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The Perils of Murky Emotions: Emotion Differentiation Moderates the Prospective Relationship Between Naturalistic Stress Exposure and Adolescent Depression

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Negative emotion differentiation (NED) refers to the ability to identify and label discrete negative emotions. Low NED has been previously linked to depression and other indices of low psychological well-being. However, this construct has rarely been explored during adolescence, a time of escalating depression risk, or examined in the context of naturalistic stressors. Further, the association between NED and depression has never been tested longitudinally. We propose a diathesis-stress model wherein low NED amplifies the association between stressful life events (SLEs) and depression. A sample of 233 community-recruited midadolescents (M_{age} = 15.90 years, 54% female) completed diagnostic interviews and reported on mood and daily stressors 4 times per day for 7 days. SLEs were assessed using a semistructured interview with diagnosis-blind team coding based on the contextual threat method. Follow-up interviews were conducted 1.5 years after baseline. Low NED was correlated with depression but did not predict prospective changes in depression as a main effect. Confirming predictions and supporting a diathesis-stress model, low NED predicted (a) within-subjects associations between daily hassles and momentary depressed mood, (b) between-subjects associations between SLE severity and depression, and (c) prospective associations between SLE severity and increases in depression at follow-up. Results were specific to negative (vs. positive) emotion differentiation. Results suggest that low NED is primarily depressogenic in the context of high stress exposure.

Keywords: negative emotion differentiation, adolescence, depression, stress, granularity

Discrete negative emotional states communicate crucial information about potential causes of distress and strategies for the remediation of environmental threats. Labeling emotional distress as *guilt* versus *irritation* or *restlessness* can help to illuminate sources of distress, relational contexts, expected physiological experiences, and identify appropriate behavioral responses. *Negative emotion differentiation* (NED, also referred to as emotional granularity) refers to the ability to make fine-grained distinctions between negative emotions (NEs) and apply precise labels for negative emotional states (Barrett, Gross, Christensen, & Benvenuto, 2001; Kashdan, Barrett, & McKnight, 2015). People low on NED tend to use more global terms to describe their NEs (e.g., *bad*, *upset*) and are consequently less able to extract and benefit from the potentially useful information encoded in their NEs.

Given the centrality of emotions to psychological functioning (Kring & Sloan, 2011), it is not surprising that the inability to distinguish between negative emotional states is associated with a broad range of negative outcomes. Consistent with the notion that the identification and labeling of NEs facilitates the retrieval of appropriate regulatory strategies, low NED predicts poorer emotion regulation in laboratory and naturalistic settings (Barrett et al., 2001; Hill & Updegraff, 2012; O'Toole, Jensen, Fentz, Zachariae, & Hougaard, 2014; Tong & Keng, 2017) as well as increased maladaptive behavioral responses to emotional states in daily life (e.g., Kashdan, Ferssizidis, Collins, & Muraven, 2010; Pond et al., 2012). Low NED is also broadly associated with disruptions in psychological well-being (Erbas, Ceulemans, Lee Pe, Koval, & Kuppens, 2014), including the presence of psychological symptoms and disorders (e.g., Erbas,

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Ceulemans, Boonen, Noens, & Kuppens, 2013; Kashdan & Farmer, 2014; O'Toole et al., 2014).

NED and Its Association With Depression

Depression is a prevalent, recurrent condition that contributes to massive global health burden and predicts a host of negative outcomes, even at the subsyndromal level (Judd et al., 1998; Murray et al., 2012). Mapping emotional dynamics associated with depression may be key to devising effective treatments. Multiple studies link low NED to depression and depressive symptom severity (Demiralp et al., 2012; Erbas et al., 2014; Goldston, Gara, & Woolfolk, 1992; Plonsker, Gavish Biran, Zvielli, & Bernstein, 2017; Starr, Hershenberg, Li, & Shaw, 2017). Importantly, this association is not better accounted for by the fact that depressed people tend to experience more intense emotions, which is itself associated with NED (Demiralp et al., 2012). The putative link between low NED and depression makes conceptual sense, as depression is associated with marked aberrations in the experience and contextual modulation of NEs (Rottenberg, Gross, & Gotlib, 2005; Thompson et al., 2012). Moreover, the act of labeling emotions has inherent regulatory properties (Lieberman et al., 2007), which may protect against depression. Finally, as improvement of emotion differentiation is a plausible target of intervention, better understanding NED's link to depression may have important implications for both etiological and treatment models. However, there are important gaps in our knowledge about NED and its association with depression.

First, research on NED and depression to date has largely been cross-sectional, showing that NED correlates with depressive symptoms or predicts group differences between depressed and nondepressed groups (e.g., Demiralp et al., 2012; Starr, Hershenberg et al., 2017). Although an important first step, the reliance on cross-sectional data leaves crucial questions unanswered about temporal sequencing. Indeed, though low NED may be an etiological factor for depression, an alternative possibility is that low NED is a state-related correlate of depressive symptoms, perhaps a consequence of the concentration problems, negative thinking, or other impairments characteristic of depression. These two possibilities are not necessarily mutually exclusive, but to the extent that NED is affected by state depression, cross-sectional associations may be inflated and conclusions about the etiological importance of low NED may be overstated. Thus, longitudinal data that clarifies whether low NED predicts increasing depression is badly needed. Addressing this important gap, the current study will examine whether NED predicts increased depressive symptoms over a 1.5-year follow-up during adolescence, a key developmental period.

Emotion Differentiation in Adolescents

Almost all research on emotion differentiation has focused on adults; to date, only two studies have examined this construct in younger samples (Lennarz, Lichtwarck-Aschoff, Timmerman, & Granic, 2018; Nook, Sasse, Lambert, McLaughlin, & Somerville, 2018). Adolescence may be a particularly critical time to examine this construct. Depression rates climb steadily during adolescence (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015), corresponding with rapid, nonlinear neural maturation in emotion-related regions (e.g., Yurgelun-Todd, 2007), increased reports of daily negative emotions (Larson & Asmussen, 1991), and developmental shifts that introduce novel and emotionally challenging interpersonal contexts (e.g., Collins, 1990, 2003). Further, compared to adults, adolescents show amplified amygdala responses to emotional stimuli (Hare et al., 2008), potentially making top-down regulation (which may be supported by the ability to effectively differentiate between NEs) even more important for maintaining emotional well-being.

