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Repetitive Negative Thinking As a Transdiagnostic Prospective Predictor of Depression and Anxiety Symptoms in Neurodiverse First-Semester College Students

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Abstract

Background: Improving the understanding and treatment of mental health concerns, including depression and anxiety, are significant priorities for autistic adults. While several theories have been proposed to explain the high prevalence of internalizing symptoms in autistic populations, little longitudinal research has been done to investigate potential causal mechanisms. Additional research is needed to explore how proposed contributors to depression from general population research predict and/or moderate the development of internalizing symptoms in autistic individuals. In this study, we investigated the relation of one established risk factor, repetitive negative thinking (RNT), to internalizing symptoms over the course of college students' first semester, additionally examining whether this association is moderated by a measure of autistic traits.

Methods: Students were recruited from 4 northeastern U.S. universities: 144 participating students included 97 nonautistic students and 47 participants who either reported a formal autism diagnosis ($n = 15$) *o*r endorsed a history of self and/or others thinking that they may be autistic ($n = 32$). Participants completed a baseline survey battery within their first 2 weeks of starting college, a brief biweekly survey throughout their first semester (up to 24 times across 12 weeks), and an endpoint packet.

Results: Elevated trait-like RNT at baseline was prospectively associated with biweekly ratings of depression and anxiety symptoms across the semester. In addition, greater RNT was synchronously related to elevated sadness, anhedonia, and anxiety throughout the semester. Contrary to hypotheses, a shorter term predictive relationship between RNT at one timepoint and mood symptoms at the next was largely unsupported. While these patterns were observed across neurotypes, students with higher self-reported autistic traits were more likely to experience RNT, as well as depressive and anxiety symptoms.

Conclusions: These preliminary findings highlight RNT as a specific mechanism that may be a useful prevention and/or intervention target toward reducing the elevated depression and anxiety rates in the autistic community.

Keywords: autism, depression, anxiety, college transition, college mental health

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Community Brief

Why was this study done?

Many autistic people have depression and anxiety. However, we know very little about why autistic people are more likely to have these mental health concerns than people who are not autistic. We also do not know what leads to these symptoms over time. One theory is that repetitive negative thinking (RNT; or thinking repeatedly about problems and worries) might cause depression and anxiety. Autistic people might do more RNT than nonautistic people.

What was the purpose of this study?

In this study, we wanted to see how RNT might relate to depression and anxiety over the first semester of college. We looked at how this might be related to autistic traits.

What did the researchers do?

The researchers gave surveys to 144 students about their experiences with depression, anxiety, and RNT. The participants answered these surveys at the beginning and end of their first semester at their university. They also completed a brief survey 24 times (twice per week for 12 weeks) during the semester.

What were the results of the study?

We found that overall RNT levels at the beginning of the semester were related to sadness, anhedonia (lack of pleasure), and anxiety later. In-the-moment RNT reported on the twice-weekly survey was also related to sadness, anhedonia, and anxiety. However, RNT on biweekly surveys did not seem to predict mood symptoms a few days later. Students with higher levels of autistic traits tended to report more depression and anxiety, as well as more RNT.

What do these findings add to what was already known?

This study helps us to understand that RNT might be related to depression and anxiety, regardless of whether or not someone is autistic. This might mean that reducing RNT could help prevent or treat depression and anxiety, especially in autistic adults.

What are potential weaknesses in the study?

Our study had a low number of participants with formal autism diagnoses (15 people), so it might not represent the broader population of autistic adults with formal diagnoses as well as we would like. Nevertheless, we had a larger group with self-reported or suspected autism (32 people).

How will these findings help autistic adults now or in the future?

These findings help us to better understand risk factors for depression and anxiety in autistic adults. Since RNT was related to depression and anxiety in the same way regardless of levels of autistic traits in our study, we hope that clinicians will feel more comfortable providing therapy to people with mood disorders, regardless of whether they are autistic and/or have high autistic traits. This could be a small step toward increasing equity and accessibility of mental health services for autistic adults.

Background

In the autistic community, depression and anxiety are prominent concerns, with detrimental effects on well-being. Meta-analyses have estimated that the prevalence of depression in autistic adults is approximately three to four times higher than in nonautistic adults.[1] These high rates of depression have been associated with distress and lower quality of life,[2,3] greater service use,[4] lost work days,[3] and self-injury and suicidality in the autistic community.[5,6] Autistic adults are also at an increased risk of anxiety disorders, with a lifetime prevalence estimated to be approximately 42%.[7]

Although anxiety is more weakly associated with reduced quality of life than depression,[3] anxiety is related to difficulty coping with change[8] and increased physical health concerns in autistic people.[9–11] In addition, the development of both depression and anxiety is highly intertwined and, therefore, may affect one another.[12] Given the magnitude of health and quality-of-life concerns associated with depression and anxiety, it is perhaps unsurprising that autistic adults and other stakeholders frequently identify better understanding and treatment of these conditions as top clinical and research priorities.[13–16]

While this call for mental health prioritization has come from stakeholders across the lifespan,[15] autistic young adults,

including college students, may face unique challenges that put them at heightened risk for difficulties with mental health.[17,18] An increasing number of students diagnosed as autistic have entered college[19] and have reported salient mental health concerns.[16]

Autistic adults have described ''feeling overwhelmed, stressed, anxious, depressed, tired and isolated'' as university students[16(p19)] and recent larger scale comparisons consistently document heightened mental health issues among autistic students, relative to nonautistic university students.[20–22] Given that autistic students' ability to access and benefit from mental health resources varies greatly,[14,18] further research is needed to learn how to best support autistic university students, including by preventing and treating mental health concerns.

Existing research suggests that autistic students have greater difficulty transitioning to college than their nonautistic peers.[20,23–25] Given that lower educational status is associated with increased emotional and physical distress later in life, the consequences of difficulties transitioning into college may be long lasting[24,25] and exacerbate the challenges facing the already underserved community of autistic adults. Therefore, work is needed to understand and eventually address mental health issues that may prevent autistic students from successfully transitioning into college.

By focusing on factors that contribute to mental health concerns among autistic and nonautistic students who are transitioning into college, we may be able to develop supports to help them navigate this transition more successfully. Importantly, this work cannot exclusively focus on individuals who have had access to formal autism assessments and diagnoses, particularly in the context of a system wherein access to appropriate evaluations is unequally distributed.[26,27]

Therefore, adult autism research may be more inclusive and beneficial to the full autistic community if there is greater opportunity for understanding the experiences of not only those formally diagnosed in childhood, but also young adults who have self-diagnosed and/or are still exploring their autistic identity. In line with this goal of greater inclusion of people with a variety of diagnostic histories, this study does not require a formal diagnosis and instead explores the effects of autistic traits dimensionally, rather than categorically.

The role of repetitive negative thinking in depression and anxiety in autistic adults

In the general population, repetitive negative thinking (RNT)—thinking excessively and recurrently about concerns, past experiences, and/or future worries[28]—has been shown to be associated with depression and anxiety.[29–31] In particular, much of the prior literature has emphasized the role of rumination, a type of RNT that focuses on one's distress without making active efforts to solve the problem.[32] Rumination is related to worry and other forms of repetitive thought, but is distinct particularly due to its lack of problem-solving orientation and its specific focus on stressors or contributors to low mood.[33]

These contributors are often in the past, thus there is little opportunity to productively plan and change outcomes (unlike with worry, which is often anticipatory). Prior literature has viewed rumination transdiagnostically[33] across a variety of temporal relationships—such that rumination has been conceptualized as including both a longer term more stable cognitive tendency that may be learned early in life through parenting and other environmental influences,[33–35] and shorter term moments of heightened repetitive cognition that may arise in response to stressors,[36] with both likely playing a role in functioning.

Across these conceptualizations, a large body of evidence suggests that rumination both predicts the onset of and maintains depression and anxiety symptoms in the general population.[32,35,37,38] Furthermore, rumination is related to numerous other negative outcomes such as physical health concerns,[39,40] suicidality,[41] and reduced response to therapeutic interventions.[33]

There is reason to believe that repetitive thought processes may be even more common in autistic people[42,43]—and similarly related to internalizing symptoms in autistic populations as in the general population.[42,44] There are many potential causal explanations for this elevated rate of repetitive thought within autistic people, although there is not clear consensus on whether any one theory can fully explain the phenomena. Some reports have suggested that there may be differences in activation at a neural level that contribute to greater instances of cognitive inflexibility within autism.[45] Cognitive inflexibility describes difficulties in executive functioning, in which one may struggle to disengage from and switch tasks or thought processes.[46–49]

This tendency is more common in autistic people[45,50,51] and, similar to rumination, heightened cognitive inflexibility has been associated with greater depressive and anxiety symptoms in autistic youth[46,48] (although potentially indirectly through intolerance of uncertainty).[49] This connects well with general population literature that identifies executive dysfunction as a key contributing factor toward a ruminative response style.[33]

Another explanation may be that autism is defined diagnostically by repetitive behavior (such as in speech, interests, and other manifest behavior),[52] and thus these behavioral and cognitive domains of repetition may be interrelated. For example, insistence on sameness is likely to involve both a more rigid cognitive style and related routine-protecting behaviors.[53] In this proposed view, a more repetitive, inflexible, or detail-focused cognitive style may underlie both repetitive thinking in autistic individuals and many of the observable repetitive behaviors associated with autism.[44,46,47]

Finally, it is also possible that greater reported repetitive thinking in autistic adults is related to the elevated rates of stressors that autistic people face. Marginalized individuals across a variety of identities appear to engage in more ruminative processes, as a maladaptive method of coping with their increased rates of stressors.[54–57] Ableism and other forms of oppression related to intersectional autistic identities may increase autistic adults' risk of rumination.

