



Identifying Predictors of Momentary Negative Affect and Depression Severity in Adolescents with Autism: An Exploratory Ecological Momentary Assessment Study

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Accepted: 15 February 2021 / Published online: 7 March 2021

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Abstract

Depression is a common comorbidity in autism spectrum disorder (ASD). Little is known about risk factors for depression and depressive symptoms in this population. Ecological momentary assessment (EMA) has been used in the typically developing population to identify risk factors for depression, but has been rarely applied in ASD populations. In this exploratory study, 17 autistic adolescents participated in an EMA protocol in which they reported on their current activities and emotions six times per day for seven consecutive days. Results suggested that negative affect is predicted by momentary quality of social interaction and enjoyment of the current activity ($p < 0.05$). Additionally, affective instability predicted depressive symptoms. These results provide insights into risk factors for depression in this vulnerable population.

Keywords Autism · Ecological momentary assessment · Depression · Adolescence · Social interaction

Background

Depression is a commonly reported and concerning mental health comorbidity in autism spectrum disorder (ASD; Mazefsky et al., 2011). Estimates of depression in the non-psychiatrically referred autistic population range from 2 (Ghaziuddin et al., 1992) to 44% (Strang et al., 2012) and as high as 56% in a clinically referred ASD population (Joshi et al., 2010). While there is no consensus regarding the prevalence of depression, researchers generally agree that autistic individuals are at high risk for depression, especially compared to their typically developing peers (who have a prevalence of 7.5%) (Avenevoli et al., 2015). In general, depression is often overlooked in adolescence (Leaf et al., 1996), but autistic adolescents are at an even greater risk of a missed diagnosis. The goal of this study was to characterize

risk factors for depressive symptomatology among adolescents with autism spectrum disorder, using self-report.

The variance in prevalence, and potential failure of identifying at-risk autistic individuals, can be explained for several reasons. (1) It is challenging to differentiate prototypical ASD symptoms from comorbid depression due to overlapping symptomatology such as obsessive and ritualistic thinking, agitation, self-injury, sleep disturbances and a decrease in former interests and adaptive functioning (DeFilippis, 2018). These overlapping features can mask the onset of depression thereby making its diagnosis in ASD difficult, particularly during adolescence (DeFilippis, 2018). (2) While self-report is generally preferred to identify internalizing symptoms, self-report measures frequently under-identify cases in this population (Mazefsky et al., 2011). A recent meta-analysis of depression prevalence among adults on the autism spectrum found that clinical interviews tend to identify more cases of depression than questionnaires (Hollocks et al., 2019). In some studies caregivers (Taylor et al., 2020) and clinicians (Mazefsky et al., 2011) reported higher rates of depression than those reported via self-report in the ASD population, while, in other cases, caregivers and children both reported similar levels of depression (Ozsivadjian et al., 2014). Reporter type and other measurement effects have led to wide degrees of variance in previously observed pediatric depression rates in ASD (Wigham et al., 2017). To overcome

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measurement and identification issues best-practices suggest that self-report, parent-report and clinical-rated scales are needed to evaluate depression severity. Given that the US Preventative Services Task Force (USPSTF) (Siu et al., 2016) has recommended that all adolescents be screened for depressive symptomatology, efforts to develop effective and sensitive tools to measure risk factors for depressive symptomatology among autistics are needed in the field.

Two-Hit Model of ASD

While adolescence can be a challenging time physically, socially, and academically for those with and without developmental disabilities, adolescence may be a particularly vulnerable time for autistic individuals (Picci & Scherf, 2015). The two-hit model of ASD (Picci & Scherf, 2015) proposes that autistic individuals experience the first *hit* with biological genetic variations that predispose them to typical challenges associated with the diagnosis such as deficits in social-emotional reciprocity, nonverbal communication, development, maintenance, and understanding of relationships (American Psychiatric Association [APA], 2013), and altered sensory processing (Serafini et al., 2017). These initial vulnerabilities pose unique challenges for children in their social development.

The second *hit* occurs when children transition into the period of adolescence when their initial vulnerabilities worsen with the onset of the hormonal, social, and academic challenges typical of adolescence. Social challenges such as bullying (Cook et al., 2010; Espelage & Holt, 2001), a common experience during adolescence, further expose individuals at this developmental stage to risks for emotional disorders such as depression. These increased social demands may heighten emotional regulation impairments (Mazefsky & White, 2014; Mazefsky et al., 2014), further increasing risk for depression (Gross & Thompson, 2006). Emotional regulation (ER) is defined as the adaptive ability to modulate one's emotions to fit changing situations (see Mazefsky & White, 2014 for a review). Since the experiential characteristics of the *second hit* include situational challenges, and ER is contingent on changing situations (Gupta & Gehlawat, 2020), then the challenges posed during the *second hit* are likely to exacerbate ER deficits. Recent empirical research has also related social experiences to ER and mental health among autistic individuals. For example, when autistic individuals experience game-related trade offers they deem to be unfair, they experience more negative moods than their non-autistic peers, suggesting that ER is linked to perceived social experiences (Tei et al., 2018). Autistic individuals who are aware of their social deficits demonstrate increased depression (Day et al., 2019) suggesting that social experiences, such as those that increase one's awareness of social deficits, may be implicated in psychopathology. While

increasing ASD symptom severity has been linked to depression, loneliness and satisfaction with social support mediate this relationship (Hedley et al., 2018), further implicating social experiences in the severity of depression.

