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Research paper

Prospective predictors of first-onset depressive disorders in adolescent females with anxiety disorders

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ABSTRACT

Background: Anxious youth are at increased risk for later depressive disorders, but not all anxious youth develop depression. Sequential comorbidity models emphasize shared risk factors and anxiety sequelae, but some anxious youth who later develop depression may have risk factors that are relatively specific to depression, in addition to a liability to anxiety. We examined several variables that appear relatively specific to risk for depression—the personality traits of low positive affectivity and high sadness, and an electrophysiological measure of blunted response to reward - in predicting first-onset depressive disorders and depressive symptoms in clinically anxious adolescent girls.

Methods: A sample of 114 adolescents with baseline anxiety disorders completed personality and psychopathology measures, psychophysiology tasks, and diagnostic interviews. Interviews and a measure of depressive symptoms were re-administered over 27 months.

Results: After controlling for baseline depressive symptoms, blunted reward sensitivity uniquely predicted first-onset depressive disorders and depressive symptoms 27 months later. Post-hoc analyses indicated that blunted reward sensitivity only predicted first-onset depressive disorders and depressive symptoms in girls with high social anxiety symptoms.

Limitations: Analyses were unable to account for concurrent anxiety symptoms and disorders.

Conclusions: The depression-specific risk factor, blunted reward sensitivity, may comprise one pathway to subsequent depressive disorders and symptoms in anxious youth and indicate which anxious youth need intervention to prevent later depression, particularly in socially anxious girls.

Depressive and anxiety disorders are common in youth, particularly in females, and associated with significant impairment (e.g., school failure, social difficulties, substance use, and suicide) (Cummings et al., 2014; Merikangas et al., 2010; Rohde et al., 2013). Depressive and anxiety disorders commonly co-occur (Cummings et al., 2014; Schleider et al., 2014). Three-quarters of depressed adolescents have comorbid anxiety disorders (Avenevoli et al., 2001; Kessler et al., 2001) and over half of anxious adolescents have comorbid depressive disorders (Lewinsohn et al., 1997). This comorbidity is often sequential, with anxiety disorders frequently preceding the onset of depression (Costello et al., 2005; Cummings et al., 2014; Jacobson and Neuman, 2017). Indeed, the onset of anxiety disorders is often in childhood (Beesdo et al., 2009), whereas depressive disorders do not typically emerge until adolescence (Rohde et al., 2013). There is some evidence that girls may be more likely to develop later depression following the onset of anxiety disorders than boys (e.g., Costello et al., 2003; Keenan & Hipwell, 2005), although findings have been mixed (e.g., Breslau et al., 1995; Gallerani et al., 2010; Pine et al., 1998; Vaananen et al., 2010). Because childhood anxiety disorders are also more common in girls (Beesdo et al., 2009), several authors (e.g., Bittner et al., 2004; Breslau et al., 1995; Silk et al., 2012) have suggested that increased risk for later depression in anxious girls may contribute to sex differences in depression in early adolescence (Salk et al., 2017).

Although youth with anxiety disorders are at substantially increased risk for later depressive disorders, many do not develop depression (Costello et al., 2005; Costello et al., 2003). Comorbid depression in youth with primary anxiety disorders is associated with substantially worse psychosocial functioning than anxiety alone (Cummings et al., 2014). Further, prevention of anxiety disorders does not reduce depressive symptoms (Garber et al., 2016). Therefore, identifying which

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anxious youth will develop depressive symptoms and disorders may contribute to understanding the heterogeneity of anxiety and depression and allow for more targeted prevention of depression in at-risk youth.

Theoretical models of sequential comorbidity for later depression have emphasized two pathways, that a) shared risk factors contribute to both anxiety and depression and b) that greater severity of anxiety or its consequences lead to later depression (Cummings et al., 2014; Garber and Weersing, 2010; Schleider et al., 2014; Silk et al., 2012). Empirical support has emerged for both of these pathways to later depression. Studies examining shared risk factors have demonstrated that cognitive (e.g., repetitive thinking, attentional avoidance of threat; McLaughlin and Nolen-Hoeksema, 2011; Price et al., 2016; Starr et al., 2016), behavioral (e.g., avoidance; Jacobson and Newman, 2014), and interpersonal (e.g., low sociability, interpersonal oversensitivity, chronic interpersonal stress, feeling unloved or unaccepted in interpersonal relationships; Jacobson and Newman, 2016; Starr et al., 2014) factors predict the sequential comorbidity of anxiety and depressive symptoms and disorders in adolescents and young adults. In addition, researchers have reported that panic attacks (Beesdo et al., 2007), hopeless cognitions about anxiety symptoms (Starr et al., 2016), and severe functional impairment from anxiety (Bittner et al., 2004) predict later depressive symptoms and disorders in anxious adolescents and young adults.