Further, recent evidence suggests that emotion differentiation reaches its nadir during adolescence, with adolescents showing lower NED compared to either younger children or adults (Nook et al., 2018). That NED decreases at a point in development when depression rates steadily climb suggests that examining the relationship between depression and NED in an adolescent sample may be an important direction for investigation. To date, only one study has cross-sectionally examined adolescent NED in relation to well-being (Lennarz et al., 2018). Surprisingly, that study did not find significant associations between NED and depression; however, higher differentiation was linked to other aspects of emotional well-being (e.g., lower emotional intensity), suggesting additional investigation is merited. Further, Lennarz et al. (2018) examined only the main effect of NED on depression, and it is possible that differentiation is more strongly linked to depression under certain moderating conditions.

A Diathesis–Stress Model of NED and Depression

Given increased environmental demands during adolescence, one important moderating context to examine is environmental stress. Environmental stress is a central etiological component of depression, with the majority of initial major depressive episode onsets preceded by stressful life events (Hammen, 2005). However, there is substantial heterogeneity in the relationship between stress exposure and depression; while some individuals spiral into depressive episodes following even mild stress exposures, other remain resilient in the face of stunning adversity. As such, researchers have long argued that identifying diatheses that modulate the impact of stressful life events (SLEs) on depression is key to understanding depression etiology (i.e., diathesis–stress model; Monroe & Simons, 1991).

We propose that NED interacts with SLE exposure to predict depression, such that youth who are poor at differentiating emotions are more susceptible to depressive symptoms following stressful experiences. As theory and research suggest that emotion differentiation promotes the selection and implementation of appropriate regulatory strategies (Barrett et al., 2001; Kashdan et al., 2015), we hypothesize that adolescents with high NED may be better prepared to manage the emotional and behavioral aftermath of stress exposure, reducing the likelihood that negative emotions will escalate into clinically significant depression over time. Aligning with this model, Flynn and Rudolph (2010) found that low self-perceived emotional clarity predicted maladaptive stress responses among children, which in turn predicted depression. Likewise, Stange, Alloy, Flynn, and Abramson (2013) found that high self-reported emotional clarity protected against increases in depression following SLEs among adolescents with high cognitive vulnerability. Importantly, self-perceived emotional clarity, although conceptually very similar to NED, has shown surprisingly

limited empirical overlap with behaviorally defined NED (Boden, Thompson, Dizén, Berenbaum, & Baker, 2013) However, consistent with the idea that low NED itself predicts stronger responses to stressful experiences, Kashdan et al. (2014) found that, among those with low self-esteem, low NED predicted higher neural reactivity to a virtual social ostracization task. Taken together, this evidence aligns with the hypothesis that low NED may amplify the association between naturalistic stress exposure and depression.

In the most direct test of our proposed model to date, Starr, Hershenberg et al. (2017) found that NED moderated within-person associations between daily stress exposure and daily fluctuations in depressed mood in a sample of help-seeking veterans, with elevated daily stress predicting higher depressed mood at low but not high levels of NED. However, authors showed no support for a daily stress–NED interaction in a sample of college students; this inconsistency in findings suggests that further replication is warranted. Following growing recognition of the need for direct replications in psychological science (Simons, 2014), one goal of our study is to reevaluate the within-subjects model supported in the Starr, Hershenberg et al. (2017) study, using intensive longitudinal data tracking fluctuations between daily, low-level stress (i.e., hassles) and depressed mood.

Daily hassles (e.g., arguments with family members, parking tickets) strongly affect negative mood (Eckenrode, 1984), and mood reactivity to daily hassles has been shown to longitudinally predict depressive symptoms (Cohen, Gunthert, Butler, O'Neill, & Tolpin, 2005). That said, SLEs (e.g., parental divorce, romantic break-ups, or major academic failures) are more likely to directly spur clinically significant depressive episodes and are considered more central to depression etiology (Hammen, 2005). Importantly, gold-standard research on SLEs uses objective coding that can distinguish between the intensity of the stress occurrence and the stress response (i.e., emotional reactions to stress such as depression; (Harkness & Monroe, 2016). To our knowledge, no prior research has examined if NED moderates the association between objectively coded SLEs and symptoms of depression, which we examine in the present study.

The Present Study

In the current study, we examined the longitudinal relationship between NED and depression in a sample of adolescents drawn from the community. Importantly, we used gold-standard assessment methods for all constructs of interest: depression was evaluated using clinical interviews, NED was assessed behaviorally utilizing intensive longitudinal reports of negative emotions (Tugade, Fredrickson, & Barrett, 2004), and SLEs were assessed with an interview-based approach using diagnosis-blind team-based coding of contextual threat (a method that effectively segregates stress exposure from stress response to reduce spurious findings; Harkness & Monroe, 2016).

First, we hypothesized that NED and depression would be inversely related. As a conservative step, we examined whether NED's association with depression held controlling for mean emotional intensity, as depressive symptoms are associated with more intense daily NEs, which in turn may be plausibly linked to lower differentiation due to restricted range. Second, as a test of the temporal precedence of NED, we also hypothesized that NED would longitudinally predict increasing depressive symptoms over time, controlling for baseline symptoms.

We next examined a diathesis–stress model of NED and depression under several different scopes of time. First, we attempted to replicate previous findings (Starr et al., 2017) suggesting that low NED intensifies the association between daily hassles and fluctuations in depressed mood. Second, we hypothesized low NED would intensify cross-sectional associations between SLE exposure and depression. Finally, for a more rigorous test of this hypothesis, we addressed this question longitudinally, hypothesizing that low NED would predict stronger associations between stress exposure and prospective increases in depression.

Although our primary hypotheses focus on NED, some research has examined differentiation of positive emotional states (positive emotion differentiation [PED]) in relation to maladaptive behaviors in psychopathology (e.g., Dixon-Gordon, Chapman, Weiss, & Rosenthal, 2014; Selby et al., 2014). However, studies have generally not supported an association between PED and depression (Demiralp et al., 2012; Starr, Hershenberg et al., 2017). Further, in contrast to positive emotions (PEs; Fredrickson, 2001), NEs arguably explicitly function to avoid and manage threats (Parrott, 2002); therefore, NED may be more relevant than PED to managing the emotional consequences of stressors. As supplemental analyses, we reexamined hypotheses with regard to PED to explore whether findings are specific to NED or apply to emotion differentiation more broadly.

