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Stress exposure in at-risk, depressed, and suicidal adolescents

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Background: Stress exposure contributes to the onset, maintenance, and recurrence of major depressive disorder (MDD) in adolescents. However, the precise stress facets (e.g. chronicity, domain) most strongly linked to outcomes at different stages along the depression severity continuum remain unclear. Across two studies, chronic and episodic stressors were comprehensively assessed among: (a) healthy youth with (High-Risk [HR]) and without (Low-Risk [LR]) a maternal history of MDD and (b) adolescents with current MDD and suicide ideation and healthy controls (HC). **Method:** Study 1 included LR (n = 65) and HR (n = 22) 12- to 14-year-olds (49 females; 56.32%) with no lifetime history of mental disorders. Study 2 enrolled 87 mid-to-late adolescents (64 females; 73.56%), including 57 MDD youth from a short-term intensive treatment service and 30 HCs from the community. All depressed youth reported recent suicide ideation; some had no lifetime history suicide attempts (SI; n = 31) and others reported at least one past year attempt (SA; n = 26). The Life Events and Difficulties Schedule was used to capture stressor severity in both studies. **Results:** We used multiple linear regression models that adjusted for demographic and clinical covariates. Being in the HR versus LR group was associated with more severe chronic (β = .22, CI_{95} = 0.01–0.42, p = .041), independent (β = .34, CI_{95} = 0.12–0.56, p = .003), and interpersonal (β = .23, CI_{95} = 0.004–0.45, p = .047) stress severity. By contrast, the MDD group reported significantly more severe chronic (β = .62, CI_{95} = 0.45–0.79, p < .001) and dependent (β = .41, CI_{95} = 0.21–0.61, p < .001) stress than the HC group, but not independent (p = .083) stress. Stress severity did not differ between recent attempters versus youth who reported suicide ideation alone (SA vs. SI contrast). However, the SA group reported a higher rate of targeted rejection events (RR = 3.53, CI_{95} = 1.17–10.70, p = .026). **Conclusions:** Our findings clarify the stressor features that may most strongly contribute to adolescent depression and its clinical correlates at two important points along depression's clinical course. **Keywords:** Major depressive disorder; life events; stress; maternal depression; suicidal behaviour.

Introduction

Decades of research show that life stress exposure is linked to the onset and maintenance of major depressive disorder (MDD) in adolescents (Rnic et al., 2023; Vrshek-Schallhorn, Ditcheva, & Corneau, 2020). However, the empirical foundation of the basic stress–depression relationship rests on research in middle-to-late adolescent community samples (Hammen, 2016; Rnic et al., 2023). We lack a fine-grained understanding of the stressor features (e.g. domain, chronicity) that may contribute to depression at other points along its clinical course. At the earlier/milder end of the depression continuum, this involves examining differences in stress exposure among nondepressed youth with and without familial depression risk (Goodman, 2020). At the later/severe end of the continuum, few studies have examined patterns of stress exposure in severely depressed adolescents. Further, it is unclear how stressor features may be differentially related to clinical characteristics (e.g. suicidal thoughts and behaviors; STBs) among depressed youth (Vrshek-Schallhorn et al., 2020). Focusing on youth occupying understudied poles of the depression continuum is critical because the types of stressors most implicated in adolescent depression may change over time and with experience (Hammen, 2016).

We address empirical gaps regarding the type(s) of stress most strongly associated with MDD using a sample of nondepressed youth (12–14 years old) with and without a maternal history of MDD (Study 1) and hospitalized adolescents with current depression and suicide ideation and nondepressed controls (Study 2). We build on prior work by rigorously characterizing clinical history and using gold-standard measures of life stress and suicidal behaviors (Study 2) that reliably capture their nature and timing. The studies share two important methodological features. First, chronic difficulties (stressors lasting 1 month or more) are seldom examined among at-risk or currently depressed adolescents, particularly alongside episodic events. This omission has persisted despite evidence that chronic stressors uniquely predict MDD in young adults (Vrshek-Schallhorn et al., 2015). Second, the relative contributions of different types of stress to depression are rarely tested (c.f., Rnic et al., 2023; Vrshek-Schallhorn et al., 2015); stressors with different features must be examined 'head-to-head' to determine if a particular domain of stress is more salient

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to depression risk and/or MDD. Together, the present studies build a more comprehensive picture of how life stress is linked to depression and its clinical correlates across a continuum of severity.

Life stress exposure among offspring of depressed parents

Having a depressed parent confers a threefold risk for MDD among offspring (Weissman et al., 2006). Maternal depression is related to elevated chronic and episodic stress (Vrshek-Schallhorn et al., 2020), and consequently, offspring of mothers with MDD (i.e. HR children) encounter more stressful environments (Goodman, 2020). Many of these stressors are outside of the child's control (*independent*), such as parental job loss and unemployment. Maternal depression may also contribute indirectly to *dependent* stressors, which are at least partly a product of offspring characteristics or behaviors (e.g. a major argument). Indeed, children of depressed mothers may learn maladaptive communication patterns and poor interpersonal problem-solving that contribute to dependent stressors, particularly in the interpersonal domain (Hammen, 1991, 2009).

Studies testing the link between maternal risk for depression and stressors measured with contextual, interview-based assessments are mixed. Generally, HR youth experience more severe total episodic life stress relative to those with no maternal history of MDD (LR youth) (Adrian & Hammen, 1993; Feurer et al., 2018; Hammen, Brennan, & Shih, 2004; Hammen, Hazel, Brennan, & Najman, 2012; Morris, Kouros, Hellman, Rao, & Garber, 2014). However, among studies that separate stressors into domains, some find that HR youth experience more frequent and/or severe dependent interpersonal events than LR youth (Feurer, Hammen, & Gibb, 2016; Hammen & Brennan, 2001), while others find no group differences (Carter & Garber, 2011; Gershon et al., 2011) or find that differences are not specific to interpersonal events (Feurer, Burkhouse, Siegle, & Gibb, 2017).

Two methodological limitations have contributed to the mixed findings above. First, some prior studies use broad age ranges (i.e. 8- to 15-years-old; Adrian & Hammen, 1993; Feurer et al., 2016; Gershon et al., 2011). The frequency and content of life stress changes from mid-childhood to mid-adolescence (Rudolph, 2009) and the salience of certain stressors (e.g. peer conflict) may also increase in this period (Pfeifer & Allen, 2021). Age-related differences in stress exposure and reactivity could explain some of the inconsistent effects of maternal depression. Second, chronic difficulties and episodic stressors are correlated, and are temporally dependent in some cases (Bifulco et al., 1989). When including chronic stress, extant research has typically *only* measured interpersonal domains (e.g. peer conflict), finding that HR youth have more frequent and severe stressors relative to their LR peers (Hammen et al., 2004, 2012; Herr, Hammen, & Brennan, 2007). Studies that have defined chronic stress more broadly are mixed; Gershon et al. (2011) found that relative to LR youth, HR youth reported more severe interpersonal and non-interpersonal chronic stress, while Feurer et al. (2016) found differences for interpersonal, but not academic, chronic stress. Another study found that children of depressed mothers had greater combined chronic and episodic stress severity relative to children of nondepressed mothers (Adrian & Hammen, 1993). This ignores the unique links that chronic and episodic stressors may have with maternal MDD (Goodman, 2020). Overall, chronic stress is seldom considered in research on the impacts of maternal depression, and the effects of chronic versus episodic stressors have not been compared. Omitting stressor chronicity and type may contribute to misleading conclusions about the stressors experienced by HR relative to LR youth.

