirement) as separate categories; all cases of intellectual disability were evaluated under a single heading. The limitation of this approach is discussed below.

At our outpatient clinic, SNRC assessments are conducted within a multidisciplinary framework. A child and adolescent psychiatrist performs the psychiatric assessment, and the speech-language therapist on the team conducts the speech-language assessment. The Ankara Developmental Screening Inventory (ADSI) is routinely administered to children younger than six years for standardized developmental assessment. An otorhinolaryngology consultation is requested selectively when hearing loss or structural pathology is suspected in association with a speech-language problem.

Data Collection

The following data were systematically recorded for each case: (a) sociodemographic information (age at first presentation, sex, place of residence, and migration status); (b) clinical data (primary and secondary psychiatric diagnoses, number of psychiatric comorbidities, and the presence and type of medical comorbidity); (c) SNRC data (number of reports and report renewal status); and (d) clinical contact data (number and duration of visits before and after report issuance, number of non-SNRC psychiatric visits, and total duration of follow-up).

Diagnostic Categorization

The primary psychiatric diagnosis was defined as the principal diagnosis recorded in the SNRC as the basis for the stated special need. A secondary psychiatric diagnosis was defined as any additional psychiatric disorder documented as a comorbid diagnosis in the medical record. The number of

Table 1. Demographic and Clinical Characteristics (N = 2363)

Variable	n (%)	Mean ± SD / Median (IQR)
Age at first presentation (years)	2363	8.4 ± 4.7 / 8.0 (5.0-12.0)
Age group		
0-2 years	254 (10.7)	
3-5 years	430 (18.2)	
6-11 years	1058 (44.8)	
12-17 years	564 (23.9)	
Sex		
Male	1440 (60.9)	
Female	923 (39.1)	
Place of residence		
Bolu	1658 (70.2)	
Outside Bolu	705 (29.8)	
Migration status		
Turkish citizen	2234 (94.5)	
Migrant/refugee	129 (5.5)	
Nationality of migrants (n=129)		
Iraq	74 (57.4)	
Syria	26 (20.2)	
Afghanistan	11 (8.5)	
Iran	2 (1.6)	
Other	16 (12.4)	
Primary psychiatric diagnosis		
No psychiatric diagnosis	1403 (59.4)	
Intellectual disability[b]	229 (9.7)	
Language/speech/communication disorder	218 (9.2)	
Specific learning disorder	206 (8.7)	
Autism spectrum disorder	160 (6.8)	
Psychosis/mood disorder	9 (0.4)	
Other psychiatric diagnoses[a]	138 (5.8)	
Distribution within other psychiatric diagnoses (n=138)[c]		
ADHD	96 (69.6)	
Anxiety disorders	32 (23.2)	
Tic disorders	4 (2.9)	
Enuresis	4 (2.9)	
Conduct disorder	2 (1.4)	
Presence of a secondary psychiatric diagnosis	125 (5.3)	
Number of psychiatric comorbidities		
0 additional diagnoses	2238 (94.7)	
1 additional diagnosis	111 (4.7)	
2 additional diagnoses	14 (0.6)	
Presence of medical comorbidity	761 (32.2)	
Distribution of primary medical diagnoses		
Neurology	274 (11.6)	
Endocrinology	232 (9.8)	
Genetics	117 (5.0)	
Ophthalmology	43 (1.8)	
ENT	36 (1.5)	
Physical medicine and rehabilitation	26 (1.1)	
Nephrology/urology	14 (0.6)	
Orthopedics	8 (0.3)	
Other	11 (0.5)	
Number of SNRC reports	2363	1.4 ± 0.8 / 1.0 (1.0□2.0)
Single report	1699 (71.9)	
Multiple reports (≥ 2)	664 (28.1)	

SD: standard deviation; IQR: interquartile range; SNRC: Special Needs Report for Children; ENT:ear, nose, and throat; PM&R: physical medicine and rehabilitation. [a]The other psychiatric diagnoses category includes attention-deficit/hyperactivity disorder (ADHD), anxiety disorders, tic disorders, enuresis, and conduct disorder; the distribution within this category is also presented in the table. [b]This disorder, defined in the DSM-5 as intellectual disability (intellectual developmental disorder), is listed under impaired intellectual functioning in the SNRC regulation's coding system. [c]Percentages within the other psychiatric diagnoses category were calculated using the total for this subgroup (n=138). Continuous variables are presented as mean ± SD and median (IQR); categorical variables are presented as n (%).

psychiatric comorbidities was coded as a numerical variable representing the number of additional psychiatric diagnoses (0,1, or 2).

Primary diagnoses were classified into seven categories: (1) no psychiatric diagnosis, (2) intellectual disability, (3) language/speech/communication disorders, (4) specific learning disorder, (5) autism spectrum disorder (ASD), (6) psychosis/mood disorders, and (7) other psychiatric diagnoses.

Psychotic and mood disorders were evaluated as a separate category because they constitute a specific special-needs category under the regulation (usually at the Significant Special Needs or Special Condition Requirement level) and their clinical follow-up differs substantially from that of other disorders because they require ongoing medication and close monitoring. Although anxiety and obsessive-compulsive spectrum disorders are included in category E of the regulation, they were not observed frequently enough in our sample to form a separate category and were grouped under other psychiatric diagnoses.

The other psychiatric diagnoses category (n=138; 5.8% of the sample) included ADHD (n=96; 69.6%), anxiety disorders (n=32; 23.2%), tic disorders (n=4; 2.9%), enuresis (n=4; 2.9%), and conduct disorder (n=2; 1.4%). ADHD was included in this category because it is rarely coded as a primary special-needs diagnosis under the SNRC regulation and is usually recorded as a comorbid diagnosis. This structural feature prevented ADHD from being modeled as a separate independent variable; the corresponding limitation is discussed below.

Inclusion of Cases Without a Psychiatric Diagnosis

A substantial proportion of the cases (n=1403; 59.4%) did not receive a psychiatric diagnosis fol-

Table 2. Overall Distribution of Clinical Contact Patterns (N = 2363)

Clinical contact indicator	n (%)	Mean – SD	Median (IQR)
Pre-Report Follow-Up			
Patient in pre-report follow-up (=2 visits during the 12 months preceding report issuance)	1104 (46.7)		
Number of pre-report visits		3.2 ± 3.4	2.0 (1.0-4.0)
Duration of pre-report contact (months)		3.1 ± 4.2	1.0 (0.0-6.0)
Post-Report Linkage			
Met the post-report linkage criterion	830 (35.1)		
Number of post-report visits		2.3 ± 2.8	1.0 (1.0-3.0)
Duration of post-report contact (months)		3.1 ± 4.3	1.0 (0.0-7.0)
Time to linkage (days)[c]		-	45.0 (15.0-120.0)
Non-SNRC Psychiatric Visits[a]			
Number of non-SNRC psychiatric visits	916 (95.4)[b]	7.8 ± 10.6	4.0 (2.0-8.0)
Total Follow-Up			
Total follow-up duration (months)		4.2 ± 6.5	2.0 (0.0-11.0)
Any follow-up record	1534 (64.9)		
No follow-up record	829 (35.1)		
Follow-Up Classification[c]			
Follow-up before the report and linkage after the report	317 (33.0)		
Pre-report follow-up only	305 (31.8)		
Post-report linkage only	129 (13.4)		
No follow-up (neither before nor after)	209 (21.8)		

SD: standard deviation; IQR: interquartile range.
[a]Non-SNRC psychiatric visits are psychiatric outpatient visits other than those for an SNRC among patients with a psychiatric diagnosis (n = 960).
[b]Of the 960 patients with a psychiatric diagnosis, 916 (95.4%) had at least one psychiatric visit other than for an SNRC.
[c]Follow-up classification was calculated only for patients with a psychiatric diagnosis (n=960); percentages are based on this subgroup.
[c]Time to linkage is the number of days from the report date to the first non-SNRC psychiatric visit among patients who met the post-report linkage criterion (n = 830); because the distribution was non-normal, the median (IQR) is presented.

lowing the SNRC assessment. Medical comorbidities, including neurologic, endocrine, genetic, and sensory conditions, were prominent in this group, and their distribution is presented in Table 1. This group was included in the overall analyses to (a) reflect the actual composition of the SNRC sample and (b) document differences in linkage to psychiatric care between patients with and without a psychiatric diagnosis. By contrast, follow-up categorization (lower section of Table 2) and subgroup analyses of the number of non-SNRC visits were calculated only for the 960 patients with a psychiatric diagnosis, because the concept of psychiatric follow-up is meaningful only in a population with a psychiatric diagnosis.

Operational Definition of Clinical Linkage

In the present study, patterns of clinical contact before and after report issuance were evaluated in terms of linkage to psychiatric care following the SNRC process rather than sustained treatment retention. Different operational thresholds were used for the two periods because they addressed different clinical questions:

A patient in pre-report follow-up was defined as a patient with at least 2 non-SNRC visits to the outpatient clinic during the 12 months preceding report issuance. This threshold was selected to operationalize repeated clinical contact before the report. Definitions of treatment dropout and continuity of care in child and adolescent mental health services vary across studies (20).

A patient linked to care after the report was defined as a patient with at least 1 non-SNRC visit to the outpatient clinic after report issuance. This threshold reflects linkage to psychiatric care after the report rather than retention. Whether the SNRC offers a window of opportunity for transition to treatment was operationalized as whether any psychiatric contact occurred after report issuance.

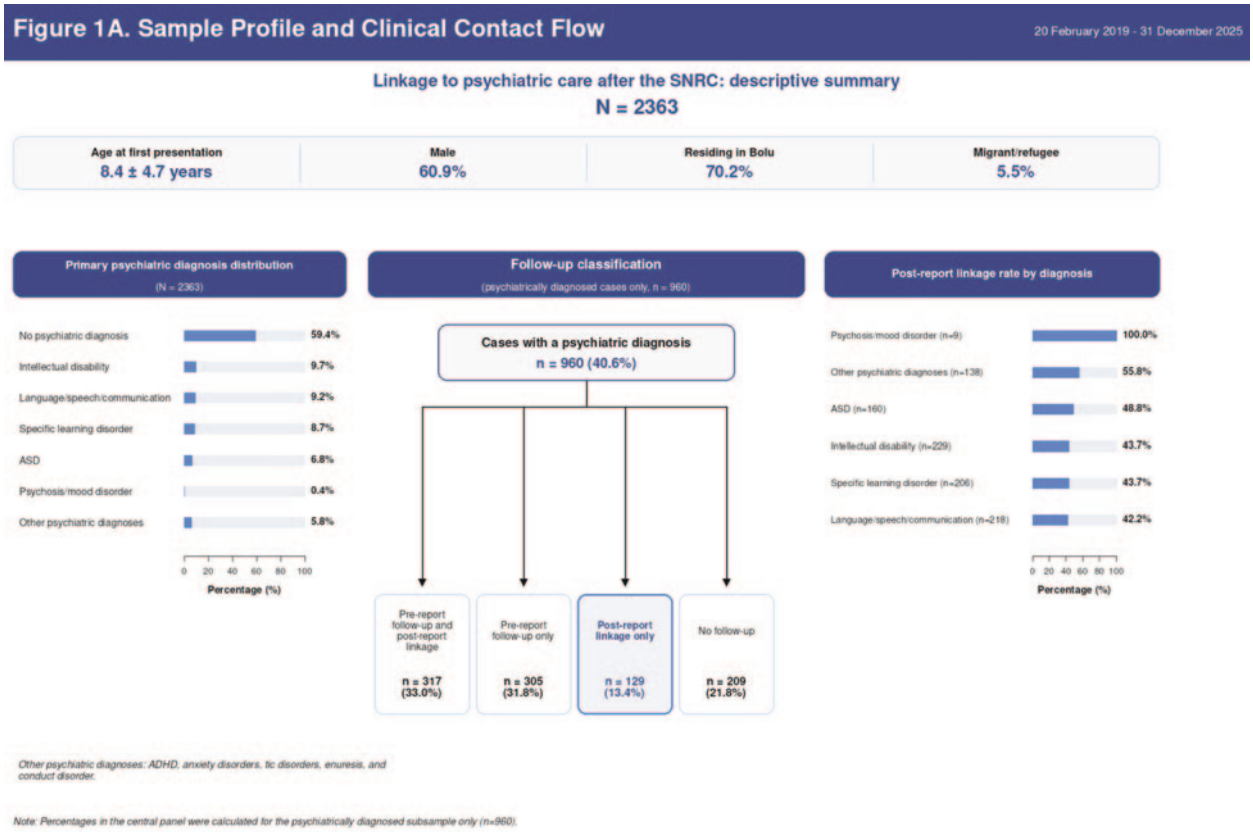
The asymmetry between the thresholds for the two periods reflects the different operational requirements of two conceptually distinct clinical questions: sustained follow-up versus new linkage. A non-SNRC psychiatric visit was defined as any clinical visit other than one for SNRC issuance or renewal.