The constellation of potential factors impacting depressive symptomatology in this population indicates there is likely no singular cause of depressive symptomatology in ASD. However, theoretical and empirical research aligns in implicating social experiences in the onset of depression (Day et al., 2019; Picci & Scherf, 2015). Recently, researchers investigating depressive symptomatology have challenged monolithic research approaches (i.e., those that examine only a singular cause of depression) and suggested that network approaches better identify the constellation of factors that increase the risk for depressive symptomatology (Koval et al., 2013).

Network View on Emotional Dynamics

In the neurotypical population, features of decreased psychological functioning and emotional dysregulation (i.e., emotional instability, emotional inertia) are believed to increase vulnerability for depression (Koval et al., 2013). *Emotional instability* refers to the magnitude of variation in an individual's emotional states across time and contexts (Houben et al., 2015). *Emotional Inertia* refers to the continuance of emotions from one moment to the next (Koval et al., 2012). The inertia of positive emotions, such as happiness, can be protective of psychological disorders, while the inertia of negative emotions, such as sadness, can result in the onset of depression (Koval et al., 2013). Within the network view on emotional dynamics, microlevel emotional states can change over time, unnoticed by parents or caregivers. These microlevel changes are expected to vary across contexts in a way that would prevent parents or other individuals from bearing witness to the changes, because they will occur at school, at the mall, and in other settings where parents are not present. Accordingly, successful identification of situations, such as positive social experiences, that prevent the inertia of negative emotions or reduce instability of emotions can be used to prevent the cascade of subsequent negative emotional states in an intervention (Fried et al., 2017, p. 6). The network theory of emotional dynamics integrates with other perspectives of mental health in arguing that decreasing psychological functioning (i.e., as demonstrated by the presence of affective instability) increases risk for mental illness (i.e., depression) (Caspi et al., 2014; Ward et al., 2017). While researchers have traditionally investigated emotional instability assuming a linear relationship between instability and depression, researchers have recently questioned whether this relationship would be better characterized using a model that better

incorporates the nuances of emotional instability (Houben et al., 2015). It may be that there are optimal levels of emotional instability, which would reflect normal patterns of emotional variation. However, when emotional instability reaches a particular threshold, it leads to increased depression vulnerability.

High emotional instability coupled with high emotional inertia has been linked to depression in the typically developing population (Koval et al., 2013); however, this theory remains untested in the ASD population. In contrast to trait emotion, intra individual state emotion reflects one's emotional regulation in response to everyday contexts and situations. Given that autistic individuals are believed to have impaired emotional regulation (Mazefsky et al., 2013), we believe that the network theory may provide insight into the depressive profile of autistic adolescents. While it may seem that inertia of emotions may be protective against emotion instability, researchers have found that both features are present among depressed individuals (Kuppens et al., 2010). The combination of negative emotional inertia and instability reflects a non-adaptive emotional pattern "that reaches more extreme emotional intensities and involves relatively large moment-to-moment fluctuations, but at the same time shows a stronger self-predictive lingering effect that makes the emotion slower to recover or be pulled back to a normative state" (Houben et al., 2015, p. 922).

Ecological momentary assessment (EMA) is an intensive-sampling longitudinal research approach that is well-suited to understand micro-level changes in emotion among autistic adolescents. While EMA approaches are varied in their design, they all share three common characteristics. First, all EMA assessment involve sampling a behavior or phenomenon over multiple occurrences. In our effort to understand daily mood fluctuations, it is important to assess multiple mood shifting events. Second, subjects are sampled in their natural environments. Lastly, EMA aims to minimize recall bias by assessing a targeted phenomenon as close to the event as possible (see Shiffman et al., 2007 for a review). Because autistic individuals are known to have less accurate recall of autobiographical information than the typically developing population (Bowler et al., 2000, 2004; Toichi & Kamio, 2002), this feature of EMA may be particularly advantageous for this group. Although, EMA has rarely been employed with the ASD population, it has been used to measure positive emotions (Kovac et al., 2016), social experiences (Chen et al., 2014, 2017), emotional regulation (Cai et al., 2020), and coping (Khor et al., 2014).

This study sought to address the observed gap in understanding depressive symptomatology in ASD. We utilized EMA to accomplish the following aims: (1) identify both state (momentary) and trait factors that are protective of depressive symptomatology by eliciting positive affect and

factors that are risk factors for depressive symptomatology by eliciting negative affect, (2) evaluate and characterize the role of adolescent emotional instability and emotional autocorrelation in predicting parent-reported adolescent depression.