Stress exposure, adolescent depression, and suicide

Adolescents with MDD experience more frequent and severe life stressors than nondepressed youth. This effect has been explained using the stress generation framework (Hammen, 1991), which proposes that characteristics of depressed individuals contribute to their experience of subsequent stressors. The model hypothesizes depression is specifically linked to dependent stressors but does not impact exposure to independent stressors (Hammen, 2020; Rnic et al., 2023). Several studies have supported this association (see Hammen, 2020; Liu & Alloy, 2010) and there is some evidence that the effect might be strongest for *interpersonal* dependent stress in youth (Conway, Hammen, & Brennan, 2012). Nonetheless, depressed adolescents also experience higher rates of *independent* stressors in some studies (Harkness & Stewart, 2009; Kercher, Rapee, & Schniering, 2009) and the relative strength of associations among depression and distinct types of stress are rarely tested. Further, adolescent depression may be linked to chronic stress (Mineka et al., 2020; Rudolph et al., 2000; Vrshek-Schallhorn et al., 2015), but chronic and episodic stress are rarely examined simultaneously. Addressing these gaps will deepen knowledge regarding types of stress that most contribute to persistent depression in youth.

Among depressed adolescents, the contribution of stress toward the occurrence and severity of STBs – key clinical correlates of depression – has also received relatively limited attention. Depression is a strong correlate of suicide ideation but does not predict suicide attempts among ideators (Alqueza et al., 2023). Theories of suicide posit that stress exposure may generate suicidogenic cognitive–affective states and may thus contribute to more severe suicidal ideation. Further, stressors may trigger suicide attempts among ideators directly or

indirectly by increasing the person's capability to engage in potentially lethal, self-directed harm (see Klonsky, Saffer, & Bryan, 2018). Life stress is generally associated with a range of suicide-related outcomes (Liu & Miller, 2014). However, studies rarely elucidate: (a) the differential importance of distinct types of life stress vis-à-vis STBs and (b) whether stress exposure is related to ideation, in general, or suicide attempts, specifically. This is critical because many putative risk factors for adolescent STBs (depression, hopelessness, and psychiatric disorders) are associated with suicidal thinking but do not distinguish those who have and have not made a prior attempt (Allison et al., 2021; Alqueza et al., 2023; Stewart, Esposito, et al., 2017; Stewart et al., 2019).

Studies examining stress exposure in adolescent ideators versus attempters have used questionnaire measures of stress that are limited by response biases (Harkness & Monroe, 2016). They find that more subjectively severe general life stress is associated with greater odds of being a suicide attempter versus ideator (King et al., 2001; Liu & Tein, 2005; Mars et al., 2019; O'Connor, Rasmussen, & Hawton, 2012). These studies have applied broad (e.g. past year) and overlapping STB and stress assessment periods. This approach cannot determine whether effects are driven by stressors that precede or follow STBs. Longitudinal studies that overcome this limitation have focused on a narrow set of stressors and generally have not found a link between stress and subsequent suicidal behaviors (Daniel, Goldston, Erkanli, Heilbron, & Franklin, 2017; Massing-Schaffer et al., 2019; Pettit, Green, Grover, Schatte, & Morgan, 2011; Stone, Liu, & Yen, 2014). Studies must comprehensively assess the severity and timing of a broad range of stressors to advance understanding of the link between stressors and suicide.

Present studies

The present studies investigated the relative associations of stressors with distinct features (domain; chronicity) at underexamined points in the course of adolescent MDD. Study 1 enrolled healthy 12- to 14-year-olds with and without a maternal MDD history. We used the Life Events and Difficulties Schedule (LEDS-II; Bifulco et al., 1989) to comprehensively assess key domains of episodic and chronic stressors and used directly compared the magnitude of the association between risk status and each stress variable. We hypothesized that, relative to LR youth, HR youth would experience more severe stressors. Given mixed findings in extant research, we did not make hypotheses regarding specific types of stress.

Study 2 enrolled adolescents with MDD and current suicide ideation receiving treatment and healthy controls (HCs) from the community. We hypothesized that, relative to HC youth, adolescents with MDD and suicide ideation would experience more severe stress, particularly in the interpersonal domain. Among the depressed adolescents with current suicide ideation, we also examined differences in stress exposure between those with prior suicide attempts (i.e. suicide attempters; SA) and those without (i.e. suicide ideators; SI). We hypothesized that being in the SA versus SI group would be associated with greater interpersonal stress severity. Last, stressors involving rejection may be particularly potent contributors to both depression (Slavich, Thornton, Torres, Monroe, & Gotlib, 2009) and adolescent suicidal behavior (Cheek, Goldston, Erkanli, Massing-Schaffer, & Liu, 2020; Cheek, Reiter-Lavery, & Goldston, 2020; Stewart et al., 2019). Thus, we tested where targeted rejection (TR) – events involving social demotion (e.g. losing relationships) that are intentionally and exclusively directed at the person (Slavich et al., 2009) – was associated with suicide attempts among ideators. We expected that the SA group would experience more frequent TR than the SI group.[1]

Study 1

Method

Study 1 and 2 procedures were approved by the Partners Institutional Review Board. Sample size and statistical power considerations for both studies are discussed in Appendix S1.

Participants. Participants were a subset of 93 mother–child dyads who participated in a larger longitudinal study investigating neural predictors of first onset MDD in early adolescents (Belleau et al., 2021; Pagliaccio, Kumar, Kamath, Pizzagalli, & Auerbach, 2023). At an initial laboratory appointment, dyads recruited to the umbrella study from the community provided consent, and then mothers and youth (12–14 years old) were administered clinical interviews – the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I/P; First, Spitzer, Gibbon, & Williams, 2002) and Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) – to assess the presence/absence of lifetime depressive disorders in mothers, and to confirm the absence of lifetime psychopathology in youth. Full inclusion and exclusion procedures are detailed in Appendix S2.

Of the 125 participants who were eligible to participate after baseline, 92 (73.60%) had mothers who reported no lifetime history of depression at baseline (LR) and 33 (26.40%) had mothers who reported a lifetime unipolar depressive disorder sometime in their lifetimes (HR). The LEDS-II was administered at a follow-up approximately 6 months later and 93 youth completed the interview. Six additional participants had complete missing data

on at least one psychiatric symptom measure; these participants were excluded from primary analyses, leaving a final sample of 87 (LR = 65, HR = 22) adolescents (see Table 1 for demographic and clinical characteristics). In the final sample, HR mothers reported the following unipolar mood disorders: MDD (n = 20, 90.91%), depressive disorder NOS (n = 1, 4.54%), and dysthymia (n = 1, 4.45%)[2] (see Table 2). Participants who were excluded because they did not complete the LEDS-II or other measures (n = 38) did not significantly differ from included participants on sociodemographic, clinical, or grouping variables (all ps >.16).

Measures. Questionnaires: Adolescents completed the Mood and Feelings Questionnaire (MFQ; Costello & Angold, 1988), the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997), and the Snaith-Hamilton Pleasures Scale (SHAPS; Snaith et al., 1995) to measure their recent depression, anxiety, and anhedonia symptom severity, respectively. Additionally, youth completed the self-report version of the Tanner Scale (Tanner & Davies, 1985) to assess pubertal status. Mothers completed the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) and SHAPS to assess depression and anhedonia symptom severity. The internal consistency of these measures in our sample ranged from good to excellent (Table 3).

Table 2 Diagnostic characteristics for mothers in the HR group (n = 22) based on the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I/P) assessment administered in Study 1

	Current		Past	
	n	%	n	%
Major depressive disorder	1	4.55	19	86.36
Depressive disorder NOS	0	0.00	1	4.55
Dysthymia	0	0.00	1	4.55
Any unipolar mood disorder[a]	1	4.55	20	90.91
Generalized anxiety disorder	3	13.64	–	–
Specific phobia	2	9.09	2	9.09
Social phobia	2	9.09	5	22.73
Panic disorder	0	0.00	1	4.55
Post traumatic stress disorder	2	9.09	4	18.18
Anorexia nervosa	0	0.00	2	9.09
Bulimia nervosa	0	0.00	2	9.09
Alcohol use disorders	0	0.00	5	22.73
Substance use disorders	0	0.00	2	9.09

NOS, not otherwise specified.
[a]One mother met for past subthreshold major depressive disorder according to the SCID-I/P.