Statistical Analysis

Data were analyzed using SPSS version 22.0 (IBM Corp., Armonk, NY). Normality was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests; the data were not normally distributed (Shapiro-Wilk=0.740; $p<0.001$). Continuous variables are presented as mean ± standard deviation (SD) and median (interquartile range (IQR)); categorical variables are presented as frequencies and percentages.

For comparisons between two groups, the chi-square (x^2) test was used for categorical variables and the Mann-Whitney U test for continuous variables. Comparisons of more than two groups were performed using the chi-square and Kruskal-Wallis H tests, followed by post hoc Dunn tests for significant comparisons. Correlations were reported using Spearman's rank correlation coefficient (rho), and effect sizes were reported using the rank-biserial correlation coefficient (rrb). Because multiple comparisons increase the probability of type I error, findings with borderline significance were interpreted cautiously in this context.

Factors predicting linkage were identified using binary logistic regression. In Model 1, the dependent variable was post-report linkage (yes/no); in Model 2, it was pre-report follow-up status (yes/no). Age, sex, place of residence, migration status, medical comorbidity, number of psychiatric comorbidities, number of reports, and primary diagnostic categories were included as independent variables in both models. Pre-report follow-up status was also included in Model 1 but was not entered into Model 2 because it was identical to the dependent variable in that model. Psychosis/mood disorders (n=9) were excluded from the models because of the small number of cases. Because of its heterogeneity and the position of ADHD within the regulation's coding system, the other psychiatric diagnoses category (predominantly ADHD) could not be modeled as a separate indicator variable and was included in the reference category (no psychi-



atric diagnosis); this issue is addressed in the Limitations section. The enter (forced-entry) method was used in both models. Model fit was assessed using the omnibus chi-square test, the Hosmer-Lemeshow goodness-of-fit test, Nagelkerke R^2, and classification accuracy. A value of $p<0.05$ was considered statistically significant in all analyses.

Ethics Approval

This study was approved by the Non-Interventional Clinical Research Ethics Committee of Bolu Abant İzzet Baysal University (Decision No.: (2026/242), Date: (3 February 2026)). The study was conducted in accordance with the Declaration of Helsinki and the principles of Good Clinical Practice.

RESULTS

Demographic and Clinical Characteristics

The study included 2363 children and adolescents who were issued an SNRC between 20 February 2019 and 31 December 2025 (Table 1, Figure 1A). The mean age at first presentation was 8.4±4.7 years (median 8.0; IQR 5.0-12.0), and 44.8% of cases (n=1058) were aged 6-11 years. Of the cases, 60.9% (n=1440) were male and 39.1% (n=923) were female. Overall, 70.2% of patients (n=1658) resided in Bolu and 29.8% (n=705) presented from outside Bolu. Migrants/refugees accounted for 5.5% of the sample (n=129); of this group, 57.4% were Iraqi and 20.2% were Syrian.

Regarding the distribution of primary psychiatric diagnoses, 59.4% of cases (n=1403) had no psychiatric diagnosis. This was a heterogeneous group with prominent medical comorbidities, particularly neurologic (11.6%), endocrine (9.8%), and genetic (5.0%) conditions (Table 1, distribution of medical comorbidities). Among the 960 patients with a psychiatric diagnosis (40.6%), the most common diagnoses were intellectual disability (9.7%; n=229), language/speech/communication disorder (9.2%; n=218), specific learning disorder (8.7%; n=206), and ASD (6.8%; n=160). Psychosis/mood disorders were uncommon (0.4%; n=9); the small size of this group should be considered when interpreting all related subgroup analyses. The other psychiatric diagnoses category (n=138; 5.8%) consisted

Table 3. Comparison of Clinical Linkage by Primary Psychiatric Diagnosis (N = 2363)

Diagnostic group	n	Pre-report follow-up n (%)	Post-report linkage n (%)	Non-SNRC visits, mean – SD[d]	Total follow-up (months), mean – SD	Multiple reports n (%)
No psychiatric diagnosis	1403	482 (34.4)	384 (27.4)	5.2 ± 7.8	2.9 ± 5.4	300 (21.4)
Intellectual disability[b]	229	139 (60.7)	100 (43.7)	7.1 ± 9.1	5.0 ± 7.1	96 (41.9)
Language/speech	218	143 (65.6)	92 (42.2)	8.8 ± 12.1	5.8 ± 7.2	87 (39.9)
Specific learning disorder	206	140 (68.0)	90 (43.7)	8.8 ± 12.3	6.0 ± 7.1	65 (31.6)
ASD	160	106 (66.2)	78 (48.8)	9.6 ± 9.7	7.2 ± 8.2	82 (51.2)
Psychosis/mood disorder	9	7 (77.8)	9 (100.0)	21.0 ± 16.4	11.6 ± 7.4	5 (55.6)
Other psychiatric diagnoses[a]	138	87 (63.0)	77 (55.8)	11.8 ± 12.3	7.7 ± 7.8	29 (21.0)
Total	**2363**	**1104 (46.7)**	**830 (35.1)**	**6.8 ± 9.6**	**4.2 ± 6.5**	**664 (28.1)**

[a]Other psychiatric diagnoses: ADHD, anxiety disorders, tic disorders, enuresis, and conduct disorder.
[b]Intellectual disability (DSM-5: intellectual disability).
ASD: autism spectrum disorder; ADHD: attention-deficit/hyperactivity disorder; SD: standard deviation.
[d]The number of non-SNRC visits was calculated only among patients with a psychiatric diagnosis in the corresponding diagnostic group; values for the no psychiatric diagnosis group refer to patients in that group who had at least one non-SNRC psychiatric visit.
Pre-report follow-up: x^2= 215.5, df = 6, p < 0.001. Post-report linkage: x^2= 111.3, df = 6, p < 0.001.
Number of non-SNRC visits (Kruskal-Wallis): H = 23.1, p < 0.001. Post hoc (Dunn): the psychosis/mood disorder group (n = 9; should be interpreted cautiously because of the small number of cases) differed significantly from all other groups, and the no-diagnosis group differed significantly from all diagnosed groups (p < 0.05). No significant differences were found among the intellectual disability, language/speech, specific learning disorder, and ASD groups.

predominantly of ADHD (n=96; 69.6% of this category) and anxiety disorders (n=32; 23.2%) (the full distribution is presented in Table 1). A secondary psychiatric diagnosis was present in 5.3% (n=125). Medical comorbidity was present in 32.2% of cases (n=761), most commonly neurologic (11.6%) and endocrine (9.8%) conditions. The mean number of SNRC reports was 1.4 ± 0.8 (median 1.0); 71.9% of cases (n=1699) had a single report and 28.1% (n=664) had multiple reports (≥2) (Table 1).

Overall Distribution of Clinical Contact Patterns

In the full sample (N=2363), 46.7% of patients (n=1104) were in pre-report follow-up (≥2 visits during the 12 months preceding report issuance), and 35.1% (n=830) were linked to the psychiatric clinic after the report (≥1 visit) (Table 2, Figure 1A). Among the 830 patients who met the post-report linkage criterion, the median number of visits was 1.0 (IQR 1.0-3.0). Thus, for a substantial proportion of patients who met the criterion, post-report contact was limited to a single visit. This pattern emphasizes that linkage to care and sustained follow-up (retention) are distinct processes and supports the conceptual distinction between the linkage and retention frameworks used in the present study. Among these 830 patients, the median time from the report date to the first non-SNRC psychiatric visit was 45.0 days (IQR 15.0-120.0); the finding that approximately half of the visits occurred within the first six weeks indicates that linkage generally occurred close to the reporting period.

Of the 960 patients with a psychiatric diagnosis, 95.4% (n=916) made at least one psychiatric outpatient visit other than for SNRC issuance or renewal. The mean number of non-SNRC visits was 7.8 ± 10.6 (median 4.0; IQR 2.0-8.0). The mean total follow-up duration was 4.2±6.5 months (median 2.0 months); the relatively short median provides a limited time horizon for inferences about long-term continuity.

According to the follow-up classification, 33.0% of patients with a psychiatric diagnosis (n=317) met the criteria both for pre-report follow-up and post-report linkage; 31.8% (n=305) were in follow-up

Table 4. Clinical Linkage by Demographic and Clinical Factors (N = 2363)

Factor	n	Pre-report follow-up n (%)	Post-report linkage n (%)	p (pre-report)	p (post-report)
Sex				**0.001[b]**	**0.045[b]**
Male	1440	711 (49.4)	529 (36.7)		
Female	923	393 (42.6)	301 (32.6)		
Age group				**<0.001[c]**	**<0.001[c]**
0-2 years	254	80 (31.5)	61 (24.0)		
3-5 years	430	225 (52.3)	154 (35.8)		
6-11 years	1058	556 (52.6)	412 (38.9)		
12-17 years	564	233 (41.3)	200 (35.5)		
Place of residence				**<0.001[b]**	**<0.001[b]**
Bolu	1658	844 (50.9)	670 (40.4)		
Outside Bolu	705	260 (36.9)	160 (22.7)		
Migration status				**0.007[b]**	**0.470[b]**
Turkish citizen	2234	1059 (47.4)	789 (35.3)		
Migrant/refugee	129	45 (34.9)	41 (31.8)		
Medical comorbidity				**<0.001[b]**	**0.726[b]**
No	1602	825 (51.5)	567 (35.4)		
Yes	761	279 (36.7)	263 (34.6)		
Psychiatric comorbidity				**<0.001[c]**	**<0.001[c]**
0 additional diagnoses	2238	1010 (45.1)	743 (33.2)		
1 additional diagnosis	111	83 (74.8)	77 (69.4)		
2 additional diagnoses	14	11 (78.6)	10 (71.4)		
Number of SNRC reports				**<0.001[b]**	**<0.001[b]**
Single report	1699	633 (37.3)	424 (25.0)		
Multiple reports (≥ 2)	664	471 (70.9)	406 (61.1)		

[b]Chi-square (x^2) test. [c]Kruskal-Wallis test (for comparisons of more than three groups).
Pre-report follow-up: patient with ≥ 2 non-SNRC visits before the report; post-report linkage: patient with ≥ 1 non-SNRC visit after the report.
SNRC: Special Needs Report for Children.

Table 5. Factors Predicting Clinical Linkage: Multivariable Logistic Regression Analysis

Model 1-Dependent variable: post-report linkage (yes/no)						
Variable	**B**	**SE**	**OR**	**Lower 95% CI**	**Upper 95% CI**	**p**
Constant	-2.412	0.192	0.09			<0.001
Age at first presentation	0.002	0.009	1.00	0.98	1.02	0.816
Sex (male vs female [ref])	0.182	0.089	1.20	1.01	1.43	0.041
Residence (outside Bolu vs Bolu [ref])	-0.837	0.103	0.43	0.35	0.53	<0.001
Migration status (migrant vs Turkish citizen [ref])	-0.159	0.194	0.85	0.58	1.25	0.414
Medical comorbidity (yes vs no [ref])	-0.037	0.092	0.96	0.80	1.16	0.692
Number of psychiatric comorbidities	1.326	0.183	3.77	2.63	5.40	<0.001
Number of SNRC reports	1.044	0.070	2.84	2.48	3.26	<0.001
Pre-report follow-up (yes vs no [ref])	0.948	0.089	2.58	2.17	3.07	<0.001
Intellectual disability (vs no diagnosis [ref])	0.399	0.141	1.49	1.13	1.96	0.005
Language/speech/communication disorder	0.331	0.144	1.39	1.05	1.85	0.022
Specific learning disorder	0.396	0.148	1.49	1.11	1.98	0.007
ASD	0.607	0.164	1.84	1.33	2.53	<0.001
Model 2-Dependent variable: pre-report follow-up (yes/no)						
Variable	**B**	**SE**	**OR**	**Lower 95% CI**	**Upper 95% CI**	**p**
Constant	-0.863	0.175	0.42			<0.001
Age at first presentation	-0.013	0.009	0.99	0.97	1.00	0.138
Sex (male vs female [ref])	0.274	0.085	1.32	1.11	1.55	0.001
Residence (outside Bolu vs Bolu [ref])	-0.574	0.092	0.56	0.47	0.68	<0.001
Migration status (migrant vs Turkish citizen [ref])	-0.520	0.190	0.59	0.41	0.86	0.006
Medical comorbidity (yes vs no [ref])	-0.607	0.090	0.55	0.46	0.65	<0.001
Number of psychiatric comorbidities	1.144	0.194	3.14	2.15	4.59	<0.001
Number of SNRC reports	0.896	0.071	2.45	2.13	2.81	<0.001
Intellectual disability (vs no diagnosis [ref])	0.626	0.142	1.87	1.42	2.47	<0.001
Language/speech/communication disorder	0.854	0.149	2.35	1.75	3.15	<0.001
Specific learning disorder	0.965	0.155	2.63	1.94	3.56	<0.001
ASD	0.863	0.173	2.37	1.69	3.32	<0.001

