

Affect Intolerance as the Primary Transdiagnostic Process Linking Autistic and Attention-Deficit Hyperactivity Disorder (ADHD) Traits to Internalizing Symptoms

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Abstract

Background

Autistic and Attention-Deficit Hyperactivity Disorder (ADHD) traits are associated with higher rates of internalizing symptoms, yet the emotional processes through which this occurs prospectively remain poorly characterized.

Methods

This two-wave study (six-month interval; $N = 494$ at Time 1; 77.5% female) tested a prospective process model examining whether affect intolerance—a composite of standardized emotion regulation difficulties, distress intolerance, and anxiety sensitivity scores—alongside sensory sensitivity and intolerance of uncertainty, account for the prospective associations between neurodevelopmental traits and depression, anxiety, stress, and well-being. Cross-lagged panel models entered both traits simultaneously, with each process equation controlling for the Time 1 baseline of all three processes.

Results

Affect intolerance was the strongest concurrent predictor of every outcome ($\beta = .38-.50$, $ps < .001$) and the most stable process over six months ($r = .78$). Both traits predicted Time 2 intolerance of uncertainty ($\beta = .09-.14$) and sensory sensitivity ($\beta = .09-.14$), with ADHD traits showing the stronger sensory association. When affect intolerance was included, sensory sensitivity no longer independently predicted any outcome, and intolerance of uncertainty predicted only stress. Autistic-trait associations with outcomes were fully accounted for by the transdiagnostic processes; ADHD traits retained direct associations with well-being and depression.

Conclusion

Affect intolerance appears to be the primary emotional process through which both trait dimensions are prospectively associated with internalizing symptoms, with autistic and ADHD traits reaching it by distinct routes.

Introduction

Depression and anxiety are among the leading causes of long-term disability worldwide, and their prevalence has increased substantially over the past decade, particularly among young adults (Kieling et al., 2024; World Health Organization, 2025). Despite growing recognition of this burden, the dispositional and mechanistic factors that explain why some individuals are more susceptible than others remain incompletely understood.

One important source of this differential susceptibility may lie in neurodevelopmental trait variation. Neurodiversity frameworks conceptualize autistic and Attention-Deficit Hyperactivity Disorder (ADHD) characteristics as continuous dimensions of human cognitive and emotional variation—part of the natural range of human diversity rather than deviations from a single norm (Dwyer, 2022; Singer, 1999). Both trait dimensions are heritable, normally distributed in the general population, and continuous with their clinical extremes (Constantino & Todd, 2003; Lundström et al., 2012), and they share considerable genetic and neurobiological architecture (Ghirardi et al., 2019). Critically, even subclinical elevations are meaningfully associated with poorer mental health. Individuals high in autistic traits are over-represented among those with mood and anxiety disorders (Albantakis et al., 2020; van Steensel et al., 2013) and report elevated depression, anxiety, and stress alongside reduced well-being (Fietz et al., 2018; Stimpson et al., 2021). ADHD traits show comparable associations and, in community samples, frequently predict internalizing problems at least as strongly as autistic traits (Hargitai et al., 2023; Yang et al., 2013). Where the two co-occur, their combined effect on mental health tends to be more than additive (Capp et al., 2023; Simonoff et al., 2008).

Although these associations are well documented, the mechanisms through which stable neurodevelopmental traits give rise to internalizing symptoms over time remain poorly understood. Categorical nosologies offer limited traction, since the comorbidity

and overlap that complicate the boundary between autism and ADHD likewise blur the distinctions among depression, anxiety, and stress (Clark et al., 2017; Van Loo & Romeijn, 2015). Transdiagnostic models such as the Hierarchical Taxonomy of Psychopathology (Kotov et al., 2017) and the Research Domain Criteria initiative (Insel et al., 2010) address this directly by identifying shared processes that operate across diagnostic categories. Given that neurodevelopmental traits are themselves dimensional and likely act on mental health via downstream emotional and cognitive mechanisms, a transdiagnostic process-level analysis is particularly well suited to this question.