Life stress: The LEDS-II (Bifulco et al., 1989; adolescent version, Frank, Matty, & Anderson, 1997). The interview queried life events and difficulties across 10 domains (e.g. romantic relationships, housing) that occurred between adolescents' baseline and follow-up appointments (M_{days} = 185.87, SD_{days} = 32.78, range = 148–429 days). Given the

Table 1 Descriptive statistics and clinical characteristics for the Study 1 sample of LR (n = 65) and HR (n = 22) adolescents

	Descriptive statistics (M [SD] or n [%])				
	LR	HR	t/χ^2	p	Φ/d
Demographics					
Sex (Female)	36 (55.38)	13 (59.09)	0.09	.762	0.03
Age (in years)	12.88 (0.80)	12.95 (0.79)	0.39	.694	0.09
Tanner pubertal rating	3.03 (0.63)	2.95 (0.68)	0.55	.582	
Race					
White	56 (86.15)	19 (86.36)	1.99	.371	0.15
Black	0.00	0.00			
Asian	4 (4.60)	0 (0.00)			
Pacific Islander	0 (0.00)	0 (0.00)			
More than one race	5 (7.69)	3 (13.64)			
Psychiatric symptoms					
MFQ	4.10 (4.73)	8.17 (8.73)	2.09^	.047	0.58
MASC	29.43 (9.01)	38.51 (14.92)	2.69^	.012	0.74
SHAPS-CR	20.05 (6.33)	20.95 (4.26)	0.62	.536	0.17
Mothers' psychiatric symptoms					
BDI-II	2.79 (3.11)	6.25 (6.99)	2.25^	.034	0.64
SHAPS-M	18.21 (4.42)	18.50 (4.45)	0.26	.794	0.07
Severity of life stress exposure					
Chronic	0.65 (1.24)	2.00 (2.07)	2.90^	.008	0.91
Independent	0.46 (0.87)	1.77 (2.39)	2.52^	.019	0.93
Non-interpersonal	0.85 (2.03)	0.91 (1.19)	0.14	.891	0.03
Interpersonal	0.42 (0.68)	1.23 (2.18)	1.72^	.100	0.66
Targeted rejection	1 (1.54)	3 (13.64)	–	–	–

Statistics computed using degrees of freedom that were adjusted due to significantly unequal variances are denoted with ^. CR, child/adolescent report; M, mother; MASC, Mood and Anxiety Symptom Questionnaire; MFQ, Mood and Feelings Questionnaire; SHAPS, Snaith-Hamilton Pleasures Scale.

Table 3 Correlations (Spearman's rho [ρ]) and descriptive statistics for stress and symptom variables in the Study 1 sample (n = 87)

	1.	2.	3.	4.	5.	6.	7.	8.	9.
Bivariate correlations									
1. Independent stress	–	.05	.13	.30**	.08	.21*	.09	−.10	.005
2. Non-interpersonal stress		–	.03	.31**	.07	.18	−.01	.10	.02
3. Interpersonal stress			–	.12	.21	.05	.05	−.09	−.27*
4. Chronic stress				–	.40***	.38***	.16	.03	.07
5. MFQ					–	.52***	.27*	.03	−.09
6. MASC						–	.16	−.02	−.19
7. SHAPS-CR							–	.10	−.06
8. BDI-II-M								–	.17
9. SHAPS-M									–
Descriptive statistics									
Mean	0.79	0.86	0.62	0.99	5.13	31.73	20.28	3.66	18.29
Standard deviation	1.51	1.84	1.28	1.60	6.20	11.42	5.87	4.63	4.40
Range	0–10	0–15	0–9	0–6	0–39	6–59	14–56	0–24	14–30
Skewness	3.30	5.62	4.03	1.55	2.52	0.26	2.72	1.99	0.73
Kurtosis	15.69	40.61	22.12	1.44	9.98	−0.06	14.69	4.87	−0.43
Reliability (α)	–	–	–	–	0.902	0.843	0.913	0.854	0.890

BDI-II, Beck Depression Inventory-II; M, mother; MASC, Mood and Anxiety Symptom Questionnaire; MFQ, Mood and Feelings Questionnaire; SHAPS, Snaith–Hamilton Pleasure Scale; CR, child/adolescent report.
*p < .05; **p < .01; ***p < .001.

variability in the amount of time the LEDS-II interviews covered, we analyzed data extracted from the 4 months prior to the interview. This period maximized participant retention in analyses while ensuring reliable retrospective recall (Johnson, 2005; Paykel, 1997), consistent with past adolescent research (Harkness, Bruce, & Lumley, 2006).

Interviews were condensed into stressor vignettes and presented to two independent raters who used the LEDS-II manual – which includes detailed rules and over 5,000 examples – to standardize ratings. Life events were scored on a 5-point severity scale: 1 = *little/none*, 2 = *some*, 3 = *low moderate*, 4 = *high moderate*, and 5 = *marked*. Raters also classified events as *independent, dependent interpersonal*, or *dependent non-interpersonal*. We computed separate total severity scores for each category of event; participants with no events in a category were assigned scores of 0. Difficulties (i.e. stressors persisting for at least 1 month) were rated on a 6-point severity scale: 1 = *very mild*, 2 = *mild*, 3 = *low moderate*, 4 = *high moderate*, 5 = *low marked*, and 6 = *high marked*. Total chronic stress was the sum of scores for all difficulties adolescents endorsed (see Appendix S3 for further detail on LEDS-II procedures).

We also rated life events for the presence or absence of targeted rejection (TR). TR is present when the following criteria are met (Slavich et al., 2009): (a) rejection of the interviewee by another person or group is intentional; (b) only the interviewee experiences the direct impacts of rejection; and (c) the event results in the interviewee losing social status (e.g. going from having a group of friends to not having them). TR events occurred infrequently (n = 4, 4.60%) in this sample; thus, we included TR in Table 1 for descriptive purposes, but group differences were not tested.

Data analysis. We fit four multiple linear regression models to test the association between familial depression risk (LR vs HR) and stressor severity. The dependent variables in our models were chronic, independent, dependent non-interpersonal, and dependent interpersonal stress severity. In each model, we first entered a dummy-coded familial depression risk variable on Step 1. We then entered covariates on Step 2 to test the robustness of the familial depression effect. To ascertain which stressors were most strongly associated with depression risk, we tested differences between standardized regression coefficients using the following formula: $Z = (\beta_1 - \beta_2)/\sqrt{(SE_1^2 + SE_2^2)}$ (see Armstrong et al., 2022).

Results

Preliminary analyses. Table 1 summarizes participants' sociodemographic and clinical characteristics. Age (rs < |.21|, ps > .053), sex (ps > .308, ds <.23), and racial/ethnic minority status (ps > .346, ds < .32) were not significantly associated with stressor severity. Adolescent depression severity was associated with greater chronic stress, adolescent anxiety severity was associated with greater chronic and independent stress, and mother's anhedonia severity was inversely associated with interpersonal stress (Table 3). To maintain a consistent set of covariates, we tested the robustness of effects in all models by entering these three variables on Step 2.