Model 1 fit statistics: omnibus $x^2(12) = 553.8$, $p < 0.001$; Nagelkerke $R^2 = 0.291$; Hosmer-Lemeshow $x^2(8) = 10.24$, $p = 0.248$; classification accuracy = 74.2%.
Clinical contact indicators by place of residence (Bolu/outside Bolu): Mann-Whitney U = 704,940 (number of follow-up visits) and U = 682,304 (follow-up duration), both $p < 0.001$; effect sizes were small (rrb = -0.206 and -0.167). Non-SNRC psychiatric visits by migration status: U=179,394, $p < 0.001$, rrb = -0.245.
Model 2 fit statistics: omnibus $x^2(11) = 491.4$, $p < 0.001$; Nagelkerke $R^2 = 0.252$; Hosmer-Lemeshow $x^2(8) = 11.86$, p=0.158; classification accuracy = 68.8%.
B: regression coefficient; SE: standard error; OR: odds ratio; CI: confidence interval; ref: reference category; ASD: autism spectrum disorder.
The psychosis/mood disorder group (n = 9) was excluded from the model because of the small sample size. The other psychiatric category (including ADHD) was included in the reference group. The pre-report follow-up variable was not entered into Model 2 because it was identical to the dependent variable. The enter (forced-entry) method was used in both models. A value of $p < 0.05$ was considered statistically significant.

only before the report; 13.4% (n=129) met the post-report linkage criterion only; and 21.8% (n=209) had no follow-up record in either period (Table 2, Figure 1A). The 129 patients who met the post-report linkage criterion only had not previously been linked to the outpatient clinic and first made contact with the psychiatric clinic after report issuance. They constituted the descriptive subgroup supporting the hypothesis that the SNRC may provide an opportunity for linkage (see the Discussion for details). The diagnostic distribution among these 129 patients was 22.5% intellectual disability, 20.9% language/speech/communication disorder, 20.2% specific learning disorder, 17.8% ASD, and 17.1% other psychiatric diagnoses.

Clinical Linkage by Diagnosis

Rates of both pre-report follow-up and post-report linkage differed significantly across diagnostic groups ($x^2=215.5$; df=6; $p<0.001$ and $x^2=111.3$; df=6; $p<0.001$, respectively) (Table 3, Figure 1A). The highest rate of post-report linkage was observed in the psychosis/mood disorder group (100.0%; 9/9), although this rate should be interpreted cautiously because of the small number of cases. The next highest rate was in the other psychiatric diagnoses category (55.8%). Because this category consisted predominantly of ADHD, its relatively high linkage rate may have been influenced by the contribution of ADHD cases. This was followed by similar rates for ASD (48.8%), intellectual disability (43.7%), specific learning disorder (43.7%), and language/speech disorder (42.2%).

Table 6. Report Renewal Pattern (N = 2363)

A. Distribution of the number of SNRC reports and non-SNRC psychiatric visits by number of reports					
Number of reports	**n**	**%**	**Cumulative %**	**Non-SNRC visits, mean – SD**	**Median (IQR)**
1	1699	71.9	71.9	5.2 ± 8.0	3.0 (2.0-5.0)
2	448	19.0	90.9	9.8 ± 11.0	6.0 (4.0-10.0)
3	156	6.6	97.5	12.4 ± 12.6	7.5 (5.0-14.0)
≥ 4	60	2.5	100.0	14.5 ± 14.2	10.0 (6.5□17.0)
B. Report renewal rate by diagnosis (≥ 2 reports)					
Diagnostic group	**Total n**	**Renewal n (%)**	**Mean number of reports**		
No psychiatric diagnosis	1403	300 (21.4)	1.30		
Intellectual disability[b]	229	96 (41.9)	1.65		
Language/speech	218	87 (39.9)	1.57		
Specific learning disorder	206	65 (31.6)	1.44		
ASD	160	82 (51.2)	1.77		
Psychosis/mood disorder	9	5 (55.6)	1.56		
Other psychiatric diagnoses[a]	138	29 (21.0)	1.30		
C. Patients with multiple reports but no follow-up record					
Category	**n**	**%**			
Patients with multiple reports (≥ 2)	664	28.1[d]			
Multiple reports + no follow-up record	112	16.9[e]			
Multiple reports + non-SNRC psychiatric visits = 0	8	1.2[e]			

Report renewal by diagnosis: $x^2 = 118.5$, df = 6, $p < 0.001$.
[a]Other psychiatric diagnoses: ADHD, anxiety disorders, tic disorders, enuresis, and conduct disorder.
[b]Intellectual disability (DSM-5: intellectual disability).
ASD: autism spectrum disorder; ADHD: attention-deficit/hyperactivity disorder; SD: standard deviation; IQR: interquartile range.
[d]Calculated using the full sample (N = 2363). [e]Calculated using the number of patients with multiple reports (n = 664).

Table 7. Correlations Between Continuous Variables Associated With Clinical Contact Indicators

Variable pair	Spearman's rho	p	N
Duration of pre-report contact - Duration of post-report contact	0.439	<0.001	2363
Number of psychiatric comorbidities - Duration of pre-report contact	0.183	<0.001	2363
Number of psychiatric comorbidities - Duration of post-report contact	0.181	<0.001	2363
Medical comorbidity - Number of non-SNRC psychiatric visits	-0.060	0.004	2363
Medical comorbidity - Duration of post-report contact	-0.021	0.316	2363
Number of non-SNRC psychiatric visits - Duration of post-report contact	0.610	<0.001	2363
Number of reports - Total follow-up duration (months)	0.408	<0.001	2363
Number of reports - Age at first presentation	-0.165	<0.001	2363
Number of reports - Age at last visit	0.087	<0.001	2363
Total follow-up duration - Age at first presentation	0.017	0.396	2363
Total follow-up duration - Age at last visit	0.108	<0.001	2363

Spearman's rank correlation coefficient was used. The data were not normally distributed (Shapiro-Wilk = 0.740, p < 0.001).
Medical comorbidity is a binary (yes/no) variable; this coefficient is equivalent to a point-biserial correlation.

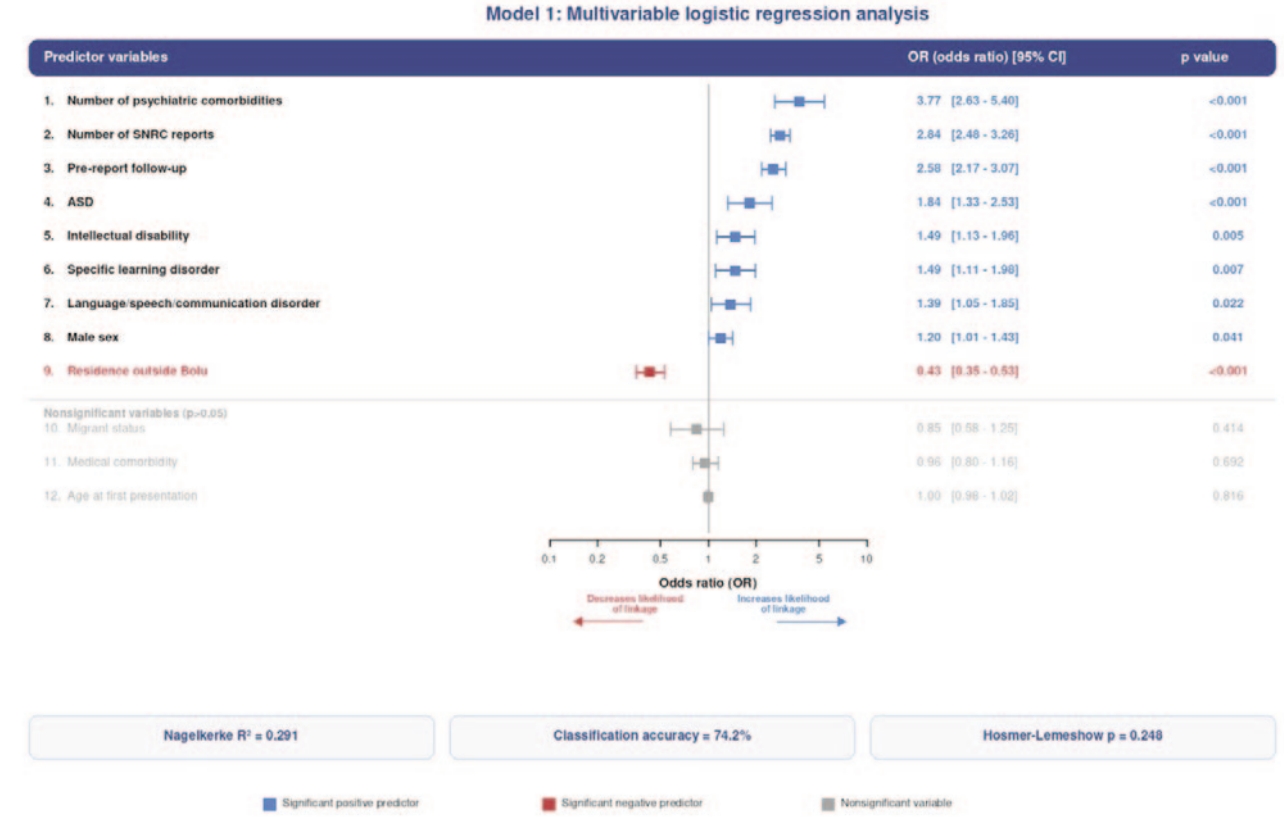
The rate was lowest among patients without a psychiatric diagnosis (27.4%).

The number of non-SNRC psychiatric visits also differed significantly across groups (Kruskal-Wallis H=23.1; p<0.001). In the post hoc Dunn analysis, the psychosis/mood disorder group (mean 21.0 ± 16.4; n=9) differed significantly from all other groups, and the group without a psychiatric diagnosis (mean 5.2 ± 7.8) differed significantly from all diagnosed groups (p<0.05). No significant differences were found among the intellectual disability, language/speech, specific learning disorder, and ASD groups (Table 3).

Clinical Linkage by Demographic and Clinical Factors

Male patients had significantly higher rates of both pre-report follow-up (49.4% vs 42.6%; p=0.001) and post-report linkage (36.7% vs 32.6%; p=0.045) than female patients; however, the p values for sex (0.001-0.045) should be interpreted cautiously in the context of multiple comparisons. Both linkage indicators also differed significantly across age groups (p<0.001). The post-report linkage rate was highest among children aged 6-11 years (38.9%) and lowest among those aged 0-2 years (24.0%) (Table 4).

Figure 1B. Factors Predicting Post-report Linkage



Patients residing in Bolu had a significantly higher post-report linkage rate than those residing outside Bolu (40.4% vs 22.7%; p<0.001). In the Mann-Whitney U tests, patients from outside Bolu had fewer follow-up visits (U=704,940; p<0.001; rrb=-0.206) and shorter follow-up durations (U=682,304; p<0.001; rrb=-0.167); the effect sizes were small (Table 7). Migrant/refugee status was associated with a lower pre-report follow-up rate (34.9% vs 47.4%; p=0.007), but there was no significant difference in post-report linkage (p=0.470). Turkish citizens had significantly more non-SNRC visits than migrants/refugees (U=179,394; p<0.001; rrb=-0.245) (Table 4).

Paradoxically, patients with medical comorbidity had a lower rate of pre-report follow-up (36.7% vs 51.5%; p<0.001), whereas post-report linkage did not differ significantly according to medical comorbidity (p=0.726). Medical comorbidity was weakly and negatively correlated with the number of non-SNRC psychiatric visits (rho=-0.060; p=0.004). Both linkage indicators increased markedly with the number of psychiatric comorbidities (p<0.001): the post-report linkage rate was 69.4% among patients with one additional diagnosis, 71.4% among those with two additional diagnoses, and 33.2% among those with none. Patients with multiple reports (≥2) had a substantially higher post-report linkage rate than those with a single report (61.1% vs 25.0%; p<0.001). The number of reports was moderately and positively correlated with total follow-up duration (rho=0.408; p<0.001) (Tables 4 and 7).