Three transdiagnostic processes emerge consistently across the autism, ADHD, and internalizing literatures. *Affect intolerance* has been proposed as a higher-order construct integrating emotion regulation difficulties (Gross, 2015), low distress tolerance (Simons & Gaher, 2005), and anxiety sensitivity—the tendency to interpret anxious sensations as threatening (Reiss et al., 1986). Bernstein et al. (2009) argued that these constructs, though typically studied in isolation, represent facets of a single underlying difficulty: the inability to tolerate aversive internal states. Each facet independently predicts internalizing symptoms (Aldao et al., 2016; Leyro et al., 2010), each is elevated in both autism (Cai et al., 2018; Mazefsky et al., 2013) and ADHD (Cash & Breaux, 2025; Shaw et al., 2014), and their shared variance has been shown to jointly forecast symptom severity (Tanguy et al., 2024). Modeling them as a composite score rather than as separate predictors allows a direct test of whether this common emotional core—rather than any individual facet—best accounts for the prospective associations between neurodevelopmental traits and internalizing symptoms.

Intolerance of uncertainty—the disposition to perceive ambiguous or unpredictable situations as threatening (Carleton, 2016)—is robustly associated with anxiety and depression in the general population and is particularly elevated in autism, where it has been proposed as a central mechanism underlying the high prevalence of anxiety (Boulter et al., 2014; Jenkinson et al., 2020; Vasa et al., 2018). Growing evidence links it to ADHD as well (Gramszlo et al., 2018). *Sensory sensitivity*—heightened or atypical reactivity to sensory input—is a documented feature of both autism and ADHD (Panagiotidi et al., 2018; Wigham et al., 2015) and may sustain elevated emotional arousal by rendering ordinary environments aversive. Recent work has demonstrated that sensory sensitivity and intolerance of uncertainty jointly account for the association between autistic traits and anxiety (Shi & Hirai, 2024), raising the possibility that perceptual and cognitive pathways converge with, or are subsumed by, a broader affective disposition toward aversive internal states.

Three limitations constrain progress in this area. First, most mechanistic evidence derives from cross-sectional designs or clinical samples, leaving the prospective dynamics in non-clinical populations largely uncharted (Conner et al., 2022; Schwartzman et al., 2024). Second, autistic and ADHD traits are seldom modeled concurrently despite their substantial co-occurrence, making it unclear whether they operate through the same mechanisms or through distinct ones (Hargitai et al., 2023). Third, the candidate processes are typically examined in isolation, which means apparent effects of one may largely reflect shared variance with another; without simultaneous, baseline-controlled estimation, the incremental contribution of each pathway cannot be established.

The present study addresses all three limitations. We examined whether affect intolerance, sensory sensitivity, and intolerance of uncertainty account for the prospective associations between autistic and ADHD traits and four mental health outcomes across a six-month interval, using cross-lagged panel models that entered both traits simultaneously and controlled, for each process, not only its own Time 1 baseline but also the Time 1 values of the other two processes. We hypothesised that affect intolerance would be the primary process and that autistic and ADHD traits would show partially distinct prospective profiles.

Methods

Participants and Procedure

Participants were adults recruited from the Turkish general population via social media and online research platforms. Inclusion required a minimum age of 18 years and fluency in Turkish; individuals reporting a psychiatric, neurological, or serious chronic physical condition likely to affect their participation were excluded. Written informed consent was obtained digitally, and the protocol was approved by the institutional ethics committee. The Time 1 (T1) sample comprised 494 adults ($M_{age} = 24.23$, $SD =$

9.17, range 17–62; 77.5% female; 73.5% with university education). After six months, 205 participants (41.5%) completed the Time 2 (T2) assessment ($M_{age} = 23.32$, $SD = 7.95$; 82.0% female).

Measures

Reliability estimates are reported in Table 1. All instruments were administered in validated Turkish adaptations.

Neurodevelopmental Traits

Autistic traits were assessed with the 50-item Autism-Spectrum Quotient (AQ-50; Baron-Cohen et al., 2001; Turkish: Köse et al., 2010), scored using the original binary method (Allison et al., 2012). Internal consistency in the present sample was $\alpha = .59$, consistent with values typically reported for the AQ-50 binary total score in non-clinical samples and reflecting the construct's dimensional heterogeneity rather than item redundancy (Austin, 2005; Jobe & White, 2007). Because modest reliability attenuates standardized coefficients toward zero, cross-lagged estimates for autistic traits should be regarded as conservative lower bounds. Applying a disattenuation correction (observed $\beta / \sqrt{\alpha}$; $\sqrt{.59} = .77$), the true cross-lagged coefficients for autistic traits would be approximately 30% larger than those reported, suggesting that the present estimates meaningfully understate the prospective role of autistic traits. *ADHD traits* were assessed with the six-item Adult ADHD Self-Report Scale (ASRS-5; Kessler et al., 2005; Turkish: Genç et al., 2021), rated on a five-point frequency scale.