Stress exposure among LR versus HR youth. The distributions of stressor severity variables were nonnormal (Table 3). Following recommendations for transformation moderately positively skewed data (Tabachnick & Fidell, 2019), stress variables

Table 4 Multiple linear regression models testing the association between life stress severity variables and depression risk status in Study 1 (n = 87), controlling for clinical and demographic covariates

	β	CI_{95}	t	p	r_{sp}
Model 1: Chronic difficulty threat					
Step 1 (F[1, 85] = 11.79, p < .001, R^2 = .122					
LR versus HR	.35	0.15, 0.55	3.43	<.001	.35
Step 2 (ΔF[3, 82] = 4.39, p = .006, ΔR^2 = .121[a]					
LR versus HR	.22	0.01, 0.42	2.08	.041	.20
MFQ	.25	0.02, 0.48	2.17	.033	.21
MASC	.17	−0.07, 0.41	1.41	.161	.14
SHAPS-M	.07	−0.13, 0.26	0.69	.494	.07
Model 2: Independent event threat					
Step 1 (F[1, 85] = 13.45, p < .001, R^2 = .137)					
LR versus HR	.37	0.17, 0.57	3.67	<.001	.37
Step 2 (ΔF[3, 82] = 0.27, p = .847, ΔR^2 = .008)[b]					
LR versus HR[c]	.34	0.12, 0.56	3.05	.003	.31
MFQ	.06	−0.19, 0.31	0.48	.630	.05
MASC	.05	−0.21, 0.30	0.37	.710	.04
SHAPS-M	−.02	−0.22, 0.19	−0.14	.885	−.01
Model 3: Non-interpersonal event threat					
Step 1 (F[1, 85] = 0.62, p = .433, R^2 = .007)					
LR versus HR	.09	−0.13, 0.30	0.79	.433	.09
Step 2 (ΔF[3, 82] = 0.48, p = .699, ΔR^2 = .017)					
LR versus HR	.05	−0.18, 0.28	0.43	.666	.05
MFQ	−.08	−0.34, 0.19	−0.58	.564	−.06
MASC	.16	−0.11, 0.43	1.18	.242	.13
SHAPS-M	.002	−0.22, 0.22	0.02	.988	.002
Model 4: Interpersonal event threat					
Step 1 (F[1, 85] = 4.34, p = .040, R^2 = .049)					
LR versus HR	.22	0.01, 0.43	2.08	.040	.22
Step 2 (ΔF[3, 82] = 1.85, p = .144, ΔR^2 = .060)[d]					
LR versus HR	.23	0.004, 0.45	2.02	.047	.21
MFQ	.07	−0.18, 0.32	0.55	.584	.06
MASC	−.06	−0.32, 0.20	−0.44	.663	−.05
SHAPS-M	−.24	−0.46, −0.03	−2.30	.024	−.24

CI_{95} = 95% confidence interval; MASC, Mood and Anxiety Symptom Questionnaire; MFQ, Mood and Feelings Questionnaire; SHAPS-M, Snaith–Hamilton Pleasure Scale (mother). LR versus HR = Dummy coded variable that compares low-risk (LR) adolescents to high-risk (HR) adolescents. The maximum Variance Inflation Factor (VIF_{max}) in the models was 1.58. The lowest tolerance value in the model was 0.63.

[a]The direction and significance of effects were similar adjusting only for the MFQ and MASC.

[b]The direction and significance of effects were similar adjusting only for the MASC.

[c]The effect remained statistically significant in a model that further adjusted for chronic difficulty threat.

[d]The direction and significance of results were similar adjusting only for the SHAPS-M.

were log transformed. The transformed variables had acceptable skewness and kurtosis values (Skewness < 1.52; Kurtosis <2.72) and approximated normal distributions.

Results of regression models predicting stressor severity are presented in Table 4. Being in the HR versus LR group was associated with more severe chronic, independent, and interpersonal stress severity; these effects were robust when covariates were included. By contrast, adolescents' maternal history of MDD was not associated with non-interpersonal event severity. Given the importance of accounting for the overlap among stress variables in depression research (Vrshek-Schallhorn et al., 2020), we further controlled for chronic stress severity in the independent event model. The effect of maternal depression history persisted on independent stress persisted adjusting for chronic stress, β = .28, CI_{95} = 0.06, 0.50, t(81) = 2.55, p = .013, r_{sp} = .25. For models in which the maternal depression history effect was significant, the grouping variable explained between 5% (interpersonal stress) and 14% (independent stress) of the variance in outcomes, corresponding to small-to-medium effect sizes (R^2s = .049–.137; see Table 4).

Between-model group effect comparisons: Using unadjusted effects, the association between being in the HR versus LR group and stress severity in the chronic difficulty model (z = 1.78, p = .038) and independent stress (z = 1.92, p = .027) were significantly larger than the same effect for non-interpersonal stress. The group effects in the interpersonal and non-interpersonal models were not significantly different (z = 0.89, p = .186). Additionally, the group effects in the chronic, independent, and interpersonal models were not significantly different from one another, |z|s < 1.03, ps >.152.

Study 2

Method

Participants. Participants were 87 adolescents (64 female), aged 12–19-years-old (M = 15.48, SD = 1.86). Fifty-seven (65.52%) adolescents were recruited from a short-term (10–14 days) intensive treatment service at McLean Hospital for youth with internalizing disorders (van Alphen et al., 2017; Zambrowicz et al., 2019). Youth completed a clinical assessment within 48-hr of admission that was used to determine eligibility. Inclusion criteria included: (a) current DSM-IV-TR MDD or dysthymia confirmed using the Mini International Neuropsychiatric Interview for Children and Adolescents (MINI-KID; Sheehan et al., 2010), (b) depressive symptoms in the clinical range (≥16) on the Center for Epidemiological Studies-Depression (CES-D; Radloff, 1977), and (c) were experiencing clinical significant suicidal ideation, indexed by scores of ≥4 on the Scale for Suicide Ideation (SSI; Beck, Kovacs, & Weissman, 1979), in line with prior studies (Stewart, Glenn, et al., 2017; Stewart et al., 2019).

Depressed youth (MDD; n = 57) in treatment were classified into one of two groups using responses to the Self-Injurious Thoughts and Behaviors Interview–Short Form (SITBI-SF; Nock, Holmberg, Photos, & Michel, 2007). Suicide attempters (SA; n = 26) reported at least one suicide attempt in the past year and suicide ideators (SI; n = 31) reported no lifetime history of suicide attempts. See Appendix S4; Tables S1 and S2 for additional clinical information for the SI and SA groups.

The healthy controls (HC; n = 30) were recruited from the community and had no lifetime history of DSM-IV-TR, lifetime psychotropic medication use, STBs, or nonsuicidal self-injury. Eligible adolescents provided their written informed consent prior to participation; for youth 12–17-years-old, a parent/guardian also provided consent for their child to participate.

Ninety-four adolescents (MDD = 61, HC = 33) consented to participate. In the MDD group, one (1.64%) participant's care was stepped up before participation, two (3.28%) did not complete the LEDS-II, and one (1.64%) had missing data on key symptom measures; these youth were not included in primary analyses. Further, three (9.09%) HC adolescents had histories of psychopathology and were thus excluded. Retained and excluded youth did not differ significantly on sociodemographic, clinical, or grouping variables (all ps > .343).

Measures. Adolescents in the HC group completed all interviews and questionnaires in a single laboratory session. Youth in the MDD group completed the MINI-KID, SITBI-SF, and questionnaires within 48 hr of beginning treatment, and then, once enrolled, they were administered the LEDS-II separately 0–18 days later (M = 8.18, Mdn = 8.00, SD = 3.88).

Interviews: Training procedures for the MINI-KID and SITBI-SF, as well as evidence for their reliability and validity in adolescent clinical samples, have been described elsewhere (Nock et al., 2007; Stewart, Valeri, Esposito, & Auerbach, 2018).

Questionnaires: Adolescents completed the CES-D (Radloff, 1977), MASC (March et al., 1997), the SHAPS (Snaith et al., 1995), and the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974) to measure their depression, anxiety, anhedonia, and hopelessness symptoms, respectively. The 19-item version of the SSI (Beck et al., 1979) was used as a continuous measure of suicide ideation severity in the past week. Internal consistency for these symptom measures was good to excellent (Table 5). Adolescents also completed the Childhood Trauma Questionnaire–Short Form (CTQ-SF; Bernstein et al., 2003) to assess their lifetime experiences of abuse and neglect. We used recommended cutoff scores (Bernstein & Fink, 1998) to compute dichotomous variables representing the presence versus absence of physical and sexual abuse. Specifically, physical and sexual abuse were coded as 'present' for youth with scores of ≥8 and ≥6, respectively (i.e. the 'slight to moderate' range or above; Bernstein et al., 2003).

Life stress: We assessed independent, dependent non-interpersonal, and dependent interpersonal life event severity, as well as chronic difficulty severity. For participants in the HC group, the LEDS-II interview covered the 6 months prior to the interview date. For the SA and SI group, the interview covered the 6 months prior to treatment admission.