Method

Participants

Participants were recruited from community settings in a mid-sized metropolitan area of the northeastern United States (for full recruitment details, see Starr, Dienes et al., 2017). Eligible participants were aged 14–17 years and fluent in English. Exclusion criteria included evidence of pervasive developmental disorder, prior diagnosis of bipolar or psychotic disorder, any major physical or neurological disorder, and prior enrollment of another household member.

The full sample for the study was comprised of 241 adolescents (M_{age} = 15.90 years, SD = 1.09; 53% female, 46% male, 1% nonbinary gender) who participated with their primary caregiver. Eight subjects from the overall sample were excluded from analyses due to insufficient daily survey information to compute NED (i.e., fewer than two diaries with complete NE data), resulting in an NED sample of 233 adolescents. There were no significant differences on key demographic variables (i.e., age, race, sex), depression, or episodic stress (all $ps > .05$) between adolescents excluded for insufficient data and those included. Approximately 1.5 years after their initial participation (mean follow-up period 19.09 months, SD = 4.14), adolescents were invited to participate in follow-up interviews. Of the original 241 adolescents, 193 participated in interviews (80.1% retention). There was no evidence of differential attrition by age, race, sex, or Time 1 (T1) depression, episodic stress, or emotional intensity levels. However, adolescents with lower NED at T1 were less likely to participate at Time 2 (T2), $t(51.23) = -2.29$, $p = .026$. All procedures were approved by the University of Rochester Research Subjects Review Board.

Procedure

Baseline (T1) lab visit. Participants and their primary caregivers attended a lab session during which they provided consent and assent, then completed separate interviews and a battery of questionnaires. At the end of each lab visit, adolescents were given detailed instructions about the completion of daily surveys during the follow-up period. Families were compensated with up to $160 (depending on compliance) for completion of all study procedures.

Daily surveys. Adolescents were asked to complete brief surveys using their personal smartphones, computers, or study-provided tablets four times per day for 7 days, beginning the day after the baseline assessment. Because participants were drawn from a large number of school districts, it was not feasible to obtain school permission for in-class participation; therefore, surveys were scheduled to be completed at designated times corresponding to each adolescent's school schedule, at approximately 4–6 hr intervals. First, adolescents completed a brief survey after waking up in the morning (Prompt 1). Second, they were asked to complete two midday surveys (Prompts 2 and 3). These were finished at lunchtime (on school days, these were done during breaks between instruction periods in accordance with their school's policies) and in the afternoon between 2:30 p.m. and 5:00 p.m. Finally, adolescents completed a 10-min survey before their bedtime (Prompt 4). Compliance was incentivized with a prorated compensation system and with additional raffles based on diary completion. Participants were eligible to receive up to $50 depending on their dairy completion and received one raffle entry per completed survey. Study staff monitored diary compliance daily and contacted noncompliant participants to troubleshoot difficulties and encourage survey completion. Compliance was good; among the 233 participants included in NED computations, the mean number of valid diaries completed was 24.05 (SD = 6.15) out of 28 (86% compliance), with 81% of the sample completing at least 75% of surveys. Compliance rates were comparable across the four prompts. Among participants included in the NED calculations, lowest compliance rate was two diaries (n = 2; excluding these participants did not alter results) and 27% of participants completed 100% of diaries. The mean lag between surveys (within day) was 4.84 hr (SD = 1.41).

T2 follow-up. At T2, adolescents completed interviews over the phone to reduce burden and allow for geographically remote participation (e.g., for adolescents beginning college). Adolescents were compensated with $50 for all T2 study components.

Measures

SLEs. Episodic life stress exposure was assessed at T1 and T2 using a version of the UCLA Life Stress Interview modified for use with adolescents (LSI; Hammen, Henry, & Daley, 2000; Shih, Eberhart, Hammen, & Brennan, 2006). The LSI is a semistructured interview that assesses adolescents' exposure to chronic and episodic life stress. For episodic life stress, the LSI uses a contextual threat approach (Brown & Harris, 1978) to produce objective ratings of the severity of SLEs while considering the surrounding circumstances. Trained interviewers elicited information from adolescents about discrete stressful events that occurred within the last 12 months, including details about timing, duration, and circumstances surrounding each event. Afterward, interviewers prepared a written narrative for each event and presented them to a team of coders who were blind to adolescents' diagnostic status and emotional reactions to the event. These coders considered the context surrounding each event and provided a consensus rating of objective severity on a 5-point scale, with half-point intervals from 1 (*no negative impact*) to 5 (*extremely severe impact*). Total episodic stress was computed by summing severity scores for all events (following convention, events coded as 1, indicating nonevents, were excluded). A second team of blind coders recoded a subset of events, with excellent reliability (ICC = .87 at T1, .86 at T2). Adolescents reported a mean of 2.95 events (SD = 2.09) at T1 and 2.78 events (SD = 1.20) at T2.

Depression. Trained interviewers assessed adolescents' symptoms of depression using the depressive disorders module of the Schedule for Affective Disorders and Schizophrenia for School-Aged Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997). The K-SADS-PL is a semistructured diagnostic interview that has demonstrated high validity and reliability. Similar to prior research (Rao, Daley, & Hammen, 2000; S. J. Steinberg & Davila, 2008), symptoms were captured at the disorder and subsyndromal levels using a 5-point rating scale: 0 = *no symptoms*; 1 = *mild symptoms*; 2 = *moderate, subthreshold symptoms*; 3 = *DSM–IV criteria met*; 4 = *DSM–IV criteria met with high severity/impairment*. Maximum ratings for major depressive disorder and dysthymia were utilized as an index of depression. Current episodes were included as our T1 variable. At T2, current episodes were assessed, as well those experienced since T1. In longitudinal analyses (except where otherwise noted), we used as an outcome variable the most severe depression rating reported during the follow-up period. Blind raters recoded audiotaped interviews with outstanding reliability (ICCs = 1.00 at T1, .97 at T2). Consistent with the community sample and the inclusion of only current symptomology, at T1 15% of the sample reported current clinically significant symptoms (4% meeting diagnostic criteria). At T2 (with symptoms assessed over a larger time span), 37% reported clinically significant samples, with 16% meeting diagnostic criteria).

Daily diary measures.

