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The Impact of Resilience and Subsequent Stressful Life Events on MDD and GAD

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Abstract

Background—There remains a dearth of research examining the "buffering" effect of resilience, wherein resilience at one point in time would be expected to protect an individual against development of psychopathology following future adverse life events.

Methods—Using longitudinal data from an epidemiological twin sample (N = 7463), this study tested whether resilience would act as a buffer for stressful life events (SLEs) against risk for major depressive disorder (MDD) and generalized anxiety disorder (GAD). Resilience, demographics, and psychopathology were measured at Time 1 and recent SLEs and current MDD and GAD were measured at Time 2.

Results—Final models, controlling for demographic covariates and Time 1 diagnosis, examined the impact of Time 1 resilience, recent SLEs, their interaction, and a three-way interaction adding sex, on MDD and GAD.

Conclusions—The pattern of findings was the same for MDD and GAD, wherein main effects and two-way interactions of resilience and SLEs were significant, such that greater resilience was protective, even in the context of high numbers of past-year SLEs. The three-way interaction was not significant, suggesting that the relationship between SLEs and resilience on psychopathology was the same for both men and women. Findings support the conceptualization of resilience as a buffer against the impact of future life stressors on common internalizing psychopathology. Longitudinal designs and trajectory-based studies that include recurring measures of SLEs could inform conceptualizations of resilience in the context of ongoing adversity and aid in developing interventions aimed at fostering healthy adaptation in the face of stressors.

Keywords

resilience; psychological; longitudinal studies; depression; anxiety; life stress

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The experience of stressful life events (SLEs) is a common occurrence and includes traumatic experiences (e.g., physical assault; Benjet et al., 2016; Liu et al., 2017) stressors in the aftermath of trauma (e.g., recovering after disasters; Galea et al., 2007) and personal and network events (e.g., divorce, illness of family member; Kendler, Karkowski, & Prescott, 1999). A wealth of literature demonstrates a relationship between SLEs and psychopathology, particularly internalizing disorders such as major depressive disorder (MDD; Hammen, 2005; Kendler & Gardner, 2010; Park et al., 2015) and generalized anxiety disorder (GAD; Kendler et al., 2003; Bonanno 2012). However, many individuals cope well following SLE exposure, and are generally termed "resilient." Resilience is a concept that has garnered recent theoretical and empirical attention (Bonanno, 2012; Rutter, 2012; Rivers et al., 2016) yet critical questions remain to be answered, particularly with regard to the role resilience may have in buffering against the effects of future SLEs. Thus, the goal of the present study was to utilize a longitudinal sample to test the hypothesis that resilience buffers against the development of psychopathology (i.e., MDD, GAD) in the context of new SLEs.

The concept of resilience, generally defined as positive adaptation and outcomes in the face of adversities (Bonanno, 2012; Rutter, 2012), has been applied in numerous ways. Despite the differing operational definitions of resilience in the literature (Masten, 2001; Luthar, Cicchetti, & Becker, 2000, Hoge et al 2007, Bensimon, 2012; Connor & Davidson, 2003), a tenant generally inferred across applications of the concept is that resilience should act as a buffer against the harmful effects of future stressors. Extant findings support this hypothesis (e.g., findings that individuals high in self-reported resilience scores, capturing trait-level characteristics and environmental supports, remained unchanged with regard to psychiatric symptoms in the face of future SLEs; Hjemdal et al., 2006), yet this research is limited. Further research testing the "buffering" theory is needed, particularly as a majority of studies examining resilience have been cross-sectional in design, and of the existing longitudinal studies (e.g., Bonanno et al., 2012; Norris et al., 2009), most do not also examine new occurrences of stressors.

If resilience can buffer against the effects of adversity, to test the construct validity, it follows that resilience at one point in time would be expected to protect an individual against the development of psychopathology in the aftermath of future adverse events. Individual differences in self-reported trait resilience have been shown to influence the threshold at which individuals react to ongoing daily stressors (Ong, Bergeman, Bisconti, & Wallace, 2006). Additionally, there is a need to examine the potential differential effects of resilience between the sexes. Given prior findings regarding sex differences in psychopathology (e.g., Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Carter, Wittchen, Pfister, & Kessler, 2001) and following traumatic or SLEs (Goldstein et al., 2016), it may be that resilience has a stronger effect in preventing future psychopathology among men than women. Evidence for sex differences in resilience have been noted, using varying metrics (e.g., involvement in extracurricular activities as protective against alcohol use in males, Habib et al., 2014; greater resilience to delinquency in females, Newsome et al., 2016; less resilience to adult psychopathology following childhood maltreatment in females, Samplin et al., 2013).