A third potential pathway that has received less theoretical and empirical attention is that some anxious youth may also have depression-specific vulnerabilities. Cummings et al. (2014) raised the possibility of depression-specific predictors contributing to concurrent comorbidity, but did not discuss this potential pathway in the context of sequential comorbidity. Two sets of risk factors for depression that are fairly distinct from anxiety include selected facets of personality and low reward sensitivity. Personality traits, such as extraversion and neuroticism, are relatively stable and have been well established as predictors of later depressive disorders (Klein et al., 2011). Although these broad personality constructs also relate to anxiety (Khazanov and Ruscio, 2016; Kotov et al., 2010), there is emerging evidence that some facets of extraversion and neuroticism are unique to depression. Low positive affectivity (PA) is a facet of extraversion that has generally been shown to distinguish anxiety from depression (Naragon-Gainey et al., 2009; Klein et al., 2011; Watson et al., 2015). Low PA predicts increased risk for depression in adolescents prospectively (Goldstein et al., 2017; Neumann et al., 2011). Trait sadness, or depressivity, is a facet of neuroticism that also appears to be specific to depression (Rector et al., 2012), and prospectively predicts higher depressive symptoms and disorders (Goldstein et al., 2017; Klein et al., 2011; Naragon-Gainey and Watson, 2014; Zinbarg et al., 2016).[1] However, neither low PA nor sadness have been investigated as predictors of which anxious youth will develop depression.

Anhedonia and diminished sensitivity to reward are considered core features of depression, but are not generally viewed as integral to anxiety (Kujawa and Burkhouse, 2016; Olino, 2016). Low or blunted reward sensitivity has been consistently associated with depression using both functional magnetic imaging and event-related potential (ERP) methods (Kujawa and Burkhouse, 2016; Stringaris et al., 2015). In contrast, some studies find relationships between enhanced neural reward sensitivity and anxiety (Kessel et al., 2015; Shechner et al., 2012). For these reasons, blunted neural response to reward may distinguish which anxious youth become depressed (Silk et al., 2012); however, there have been no empirical investigations of this premise to date.

[1] Although there is some overlap between personality traits and depressive symptoms and disorders, they differ in their time-course of change. Personality and its facets, while relatively stable in terms of rank-order stability (Roberts & DelVecchio, 2000), change gradually over the lifespan (Roberts et al., 2006). In contrast, depressive symptoms and disorders change much more rapidly, and are thus, less stable over shorter intervals, such as weeks or months, and more episodic in nature (Klein et al., 2011).

The reward positivity (RewP) is an ERP component that is sensitive to the difference between reward and non-reward. A blunted RewP is associated with greater depressive disorders and symptoms (Belden et al., 2016; Bress et al., 2012), but is unrelated to anxiety in youth (Bress et al., 2015). Several studies have also demonstrated that a blunted RewP prospectively predicts first onsets of depressive disorders in adolescents (Bress et al., 2013; Bress et al., 2012; Nelson et al., 2016).

The present study prospectively examined predictors of first-onset DSM-IV depressive disorders and depressive symptoms in a sample of 114 adolescent females with lifetime DSM-IV anxiety disorders who were assessed at baseline and re-evaluated every 9 months for 27 months. We focused on girls due to their greater risk for anxiety and depression (e.g., Beesdo et al., 2009; Costello et al., 2003; Rohde et al., 2013; Salk et al., 2017). Further, we utilized a sample with anxiety disorders to enhance the clinical relevance of the study for secondary prevention of depression in anxious youth (Cummings et al., 2014; Eaton et al., 1995; Kessler and Price, 1993). Based on existing gaps in the literature on sequential comorbidity for later depression in youth, we examined a potential depression-specific risk pathway. We hypothesized that depression-specific risk factors, including low PA, high sadness, and a blunted RewP, would predict first-onset depressive disorders and depressive symptoms in adolescent girls with anxiety disorders. As a comparison, we also examined whether greater severity of anxiety, current anxiety disorder, and anxiety-related characteristics predicted increased risk for depression. In addition, we also included personality and electrophysiological predictors that are more strongly associated with risk for anxiety than depression for comparison purposes, including trait anxiousness, a facet of neuroticism (Goldstein et al., 2017; Zinbarg et al., 2016), and the error related negativity (ERN), an ERP component elicited by errors on speeded response tasks that is associated with risk for anxiety symptoms and disorders (Bress et al., 2015; Hajcak, 2012; Meyer et al., 2015).

1. Method

1.1. Participants

Participants were 114 adolescent girls with lifetime DSM-IV anxiety disorders recruited through the adolescent development of emotions and personality traits (ADEPT) study (see Nelson et al., 2016 for more details on the full sample and recruitment procedures). Briefly, the full sample included 550 adolescent females aged 13–15 years (M = 14.4, SD = 0.63) who were recruited using commercial mailings and postings and by word of mouth. Girls were eligible to participate in the study if they had a biological parent willing to participate, were fluent in English, were able to complete questionnaire measures, did not have an intellectual disability, and did not have a history of either major depressive disorder or dysthymia. Families received financial compensation for their participation. All procedures were approved by the Stony Brook University Institutional Review Board.