NED and other mood variables. At each diary prompt, participants rated their current emotions using the 12 NE adjectives on the 15-item version of the Profile of Mood States (POMS-15; Cranford et al., 2006). This version of the POMS assesses NEs across five domains using three items each: anxious mood (anxious, on edge, uneasy); depressed mood (sad, hopeless, discouraged); anger (angry, resentful, annoyed); and fatigue (fatigued, worn out, and exhausted). Five positive emotion (PE) items (happy, proud, cheerful, lively, joyful) were drawn from the positive affect subscale of the 10-item version of the Positive and Negative Affect Scale for Children (Ebesutani et al., 2012). Each item was rated on a Likert-type scale ranging from 1 (*not at all*) to 5 (*extremely*).

Using established procedures (Shrout & Fleiss, 1979; Tugade et al., 2004), NED was calculated by taking the average intraclass correlation for all NE items across all assessment points. This computational method has been validated across many studies (e.g., Hill & Updegraff, 2012; Selby et al., 2014; Starr, Hershenberg et al., 2017; Tugade & Fredrickson, 2007). To aid interpretation (and consistent with most other studies), we subtracted ICCs from 1 to create a NED variable where higher values reflect greater differentiation. Because negative ICC values are sometimes gen-

erated but difficult to interpret (Giraudeau, 1996), NED values were truncated at 1.0; results were unchanged if these participants were excluded from analyses. We computed NED using all 12 POMS-15 NE items, rather than taking ICCs across the four validated subscales (anxious mood, depressed mood, anger, and fatigue), because recent evidence suggests that NED indices computed with a range of NEs that are both similar to each other (i.e., fitting under the same overarching category) and different (i.e., representing different categories of emotions) are most strongly associated with well-being (Erbas et al., 2019). This may be because it captures both the ability (or lack thereof) to make fine-grained distinctions between related emotional states as well as broader differentiation between highly disparate emotions. However, using an NED index computed from the four NE subscales produced very comparable results (as shown in the corresponding footnotes). PED was calculated using the five PE items, using identical methods.

In addition, NE intensity was derived by taking the mean of all NE items across all diary data points. Depressed mood was calculated by averaging the three depressed mood items, as were anxious mood, anger, and fatigue (used in supplemental analyses).[1] Internal reliability was taken for both these scales by calculating Cronbach's alpha at each of the 28 prompts and then averaging; *M* alpha was .86 for total NE, .83 for depressed mood, .80 for anxious mood, .75 for anger, and .85 for fatigue.

Daily hassles. Daily hassles were assessed at Prompts 2, 3, and 4 using a scale modeled loosely on existing measures (e.g., Totenhagen, Serido, Curran, & Butler, 2012) but tailored for adolescents and study time constraints. Participants reported whether any negative events occurred across six domains: interactions with friends, romantic life, schoolwork, extracurricular activities or job, interactions with family members, or another area of their life (e.g., "Something negative, unpleasant, or stressful happened in my interactions with my friends"). If they indicated "yes" for the occurrence of an event within the past 5 hrs or since their last survey (whichever was sooner), they were asked to rate how significant the event was for them on a 1 to 5 scale from 1 (*not a big deal at all*) to 5 (*a really big deal*), with those endorsing no events rated as 0. We calculated the mean severity using significance ratings for negative events across all domains.

Data Analytic Approach

Within-subjects moderation hypothesis. Tests of the within-persons hypothesis (NED × Daily Hassles predicting concurrent depressed mood) were conducted using multilevel modeling (MLM) in SPSS 25 MIXED. MLM is recommended for intensive longitudinal data because it accounts for the inherent nonindependence of nested data. MLM also copes well with missing data and has superior statistical power than traditional approaches. For simplicity, we present findings using a two-level approach (repeated measures nested within participants). We also conducted analyses using a three-level structure (observations within days within participants), but this produced near-identical coefficients and no differences in significance or direction of effects, so we present the two-level results as a more parsimonious model. The hypothesis tested a slopes-as-outcomes model (i.e., a cross-level interaction) between daily hassles (Level 1, within-subjects predictor) and NED (Level 2, between-subjects moderator). The hassles variable was person-mean centered, and all Level 2 variables and covariates were grand-mean centered. All effects were entered as fixed effects, and daily hassles and the intercept were entered as random effects. Following recommendations of Bolger and Laurenceau (2013), to best isolate within-subjects variance, we partitioned our within-subjects variable (hassles) into two orthogonal components: a between-persons means component (the person's mean hassles score aggregated over all observations and grand-mean centered, $\breve{X}_{\cdot j}$) and a within-persons component (the deviation from the means component at each observation point, $\breve{X}_{ij} - X_{\cdot j}$). We entered both of these components as fixed effect predictors, as both main effects and interactions with NED. Note that the inclusion of the between-subjects means component is intended to enhance interpretability of the within-subjects component, and corresponding results are not considered interpretable (Bolger & Laurenceau, 2013). Exclusion of the means component variables did not impact results.

Time elapsed from the first completed diary survey was included as the repeated variable and was also included as a covariate to control for temporal change. Time of day was also entered as a covariate to account for diurnal mood variation. A first-order autoregressive (AR[1]) model was specified to account for autocorrelation for residuals, and an unstructured covariance matrix was applied for random effects. Using the notation of Bolger and Laurenceau (2013), the resulting model can be summarized in the following equations:

Level 1:

$$Y_{ij} = \beta_{0j} + \beta_{1j}\left(\breve{X}_{ij} - \breve{X}_{\cdot j}\right) + \beta_{2j}(\text{Time}) + \varepsilon_{ij} \quad (1)$$

Level 2:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}W_j + \gamma_{02}\breve{X}_{\cdot j} + \gamma_{03}W_j\breve{X}_{\cdot j} + u_{0j} \quad (2)$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}W_j + u_{1j} \quad (3)$$

$$\beta_{2j} = \gamma_{20} \quad (4)$$

Here, W_j represents NED, $\breve{X}_{\cdot j}$ and $\breve{X}_{ij} - X_{\cdot j}$ respectively denote main effects for $\text{hassles}_{\text{between}}$ and $\text{hassles}_{\text{within}}$, and $W_j\breve{X}_{\cdot j}$ and $W_j(\breve{X}_{ij} - X_{\cdot j})$ respectively represent NED × $\text{hassles}_{\text{between}}$ and NED × $\text{hassles}_{\text{within}}$ interactions. Of interest is the NED × $\text{hassles}_{\text{within}}$ interaction ($W_j(\breve{X}_{ij} - X_{\cdot j})$). The interaction was decomposed at ±1 SD using simple slope tests (Aiken & West, 1991; Preacher, Curran, & Bauer, 2006).