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In summary, the potential "buffering" effects of resilience are best tested using a longitudinal framework in which resilience is assessed prior to prospective SLEs and related outcomes in order to examine how resilience may influence the impact of future stressors on an individual. To that end, the current study utilized longitudinal data from a large population-based twin sample to address these gaps in the resilience literature, using a quantitative, continuous definition of resilience employed by our research group. This model captures the difference between actual and predicted psychiatric distress, given stressors experienced by quantifying resilience as the residuals of internalizing symptoms left over after the effect of number of previous SLEs has been regressed out. This resilience conceptualization has demonstrated moderate, stable heritability (Amstadter, Myers, & Kendler, 2014) and has been shown to be related to, but distinct from, internalizing psychopathology (Amstadter, Maes, Sheerin, Myers, & Kendler, 2016) and traits that are often associated with "good" outcomes (e.g., self-esteem, optimism; Amstadter, Moscati, Maes, Myers, & Kendler, 2016).

Our primary aim was to examine the impact of resilience, in combination with the experience of later stressors, on future internalizing psychopathology (MDD and GAD). We hypothesized that resilience at Time 1 would protect against the development of subsequent psychopathology at Time 2 in the presence of new SLEs (i.e., high levels of resilience will buffer against psychopathology even in the presence of a large number of new SLEs). Additionally, given previous work suggesting that sex differences in SLE exposure and sensitivity do not account for sex differences in rates of psychopathology, and the mixed literature of sex differences in resilience, an exploratory aim sought to determine whether or not sex modified the interaction between resilience and SLEs on future psychopathology.

Methods

Sample

Participants for the current study were taken from the Virginia Adult Twin Studies of Psychiatric and Substance Use Disorders (VATSPSUD; total N ~ 7500), a large longitudinal twin study of Caucasian adults, described in detail elsewhere (Kendler & Prescott, 2006). Similar to existing papers with this dataset (e.g., Kendler et al., 2000; Lind et al., 2016; Lind et al, 2017), this sample is being used as an epidemiological sample, with analyses controlling for twin status. We utilized data from female-female (FF) and male-male and male-female (MMMF) twins assessed at two separate interview waves (referred to here as Time 1 [study wave 1 for FF and MMMF] and Time 2 [study wave 3 for FF and wave 2 for MMMF]). The average number of years between Time 1 and 2 was 5.11 (0.42) for FF and 1.59 (0.73) for MMMF. Mean (SD) age at Time 1 was 29.3 (7.7) for FF and 35.1 (9.1) for MMMF. For analyses, we restricted the sample to individuals with non-missing data on both resilience and SLEs.

Measures

Demographic variables—Age and income level were used as covariates in all analyses. Income level was assessed using an ordinal scale comprised of 16 income ranges, beginning at 'No income' and ending at '$200,000 and over.'

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Resilience—Participants completed a shortened version of the Symptom Checklist-90, (SCL-90; Derogatis, Lipman, & Covi, 1973) to assess for past-month distress symptoms. The SCL uses a Likert-scale with options ranging from 0 ("not at all") to 4 ("extremely"). The shortened version used 27 items from four of the SCL subscales: depression (10 items), somatization (5 items), anxiety (7 items), and phobic anxiety (5 items). A composite score was created for symptoms at Time 1 and used in the creation of the resilience variable. Participants were also assessed for exposure to SLEs at Time 1. The presence of a variety of SLEs that were both personal in nature (e.g., assault, marital problems, job loss) and "network" events (i.e., events that occurred primarily to, or in interaction with, an individual in the participant's social network; e.g., death or severe illness of participant's spouse, child, or parent, serious trouble getting along with others) were assessed during personal interview. A count of the total number of SLE types experienced (out of 15 items) over the past 90 days (to be proximal to distress ratings) was computed.

The resilience variable utilized the Time 1 SCL score (past month) and Time 1 SLE count (past 90 days) and was operationalized as the residual of SCL score after the effect of recent number of SLEs has been regressed out (i.e., the difference between actual and predicted SCL), as has been done in prior studies using this dataset and method (e.g., Amstadter et al., 2016). This resulted in a range of residual scores that varied from responding much worse or much better than expected given the number of stressors experienced; lower than predicted residuals resulted in a negative score and higher than predicted residuals resulted in a positive score. For clarity in interpretation of subsequent analyses, scores were then reverse-coded, such that positive scores reflected higher resilience and negative scores represented lower resilience.