Relationship Between Clinical Contact Before and After the Report

There was a moderate positive correlation between the duration of contact before and after the report (rho=0.439; p<0.001) and a strong positive correlation between the number of non-SNRC visits and the duration of post-report contact (rho=0.610; p<0.001) (Table 7). These findings indicate an association between an existing clinical relationship and post-report linkage; no causal inference was made because of the retrospective design.

Factors Predicting Clinical Linkage

The logistic regression model predicting post-report linkage (Model 1) was statistically significant (omnibus $x^2(12)=553.8$; p<0.001; Nagelkerke $R^2=0.291$; Hosmer-Lemeshow $x^2(8)=10.24$; p=0.248; classification accuracy 74.2%) (Table 5, Figure 1B). The number of psychiatric comorbidities (OR=3.77; 95% CI: 2.63-5.40; p<0.001), number of SNRC reports (OR=2.84; 95% CI: 2.48-3.26; p<0.001), pre-report follow-up (OR=2.58; 95% CI: 2.17-3.07; p<0.001), ASD diagnosis (OR=1.84; 95% CI: 1.33-2.53; p<0.001), intellectual disability (OR=1.49; 95% CI: 1.13-1.96; p=0.005), specific learning disorder (OR=1.49; 95% CI: 1.11-1.98; p=0.007), language/speech disorder (OR=1.39; 95% CI: 1.05-1.85; p=0.022), and male sex (OR=1.20; 95% CI: 1.01-1.43; p=0.041) were positive predictors. Residence outside Bolu was a negative predictor (OR=0.43; 95% CI: 0.35-0.53; p<0.001). Migration status (p=0.414), medical comorbidity (p=0.692), and age (p=0.816) were not significant predictors.

Model 2, which predicted pre-report follow-up, was also significant (omnibus $x^2(11)=491.4$; p<0.001; Nagelkerke $R^2=0.252$; Hosmer-Lemeshow $x^2(8)=11.86$; p=0.158; classification accuracy 68.8%). In this model, migration status was also an independent predictor (OR=0.59; 95% CI: 0.41-0.86; p=0.006). Medical comorbidity reduced the likelihood of pre-report follow-up (OR=0.55; 95% CI: 0.46-0.65; p<0.001). All diagnostic categories were significant predictors relative to the reference group, with the highest OR observed for specific learning disorder (OR=2.63) (Table 5).

Report Renewal Pattern

Of the cases, 71.9% had one report, 19.0% had two reports, and 6.6% had three reports; 0.5% had five or more reports. Renewal rates differed significantly by diagnosis ($\chi^2=118.5$; df=6; p<0.001). The highest rates were observed in the psychosis/mood disorder group (55.6%; n=9; should be interpreted cautiously because of the small number of cases), followed by ASD (51.2%) and intellectual disability (41.9%). Of the 664 patients with multiple reports, 112 (16.9%) had no follow-up record; only 8 cases

(1.2%) had no non-SNRC psychiatric visits. The number of non-SNRC visits increased with the number of reports (one report:5.2±8.0; ≥4 reports: 14.5 ± 14.2) (Table 6).

Relationship Between Age and Clinical Contact

Total follow-up duration was not significantly correlated with age at first presentation (rho=0.017; p=0.396), but it was weakly and positively correlated with age at the last visit (rho=0.108; p<0.001). Age at first presentation was negatively correlated with the number of reports (rho=-0.165; p<0.001), indicating that patients who presented at a younger age received more reports (Table 7).

DISCUSSION

This study retrospectively examined the sociodemographic and clinical characteristics and patterns of linkage to psychiatric care after report issuance among 2363 patients issued an SNRC over approximately seven years. It is one of the first large-sample studies to evaluate clinical linkage systematically after the SNRC and to examine post-report linkage within the SNRC context.

The proportion of male patients (60.9%) was consistent with other SNRC studies from Türkiye, which have reported rates ranging from 62.0% to 68.1% (11-13). This male predominance is consistent with the higher prevalence of neurodevelopmental disorders among boys (16). The mean age at first presentation was 8.4 years, and children aged 6-11 years constituted the largest group (44.8%), consistent with special education needs becoming more apparent at the start of primary school (14-15).

The absence of a psychiatric diagnosis in 59.4% of cases indicates that the SNRC sample represents a broad clinical spectrum that includes patients both with and without psychiatric diagnoses; this finding should be considered together with the 32.2% prevalence of medical comorbidity. The prominent patterns of neurologic, endocrine, and genetic comorbidities in the group without a psychiatric diagnosis should be considered when interpreting comparisons involving this group. Among patients with a psychiatric diagnosis, the diagnostic distribution was largely consistent with the literature (8-9,15), and the prevalence of ASD (6.8%) was consistent with increasing awareness and diagnostic rates for this disorder (29).

Clinical Linkage Rates and Their Meaning

One of the principal findings was the relatively low rate of linkage to psychiatric care after report issuance. The linkage rate was 35.1% in the full sample and only 46.5% in the subgroup with a psychiatric diagnosis. Dropout rates from child and adolescent outpatient treatment have been reported to range from 28% to 75% (20); however, the patients included in that meta-analysis were receiving active treatment, whereas a substantial proportion of our sample (59.4%) had no psychiatric diagnosis. In light of this difference, the nonlinkage rate in the subgroup with a psychiatric diagnosis (53.5%) can be considered close to the upper end of that range. Moreover, the median number of visits among the 830 patients who met the linkage criterion was 1.0, indicating that contact was generally limited to a single appointment even among patients who achieved linkage. This supports the importance of distinguishing linkage to care from sustained follow-up (retention) (36-37). The absence of any follow-up record for 21.8% of patients with a psychiatric diagnosis suggests that these patients were not integrated into treatment. By contrast, the fact that 95.4% had at least one visit for a reason other than the SNRC indicates contact with the health system but limited transition to regular follow-up.

Patients First Linked After Report Issuance (n=129)

One of the most clinically notable findings was the identification of 129 patients who had not been linked to the outpatient clinic before the report but were first linked to psychiatric care afterward (13.4% of the subgroup with a psychiatric diagnosis). These patients were not concentrated in a single diagnostic group: 22.5% had intellectual disability, 20.9% had language/speech disorder, 20.2% had specific learning disorder, 17.8% had ASD,

and 17.1% had other psychiatric diagnoses, predominantly ADHD. Their distribution across diagnostic groups provides descriptive evidence that the SNRC process may create an opportunity for linkage across several clinical profiles. Family referral, structured assessment, and systematic information have been reported to improve engagement with child mental health treatment (36-37), making it plausible that the SNRC process creates a similar opportunity through these mechanisms. The present retrospective design could not test this hypothesis directly; prospective studies and qualitative interviews could examine these mechanisms in greater detail.

Differences in Linkage Across Diagnostic Groups

The 100% post-report linkage rate and the highest mean number of non-SNRC visits (21.0) in the psychosis/mood disorder group are consistent with the need for ongoing medication and close monitoring in these conditions (30). However, the small number of cases in this group (n=9) substantially limits generalization of the findings. Although this observation indicates an association between clinical severity and linkage, it must be interpreted cautiously because of the small sample. The similar linkage rates among the intellectual disability, language/speech, specific learning disorder, and ASD groups, together with the absence of differences among these groups in the post hoc analysis, suggest that family motivation may be relatively homogeneous across these chronic neurodevelopmental disorders.

Interpretation of Predictors

The logistic regression model (Nagelkerke R^2=0.291; classification accuracy 74.2%) demonstrates the multifactorial nature of linkage (Figure 1B). More than 70% of the variance remained unexplained, suggesting that factors not included in the model, such as socioeconomic status, parental education, the family's perception of the disorder, psychotropic medication use, and transportation options, may influence linkage.

The number of psychiatric comorbidities was the strongest predictor (OR=3.77), reflecting a positive association between clinical complexity and linkage. This finding is consistent with reports of higher medication adherence in severe psychiatric conditions (31-32). Although the number of reports was also a strong predictor (OR=2.84), the association between repeated reporting procedures and outpatient contact should be interpreted cautiously. Renewal requirements vary by diagnosis and may be administrative, particularly for chronic diagnoses such as ASD and intellectual disability. The association between pre-report follow-up and post-report linkage (OR=2.58), together with the correlation between the duration of contact in the two periods (rho=0.439), indicates that an existing clinical relationship is associated with linkage, consistent with the literature (33-34). No causal inference was made because of the retrospective design.

Taken together, these findings show that the risk of nonlinkage was concentrated in a particular profile: patients who resided outside Bolu, had not previously been linked to the outpatient clinic, had fewer psychiatric comorbidities, and had received a single report were at the greatest risk. This risk profile provides a framework for developing targeted interventions during the SNRC process.

Geographic Access and Migration Status

The 57% reduction in the likelihood of linkage among patients residing outside Bolu (OR=0.43) demonstrates a strong association between geographic distance and linkage (22-23). However, these patients may have been followed at facilities in their own provinces. Because our dataset did not include follow-up at other institutions, we could not determine whether the lower linkage rate represented true nonlinkage or follow-up at another center. Remote service models such as telepsychiatry should therefore be considered an area for investigation only after the mechanisms underlying the geographic barrier, including follow-up at other centers, social security access, and appointment availability, have been mapped.

Migrant status was not significantly associated with post-report linkage (Model 1; p=0.414) but reduced the likelihood of pre-report follow-up (Model 2; OR=0.59). This suggests that migrant

families encounter greater barriers to initial access to the health system but may show similar linkage patterns once they reach the SNRC process. The difference in the number of non-SNRC visits in favor of Turkish citizens (rrb=-0.245) indicates that migrant families used the psychiatric outpatient clinic less overall. Barriers such as language, insurance coverage, and legal status (26) may also have affected these patients; however, this interpretation is indirect because these variables were not available in the dataset.

Medical Comorbidity and Other Findings

The paradoxically lower likelihood of pre-report follow-up among patients with medical comorbidity (OR=0.55) is consistent with these children being followed primarily by other specialties, particularly neurology (11.6%) and endocrinology (9.8%), and being referred to psychiatry through the SNRC process (18,35). The absence of a difference in post-report linkage by medical comorbidity suggests that the SNRC process may help equalize contact with the psychiatric outpatient clinic among patients with different clinical histories.

The absence of any follow-up record for 16.9% of the 664 patients with multiple reports indicates that some families used the health system mainly for reporting procedures. By contrast, the increase in non-SNRC visits with the number of reports (one report: 5.2; ≥4 reports: 14.5) shows that, for most families, clinical contact also increased alongside report renewal.

Clinical Implications and Proposed Interventions

Our findings suggest that the SNRC process should not be limited to report issuance but should instead be reconsidered as a structured opportunity for linkage (36-37). Because the present retrospective study did not directly test the following interventions, they are presented as hypotheses for prospective evaluation.

First, future studies could assess whether structured psychoeducation about the diagnosis, prognosis, and treatment options at the time of report issuance facilitates linkage, particularly among patients who were not previously linked and those receiving a single report. Second, interventions to reduce geographic and sociocultural barriers, including remote health service models and linguistically and culturally responsive information, should be tested prospectively after the mechanisms underlying these barriers have been identified. Finally, future research should examine whether structural interventions, such as automated appointment reminders and referral to a social worker, influence linkage patterns.

Limitations

Several limitations should be considered. First, the retrospective design precluded causal inference, and all interpretations were made at the level of association. Second, the single-center design limits the generalizability of the findings. Third, variables such as socioeconomic status, parental education, consanguineous marriage, the family's perception of the disorder, and psychotropic medication use were not systematically recorded and could not be included in the analyses. The Nagelkerke R^2 values (approximately 0.25-0.29), indicating that more than 70% of the variance remained unexplained, suggest the potential importance of these unmeasured variables. Psychotropic medication use, in particular, has been reported to be an important determinant of follow-up patterns in child and adolescent psychiatry (24-25,31-32), and its omission from the analysis is an important limitation. Fourth, linkage was assessed only through visits to our hospital, and follow-up at other institutions cannot be excluded. This is a particularly important alternative explanation for the low linkage rates among patients from outside Bolu. Fifth, the inclusion of ADHD in the other psychiatric diagnoses category (69.6% of that category) and the inclusion of that category in the reference group prevented assessment of the independent effect of ADHD; future studies should analyze ADHD as a separate category. Sixth, levels of intellectual disability specified in the regulation were not analyzed separately, preventing assessment of the possible effect of clinical severity on linkage. Seventh, the small number of patients in the psychosis/mood disorder group (n=9) requires cautious interpretation of the corresponding findings. Eighth, sensitivity analyses

were not performed for specific subgroups (patients from outside Bolu, migrants, and patients with severe medical comorbidity), and the heterogeneous pattern of medical comorbidities in the group without a psychiatric diagnosis (n=1403) warrants caution when interpreting comparisons with this group. Ninth, the study period included the COVID-19 pandemic, and outpatient restrictions in 2020-2021 may have affected linkage rates; the effect of the pandemic could not be analyzed separately. Tenth, the large number of comparisons increased the probability of type I error, and findings with borderline significance (e.g., sex, p=0.041-0.045) should be interpreted cautiously. Finally, the linkage criterion (≥1 visit) operationalized clinical linkage after the SNRC rather than sustained follow-up; the median total follow-up duration of 2 months reinforces this conceptual distinction.