Data analysis. Regression models tested the association between group (HC, SI, and SA) and life stress. We used contrast coding to examine mean differences between the groups and combinations of these groups in a linear regression context (see Cohen, Cohen, West, & Aiken, 2003). Specifically, we created two (k–1) orthogonal contrast code variables to test hypotheses relating to the three groups, following recommended procedures (Cohen et al., 2003; Tabachnick & Fidell, 2019). We created contrast code variables following the formal statistical definitions of contrasts (rules described by Cohen et al., 2003); therefore, the regression coefficients in the model are directly interpretable as the difference between the means of the group(s) being compared in the contrast. The first contrast code variable tested whether adolescents in two clinical groups (SI and SA participants; labeled MDD) had higher mean scores on the stress outcome variables than adolescents in the HC group. The second contrast code tested whether the SI and SA groups had

Table 5 Bivariate correlations and descriptive statistics for stress and symptom variables in the full Study 2 sample (n = 87)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
Bivariate correlations											
1. Independent stress	–	.35***	.18	.28**	−.04	.25*	.27*	.29**	.23*	.16	.18
2. Non-Interpersonal stress		–	.25*	.39***	.18	.11	.40***	.32**	.34**	.35***	.38***
3. Interpersonal stress			–	.32**	.56***	.30**	.23*	.22*	.27*	.21	.26*
4. Chronic stress				–	.17	.32**	.63***	.64***	.58***	.48***	.60***
5. Targeted rejection					–	.07	16	.11	.16	.17	.29**
6. Childhood abuse						–	.32**	.40***	.30**	.17	.24*
7. CES-D							–	.84***	.74***	.78***	.81***
8. MASC								–	.66***	.61***	.73***
9. SHAPS									–	.75***	.74***
10. BHS										–	.79***
11. SSI											–
Descriptive statistics											
Mean/N	2.41	1.59	2.09	4.91	14[a]	13	25.92	54.25	27.11	8.45	8.87
Standard deviation/%	3.12	2.34	3.19	4.85	16.09[a]	14.94	18.96	22.43	8.52	6.34	8.38
Range	0–17	0–11	0–17	0–21	0–4[b]	–	0-58	2-97	14–44	0–20	0–26
Skewness	1.91	1.92	2.37	1.15	–	–	−0.01	−0.13	−0.08	0.35	0.35
Kurtosis	5.14	4.11	6.89	0.98	–	–	−1.47	−0.75	−1.20	−1.21	−1.29
Reliability (α)	–	–	–	–	–	–	0.970	0.943	0.926	0.934	0.943

BHS, Beck Hopelessness Scale; CES-D, Center for Epidemiological Studies–Depression; MASC, Mood and Anxiety Symptom Questionnaire; SHAPS, Snaith–Hamilton Pleasure Scale; SSI, Scale for Suicide Ideation. Values in rows 1–5 are Spearman's rho coefficients (ρ) given the moderate positive skew in the stress variables. Values in 6 are point biserial correlations, as childhood maltreatment is a binary variable. Values in rows 7–11 are Pearson's product–moment correlation coefficients (r).
[a]Values indicate the number and percentage of participants in the sample who experienced at least one targeted rejection event.
[b]The range refers to the number of targeted rejection events participants experienced. Twelve (13.79%) participants reported one TR event, one (1.14%) participant reported two TR events, and one (1.14%) participant reported four TR events.
*p < .05; **p < .01; ***p < .001.

significantly different mean scores on the stress outcome variables. Contrast code variables were entered simultaneously on Step 1; this follows recommended procedures and is appropriate because the contrast codes are orthogonal and thus share minimal overlapping variance (Cohen et al., 2003). Covariates were added on Step 2 to test the robustness of the group effects.

Our first group of models used total stressor severity scores for events and difficulties that occurred any time in the 6-month interview period. We expected adolescents experiencing depression and suicidal ideation (MDD group) to report more severe stress overall, and interpersonal stress particularly. This was captured by the HC versus MDD contrast code.

We ran a second set of time-locked analyses. For attempters, we computed stress severity variables based on 3 months preceding their most recent attempt. This approach separates stress that may contribute to attempts from the negative sequelae related to attempts (Liu & Spirito, 2019). The 3-month period maximized participant retention; youth with attempts closer to the start of the LEDS-II period (or before it) were removed. Our period is consistent with research demonstrating that stressors increase the likelihood of future attempts within the 3 months after the stressor (Cooper, Appleby, & Amos, 2002; Foster, 2011) For the SI group, we computed stress variables for the 3 months prior to admission and for the HC group, we time-locked exposure to the interview date. Time-locked analyses provided the strongest test of the link between stress and attempts among ideators, captured by the SI versus SA contrast code.

Target rejection events: Our analysis of TR events focused on stressors reported in the full 6-month interview period.[3] We operationalized TR as the number of events participants experienced, in line with prior studies (Cheek, Reiter-Lavery, et al., 2020; Massing-Schaffer et al., 2019; Slavich et al., 2009); this approach has been used because TR events tend to be major stressors that occur rarely. The count of TR events was a good fit to a Poisson distribution, D (57) = 0.30, z = 0.23, p = .743. As with models above, we entered the contrast-coded variables simultaneously on Step 1 as predictors of TR event frequency in a Poisson regression model with log link. Covariates were entered on Step 2.

Results

Preliminary analyses. Table 6 summarizes: (a) demographic and clinical characteristics of the sample and (b) differences among HC, SI, and SA (see also Tables S1 and S2). Older age was associated with more severe non-interpersonal threat, r (85) = .36, p < .001, but not the other stress variables, ps >.082. Relative to males, females reported more severe event threat in all domains, ts >2.10, ps < .038, ds > .49, and more severe chronic

difficulties $t(85) = 2.81$, $p = .006$, $d = .68$. Identifying as a racial/ethnic minority (relative to identifying as White) was not associated with severity of the stressors we assessed, ps > .123, ds < .35. Finally, the presence of childhood abuse was associated with more severe chronic, independent, and interpersonal stress (Table 5).

Symptom variables were associated with all stress outcomes. However, due to recruitment and grouping procedures, these correlations reflected expected group effects (e.g. HC vs. MDD). Thus, covariates entered in our models were age, sex, and childhood abuse (presence/absence); symptom variables were not used to avoid multicollinearity.

Six-month stress exposure. Participants reported between one and 15 life events. Moderate-to-severe stressors are more etiologically relevant to stress-related psychopathology and these events were common in our sample (71 youth [81.61%] had one or more events rated above 'little/none'). In line with prior research on depression (Stroud, Davila, Hammen, & Vrshek-Schallhorn, 2011) and suicide (Cheek, Goldston, et al., 2020; Massing-Schaffer et al., 2019), we excluded events rated 'little/none' from total scores.

Like Study 1, the distributions of stress severity variables were nonnormal (Table 5). We again log-transformed data per recommendations (Tabachnick & Fidell, 2019), and distributions of the new variables were approximately normal (Skewness <0.66; −0.86 < Kurtosis <1.23).

Table 7 shows results of regression models predicting stress severity. Adolescents in the MDD group reporting significantly more severe mean chronic, non-interpersonal, and interpersonal episodic stress compared to adolescents in the HC group. The difference between stress exposure in the MDD and HC groups was also significant in the independent stress model but was nonsignificant when covariates were added.