The present sample included 114 adolescent females (Mage = 14.3 years, SD = 0.59) who met criteria for lifetime DSM-IV anxiety disorders at baseline, as assessed by a semi-structured diagnostic interview (described below). As shown in Table 1, lifetime anxiety disorders at baseline included Specific Phobia (N = 55, 48.2%), Social Phobia (N = 46, 40.4%), Generalized Anxiety Disorder (N = 14, 12.3%), Separation Anxiety Disorder (N = 11, 9.6%), Anxiety Disorder Not Otherwise Specified (N = 13, 11.4%), Obsessive-Compulsive Disorder (N = 6, 5.3%), Panic Disorder (N = 2, 1.8%), and Agoraphobia (N = 2, 1.8%). There were no cases of Posttraumatic Stress Disorder. Twenty-eight participants (24.6%) had two or more lifetime anxiety disorders. The majority of participants had at least one current anxiety disorder diagnosis at baseline (N = 101, 88.6%). Most participants were non-Hispanic Caucasian (79.8%), lived with both parents (83.3%), and had a parent with at least a bachelor's degree (67.3%).

Table 1
Baseline participant characteristics.

Baseline variable	N (%)
Age *M* (*SD*)	14.3 (0.59)
Lifetime DSM-IV anxiety disorders	
Specific phobia	55 (48.2%)
Social phobia	46 (40.4%)
Panic disorder	2 (1.8%)
Separation anxiety	11 (9.6%)
Agoraphobia	2 (1.8%)
Generalized anxiety disorder	14 (12.3%)
Posttraumatic stress disorder	0 (0.0%)
Obsessive-compulsive disorder	6 (5.3%)
Anxiety disorder not otherwise specified	13 (11.4%)
Current DSM-IV anxiety disorder diagnoses	101 (88.6%)
Race/Ethnicity	
non-Hispanic Caucasian	91 (79.8%)
Hispanic	14 (12.3%)
Hispanic Caucasian	7 (6.1%)
Hispanic black	2 (1.7%)
Hispanic other	5 (4.4%)
American Indian	1 (0.9%)
Asian	1 (0.9%)
African-American/black	5 (4.4%)
Other	2 (1.7%)
Living with both parents	95 (83.3%)
Parent with at least a bachelor's degree	74 (67.3%)

1.2. Procedure

Participants were assessed every 9 months. They completed all personality, psychophysiology, and psychopathology measures, including the expanded version of the Inventory of Depression and Anxiety Symptoms (IDAS-II) and the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime Version (K-SADS-PL), in the lab at baseline. Participants completed the K-SADS-PL depression module by phone at 9 months (Wave 2) and at 27 months (Wave 4). The IDAS-II was also administered at Wave 4 in an online survey format (www.limesurvey.com). At the 18-month follow-up (Wave 3), participants completed the K-SADS-PL in person. Each follow-up diagnostic interview (both phone and in person) assessed the period since the participant's last diagnostic assessment to reduce the likelihood of missing first onsets of depressive disorders. 87.8% of the full sample (N = 483) was retained over all four waves.

1.3. Psychopathology measures

The expanded version of the Inventory of Depression and Anxiety Symptoms, the IDAS-II (Watson et al., 2012), is a 99-item self-report measure of depression and anxiety symptoms. Its scales were factor analytically derived from a large item pool; it is one of the most comprehensive measures of mood and anxiety symptoms available and has the added advantage of offering nonoverlapping scales. IDAS-II scales scores also correspond well to other self-report measures and interview measures of depression and anxiety (Watson et al., 2012). Participants rate symptoms based on the past two weeks on a 5-point Likert scale (1 = *not at all,* 5 = *extremely)*. We used the 20-item Depression subscale to assess depressive symptoms and the Panic (8 items), Social Anxiety (6 items), Claustrophobia (5 items; assessing agoraphobia and situational phobia symptoms, including small spaces and tunnels), Checking (3 items), Ordering (5 items), and Cleaning subscales (7 items) to assess anxiety symptoms. Internal consistencies for these scales were good to excellent (α = 0.76–0.91) in the full sample.

Lifetime anxiety and depressive disorder diagnoses were assessed using the K-SADS-PL (Kaufmann et al., 1997), a widely used semi-structured diagnostic interview. Interviews were videorecorded and conducted by trained research staff under the supervision of clinical psychologists (R. K., D. N. K., and G. P.). A subset of interviews were rescored by a second interviewer blind to the original diagnoses to evaluate interrater reliability of diagnoses, which ranged from fair (kappa = 0.64; social phobia) to excellent (kappa = 0.91; generalized anxiety disorder) in the full sample. Interrater reliability was adequate for anxiety disorders (kappa = 0.75) and excellent (kappa = 0.81) for depressive disorders in particular. As only a subset of interviews were rated for reliability, the index interviewer's diagnosis was used for consistency, when there was a disagreement.