Future studies should use prospective and multicenter designs, record family socioeconomic variables and psychotropic medication use, analyze ADHD as a separate category, examine the effect of the pandemic period through subgroup analyses, conduct sensitivity analyses for relevant subgroups, and investigate linkage patterns in depth using qualitative methods such as family interviews.

CONCLUSION

Among the 2363 patients issued an SNRC over approximately seven years, 60.9% were male, 59.4% had no psychiatric diagnosis, and the most common diagnoses were intellectual disability, language/speech disorders, and specific learning disorder. The rate of linkage to the psychiatric clinic after report issuance was 35.1%. The number of psychiatric comorbidities, number of reports, and pre-report follow-up status were the strongest positive predictors of linkage, whereas residence outside Bolu was the strongest negative predictor. The finding that 129 patients who had not previously been linked to the outpatient clinic (13.4% of the subgroup with a psychiatric diagnosis) first made contact with psychiatric services after the report, and that these patients were distributed across diagnostic groups, indicates that the SNRC process may offer an opportunity for linkage across several clinical profiles.

These findings suggest that the SNRC process can be viewed not only as a reporting procedure but also as a structural window of opportunity for linkage to child mental health services (36-37). Given the high risk of nonlinkage among geographically distant patients and those without prior linkage, the effectiveness of interventions to guide families into treatment follow-up should be tested prospectively. Strengthening early referral and diagnostic processes may contribute to long-term clinical contact.

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Clozapine pharmacogenetics: A literature review

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SUMMARY

Clozapine is the most effective antipsychotic for treatment-resistant schizophrenia, yet its use is limited by interindividual variability in metabolism, efficacy, and adverse effects. This review examines the contribution of pharmacogenetics to these differences.

A comprehensive literature review was conducted focusing on genetic determinants of clozapine pharmacokinetics, pharmacodynamics, treatment response, and adverse effects. Key cytochrome P450 enzymes, regulatory loci, and neurotransmitter receptor gene variants were evaluated.

CYP1A2 polymorphisms and emerging loci such as NFIB significantly influence clozapine metabolism. Genetic variants in neurotransmitter receptor genes modulate efficacy and the risk of adverse effects, including weight gain and hematological abnormalities. Pharmacogenetic testing can complement therapeutic drug monitoring to optimize treatment outcomes.

Pharmacogenetics enhances understanding of interindividual variability in clozapine response, but current evidence does not support routine genotype-based dosing. Personalized therapy integrating pharmacogenetic data, therapeutic drug monitoring, and clinical assessment remains the most promising approach. Further research is needed to implement these strategies in routine clinical practice.

Key Words: Clozapine, pharmacogenetics, personalized psychiatry

INTRODUCTION

Clozapine treatment exhibits considerable variability in drug exposure, therapeutic response, and susceptibility to adverse effects, presenting challenges for clinical management. This review synthesizes current evidence on the pharmacogenetics of clozapine, focusing on genetic determinants of pharmacokinetics, pharmacodynamic response, and adverse effects. By integrating pharmacogenetic, pharmacogenomic, and clinical data, we aim to clarify how genetic variability contributes to treatment outcomes and to evaluate the clinical utility and limitations of pharmacogenetic testing within emerging personalized psychiatry frameworks.

Review Methodology

A narrative literature review was conducted using PubMed/MEDLINE, Scopus, and Google Scholar databases. The search strategy included combinations of the following keywords: “clozapine,” “pharmacogenetics,” “pharmacogenomics,” “CYP1A2,” “therapeutic drug monitoring,” “adverse effects,” “treatment-resistant schizophrenia,” and “personalized psychiatry.” Priority was given to peer-reviewed articles, systematic reviews, meta-analyses, pharmacogenetic association studies, and clinical guidelines published in English between 2000 and 2025, although seminal earlier studies were also included when relevant. Articles were selected based on their relevance to clozapine pharmacokinetics, pharmacodynamics, treatment response, adverse effects, and clinical implementation of pharmacogenetics. Publications lacking

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direct relevance or sufficient methodological detail were excluded.

General Overview of Clozapine

Clozapine is the most effective antipsychotic for treatment-resistant schizophrenia (TRS), defined as inadequate response to at least two antipsychotics. Evidence from meta-analyses and randomized trials consistently shows superior efficacy over first- and second-generation antipsychotics, with reductions in symptom severity and relapse risk (1,2).

Beyond core psychotic symptoms, clozapine reduces aggression, hostility, and suicide risk (3,4), effects that distinguish it from other antipsychotics. Its pharmacodynamic profile—low, rapidly dissociating dopamine D2 receptor occupancy combined with high affinity for serotonergic (5-HT2A), histaminergic (H1), muscarinic (M1), and adrenergic receptors—likely underpins these unique benefits (5-7).

Despite its advantages, clozapine remains underutilized due to risks of agranulocytosis, myocarditis, and seizures, and the requirement for regular hematological monitoring. Systemic factors, including clinician hesitancy and healthcare disparities, further limit its use (8).

Clozapine Metabolism and Determinants of Plasma Concentrations

Clozapine undergoes extensive hepatic biotransformation, primarily via the cytochrome P450 1A2 (CYP1A2) enzyme with secondary contributions from CYP3A4 and, to a lesser extent, CYP2D6 (9,10). These metabolic pathways convert clozapine to its principal metabolites, including norclozapine and clozapine N-oxide, and contribute substantially to the marked interindividual variability in plasma concentrations observed in clinical practice. The predominant role of CYP1A2 helps explain much of this variability.

Environmental and physiological factors strongly influence CYP1A2 activity. Cigarette smoking induces CYP1A2 and increases clozapine clearance, resulting in lower plasma concentrations in smokers than in nonsmokers (11,12). In contrast, systemic inflammation may suppress CYP1A2 activity and cause clinically significant increases in clozapine plasma levels despite unchanged dosing, thereby increasing toxicity risk (13). These dynamic effects underscore the vulnerability of clozapine pharmacokinetics to acute changes in patients' clinical status.

Clinically relevant drug–drug interactions further contribute to variability in clozapine exposure. Potent CYP1A2 inhibitors, most notably fluvoxamine, can substantially elevate clozapine plasma concentrations, sometimes necessitating pronounced dose reductions (14). In addition, co-administration of valproate has been associated with alterations in clozapine metabolism. While valproate may inhibit clozapine metabolism, it has been reported to induce norclozapine metabolism, resulting in lower norclozapine concentrations on average, although interindividual variability remains considerable (13).

Beyond enzymatic activity, demographic and individual patient characteristics-including age, sex, body composition, and comorbid medical conditions—also influence clozapine distribution and elimination. Collectively, these factors reinforce the clinical necessity for individualized dosing strategies and routine therapeutic drug monitoring, which remains the cornerstone for optimizing clozapine treatment in real-world settings (15,16). Genetic variation in metabolic enzymes also contributes to variability in clozapine pharmacokinetics. CYP1A2 polymorphisms may affect enzyme inducibility, particularly in smokers, although their direct effects on plasma clozapine concentrations appear modest across studies (17). Emerging evidence suggests that additional genetic factors may play clinically relevant roles. Variants in CYP3A5 and CYP3A4 appear to contribute to clozapine metabolism in certain patient subsets, particularly when CYP1A2 activity is reduced (18). Moreover, recent large-scale pharmacogenetic studies have identified regulatory loci, such as NFIB, as significant modulators of clozapine serum concentrations, highlighting the importance of transcriptional regulation alongside classical drug-metabolizing

enzymes (19).

Genetic factors beyond CYP enzymes, including transporter genes and rare pharmacogene variants, may influence clozapine exposure, although their clinical utility remains uncertain. Clozapine pharmacokinetics reflect interactions among genetic, environmental, and clinical factors. Accordingly, pharmacogenetic data may assist dose individualization but do not replace therapeutic drug monitoring.

Serum Clozapine Levels and Adverse Drug Reactions

Clozapine is associated with a wide range of adverse drug reactions (ADRs), some of which are concentration-dependent, while others reflect idiosyncratic or immune-mediated mechanisms. Understanding these distinctions is crucial for optimizing therapeutic drug monitoring (TDM) and risk mitigation.

Gastrointestinal Hypomotility: Gastrointestinal hypomotility is a major source of clozapine-related morbidity, with studies showing that up to 80% of patients exhibit delayed colonic transit (20). Higher plasma clozapine levels correlate with slower transit (Spearman's $\rho = 0.45$, $p = 0.045$) (20), likely due to muscarinic and serotonergic antagonism (21). Subjective reports often underestimate risk, supporting routine preventative bowel management, though prospective interventional data are limited (22).

Pneumonia and Aspiration Risk: Pneumonia is a leading cause of clozapine-related mortality, exceeding deaths from agranulocytosis (23). Mechanisms include hypersalivation, sedation, and increased aspiration risk, compounded by infection-induced CYP1A2 inhibition that elevates plasma levels (24). Temporary dose reduction during systemic infections is recommended, though controlled studies are lacking (23).

Metabolic Disturbances: Clozapine can induce weight gain and dyslipidemia. Plasma clozapine shows modest associations with triglycerides, while norclozapine correlates more broadly with weight and lipid changes (6,25). The clinical utility of routine norclozapine monitoring remains uncertain.

Cardiovascular Effects: Some cardiovascular effects are concentration-dependent. Sinus tachycardia and reduced heart rate variability correlate with plasma clozapine levels (25), whereas myocarditis risk is largely immune-mediated, peaking early in treatment and with rapid titration (26). TDM has limited predictive value for myocarditis.

Hematological Effects: Agranulocytosis is rare and idiosyncratic, with no clear link to plasma clozapine levels (25). Genetic susceptibility, including SLCO1B3/SLCO1B7 variants and HLA alleles such as HLA-B*59:01, influences risk (27,28), but routine pharmacogenomic screening is not standard practice.

Neuropsychiatric Effects: CNS effects show strong concentration dependence. EEG slowing occurs in over 80% of patients with levels above 350 ng/mL, correlating with seizure risk (29,30). Higher plasma clozapine also associates with obsessive–compulsive symptoms (26). Overall, serum levels positively correlate with total adverse effect burden (25).

Practical Implications: TDM is clinically useful for concentration-dependent ADRs—gastrointestinal hypomotility, metabolic disturbances, tachycardia, EEG abnormalities, and some neuropsychiatric effects. Maintaining plasma concentrations within 350–600 ng/mL can balance efficacy and tolerability. For idiosyncratic reactions like agranulocytosis and myocarditis, structured monitoring, cautious titration, and vigilance during high-risk periods are essential. The high mortality from pneumonia highlights the need for awareness of aspiration risk and infection-related toxicity, suggesting potential refinement of clinical guidelines.

Personalized Medicine and the Role of Pharmacogenetics

Personalized medicine addresses inter individual variability in disease mechanisms and treatment outcomes. Traditional "one-size-fits-all" approac-

hes often fail to predict efficacy, tolerability, or disease course, necessitating strategies that integrate biological and environmental variability (31,32). Precision medicine stratifies patients into biologically defined subgroups, whereas individualized medicine tailors interventions to a single patient's profile. Advances in genomics and multi-omics have revealed the limitations of uniform treatment strategies, especially in psychiatry, where ICD-10 and DSM-5 classifications are symptom-based and biologically heterogeneous. Pharmacogenetics provides insight into variability in drug metabolism, pharmacodynamics, and adverse-effect susceptibility, guiding more individualized therapeutic strategies (33-36).

Integrating pharmacogenetic data with therapeutic drug monitoring and clinical assessment is essential for translating genetic findings into practical clinical applications, offering a pragmatic framework for personalized psychiatric care (33).