Between-subjects hypothesis. The between-subjects moderation hypothesis (NED × episodic stress, predicting depression) was tested using the PROCESS Macros for SPSS (Hayes, 2013). The interaction was probed using simple slope tests at ±1 *SD* (Aiken & West, 1991). For all longitudinal data analyses, we used Mplus (Muthén & Muthén, 1998–2012) to conduct path analysis. All models were fully saturated, making them equivalent to linear regression, but with the application of full-information maximum-

[1] For depressed mood (and other NE outcomes), we utilized items assessing "recent" (instead of current) depressed mood, covering the time span since their last prompt (or over the past 5 hours if they missed the prior prompt). This ensured that depressed mood (used as an outcome) was statistically independent from NED and NE intensity (calculated with current NE items). Results were comparable when using current depressed mood.

likelihood (FIML) data estimation procedures for missing data (Arbuckle, 1996). As a gold-standard missing data approach, FIML directly estimates parameters from the full sample; cases with incomplete data are included in computations, thus ensuring all available information is utilized in obtaining optimal parameter estimates (results were comparable using listwise deletion).

Sample size justification. Power analyses were conducted in G*Power (Faul, Erdfelder, Buchner, & Lang, 2009) and Optimal Design (Spybrook et al., 2011). Using estimated parameters derived from previous studies (Demiralp et al., 2012; Starr, Hershenberg et al., 2017), our N of 233 was powered at >95% for identifying effects for the association between NED and depression and for the within-subjects moderation of the association between daily hassles and depressed mood. There was insufficient basis to estimate parameters for testing the NED's moderation of SLEs and depression, but our sample size was comparable to that used in previous research examining self-reported emotional clarity as a moderator of the association between SLEs and depression (Stange et al., 2013).

Results

Descriptive Statistics, Bivariate Correlations, and Association Between NED and Depression

Descriptive statistics and bivariate correlations among study variables are presented in Table 1. As reported elsewhere (e.g., Starr & Huang, 2018), episodic stress was correlated with depression. Mean daily hassles were associated with mean daily depressed mood (both variables were aggregated across the full diary period), but not with episodic stress or depression, suggesting that SLEs and daily hassles represent unique forms of environmental stress, with SLEs more relevant to clinically significant depression. Supporting predictions, NED was significantly negatively related to depression, such that adolescents with lower NED reported higher depression, $b = -2.38$, $SE = .50$, $\beta = -.30$, $p < .001$, 95% CI [−3.37, −1.40]. This association was retained when controlling for NE intensity, $b = -1.53$, $SE = .55$, $\beta = -.19$, $p = .006$, 95% CI [−2.61, −.45].[2] Inclusion of demographic covariates (age, gender, race) did not alter results. NED was also associated with mean daily depressed mood and mean daily hassles, although the latter association became marginally significant ($p = .059$) when controlling for mean NE intensity. Further, youth meeting DSM–IV criteria for depression had significantly lower NED ($M = .76$, $SD = .09$) than those not meeting criteria ($M = .89$, $SD = .09$), $t(231) = -4.298$, $p < .001$.

Longitudinal Prediction of Depression Over Time

As shown in Table 1, NED was negatively correlated with T2 depression; however, in the path analysis model controlling for baseline depression, low NED only marginally predicted increased depression, $\beta = -.13$, $b = -1.68$, $SE = 1.00$, $p = .094$.[3] Moreover, controlling for emotional intensity reduced this association further, $\beta = -.04$, $b = -.49$, $SE = 1.08$, $p = .654$. Thus, NED does not appear to predict longitudinal changes in depression as a main effect in this sample.

Within-Subjects Moderation of Association Between Daily Hassles and Daily Depressed Mood (Replication of Starr, Hershenberg, et al., 2017 Results)

As described in the Data Analytic Plan section, we constructed a model wherein fluctuations in hassles, NED, and their interaction were entered into a multilevel model, with time since the first completed survey and time of day included as covariates, and depressed mood as the outcome. Of note, the interaction between $\text{hassles}_{\text{within}}$ and NED was significant, $b = -.36$, $SE = .06$, $p < .001$.[4] Decomposition revealed that, although hassles were significant in predicting depressed mood regardless of NED level, hassles more strongly predicted depressed mood at low NED ($b = .10$, $SE = .01$, $p < .001$, 95% CI [.07, .10]) than at high NED ($b = .03$, $SE = .01$, $p = .003$, 95% CI [.02, .05], consistent with predictions and prior findings (Starr, Hershenberg et al., 2017). Figure 1 illustrates the interaction. All results were retained when controlling for mean NE intensity (NED × $\text{hassles}_{\text{within}}$ $b = -.36$, $SE = .06$, $p < .001$, CI [−.47, −.24]), or for baseline depression ($p < .001$) as well as when demographic covariates (age, race, gender) were entered ($p < .001$).[5]

As a conservative measure, we ran an additional model that controlled for the effects of depressed mood reported at that morning's Prompt 1 survey (taken shortly after awakening), to examine if the interaction between NED and hassles represented a

[2] NED was similarly negatively correlated with depression when calculated using POMS subscales, $r = -.31$, $p < .001$.

[3] POMS subscale-calculated NED similarly did not predict longitudinal changes in depression: $\beta = -.04$, $p = .670$.

[4] NED calculated using the POMS subscales generated comparable results, NED × $\text{hassles}_{\text{within}}$ $b = -.20$, $SE = .04$, $p < .001$, with stronger associations between hassles and depressed mood for those at low levels of NED.

[5] We selected depressed mood as the a priori outcome in these analyses to directly replicate the Starr, Hershenberg et al. (2017) findings and to align analyses with the depressive outcomes in the between-subject depressive outcomes. As a post hoc exploratory test, we examined whether results replicated with the other three POMS-15 NE subscales: anxious mood, anger, and fatigue, or if they were unique to depression. Each subscale, when entered as an outcome, initially yielded significant interaction terms in initial models (anger: $b = -.51$, $SE = .21$, $p = .016$, anxious mood: $b = -.35$, $SE = .07$, $p < .001$, fatigue: $b = -.24$, $SE = .07$, $p < .001$). However, when controlling for concurrent depressed mood (as a fixed and random effect), the models predicting anger and anxious mood were no longer significant (anger $b = .14$, $SE = .19$, $p = .447$; anxious mood $b = -.02$, $SE = .06$, $p = .723$), suggesting initial results were inflated by these NE variables' concurrent associations with depressed mood. The model predicting fatigue remained significant ($b = -.13$, $SE = .07$, $p = .046$) when controlling for depressed mood, but became nonsignificant ($p = .129$) when controlling for other NEs. In contrast, the model predicting depressed mood remained robustly significant when controlling simultaneously for fatigue, anger, and anxious mood (interaction $b = -.22$, $SE = .05$, $p < .001$; note that NE covariates were entered only as fixed effects in this model because including all three as random effects prevented model convergence, but near-identical results were generated when entering NE random effects individually or in pairs). We conclude depressed mood findings uniquely hold over and above other NE mood states, although further research should consider exploring the intriguing interplay between stress, NED, and fatigue.