1.4. Personality measures

Personality facets were assessed using several self-report measures of personality, including the Faceted Inventory of The Five Factor Model (FI-FFM; Naragon-Gainey et al., 2009; Simms, 2009; Watson et al., 2017), the International Personality Item Pool (IPIP; Goldberg et al., 2006), and the Schedule of Nonadaptive and Adaptive Personality (SNAP; Clark, 1993). The FI-FFM Melancholia facet scale (10 items) and the IPIP Sadness scale (10 items) were used as measures of sadness. The FI-FFM Anxiousness facet scale (10 items) and IPIP Anxiousness facet scale (10 items) were included as measures of anxiousness. The FI-FFM Positive Temperament facet scale (8 items), IPIP Cheerfulness scale (10 items), and SNAP Positive Temperament scale (27 items) were used as measures of PA. Internal consistencies ranged from 0.87 to 0.91 for the sadness scales, 0.83–0.86 for the anxiousness scales, and 0.79–0.87 for the positive temperament scales.

Because of high correlations among subscales assessing the same facet (*r*s = 0.58–0.84; mean *r* = 0.74), three personality facet composite scores were created, as these were thought to be capturing the same trait. The Sadness and Melancholia subscales of the IPIP and FFM were used to create the Sadness composite. The Anxiousness scales of the IPIP and FI-FFM were used to create the Anxiousness Composite. Finally, the IPIP Cheerfulness, FFM Positive Temperament, and SNAP Positive Temperament subscales were used to create the PA composite. Subscale scores were first *z*-scored then averaged to calculate composite scores.

1.5. ERP measures

The reward task was administered using Presentation version 17.2 (Neurobehavioral Systems, Albany, Calif.). Participants were instructed to select one of two doors, in which they could either gain $0.50 or lose $0.25, by clicking the corresponding left or right mouse button. Participants could win a maximum of $5. In each trial, two identical doors were presented until participants selected a door, followed by a fixation cross displayed for 1000 ms. Participants then received feedback on their performance for 2000 ms. Gains were depicted by a green arrow pointing upward and losses depicted by a red arrow pointing downward. A fixation cross was then displayed again for 1500 ms. Finally, the message “Click for next round” was presented until the participant pressed the button to begin the next trial. Each participant was presented with a total of 30 gain trials and 30 loss trials in random order, in 20 trial blocks.

Participants also completed an arrowhead version of the flankers task (Eriksen and Eriksen, 1974) while EEG was recorded. On each trial, horizontally aligned arrowheads were presented for 200 ms, followed by an intertrial interval (ITI) varying randomly between 2300 and 2800 ms. Half of the trials were compatible (“> > > > >” or “< < < < <”) and half were incompatible (“< < > < <” or > > < > >”); the order of trials was randomly determined. Participants were told to press the right mouse button if the center arrow faced right and to press the left mouse button if the center arrow faced left. After a 30 trial practice block, participants completed 11 blocks of 30 trials (330 trials). Each block was initiated by the participant and participants received feedback based on their performance at the end of each block. If performance was 75% correct or lower, the message “Please try to be

more accurate" was displayed; if performance was above 90% correct, the message "Please try to respond faster" was displayed; otherwise the message "You're doing a great job" was displayed.

EEG recording and processing procedures were consistent with previous investigations of the RewP and ERN (e.g., Bress et al., 2013). Continuous EEG was recorded using an elastic cap with 34 electrode sites placed according to the 10/20 system. Electro-oculography (EOG) was recorded using four additional facial electrodes: two placed approximately 1 cm outside of the right and left eyes and two placed approximately 1 cm above and below the right eye. Sintered Ag/AgCl electrodes were used. EEG and EOG were recorded using the ActiveTwo system (BioSemi, Amsterdam). Data were digitized with a sampling rate of 1024 Hz using a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz. For the EEG electrodes, a common mode sense active electrode producing monopolar (nondifferential) channel was used as a recording reference. The EOG electrodes produced two bipolar channels that measured horizontal and vertical eye movements.

EEG data were analyzed using BrainVision Analyzer, version 2.1 (Brain Products, Gilching, Germany). Data were referenced offline to the average of left and right mastoids, band-pass filtered (0.1 to 30 Hz), and corrected for eye movement artifacts (Gratton et al., 1983). Epochs with a voltage greater than 50 μV between sample points, a voltage difference of 300μV within a segment, or a maximum voltage difference of less than 0.5 μV within 100 ms intervals were automatically rejected. Additional artifacts were identified and removed based on visual inspection.

For the reward task, feedback-locked epochs were extracted with a duration of 1000 ms, beginning 200 ms before feedback presentation. The 200 ms prestimulus interval served as the baseline. Feedback-locked ERPs were averaged separately for gains and losses; the RewP was quantified as the difference between gain and loss trials (gains minus losses) as the mean amplitude from 250–350 ms following feedback at FCz, where the difference between gains and losses was maximal. It should be noted that this measure has been previously used to predict depression in the full sample at the 18-month follow-up (Nelson et al., 2016).

For the flankers task, response-locked epochs were extracted with a duration of 1500 ms, beginning 500 ms before the response. The 200 ms interval prior to the response served as the baseline. Feedback-locked ERPs were averaged separately for correct for error and correct trials. The ERN was quantified as the average activity from 0–100 ms after error commission, at FCz, where error related activity was maximal. Additionally, the correct response negativity (CRN) was quantified in the same time window at FCz, after correct responses. To isolate error-specific brain activity, analyses focused on the ΔERN—quantified as the ERN minus the CRN.