Regardless of the cause(s), autistic individuals appear to engage in more repetitive thinking than their nonautistic peers. While much of the repetition associated with autism may not be focused on negative information, repetitive thinking may be a cognitive characteristic of autism that, when focused on negative events or beliefs, contributes to depression through means similar to rumination.[42,44]

In addition to engaging in more repetitive cognitive patterns overall, cross-sectional evidence suggests that autistic

adults engage in more RNT than their nonautistic peers,[42,43] and that this elevated RNT is related to depression symptom endorsement in both autistic adults and adults with high levels of autistic traits.[58,59] Importantly, only one longitudinal study has investigated potential links between rumination and depression in autism, finding that rumination prospectively predicts depression scores in autistic children.[60]

To our knowledge, previous research has not evaluated this longitudinal link in an autistic adult sample. Furthermore, research has underexplored the association between RNT and anxiety in autistic people. Anxiety and depression are often co-occurring,[61] contributing to the theorization of similar cognitive mechanisms for both internalizing problems.[62] However, to our knowledge, no longitudinal studies have evaluated the relationship between RNT and anxiety in autistic populations.

Objectives

In this study, we aimed to evaluate how RNT may predict and/or maintain depression and anxiety symptoms in first-semester undergraduate students. We compared potential associations across levels of autistic traits, to see whether the strength of RNT's contribution to depression and anxiety symptoms varied by potential autism status. By understanding RNT's contribution to the development and/or maintenance of internalizing symptoms, we may be able to better tailor mental health interventions for greater effectiveness or find more precise ''points of entry'' through which to intervene.

In line with these goals and previous research, the following *a priori* hypotheses were formed:

1. Incoming college students who reported greater RNT at baseline and throughout the semester would report increased depression (sadness and anhedonia) and anxiety symptoms over the course of their first semester compared with those with lower RNT.
2. We hypothesized no difference in the model of this mechanism as a function of autistic traits (i.e., a lack of moderation by levels of autistic traits).* However, we anticipated that students who reported higher autistic traits would exhibit greater repetitive thinking scores, as well as greater depression and anxiety scores, compared with those who reported lower autistic traits.

*When forming *a priori* hypotheses, we decided that we would use SRS-2 scores as a dimensional representation of autistic traits, instead of relying on self-report of autism, if the majority of our autistic participants did not report a formal autism diagnosis. Therefore, this hypothesis was written in two different ways initially: first, we hypothesized that students in our self-described autistic group would report high prevalence of RNT, depression, and anxiety, and, second, we also hypothesized that students with higher autistic traits would report heightened RNT, depression, and anxiety.

In both cases, we assumed that autism/autistic traits would not moderate how RNT affected depression/anxiety. For parsimony, we focus on the second iteration of this hypothesis in this article due to our sample characteristics. Both hypotheses were formed *a priori*.

Methods

We recruited incoming undergraduate students from four similarly sized public universities in the northeastern United States (Rowan University, Montclair State University, Stony Brook University, and College of Staten Island) to participate in this fully online study. After confirming eligibility, participants completed a baseline questionnaire battery about their diagnostic and mental health history, a brief survey twice per week throughout their first semester on campus, and then an endpoint battery.

Participants

We collected the data presented in two waves—first from a pilot group of Rowan University students in Fall 2020 ($n = 41$ at baseline), and then from a larger group from all four universities in Fall 2021 ($n = 103$ at baseline). Somewhat contrary to our expectations given the heightened COVID-19 precautions and potentially reduced social opportunities in Fall 2021, analyses found no significant historical effects when comparing these groups: primary outcome scores of interest did not differ significantly between the 2 years, for example, there was no significant difference in baseline Beck Depression Inventory-II[63] [BDI-II; baseline: $F(1, 138) = 0.18$, $p = 0.67$, $r = 0.04$] or Generalized Anxiety Disorder 7-item scale (GAD-7)[64] [GAD-7; baseline: $F(1, 137) = 0.53$, $p = 0.47$, $r = 0.06$] scores between the two waves (further details and data provided upon request). Therefore, we collapsed data across the two data collection waves for further analyses.

For both waves of data collection, we recruited autistic and nonautistic students during the summer before their first semester at their respective universities. At each university, we recruited participants through emails to incoming first-year student listservs, flyers posted in common gathering spaces, and invitations sent through institutional offices of disability services.

Eligible participants were undergraduate students, 18 years or older, and in their first semester at each respective campus (experience at a different university was permitted). Exclusion criteria included self-report of current concerns of psychosis, bipolar disorder, or significant substance-use disorders, as these may obscure comparisons across our cohorts of interest by confounding influences on mood. We first enrolled all eligible autistic students—this included both those who self-reported a formal autism diagnosis and those who indicated an affirmative response to the screener survey question, ''Have you or others around you ever suspected that you were autistic/had an autism spectrum disorder?''

By including these participants, we intended to better represent historically under-represented groups of autistic adults who have lower access to formal diagnoses,[26,27] however, primary analyses of the effects of autism traits relied on Social Responsiveness Scale, second edition[65] (SRS-2) T-scores, not this self-assigned autism status (see Statistical Analyses section). Then, we matched nonautistic students as closely as possible on university, age, gender, and race/ethnicity, through a one-to-one match, prioritizing approximate age in years and the university they are attending (matching 18-year-old autistic Rowan University students to 18-year-old nonautistic Rowan University students, etc.), when exact matches were not possible.

Given the increased prevalence of depression in autistic individuals, we chose to oversample nonautistic participants to increase the likelihood of having a comparable number of students within each diagnostic cohort who would go on to develop clinically significant symptoms of depression during the semester. When enrolling these additional students, we oversampled nonautistic students who were older than 18 years, nonbinary, and/or transgender to better match the demographic characteristics of the autistic group.[23,66] Otherwise, these students were enrolled in order of screener survey response date.

These procedures resulted in an overall sample of $N = 144$ participants who completed the baseline survey packet: 47 autistic participants ($n = 15$ reporting formal diagnoses) and 97 nonautistic participants (Table 1). Given that we anticipated recruiting a relatively low number of participants with a formal autism diagnosis—and explicitly wanted to also represent those without access to a formal diagnosis—we used the dimensional variable of SRS-2 scores instead of diagnostic categories for analyses of the effect of autism status.

Of note, however, we did expect and observe that the autistic cohort had significantly higher SRS-2 scores ($M = 63.33$, standard deviation [SD] = 10.00) than the nonautistic group ($M = 53.43$, SD = 8.98) as expected (Cohen's $d = 1.06$, 95% confidence interval, CI [0.67 to 1.41]), despite significant variability in SRS-2 T-scores within both groups (Supplementary Fig. S1). High distributional overlap in SRS-2 scores is evident between those reporting professional autism diagnoses and those who indicated that they/others have thought they might be autistic but have not received a formal diagnosis.

The probability of superiority statistic (A[67]) showed that a randomly selected individual from the diagnosed group would have a higher total SRS score than a randomly selected individual from the suspected autism group only about half the time ($A = 0.51$, 95% CI [0.32 to 0.66]). Comparatively, there is low distributional overlap between both of these categories and our nonautistic group, with a randomly selected student with suspected or diagnosed autism having higher SRS scores than a randomly selected nonautistic student nearly three-fourth of the time ($A = 0.73$, 95% CI [0.63 to 0.80]).

Participation at each biweekly survey timepoint ranged from 54 to 97 students, with an average of ~ 80 respondents per timepoint (SD = 13.44).[†] A total of 96 participants then completed the endpoint survey.

[†]This variability is largely due to postbaseline attrition and an unexpected change to one major phone carrier's spam filter mid-study in Fall 2021, which resulted in 27 participants missing a portion of the biweekly survey alerts (5–7 survey points from November 7 to December 1, 2021). We were unaware of the spam filter change, and thus the reason for this sudden attrition, until after the conclusion of data collection.

However, there were no identifiable significant differences on baseline variables of interest (i.e., Social Responsiveness Scale, second edition, Beck Depression Inventory, General Anxiety Disorder-7, and laboratory-made repetitive thinking battery) between those who completed both the baseline packet and biweekly survey and those who only completed the baseline packet. Similarly, differences did not seem to exist by phone carrier: the relationship between phone carrier and each measure of interest revealed correlations approaching 0, $p > 0.90$ in all cases. This suggests that attrition bias is of relatively minimal concern.

Procedures

Interested students first completed a brief eligibility screener, where they noted the university they attend, their age, year in school, gender, and relevant diagnostic history. We used this survey to match participant groups and ensure all participants met eligibility criteria, as previously described (Table 1). Participants deemed eligible then gained access to the online baseline ''packet'' 1 week before the start of their semester and had approximately 3 weeks to complete it.

Since the timeline was based around each university's semester calendar, the initial start date of survey participation differed by 1 week between the New York and New Jersey universities. The baseline packet took approximately 45 minutes to complete, and participants had the option to complete the packet all in one session or to complete a portion and then return to it at a later time.

After completion of the baseline packet, participants received a link to a single brief survey through text every Sunday and Wednesday evening throughout the semester. This biweekly survey consisted of 12 questions and took approximately 2 minutes to complete on a smartphone (see Measures section for more details). At the conclusion of the semester (13–15 weeks after baseline), participants completed a final battery of surveys, similar to baseline. Participants received up to a total of $75 in emailed Visa gift codes for completing all study procedures.

We received approval for this study and measures from the Rowan University School of Osteopathic Medicine Institutional Review Board, which was the IRB of record for most recruitment sites (Study ID Pro2020001172), as well as Stony Brook University (IRB2021-00266). All participants were age 18 years or older and electronically provided their own informed consent.

Measures

Participants completed all measures online through Research Electronic Data Capture (REDCap), a survey and data management platform developed specifically for use in electronic acquisition and storage of sensitive data.[68,69]

Baseline. The initial survey battery, completed by participants within 2 weeks of the start of their semester, collected baseline data about key constructs related to emotional, social, behavioral, and physical health, as well as demographics. We selected measures based on their usability in both the autistic and general population.

Measures relevant for the current analyses included the SRS-2[65] to assess autistic traits, BDI-II[63,70,71] to assess recent depression symptoms, and GAD-7[64,72–74] to measure recent anxiety symptoms. Internal consistency for all measures was strong: SRS-2 Cronbach $\alpha = 0.97$, BDI-II Cronbach $\alpha = 0.92$, GAD-7 Cronbach $\alpha = 0.91$. We also collected information on participants' repetitive thinking; this was primarily accomplished through a novel repetitive thinking questionnaire.