Table 1
Bivariate Correlations Among Primary Study Variables and Descriptive Statistics

Study variables	1	2	3	4	5	6	7	8
1. NED	—							
2. Depression rating	−.30***	—						
3. Total episodic stress	−.09	.27***	—					
4. NE intensity	−.46***	.31***	.17**	—				
5. Mean daily hassles	−.24***	.09	.10	.69***	—			
6. Mean daily depressed mood	−.46***	.34***	.14*	.83***	.59***	—		
7. T2 Depression rating	−.20**	.26***	.27***	.29***	.15*	.26***	—	
8. T2 Total episodic stress	−.24**	.22**	.24**	.31***	.17*	.25**	.46***	—
M	.88	.29	.17	1.52	1.64	1.36	.82	5.18
SD	.10	.79	.72	.44	1.74	.53	1.24	4.22

Note. NED = negative emotion differentiation; NE = negative emotion.
* $p < .05$. ** $p < .01$. *** $p < .001$.

shift in mood beyond what was reported earlier in the day[6] (White & Shih, 2012). Results were unchanged when morning mood was included (interaction $p < .001$, same decomposition pattern).

Between-Subjects Moderation of Association Between Episodic Stress and Depression

Cross-sectional model. Total episodic stress and NED (both centered) and their interaction were entered as predictors and depression was entered as the outcome. Of note, the interaction term was significant, β = −.22, $b = -.22$, $SE = .09$, $p = .019$, 95% CI [−.40, −.04].[7] Decomposition supported the hypothesized pattern: episodic stress was strongly associated with depression at low levels of NED ($b = .06$, $SE = .01$, $p < .001$) but not at high NED ($b = .01$, $SE = .01$, $p = .343$), illustrated in Figure 2. These results were retained when controlling for NE intensity (interaction $p = .021$), with a similar decomposition pattern, and were also robust following inclusion of demographic variables.[8]

Longitudinal models. In our primary longitudinal model, NED and T1 episodic stress (both centered) and their interaction were entered as predictors of T2 depression, with T1 depression included as a covariate. The interaction term was significant; β = −.19, $b = -.46$, $SE = .17$, $p = .010$, 95% CI [−.81, −.11].[9]

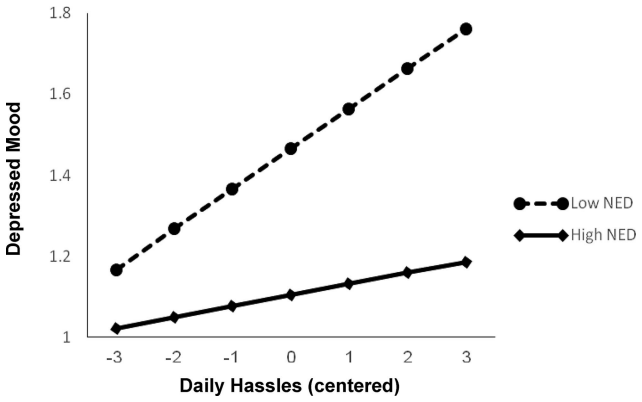

Figure 1. Within-subjects associations between recent daily hassles and depressed mood, at high and low levels of negative emotion differentiation (NED). High and low NED corresponds to 1 *SD* above and below the mean.

Decomposition was consistent with the hypothesized pattern; at low levels of NED, episodic stress predicted increases in depression at T2 ($b = .11$, $SE = .03$, $p < .001$), whereas at high NED, T1 episodic stress did not predict changes in depression at T2 ($b = .02$, $SE = .03$, $p = .467$). These findings are illustrated in Figure 3. As an alternative explanation, we considered whether NED was more predictive of increases of depression over time under stressful contexts. At low levels of stress, NED did not predict increases in depression over time ($b = .93$, $SE = 1.38$, $p = .499$), but at high levels of stress it did ($b = -3.40$, $SE = 1.18$, $p = .004$). These findings were unchanged when controlling for emotional intensity (interaction $p = .012$) and demographic variables (interaction $p = .005$).

Although episodic stress and NED were not correlated in our sample, some evidence suggests that stress exposure influences within-subject variations in NED (Erbas et al., 2018), which could have potentially affected the above models as NED and episodic stress were both assessed at baseline. Therefore, we conducted a final model wherein T1 NED moderated the association between T2 episodic stress and T2 depression, using current T2 depression ratings as the outcome variable. We also controlled for T1 depression, so the outcome represented increase in depression prior to the SLE exposure. Results were consistent with other reported models, with significant interaction term (β = −.26, $b = -.33$, $SE = .08$, $p < .001$, CI [−.48, −.17],[10] and a similar pattern of decomposition as described above.

[6] Note that Prompt 1 depressed mood was not incorporated as an outcome in our models (as hassles were not assessed at Prompt 1), so its inclusion did not constitute a violation of nonindependence of the random intercept and dependent variables that can occur when lagged dependent variables are entered as predictors in multilevel models (Allison, 2015).

[7] Comparable results were generated using NED calculated with the POMS subscale: $b = -.18$, $SE = .07$, $p = .008$.

[8] Though we were underpowered to replicate analyses with categorical depression diagnoses (given low rates of depression in our community sample), we did nevertheless examine if findings would replicate using depression diagnosis (i.e., meeting DSM-criteria) as a categorical outcome. Findings were not significant when including categorical depression diagnosis as an outcome.

[9] Results were comparable using NED calculated using POMS subscales: $b = -.31$, $SE = .14$, $p = .025$.

[10] Using NED computed with POMS subscales, results were comparable: $b = -.27$, $SE = .06$, $p < .001$.