Group effects in the non-interpersonal and interpersonal stress models were of similar magnitude and direction. Given the primacy of dependent stressors in the literature on adolescent depression (Vrshek-Schallhorn et al., 2020) and suicide (Liu & Miller, 2014), we conducted additional analyses wherein overall dependent event threat was the outcome variable. Relative to the HC group, the MDD group had significant higher mean dependent stress severity (Table 7). Dependent event threat and chronic difficulty threat were significantly

Table 6 Descriptive statistics and clinical characteristics for healthy controls (HC; $n = 30$), suicide ideators (SI; $n = 31$), and suicide attempters (SA; $n = 26$) in Study 2

	Descriptive statistics (M [SD] or n [%])					
	HC	SI	SA	F/χ^2	p	Φ/η_p^2
Demographics						
Sex (female)[b]	18 (60.00)	23 (74.19)	23 (88.46)	5.81	.060	0.26
Age (years)[a,b]	14.77 (2.24)	16.26 (1.59)	15.38 (1.30)	5.46	.006	0.12
Race						
White	22 (73.33)	23 (74.19)	20 (76.92)	4.78	.951	0.23
Black	0 (0.00)	1 (3.23)	0 (0.00)			
Asian	3 (10.00)	2 (6.45)	1 (3.85)			
Pacific Islander	0 (0.00)	1 (3.23)	0 (0.00)			
>1 race	5 (16.67)	4 (12.90)	5 (19.23)			
Psychiatric symptoms						
CES-D[a,b]	3.15 (3.81)	36.23 (11.05)	39.89 (10.79)	143.65	<.001	0.77
MASC[a,b]	31.83 (13.30)	63.00 (17.28)	69.68 (14.88)	50.60	<.001	0.55
SHAPS[a,b]	17.97 (4.06)	31.39 (5.17)	32.54 (6.68)	67.37	<.001	0.62
BHS[a,b]	1.84 (1.29)	11.03 (5.11)	12.99 (4.80)	61.15	<.001	0.59
SSI[a,b]	0.00 (0.00)	12.68 (5.92)	14.58 (7.28)	64.60	<.001	0.61
Childhood abuse (presence)						
Physical[b]	0.00 (0.00)	2 (6.45)	5 (19.23)	7.13	.025	0.29
Sexual[a,b]	0.00 (0.00)	5 (16.13)	5 (19.23)	6.08	.045	0.26
Either[a,b]	0.00 (0.00)	5 (16.13)	8 (30.77)	10.43	.005	0.35
Severity of life stress exposure						
Chronic[a,b]	1.13 (1.83)	6.65 (4.54)	7.19 (5.11)	20.15	<.001	0.32
Independent[a,b]	1.17 (1.58)	3.13 (3.36)	3.00 (3.75)	3.93	.023	0.09
Non-interpersonal[a,b]	0.47 (1.01)	2.42 (2.42)	1.88 (2.85)	6.33	.003	0.13
Interpersonal[a,b]	0.67 (1.30)	2.26 (2.94)	3.54 (4.25)	6.41	.003	0.13

BHS, Beck Hopelessness Scale; CES-D, Center for Epidemiological Studies-Depression; HC, healthy control; MASC, Mood and Anxiety Symptom Questionnaire; SA, suicide attempter; SHAPS, Snaith–Hamilton Pleasure Scale; SI, suicide ideator; SSI, Scale for Suicide Ideation. Life stress values are the mean severity in the domains listed for each of the groups in the 6 months prior to hospitalization (SI and SA groups) or prior to the interview (HC group). Event threat severity did not include mild events (i.e. those rated 4 [*little/none*] for long-term threat).
[a]HC < SI.
[b]HC < SA.

Table 7 Multiple linear regression models testing the association between life stress variables in the 6-month interview period (n = 87) and group (HC vs. MDD; SI vs. SA) in Study 2, controlling for clinical and demographic covariates

	β	CI_{95}	t	p	r_{sp}
Model 1: Chronic difficulty threat					
Step 1 (F[2, 84] = 38.59, p < .001, R^2 = .479)					
HC versus MDD	0.69	0.54, 0.85	8.78	<.001	.69
SI versus SA	0.05	−0.11, 0.21	0.65	.515	.05
Step 2 (ΔF[3, 81] = 1.98, p = .123, ΔR^2 = .036)[a]					
HC versus MDD	0.62	0.45, 0.79	7.16	<.001	.55
SI versus SA	0.02	−0.14, 0.18	0.24	.808	.02
Age	0.03	−0.14, 0.19	0.35	.728	.03
Sex (Female)	0.15	−0.01, 0.31	1.87	.065	.14
Childhood abuse (present)	0.11	−0.06, 0.27	1.28	.204	.10
Model 2: Independent event threat					
Step 1 (F[2, 84] = 3.45, p = .036, R^2 = .076)					
HC versus MDD	0.27	0.07, 0.48	2.62	.011	.27
SI versus SA	−0.01	−0.22, 0.20	−0.09	.930	−.01
Step 2 (ΔF[3, 81] = 2.78, p = .046, ΔR^2 = .086)[a]					
HC versus MDD	0.20	−0.03, 0.42	1.75	.083	.18
SI versus SA	−0.09	−0.30, 0.12	−0.83	.407	−.08
Age	−0.11	−0.33, 0.10	−1.02	.309	−.10
Sex (Female)	0.19	−0.02, 0.40	1.77	.081	.18
Childhood abuse (present)	0.20	−0.01, 0.42	1.86	.066	.19
Model 3: Non-interpersonal event threat					
Step 1 (F[2, 84] = 8.58, p < .001, R^2 = .170)					
HC versus MDD	0.38	0.18, 0.57	3.77	<.001	.38
SI versus SA	−0.15	−0.35, 0.05	−1.52	.133	−.15
Step 2 (ΔF[3, 81] = 2.46, p = .068, ΔR^2 = .069)[b]					
HC versus MDD	0.28	0.07, 0.50	2.62	.010	.25
SI versus SA	−0.12	−0.32, 0.08	−1.15	.253	−.11
Age	0.25	0.05, 0.46	2.46	.016	.24
Sex (Female)	0.12	−0.08, 0.32	1.22	.226	.12
Childhood abuse (present)	−0.01	−0.22, 0.19	−0.13	.898	−.01
Model 4: Interpersonal event threat					
Step 1 (F[2, 84] = 7.99, p < .001, R^2 = .160)					
HC versus MDD	0.38	0.18, 0.58	3.81	<.001	.38
SI versus SA	0.14	−0.06, 0.34	1.42	.160	.14
Step 2 (ΔF[3, 81] = 1.18, p = .322, ΔR^2 = .035)[a]					
HC versus MDD	0.29	0.07, 0.51	2.63	.011	.26
SI versus SA	0.13	−0.08, 0.34	1.24	.331	.12
Age	0.11	−0.10, 0.32	1.01	.336	.10
Sex (Female)	0.07	−0.14, 0.27	0.64	.502	.06
Childhood abuse (present)	0.15	−0.07, 0.36	1.36	.143	.14
Model 5: Dependent event threat					
Step 1 (F[2, 84] = 15.74, p < .001, R^2 = .273)					
HC versus MDD	0.52	0.34, 0.71	5.61	<.001	.52
SI versus SA	0.03	−0.15, 0.22	0.34	.736	.03
Step 2 (ΔF[3, 81] = 2.49, p = .066, ΔR^2 = .061)[c]					
HC versus MDD	0.41	0.21, 0.61	4.06	<.001	.37
SI versus SA	0.04	−0.15, 0.23	0.44	.663	.04
Age	0.21	0.02, 0.40	2.20	.030	.20
Sex (Female)	0.10	−0.09, 0.28	1.02	.311	.09
Childhood abuse (present)	0.11	−0.08, 0.30	1.12	.266	.10

CI_{95} = 95% confidence interval; HC versus MDD = contrast code variable that compares HC to MDD (SI + SA). Positive, statistically significant beta values indicate that the MDD (SI + SA) group has a significantly higher mean score on the outcome (i.e. stress severity) than the HC group. SI versus SA = Contrast code variable that compares the SA and SI groups that the SA group has a significantly higher mean score on the outcome (i.e. stress severity) than the SI group. Events rated 'little/none' for event threat severity were not included in total threat scores. The maximum Variance Inflation Factor (VIF_{max}) in the models was 1.24. The lowest tolerance value in the model was 0.81.

[a]The direction and significance of effects were similar in a model that only adjusted for sex and childhood abuse.

[b]The direction and significance of effects were similar in a model that only adjusted for sex and age.