1.6. Data analysis

A total of 152 girls had lifetime anxiety disorders at baseline; however, participants were excluded from analyses if they had a lifetime depressive disorder not otherwise specified diagnosis at baseline (N = 13), were missing diagnostic interview data at Wave 4 and had never met criteria for a depressive disorder (N = 12), or had an outlier ERP value (N = 13). This provided a final sample of 114 adolescent girls.

We first examined bivariate correlations of first-onset depressive disorders and Wave 4 depressive symptoms with baseline anxiety and depressive symptoms, number of lifetime anxiety disorders, current vs. past anxiety disorder status, personality facets, and ERP predictors. In addition to the depression specific risk factors, significant bivariate predictors were included in multivariate analyses. Logistic and linear regression analyses were used to determine which bivariate predictors contributed unique variance in predicting first-onset depressive disorder and Wave 4 depressive symptoms. Baseline IDAS Depression was included as a covariate in all multivariate analyses to account for baseline depressive symptoms. Predictors were standardized prior to the logistic regression analysis to allow for direct comparison of odds ratios. In post-hoc analyses, we included two-way interactions of significant depression-specific risk factors and IDAS social anxiety in the last step of our models to examine whether these relationships varied by the degree of anxiety symptoms. We focused on social anxiety symptoms because social anxiety and peer relationships are particularly relevant for this development period (Crone and Dahl, 2012; Steinberg and Morris, 1991), social anxiety was one of the most prevalent baseline anxiety disorders in our sample, and such symptoms have been strongly linked to later depression (Cummings et al., 2014; Silk et al., 2012). All independent variables were centered prior to creating interaction terms (Aiken and West, 1991). Interactions were interpreted by comparing simple slopes at high and low levels (± 1 *SD*) of the moderator.

2. Results

Over 27 months, 24 participants (21.1%) had a first-onset depressive disorder, including major depressive disorder (N = 11, 9.6%), dysthymia (N = 6, 5.3%), and depressive disorder not otherwise specified (N = 9, 7.9%). Two participants were diagnosed with both major depressive disorder and dysthymia over the study interval.

Bivariate correlations of first-onset depressive disorders and Wave 4 depressive symptoms with baseline depressive and anxious symptoms, number of lifetime anxiety disorders, current vs. past anxiety disorder status, personality composites, and psychophysiology are presented in Table 2. First-onset depressive disorders from Waves 2 to 4 were related to higher baseline trait sadness ($r = 0.31$, $p < .01$), trait anxiousness ($r = 0.21$, $p < .05$), a blunted RewP ($r = -0.23$, $p < .05$), and higher baseline IDAS depression ($r = 0.25$, $p < .01$) and IDAS social anxiety symptoms ($r = 0.22$, $p < .05$). Wave 4 IDAS depression symptoms was related to higher baseline sadness ($r = 0.42$, $p < .001$), anxiousness ($r = 0.37$, $p < .001$), a blunted RewP ($r = -0.19$, $p = .05$), and higher baseline IDAS depression ($r = 0.40$, $p < .001$), IDAS panic ($r = 0.34$, $p < .001$), and IDAS social anxiety symptoms ($r = 0.28$, $p < .01$).

Baseline personality composites, the RewP, and anxiety symptoms were included as predictors in logistic and linear regression analyses. Baseline depressive symptoms were included as a covariate in analyses. As shown in Fig. 1, a blunted RewP uniquely predicted a greater likelihood of developing first-onset depressive disorders (OR = 0.50, 95% CI = 0.27–0.91, $p < .05$). A blunted RewP ($\beta = -0.18$, $p < .05$) also uniquely predicted greater depressive symptoms at 27 months.

Table 2
Bivariate correlations of baseline measures with first-onset depressive disorder and IDAS depression.

Baseline variable	First-onset depressive disorder (1 = Present) *r*	Wave 4 IDAS depression *r*
Sadness	0.31**	0.42***
Anxiousness	0.21*	0.37***
Positive affectivity	−0.17	−0.08
RewP	−0.23*	−0.19
ERN	0.06	0.11
Number of lifetime anxiety disorders	0.17	0.09
Current vs. past anxiety disorder (1 = Current)	−0.02	0.04
IDAS depression	0.25**	0.40***
IDAS panic	0.10	0.34***
IDAS social anxiety	0.22*	0.28**
IDAS claustrophobia	0.07	0.09
IDAS checking	−0.04	0.15
IDAS ordering	0.03	0.17
IDAS cleaning	0.02	0.16

*$p < .05$, **$p < .01$, ***$p < .001$; RewP = Reward positivity; ERN = Error-related negativity.