Stressful life events (SLEs)—SLEs were also assessed at Time 2, as described above, but assessed for SLE count over the past year. This variable count was collapsed into 0–6 or more SLEs endorsed for use in regression analyses.

Assessment of psychiatric disorders—MDD and GAD were assessed during personal interview by trained mental health professionals using modifications of the SCID interview and DSM-III-R criteria (American Psychiatric Association, 1994; Spitzer, Williams, Gibbon, & Williams, 1995). A minimum duration of two weeks was required for MDD and a minimum duration of one month was required for GAD (shown to perform similarly to the six-months diagnostic requirement; e.g., Kessler et al., 2005). We utilized past-year diagnoses for MDD and GAD, assessed at Time 1 and Time 2. During assessment of past-year diagnoses at Time 2, in instances in which Time 2 was less than a year later than Time 1, Time 2 was kept completely independent from Time 1 (i.e., symptoms were assessed for past year "since the last assessment" so Time 2 does not include Time 1 information). MDD and GAD at Time 2 represent the primary outcome measures, while MDD and GAD at Time 1 were included as covariates.

Trait-based variables—Additional trait measures that tap into coping/strength were also examined. Dispositional optimism was measured using 5 items from the Life Orientation Test (Scheier & Carver, 1985); self-esteem was measured with the 10-item Rosenberg Self-Esteem Scale (Rosenberg, Schooler, Schoenbach, & Rosenberg, 1995); and mastery was

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measured using 6 items from the powerlessness subscale of the Alienation scale (Maddi, Kobasa, & Hoover, 1979). Sum scores for each variable were used.

Data analytic plan

Stepwise regressions that modeled probability outcomes were conducted in SAS 9.4 (SAS Institute; Cary, NC) on two outcomes: Time 2 MDD and GAD. Model 1 tested the main effect of resilience on diagnostic status, as well covariates of age, income, sex, and Time 1 diagnostic status. In Model 2, past year SLEs were added to examine the effect of SLEs on outcome. In Model 3, to test the hypothesis that resilience at Time 1 would protect against negative outcomes in the presence of new SLEs, an additive interaction term was included to test the moderation of resilience and recent SLEs. Finally, in Model 4, to examine whether or not sex may modify the interaction between resilience and SLEs on future psychopathology, a three-way interaction term was added.

A generalized estimating equations (GEE) approach (invoked using the REPEATED statements in SAS PROD GENMOD) was used in all models to account for the non-independence of the nested twin structure. In order to examine the interactions on an additive scale, we chose to investigate a risk difference model (i.e. model the difference in risk of being diagnosed with MDD or GAD for individuals with varying levels of resilience and exposure to SLEs). Thus, all analyses used an additive probability scale, as opposed to multiplicative (e.g., odds ratio or risk ratio; see Kendler & Gardner, 2010). These models utilized the sampling variance of the binomial distribution for parameter estimation, with constraints to keep the predicted probabilities between 0 and 1. The regression coefficient can be interpreted as the increase in probability of being diagnosed with MDD or GAD at Time 2.

Follow-up analyses were conducted in order determine whether our resilience variable acted as a buffer after adjusting for the main effects of other traits demonstrated to be associated with MDD/GAD (optimism, self-esteem, mastery). These variables were added simultaneously to the final MDD and GAD models. Finally, as the length of time between Time 1 and Time 2 was not consistent across subsamples, follow-up analyses were conducted by sex and wave.

Results

Descriptives

A total of 7463 individuals (43.8% female) from the combined FF and MMMF samples were included in analyses. The mean (SD) age at Time 1 for individuals included in analyses was 34.0 (9.0) and the median income level was $40,000 – $49,999. The mean (SD) SLE count was 1.98 (1.70). Mean SLE count differed by sex ($t = 5.75$, $p < 0.0001$), with men endorsing more SLEs on average (M = 2.08, SD = 1.71) than women (M = 1.85, SD = 1.68). Mean resilience score was lower for women (t = 4.66, $p < 0.0001$; mean [SD] = 0.04 [0.97] for men and −0.07 [0.97] for women). The prevalence of psychiatric disorders was as follows: 11.4% (N = 852) MDD at Time 1, 10.9% (N = 813) MDD at Time 2, 6.0% (N =

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449) GAD at Time 1, and 8.4% (N = 628) GAD at Time 2. Significantly more females endorsed MDD or GAD at each time point (X^2 ranging from 9.94–71.91).