Method

Participants

Participants were 17 male autistic adolescents aged 11–17 years ($M = 14$ years, $SD = 2$ years) (see Table 1). Participants were recruited via The University of North Carolina at Chapel Hill ASD Research Registry. Registry participants are referred through collaborative relationships with other university affiliated institutions that serve individuals with autism. To qualify for the research registry, all participants had undergone extensive observation and clinical testing for an autism diagnosis, including gold-standard diagnostic assessments and interviews such as the ADI (define and cite), ADOS (define and cite), and CARS (define and cite). Inclusion criteria were as follows: participants resided in Central and Eastern North Carolina, confirmed clinical diagnosis of ASD, had a composite abbreviated intelligence quotient greater than 85, spoke fluent English, and had regular access to a mobile phone. Participants with co-occurring genetic syndromes were excluded from the study. Females were excluded due to the small sample size, the observed prevalence of ASD being much higher in males than females (1:4), and the exploratory nature of this study.

Procedure

The Institutional Review Board of The University of North Carolina at Chapel Hill granted ethical approval for this research study. Parents were informed about the study through an ASD research registry, flyers posted on social media, and a central database of university-affiliated employees/students. Parents from the research registry mailed in a postcard indicating interest in the study and giving the researcher permission to contact them. Interested parents were then contacted and invited for participation. All participants were evaluated by the first author and a research assistant (Fig. 1).

During the initial screening visit, participants completed the Stanford-Binet Intelligence Test, fifth edition (Roid, 2003) and a set of demographic questionnaires and assessments. Caregivers also completed a set of demographic questionnaires about themselves and their child.

Table 1 Participant demographics

	Mean (SD)
Child age in years	14 (2)
Number of surveys completed of 42	31.53 (9.73)
Co-occurring diagnoses	ADHD (n = 15) Depression (n = 8)
ABIQ	106.18 (14.60)
CDI 2: SR Total	12.65 (8.75)
Participants with t-score above 60 based on CDI 2:SR ^a	4 (24%)
CDI 2: PR Total	18.00 (6.05)
Participants with t-score above 60 based on CDI 2:PR ^a	9 (53%)
Std. differences in CDI (S/P)	− 0.59 (0.31)

Std. differences in CDI (S/P) is calculated as the standardized CDI 2: SR sum score divided by the standardized CDI 2: PR score

ABIQ abbreviated intelligence quotient, *CDI 2 SR* child depression inventory second edition: self report, *CDI 2 PR* child depression inventory second edition: parent report

^aNumber of participants (percentage)

Participant characteristics are reported in Table 1. Once eligibility was established.

Measures

To characterize our sample, we administered clinical assessments of depression, cognitive ability, parent-rated autism severity, and adaptive behavior. Participants also completed a mobile assessment questionnaire, delivered as an EMA, providing us further information about the participants.

Stanford-Binet Intelligence Test, Fifth Edition (SB-5)

All participants were administered the abbreviated SB-5 (Roid, 2003) by a graduate student or undergraduate research assistant to determine verbal, nonverbal, and general IQ (Table 2) to verify inclusion criteria. Participants completed the object series/matrices subtest, to assess nonverbal fluid reasoning, and the vocabulary subtest, to assess verbal knowledge. Raw scores for verbal knowledge and nonverbal fluid reasoning were summed and standardized to generate abbreviated IQ (ABIQ) scores (Fig. 1).

Child Depression Inventory 2nd Edition: Self Report (CDI 2: SR)

The CDI 2:SR is a standardized and clinically-validated self-report measure of depressive symptoms (Kovacs, 2010; Saylor et al., 1984) that has been used in the ASD population (Mazefsky et al., 2011). The 27 items of the CDI 2: SR are scored on a scale of 0–2 (anchored by behavior

frequency, e.g., I have trouble sleeping many nights, every night, or I sleep pretty well) and was divided into four factors including: negative mood/physical symptoms, negative self-esteem, interpersonal problems, and ineffectiveness. The CDI 2: SR was originally developed to be a measure of depression severity and not as a diagnostic instrument.

Child Depression Inventory 2nd Edition: Parent-Report (CDI 2: PR)

The CDI 2: PR is a 17-item, Likert-type, parental or primary caregiver reported measure of a child's depressive symptoms (Kovacs, 2010). The CDI 2: PR items align with items on the self-report version of the CDI 2:SR. Respondents indicate if a behavior (i.e., crying) is observed in their child—not at all, a little, some of the time, moderately, or a lot. Similar to the CDI 2: SR, the CDI 2: PR was also developed to be a measure of depression severity and not as a diagnostic instrument. Additionally, the CDI 2 has been used previously to understand depressive symptomatology in this population (Gotham et al., 2015).

Pubertal Developmental Scale (PDS)

In addition, parents completed the Pubertal Development Scale (PDS; Petersen et al., 1988) to assess the development of secondary pubertal development characteristics, such as body hair growth, skin changes, and voice changes. Parents completed the five-item measure, indicating on a four-point scale ranging from: not yet begun to complete. The PDS was originally developed as a self-report measure of pubertal development; however, it also has been used as a parent-report pubertal development measure (Muscatello & Corbett, 2018). While parents were the ones completing the measure, we encouraged all parents to consult with their child to confirm their answers. Given that depression has been found to be related to pubertal status, and not age, in adolescents (Mendle et al., 2010), pubertal status will be used as a potential predictor variable in data-driven approaches and as a covariate in theory-driven analyses.