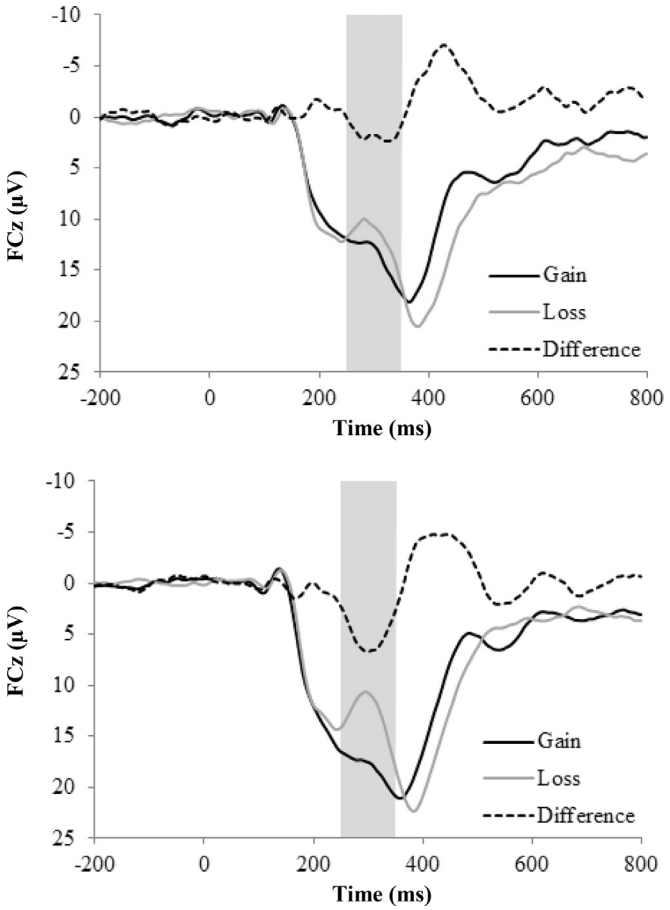

Fig. 1. Feedback locked ERPs at the FCz electrode site in response to losses and gains, as well as the gain-loss difference. Results are shown for participants with first onsets of depressive disorders (top) and for participants who did not have a first onset. Negative values are plotted up.

Post-hoc,[2] we then included the interaction of the RewP and IDAS social anxiety in the logistic and linear regression models to explore whether these relationships varied as a function of social anxiety symptom severity, as shown in Table 3. The interaction of IDAS social anxiety and the RewP uniquely predicted both first-onset depressive disorders (OR = 0.48, 95% CI = 0.24–0.96, $p < .05$) and depressive symptoms at 27 months ($\beta = -0.12$, $p < .05$). As shown in Fig. 2, at high levels of social anxiety symptoms, the effect of a blunted RewP on both first-onset depressive disorder (OR = 1.37, 95% CI = 0.37–2.37, $p < .01$) and depressive symptoms ($\beta = -0.25$, $p < .01$) was significant, but not at low levels for either first-onset depressive disorders (OR = 0.08, 95% CI = −0.74–0.91, $p = .84$) or depressive symptoms ($\beta = 0.00$, $p = .98$).

3. Discussion

The current study prospectively tested predictors of first-onset depressive disorders and depressive symptoms in a sample of 114 adolescent girls with baseline anxiety disorders. Models of sequential comorbidity have primarily focused on shared and anxiety-specific risk factors for later depressive disorders and symptoms. In contrast, we examined depression-specific risk factors (trait sadness, low PA, blunted reward sensitivity) as predictors of first-onset depressive disorders and depressive symptoms. As a comparison, we included some corresponding features unique to anxiety (trait anxiousness, error-related negativity), current anxiety disorder status, and several indicators of anxiety severity, including baseline anxiety symptoms and number of lifetime anxiety disorders. In bivariate analyses, baseline sadness, anxiousness, blunted reward sensitivity, and depression, panic, and social anxiety symptoms were associated with first-onset depressive disorders and/or depressive symptoms 27 months later. However, in multivariate models that adjusted for baseline depressive symptoms, a single depression-specific predictor, blunted reward sensitivity, emerged as a unique predictor of first-onset depressive disorders and depressive symptoms in anxious adolescent girls. This finding is consistent with Cummings et al.'s (2014) notion of depression-specific risk factors influencing concurrent comorbidity, and is the first of its kind in demonstrating that depression-specific risk contributes to sequential comorbidity. Furthermore, post-hoc analyses indicated that blunted reward sensitivity predicted first-onset depressive disorder and later depressive symptoms only at high levels of social anxiety symptoms.

Our results are consistent with previous studies reporting that a blunted RewP predicts first-onset and symptoms of depression in unselected samples (e.g., Bress et al., 2013; Nelson et al., 2016). Our findings are also consistent with Silk et al. (2012)'s proposal that low reward sensitivity influences which anxious youth develop depression. Specifically, they hypothesized that, due to the salience of social experiences for adolescents (Crone and Dahl, 2012), blunted responses to social rewards predispose anxious youth to later depression. In the present study, a blunted RewP, an ERP component elicited by both monetary and social reward (Kujawa et al., 2017), emerged as a predictor in response to monetary rewards of later depressive symptoms and diagnoses in anxious youth.