[c]The covariates included on Step 2 were all significantly bivariately associated with the total severity of dependent events.

associated, r(85) = .47, p < .001; nonetheless, MDD versus HC difference in the dependent stress model remained significant after further adjusting for chronic stress, β = .31, CI_{95} = 0.06, 0.57, t(80) = 2.42, p = .018, r_{sp} = .22. The SI versus SA contrast code variable was nonsignificant in all models.

In general, for models in which the HC versus MDD contrast code variable was significant, the contrasts

explained between 8% (independent stress) and 48% (chronic stress) of the variance in outcomes, corresponding to small-to-large effect sizes (R^2s = .076–.479; see Table 7).

Between-model group effect comparisons: The unadjusted effect reflecting the mean difference between the MDD and HC groups was significantly larger in the chronic stress model than the independent, non-interpersonal, and interpersonal models (*z*s = 2.50–3.95, *p*s <.007). In contrast, there were no significant differences among the effects in the independent, non-interpersonal, and interpersonal models, *z*s <1.02, *p*s >.056. However, the comparison of the effect of the MDD versus HC contrast code variable between the chronic and dependent stress model was nonsignificant ($z = 1.39$, $p = .082$) and the effect in the dependent model was significantly larger than the independent stress model ($z = 1.77$, $p = .039$).

Three-month event-locked stress exposure. The pattern of effects in the 3-month models was like the 6-month models. The mean difference between adolescents in the MDD versus HC group (i.e. first contrast coded variable) was significant in the chronic, non-interpersonal and interpersonal stress models, but not the independent model. The mean difference between the SI and SA group (i.e. second contrast coded variable) was nonsignificant in all models. A complete report of the 3-month event-locked analyses is provided in Appendix S5 and Tables S3–S5.

Targeted rejection. One (3.33%) HC, four (12.90%) SI, and nine (34.62%) SA adolescents reported at least one TR event. The SI versus SA contrast code was significant in the unadjusted model; relative to the SI group, the SA group experienced a significantly higher rate of TR events, $b = 1.05$, $SE = 0.53$, $Wald(1) = 3.90$, $p = .048$, $RR = 2.86$, and CI [1.01, 8.12]. This effect was robust when controlling for covariates and persisted in a model that excluded the HC group (Table S6).

The HC versus MDD contrast code variable was also significant in the model; the MDD group experienced a significantly higher rate of TR relative to the HC group, $b = 2.10$, $SE = 1.03$, $Wald(1) = 4.13$, $p = .042$, $RR = 8.19$, and C I[1.08, 62.24]. However, this effect should be interpreted with particular caution because of the very low rate of TR in the HC group. Additionally, the HC versus MDD contrast code variable was nonsignificant when covariates were added to the model.

Discussion

Life stress is implicated in the onset and worsening of depression in adolescents. However, limitations in stress assessment have left open questions about which forms of stress contribute to adolescent depression at understudied points along its clinical course. Further, few studies have identified types of stress most strongly linked to clinical features among depressed adolescents. We used the LEDS-II to address these gaps in two well-characterized samples.

There were four primary findings. First, across samples, chronic stress severity was associated with depression and relations were sometimes larger than those for episodic stress. Second, independent and interpersonal stress was associated with familial risk for depression, but non-interpersonal stress was not. Generally, the effect of familial risk for depression on stress outcomes were small-to-medium; this range is consistent with recent studies using interview-based stress assessment methods (e.g. Feurer et al., 2016). Third, relative to the HC group, youth with MDD and suicide ideation had greater mean dependent event severity, and this effect was stronger than the relation with independent stress. The effect sizes associated with the HC versus MDD comparison were small-to-large, and the medium effects (R^2s = .160–.273) in the dependent stress models are in line with a recent meta-analysis of relevant literature (Rnic et al., 2023). Last, the SI and SA groups did not significantly differ in mean stress severity in any domain; however, youth with recent attempts reported more TR events than ideators.

Chronic stress

Rigorously capturing episodic *and* chronic stressors to achieve a more complete picture of the stress–depression relationship has long been recommended (Hammen, 2016; Harkness & Monroe, 2016) but is rarely implemented (Vrshek-Schallhorn et al., 2020). Accordingly, we assessed chronic stress and tested the magnitude of its associations with depression outcomes relative to episodic stressors. Prior work that *has examined* chronic stress and maternal depression history (Adrian & Hammen, 1993; Feurer et al., 2016; Gershon et al., 2011; Hammen et al., 2004, 2012; Herr et al., 2007) has been limited by its exclusively use of the Life Stress Interview (LSI; Hammen et al., 1987). The LSI operationalizes chronic difficulties as general adjustment in areas assessed (e.g. finances), which may overlap substantially with impairment related to depression (Harkness & Monroe, 2016). In contrast, the LEDS-II confirms exposure to specific difficulties using inclusion rules. We found that chronic stress was related to familial risk for depression and the effect was equivalent to, or stronger than, effects for episodic stress, methodologically extending prior work. Our results replicate prior work showing that HR youth experience more severe chronic interpersonal stress relative to LR youth (Gershon et al., 2011; Hammen et al., 2004, 2012; Herr et al., 2007), but not non-interpersonal chronic

stress (Feurer et al., 2016). Our operationalization of chronic stress included dependent and independent difficulties (see Appendix S3). Assessing a full range of chronic stress may optimally capture the diverse etiological pathways through which depression may be transmitted across generations (Goodman, 2020).

In Study 2, adolescents with current MDD and suicide ideation experienced more severe chronic stress than HCs. These results replicated prior work (using the LSI) that has highlighted a potential bidirectional relation between chronic stress and depression in young adults (Vrshek-Schallhorn et al., 2015) and adolescents (Mineka et al., 2020; Rudolph et al., 2000; Uliaszek et al., 2012), as well as relations between chronic stress and suicide ideation (Massing-Schaffer et al., 2019; Pettit et al., 2011; Stewart et al., 2018). The centrality of chronic stress in samples from earlier (Study 1) and later (Study 2) in the course of MDD conceptually aligns studies with the high stability of chronic stress among depressed adolescents (Uliaszek et al., 2012) and the continuity of stress exposure in this group (Hammen et al., 2012; Hazel, Hammen, Brennan, & Najman, 2008). Future work should test potential interactions between chronic stress and risk factors and/or episodic stress (Vrshek-Schallhorn et al., 2020) to predict depression-related outcomes in youth.

Life events among HR and LR youth

A maternal history of depression was associated with independent and interpersonal event severity; the former effect was stronger than it was for non-interpersonal stress. The effect for independent events may reflect the environmental features associated with maternal depression that persist outside of depressive episodes. For example, MDD may elicit pathophysiological changes that contribute to chronic medical conditions (Machado et al., 2018), increasing the likelihood youth are exposed to stress related to parental illness and disability. Maternal depression is also linked to parental unemployment (Jefferis et al., 2011), lower family income (Lorant et al., 2003), and marital conflict (Essex, Klein, Cho, & Kraemer, 2003). In the LEDS-II, stressors related to these circumstances (e.g. parent job loss) are classified as independent. Youth at risk for depression may also select environments in which independent stressors are more likely to occur (Hammen, 2020; Harkness & Stewart, 2009). For instance, entering relationships with peers with shared experiences of depression may increase the probability that youth are involved in a close others' health and/or psychiatric crisis.

The effect of maternal depression history was modest, but significant, for interpersonal stress. Mothers in the HR group had lifetime but not current MDD, and we controlled for symptoms that were associated with stress outcomes. This extends prior research that has focused on mothers with current MDD (Adrian & Hammen, 1993; Gershon et al., 2011; Hammen & Brennan, 2001) or that did not consider the timing of maternal MDD in analyses (Carter & Garber, 2011; Feurer et al., 2017; Morris et al., 2014). Further, mothers' MDD episodes did not necessarily occur during their offspring's lifetime. Thus, heightened interpersonal stress among HR adolescents is not entirely explained by direct effects of current maternal depression (e.g. greater parent–child conflict; Feurer et al., 2016; Hammen et al., 2004). Youth in the HR group may possess depressogenic cognitive and/or interpersonal styles that drive the intergenerational transmission MDD. Maternal depression is linked to offspring difficulties with emotion regulation, communication, and problem-solving (Goodman, 2020), and these may have contributed to the generation of interpersonal stress among HR youth (Hammen, 1991, 2009).