Positive and Negative Affect Scale, Children's Version, Short Form (PANAS-C-SF)

Adolescents completed the positive and negative affect scale, short form (PANAS-C-SF) (Sanmartín et al., 2018) to assess momentary positive affect and negative affect. Adolescents completed the 10-item measure, indicating on a four-point scale to what extent they are experiencing a particular emotion: none at all, a little, some, a lot.

Table 2 Fixed effect parameter estimation—positive score

Predictor variable	Estimate	Std. error	DF	p value
Positive				
Intercept	− 3.26	37.36	466	0.93
Day	− 0.02	0.02	466	0.23
Level of current social interaction	0.14	0.04	466	<0.01*
Quality of current social interaction	0.13	0.01	466	<0.01*
Enjoyment of current activity	0.02	0.01	466	0.11
Duration of assessment	0	0	466	0.79
Verbal IQ	− 0.38	2.74	7	0.89
Non-verbal IQ	− 0.54	2.67	7	0.85
IQ	0.12	0.91	7	0.9
Genetic sibling with ASD	0.66	0.61	7	0.32
Parent-reported overall health	− 0.77	0.26	7	<0.02*
Parent-reported pubertal skin changes	0.54	0.6	7	0.39
Parent-reported pubertal hair changes	1.34	0.46	7	0.02*
Parent-reported pubertal voice changes	− 0.96	0.51	7	0.1
Negative				
Intercept	− 13.9	22.49	466	0.5368
Day	− 0.002	0.01	466	0.8060
Level of social interaction	0.02	0.02	466	0.5280
Quality of social interaction	− 0.04	0.01	466	<0.01*
Enjoyment of current activity	0.005	0.01	466	0.4654
Duration	0	0	466	0.2402
Verbal IQ	− 1.23	1.65	7	0.4794
Non-verbal IQ	− 1.24	1.61	7	0.4646
ABIQ	0.42	0.55	7	0.4726
Genetic sibling with ASD	− 0.12	0.37	7	0.7520
Parent-reported overall health	0.05	0.16	7	0.7664
Parent-reported pubertal skin changes	− 0.3	0.36	7	0.4286
Parent-reported pubertal hair changes	− 0.45	0.28	7	0.1480
Parent-reported pubertal voice changes	0.31	0.31	7	0.3417

For brevity, only 14 variables for positive momentary affect and negative momentary affect (total of 28 variables) are presented. These variables were chosen to be presented because they explained the most variance in depressive symptomatology

Asterisk indicates a p-value significant at the 0.05 level

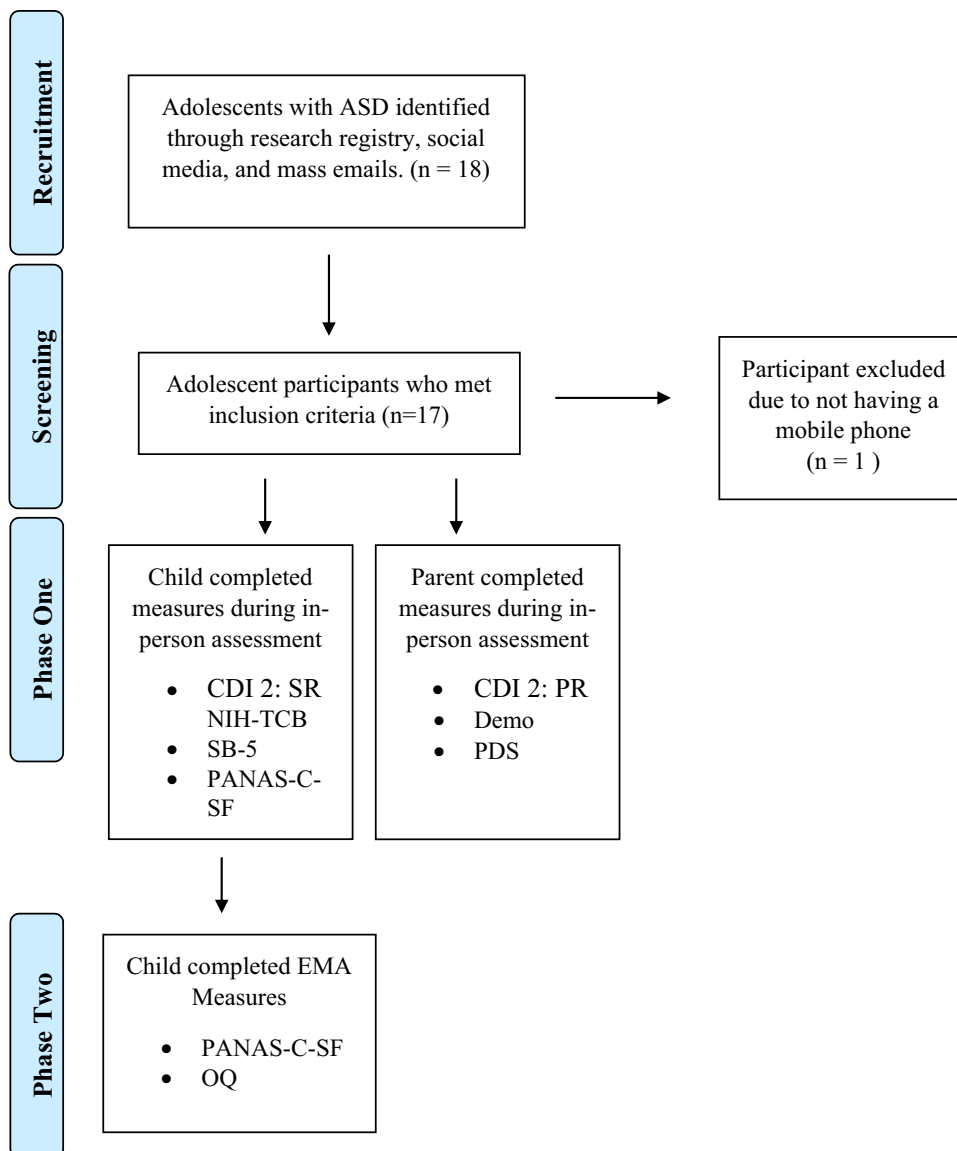
Ecological Momentary Assessment Procedure