The senior author's research team created this novel measure to assess general tendencies toward transdiagnostic RNT through several years of federally funded research on repetitive thinking in autistic adults. This measure development occurred in several stages, including community engagement studios, cognitive interviews, collecting data on the extended

Table 1. Baseline Demographics and Comparisons of Autistic and Nonautistic Participants

	Entire sample, N = *144*	*Nonautistic,* n = *97*	*Autistic (clin dx or self ID),* n = *47*	*Group differences*
Age, years, mean (SD)	19.50 (3.79)	19.23 (3.15)	20.05 (4.84)	$F(1, 147) = 1.55$, $p = 0.22$, $d = 0.22$
Range	18–43	18–39	18–43	
Gender (% women/nonbinary or other)	45/11%	47/9%	43/16%	χ^2 (3, $N = 149$) = 2.81, $p = 0.42$
Race/ethnicity, %				
Native American	3	2	6	n.s.[a], $p > 0.05$
Asian	20	22	14	
Black	15	19	8	
White	60	57	67	
Hispanic	20	24	25	
Least educated parent, %				
HS or less	35	36	34	χ^2 (7, $N = 144$) = 3.49, $p = 0.84$
Some college	17	17	18	
Associate degree	8	8	6	
Bachelor's degree	29	27	33	
Graduate degree	9	10	6	
SRS-2 T-scores, mean (SD)	56.71 (10.40)	53.43 (8.98)	63.33 (10.00)	$F(1, 134) = 33.98$, $p < 0.001$**, $d = 1.06$
Range	39–87	39–80	45–87	
BDI-II, mean (SD)	11.77 (9.92)	10.76 (9.54)	13.85 (10.44)	$F(1, 138) = 3.05$, $p = 0.08$, $d = 0.31$
Range	0–46	0–39	0–46	
GAD-7, mean (SD)	5.81 (5.18)	5.14 (5.06)	7.22 (5.19)	$F(1, 137) = 5.08$, $p = 0.03$*, $d = 0.41$
Range	0–21	0–19	0–21	
RepT, mean (SD)	23.45 (8.27)	21.57 (8.56)	27.22 (6.19)	$F(1, 133) = 15.55$, $p < 0.001$**, $d = 0.72$
Range	8–39	8–39	8–37	

The table shows the demographics of the full sample, as well as self-defined autistic and nonautistic cohorts (described further in Methods section). However, most analyses were done dimensionally, using the entire sample, with SRS-2 scores as moderator.

[a]Categories of race/ethnicity were not mutually exclusive, such that some participants selected multiple categories. As assessed by chi-square and asymptotic significance, any differences in percentages of race/ethnicity between cohorts were not significant at the $p < 0.05$ level.

*$p < 0.05$, **$p < 0.001$.

BDI-II, Beck Depression Inventory, second edition; GAD-7, Generalized Anxiety Disorder 7-item scale; RepT, repetitive thinking; SD, standard deviation; SRS-2, Social Responsiveness Scale, second edition.

version of this measure from 589 participants crowdsourced through Amazon MTurk, iteratively analyzing these data using Item Response Theory to identify best-performing items within empirically derived factors, and then further analyzing performance of the refined measure in a sample of 762 autistic adults from the SPARK national autism registry.[75]

Based on these data, we selected eight items that best represented negative repetitive thinking out of the greater repetitive thinking item pool to reduce the time burden on participants. All items are rated on a 5-point Likert scale (1 = almost never to 5 = almost always) and were best-performing items drawn from the Perseverative Thinking Questionnaire,[76] Rumination and Reflection Questionnaire Negative Events,[77] and Measure of Mental Anticipatory Processes.[78]

The resulting measure assesses general tendencies toward transdiagnostic RNT through eight self-report items, answered through 1–5 point Likert scale. For example, participants rated their agreement with items, such as ''When I have a difficult experience or problem, the same thoughts keep going through my mind.'' Possible total scores range from 8 to 40, with lower scores reflecting lower RNT.

These eight items do not represent our laboratory's final iteration of the repetitive thinking instrument, but nevertheless showed excellent internal consistency in the current sample, Cronbach $\alpha = 0.93$, strong internal consistency in the large SPARK sample already described (Cronbach $\alpha = 0.86$),[75] and correlated with expected measures such as the Penn State Worry Questionnaire,[79] $r(723) = 0.65$, $p < 0.001$, and the Ruminative Response Scale[32] total score, $r(721) = 0.64$, $p < 0.001$, and Brooding subscale [$r(721) = 0.64$, $p < 0.001$].

Brief biweekly survey. Next, participants completed the brief 12-question survey twice per week for 12 weeks (approximately equivalent to the remainder of the semester after the 2- week ''baseline'' period) to track changes in key constructs (e.g., sadness, anhedonia, anxiety, RNT) throughout each participant's semester. Developed by the senior author's laboratory, this brief survey prompted participants to provide information about their mood, thinking patterns, and social situation over the ''last few days'' (i.e., since the previous biweekly survey) through 5-point Likert scales, balancing content coverage and parsimony.

On these scales, 1 is ''almost never'' and 5 is ''almost always.'' The 1–5 Likert scale responses to the question ''In the last few days, how often have you been brooding, or thinking repetitively, about problems or negative experiences?'' were used to reflect RNT. For depressive symptoms, 1–5 Likert scale responses to ''How often have you been feeling down, sad, or empty?'' measured sadness and ''How often have you been feeling low interest or little pleasure in

doing things that you usually enjoy?'' measured anhedonia. Finally, responses to ''How often have you been feeling nervous, anxious, or on edge?'' represented anxiety.

Although sadness and anhedonia are both symptoms of depression,[52] existing evidence suggests that they are better conceptualized as related by distinct processes that may directly influence each other rather than reflective indicators of an underlying unidimensional process (''depression'').[80] Therefore, we considered both constructs separately in analyses. See Supplementary Data section for the full biweekly survey.

Endpoint. Finally, participants completed an end-of-semester online survey battery. Most measures in this battery were the same as baseline, but the endpoint was slightly shorter for feasibility and retention. We chose not to repeat demographic and diagnostic history questions, and we also did not repeat the SRS-2, as these scores were expected to be more trait-like and not change significantly over the course of a semester.[81]

Statistical analyses

Primary analyses. For all primary analyses, we used generalized least squares (GLS) regression[82] using the *rms*[83] and *nlme*[84] packages in R. GLS accounts for the fact that repeated measurements from the same individual are typically correlated. A first-order autoregressive correlation structure (AR_1) was used to capture temporal (within-person) dependence in the errors. The correlation between outcome measurements (Y) at adjacent timepoints (t) is estimated and assumed to be constant over time (stationarity). The correlation among repeated Y measurements is assumed to diminish exponentially according to the number of time units (weeks) separating the measurements.

In sum, the GLS AR_1 model assumes measurements from the same individual are correlated, and the strength of the correlation diminishes rapidly with increasing distance between the measurements. Mixed-effects modeling is an alternative strategy that provides both individual and marginal effects. However, we were primarily concerned with marginal effects rather than individual deviations around average effects; therefore, we chose to avoid the added assumptions required when including random effects parameters.[85,86] Effect sizes were calculated as the ''proportion of variance explained by the given effect relative to the proportion of outcome variance unexplained.''[87(p5)] This is notated as f^2 and interpreted as follows: 0.02 is considered a small effect, 0.15 is considered medium, and ≥ 0.35 is large.[88]

Outcome trajectories were permitted to take a flexible nonlinear shape by modeling the effect of time (in weeks) using a restricted cubic spline[89] with three knots placed at the 0.10, 0.50, and 0.90 quantiles. Restricted cubic splines require $k-1$ degrees of freedom (df), where k is the number of knots,[90] so the time effects in our models required $df=2$.

We assessed the relationship between RNT and each outcome of interest (sadness, anhedonia, and anxiety) in three ways. First, we assessed the relationship between baseline repetitive thinking levels (measured through the laboratory-made repetitive thinking battery, as previously described), and biweekly reports of the outcomes of interest (sadness, anhedonia, and anxiety) across the semester. We then evaluated the relationship between biweekly reports of RNT and internalizing symptoms in terms of a temporal predictive relationship and synchronously.

To explore the shorter term prospective association between RNT and each outcome, we created a lagged variable such that the outcome of interest was predicted by RNT at the prior timepoint (e.g., sadness at time t was regressed on RNT at time $t-1$). Finally, we evaluated the synchronous association between RNT and each outcome within one timepoint, using the nonlagged biweekly report of RNT.

SRS-2 scores were held constant in each of these primary analyses, to limit their potentially confounding effects as a contributor to both RNT and mood symptoms. Time (week of survey) was also included in the model to limit the influence of an underlying symptom trajectory. This allows us to better evaluate the effect of RNT on mood symptoms above and beyond that which may be due to a general trajectory of mood over time.

Differences by autistic trait status. Given the variability and relatively low sample size of formally diagnosed autistic participants, we used SRS-2 T-scores to evaluate how autistic traits, measured dimensionally, related to RNT, depression, and anxiety, rather than comparing groups based on self-reported autism status. We anticipated that participants with greater endorsed autistic traits would report higher sadness, anhedonia, anxiety, and RNT, compared with participants who reported fewer autistic traits. To assess this, we evaluated the cross-sectional relationship between baseline measures of SRS-2 scores and baseline mental health measures, BDI-II and GAD-7, in general linear models.

After this, we evaluated the relationship between SRS-2 scores and biweekly reports of RNT, sadness, anhedonia, and anxiety using GLS regression. Time was held constant in these models. Exploratory analyses were also conducted to evaluate relationships between dichotomous autism groups and level of internalizing symptoms.

Results

RNT and sadness

Overall baseline RNT was prospectively associated with later sadness endorsement across the semester's biweekly survey: A 1-unit increase in baseline RNT was associated with a 0.042 units increase [95% CI 0.03 to 0.05] in subsequent sadness levels, holding constant time and SRS-2 scores (see Table 2 for results from all primary analyses). Furthermore, the addition of RNT improved the ability of the model to predict sadness scores: The addition of RNT in the model of the relationship between sadness and time resulted in a 0.08 increase in the value of R^2 (from 0.25 to 0.33, while still holding SRS-2 T-scores constant).

The relationship between baseline repetitive thinking and sadness is represented in Figure 1. In this figure, tertiles of baseline RNT are depicted in separate panels, such that the relationship between week of survey participation and self-reported sadness is displayed, as it depends on baseline level of RNT (panels).