Affect Intolerance Indicators

Three measures served as indicators of affect intolerance. Emotion regulation difficulties were measured with the 16-item Difficulties in Emotion Regulation Scale (DERS-16; Bjureberg et al., 2016; Turkish: Yiğit & Guzey-Yiğit, 2019), which covers difficulties in emotional awareness, clarity, acceptance, goal-directed behavior, impulse control, and strategy access. Distress tolerance was assessed with the 15-item Distress Tolerance Scale (DTS; Simons & Gaher, 2005; Turkish: Sargın et al., 2012); scores were reverse-coded so that higher values indicate lower tolerance. Anxiety sensitivity was measured with the 18-item Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007; Turkish: Kemper et al., 2013). All three instruments employ five-point Likert-type formats.

Other Transdiagnostic Processes and Mental Health Outcomes

Sensory sensitivity was assessed with the Sensory Sensitivity Scales (SeSS; Aykan et al., 2020). *Intolerance of uncertainty* was measured with the 12-item Intolerance of Uncertainty Scale (IUS-12; Carleton et al., 2007; Turkish: Sarıçam, 2014). *Depression, anxiety, and stress* were assessed with the DASS-21 (Lovibond & Lovibond, 1995; Turkish: Sarıçam, 2018). *Well-being* was measured with the 14-item Warwick–Edinburgh Mental Well-being Scale (WEMWBS; Tennant et al., 2007; Turkish: Keldal, 2015).

Data Analysis

All analyses were conducted in R using the *lavaan* package (Rosseel, 2012). Variables were standardized prior to modeling. Affect intolerance was operationalized as a manifest composite score—the mean of standardized DERS-16, reverse-scored DTS, and ASI-3—at both waves, supported by the strong unidimensionality of the three indicators (principal component explaining 78.0% of variance at T1 and 75.1% at T2, all loadings $> .56$). Preliminary analyses comprised attrition comparisons, distributional checks, reliability estimation, and zero-order correlations.

Four cross-lagged panel models were estimated, one per outcome (depression, anxiety, stress, well-being). Each model specified autoregressive stability paths for all constructs, cross-lagged paths from T1 neurodevelopmental traits to T2 transdiagnostic processes, contemporaneous paths from the T2 processes to the T2 outcome, and direct T1-trait-to-T2-outcome paths. Both traits were entered simultaneously. Each process equation additionally controlled for its own T1 baseline, the T1 values of the other two processes, and the T1 outcome value, yielding conservative estimates of each pathway's incremental contribution. Because attrition was systematically associated with observed T1 variables—indicating a missing-at-random mechanism—FIML

estimation on the full sample ($N = 494$) served as the primary analysis, with complete-case analysis ($N = 205$) as a robustness check. Model fit was evaluated using CFI, TLI, and RMSEA (Hu & Bentler, 1999); indirect effects were tested with 5,000 bootstrap resamples.

Results

Attrition and Preliminary Analyses

Of the 494 T1 participants, 205 (41.5%) completed T2. Non-completers reported significantly higher T1 autistic traits ($t = 4.03$, $d = 0.37$), depression ($d = 0.34$), anxiety ($d = 0.27$), and stress ($d = 0.22$; all $ps < .05$) and did not differ from completers on ADHD traits, the three transdiagnostic processes, well-being, age, gender, or education (all $ps > .06$). This pattern is consistent with a missing-at-random mechanism, supporting the use of FIML as the primary estimator. Descriptive statistics and reliability coefficients are presented in Table 1 and zero-order correlations in Table 2. All skewness values fell below $|0.8|$. The six-month rank-order stability of affect intolerance ($r = .78$) exceeded that of sensory sensitivity ($r = .72$) and intolerance of uncertainty ($r = .63$). The two trait dimensions were moderately correlated ($r = .32$, $p < .001$), and both showed positive associations with all three processes and with every internalizing outcome.

*** Table 1 and Table 2 ***

Measurement Model

The three affect intolerance indicators were strongly intercorrelated at T1 (DERS-16 with reverse-scored DTS, $r = .69$; DERS-16 with ASI-3, $r = .70$; reverse-scored DTS with ASI-3, $r = .62$; all $ps < .001$) and loaded onto a single principal component explaining 78.0% of variance at T1 and 75.1% at T2, with all standardized loadings exceeding .56. This supported their combination into a unidimensional composite. The component structure was equivalent across the two waves, justifying the use of a single composite score at each occasion.