Supplemental Analyses: PED

Primary analyses focused on NED rather than PED because (a) NED has been more consistently linked to depression (Demiralp et al., 2012; Starr, Hershenberg et al., 2017), and NEs are likely more relevant in the aftermath of stressors, and (b) we administered fewer PE items (happy, proud, joyful, lively, cheerful), several of which are conceptually similar to each other (e.g., cheerful and happy) and therefore likely more difficult to differentiate. With these caveats in mind, as supplemental analyses, we explored whether results were specific to NED versus PED, following analogous data analytic procedures as described previously. PED was significantly correlated with NED, $r = .31$, $p < .001$ but not with depression, $r = -.10$, $p = .116$. PED did not predict changes in depression at T2 as a main effect ($b = -.41$, $SE = .46$, $p = .374$). In initial models, PED did significantly moderate the within-subjects association between daily hassles and depressed mood (interaction $b = -.09$, $SE = .04$, $p = .011$); however, given the correlation between NED and PED, we examined whether this held when controlling for the significant interaction between NED and hassles (and the main effect of NED). In the resulting model, the NED × Hassles effect remained significant ($b = -.34$, $SE = .07$, $p < .001$) but the PED × Hassles effect did not ($b = -.02$, $SE = .04$, $p = .563$), leading us to conclude that the originally significant model was due to the high correlation between NED and PED. Likewise, PED marginally moderated the cross-sectional association between T1 episodic stress and T1 depression ($b = -.09$, $SE = .05$, $p = .075$); however, when the NED × Stress and PED × Stress interactions were entered simultaneously (along with all main effects), the NED × SLEs interaction was significant ($b = -.20$, $SE = .10$, $p = .041$), but the PED × SLEs interaction was not ($b = -.04$, $SE = .05$, $p = .400$). PED did not moderate the prospective association between T1 SLEs and T2 depression ($b = -.09$, $SE = .08$, $p = .251$). PED marginally moderated the association between T2 SLEs and T2 depression ($b = -.08$, $SE = .06$, $p = .099$), but again, when simultaneously entering both PED × SLEs and NED × SLEs interaction models, only NED was a significant moderator (NED × SLEs $b = -.33$, $SE = .09$, $p < .001$; PED × SLEs $b = .001$, $SE = .05$, $p = .992$). Therefore, consistent with previous studies suggesting a lack of association between PED and depression (Demiralp et al., 2012; Starr, Hershenberg et al., 2017), we found no evidence that PED uniquely predicted depression alone or in interaction with stressful contexts, after taking into account covariance with NED.

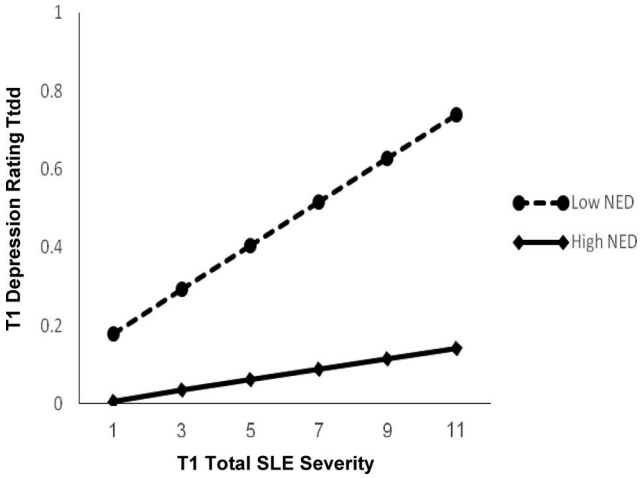

Figure 2. Severity rating of T1 depression as a function of T1 episodic stress (total stressful life event severity), at high and low levels of negative emotion differentiation (NED). High and low NED corresponds to 1 *SD* above and below the mean. Total episodic stress refers to the sum of severity ratings across all reported events. Depression rating refers to interviewer-assigned dimensional rating of major depressive disorder/dysthymia diagnoses.

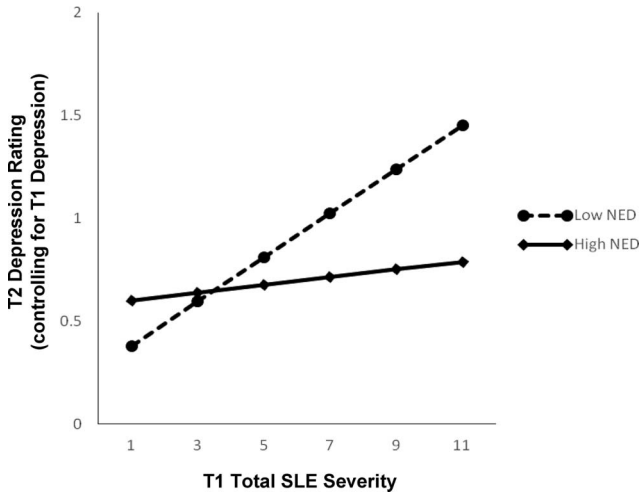

Figure 3. Changes in depression ratings at T2 (controlling for T1 depression) as a function of T1 episodic stress (total stressful life event severity), at high and low levels of negative emotion differentiation (NED). High and low NED corresponds to 1 *SD* above and below the mean. Total episodic stress refers to the sum of severity ratings across all reported events. Depression rating refers to interviewer-assigned dimensional rating of major depressive disorder/dysthymia diagnoses.

Discussion

The current study sought to clarify the relationship between NED and depression in a community sample of adolescents. The study incorporated multiple components (EMA, gold-standard stress assessment, longitudinal outcomes), with temporal resolution ranging from hours to 1.5 years, and provides strong support for a novel diathesis-stress model of NED depression. Supporting hypotheses, NED was cross-sectionally (but not longitudinally) related to depression, such that adolescents with lower NED reported higher depression, beyond the effects of emotional intensity. However, this association was qualified by exposure to stressful contexts, with low NED predicting intensified cross-sectional and longitudinal associations between acute, naturalistic stress exposure and depression. Evidence for the role of NED in modifying stress reactivity was further bolstered by within-subjects findings showing that NED moderated within-person associations between daily stressors and momentary fluctuations in depressed mood (directly replicating a prior study; Starr, Hershenberg et al., 2017). All effects were specific to NED versus PED, suggesting it is differentiation of negative emotional states, as opposed to overall emotion granularity, that confers depression risk in the context of stress (although note that PED may be better assessed using a wider range of PE variables).