Adolescents completed practice EMA procedure prior to the start of the EMA protocol. EMA is an intensive sampling method in which participants complete the same assessments at multiple times (Stone & Shiffman, 1994). Once reliability was established, adolescents and their parents scheduled a week that mirrored a *typical* week for the adolescent (i.e. not travelling). During this week, adolescents were signaled six times a day, semi-randomly for seven consecutive days to complete a questionnaire. In this study, to reduce the chances of participants anticipating prompts, each participant's initial prompt was sent semi-randomly within 2 h of their parent-reported waketime. Subsequent prompts were sent two hours after the initial message. Because the start time for each day was semi-randomly generated, we believe that participants

were unlikely to anticipate when they would receive messages each day. Participants received a total of 42 messages (six messages per day for 7 days).

Adolescents completed an electronic questionnaire that was sent to them via text message link. Each questionnaire included multiple surveys. The first survey was adapted from previous EMA studies (Kovac et al., 2016). Participants completed the positive and negative affect scale, short form (PANAS-C-SF) (Sanmartín et al., 2018) and a brief questionnaire about their current activities. The questionnaire included questions such as: “what were you doing the moment you were beeped”, “how many people are around you” (used here as an index of self-reported level of social interaction), and “how would you rate your enjoyment of your current social interactions” (used here as an index of self-reported quality of social interaction).

Fig. 1 Study procedures



Statistical Analyses

Using a data driven approach, we identified predictors of momentary affect. We generated random forests in R (Version 3.5.3), inputting all 114 numeric variables (items from PDS, CDI 2: P, CDI 2: SR, demographic questionnaires, and the mobile assessment questionnaire) as predictors to model the importance of each variable to current positive affect scores and negative affect scores separately. We chose individual items, as opposed to subscale or total scores, to balance the depth of assessment and data sensitivity. Then we selected the 10 variables with the highest relative explained variance to identify the most important predictors of momentary positive affect and negative affect. Next, we used these variables as predictors to fit a linear mixed effects model for momentary mean positive scores and momentary

mean negative scores separately, with a random intercept for each participant to understand both risk and protective factors of depressive symptomatology.

To examine the impact of the pubertal stage on depression status we create a summary pubertal status indicator by summing all pubertal development items. We categorized pubertal development by scoring those who were -1 standard deviation from the mean on the total PDS score as ‘early stage’ and those who were $+1$ from the mean PDS score as ‘late stage’ (Smith-Woolley et al., 2017). For all analyses, we used average as the reference category. Once again, we generated random forests to determine the relative variable importance and explained variance, of all numeric variables with the new categorical pubertal development variable. We subsequently fit a linear mixed effects model for momentary mean positive scores and momentary mean negative scores

separately, with the pubertal stage as a predictor and a random intercept for each participant.

Lastly, we took a theory driven approach to estimate the importance of mean square successive difference (MSSD) (see Thompson et al., 2012; von Neumann et al., 1941) and the autocorrelation of emotions to parent-reported depressive symptomatology. We use parent-reported depressive symptomatology, as opposed to child-reported depressive symptomatology because parents are believed to be better reporters of depressive symptoms (Lewis et al., 2012; Mazefsky et al., 2011; Strang et al., 2012). In this approach, we first examined the variance in momentary mean positive affect and mean negative affect. The variable with the greatest variance, mean positive affect, was chosen as the predictor for subsequent analyses. Then we fit a linear model in which MSSD of positive affect predicts parental reported depression levels for each participant, controlling for pubertal status. Since researchers have suggested a non-linear relationship between these variables (Houben et al., 2015), we explored linear, quadratic, and cubic relationships between MSSD and parent-reported depressive symptomatology using a mixed effects model, controlling for the pubertal development stage. We evaluated the model's fit using adjusted R^2 (R^2_{adj}) and Akaike's information criterion (AIC) of model evaluation (Akaike, 1998), with higher R^2_{adj} and lower AIC interpreted as better model fit. Finally, we characterized the relationship between the autocorrelation of positive affect and parental reported depression levels by using a mixed effects model, by controlling for the pubertal development stage and fitting linear, quadratic, and cubic models. Once again, we evaluated model fit using R^2_{adj} and AIC.