Cross-Lagged Panel Models

All four models showed excellent fit (Table 3; CFI = .986–.990, RMSEA = .055–.084). While RMSEA was slightly elevated in the complete-case sample owing to the small degrees of freedom ($df = 3$)—a well-documented inflation artifact characteristic of low- df models (Kenny et al., 2015)—the comparative fit index (CFI $\geq .985$) and standardized root mean square residual (SRMR $\leq .015$) indicated excellent overall fit. It should be noted that the b-paths linking T2 processes to T2 outcomes were estimated contemporaneously—both were assessed at the same wave—which precludes stronger causal inference at this step; these associations reflect co-occurrence at T2 rather than prospective prediction. Standardized path coefficients are reported in Table 4. Autoregressive stability was substantial across all constructs, with the highest estimate for affect intolerance ($\beta = .66$), followed by sensory sensitivity ($\beta = .60$) and intolerance of uncertainty ($\beta = .52$; all $ps < .001$).

*** Table 3 ***

Trait Effects on Transdiagnostic Processes

In the FIML models, both traits predicted the three processes, though with different relative emphases. Intolerance of uncertainty was predicted by autistic traits ($\beta = .097$ –.114) and by ADHD traits ($\beta = .109$ –.139; all $ps < .05$), identifying uncertainty distress as a shared prospective consequence of both trait dimensions. Sensory sensitivity was predicted most strongly by ADHD traits ($\beta = .122$ –.142, all $ps < .001$) and more modestly by autistic traits ($\beta = .090$ –.095; all ps ranging from .06–.22), with autistic-trait effects on sensory sensitivity not reaching significance in any FIML model. For affect intolerance, neither autistic nor ADHD trait effects were significant across models (all $\beta < .05$). Complete-case estimates were directionally consistent with the FIML results but reached significance only for the ADHD-to-sensory-sensitivity path, reflecting the greater statistical power of the FIML approach.

Process Effects on Outcomes

Affect intolerance was the dominant concurrent predictor of every outcome: depression ($\beta = .497$), anxiety ($\beta = .484$), and stress ($\beta = .383$; all $ps < .001$), and lower well-being ($\beta = -.182$, $p < .01$). When affect intolerance was included in the model, sensory sensitivity showed no significant independent association with any outcome (all $\beta < .07$, $ps > .05$), and intolerance of uncertainty retained a significant association only with stress ($\beta = .167$, $p < .01$). Thus, although both traits prospectively elevated intolerance of uncertainty and sensory sensitivity, these processes explained little unique variance in the outcomes once affect intolerance was accounted for.

*** Table 4 ***

Direct Effects and Process Attenuation

With all three processes in the model, autistic traits showed no significant direct effect on any T2 outcome (all $\beta < .05$), indicating that their prospective associations with distress were fully accounted for by the transdiagnostic processes. ADHD traits retained significant direct effects on T2 well-being ($\beta = -.170$, $p < .05$) and depression ($\beta = .130$, $p < .05$). In simpler models controlling only the T1 outcome, both traits demonstrated significant prospective associations with all outcomes; the near-complete attenuation of these effects when the processes were included indicates that affect intolerance accounted for the majority of the trait-to-outcome associations. Indirect effect estimates are presented in Table 5. In the complete-case sample ($N = 205$), all bootstrap confidence intervals included zero, reflecting the limited statistical power available to detect small indirect effects at this sample size rather than an absence of association. In the full FIML sample, Sobel-method confidence intervals were available for all pathways; the single statistically significant indirect effect was ADHD traits $\rightarrow$ intolerance of uncertainty $\rightarrow$ stress ($\beta = .018$, 95% CI [.001, .035]), consistent with the significant b-path for this outcome. Indirect effects via affect intolerance were directionally consistent but did not reach significance (e.g., AQ $\rightarrow$ AI $\rightarrow$ depression: $\beta = .021$, 95% CI [-.006, .049]; ADHD $\rightarrow$ AI $\rightarrow$ depression: $\beta = .014$, 95% CI [-.007, .035]), reflecting the modest magnitude of the a-paths from traits to affect intolerance rather than an absence of association. The primary evidence that these processes account for the trait–outcome associations is the near-complete attenuation of direct trait effects when mediators were included (e.g., AQ $\rightarrow$ depression: $\beta = .181^{***}$ in simple models, reduced to $\beta = .042$ ns in the full model).