When testing the lagged relationship between repetitive thinking and sadness (i.e., repetitive thinking from the previous biweekly survey as a predictor of subsequent timepoint sadness), prior timepoint RNT did not significantly predict subsequent timepoint sadness when controlling for SRS-2 T-scores and time ($b=-0.004$, 95% CI [–0.03 to 0.02], $f^2=-0.0005$). However, synchronous sadness and RNT (i.e., measured at the same

TABLE 2. RELATIONSHIP BETWEEN HYPOTHESIZED PREDICTORS AND BIWEEKLY OUTCOMES OF INTEREST

Hypothesized predictor	*Outcome*	*Estimate [95% CI]*	*Estimated standard error*	t (*df*)	*Pr(≥|t|)*	f^2
SRS-2 scores	RNT	0.045 [0.03 to 0.06]	0.005	8.14 (5, 1804)	<0.001***	0.17
SRS-2 scores	Sadness	0.052 [0.04 to 0.06]	0.005	10.54 (5, 1804)	<0.001***	0.28
SRS-2 scores	Anhedonia	0.053 [0.04 to 0.06]	0.005	11.08 (2, 1804)	<0.001***	0.31
SRS-2 scores	Anxiety	0.039 [0.03 to 0.05]	0.005	7.72 (5, 1804)	<0.001***	0.14
Baseline RNT[a]	Sadness	0.042 [0.03 to 0.05]	0.009	4.74 (3, 1895)	<0.001***	0.11
Baseline RNT[a]	Anhedonia	0.038 [0.03 to 0.05]	0.009	4.51 (3, 1895)	<0.001***	0.12
Baseline RNT[a]	Anxiety	0.043 [0.03 to 0.05]	0.009	4.87 (3, 1895)	<0.001***	0.11
Lagged RNT[a]	Sadness	−0.004 [−0.03 to 0.02]	0.01	−0.26 (6, 1803)	0.79	−0.0005
Synchronous RNT[a]	Sadness	0.361 [0.32 to 0.39]	0.02	17.95 (6, 1803)	<0.001***	0.28
Lagged RNT[a]	Anhedonia	−0.041 [−0.07 to −0.02]	0.01	−3.18 (6, 1803)	0.001*	−0.004
Synchronous RNT[a]	Anhedonia	0.283 [0.24 to 0.32]	0.02	14.33 (6, 1803)	<0.001***	0.22
Lagged RNT[a]	Anxiety	−0.002 [−0.03 to 0.03]	0.01	−0.14 (6, 1803)	0.89	−0.0002
Synchronous RNT[a]	Anxiety	0.367 [0.33 to 0.41]	0.02	17.58 (6, 1803)	<0.001***	0.27

[a]Variable was included in a model covarying SRS-2 T-scores. We also evaluated separate models covarying baseline BDI-II and GAD-7 scores (separately, each in place of SRS-2 scores). The same pattern of significance was detected; results available upon request. Effect sizes are described by f^2 and interpreted as follows: 0.02 is considered a small effect, 0.15 is considered medium, and ≥0.35 is large.

CI, confidence interval; *df*, degrees of freedom; RNT, repetitive negative thinking.

timepoint) were significantly related to one another, such that a 1-unit increase in repetitive thinking was associated with a 0.361 unit increase in sadness (95% CI [0.32 to 0.39], $f^2 = 0.28$). The predictive strength of this relationship is shown in Figure 2.

RNT and anhedonia

We also assessed the relationship between RNT and anhedonia, with similar results: overall repetitive thinking tendencies measured at baseline were significantly related to anhedonia across the semester ($b = 0.038$, 95% CI [0.03 to 0.05], $f^2 = 0.12$), when controlling for SRS-2 T-scores and time. Lagged RNT was related to anhedonia endorsement ($b = -0.041$, 95% CI [−0.07 to −0.02]), but the effect size was negligible ($f^2 = -0.004$). However, there was a significant relationship between synchronous anhedonia and RNT ($b = 0.283$, 95% CI [0.24 to 0.32], $f^2 = 0.22$).

RNT and anxiety

Finally, we investigated the relationship between RNT and anxiety symptoms. Again, we found that the baseline measure of RNT was significantly related to anxiety across the biweekly reports ($b = 0.043$, 95% CI [0.03 to 0.05], $f^2 = 0.11$). There was little evidence for an effect of lagged RNT on anxiety symptoms ($b = -0.002$, 95% CI [−0.03 to 0.03], $f^2 = -0.0002$), but we again observed a significant relationship between synchronous RNT and anxiety ($b = 0.367$, 95% CI [0.33 to 0.41], $f^2 = 0.27$).

Predictive utility of autism trait levels

The relationships between autistic traits and depression, anxiety, and RNT were explored. First, cross-sectional analyses showed significant effects of SRS-2 on depression,

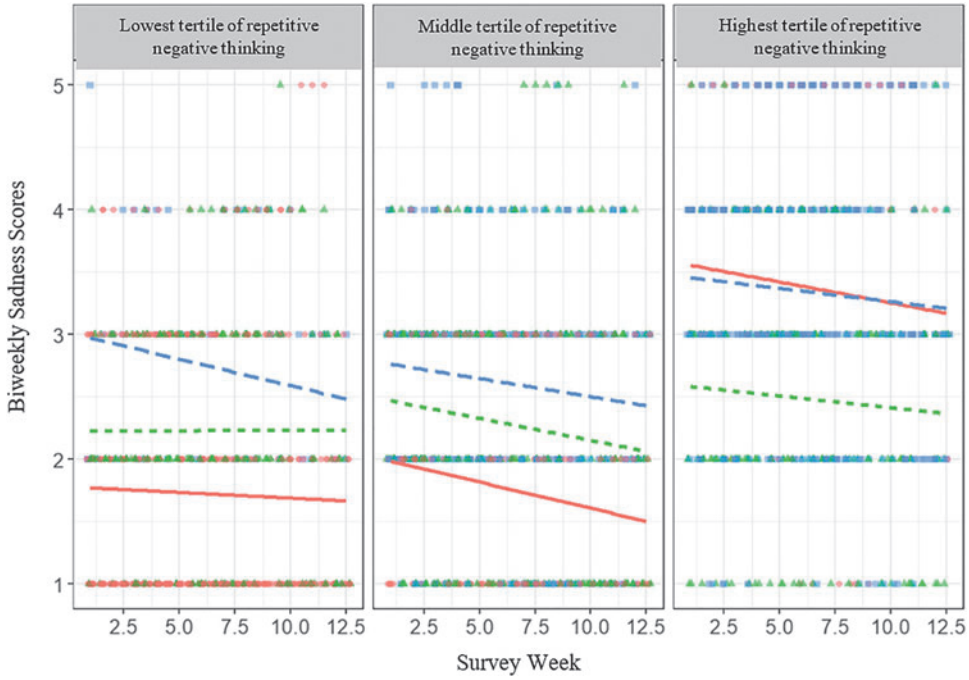

FIG. 1. Sadness scores each week by baseline RNT tertile. ■ = Participants reporting the highest SRS T-scores (61–89). ■ = Participants reporting the median level of SRS T-scores (51–61). ■ = Participants reporting the lowest SRS T-scores (39–51). RNT, repetitive negative thinking; SRS, Social Responsiveness Scale.

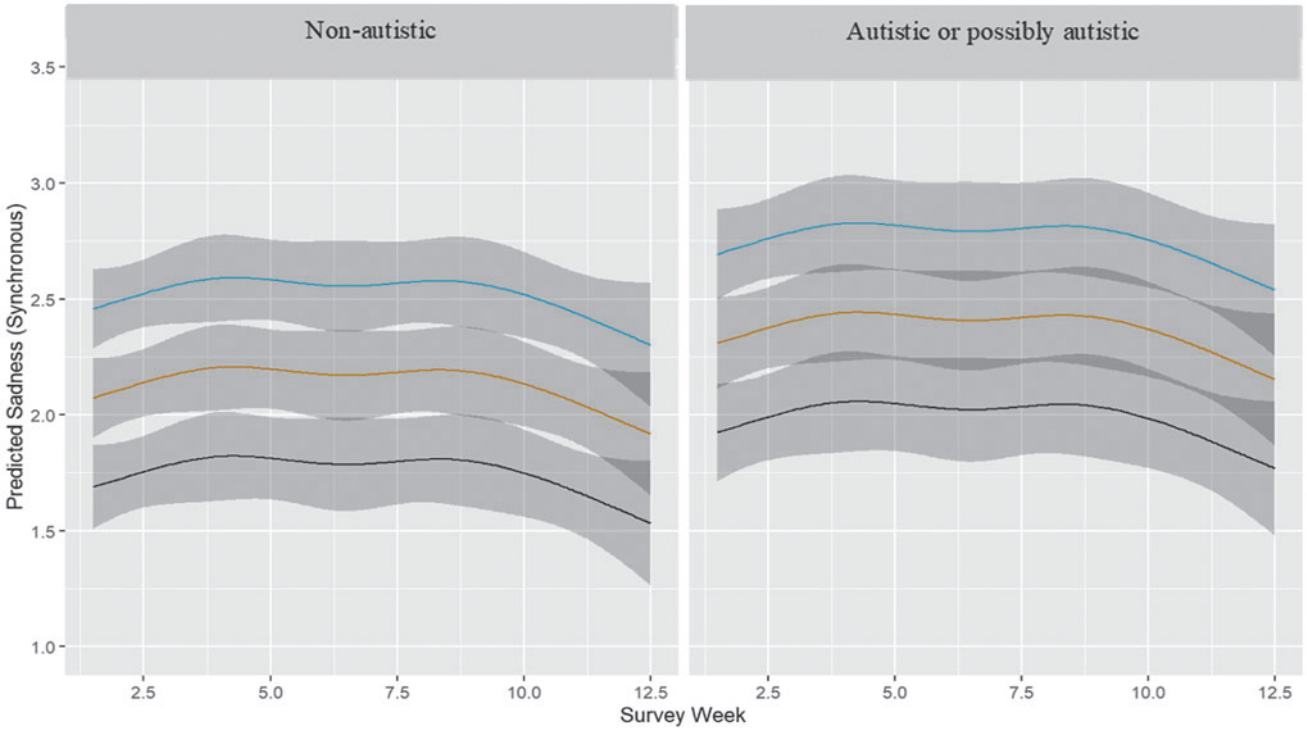

FIG. 2. Predicted relationship between sadness and RNT over time, by autism status. This figure depicts predicted biweekly reports of sadness scores over time, stratified by levels of RNT and diagnostic cohort. Predictions are based on a GLS model with a first-order autocorrelation structure, with SRS-2 T-scores held constant (at 55.0). The left panel depicts predictions for participants with no history of autism. The right panel depicts predictions for participants who are in our self-classified autism group, meaning they either have a formal diagnosis or a history of thinking or other commenting that they may be autistic. (For more information on these groups, see the first Supplementary Fig. S1.) ■ = Participants reporting the highest RNT. ■ = Participants reporting the median level of RNT. ■ = Participants reporting the lowest RNT. GLS, generalized least squares.

anxiety, and repetitive thinking at both baseline and endpoint: Participants with higher SRS-2 scores reported significantly higher depression scores at baseline [$F(1, 134) = 90.70$, $p < 0.001$, $r = 0.26$] and endpoint [$F(1, 82) = 36.15$, $p < 0.001$, $r = 0.55$]. Higher SRS-2 scores were also related to elevated anxiety symptoms at baseline [$F(1, 134) = 57.58.42$, $p < 0.001$, $r = 0.22$] and endpoint [$F(1, 82) = 23.15$, $p < 0.001$, $r = 0.47$].