Stress among youth with MDD and current ideation

Currently, depressed adolescents with recent ideation experienced more severe average dependent – interpersonal and non-interpersonal – life stress than HCs, and the effect was stronger than the independent stress model. These results are consistent with prior work showing that depression (Hammen, 2020; Liu & Alloy, 2010) and suicidal ideation (Liu & Spirito, 2019) are associated with dependent stress severity in youth. The primacy of dependent stress is also consistent with a meta-analysis of the stress generation literature (Rnic et al., 2023). The group effect persisted in models that adjusted for chronic stress and childhood maltreatment history, which contribute to depression and overlap with dependent stress (Hammen, 2016; Harkness, Lumley, & Truss, 2008). The group effect was not uniquely associated with interpersonal stress, as some models propose (Hammen, 2009). Our work distinctively examined a group of youth with MDD *and* suicide ideation; symptom severity among youth in our sample may have been greater compared to many prior studies. Non-interpersonal stressors may be more common among youth with more complicated psychiatric histories. Future research in samples of depressed youth with and without histories of STBs is needed to unpack these possibilities.

Suicide attempters versus ideators

Contrary to our hypotheses, depressed adolescent attempters did not experience more severe stress than depressed ideators in any domain assessed. These null results are inconsistent with some prior work that has implicated events involving threats to, or loss of, close relationships in adolescent suicide attempts (Beautrais, Joyce, & Mulder, 1997; Brent et al., 1993;

Cheek, Goldston, et al., 2020; Daniel et al., 2017; Fergusson, Woodward, & Horwood, 2000). However, prior research has employed questionnaire-based stress measures and has not compared ideators and attempters with similar clinical characteristics. One notable exception to the latter is Stewart et al. (2019) who found that only interpersonal loss events differentiated SA and SI groups. However, the time period in which life events were recorded was longer than this study. Notably, longitudinal studies that, by design, separate the life stress and STB assessment periods, as we did, have also found that stress is not associated with attempts (Daniel et al., 2017; Massing-Schaffer et al., 2019; Pettit et al., 2011; Stone et al., 2014). Future research should address open questions about (a) the time frame in which life events may increase the odds of attempts and (b) the potentially unique effects of perceived versus objectively defined stress severity.

Relative to ideators, adolescent attempters had a higher rate of TR events 6 months prior to treatment intake. Although TR was significantly associated with interpersonal stress severity (Table 5), TR events were uniquely relevant to differences in STB history. TR includes interpersonal and non-interpersonal events resulting in status loss (Slavich et al., 2009), and their core features, even if they are not overtly interpersonal, may be suicidogenic. Our findings should be interpreted cautiously; the standard errors of group estimates were relatively large, highlighting the importance of replication in larger samples of youth who may experience TR more frequently (e.g. adolescents with personality disorders). Tentatively, our results align with one study showing that, relative to experiencing no major stressor, social rejection increase the odds of subsequent attempts among adolescents (Cheek, Reiter-Lavery, et al., 2020). Conversely, another study did not find a link between TR and STBs (Massing-Schaffer et al., 2019). Theoretically, TR may contribute to suicide through its connection to cognitive–affective states like thwarted belongingness that fuel increases in suicidal desire (Klonsky et al., 2018).

Limitations

Our findings should be interpreted considering the following limitations. First, both studies had modest sample sizes (see Appendix S1); it is critical for future research to replicate effects we interpret here in larger samples wherein parameter estimates would be more reliable. Second, we used cross-sectional designs and retrospective assessments of stress and clinical characteristics. However, stressors can be accurately recalled in periods up to 1 year (Johnson, 2005; Paykel, 1997); therefore, memory biases likely had minimal influence on our assessments. Third, due to their infrequency, we did not separate chronic stressors into distinct domains; however, this aligns with some methodological critiques of stress classification that contend that all chronic stress is, to some extent, dependent because personal characteristics are highly likely to contribute to long-standing stressors at some point (Harkness & Monroe, 2016; Vrshek-Schallhorn et al., 2020).

Fourth, in Study 1, mothers' diagnostic status was not reassessed at the LEDS-II appointment; it is possible that some LR mothers experienced a first lifetime depressive episode at that time. However, LR mothers' BDI-II scores were in a range indicating minimal symptoms. Fifth, due to the infrequency of TR events in the Study 1 sample, we could not formally test whether HR youth experienced higher rates of these etiologically important stressors (see Slavich et al., 2009). The potential link between maternal risk for depression and TR events, specifically, should be examined in larger samples of participants in middle-to-late adolescence, where TR may be more common (e.g. Cheek, Reiter-Lavery, et al., 2020). Sixth, in Study 2, our examination of how stress impacts STBs was restricted to youth with MDD. STBs are transdiagnostic phenomena, and they also occur among youth with no formal diagnoses. Our results may not be generalizable to all adolescents who experience STBs. Last, in Study 2, most depressed youth first experienced ideation before the period in which we assessed stress. Thus, our findings cannot inform how life stress may contribute to first lifetime instances of ideation.

Overall, our findings suggest the stressor features that most strongly contribute to adolescent depression and its clinical correlates may be different for youth earlier versus later in its clinical course. Rigorously classifying and quantifying stress in its developmental context and considering the timing key clinical events (e.g. attempts) is necessary for mitigating the impacts of adolescent depression.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Sample size and power considerations.
Appendix S2. Additional details about Study 1 recruitment and participants.
Appendix S3. Additional LEDS-II details.
Appendix S4. Further clinical characteristics of Study 2 participants.
Appendix S5. Three-month event-locked stress exposure analyses.
Table S1. Diagnostic characteristics for adolescents in the SI.
Table S2. Descriptive statistics and clinical characteristics for healthy controls.
Table S3. Descriptive statistics and clinical characteristics for healthy controls.

Table S4. Bivariate correlations and descriptive statistics for stress and symptom variables in the time-locked Study 2 sample.
Table S5. Multiple linear regression models testing the association between life stress variables in 3-month, event-locked time period.
Table S6. Poisson regression models testing relations between the presence/absence of targeted rejection in the 6-month interview period and group in Study 2.

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Data availability statement
The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Key points
- Exposure to stress is implicated in major depressive disorder for many adolescents, but it is unclear what type(s) of stressors are most relevant to depression across its clinical spectrum.
- Using a gold-standard approach to comprehensively assess stress features, we show that, compared to youth without a maternal history of depression, those with a maternal history experience more severe chronic, independent, and interpersonal stress.
- Conversely, chronic and dependent (interpersonal and non-interpersonal), but not independent, stress differentiated depressed youth with current suicide ideation and nondepressed community controls.
- Only targeted rejection – a rare and generally severe form of stress involving social demotion – was associated with recent suicide attempts among depressed ideators.
- Future research should explore how the stressor features that most strongly contribute to adolescent depression and its clinical correlates (e.g. suicide-related outcomes) shift over the clinical course of major depressive disorder.
- Targeted rejection may be uniquely linked to suicidal behavior and our effects warrant replication in larger samples of youth.

Endnotes

1. When operationalized in contextual, interview-based stress assessments, TR events occur rarely, even in clinical samples (Slavich et al., 2009). In Study 1, low base-rates of TR precluded examining differences between HR and LR youth in the occurrence of these events.
2. One HR mother reported significant subthreshold symptoms of MDD on the SCID-I/P but did not meet full lifetime diagnostic criteria. They were retained following the baseline assessment. Excluding them did not impact the direction or significance of the group effect in primary models; thus, we report on the full sample here.
3. Because only one HC participant experienced TR, the estimates for the MDD versus HC contrast are unreliable. To confirm that our effects held in a model that only included clinical participants, we also ran the model without adolescents from the HC group ($n = 57$), and with a dummy-coded variable reflecting the SI versus SA comparison. The full models are presented in Table S6; the effect of being in the SI versus SA group remains significant in the model that removed HC adolescents.

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