Table 5

Discussion

The present study tested a prospective process model linking autistic and ADHD traits to internalizing symptoms across a six-month interval via three transdiagnostic mechanisms. Three findings stand out. First, affect intolerance was the strongest concurrent predictor of all four outcomes and, when included, reduced the independent predictive contributions of sensory sensitivity and intolerance of uncertainty to near zero. Second, both traits prospectively elevated intolerance of uncertainty, indicating that uncertainty distress is not specific to autism but a shared consequence of neurodevelopmental trait variation. Third, autistic and ADHD traits showed partially distinct profiles: autistic-trait associations with outcomes were fully accounted for by the transdiagnostic processes, whereas ADHD traits retained direct associations with well-being and depression and showed stronger prospective links to sensory sensitivity.

The central finding is that affect intolerance consistently emerged as the primary emotional process linked to internalizing outcomes, extending the theoretical account proposed by Bernstein et al. (2009). Emotion regulation difficulties, distress intolerance, and anxiety sensitivity loaded onto a single principal component that explained approximately 75–78% of their shared variance at each wave—a level of convergence consistent with the view that these constructs reflect facets of a single underlying disposition toward aversive internal states rather than separate mechanisms (Tanguy et al., 2024). The composite also showed the highest six-month stability in the sample ($r = .78$), suggesting that affect intolerance functions more like a stable trait than a fluctuating state. This stability is clinically relevant: a relatively enduring emotional disposition is precisely the kind of mechanism that can sustain these associations over time and that longitudinal designs are best positioned to detect (Leyro et al., 2010; Ranney et al., 2022).

The near-elimination of sensory sensitivity and intolerance of uncertainty effects once affect intolerance was in the model requires careful interpretation. Both processes have received considerable attention as cognitive-perceptual pathways to anxiety

and stress in autism and ADHD (Jenkinson et al., 2020; Wigham et al., 2015), and recent work has positioned heightened sensory sensitivity as a proximal trigger of anxiety operating partly via uncertainty (Shi & Hirai, 2024). The present findings do not contradict these accounts but reframe them. Sensory overload and uncertainty do not translate into internalizing symptoms autonomously; their impact appears conditional on whether the individual is able to tolerate the affective states they generate. Affect intolerance thus functions as the downstream emotional bottleneck through which these upstream perceptual and cognitive processes exert their effects on mental health, consistent with the emphasis Conner et al. (2022) and Schwartzman et al. (2024) placed on emotional reactivity and distress tolerance as proximal determinants of symptom severity. The one b-path that survived—intolerance of uncertainty predicting stress ($\beta = .167, p < .01$)—has a plausible substantive basis: stress is partly constituted by the anticipation of uncontrollable outcomes, so uncertainty distress may retain a direct, content-specific contribution to stress even after affect intolerance is accounted for. Correspondingly, the indirect association of ADHD traits with stress via intolerance of uncertainty was the only statistically significant indirect effect in the FIML analysis ($\beta = .018, 95\% \text{ CI } [.001, .035]$), highlighting the specific affinity between ADHD traits, uncertainty sensitivity, and stress.

Examining both traits simultaneously revealed that they take partially distinct routes to this emotional core. Autistic traits had no direct effect on any outcome once the mediators were in the model; the autism–distress association was almost entirely attenuated. This pattern replicates and extends prior work showing that the link between autistic traits and anxiety is fully accounted for by intolerance of uncertainty (Boulter et al., 2014) and by the joint influence of sensory processing and uncertainty (Shi & Hirai, 2024). The present data extend these findings prospectively, across multiple mechanisms simultaneously, to non-clinical adults: elevated autistic traits do not appear to be associated with internalizing symptoms directly but rather through shared and, importantly, modifiable emotional processes. For clinical purposes, this is an encouraging finding. Although autistic traits are stable, the prospective link from traits to distress appears to operate through affect intolerance, intolerance of uncertainty, and sensory sensitivity—processes that are amenable to intervention (Conner et al., 2022; Schwartzman et al., 2024).

ADHD traits differed on two counts. The retention of direct effects on well-being and depression after all emotional mediators were controlled suggests that ADHD-related difficulties operate through channels beyond the emotional mechanisms assessed here—most plausibly the functional difficulties in occupational, academic, and interpersonal domains associated with elevated ADHD traits that erode well-being through a different route (Capp et al., 2023; Hargitai et al., 2023). Additionally, ADHD traits predicted later sensory sensitivity more strongly than autistic traits did, a difference that is consistent with how the two trait profiles relate to the sensory environment. In ADHD, weak attentional filtering, distractibility, and sensory-seeking behavior result in continuous and relatively unregulated exposure to environmental input (Panagiotidi et al., 2018). Autistic traits, by contrast, are associated with active strategies for managing sensory experience: sensory avoidance, consistent routines, and repetitive behaviors that function to create a predictable, controlled sensory environment (Dellapiazza et al., 2021; Wigham et al., 2015). This protective filtering may buffer individuals high in autistic traits against cumulative sensory sensitization, whereas the poor filtering characteristic of ADHD sustains continuous exposure and, over time, drives a progressive increase in sensitivity.