Although blunted reward sensitivity is a risk factor for later depressive symptoms and disorders in unselected community samples of youth (Nelson et al., 2016), blunted reward sensitivity specifically predicted first-onset depressive disorders and later depressive symptoms in the presence of high levels of social anxiety symptoms in our anxious sample. Girls with high levels of both social anxiety and reactivity to reward were at lower risk for later depressive symptoms and disorders, likely due to the enhanced PA and approach behavior associated with high levels of reward sensitivity (Olino, 2016). Further, consistent with Silk et al. (2012), youth with low reward sensitivity and high social anxiety symptoms were at greatest risk for first-onset depressive disorders. Silk and colleagues also maintain that social evaluative threat responding may disrupt reward processing in anxious youth, which may then lead to later depression. Given that anxiety symptoms have been associated with enhanced, rather than blunted, reward sensitivity in children in some studies (Kessel et al., 2015; Shechner et al., 2012), prospective investigation of influences of social anxiety symptoms on changes in reward sensitivity is warranted.

Blunted reward sensitivity is a risk factor for later depression that is likely present early in life (Klein and Finsaas, 2017). This factor may reflect a trait-like vulnerability for depression that is activated in adolescence (Salk et al., 2017), possibly due to pubertal development and the stressors and challenges associated with this developmental period (Hyde et al., 2008; Silk et al., 2012). Alternatively, blunted reward sensitivity may function as an early manifestation of developing depressive pathology, perhaps as a precursor to anhedonia (Silk et al., 2012). Future work should investigate the trajectory of early blunted reward sensitivity in relation to the development of depressive symptoms and disorders in anxious youth.

The present findings also extend the literature on depression-specific facets of extraversion and neuroticism to a sample of anxious adolescent girls. In addition to predicting first-onset depressive disorders in community samples of adolescents (Goldstein et al., 2017; Zinbarg et al., 2016), sadness was associated with first-onset depressive disorders and later depressive symptoms in the present sample of anxious adolescent girls. However, sadness did not emerge as a significant predictor of first onsets or depressive symptoms in multivariate analyses. This may be in part because our sample was just entering the risk

[2] We would like to thank an anonymous reviewer for this suggestion.

Table 3
Multiple regressions with baseline measures predicting first-onset depressive disorder and IDAS depression over 27 months[a].

Baseline predictor	First-onset depressive disorder				Wave 4 IDAS depression			
	R^2	X^2	OR	95% CI	R^2	F	β	95% CI
	.28	22.26**			.30	5.05***		
IDAS depression			1.05	[0.49, 2.24]			.06	[−0.15, 0.26]
Sadness			2.22	[0.82, 5.98]			.17	[−0.04, 0.37]
Anxiousness			0.89	[0.40, 1.99]			.07	[−0.09, 0.23]
Positive affectivity			0.83	[0.43, 1.60]			.06	[−0.10, 0.22]
RewP			0.53*	[0.29, 0.97]			−0.12*	[−0.23, −0.01]
IDAS social anxiety			0.93	[0.43, 2.03]			−0.04	[−0.19, 0.11]
IDAS panic			–	–			.14	[−0.04, 0.32]
Social anxiety X RewP			0.48*	[0.24, 0.96]			−0.12*	[−0.24, −0.01]

$^{+}p = .05$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$; RewP = Reward positivity.

[a] Baseline IDAS Depression was included as a covariate in all analyses to account for baseline depressive symptoms. Predictors were standardized for the logistic regression analysis to allow for direct comparisons of odds ratios. Logistic regression was used for the dichotomous dependent first-onset depressive disorder variable and linear regression was used for the continuous dependent variable depressive symptom score at Wave 4. Chi-squares and F statistics are reported for the overall omnibus tests for the logistic and linear regressions, respectively. The Nagelkerke R^2 is reported for the logistic regression.