Impact of Resilience and SLEs on MDD Risk

Regression results for MDD are shown in Table 1. In Model 1, higher resilience at Time 1 was significantly associated with a lower likelihood of MDD at Time 2 (Beta = −0.06, $p < 0.0001$) and all covariates, including MDD endorsed at Time 1, were significant predictors (all p values <0.05). In Model 2, SLE count at Time 2 was associated with greater likelihood of MDD (Beta = 0.04, $p < 0.0001$). Resilience remained a significant predictor as did all covariates. In Model 3, there was a significant interaction between Resilience and SLE count at Time 2 (Beta = −0.02, $p < 0.0001$), such that resilience buffered the effect of high SLE count, with individuals endorsing more SLEs but higher resilience exhibiting similar likelihood of MDD as those with lower SLE counts. This interaction is presented in Figure 1a. Both resilience and SLEs remained significant predictors of MDD in this model, as did all covariates. In the final model (see Table 1, Model 4) the three-way interaction between resilience, SLEs, and sex was not significant and parameter estimates were nearly identical to those from Model 3, suggesting that sex did not further moderate the effect.

Impact of Resilience and SLEs on GAD Risk

Table 2 shows regression results for GAD. The overall pattern of results was similar to that of MDD. In Model 1, higher resilience at Time 1 significantly predicted a lower probability of endorsing GAD at Time 2 (Beta = −0.06, $p < 0.0001$), with age, income, sex, and GAD at Time 1 all significant covariates (all p values < 0.001). In Model 2, SLEs (Beta = 0.03, p <0.0001), resilience (Beta = −0.05, p <0.0001), and all covariates (all p values < 0.01) were significant predictors of GAD. In Model 3, there was a significant interaction between resilience and SLEs (Beta = −0.02, $p < 0.0001$), such that greater resilience was protective for individuals with higher SLE counts, in that those individuals endorsed similar likelihood of having GAD as those with lower SLE counts (Figure 1b). Within this model, all main effects and covariates remained significant. The 3-way interaction between resilience, SLEs, and sex was not significant in Model 4, while other variables remained significant and parameter estimates were similar to Model 3.

Follow-up Analyses

When the trait variables were added into the models, resilience was a significant buffer in the context of new SLEs, however, the main effect of resilience was no longer significant (see Supplemental Table 1). Finally, follow-up analyses conducted by sex and wave (e.g., FF and MMMF) found that the overall pattern of results was consistent, with the exception of GAD in the FF wave only, with no interaction between resilience and SLEs.

Discussion

Consistent with our hypothesis, resilience at Time 1 buffered against the effects of new-onset SLEs on risk for psychopathology at Time 2. The pattern of findings was the same for MDD and GAD, and consistent across sex. Greater resilience was protective, wherein individuals high in resilience were less likely to develop MDD or GAD even in the context of high

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numbers of past-year SLEs. Of note, resilience is accounting for variance in outcomes above and beyond pre-existing MDD/GAD history and recent stressors, and the interaction of resilience and SLEs was significant even after controlling for covariates, including other trait-based measures we know are related to these outcomes. This supports the "buffering effect" of resilience in the context of additional, or ongoing, life stressors on risk for common internalizing psychopathology. This study adds to the limited literature of longitudinal investigations of the buffering effects of resilience (Hjemdal et al., 2006) and is the first to use a quantitative method of determining resilience. Findings align with existing work that suggests resilience increases the threshold at which individuals react to ongoing daily stressors (Ong et al., 2006) and with the view of resilience as an adaptive process.

Contrary to our hypotheses for the secondary aim, although women had significantly higher probabilities of experiencing MDD or GAD and lower resilience scores than men, sex did not further moderate the interaction of SLE and resilience (i.e., resilience plays a similar buffering role in men and women). Although some literature suggests women have a greater sensitivity to the negative effects of SLEs with regard to depression (e.g., Nazroo, Edwards, & Brown, 1997) and anxiety (e.g., Phillips, Carroll, & Der, 2015), studies report mixed results. Prior work using the present sample suggested that differences in prevalence of MDD were not due to sex differences in overall rates of reported stressful life events nor to differential sensitivity to their pathogenic effects overall (Kendler et al., 2001). Present study findings suggest resilience as a protective factor against stressful life events may work in a similar manner across sexes, although additional work is needed to determine if this remains the case with regard to number, severity, and type of stressors experienced. It should be noted, however, that the range of SLEs was higher for males in this sample, but that given the low endorsements of high numbers of stressors, SLEs were binned in the same manner across sexes. It is unknown if this pattern would translate in samples with greater rates or a larger range of stressors.