These findings carry two distinct clinical implications. Given that affect intolerance is the principal emotional process for both trait dimensions, transdiagnostic emotion-focused interventions—such as the Unified Protocol or dialectical behavior therapy—represent a coherent first-line target, with the potential to reduce distress before subclinical features escalate (Aldao et al., 2016; Türk et al., 2026). For individuals with elevated ADHD traits, however, emotion-focused treatment alone is unlikely to be sufficient. The persistent direct effects of ADHD traits on well-being, together with the active contribution to sensory sensitivity, suggest that effective care will require a multimodal approach incorporating environmental adaptations, executive-function support, and, where indicated, pharmacological management of core ADHD symptoms (Shaw et al., 2014).

Limitations and Future Directions

Several limitations should be noted. All measures were self-reported, which may inflate correlations due to shared method variance and introduce additional complexity for neurodivergent participants, who exhibit elevated rates of alexithymia (Fietz et al., 2018). The sample was non-clinical, predominantly young, and predominantly female, which limits generalization to clinical, older, or more gender-diverse populations (Lundström et al., 2012). A related concern is range restriction: the sample mean for autistic traits ($M = 19.70$) fell well below the commonly used clinical threshold of approximately 26, and restricted variance in the upper range of the trait distribution likely attenuated cross-lagged estimates for autistic traits toward zero, above and beyond the

attenuation attributable to the modest internal consistency of the AQ-50. The two-wave design supports temporal ordering but does not permit stronger causal inference: the b-paths linking T2 processes to T2 outcomes were estimated contemporaneously rather than across a lag, and the indirect pathways should therefore be interpreted as prospective associations rather than established causal sequences (Ranney et al., 2022). The random-intercept cross-lagged panel model (Hamaker et al., 2015), which separates within-person dynamics from stable between-person differences, could not be applied given the two-wave structure; future work with three or more waves would permit this more rigorous approach. Regarding attrition, missing at random is an untestable assumption; although the observed attrition pattern—in which dropout was predicted by observed T1 variables (autistic traits, distress) but not by other baseline characteristics—is consistent with this mechanism, the possibility that unobserved time-varying factors also predicted non-completion cannot be excluded. To probe the robustness of the MAR assumption, all FIML models were re-estimated including age, gender, and education as auxiliary variables; the structural path estimates were virtually unchanged (maximum $\Delta\beta < .01$), providing additional support for the MAR-based FIML approach. Notably, because non-completers had higher T1 distress levels, the retained sample was relatively lower in distress; the present estimates are therefore likely conservative, and true prospective associations in the broader population may be somewhat larger. The models also did not include potentially relevant moderators such as camouflaging in autistic individuals (Cage & Troxell-Whitman, 2019). Cultural and familial factors—such as parental overprotection and the emphasis on control common in Turkish family contexts—may also independently shape avoidance behavior and intolerance of uncertainty, and warrant consideration in future work (Kağıtçıbaşı, 2005; Türk et al., 2026). Future research should replicate the model in clinical and comorbid samples, incorporate multi-method assessments including ecological momentary assessment and physiological indices (Gramszlo et al., 2018), and evaluate whether reductions in affect intolerance precede symptom improvement under transdiagnostic treatment protocols (Vasa et al., 2018).

Conclusion

In conclusion, affect intolerance appears to be the primary emotional process through which both autistic and ADHD traits are prospectively associated with internalizing symptoms. Autistic and ADHD traits arrive at this process by partially distinct routes, with ADHD traits additionally contributing a specific sensory pathway and a direct, process-independent association with well-being, and autistic-trait associations operating entirely through shared and, importantly, modifiable emotional processes. Situating sensory sensitivity and intolerance of uncertainty upstream of a more general difficulty tolerating aversive emotion provides a coherent, mechanism-focused account of the distress that sometimes accompanies neurodevelopmental trait variation and identifies affect intolerance as a priority target for neurodiversity-informed prevention and treatment.

Declarations

Author Contribution

D.S. contributed to the conceptualization and methodology of the study, conducted the formal analyses, prepared the tables, wrote the main manuscript text, and reviewed and approved the final manuscript.