Results

Predictors of Momentary Affect Based on Random Forest

From the random forest, the nine variables with the greatest impact on momentary positive affect included both stable and time-varying variables. Of these nine, five stable variables had the greatest importance for momentary positive affect score: *non-verbal IQ*, *verbal IQ*, *abbreviated IQ*, *parent-reported overall child health*, and *parent-reported pubertal skin changes*. The following four time-varying variables also impacted momentary positive affect: *quality of social interaction*, *enjoyment of the current activity*, *level of social interaction*, *survey completion time (seconds)*. Among these ten variables, the level of current social interaction ($p < 0.01$), quality of current social interaction ($p < 0.01$), parent-reported overall health ($p < 0.01$), and parent-reported

hair changes ($p = 0.02$) have statistically significant effects on participants' momentary positive affect.

From the random forest, the following seven stable variables had the greatest importance for momentary negative affect: *nonverbal-IQ*, *presence of an ASD sibling*, *parent-reported pubertal hair changes*, *parent-reported pubertal changes related to skin*, *parent-reported pubertal changes related to voice*, *degree of sadness over the past two weeks*, and *medications*. The following three time-varying variables had the greatest importance for momentary negative affect: *duration of the assessment*, *enjoyment of the current activity*, *level of present social interactions*. The only significant time-varying predictor of momentary negative affect was *the quality of current social interaction* ($p < 0.01$). Poorer quality of social interaction corresponds to a higher negative score, which is consistent with the estimation result for positive scores (Table 2).

In our next model, we examined the effect of overall current pubertal status on momentary positive and negative affect. For momentary positive score endpoints, significant effects (p value < 0.05 ; Table 2) were detected on the interaction of the level of social interaction and pubertal status. This interaction indicates that for participants in early pubertal stages, higher levels of social interaction corresponds to higher PA scores. The interaction between the quality of social interaction and late stage pubertal status indicates that for participants with "late" sum PDS scores, responses of higher quality social interaction corresponded to higher endpoint positive scores. This finding suggests that puberty status may play an important role in differentiating risk factors for negative affect (Table 3).

As expected, for momentary negative affect scores, significant effects (p value < 0.05) were detected for the interaction between quality of social interaction and late pubertal status (negative estimated coefficient). This suggests that for participants with later pubertal development, a higher score on the quality of social interaction corresponded to lower negative endpoint score.

Predictors of Parent-Reported Depressive Symptoms

We estimated a significant positive relationship between positive affect instability (as estimated by MSSD of positive affect) and parent-reported depression symptomatology. The cubic model ($R^2_{\text{adj}} = 0.45$, $AIC = 41.1$; see Fig. 2) fit the data better than either the linear model ($R^2_{\text{adj}} = 0.17$, $AIC = 46.2$) or the quadratic model ($R^2_{\text{adj}} = 0.25$, $AIC = 45.3$). Autocorrelation of positive affect, however, was not related to parent-reported depressive symptomatology when we fit a linear ($R^2_{\text{adj}} = 0.16$, $AIC = 46.36$), quadratic ($R^2_{\text{adj}} = 0.16$, $AIC = 46.49$), or cubic model ($R^2_{\text{adj}} = 0.16$, $AIC = 48.36$).

Table 3 Fixed effect parameter estimation, including pubertal status—positive score

Predictor variable	Estimate	SE	DF	p value
Positive				
Intercept	− 34.53	84.42	460	0.68
Level of social interaction	0.03	0.05	460	0.56
Quality of social interaction	0.13	0.02	460	<0.01*
Enjoyment of current activity	0.04	0.01	460	<0.01*
Non-verbal IQ	− 2.74	6.07	7	0.66
IQ	0.91	2.07	7	0.67
Genetic sibling with ASD	0.35	0.93	7	0.72
Pubertal status—early stages	− 0.25	1.09	7	0.83
Pubertal status—late stages	− 1.58	1.16	7	0.22
Level of social interaction × Pub. status—early	0.53	0.11	460	<0.01*
Level of social interaction × Pub. status—late	0.07	0.11	460	0.49
Quality of social interaction × Pub. status—early	− 0.04	0.03	460	0.16
Quality of social interaction × Pub. status—late	0.09	0.03	460	<0.01*
Enjoyment of current activity × Pub. status—early	− 0.04	0.03	460	0.17
Enjoyment of current activity × Pub. status—late	− 0.02	0.03	460	0.48
Negative				
Intercept	− 25.68	32.05	460	0.42
Level of social interaction	0.06	0.03	460	0.05*
Quality of social interaction	− 0.03	0.01	460	<0.01*
Enjoyment of current activity	− 0.01	0.01	460	0.15
Duration	0	0	460	0.29
Genetic sibling with ASD	0.32	0.35	7	0.4
Pubertal status—early stages	0.48	0.43	7	0.31
Pubertal status—late stages	0.63	0.46	7	0.21
Number of medications	− 0.19	0.32	7	0.57
Degree of sadness over the past two weeks	− 0.08	0.36	7	0.83
Level of Social Interaction × Pub. Status—Early	− 0.15	0.06	460	0.02*
Level of social interaction × Pub. status—late	− 0.1	0.06	460	0.11
Quality of social interaction × Pub. status—late	− 0.07	0.02	460	<0.01*
Enjoyment of current activity × Pub. status—early	0.03	0.01	460	0.03*
Enjoyment of current activity × Pub. status—late	0.04	0.02	460	0.06