The longitudinal nature of this study helps examine how resilient responding can be useful in the context of adversity. Additional longitudinal designs that examine alternative conceptualizations of resilience, as well as trajectory-based studies that include recurring measures of repeated SLEs or traumatic experiences are needed. Although findings were generally consistent across outcomes and different lengths of follow-up times between the two samples, the finding that resilience did not buffer against SLEs with regard to GAD in the FF sample may indicate that the process of resilience differs, or wanes over time, or differs by outcome. The examination of resilience as protective is important, particularly if resilience is indeed modifiable, given prevention and intervention implications (Hjemdal et al., 2006; Ong et al., 2006). Study findings also highlight the need for continued development of interventions aimed at fostering resilience and adaptive coping in the face of stressors as a means of relapse prevention for internalizing conditions. Evidence suggests even low levels of resilience can buffer against lower to moderate levels of anxiety and depression symptoms for individuals in stressful environments (Bitsika, Sharpley, & Bell, 2013) indicating that building resilience (e.g., by teaching adaptive coping skills) may be quite impactful as a preventative effort.

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Limitations

Despite the novel examination of resilience and leveraging a longitudinal design with inclusion of newly occurring SLEs, findings should be interpreted within the context of noted limitations. The SLE assessment was thorough, but not exhaustive. The assessment does not capture the individual's reaction to SLEs or variations in impact of SLEs, and all events were considered as a count and not weighted based on severity of potential impact. Future work would benefit from more fine-grained analyses to determine the strength of association of individual events and event types (e.g., stressful as compared to traumatic; acute as compared to chronic) to appropriately weight these events and allow for a more comprehensive examination of resilience. As has been recently discussed by Hammen (2016), there is a need to attend to differences in acute stressors as compared to chronic or continuous stressful life events. Understanding of the stress-psychopathology link will further develop as studies examine bidirectional relationships (i.e., in addition to SLEs leading to internalizing disorders, individuals with psychological disorders often have greater stressors; Hammen, 2016). This "stress generation" effect is relevant for anxiety and, to a lesser degree, depression (Phillips, Carroll, & Der, 2015). The present study did not have the data available in order to determine temporality and direction of causation between SLEs experienced and onset of MDD/GAD symptoms nor the temporal association between SLEs; longitudinal studies that also have time specific, repeated measures of stressful life events and internalizing conditions will further inform these models.

The quantitative resilience variable used here includes internalizing distress symptoms that share notable overlap, and are potentially correlated with, our outcomes of interest, and thus, could have inflated the strength of the association. However, this overlap between stressors, resilience responding, and outcomes would be expected, and indeed may be desired. A measure of self-reported resilience was not available in this dataset in order to allow for comparisons of alternate conceptualizations, but follow-up analyses using other trait-based measures suggest the robustness of the buffering effect of our resilience variable. While findings indicate the impact of a potential resilience process, our measure of resilience at Time 1 is only a snapshot of this process at one time and cannot speak to a potentially different pattern for incident as compared to recurrent MDD/GAD. This limitation is attenuated, however, as sensitivity analyses (not reported herein) demonstrated that the pattern held whether examining the full sample or new onset MDD/GAD only. It is also important to note that due to the nature of the study design, assessment waves occurred with varying lengths of time between Time 1 and Time 2 measurements across the samples. As such, while SLEs are measured for the past year for all participants, there is variability in the amount of time not captured in that assessment; however, follow-up analyses revealed that the pattern of findings was generally consistent by wave and by sex, with the exception of GAD in the FF wave only, suggesting that the findings are robust. While this exception may indicate a waning of resilience over time or with regard to GAD specifically, previous work in this sample has demonstrated the stability of resilience over time (Amstadter et al., 2014). Further, as noted earlier, the range in number of SLEs differed between males and females, and ratings were binned at the higher ends due to smaller amounts of endorsement. Finally, although we were able to examine sex differences, the sample is primarily Caucasian with a

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fairly low endorsement of SLEs. Replication of this approach in more diverse samples with regard to demographics and greater SLE exposure is warranted.