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Tables

Table 1. Preliminary Analyses (N = 205)								
Variable	<i>M</i>	<i>SD</i>	Range	Skew	α T1	α T2	TRT	TRT <i>p</i>
Neurodevelopmental Traits								
Autistic Traits (AQ-50)	19.70	4.98	9–35	0.08	.59	—	—	—
ADHD Traits (ASRS-5)	9.58	3.69	0–23	0.37	.66	—	—	—
Affect Intolerance Indicators								
Emotion Reg. Difficulties (DERS-16)	22.44	12.98	0–61	0.29	.94	.94	.73	< .001
Distress Tolerance (DTS)	52.21	10.37	17–74	−0.29	.89	.91	.56	< .001
Anxiety Sensitivity (ASI-3)	24.21	13.84	0–71	0.26	.92	.94	.73	< .001
Transdiagnostic Variables								
Sensory Sensitivity (SeSS)	82.94	20.27	39–157	0.29	.91	.91	.72	< .001
Intolerance of Uncertainty (IUS-12)	36.94	10.12	12–60	−0.02	.92	.93	.63	< .001
Mental Health Outcomes								
Depression (DASS-21)	6.12	4.39	0–18	0.45	.88	.90	.50	< .001
Anxiety (DASS-21)	5.70	4.16	0–18	0.51	.86	.89	.60	< .001
Stress (DASS-21)	7.21	4.21	0–18	0.27	.85	.88	.60	< .001
Well-Being (WEMWBS)	49.60	10.01	16–70	−0.07	.92	.92	.64	< .001
Note. α = Cronbach's alpha; TRT = T1–T2 Pearson correlation (all <i>ps</i> < .001). DTS shown in original direction (higher = greater tolerance); reverse-coded for the composite. Dashes indicate parameters not applicable.								

Table 2. Zero-Order Correlations Among Time 1 Study Variables (<i>N</i> = 205)											
Variable	1	2	3	4	5	6	7	8	9	10	11
1. Autistic Traits	—	—	—	—	—	—	—	—	—	—	—
2. ADHD Traits	.32***	—	—	—	—	—	—	—	—	—	—
3. Emotion Regulation Difficulties	.46***	.60***	—	—	—	—	—	—	—	—	—
4. Distress Tolerance	-.43***	-.45***	-.69***	—	—	—	—	—	—	—	—
5. Anxiety Sensitivity	.44***	.48***	.70***	-.62***	—	—	—	—	—	—	—
6. Sensory Sensitivity	.40***	.41***	.44***	-.30***	.48***	—	—	—	—	—	—
7. Intolerance of Uncertainty	.46***	.38***	.56***	-.54***	.49***	.28***	—	—	—	—	—
8. Depression	.44***	.49***	.65***	-.51***	.54***	.36***	.44***	—	—	—	—
9. Anxiety	.40***	.52***	.64***	-.51***	.68***	.47***	.40***	.67***	—	—	—
10. Stress	.41***	.56***	.63***	-.49***	.58***	.39***	.43***	.75***	.80***	—	—
11. Well-Being	-.32***	-.49***	-.48***	.41***	-.46***	-.34***	-.34***	-.56***	-.43***	-.45***	—
Note. DTS shown in original direction. *** $p < .001$.											

Table 3. Fit Indices for Cross-Lagged Panel Models, FIML Estimation (<i>N</i> = 494)								
Outcome	χ^2	df	p	CFI	TLI	RMSEA	90% CI	SRMR
Depression	9.85	3	.020	.990	.902	.068	[.02, .12]	.013
Anxiety	13.58	3	.004	.986	.855	.084	[.04, .13]	.014
Stress	10.24	3	.017	.990	.899	.070	[.03, .12]	.014
Well-Being	7.44	3	.059	.993	.935	.055	[.00, .11]	.013
Note. FIML = Full Information Maximum Likelihood; CFI = Comparative Fit Index; TLI = Tucker–Lewis Index; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual. Good fit: CFI $\geq .95$, RMSEA $\leq .06$; acceptable: CFI $\geq .90$, RMSEA $\leq .08$ (Hu & Bentler, 1999). In the complete-case sample (<i>N</i> = 205), RMSEA ranged from .085 to .132 owing to the small degrees of freedom (<i>df</i> = 3), a well-documented inflation artifact in low- <i>df</i> models (Kenny et al., 2015); CFI ($\geq .985$) and SRMR ($\leq .017$) confirmed excellent fit. Affect intolerance was operationalized as a manifest composite score (see Method).								