Our finding that NED was significantly, negatively associated with depression in adolescence aligns with other studies linking low NED to depression and depressive symptoms in older samples (Demiralp et al., 2012; Erbas et al., 2014; Goldston et al., 1992; Starr, Hershenberg et al., 2017). However, NED did not directly predict increases in depression over time. This critical finding is, to our knowledge, unique to the literature; prospective research on NED is limited, and we are unaware of other published studies that have examined the long-term depressive consequences of low granularity. Viewed in isolation, this may suggest that the common assumption in the literature that NED functions as an etiological variable in depression is misplaced. However, the longitudinal association between NED and depression was qualified by stress exposure. That is, NED did predict increases in depression over time, but only when the adolescent had experienced high levels of recent stress, aligning with our hypothesized diathesis–stress model. This finding was highly consistent, replicating whether stress, depression, and NED were assessed concurrently with NED or at separate longitudinal time points, and controlling for a range of covariates. These findings even emerged at the daily level in our EMA analyses. We thus conclude that low NED likely does function as a risk factor for depression, but only under high-risk environmental conditions. In contrast, low granularity appears to be a fairly inert trait (at least with respect to depression) when environmental demands are low. Note that this does not eliminate the prospect that depression also dampens emotional discrimination, as NED and depression may have a bidirectional, dynamic relationship; future research that assesses both these constructs at multiple time points should further clarify their temporal relationship.

Why does low NED make adolescents more vulnerable to depression following stress exposure? Lack of emotion differentiation has significant implications, as emotions communicate information about sources of distress, and possible attentional and behavioral regulatory strategies. People with difficulties discriminating between and labeling NEs may fail to orient to the causes and consequences of their emotional responses to stressors, leaving them less prepared to effectively down-regulate NEs. Research suggests low NED is linked to trait-level maladaptive emotion regulation styles and difficulties implementing effective emotion regulation strategies (Barrett et al., 2001; Kashdan et al., 2015). As such, low NED may be linked to deficits in emotion regulation that, in turn, may make the emotional aftermath of stressors more difficult to manage and lead to the development of depressive symptoms. Further, stress is strongly linked to higher cognitive load and reduced ability to utilize cognitive resources like decreased working memory, retrieval, and attention, including at the daily level (Kuhlmann, Piel, & Wolf, 2005; Sliwinski, Smyth, Hofer, & Stawski, 2006), which may amplify the effects of low NED. Finally, adolescents who lack emotion differentiation skills may devote excessive resources toward understanding emotional experiences (Judd et al., 1998), perhaps at the expense of managing stressful contexts.

Adolescence is a particularly key developmental period for examination of these processes. During adolescence, not only do rates of depression surge (Avenevoli et al., 2015), but changing challenges in both interpersonal and intrapersonal domains present novel and unique stressors (Compas & Phares, 1991). In addition, developmental changes in responsivity to both stressors and emotional information also occur during this period, with adolescents exhibiting increased physiological stress responsivity as well as reactivity to emotional stimuli (Silk et al., 2009; Stroud et al., 2009). These psychosocial and neurophysiological changes may place increased demands on adolescents' ability to respond adaptively to stressors, making emotion differentiation skills especially critical. For example, increased sensitivity and reactivity to affective stimuli may lead to greater sensitivity to developmentally salient stressors (e.g., peer rejection, romantic problems). Existing difficulties with emotion differentiation in the presence of elevated stress may interfere with the ability to implement adaptive coping strategies and place adolescents at particular risk for the development of internalizing symptoms. In turn, depressive symptoms occurring during adolescence predict persistent, long-term impairment (Aalto-Setälä, Marttunen, Tuulio-Henriksson, Poikolainen, & Lönnqvist, 2002). Adolescents also exhibit increased impulsivity, risk-taking, and reward-seeking behaviors (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011; Forbes et al., 2010; Galvan, 2010; L. Steinberg, 2010), some of which have been linked to particularly negative outcomes among those with poor emotion differentiation (e.g., Dixon-Gordon et al., 2014; Kashdan et al., 2010). Thus, poor NED during adolescence has potential to influence long-term trajectories of well-being across a variety of outcomes.

By elucidating associations between NED, stress, and depression, this study increases understanding of emotional dynamics associated with depression and identifies targets within interventions that may prevent onset, or reduce severity, of depressive episodes. Research suggests treatment promoting expansion of emotional vocabulary to foster flexible, contextualized identification of discrete emotions has inherent regulatory properties (Kircanski, Lieberman, & Craske, 2012). This approach, a process called *affect labeling*, reduces emotional reactivity and bolsters emotion regulation strategies, which may protect against development of depression (Lieberman et al., 2007). Mindfulness interventions also help individuals recognize, label, and accept their emotional experience, thereby reducing vulnerability to nonspecific, overwhelming emotions (Hill & Updegraff, 2012). Moreover, our finding that NED interacts with environmental stress to predict depression suggests it is especially important to target interventions at high-risk, stress-exposed youth.

Important study limitations should be noted. Although this study breaks new ground by examining the depressive consequences of low NED over a significant follow-up period, the EMA was only administered at baseline, and therefore we could not examine NED as a longitudinal outcome. As noted previously, there may be important reciprocal associations between NED and affective functioning, and further longitudinal research (e.g., measurement burst designs) are needed to clarify these associations. Moreover, daily surveys could not be randomized across the day because of school schedules. Demands of school schedules are common barriers in adolescent studies using intensive longitudinal methods, which is often addressed by limiting daily survey components to afterschool hours, weekends, or summer holidays (e.g., Ranzenhofer et al., 2014; Silk et al., 2011), but which prevents evaluation of school experiences, contexts in which stressors often occur. Our study design attempted to balance this by prompting survey completion during the school day at times that corresponded with school schedules. Finally, this was a community

sample and rates of clinical depression were low (although consistent with point prevalence rates of depression in this age group; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Investigations of NED in clinically depressed samples are rare (Demiralp et al., 2012), and this is a clear direction for future research. Finally, some differential attrition occurred, with adolescents with low NED at T1 more likely to drop out than those with higher NED. Notably, T1 depression, stress, and demographic variables did not predict attrition, and it is conceptually fascinating that NED more reliably predicted drop-out than these more traditional variables. That said, results should be interpreted with appropriate caution.

Study limitations are balanced by several strengths, including the use of gold-standard assessment methods for depression (i.e., clinical interviews), NED (i.e., intensive longitudinal reports of negative emotions), and life stress (i.e., interview-based coding of contextual threat), longitudinal design, and the examination of NED in adolescence. Findings lend further support for NED as a critical skill tied to emotion functioning, and suggest its depressive aftermath is best understood in the context of life stress.

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