Conclusion

In conclusion, through longitudinal examination of a quantitative model of resilience and its impact on future internalizing psychopathology in the context of new-onset stressful life events, findings suggest resilience does indeed buffer the negative effects of stressful life events over time. The significant interaction of resilience and SLEs for both MDD and GAD occurred in the context of main effects of resilience as a protective factor and SLEs as an independent risk factor, above and beyond prior psychopathology, suggesting their importance in future risk for internalizing disorders. The lack of an interaction for sex suggests that despite differing levels of resilience, rates of psychopathology, and stressful life events across men and women, the process of resilience as a protective mechanism works in a similar manner across sexes. Present findings align with the growing literature examining resilience as something that can possibly be bolstered and targeted for prevention efforts.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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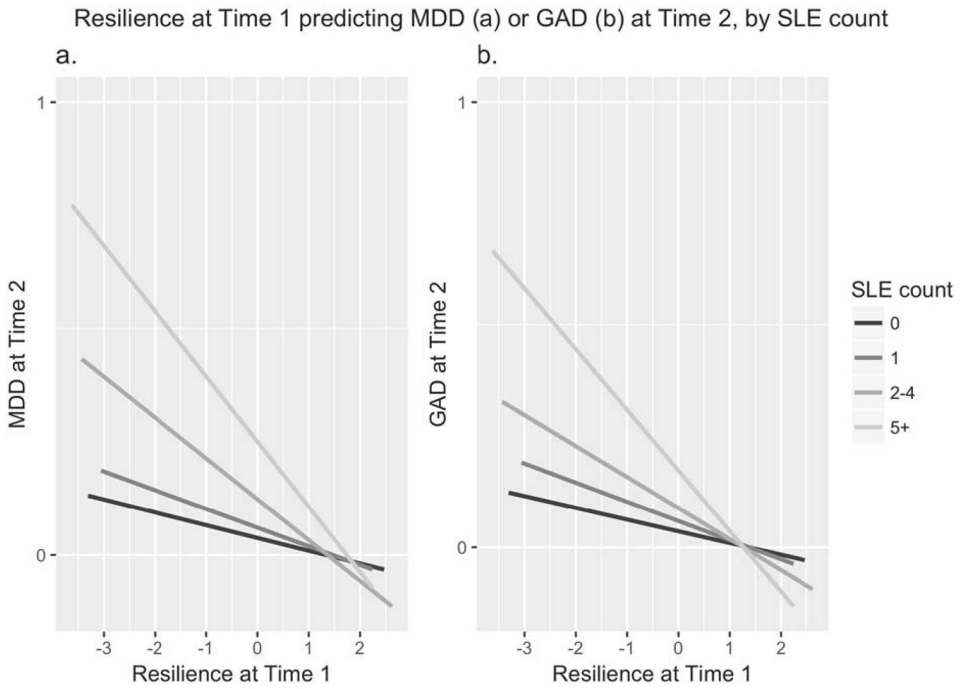

Figure 1.

Resilience at Time 1 predicting (a) MDD and (b) GAD status at Time 2, by SLE count. This figure plots regression lines for resilience at Time 1 (x-axis; resilience measured using the continuous variable quantified as the residuals of internalizing symptoms left over after the effect of number of previous stressful life events [SLEs] has been regressed out) predicting psychopathology diagnosis (y-axis; [a] major depressive disorder [MDD]; or [b] generalized anxiety disorder [GAD]) at Time 2, across different past-year SLE counts (ranging from 0 to 6 events; stressor events have been binned in this figure for readability as 0, 1, 2–4, and 5+). Note that for clarity in interpretation, residual scores are reverse-coded, such that positive scores reflect higher resilience and negative scores represent lower resilience. The significant interaction between resilience and SLEs predicting both outcomes can be visualized in the regression lines (i.e., individuals with higher resilience were less likely to endorse MDD or GAD, regardless of SLE count).