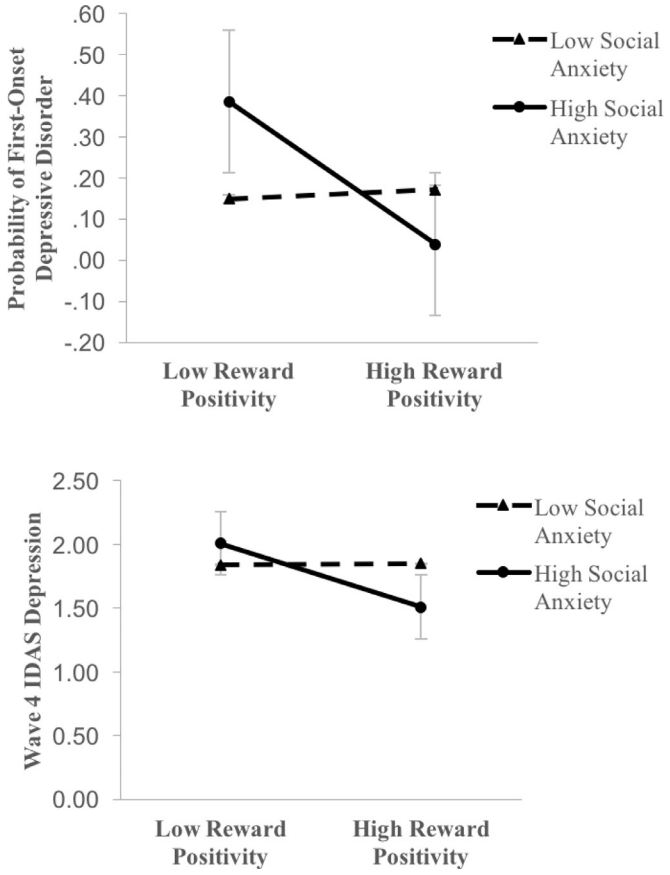

Fig. 2. Relationship between the reward positivity at baseline and later depression at high and low levels of baseline social anxiety symptoms. Results are shown for the probability of first onsets of depressive disorders (top) and Wave 4 IDAS Depression scores (bottom). Only the slopes at high social anxiety symptoms are significant. Error bars indicate standard errors.

period for depression (Salk et al., 2017), and anxious youth, like healthy youth, will likely develop further or more severe depressive symptoms and disorders with time (Klein et al., 2009; Pincus et al., 1999; Rohde et al., 2009). Alternatively, the lack of a significant unique effect in the multivariate models may have been due to shared variance with other predictors, such as anxiousness, PA, and/or baseline depressive and anxiety symptoms ($|rs| = |0.43–0.70|$; mean $|r| = |0.57|$). Interestingly, although low PA has also been shown to predict subsequent depression in adolescents generally (Goldstein et al., 2017; Neumann et al., 2011; Naragon-Gainey and Watson, 2014), it was not associated with first-onset depressive disorders or depressive symptoms. This is consistent with other investigations that generally find that neuroticism is more strongly related to depression than low extraversion (e.g., Kendall et al., 2015; Kendler et al., 2006; Kotov et al., 2010).

Clinically, because efforts to prevent anxiety disorders do not influence later depressive symptoms (Garber et al., 2016), blunted responses to reward may be a good target for prevention of depressive symptoms and disorders, particularly in socially anxious youth. Further, clinicians would be prudent to engage in ongoing monitoring of youth's depressive symptoms when treating anxious girls, particularly those who exhibit blunted responses to reward and elevated social anxiety symptoms. Our findings also raise the possibility that adolescent girls being treated for anxiety who exhibit blunted responses to rewarding stimuli may benefit from adjunctive interventions to prevent future depressive symptoms and disorders, particularly in the presence of high social anxiety symptoms. This could include CBT interventions, such as behavioral activation and mindfulness of positive stimuli (Craske et al., 2016). This may be particularly fruitful given that blunted reward sensitivity predicted a stronger response to CBT in adults with comorbid depression and anxiety (Burkhouse et al., 2016).

Despite its strengths utilizing a prospective design and careful diagnostic measures, the present study had limitations. First, the sample was limited to a relatively demographically homogenous sample of 13–15 years old girls. Therefore, results may not generalize to other samples with anxiety disorders. Second, personality and depression were assessed using only data obtained from the adolescent, who we assumed was the best source of information on internal experiences (De Los Reyes and Kazdin, 2005). Third, the IDAS Claustrophobia subscale does not assess the full range of contexts relevant for agoraphobia and specific phobia, which may limit coverage of baseline anxiety symptom severity relevant to our sample. Fourth, we were unable to control for concurrent anxiety disorders and symptoms at follow-up because they were not assessed in the second and fourth assessment waves. Fifth, we had a relatively small number of first-onset depressive disorders, many of which were depressive disorder not otherwise specified, although minor depression is a very strong predictor of later major depressive disorder (Klein et al., 2009). Finally, our sample was not large enough to examine particular anxiety disorders individually, and pathways to sequential comorbidity may differ as a function of the form of anxiety disorder (Cummings et al., 2014).

4. Conclusions

In multivariate analyses adjusting for baseline depressive symptoms, a depression-specific risk factor, specifically blunted reward sensitivity, predicted a greater likelihood of developing first-onset depression and higher depressive symptoms 27 months later in adolescent females with a history of anxiety disorder. Post-hoc analyses indicated that blunted reward sensitivity predicted first-onset depressive disorders and depressive symptoms only in adolescent females with elevated social anxiety symptoms. This suggests that depression-specific risk may indicate which anxious girls are at risk for later depressive symptoms and disorders. Anxious girls exhibiting blunted responses to positive stimuli and high social anxiety symptoms may benefit from prevention efforts targeting reward deficits to prevent subsequent depressive disorders and symptoms and reduce the associated functional impact.

Conflicts of interest

The authors have no conflicts of interest to disclose.

Author's contribution

Estee M. Hausman conducted data analyses and interpretation and drafted the manuscript. Daniel N. Klein contributed to drafting the manuscript and Ellen M. Kessel contributed to data analyses and interpretation. Roman Kotov, Daniel N. Klein, and Greg Hajcak designed the study. Roman Kotov, Greg Perlman, Greg Hajcak, Ellen M. Kessel, and Daniel N. Klein provided critical revision of the manuscript. All authors have approved the final article.

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Institutional Review Board review

All procedures in this study were approved by the Stony Brook University Institutional Review Board.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2018.04.005.

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