Finally, participants with higher SRS-2 scores endorsed higher levels of RNT at baseline [$F(1, 133) = 102$, $p < 0.001$, $r = 0.24$] and endpoint [$F(1, 80) = 29.97$, $p < 0.001$, $r = 0.52$].[‡]

Overall, autistic traits were cross-sectionally related to all baseline and endpoint measures of depression, anxiety, and RNT.

Results from GLS regressions showed that students who reported more autistic traits at baseline endorsed significantly higher sadness ($b = 0.052$, 95% CI [0.04 to 0.06], $f^2 = 0.28$; Table 2), anhedonia ($b = 0.053$, 95% CI [0.04 to 0.06], $f^2 = 0.31$), and anxiety ($b = 0.039$, 95% CI [0.03 to 0.05], $f^2 = 0.14$) on average throughout the semester. Students with higher SRS-2 scores also endorsed more RNT ($b = 0.045$, 95% CI [0.03 to 0.06], $f^2 = 0.17$).

We evaluated whether there was evidence of an interaction between SRS-2 scores and RNT. In all cases, the models without the interaction effect were preferred over those including it, as indicated by nonsignificant likelihood ratio tests and smaller Bayesian Information Criterion values.

Discussion

Autistic adults and other stakeholders have repeatedly identified mental health and the transition to adulthood as key research and clinical priorities. While others have proposed several theories to explain the high prevalence of depression and anxiety in autistic populations, virtually no longitudinal research has evaluated potential causal mechanisms. In this study, we investigated RNT as a potential prospective predictor of internalizing symptoms over the course of the first semester of college, within a transdiagnostic neurodiverse sample.

[‡]Since SRS-2 scores could be conflated with internalizing symptoms,[91–93] we also examined how depression and anxiety were related to self-selected diagnostic cohort, nonautistic versus autistic, with the latter including participants who reported an official diagnosis as well as those who thought they may be autistic or had heard from others they may be autistic. Here, we found that participants in our autism cohort did not endorse significantly more symptoms of depression at baseline [$F(1,138) = 3.05$, $p = 0.08$, $d = 0.31$, 95% CI −0.04 to 0.66] or endpoint [$F(1, 87) = 0.99$, $p = 0.33$, $d = 0.22$, 95% CI −0.20 to 0.63] than their nonautistic peers.

Autistic participants did report significantly higher levels of anxiety at baseline [$F(1,138) = 5.08$, $p = 0.03$, $d = 0.41$, 95% CI 0.06 to 0.76], although this difference was not significant at endpoint [$F(1,87) = 1.45$ $p = 0.23$, $d = 0.26$, 95% CI −0.16 to 0.69]. Autistic participants endorsed significantly higher levels of repetitive thinking at both baseline [$F(1,133) = 15.55$, $p < 0.001$, $d = 0.72$, 95% CI 0.36 to 1.08] and endpoint [$F(1,84) = 12.64$ $p < 0.001$, $d = 0.79$, 95% CI 0.35 to 1.22].

We found that overall tendency toward RNT, as measured at baseline through our novel eight-item repetitive thinking battery, predicted higher cross-semester sadness scores in incoming college students. In addition, within the biweekly survey, RNT was related to reported sadness within individual timepoints. We found evidence for these potential mechanisms across neurotypes, such that the inclusion of SRS-2 scores in the model did not eliminate significant differences in the sadness, anhedonia, or anxiety levels of those with greater RNT; however, as we hypothesized, higher SRS-2 scores were associated with higher levels of both the RNT predictor and baseline depression and anxiety scores.

Taken together, this suggests that, while this mechanism may not be autism specific, elevated rates of RNT in the autistic population may partially explain the heightened prevalence of depression and anxiety. Of note, we did not find support for the hypothesized immediate temporally predictive relationship between RNT at one timepoint and subsequent internalizing symptoms at the next timepoint.

By integrating these differing temporal findings, we may inform future hypotheses on the timing of predictive relationships of interest. First, we find there is evidence of a longer term temporally predictive relationship between more stable trait-like repetitive thinking status and short-term (''in the last few days'') sadness, anhedonia, and anxiety over the semester. In our baseline repetitive thinking battery, participants were asked to rate their agreement with items, such as ''My thoughts prevent me from focusing on other things.''

These prompts were *not* prefaced with a time frame (e.g., ''within the last week…''), therefore we may expect these responses to apply to participant's overall tendencies, rather than their behavior in any particular day or week. Again, participants (across SRS-2 scores) who endorsed more repetitive thinking on these items tended to report more internalizing symptoms throughout the semester: This is similar to the research findings from the only other known longitudinal investigation of repetitive thinking and mood concerns in autistic populations.[60] In this previous study—which was done with autistic children aged 9–15 years—tendencies toward rumination (as measured on the Worry/Rumination Questionnaire for Children, which is similarly not time bound) were related to sadness over an extended period of time (intervals of 9 months).[60]

Therefore, our current finding converges with other evidence that more stable RNT tendencies are likely to be prospectively associated with internalizing symptoms. We also found evidence of a synchronous relationship between RNT and internalizing symptoms, although we cannot currently assess the directionality (or bidirectionality) of this relationship.

Although longer term prospective relationships were observed, we had also anticipated a more immediate predictive relationship such that repetitive thinking at one biweekly timepoint would predict internalizing symptoms at the next timepoint. We did not find support for this temporal relationship—only lagged RNT to future anhedonia showed a statistically significant relationship (with no relationships observed between lagged RNT and future anxiety or sadness), and the small effect size suggests that even the relationship between lagged RNT and future anhedonia may not be a meaningful relationship.

Thus, our current data inform new hypotheses of the temporal relationship between RNT and internalizing symptoms. In this updated model, stable ongoing RNT may contribute to the development of depression and anxiety overall. In addition, in-the-moment RNT was robustly related to discrete instances of sadness, anhedonia, and anxiety, possibly with bidirectional effects. However, discrete experiences of repetitive thinking days beforehand may have little relation to internalizing symptoms at a particular timepoint. At this time, we did not have the power to test how accruing timepoints' RNT affected single instance future timepoint internalizing symptoms. We look forward to exploring this relationship further, as data collection waves are ongoing.

Cohort differences

The current research supports previous evidence that autistic adults and/or those with greater autistic traits endorse more sadness and anxiety than nonautistic adults.[7] Students with higher SRS-2 scores endorsed greater RNT, sadness, anhedonia, and anxiety in our sample. In addition, some group differences were detected by autism group, such that our broadly defined autistic group (including individuals with a formal diagnosis or who had a history of thinking or others commenting that they may be autistic) reported more anxiety at the beginning of the semester and engaged in more RNT than participants who did not identify as autistic or are likely to be autistic.

Again, our findings support the theory that known predictors of internalizing symptoms in the general population, such as RNT, may be similarly related to depression and anxiety in the autistic community, although autistic people may engage in *more* of these risk-related behaviors, which may contribute to the disproportionate rates of depression and anxiety in the autistic community.

Limitations

As noted previously, few of our participants had a formal autism diagnosis. Instead, many endorsed a history of thinking (or others commenting that) they may be autistic. SRS-2 scores ranged widely in this group, suggesting that many of these students may not have met full criteria for an autism diagnosis (though see also the Supplementary Fig. S1 showing far greater distributional overlap between suspected and diagnosed autistic participants compared with nonautistic participants). As a result, we opted to use SRS-2 scores as a dimensional proxy for autistic traits rather than using categorical groupings.

While this allowed us to assess how autistic traits affect mental health during the transition to college, it did not allow us to estimate how the *social identity* of being autistic may affect well-being. This may harm the generalizability of our research to the broader autistic population who is formally diagnosed and/or identifies more strongly as being autistic. Despite these weaknesses, treating the autism spectrum as dimensional rather than categorical did allow for greater statistical power. Furthermore, this approach may have allowed for better inclusion of historically under-represented groups of autistic adults who have lower access to formal diagnoses.[26]

Finally, this study, while longitudinal, was short term in nature. As a result, it is not possible to determine whether there is a slower growing, or longer term predictive relationship

between mechanisms of interest and subsequent depression and anxiety. Unfortunately, data suggest that many incoming college students—and perhaps particularly autistic students[94]—may have already experienced depression and anxiety in their lifetimes. Within our current study, we cannot know how the potentially bidirectional cycle of predictors of interest and negative mental health outcomes has already affected participants and at what ''stage'' of this cycle they may be in when beginning the semester.

While the extant literature does not fully indicate where research should focus, in terms of developmental stage or point in the lifespan, to identify the onset of these patterns, this highly novel work is a helpful starting point. However, the limited sample at this time limits our ability to include all potential covariates. As noted in Table 2, results followed a similar pattern when BDI-II scores or GAD-7 scores were held as covariates, instead of SRS-2 scores. We have not yet examined a combined model with all three of these potentially useful covariates, and this may be a potentially fruitful consideration as further waves of data are collected.