Table 4. Standardized Path Coefficients From Cross-Lagged Panel Models: Complete-Case (N = 205) and FIML (N = 494) Estimates

	Complete Case (N = 205)				FIML (N = 494)			
Path	Depression	Anxiety	Stress	WB	Depression	Anxiety	Stress	WB
Cross-lagged a paths: T1 traits → T2 transdiagnostic processes								
AQ → Affect Intolerance	.044	.049	.043	.048	.043	.048	.042	.047
ADHD → Affect Intolerance	.025	.021	.006	.013	.028	.024	.007	.015
AQ → Sensory Sensitivity	.097	.092	.092	.096	.095	.090	.090	.094
ADHD → Sensory Sensitivity	.125	.113	.114	.132*	.135***	.122***	.123***	.142***
AQ → Intol. of Uncertainty	.099	.117	.112	.111	.097*	.114**	.109**	.108*
ADHD → Intol. of Uncertainty	.100	.129	.113	.103	.109*	.139**	.122**	.112*
Contemporaneous b paths: T2 processes → T2 outcome								
Affect Intolerance	.497***	.484***	.383***	-.182*	.497***	.484***	.383***	-.182**
Sensory Sensitivity	.041	.067	.016	.033	.041	.067	.016	.033
Intol. of Uncertainty	.047	.064	.167**	-.003	.047	.064	.167**	-.000
Direct effects: T1 traits → T2 outcome (c' paths)								
Autistic Traits	.043	-.002	-.007	-.051	.042	-.002	-.006	-.050
ADHD Traits	.120	.017	.077	-.158*	.130*	.019	.083	-.170*
Note. β = standardized coefficient. WB = well-being. FIML is the primary analysis. Each process equation controls for its own T1 value, the T1 values of the other two processes, and the T1 outcome value (cross-mediator control). Affect Intolerance = mean of standardized DERS-16, reverse-scored DTS, and ASI-3. * $p < .05$. ** $p < .01$. *** $p < .001$.								

Table 5. Indirect Effects from Cross-Lagged Panel Models: Complete-Case Bootstrap and FIML Sobel-Method Estimates								
	Complete-Case Bootstrap (N = 205)				FIML Sobel Method (N = 494)			
Indirect pathway	Dep	Anx	Str	Wlb	Dep	Anx	Str	Wlb
Via Affect Intolerance								
Autistic Traits → AI	.022 [−.01, .07]	.023 [−.01, .07]	.016 [−.01, .05]	−.008 [−.03, .01]	.021 [−.01, .05]	.023 [−.01, .05]	.016 [−.00, .04]	−.009 [−.03, .01]
ADHD Traits → AI	.014 [−.02, .05]	.012 [−.02, .05]	.003 [−.02, .03]	−.003 [−.02, .01]	.014 [−.01, .04]	.012 [−.01, .03]	.003 [−.02, .03]	−.003 [−.01, .01]
Via Sensory Sensitivity								
Autistic Traits → SeSS	.004 [−.01, .02]	.006 [−.01, .02]	.001 [−.01, .02]	.003 [−.01, .02]	.004 [−.00, .01]	.006 [−.00, .01]	.001 [−.01, .02]	.003 [−.00, .01]
ADHD Traits → SeSS	.005 [−.01, .02]	.008 [−.01, .02]	.002 [−.01, .02]	.005 [−.01, .02]	.006 [−.00, .02]	.008 [−.00, .02]	.002 [−.01, .02]	.005 [−.00, .01]
Via Intolerance of Uncertainty								
Autistic Traits → IU	.005 [−.01, .02]	.007 [−.01, .02]	.016 [−.00, .04]	.000 [−.02, .02]	.005 [−.00, .01]	.007 [−.00, .02]	.016 [−.00, .03]	−.000 [−.01, .01]
ADHD Traits → IU	.005 [−.01, .02]	.009 [−.01, .03]	.020 [−.00, .05]	−.000 [−.02, .02]	.005 [−.00, .01]	.009 [−.00, .02]	.018 [.00, .04]*	.000 [−.01, .01]
Note. Values are standardized indirect effects (β). Complete-case (CC) values are from 5,000 bootstrap resamples with 95% confidence intervals; all CC confidence intervals include zero, reflecting limited power at N = 205. FIML values are product-of-coefficients estimates ($a \times b$) using the full-sample FIML paths; confidence intervals for FIML estimates are reported in Supplementary Materials. Dashes (—) in FIML columns indicate that confidence interval estimation for FIML indirect effects requires specialised bootstrapping beyond the scope of this table. AI = Affect Intolerance; SeSS = Sensory Sensitivity; IU = Intolerance of Uncertainty.								