For brevity, only 14 variables for positive momentary affect and negative momentary affect (total of 28 variables) are presented. These variables were chosen to be presented because they explained the most variance in depressive symptomatology

Asterisk indicates a p-value significant at the 0.05 level

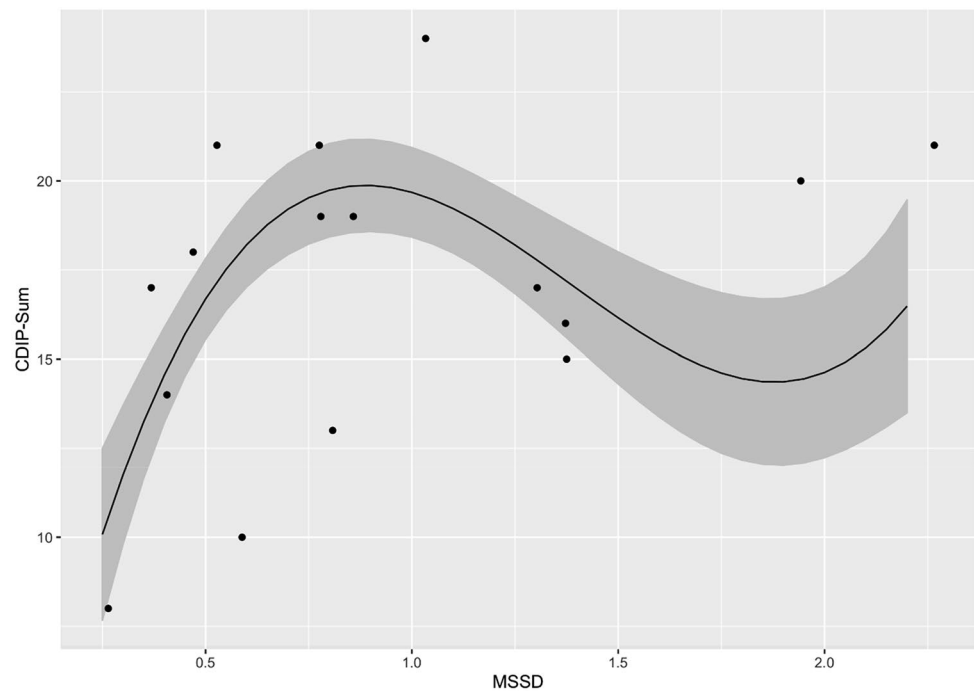
Discussion

In the present study, we identified risk factors for both momentary negative affect and depressive symptomatology in autistic adolescents. We also identified protective factors (i.e., factors that promote positive affect) that could be leveraged in the development of future interventions targeting depression in autistic adolescents.

Identifying risk factors that can increase momentary negative affect is especially important given that long periods of negative affect are a trans diagnostic symptom implicated in depression (APA, 2013). For study participants, reduced quality of social interaction had significant effects on their

momentary negative affect. In light of the two-hit model of ASD, we believe that this suggests social experiences are a key differentiating factor in the onset of depression. To our knowledge, this is the first study directly investigating the impact of negative social experiences on depressive symptomatology, though researchers have previously hypothesized this relationship (Chandrasekhar & Sikich, 2015). While increased perceived impairments and lower perceived social support had been linked to depression, the role of negative social experiences in predicting perceived social deficits remains unclear. It may be that individuals who have social experiences they perceive as negative attribute these negative experiences to their ASD diagnosis. They may then anticipate future social experiences will be negative, which

Fig. 2 Relationship between instability and depressive symptomatology. *MSSD* mean square successive difference of positive affect, *CDIP-Sum* child depression inventory second edition: parent report sum of all items



may lead to a cycle of depressive symptoms. The findings of this study suggest that autistic individuals who report feeling negative social experiences often should be further screened for potential depressive symptomatology. The development of screening tools that evaluate subjective social experiences for this population may thus be an important step in identifying those at risk for depression.

Social experiences play a significant role in the manifestation of depression for individuals on the autism spectrum (Day et al., 2019). While negative social experiences can be a risk factor for depression, we found that positive social experiences can serve as protective factors in preventing depressive symptomatology by leading to positive affect. For participants in early pubertal stages, higher levels of social interaction (i.e., being around more people) was related to positive affect. This suggests that for younger adolescents, being around more people might help decrease opportunities for negative affect. However, for those at later pubertal stages, only the quality of social interaction was identified as a significant predictor. This suggests that the types of friendships that are protective of depressive symptomatology may change throughout adolescence and interventions should be responsive and tailored to these developmental periods. Previous studies have found that younger adolescents perceive equal social support coming from both parents and friends, while older adolescents perceive more support from their friends (Bokhorst et al., 2010). In light of our findings, it may be that younger adolescents seek more friendships, including those provided by the parents, while older adolescents seek intimate friendships with only their close friends.