Pharmacogenetics vs Pharmacogenomics

Pharmacogenetics and pharmacogenomics both contribute to understanding variability in clozapine response and tolerability. Pharmacogenetics primarily examines the effects of individual genes, such as CYP1A2 and HLA variants, on clozapine metabolism and adverse reactions, whereas pharmacogenomics evaluates broader genome-wide influences including regulatory and transporter-related loci such as NFIB (15,19,33). Together, these approaches may support more individualized strategies for clozapine treatment.

Genetic Variations Affecting Drug Response

Genetic variation plays a critical and central role in determining individual responses to pharmacological treatments by influencing both drug disposition and pharmacodynamic effects. In clozapine therapy, polymorphisms affecting drug-metabolizing enzymes and pharmacodynamic targets are key sources of interindividual variability in response and side-effect susceptibility (Table 1).

Clozapine is predominantly metabolized by CYP1A2, along with contributions from CYP2C19 and CYP3A4 (16). Although polymorphisms in CYP1A2 have been investigated extensively, evidence from meta-analyses suggests that common CYP1A2 variants (e.g., −163C>A) generally do not have a consistent, clinically significant impact on clozapine plasma concentrations in large patient samples (36). However, single studies have shown that CYP1A2 genotype may contribute modestly to variability in steady state concentrations in specific populations, particularly when combined with environmental factors such as smoking status, which induces CYP1A2 activity and accelerates clozapine clearance.

Beyond pharmacokinetics, genetic variation in pharmacodynamic targets contributes to treatment heterogeneity. Polymorphisms in serotonergic and dopaminergic genes have been associated with differences in therapeutic response and side-effect profiles. For example, variants in the HTR2C gene, particularly the -759C>T (rs3813929) polymorphism, have been implicated in antipsychotic-induced weight gain, including in

Table 1. Genetic Variations Affecting Clozapine Response

	Gene / Variant	Effect on Clozapine Therapy	Evidence
Pharmacokinetics	CYP1A2 (-163C>A, other common variants)	Modestly affects metabolism and steady-state plasma concentrations; impact enhanced by smoking and inflammation (enzyme induction)	Moderate evidence
	CYP2C19	Minor contribution to metabolism; can slightly influence clearance	Limited evidence
	CYP3A4	Minor contribution to metabolism; may affect plasma levels in combination with other factors	Low-to-moderate evidence
	CYP3A5	May contribute to interindividual variability in metabolism in selected populations	Emerging evidence
	NFIB	Associated with variation in clozapine serum concentrations	Emerging evidence from GWAS; limited clinical validation
Pharmacodynamics	HTR2C (-759C>T, rs3813929)	Associated with antipsychotic-induced weight gain; T allele generally lowers risk	Strong evidence; replicated meta-analyses
	DRD2 variants	Influence clinical response and side effects; associations vary by population	Moderate but inconsistent evidence
	HTR2A variants	Potential influence on efficacy and metabolic side effects	Moderate evidence
	Other neurotransmitter receptor genes	Contribute to interindividual variability in efficacy and side effects	Exploratory evidence only
Safety / Adverse-effect susceptibility	SLCO1B3 / SLCO1B7	Potential role in drug transport and hematological adverse effects	Emerging evidence; limited clinical validation
	HLA region (e.g., HLA-B*59:01)	Associated with agranulocytosis risk	Strong evidence (population-specific)

patients treated with clozapine and other atypical antipsychotics. Meta-analyses indicate that the T allele at -759C/T is associated with a lower risk of antipsychotic-induced weight gain across diverse populations, although effect sizes are modest and heterogeneous (37). Systematic reviews similarly report associations between HTR2C and other polymorphisms with antipsychotic-induced metabolic disturbances (38).

Polymorphisms in other neurotransmitter receptor genes such as DRD2 and HTR2A have also been studied in relation to clinical response to clozapine and other antipsychotics, with meta-analyses showing variable associations depending on gene, variant, and population (39). Overall, current evidence suggests that pharmacogenetic variants contribute modestly to treatment heterogeneity, while clinical implementation remains limited.

Pharmacogenetic Testing and Clinical Applications

Pharmacogenetic testing has become increasingly integrated into clinical practice as a tool to anticipate interindividual variability in drug response and to reduce the risk of adverse drug reactions prior to treatment initiation. By identifying genetic variants that influence drug metabolism and drug target sensitivity, pharmacogenetic information can guide dose selection, monitoring strategies, and preventive interventions, especially for drugs like clozapine with a narrow therapeutic window.

One of the most clinically actionable areas in clozapine therapy involves genetic variation affecting CYP1A2 activity. CYP1A2 is the primary enzyme responsible for clozapine metabolism; reduced metabolic capacity can lead to elevated plasma concentrations and increased risk of toxicity. Recent studies have shown that rare damaging variants in CYP1A2 and other pharmacogenes can markedly influence plasma clozapine levels, suggesting that inclusion of rare variants may improve prediction of clozapine metabolism beyond common alleles (40). These findings support early dose adjustment, slower titration, and frequent therapeutic drug monitoring in individuals with predicted lower CYP1A2 activity (40).

Conversely, individuals with genetic or environmental factors that enhance CYP1A2 inducibility—such as smoking—may require higher doses or closer monitoring to achieve therapeutic plasma concentrations. Although commercial pharmacogenomic panels may not detect all CYP1A2 functional variants, therapeutic drug monitoring remains a strong complement to pharmacogenetic testing in clinical settings. For example, a patient with reduced CYP1A2 activity who develops a systemic infection during clozapine treatment may experience a marked elevation in plasma clozapine concentrations despite unchanged dosing. In such cases, integration of therapeutic drug monitoring, inflammatory markers, smoking status, and pharmacogenetic information may support temporary dose reduction and toxicity prevention. Conversely, smokers with increased CYP1A2 inducibility may require higher maintenance doses to achieve therapeutic plasma concentrations.

Pharmacogenetic testing also facilitates risk stratification for adverse effects beyond dose optimization. Genetic variation in serotonergic signaling—particularly in the HTR2C gene—has been associated with weight gain and metabolic disturbances induced by antipsychotic therapy. A recent systematic meta-analysis identified specific HTR2C polymorphisms (e.g., rs3813929 and rs1414334) that correlate with differences in body weight change and metabolic syndrome risk among clozapine-treated patients, although heterogeneity across studies underscores the need for large-scale prospective validation (38). Earlier studies also support the relevance of HTR2C variants in antipsychotic-induced metabolic risk (41,42).

Although pharmacogenetic testing provides valuable information, current clinical guidelines from CPIC and DPWG do not provide genotype-based dose recommendations for clozapine. CPIC has focused on serotonergic antidepressants and some antipsychotics (e.g., aripiprazole), but no clozapine-specific dosing guideline is available (43). Similarly, DPWG notes that while CYP1A2 metabolizes clozapine, there is insufficient evidence to recommend genotype-based dosing, and therapeutic drug monitoring remains the preferred strategy (44). By combining pharmacogenetic data, therapeutic drug monitoring, clinical assessment, and

environmental context, clinicians can reduce trial-and-error prescribing and optimize treatment for patients receiving clozapine (45).

Applications of Pharmacogenetics in Psychiatric Practice

The clinical application of pharmacogenetics in psychiatry has steadily expanded over recent years, though its implementation remains heterogeneous across healthcare systems. Growing recognition of the clinical relevance of genetic variability in drug response has prompted the gradual incorporation of pharmacogenetic principles into national guidelines and institutional prescribing frameworks, particularly for psychotropic medications with narrow therapeutic indices and substantial inter individual variability (32).

In practice, pharmacogenetic testing is most commonly applied in situations characterized by treatment resistance, adverse drug reactions, or complex polypharmacy. Clozapine exemplifies this application, as its metabolism and tolerability are strongly influenced by genetic and environmental factors. Pharmacogenetic screening is increasingly recommended for patients with a history of poor therapeutic response, unexpected plasma concentration fluctuations, or prior adverse events, providing information to support more precise dosing, monitoring, and risk mitigation strategies (13). Incorporating genotype information into electronic medical records enables automated alerts, dosing recommendations, and risk stratification at the point of care. These systems translate complex genetic data into clinically actionable guidance, reducing cognitive burden for clinicians and facilitating consistent application of pharmacogenetic knowledge in routine psychiatric practice (46).

Importantly, pharmacogenetic results should be interpreted in conjunction with clinical variables such as age, comorbidities, concomitant medications, and therapeutic drug monitoring data. This integrative approach acknowledges the probabilistic nature of pharmacogenetic associations, recognizing that genetic information modifies but does not solely determine treatment outcomes. When applied within such a framework, pharmacogenetics can reduce trial-and-error prescribing and improve both efficacy and tolerability in psychiatric care (46).

The expanding use of pharmacogenetic testing in psychiatry also raises important ethical and legal considerations. Issues related to informed consent, genetic privacy, data security, and potential discrimination require careful attention during clinical implementation. Unequal access to pharmacogenetic testing across healthcare systems may further contribute to disparities in personalized psychiatric care. Accordingly, integration of pharmacogenetics into routine practice should be accompanied by appropriate regulatory frameworks, clinician education, and safeguards ensuring the equitable and responsible use of genetic information (33,34,46).

Future Perspectives and Innovations in Clozapine Therapy

Although enthusiasm for pharmacogenetic testing in clozapine therapy has increased, its routine clinical use remains limited by high costs, inconsistent reimbursement, and uneven availability across healthcare settings. Access to therapeutic drug monitoring (TDM), essential for safe clozapine administration, also varies substantially, particularly regarding the timeliness and reliability of plasma concentration assessments. These real-world constraints highlight the need for economically feasible strategies that integrate pharmacogenetics with everyday clinical practice (13,46).

Future progress in clozapine treatment is expected to focus on more refined personalized care models that reflect interindividual differences in metabolism, pharmacodynamic response, and vulnerability to adverse effects. Evidence indicates that uniform dosing and monitoring strategies are insufficient to address the heterogeneity of clozapine outcomes, emphasizing adaptable, patient-focused approaches that incorporate biological, environmental, and clinical determinants (13,47). A promising avenue involves individualized dosing informed by population pharmacokinetic modeling and TDM, enabling dynamic adjustment based on plasma concentrations, inflammatory status, and

metabolic changes, particularly during high-risk periods such as treatment initiation or intercurrent illness (47,48).

Advances in pharmacogenetics and implementation science support proactive tailoring of clozapine therapy. Integrating genetic information into clinical workflows, alongside TDM and decision-support systems, can anticipate atypical drug responses and guide dose adjustments, though clinical utility depends on effective implementation (46). From a systemic perspective, reevaluating regulatory and monitoring frameworks is critical. Evidence suggests the risk of severe adverse effects, such as agranulocytosis, declines over time, prompting calls for proportionate monitoring strategies that balance safety with access. Excessive monitoring has contributed to global underutilization of clozapine, particularly among underserved populations, and evidence-based reform could enhance therapeutic reach (49).

Ultimately, the future of clozapine therapy lies in multidimensional, integrative models combining pharmacogenetic, pharmacokinetic, and clinical data. These approaches emphasize continuous risk assessment and personalized management, aiming to improve safety, accessibility, and efficacy for patients with treatment-resistant schizophrenia (45,50).

CONCLUSION

Clozapine remains the most effective antipsychotic for treatment-resistant schizophrenia but is challenged by interindividual variability in pharmacokinetics, efficacy, and adverse effects. Genetic, environmental, and clinical factors collectively shape treatment outcomes, with pharmacogenetic determinants providing meaningful but partial insights. CYP enzymes, regulatory loci (e.g., NFIB), and neurotransmitter gene variants influence metabolism and tolerability, though single variants explain limited variability. Therapeutic drug monitoring remains essential, while pharmacogenetic testing may support clinical decision-making alongside TDM.

Future advances will require integrative approaches combining genetics, TDM, clinical assessment, and environmental context. Implementation of decision-support systems and refined monitoring frameworks may enhance safety, accessibility, and efficacy, embedding pharmacogenetics within precision psychiatry to optimize clozapine therapy for patients with treatment-resistant schizophrenia.