In this study, we attempted to mitigate the possible limitations of not creating this combined model by including time in each of our models. This allows us to evaluate the effect of RNT on symptoms above any influence of the underlying symptom trajectory; therefore, prior depression, anxiety, and RNT scores are indirectly accounted for.

Clinical implications and future directions

To move toward translating conclusions into prevention and intervention efforts, future research may benefit from investigating both the *cause* of the increased rate of RNT in autistic adults and the *types* of RNT that are most detrimental. While previous research has supported the idea that autistic people tend to engage in more repetitive thought overall, and, therefore, it may seem logical that this population would also engage in more *negative* repetitive thinking, there may be alternative explanations to the elevated RNT noted in autistic populations.[95]

Given that autistic people are a marginalized population—and often marginalized in multiple intersecting ways[96]—the elevated use of this maladaptive cognitive processing may be partially explained by the social stress theory[56] and psychological mediation model.[57] These models suggest that minoritized groups may experience greater stress (in the psychological mediation model theory due to heterosexism; in this case, perhaps more directly due to ableism), which, according to the psychological mediation model, results in elevated negative coping strategies.

Therefore, this elevated rate of RNT may not be exclusively due to the cognitive style associated with autism, but instead the intersection of autism and stigma in society. Future studies are, therefore, necessary to disentangle these potential internal and external causal factors.

In addition, prior research has indicated that not all repetitive thinking is equally harmful. General population research has suggested that the relationship between rumination and increased depressive symptoms may be driven by the ''moody pondering'' subcomponent of rumination known as brooding.[32] Recent findings from our research team identified stagnant thought—particularly, perseverating on problems without coming to a solution—as a statistically central (''influential'') symptom within a community of maladaptive repetitive thinking items in autistic adults.[97] Therefore, future research should continue to disentangle components of repetitive thinking to find the most effective intervention targets.

Intervention efforts may include making adaptations to existing therapeutic techniques to interrupt negative thought ''spirals.'' If the relationship between these mechanisms and depressive symptoms continues to be seen as more synchronous, then interventions might focus on being accessible and useful while a client is actively distressed. Intervention strategies such as those taught in Rumination-Focused Cognitive Behavioral Therapy[98] or Dialectical Behavioral Therapy[99] may focus on making cognitive techniques more accessible during these high-stress moments, perhaps by employing emotion regulation strategies first.

In the current findings, the relationship between RNT and internalizing symptoms did not seem to differ across levels of autistic traits. This suggests that existing interventions that target RNT in the general population could be effective within autistic populations, if conducted through a neurodiversity-affirming lens.[100] Finding this overlap in clinical targets is an important step toward increasing mental health care access for autistic adults. Many clinicians express hesitancy to work with autistic clients, due to low confidence in their competency, and this contributes to autistic adults' difficulties in accessing mental health care.[101]

Therefore, future efforts should consider focusing on training clinicians to better implement adaptations of effective mental health interventions for the autistic community and, as indicated, increasing clinician confidence in using them with autistic adults.[102–108]

Conclusions

Similar to literature, the present research underscores a key mental health disparity: first-semester college students with greater autistic traits are more likely to experience depression and anxiety symptoms throughout the semester than their peers with fewer self-reported autistic traits. Our current evidence suggests that this may be in part related to the comparatively higher levels of RNT that autistic students and students with higher autistic traits are likely to engage in. This study advances our understanding of how autistic traits may affect the experience of transitioning to college and the risk factors for depression and anxiety in autistic adults more broadly.

Mental health is a crucial priority of the autistic community, and the current findings represent additional steps toward both supporting college success and identifying intervention targets for improved mental health and well-being among autistic adults.

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Authorship Confirmation Statement

E.E.M. contributed to conceptualization, formal analysis, investigation, and writing—original draft (lead); S.M.B. was

involved in conceptualization, methodology, formal analysis, and writing—review and editing; J.K.R. assisted in conceptualization and project administration; T.C.D. contributed to investigation and writing—review and editing; B.K. contributed to investigation and writing—review and editing; R.G.M. was involved in investigation and writing—review and editing; Z.J.W. assisted in writing—review and editing; K.G.-L. was involved in investigation, supervision, and writing—review and editing; E.K. contributed to investigation, supervision, and writing—review and editing; M.D.L. assisted in investigation and supervision; K.O.G. carried out conceptualization, methodology, resources, and writing—original draft (supporting), writing—review and editing, supervision, and funding acquisition.

Author Disclosure Statement

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Supplementary Material

Supplementary Data
Supplementary Figure S1

References

1. Hudson CC, Hall L, Harkness KL. Prevalence of depressive disorders in individuals with autism spectrum disorder: A meta-analysis. *J Abnorm Child Psychol*. 2019;47(1):165–175. https://doi.org/10.1007/s10802-018-0402-1.
2. Lawson LP, Richdale AL, Haschek A, et al. Cross-sectional and longitudinal predictors of quality of life in autistic individuals from adolescence to adulthood: The role of mental health and sleep quality. *Autism*. 2020;24(4):954–967. https://doi.org/10.1177/1362361320908107.
3. Park SH, Song YJC, Demetriou EA, et al. Disability, functioning, and quality of life among treatment-seeking young autistic adults and its relation to depression, anxiety, and stress. *Autism*. 2019;23(7):1675–1686. https://doi.org/10.1177/1362361318823925.
4. Joshi G, Wozniak J, Petty C, et al. Psychiatric comorbidity and functioning in a clinically referred population of adults with autism spectrum disorders: A comparative study. *J Autism Dev Disord*. 2013;43(6):1314–1325. https://doi.org/10.1007/s10803-012-1679-5.
5. Cassidy S, Bradley L, Shaw R, Baron-Cohen S. Risk markers for suicidality in autistic adults. *Mol Autism*. 2018;9(1):42. https://doi.org/10.1186/s13229-018-0226-4.
6. Cassidy SA, Bradley L, Cogger-Ward H, Rodgers J. Development and validation of the suicidal behaviours questionnaire—Autism spectrum conditions in a community sample of autistic, possibly autistic and non-autistic adults. *Mol Autism*. 2021;12(1):46. https://doi.org/10.1186/s13229-021-00449-3.
7. Hollocks MJ, Lerh JW, Magiati I, Meiser-Stedman R, Brugha TS. Anxiety and depression in adults with autism spectrum disorder: A systematic review and meta-analysis. *Psychol Med*. 2019;49(4):559–572. https://doi.org/10.1017/S0033291718002283.
8. Gillott A, Standen PJ. Levels of anxiety and sources of stress in adults with autism. *J Intellect Disabil*. 2007;11(4):359–370. https://doi.org/10.1177/1744629507083585.
9. Grant S, Norton S, Weiland RF, Scheeren AM, Begeer S, Hoekstra RA. Autism and chronic ill health: An observational study of symptoms and diagnoses of central sensitivity syndromes in autistic adults. *Mol Autism*. 2022;13(1):7. https://doi.org/10.1186/s13229-022-00486-6.
10. Taylor EC, Livingston LA, Callan MJ, Ashwin C, Shah P. Autonomic dysfunction in autism: The roles of anxiety, depression, and stress. *Autism*. 2021;25(3):744–752. https://doi.org/10.1177/1362361320985658.
11. Williams ZJ, Gotham KO. Assessing general and autism-relevant quality of life in autistic adults: A psychometric investigation using item response theory. *Autism Res*. 2021;14(8):1633–1644. https://doi.org/10.1002/aur.2519.
12. Lamers F, Oppen P van, Comijs HC, et al. Comorbidity patterns of anxiety and depressive disorders in a large cohort study: The Netherlands study of depression and anxiety (NESDA). *J Clin Psychiatry*. 2011;72(3):341–348. https://doi.org/10.4088/JCP.10m06176blu.
13. Benevides TW, Shore SM, Palmer K, et al. Listening to the autistic voice: Mental health priorities to guide research and practice in autism from a stakeholder-driven project. *Autism*. 2020;24(4):822–833. https://doi.org/10.1177/1362361320908410.
14. Crane L, Adams F, Harper G, Welch J, Pellicano E. 'Something needs to change': Mental health experiences of young autistic adults in England. *Autism*. 2019;23(2):477–493. https://doi.org/10.1177/1362361318757048.
15. Pellicano E, Dinsmore A, Charman T. What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*. 2014;18(7):756–770. https://doi.org/10.1177/1362361314529627.
16. Van Hees V, Moyson T, Roeyers H. Higher education experiences of students with autism spectrum disorder: Challenges, benefits and support needs. *J Autism Dev Disord*. 2015;45(6):1673–1688. https://doi.org/10.1007/s10803-014-2324-2.
17. Gunin GB, Gravino A, Bal VH. Advancing mental health supports for autistic postsecondary students: A call for research. *Autism Adulthood*. 2021;3(1):30–36. https://doi.org/10.1089/aut.2020.0044.
18. McMorris CA, Baraskewich J, Ames MA, Shaikh KT, Ncube BL, Bebko JM. Mental health issues in postsecondary students with autism spectrum disorder: Experiences in accessing services. *Int J Ment Health Addict*. 2019;17(3):585–595. https://doi.org/10.1007/s11469-018-9988-3.
19. Bakker TC, Krabbendam L, Bhulai S, Begeer S. First-year progression and retention of autistic students in higher