To further understand the profile of depressive symptomatology in ASD, we verified the network theory of depression. We found that increased affective instability leads to increased parent-reported depressive symptomatology. The significant relationship between emotional instability and depressive symptoms may suggest that reducing the overall variability of emotions may be an important step in targeting depression in this population, regardless of the cause of instability (i.e., ruminative thoughts lead to instability, certain sensory experiences may lead to instability, or increased perceptions of ASD related social deficits). Further, this suggests that emotional instability, as manifest by high variations in emotions, may be a key factor in the emergence of depressive symptomatology in the ASD population. While future research will be needed to explore to what extent emotional instability is normal and expected versus unhealthy, these findings suggest that emotional instability may be an appropriate intervention target in future intervention research.

While it is important to interpret these results with caution, our finding of the cubic relationship between emotional instability and depressive symptomatology has important implications for emotional regulation research. While more research is needed to determine exact cutoffs, our findings suggest there may be an optimal level of emotional instability in reducing depressive symptoms. This would indicate that ideal emotional regulation is characterized by subtle fluctuations in emotional variability (i.e., not happy all the time, sad all the time, nor characterized by extreme patterns of emotional variation). Instead, subtle variations may be a

key factor in emotional functioning patterns that are protective of depressive symptomatology. Although more research is needed to verify this hypothesis in larger samples and in other populations, we believe that, in general, child-reported emotional stability may serve as a useful target and outcome measure in intervention studies.

We were surprised to find that autocorrelation of affect did not predict depressive symptomatology in this population. This finding is in contrast to previous literature, which suggests that autocorrelation of affect is a hallmark feature of depressive symptomatology (Wichers et al., 2016). Our failure to find a significant effect may be related to our small sample size. It may also be that the emotional regulation profile in ASD is characterized by greater instability than in other clinical populations associated with depressive disorders, leading to a decrease in affective autocorrelation. This would suggest that autistic individuals experience depression differently, and consequentially, researchers must use caution in the application of depression measures normed on other populations to the ASD population.

Limitations

There are several limitations to the present study. The small sample size of this study makes these findings exploratory and future studies should verify the findings from the present study using a larger sample size. In particular, studies should concentrate on the recruitment of samples from diverse socioeconomic backgrounds. A limitation of the present study is that adolescents were required to have regular access to a mobile phone to participate in EMA procedures. This required participants to have sufficient economic resources to own a phone, and thus, these results cannot be interpreted as describing the full population. Further, females were excluded from this study. Future studies should investigate emotional patterns among autistic females, especially given reports of greater depression in females (Gotham et al., 2015). The present study is also limited in that no formal diagnostic assessments were administered to confirm ASD diagnosis. All participants were recruited from an IDDRC-funded ASD registry that requires a clinical diagnosis via gold-standard assessments for membership, therefore these were not repeated for this study. Approximately 50% of parents returned social communication questionnaires (SCQ; Chandler et al., 2007) following participation, however due to the COVID-19 pandemic, the remaining 50% were not returned. Due to the swift shift to remote schooling and working from home, we did not re-contact families for outstanding forms during this stressful time. However, future studies may benefit from the use of gold-standard diagnostic instruments, such as the ADOS-2 (Lord et al., 2012), to examine how autism symptom presentation and severity may impact depressive symptomatology. Likewise, we

used parent-reported pubertal status, which is preferred over self-reported pubertal status, but less reliable when compared to physician administered assessments (Rasmussen et al., 2015). Future studies should augment parent- and self-reported pubertal status with physician-measured pubertal status. Additionally, this study was limited in that we only included autistic individuals and an average ABIQ. It remains unclear to what extent individuals with lower IQs may experience depression like symptoms in comparison to their higher-functioning peers. Moreover, the absence of a control group prevents us from indicating whether these findings are limited to the ASD population or characteristics of adolescence in general.

Conclusions

In this study, we identified risk factors for depressive symptomatology in autistic adolescents. We found that social experiences are implicated in the mental health of autistic adolescents. For adolescents at earlier pubertal stages, larger social networks and friend groups may be important for positive mental health. However, as adolescents age, it may be important for them to develop deeper, more meaningful friendships. The type of friendships that promote optimal psychological functioning may shift throughout the lifespan, which would suggest that the types of skills needed to develop these friendships are also likely to change. Therapists who aim to optimize adolescents' mental health must consider what types of friendships are most important for that individual. The social skills required to develop and form large groups are different than those required to foster intimate friendships (Glick & Rose, 2011). Accordingly, interventions should be tailored to meet the specific needs for the types of friendships an autistic adolescents might be seeking.

Acknowledgments We thank Yueting Wang (University of Chapel Hill) for her help designing our statistical plan and completing our statistical analysis. This work was supported by the Organization for Autism Research Graduate Research Grant. Lastly, this research was drawn from the doctoral dissertation of the first author which was completed at the University of North Carolina at Chapel Hill.

Author contributions A.D. conceived of the presented idea. A.B. provided mentorship and guidance for refining the idea by suggesting an increased emphasis on examining participant social experiences. A.D. then developed the theory, recruited participants, collected data, and analyzed the data. A.B. and C.H. supervised the findings of this work. All authors discussed the results and contributed to the final manuscript.

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