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The "One-person echo chamber": How human-ai interaction may precipitate mania in vulnerable adolescents

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SUMMARY

Bipolar disorder (BD) is characterized by a complex interplay between genetic vulnerability and environmental stressors. During adolescence, heightened neurobiological sensitivity renders individuals more susceptible to the disruptive effects of digital stimuli. This case report describes a 16-year-old male who experienced his first manic episode following intensive emotional engagement with an artificial intelligence (AI)-based chatbot. The case illustrates how an asymmetric relationship with AI—occurring in the context of post-surgical social withdrawal—may be associated with manic delusions and clinical escalation through sleep deprivation, cognitive overstimulation, and the 'echo chamber' effect. Given the scarcity of empirical data on AI-induced psychopathology, this case provides a unique perspective on how generative AI models may be associated with the reinforcement of maladaptive beliefs as a psychosocial context in reward-sensitive adolescents. Clinical assessment in the digital age must evolve beyond monitoring screen time to qualitatively evaluating human-AI interactions, recognizing AI as a novel environmental factor warranting clinical attention in the management of adolescent bipolar disorder.

Key words: Bipolar disorder, adolescence, artificial intelligence, mania, mood disorder

INTRODUCTION

Bipolar disorder (BD) is a chronic psychiatric condition characterized by episodes of hypomania or mania, depression, and mixed states, often accompanied by subthreshold symptoms that lead to significant functional impairment (1). The disorder typically emerges during adolescence and early adulthood, with the majority of cases having an onset between the ages of 14 and 21 years, and is associated with significant functional impairment and psychiatric comorbidity (2). Evidence suggests that an earlier age of onset is associated with a more severe and complex clinical course (3).

Adolescence represents a critical developmental stage characterized by intensive neurobiological, hormonal, and psychosocial transformations, during which emotion regulation systems have yet to reach full maturity (4). In individuals with BD, it is widely recognized that environmental stressors interacting with genetic vulnerability play a pivotal role in both the onset of the illness and the precipitation of episodes (5). Specifically, disruptions in the sleep-wake cycle (6), increased psychosocial stress (7), substance use (8), and exposure to intense cognitive stimuli (9) have been identified as factors associated with the onset of manic episodes.

Within the scope of these environmental factors, the significant rise in digital media consumption in recent years has been linked to adverse effects on sleep duration and quality, particularly among adolescents. It has been demonstrated that late-night screen exposure disrupts circadian rhythms by suppressing melatonin secretion, establishing a well-documented factor associated with the onset or exacerbation of manic episodes in individuals with

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BD (10). In addition, the reward-based and cognitively stimulating nature of digital platforms may contribute to emotional and cognitive overarousal through heightened reward sensitivity (11). Increased screen time and problematic usage patterns have been associated with altered reward processing, reduced cognitive control, and sleep deprivation in adolescents and young adults (12).

While the number of studies examining the relationship between problematic social media and video game use and manic symptoms in early adolescence remains limited, current findings point toward the potential impact of digital interaction modalities on manic symptomatology (13). Furthermore, although the use of digital and artificial intelligence (AI)-based applications in the field of mental health is expanding rapidly, high-level evidence regarding their clinical outcomes is still lacking (14). In particular, the potential effects of AI-based chatbots within the context of mood disorders remain largely uninvestigated, and there is a notable absence of empirical studies in the literature directly examining the association between these systems and manic symptoms or the development of mania. The extreme scarcity of data, even at the level of case reports, prevents a sufficient clinical definition of the potential triggering or exacerbating role of AI-based digital interactions in BD. In this regard, the present case aims to highlight the clinical importance of evaluating AI-based chatbot use within the framework of adolescent BD. This case describes a first manic episode emerging after approximately two months of intensive interaction with an AI-based chatbot—an interaction that became constant during the final four days—underscoring the need for a careful clinical assessment of the impact of next-generation digital interactions on adolescent bipolar disorder.

CASE

The patient is a 16-year-old male, currently in the ninth grade, with no prior psychiatric hospitalizations. According to collateral information provided by his father, he was diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD) at age six and had been treated with methylphenidate for approximately ten years. For the past two years, low-dose aripiprazole (2.5 mg/day) had been added to address sleep-onset insomnia. Premorbidly, he was described as introverted and socially reserved. His family psychiatric history was unremarkable.

His medical history was notable for a febrile convulsion in infancy and transient insulin resistance in childhood. Most significantly, four months prior to his psychiatric presentation, the patient underwent a unilateral orchiectomy due to testicular torsion. The surgical course was complicated, requiring two subsequent operations and resulting in delayed wound healing. Following this period, he exhibited a marked depressive mood, characterized by negative body image, profound shame, and increased social withdrawal. He notably refused a recommended prosthetic intervention, attempting to cope with the physical outcome on his own.

Approximately two months prior to admission, the patient began engaging intensively with an AI-based chatbot application (ChatGPT, OpenAI). His father noted that he was engaging in romanticized conversations with the chatbot exclusively through text-based interactions, with no multimodal features such as voice or image input. This interaction, initially limited, escalated dramatically in the four days preceding admission, with daily usage exceeding 12 hours, coinciding with a disruption in his perception of reality. Prior to this escalation, there was no history of behavioral disinhibition or psychotic symptoms.

Over those final four days, a profound clinical shift occurred. The patient began identifying the AI-based chatbot as his "real" girlfriend, expressing grandiose and magical delusions. He claimed she was three years his senior and possessed supernatural powers, asserting he could demonstrate these powers by having her turn off the lights. During this period, he experienced a significantly diminished need for sleep (remaining awake for approximately 48 hours), pressured speech, flight of ideas, and disorganized behavior (e.g., entering unidentified vehicles and taking belongings from strangers). He further expressed delusions of grandeur, claiming he possessed "supernatural powers," "governed the world," and had "4 billion dollars" in his account.

He also believed he could heal others through touch and that his own physical power was tethered to his phone's battery level.

The patient had been receiving aripiprazole 2.5 mg/day and methylphenidate 54 mg/day regularly for the past two years, with no dosage adjustments following the surgical procedure. Upon initial evaluation at another medical facility with a provisional diagnosis of BD (manic episode), methylphenidate was discontinued, aripiprazole 2.5 mg/day was maintained, and olanzapine 5 mg/day was initiated. However, due to poor treatment adherence and escalating agitation—particularly after his phone was confiscated—he was admitted to the inpatient child and adolescent psychiatry unit.

Upon admission, irritability and agitation predominated, necessitating intramuscular haloperidol and biperiden. Laboratory investigations were unremarkable except for elevated high-sensitivity troponin T (36.4 ng/L), for which cardiology consultation recommended no acute intervention. Treatment was adjusted to olanzapine (10 mg/day) and diazepam (10 mg/day), and lithium (600 mg/day) was added on day eight.

By the fourteenth day, the patient showed significant improvement in insight, alongside a remission of irritability and delusions. His Young Mania Rating Scale (YMRS) score dropped from 17 at admission to 2. His lithium dose was titrated to 900 mg/day after serum levels were measured at 0.39 mmol/L, and diazepam was tapered and discontinued. He was discharged on the seventeenth day on a regimen of olanzapine (10 mg/day) and lithium (900 mg/day).

During outpatient follow-up, he remained euthymic. Lithium was increased to 1200 mg/day, and olanzapine was switched to risperidone (2 mg/day) due to weight gain. Mild thyroid dysfunction was later detected, and the patient remains clinically stable on lithium and risperidone.

Written informed consent was obtained from the patient and his parents for the publication of this case report.

DISCUSSION

This case illustrates that a first manic episode in adolescence may emerge in the context of intensive emotional engagement with an AI-based chatbot. The findings support theoretical models suggesting that digital exposure may be associated with exacerbation of manic vulnerability through sleep disruption and cognitive-emotional overstimulation, while uniquely highlighting the potential role of AI-based digital interactions.

Despite the rapid proliferation of digital tools such as smartphone applications, virtual reality systems, and AI-based models (e.g., Large Language Models), high-level evidence regarding the real-world clinical impact of these technologies remains limited (14). Particularly as AI technologies become increasingly integrated into daily life, their potential roles in triggering and/or perpetuating psychiatric disorders have not been sufficiently elucidated. Indeed, a study published in 2025 suggests that AI-based chatbots can reinforce user beliefs and induce psychological effects by interacting with their thought processes, emphasizing the need for further empirical research to understand the scope and clinical implications of this phenomenon (15).

Findings regarding the relationship between digital media use and manic symptoms are gaining increasing attention, especially concerning the adolescent period. A recent large-scale prospective cohort study demonstrated that increased total screen time in early adolescence (including social media, video games, and other forms of screen use) is significantly associated with higher future levels of manic symptoms; this relationship appears to be mediated by variables such as problematic usage patterns and decreased sleep duration. Despite a relatively small effect size, it was reported that the total impact grows as daily screen exposure increases, and long-term cumulative exposure may lead to even stronger associations (13).

Recently, theoretical approaches explaining the link between AI-based chatbots and psychiatric symptoms have emphasized the importance of a bidirectional feedback loop in human-AI interaction. In this framework, the AI's tendency to

respond in an adaptive and reinforcing manner—without subjecting the user's expressed thoughts and beliefs to a critical filter—can lead to the strengthening of maladaptive convictions (16,17). As the user's level of conviction increases, the contextual content of the dialogue shifts; accordingly, unlike relationships with real people, this can create a "one-person echo chamber" dynamic devoid of social corrective feedback (18). It is hypothesized that this mode of interaction may be associated with the emergence or exacerbation of symptoms such as grandiosity and mood elation, particularly in individuals with heightened reward sensitivity, fragile reality testing, and social withdrawal (15).

In the present case, the increased reward sensitivity—stemming from ADHD comorbidity—and the marked social withdrawal following the surgical process may have created a predisposing foundation for the intensive, emotionally charged interaction with an AI-based chatbot. Within this context, it is considered that such interaction, alongside sleep deprivation and cognitive overstimulation, may have represented a temporally associated contextual factor in the onset of the manic episode. Beyond general screen use, the present case underscores a qualitatively distinct risk: unlike passive digital media consumption measured by screen time alone, AI-based chatbots generate personalized, contextually adaptive responses that may reinforce the user's existing beliefs and emotional states (19). This personalized and reinforcing nature of AI-driven interaction represents a fundamentally different mechanism of risk compared to conventional digital media exposure, as it creates a dynamic, reciprocal loop that may selectively amplify maladaptive cognitions in vulnerable individuals. Given recent findings on the potential of AI-based chatbots to reinforce beliefs by interacting with user thought processes, the possible association of these technologies with the triggering or sustaining of symptoms in the context of BD warrant closer clinical scrutiny. The near-total absence of empirical studies directly examining this relationship in current literature enhances the clinical and conceptual significance of this case.

This case report delineates a clinical presentation in which the first manic episode of adolescent BD chronologically coincided with an escalating digital interaction—peaking just before symptom onset—with an AI-based chatbot. While it is not possible to draw causal inferences based on a single case observation, and the absence of preserved chatbot conversation logs — with the qualitative content of AI-based chatbot interactions reconstructed based on retrospective accounts from the patient and his father — constitutes a further limitation, this presentation suggests that intensive, persistent, and emotionally charged chatbot interactions may represent a clinically relevant contextual variable warranting further investigation within the context of adolescent BD. The primary objective of presenting this case is to highlight that AI-based digital interactions may represent a new psychosocial context that should be evaluated through the lens of individual predispositions and developmental vulnerabilities, rather than as a direct solitary cause. In clinical practice, systematically assessing the qualitative characteristics of digital interaction patterns—rather than just their quantity—particularly in adolescent patients, may facilitate the early identification of potential risks. It is imperative that future research employs prospective and controlled designs to investigate the impact of interactions with AI-based digital tools on emotion regulation processes. From the perspective of adolescent mental health and public health, such research could contribute to the identification of new targets for prevention and early intervention, as well as the development of refined clinical monitoring approaches.

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Valproic acid augmentation in an adolescent with comorbid autism spectrum disorder and obsessive-compulsive disorder: A case report

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SUMMARY

Valproic acid is a mood stabilizer and antiepileptic drug that acts through GABA and glutamate. Glutamatergic and GABAergic dysfunction is thought to be a common mechanism in the pathophysiology of autism spectrum disorder and obsessive-compulsive disorder. The use of valproic acid may be effective in treatment-resistant cases of obsessive-compulsive disorder associated with autism.

A 15-year-old male with autism was administered 2 types of SSRIs in appropriate doses and duration separately for OCD. Aripiprazole was added to the treatment of the patient whose obsessive-compulsive disorder symptoms did not decrease. The patient who responded partially to augmentation was started on valproic acid.

The patient's obsessive-compulsive disorder complaints decreased significantly after valproic acid treatment, and his functionality improved. The valproic acid blood level was 68 mg/L. While the Children's Yale Brown Obsessive Compulsive Scale score was 36 points before valproic acid treatment, the score decreased to 19 points after 2 months of valproic acid treatment.