education: A propensity score-weighted population study. *Autism Adulthood*. 2020;2(4):307–316. https://doi.org/10.1089/aut.2019.0053.
20. Bakker TC, Krabbendam L, Bhulai S, Begeer S. Background and enrollment characteristics of students with autism in higher education. *Res Autism Spectr Disord*. 2019;67:101424. https://doi.org/10.1016/j.rasd.2019.101424.
21. McLeod JD, Meanwell E, Hawbaker A. The experiences of college students on the autism spectrum: A comparison to their neurotypical peers. *J Autism Dev Disord*. 2019; 49(6):2320–2336. https://doi.org/10.1007/s10803-019-03910-8.
22. Sturm A, Kasari C. Academic and psychosocial characteristics of incoming college freshmen with autism spectrum disorder: The role of comorbidity and gender. *Autism Res*. 2019;12(6):931–940. https://doi.org/10.1002/aur.2099.
23. Shattuck PT, Narendorf SC, Cooper B, Sterzing PR, Wagner M, Taylor JL. Postsecondary education and employment among youth with an autism spectrum disorder. *Pediatrics*. 2012;129(6):1042–1049. https://doi.org/10.1542/peds.2011-2864.
24. Kaplan RM, Fang Z, Kirby J. Educational attainment and health outcomes: Data from the Medical Expenditures Panel Survey. *Health Psychol*. 2017;36(6):598–608. https://doi.org/10.1037/hea0000431.
25. Ross CE, Van Willigen M. Education and the subjective quality of life. *J Health Soc Behav*. 1997;38(3):275–297. https://doi.org/10.2307/2955371.
26. Huang Y, Arnold SR, Foley KR, Trollor JN. Diagnosis of autism in adulthood: A scoping review. *Autism*. 2020;24(6): 1311–1327. https://doi.org/10.1177/1362361320903128.
27. Wiggins LD, Durkin M, Esler A, et al. Disparities in documented diagnoses of autism spectrum disorder based on demographic, individual, and service factors. *Autism Res*.2020;13(3):464–473. https://doi.org/10.1002/aur.2255.
28. Ehring T, Watkins ER. Repetitive negative thinking as a transdiagnostic process. *Int J Cogn Ther*. 2008;1(3):192–205. https://doi.org/10.1521/ijct.2008.1.3.192.
29. McEvoy PM, Watson H, Watkins ER, Nathan P. The relationship between worry, rumination, and comorbidity: Evidence for repetitive negative thinking as a transdiagnostic construct. *J Affect Disord*. 2013;151(1):313–320. https://doi.org/10.1016/j.jad.2013.06.014.
30. McLaughlin KA, Nolen-Hoeksema S. Rumination as a transdiagnostic factor in depression and anxiety. *Behav Res Ther*. 2011;49(3):186–193. https://doi.org/10.1016/j.brat.2010.12.006.
31. Spinhoven P, Drost J, van Hemert B, Penninx BW. Common rather than unique aspects of repetitive negative thinking are related to depressive and anxiety disorders and symptoms. *J Anxiety Disord*. 2015;33:45–52. https://doi.org/10.1016/j.janxdis.2015.05.001.
32. Treynor W, Gonzalez R, Nolen-Hoeksema S. Rumination reconsidered: A psychometric analysis. *Cogn Ther Res*. 2003;27(3):247–259. https://doi.org/10.1023/A:1023910315561.
33. Watkins ER, Roberts H. Reflecting on rumination: Consequences, causes, mechanisms and treatment of rumination. *Behav Res Ther*. 2020;127:103573. https://doi.org/10.1016/j.brat.2020.103573.
34. Nolen-Hoeksema S, Davis CG. ''Thanks for sharing that'': Ruminators and their social support networks. *J Pers Soc Psychol*. 1999;77(4):801–814. https://doi.org/10.1037/0022-3514.77.4.801.
35. Nolen-Hoeksema S. Responses to depression and their effects on the duration of depressive episodes. *J Abnorm Psychol*. 1991;100(4):569–582. https://doi.org/10.1037/0021-843X.100.4.569.
36. Hoebeke Y, Blanchard A, Contreras A, Heeren A. An experience sampling measure of the key features of rumination. *Clin Neuropsychiatry*. 2022;19:288–297. https://doi.org/10.36131/cnfioritieditore20220504.
37. Abela JRZ, Hankin BL. Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: A multiwave longitudinal study. *J Abnorm Psychol*. 2011;120(2):259–271. https://doi.org/10.1037/a0022796.
38. Nolen-Hoeksema S. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *J Abnorm Psychol*. 2000;109(3):504–511. https://doi.org/10.1037/0021-843X.109.3.504.
39. Thomsen DK, Mehlsen MY, Olesen F, et al. Is there an association between rumination and self-reported physical health? A one-year follow-up in a young and an elderly sample. *J Behav Med*. 2004;27(3):215–231. https://doi.org/10.1023/B:JOBM.0000028496.41492.34.
40. Watkins ER. Constructive and unconstructive repetitive thought. *Psychol Bull*. 2008;134(2):163–206. https://doi.org/10.1037/0033-2909.134.2.163.
41. Miranda R, Nolen-Hoeksema S. Brooding and reflection: Rumination predicts suicidal ideation at 1-year follow-up in a community sample. *Behav Res Ther*. 2007;45(12): 3088–3095. https://doi.org/10.1016/j.brat.2007.07.015.
42. Gotham K, Bishop SL, Brunwasser S, Lord C. Rumination and perceived impairment associated with depressive symptoms in a verbal adolescent–adult ASD sample. *Autism Res*. 2014;7(3):381–391. https://doi.org/10.1002/aur.1377.
43. Crane L, Goddard L, Pring L. Autobiographical memory in adults with autism spectrum disorder: The role of depressed mood, rumination, working memory and theory of mind. *Autism*. 2013;17(2):205–219. https://doi.org/10.1177/1362361311418690.
44. Williams ZJ, McKenney EE, Gotham KO. Investigating the structure of trait rumination in autistic adults: A network analysis. *Autism*. 2021;25(7):2048–2063. https://doi.org/10.1177/13623613211012855.
45. Dajani DR, Uddin LQ. Demystifying cognitive flexibility: Implications for clinical and developmental neuroscience. *Trends Neurosci*. 2015;38(9):571–578. https://doi.org/10.1016/j.tins.2015.07.003.
46. Hollocks MJ, Charman T, Baird G, Lord C, Pickles A, Simonoff E. Exploring the impact of adolescent cognitive inflexibility on emotional and behavioural problems experienced by autistic adults. *Autism*. 2022;26(5):1229–1241. https://doi.org/10.1177/13623613211046160.
47. Geurts HM, Corbett B, Solomon M. The paradox of cognitive flexibility in autism. *Trends Cogn Sci*. 2009;13(2):74–82. https://doi.org/10.1016/j.tics.2008.11.006.
48. Stange JP, Alloy LB, Fresco DM. Inflexibility as a vulnerability to depression: A systematic qualitative review. *Clin Psychol Sci Pract*. 2017;24(3):245–276. https://doi.org/10.1111/cpsp.12201.
49. Ozsivadjian A, Hollocks MJ, Magiati I, Happé F, Baird G, Absoud M. Is cognitive inflexibility a missing link? The role of cognitive inflexibility, alexithymia and intolerance of uncertainty in externalising and internalising

behaviours in young people with autism spectrum disorder. *J Child Psychol Psychiatry*. 2021;62(6):715–724. https://doi.org/10.1111/jcpp.13295.
50. Lopez BR, Lincoln AJ, Ozonoff S, Lai Z. Examining the relationship between executive functions and restricted, repetitive symptoms of Autistic Disorder. *J Autism Dev Disord*. 2005;35(4):445–460. https://doi.org/10.1007/s10803-005-5035-x.
51. Panerai S, Tasca D, Ferri R, Genitori D'Arrigo V, Elia M. Executive functions and adaptive behaviour in autism spectrum disorders with and without intellectual disability. *Psychiatry J*. 2014;2014:e941809. https://doi.org/10.1155/2014/941809.
52. American Psychiatric Association, ed. *Desk Reference to the Diagnostic Criteria from DSM-5-TR*. Arlington, TX: American Psychiatric Association Publishing; 2022.
53. Chen YH, Rodgers J, McConachie H. Restricted and repetitive behaviours, sensory processing and cognitive style in children with autism spectrum disorders. *J Autism Dev Disord*. 2008;39:635–642. https://doi.org/10.1007/s10803-008-0663-6.
54. Frith U. *Autism: Explaining the Enigma,* 2nd ed. Oxford: Blackwell Publishing; 2003.
55. Snidman HR, Rice L, Sparger K. Representation, belongingness, and rumination in underrepresented psychology students. *Teach Psychol*. 2022:00986283221095115. [Epub ahead of print]; https://doi.org/10.1177/00986283221095115.
56. Meyer IH. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: Conceptual issues and research evidence. *Psychol Bull*. 2003;129(5):674–697. https://doi.org/10.1037/0033-2909.129.5.674.
57. Hatzenbuehler ML. How does sexual minority stigma ''get under the skin''? A psychological mediation framework. *Psychol Bull*. 2009;135(5):707–730. https://doi.org/10.1037/a0016441.
58. Gotham K, Siegle GJ, Han GT, et al. Pupil response to social-emotional material is associated with rumination and depressive symptoms in adults with autism spectrum disorder. *PLoS One*. 2018;13(8):e0200340. https://doi.org/10.1371/journal.pone.0200340.
59. Keenan EG, Gotham K, Lerner MD. Hooked on a feeling: Repetitive cognition and internalizing symptomatology in relation to autism spectrum symptomatology. *Autism*. 2018;22(7):814–824. https://doi.org/10.1177/1362361317709603.
60. Rieffe C, De Bruine M, De Rooij M, Stockmann L. Approach and avoidant emotion regulation prevent depressive symptoms in children with an autism spectrum disorder. *Int J Dev Neurosci*. 2014;39:37–43. https://doi.org/10.1016/j.ijdevneu.2014.06.003.
61. Mayes SD, Calhoun SL, Murray MJ, Zahid J. Variables associated with anxiety and depression in children with autism. *J Dev Phys Disabil*. 2011;23(4):325–337. https://doi.org/10.1007/s10882-011-9231-7.
62. Burrows CA, Timpano KR, Uddin LQ. Putative brain networks underlying repetitive negative thinking and comorbid internalizing problems in autism. *Clin Psychol Sci J Assoc Psychol Sci*. 2017;5(3):522–536. https://doi.org/10.1177/2167702616683506.
63. Beck AT, Steer RA, Brown GK. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1996.
64. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: The GAD-7. *Arch Intern Med*. 2006;166(10):1092–1097. https://doi.org/10.1001/archinte.166.10.1092.
65. Constantino JN, Gruber CP. *Social Responsiveness Scale,* 2nd ed. Torrance, CA: Western Psychological Services; 2012.
66. Murphy J, Prentice F, Walsh R, Catmur C, Bird G. Autism and transgender identity: Implications for depression and anxiety. *Res Autism Spectr Disord*. 2020;69:101466. https://doi.org/10.1016/j.rasd.2019.101466.
67. Grissom RJ, Kim JJ. Review of assumptions and problems in the appropriate conceptualization of effect size. *Psychol Methods*. 2001;6(2):135–146. https://doi.org/10.1037/1082-989X.6.2.135.
68. Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, Conde JG. Research electronic data capture (REDCap)—A metadata-driven methodology and workflow process for providing translational research informatics support. *J Biomed Inform*. 2009;42(2):377–381. https://doi.org/10.1016/j.jbi.2008.08.010.
69. Harris PA, Taylor R, Minor BL, et al. The REDCap consortium: Building an international community of software platform partners. *J Biomed Inform*. 2019;95:103208. https://doi.org/10.1016/j.jbi.2019.103208.
70. Wang YP, Gorenstein C. Psychometric properties of the Beck Depression Inventory-II: A comprehensive review. *Braz J Psychiatry*. 2013;35:416–431. https://doi.org/10.1590/1516-4446-2012-1048.
71. Williams ZJ, Everaert J, Gotham KO. Measuring depression in autistic adults: Psychometric validation of the Beck Depression Inventory–II. *Assessment*. 2021;28(3):858–876. https://doi.org/10.1177/1073191120952889.
72. Williams ZJ, Gotham KO. Current and lifetime somatic symptom burden among transition-aged autistic young adults. *Autism Res*. 2022;15(4):761–770. https://doi.org/10.1002/aur.2671.
73. Hull L, Levy L, Lai MC, et al. Is social camouflaging associated with anxiety and depression in autistic adults? *Mol Autism*. 2021;12(1):13. https://doi.org/10.1186/s13229-021-00421-1.
74. Kroenke K, Spitzer RL, Williams JBW, Löwe B. The Patient Health Questionnaire Somatic, Anxiety, and Depressive Symptom Scales: A systematic review. *Gen Hosp Psychiatry*. 2010;32(4):345–359. https://doi.org/10.1016/j.genhosppsych.2010.03.006.
75. Feliciano P, Daniels AM, Green Snyder L, et al. SPARK: A US cohort of 50,000 families to accelerate autism research. *Neuron*. 2018;97(3):488–493. https://doi.org/10.1016/j.neuron.2018.01.015.
76. Ehring T, Zetsche U, Weidacker K, Wahl K, Schönfeld S, Ehlers A. The Perseverative Thinking Questionnaire (PTQ): Validation of a content-independent measure of repetitive negative thinking. *J Behav Ther Exp Psychiatry*. 2011;42(2):225–232. https://doi.org/10.1016/j.jbtep.2010.12.003.
77. Trapnell PD, Campbell JD. Private self-consciousness and the five-factor model of personality: Distinguishing rumination from reflection. *J Pers Soc Psychol*. 1999;76(2):284–304. https://doi.org/10.1037/0022-3514.76.2.284.
78. Feldman G, Hayes A. Preparing for problems: A measure of mental anticipatory processes. *J Res Personal*. 2005;39(5):487–516. https://doi.org/10.1016/j.jrp.2004.05.005.