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Table 1

Linear regression models for resilience at Time 1 predicting MDD at Time 2

			95% CI			
		Beta	**Lower**	**Upper**	**Z**	***p*-value**
Model 1	Resilience, T1	−0.06	−0.07	−0.05	−13.73	<0.001
	Age, T1	0.001	0.001	0.002	3.83	<0.001
	Income, T1	−0.15	−0.01	−0.002	−3.86	<0.001
	Female sex	0.02	0.002	0.03	2.28	0.023
	PY MDD, T1	0.22	0.19	0.26	12.86	<0.001
Model 2	Resilience, T1	−0.05	−0.06	−0.04	−11.63	<0.001
	Age, T1	0.002	0.001	0.002	4.50	<0.001
	Income, T1	−0.004	−0.01	−0.002	−3.23	0.001
	Female sex	0.03	0.01	0.04	3.83	<0.001
	PY MDD, T1	0.20	0.17	0.24	11.95	<0.001
	SLEs, T2	0.04	0.03	0.04	15.31	<0.001
Model 3	Resilience, T1	−0.01	−0.02	−0.001	−2.13	0.033
	Age, T1	0.002	0.001	0.002	4.28	<0.001
	Income, T1	−0.004	−0.01	−0.001	−3.05	0.002
	Female sex	0.03	0.01	0.04	3.96	<0.001
	PYMDD, T1	0.19	0.16	0.22	11.12	<0.001
	SLEs, T2	0.04	0.03	0.04	14.78	<0.001
	Resilience, T1*SLEs, T2	−0.02	−0.02	−0.01	−7.31	<0.001
Model 4	Resilience, T1	−0.01	−0.02	−0.001	−2.14	0.032
	Age, T1	0.002	0.001	0.002	4.30	<0.001
	Income, T1	−0.004	−0.01	−0.001	−3.06	0.002
	Female sex	0.03	0.02	0.04	4.26	<0.001
	PYMDD, T1	0.19	0.16	0.22	11.13	<0.001
	SLEs, T2	0.035	0.03	0.04	14.80	<0.001
	Resilience, T1*SLEs, T2	−0.02	−0.02	−0.01	−6.64	<0.001
	Resilience, T1*SLEs, T2*sex	0.002	−0.01	0.01	0.46	0.644

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Abbreviations: PY MDD = past year major depressive disorder, SLEs = stressful life events; T1 = time 1

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Table 2

Linear regression models for resilience at Time 1 predicting GAD at Time 2

			95% CI			
		Effect	**Lower**	**Upper**	**Z**	***p*-value**
Model 1	Resilience, T1	−0.06	−0.07	−0.05	−15.23	<0.001
	Age, T1	0.001	0.001	0.002	3.41	<0.001
	Income, T1	−0.004	−0.01	−0.002	−3.72	<0.001
	Female sex	0.02	0.01	0.04	3.35	<0.001
	PY GAD, T1	0.11	0.07	0.16	5.43	<0.001
Model 2	Resilience, T1	−0.05	−0.06	−0.05	−13.67	<0.001
	Age, T1	0.001	0.001	0.002	3.84	<0.001
	Income, T1	−0.003	−0.01	−0.001	−3.19	0.001
	Female sex	0.03	0.02	0.04	4.50	<0.001
	PY GAD, T1	0.11	0.07	0.147	5.09	<0.001
	SLEs, T2	0.03	0.02	0.03	11.23	<0.001
Model 3	Resilience, T1	−0.02	−0.03	−0.01	−3.16	0.002
	Age, T1	0.001	0.001	0.002	3.59	<0.001
	Income, T1	−0.003	−0.01	−0.001	−2.95	0.003
	Female sex	0.03	0.02	0.04	4.63	<0.001
	PYGAD, T1	0.10	0.06	0.14	4.87	<0.001
	SLEs, T2	0.02	0.02	0.03	10.86	<0.001
	Resilience, T1*SLEs, T2	−0.02	−0.02	−0.01	−7.14	<0.001
Model 4	Resilience, T1	−0.02	−0.03	−0.01	−3.16	0.002
	Age. T1	0.001	0.001	0.002	3.62	<0.001
	Income, T1	−0.003	−0.01	−0.001	−2.95	0.003
	Female sex	0.03	0.02	0.04	4.96	<0.001
	PYGAD, T1	0.10	0.06	0.14	4.88	<0.001
	SLEs, T2	0.02	0.02	0.03	10.85	<0.001
	Resilience, T1*SLEs, T2	−0.02	−0.02	−0.01	−6.49	<0.001
	Resilience, T1*SLEs, T2*sex	0.001	−0.01	0.01	0.33	0.744

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Abbreviations: PY GAD = past year generalized anxiety disorder, SLEs = stressful life events; T1 = time 1