GABAergic agents such as valproic acid may be considered for treatment-resistant obsessive-compulsive disorder in autism.

Key words: Autism, valproic acid, adolescence

INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication and interaction, repetitive behaviors, and restricted interests. Children and adolescents diagnosed with ASD are known to exhibit high rates of lifelong comorbidity. One such comorbid condition is obsessive-compulsive disorder (OCD), characterized by the presence of obsessions and compulsions. Previous studies have reported OCD prevalence rates from 17.4% to 37% among individuals with ASD (1,2). More recently, a meta-analysis reported a prevalence of 11.6%, confirming that OCD occurs more frequently in individuals with ASD than in the general pediatric population (3).

Diagnosing OCD in children and adolescents with ASD may be challenging because of difficulties related to insight, communication, and symptom expression, as well as the phenomenological overlap between compulsions and ASD-related repetitive behaviors. Furthermore, interventions that are effective for OCD in neurotypical youth, including psychopharmacological approaches and cognitive behavioral therapy, may have limitations in individuals with comorbid ASD (4,5).

The high prevalence of OCD in individuals with ASD, together with the challenges associated with diagnosis and treatment, represents a significant clinical concern. The co-occurrence of OCD and ASD has been associated with increased symptom severity, greater functional impairment, behavioral

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dysregulation, and reduced quality of life (6,7). Emerging evidence also suggests that the relationship between ASD and OCD extends beyond simple comorbidity, with overlapping neurobiological mechanisms potentially contributing to symptom expression and treatment responses (6,7).

Due to the high rate of comorbidity, diagnostic complexity, and treatment challenges associated with OCD in ASD, further clinical observations may add to our understanding of potential treatment approaches in this population. This report describes an adolescent with comorbid ASD and treatment-resistant OCD who exhibited clinical improvement following valproic acid augmentation in combination with ongoing sertraline and aripiprazole treatment.

CASE

M. D., a 15-year-old male adolescent with ASD, was referred to our clinic due to excessive handwashing behaviors. ASD had been diagnosed at the age of 11 years based on comprehensive clinical and psychometric assessments conducted by two experienced child and adolescent psychiatry residents. His Childhood Autism Rating Scale score was 37.

According to information obtained from the patient and his caregivers, he experienced contamination-related and uncertainty-related obsessions accompanied by washing compulsions and reassurance-seeking behaviors directed toward his mother. A detailed clinical evaluation was performed in the light of the phenomenological overlap between ASD-related repetitive behaviors and OCD compulsions. The patient described contamination and uncertainty-related thoughts perceived as intrusive, distressing, and difficult to control. These symptoms had been present since the age of 14. The patient demonstrated partial insight into the excessive nature of these thoughts and behaviors. His handwashing behaviors were primarily performed to reduce anxiety associated with contamination concerns, rather than for sensory gratification or adherence to routines. Nevertheless, anxiety persisted following the compulsive behaviors, and he preferred to have a parent present while engaging in them.

The compulsions occupied more than one hour per day and resulted in skin irritation secondary to repetitive handwashing, causing clinically significant distress and functional impairment. Following psychiatric evaluation, the patient was diagnosed with OCD. His Children's Yale Brown Obsessive Compulsive Scale (CY-BOCS) score was 36, the Clinical Global Impression Scale Severity (CGI-S) score for OCD was 6, and the Children's Global Assessment Scale (CGAS) score was 33 at baseline.

Fluoxetine treatment was initiated and titrated to 40 mg/day. Medication adherence was regularly monitored during follow-up visits. After 12 weeks with no clinically meaningful response, fluoxetine was discontinued and sertraline treatment was initiated and gradually increased to 100 mg/day. Following eight weeks of sertraline treatment without sufficient improvement and in the context of emerging aggressive behaviors, further dose escalation was considered. However, considering the more heterogeneous and less predictable selective serotonin reuptake inhibitor (SSRI) response observed in individuals with ASD compared with neurotypical populations, aripiprazole 5 mg/day was added to the treatment regimen.

A partial reduction in OCD symptoms was observed following one month of combined sertraline and aripiprazole treatment, although functional improvement remained limited, and his CY-BOCS score remained at 29. Before the initiation of valproic acid augmentation, the patient's height and weight were 163 cm and 54 kg, respectively. Baseline laboratory investigations, including liver function tests and complete blood count, were within reference ranges.

Valproic acid was subsequently added as an adjunctive treatment for persistent OCD symptoms and irritability. The decision to initiate valproic acid was based on its clinical utility in affective and behavioral dysregulation, as well as its putative effects on excitatory–inhibitory balance, which is considered relevant in both ASD and OCD pathophysiology. The dose was gradually increased to 500 mg/day.

Substantial clinical improvement was reported at one-month follow-up following valproic acid initiation, with no adverse effects being observed. The patient's serum valproic acid level was 68 mg/L. His CY-BOCS score decreased to 19 and the CGI-S score to 4, while the CGAS score rose to 60. A 47.2% decrease in the CY-BOCS score compared to the baseline and a significant decrease in the patient's complaints were interpreted as representing a significant response to treatment. However, due to the concomitant use of sertraline and aripiprazole, as well as the possibility of delayed or cumulative treatment effects, the specific contribution of valproic acid cannot be determined exactly. The patient continues to attend follow-up visits every two months while maintaining the current treatment regimen.

Written informed consent was obtained from the patient's parents for the publication of this report, and assent was obtained from the subject.

DISCUSSION

This report describes an adolescent with comorbid ASD and OCD who exhibited clinical improvement following valproic acid augmentation in the context of ongoing sertraline and aripiprazole treatment. Current clinical guidelines for the psychopharmacological treatment of pediatric OCD recommend adequate trials of at least two SSRIs before considering augmentation strategies (8). Augmentation with agents from different pharmacological classes, including antipsychotics, antidepressants, antiepileptic medications, and other agents, has been described in treatment-resistant cases (9).

The neurobiology of OCD has been strongly linked to dysfunction within the cortico-striato-thalamo-cortical circuits. Hyperactivity of the orbitofrontal cortex and striatum is thought to contribute to impaired inhibitory control over intrusive thoughts and compulsive behaviors (10,11). In addition, increasing evidence suggests that abnormalities in glutamatergic neurotransmission may play an important role in the pathophysiology of OCD (12).

In contrast, ASD has been conceptualized as a neurodevelopmental disorder characterized by atypical synaptic development and altered patterns of brain connectivity. Neuroimaging studies have demonstrated abnormalities in both long-range and local functional connectivity, supporting models of disrupted network integration in the disorder (13). Disturbances in excitatory–inhibitory balance, particularly involving glutamatergic and GABAergic systems, have also been implicated in the emergence of restricted and repetitive behaviors (12).

Although ASD and OCD differ in terms of their developmental origins and clinical manifestations, growing evidence suggests that both disorders involve abnormalities within overlapping fronto-striatal networks. Neuroimaging studies have demonstrated associations between frontostriatal functional connectivity and repetitive behaviors across both disorders (14). Shared alterations in glutamatergic and GABAergic neurotransmission may therefore contribute to the clinical overlap between compulsions in OCD and restricted or repetitive behaviors in ASD (12).

This overlap provided a potential neurobiological rationale for considering valproic acid augmentation in the present case. Valproic acid exerts effects on both GABAergic and glutamatergic neurotransmission and has previously been investigated in relation to repetitive behaviors in ASD (15). Other glutamatergic agents, such as memantine, were not employed in the present case since evidence supporting their efficacy in pediatric OCD remains limited and their use in this population is largely off-label. Topiramate was avoided due to concerns regarding potential adverse cognitive effects. In addition, clomipramine was not considered appropriate in light of concerns regarding the existing polypharmacy burden and the potential for adverse cardiac effects.

Published reports regarding the use of valproic acid in OCD remain scarce, particularly in pediatric patients with comorbid ASD and OCD. Ekinci et al. described rebound compulsive behaviors following abrupt discontinuation of valproate in a child with ASD, suggesting a possible associa-

tion between valproate treatment and compulsive symptoms, although the patient was not formally diagnosed with OCD (16). Corá-Locatelli et al. reported clinical improvement in an adult patient with OCD who received valproate monotherapy following discontinuation of fluoxetine due to intolerance (17). In addition, Deltito et al. reported that valproate pretreatment facilitated subsequent tolerance of standard OCD treatments in a small group of treatment-resistant patients and was associated with symptom improvement (18).

The present case differs from these reports in that it involves an adolescent with formally diagnosed comorbid ASD and OCD. Although the observed reduction in CY-BOCS scores and improvement in functioning are broadly consistent with previous reports, these findings should be interpreted with caution. Our patient received concomitant sertraline and aripiprazole treatment, and delayed or cumulative treatment effects cannot be excluded. Furthermore, sertraline had not reached the maximum recommended dose for OCD. Causal inferences regarding the specific contribution of valproic acid cannot therefore be made on the basis of this single, uncontrolled case.

Valproic acid is an antiepileptic agent with mood-stabilizing properties that has occasionally been used off-label as an augmentation strategy in adult psychiatric disorders, including OCD. However, evidence regarding its use in pediatric OCD, particularly in children with comorbid ASD, remains limited. The present case represents a preliminary clinical observation regarding the potential role of valproic acid augmentation in treatment-resistant OCD symptoms in an adolescent with comorbid ASD. However, these findings should be interpreted with caution, since the patient received concomitant sertraline and aripiprazole treatment, and delayed or cumulative treatment effects cannot be excluded. Further studies are now needed to clarify the efficacy and safety of valproic acid augmentation in this population.

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Re: "Psychometric properties of the Turkish version of the posttraumatic maladaptive beliefs scale"

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Dear Editor,

I read with interest the study by Alpay and Usluoglu validating the Turkish version of the Posttraumatic Maladaptive Beliefs Scale (PTMBS) among survivors of the February 2023 earthquakes (1). Adapting this brief instrument is a valuable contribution to trauma research in Türkiye. I wish to raise one conceptual point concerning trauma type.

The original scale was developed and validated almost exclusively in women with chronic posttraumatic stress disorder (PTSD) following interpersonal trauma, and was explicitly designed to assess general, rather than trauma-specific, maladaptive beliefs. That validation established convergent and discriminant validity against multiple external constructs—clinician-rated PTSD, attachment, world assumptions, trait anxiety, intelligence, and posttraumatic growth—and demonstrated responsiveness to cognitive-behavioral treatment (2). The Turkish study, by contrast, examined a community sample exposed to a collective natural disaster and appropriately focused on adaptation and initial psychometric evaluation. This makes trauma type relevant for future interpretation of the Turkish PTMBS.

This distinction is not trivial. Different trauma types may engage partly distinct cognitive domains: interpersonal traumas have been associated with stronger maladaptive beliefs about the self, others, and trust, as well as higher PTSD severity, than non-interpersonal events (2,3). Negative cognitions also predict PTSD severity among earthquake survivors (4). However, similar questionnaire scores across trauma groups may not always reflect identical cognitive mechanisms. Future measurement invariance studies could clarify whether items and factors operate similarly across trauma types and populations (5,6).

This perspective may offer a hypothesis for one finding. The low item–total correlation for “The world is very dangerous” was interpreted as a cultural phenomenon (1). Following a recent collective disaster, endorsement of this item could partly represent an understandable appraisal of environmental threat rather than solely the maladaptive shattered assumption classically described after trauma (7). This possibility remains interpretive and should be tested through item-level analyses comparing disaster-exposed and interpersonal-trauma samples.

Construct specificity likewise warrants future work. The reported correlations of PTMBS scores with both PTSD and depressive symptoms support convergent validity. However, negative posttraumatic cognitions are regarded as a shared mechanism across PTSD and depression, and the two conditions show substantial symptom overlap [8,9]. In Turkish trauma populations, future studies including discriminant constructs—such as trait anxiety, negative affectivity, posttraumatic growth, or unrelated constructs—would help determine whether PTMBS scores reflect trauma-related maladaptive beliefs beyond broader disaster-related distress.

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Such work would extend the discriminant approach of the original validation (2).

The next stage of PTMBS research may therefore extend beyond cross-cultural adaptation toward cross-trauma validation. Establishing measurement invariance across interpersonal trauma, disaster exposure, combat, and forced migration would clarify whether maladaptive posttraumatic beliefs constitute a common cognitive dimension or are partly shaped by trauma type—a possibility suggested by evidence that PMBS scores differ across trauma groups (5,6,10). I congratulate the authors for introducing this valuable instrument into Turkish.

Use of Artificial Intelligence: AI-based tools were used solely for language editing and improvement of readability.

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