79. Meyer TJ, Miller ML, Metzger RL, Borkovec TD. Development and validation of the Penn State Worry Questionnaire. *Behav Res Ther*. 1990;28(6):487–495. https://doi.org/10.1016/0005-7967(90)90135-6.
80. Fried EI, Nesse RM. Depression sum-scores don't add up: Why analyzing specific depression symptoms is essential. *BMC Med*. 2015;13(1):72. https://doi.org/10.1186/s12916-015-0325-4.
81. Wagner RE, Zhang Y, Gray T, et al. Autism-related variation in reciprocal social behavior: A longitudinal study. *Child Dev*. 2019;90(2):441–451. https://doi.org/10.1111/cdev.13170.
82. Pinheiro JC, Bates DM. Linear Mixed-Effects Models: Basic Concepts and Examples. In: Pinheiro JC, Bates DM, eds. *Mixed-Effects Models in S and S-PLUS. Statistics and Computing*. New York: Springer; 2000;3–56.
83. Harrell Jr. FE. rms: Regression Modeling Strategies. Published online April 22, 2022. https://CRAN.R-project.org/package=rms Accessed August 2, 2022.
84. Pinheiro J, Bates D, DebRoy S, etal. nlme: Linear and nonlinear mixed effects models. Published online August 9, 2022. https://CRAN.R-project.org/package=nlme Accessed August 23, 2022.
85. McNeish D, Stapleton LM, Silverman RD. On the unnecessary ubiquity of hierarchical linear modeling. *Psychol Methods*. 2017;22(1):114–140. https://doi.org/10.1037/met0000078.
86. Pekár S, Brabec M. Marginal models via GLS: A convenient yet neglected tool for the analysis of correlated data in the behavioural sciences. *Ethology*. 2016;122(8):621–631. https://doi.org/10.1111/eth.12514.
87. Lorah J. Effect size measures for multilevel models: Definition, interpretation, and TIMSS example. *Large Scale Assess Educ*. 2018;6(1):8. https://doi.org/10.1186/s40536-018-0061-2.
88. Selya AS, Rose JS, Dierker LC, Hedeker D, Mermelstein RJ. A practical guide to calculating Cohen's f2, a measure of local effect size, from PROC MIXED. *Front Psychol*. 2012;3:111. https://doi.org/10.3389/fpsyg.2012.00111.
89. Gauthier J, Wu QV, Gooley TA. Cubic splines to model relationships between continuous variables and outcomes: A guide for clinicians. *Bone Marrow Transplant*. 2020;55(4):675–680. https://doi.org/10.1038/s41409-019-0679-x.
90. Harrell J. *Regression Modeling Strategies: With Applications to Linear Models, Logistic and Ordinal Regression, and Survival Analysis*, 2nd ed. Springer International Publishing: Imprint: Switzerland: Springer; 2015.
91. South M, Carr AW, Stephenson KG, Maisel ME, Cox JC. Symptom overlap on the SRS-2 adult self-report between adults with ASD and adults with high anxiety. *Autism Res*. 2017;10(7):1215–1220. https://doi.org/10.1002/aur.1764.
92. Havdahl KA, Bal VH, Huerta M, et al. Multidimensional influences on autism symptom measures: Implications for use in etiological research. *J Am Acad Child Adolesc Psychiatry*. 2016;55(12):1054–1063.e3. https://doi.org/10.1016/j.jaac.2016.09.490.
93. Hus V, Bishop S, Gotham K, Huerta M, Lord C. Factors influencing scores on the social responsiveness scale. *J Child Psychol Psychiatry*. 2013;54(2):216–224. https://doi.org/10.1111/j.1469-7610.2012.02589.x.
94. Magnuson KM, Constantino JN. Characterization of depression in children with autism spectrum disorders. *J Dev Behav Pediatr*. 2011;32(4):332–340. https://doi.org/10.1097/DBP.0b013e318213f56c.
95. Botha M, Frost DM. Extending the minority stress model to understand mental health problems experienced by the autistic population. *Soc Ment Health*. 2020;10(1):20–34. https://doi.org/10.1177/2156869318804297.
96. Pearson A, Rose K. A conceptual analysis of autistic masking: Understanding the narrative of stigma and the illusion of choice. *Autism Adulthood*. 2021;3(1):52–60. https://doi.org/10.1089/aut.2020.0043.
97. Gotham K, Williams ZJ. A network analysis of repetitive thinking in autistic adults. 2021. International Society for Autism Research (INSAR), virtual conference. https://doi.org/10.13140/RG.2.2.13107.71204.
98. Watkins ER. *Rumination-Focused Cognitive-Behavioral Therapy for Depression*. New York: Guilford Publications; 2018.
99. Linehan MM, Wilks CR. The course and evolution of dialectical behavior therapy. *Am J Psychother*. 2015;69(2):97–110. https://doi.org/10.1176/appi.psychotherapy.2015.69.2.97.
100. Chapman R, Botha M. *Neurodivergence-Informed Therapy*. Developmental Medicine & Child Neurology, In print, 2022.
101. Maddox BB, Crabbe S, Beidas RS, et al. ''I wouldn't know where to start'': Perspectives from clinicians, agency leaders, and autistic adults on improving community mental health services for autistic adults. *Autism*. 2020;24(4):919–930. https://doi.org/10.1177/1362361319882227.
102. Bal VH, Wilkinson E, Glascock V, Hastings RP, Jahoda A. Mechanisms of change in behavioral activation: Adapting depression treatment for autistic people. *Cogn Behav Pract*. 2022. [Epub ahead of print]; https://doi.org/10.1016/j.cbpra.2022.03.006.
103. Bemmer ER, Boulton KA, Thomas EE, et al. Modified CBT for social anxiety and social functioning in young adults with autism spectrum disorder. *Mol Autism*. 2021;12(1):11. https://doi.org/10.1186/s13229-021-00418-w.
104. Cooper K, Loades ME, Russell A. Adapting psychological therapies for autism. *Res Autism Spectr Disord*. 2018;45:43–50. https://doi.org/10.1016/j.rasd.2017.11.002.
105. Keefer A, White SW, Vasa RA, Reaven J. Psychosocial interventions for internalizing disorders in youth and adults with ASD. *Int Rev Psychiatry*. 2018;30(1):62–77. https://doi.org/10.1080/09540261.2018.1432575.
106. McVey A, Schiltz H, Boeh H. A call for compassion: Evidence-based strategies for working with autistic clients without intellectual disability in therapy. 2021;44:23–31.
107. Spain D, Milner V, Mason D, et al. Improving cognitive behaviour therapy for autistic individuals: A Delphi survey with practitioners. *J Ration Emot Cogn Behav Ther*. 2022;1–19. [Epub ahead of print]; https://doi.org/10.1007/s10942-022-00452-4.
108. White SW, Simmons GL, Gotham KO, et al. Psychosocial treatments targeting anxiety and depression in adolescents and adults with autism spectrum disorder: Review of the latest research and recommended future directions. *Curr Psychiatry Rep*. 2018;20(10):82. https://doi.org/10.1007/s11920-018-0949-0.

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