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RESEARCH ARTICLE

Negative caregiving and stress reactivity moderate the relation between early life stress and externalizing in adolescence

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Abstract

Exposure to early life stress (ELS) is common and has been implicated in the development of psychopathology; importantly, however, many individuals who experience ELS do not develop emotional or behavioral difficulties. Prior research implicates stress exposure, negative caregiving behaviors, and patterns of physiological reactivity in predicting psychological well-being; however, the precise factors that contribute to resilience versus vulnerability to the adverse effects of stress exposures are not well understood. In a longitudinal study of adolescents (N = 120) assessed at three timepoints approximately every 2 years beginning at the ages of 913 years, we examined the roles of autonomic reactivity to social stress (assessed through skin conductance during the Trier Social Stress Task) and negative caregiving behaviors as moderators of the association between exposure to ELS and internalizing and externalizing symptoms. We found that the relation between ELS and externalizing symptoms was moderated by both negative caregiving and autonomic reactivity, such that the relation between ELS and externalizing was positive at low levels of negative caregiving and at high levels of autonomic reactivity; interactions predicting internalizing symptoms were not statistically significant. These findings highlight the importance of considering physiological and environmental variables that might contribute to susceptibility or resilience to symptoms of psychopathology following exposure to ELS.

KEYWORDS
early life stress, externalizing, parenting behaviors, skin conductance

1 | INTRODUCTION

A significant proportion of the population experiences early life stress (ELS) (McLaughlin, Greif Green, et al., 2012); in fact, ELS has been estimated to play a role in the development of almost 30% of all psychological disorders (Kessler et al., 2010). ELS exposure has widely been associated with increases in psychopathology symptoms in the domains of internalizing (e.g., anxiety and depressive symptoms) and externalizing (e.g., aggression, rule breaking) (e.g., LeMoult et al., 2019; McLaughlin et al., 2014; Winiarski et al., 2018). It is important to note, however, that not everyone who experiences early adversity subsequently develops emotional or behavioral difficulties. It is likely that environmental factors (e.g., parenting) and individual differences in psychobiological functioning (e.g., neural, endocrine, autonomic stress reactivity) influence emotional development following exposure to ELS, which in turn may attenuate or magnify the negative consequences of adversity. Given these formulations, a growing body of literature is beginning to elucidate the interplay among ELS, environmental factors, and individual differences in psychobiological functioning in the development and expression of psychopathology.

Researchers have implicated individual differences in specific domains of psychobiological functioning in explaining why some

individuals who experience ELS develop symptoms of psychopathology while others do not. For example, investigators have documented that differences in physiological stress responsivity, indexed by the autonomic nervous system (ANS), moderate the relation between ELS and the development of emotion dysregulation. Parasympathetic nervous system activity has been shown to buffer the association between stressors and symptoms of psychopathology (El-Sheikh & Whitson, 2006; Leary & Katz, 2004). Sympathetic nervous system (SNS) function, however, has less consistently been shown to moderate this association. Skin conductance level (SCL) reactivity, a measure of functioning of the SNS, has been found to be potentiated in children with internalizing symptoms (Baker et al., 2013) but blunted in children with externalizing symptoms (Scarpa et al., 1997; Snoek et al., 2004). In the context of stress exposure, higher levels of SCL reactivity have been found to potentiate the relation between stress and internalizing symptoms (Cushman et al., 2021; Fletcher et al., 2019), potentially demonstrating an innate sensitivity to environmental stressors (Boyce & Ellis, 2005). However, the direction of these associations is less clear when predicting externalizing symptoms. For example, exposure to bullying and increased externalizing symptoms have been reported in children with lower levels of SCL reactivity (Gregson et al., 2014). Exposure to parental marital conflict has been found to be associated with increased externalizing behavior among boys with lower SCL reactivity; however, this relation was found across girls regardless of SCL (El-Sheikh et al., 2007). Living in a single-parent household has also been associated with more severe externalizing symptoms in boys with higher, rather than lower, SCL reactivity (Diamond et al., 2012). A stronger association between ELS and externalizing in those with higher SCL reactivity may demonstrate biological sensitivity to stressors (Boyce & Ellis, 2005), whereas studies finding a stronger association in those with low SCL reactivity may demonstrate an insensitivity to stressors, punishment, social learning, and increased risk taking (Del Giudice et al., 2012). Given these varied findings, the moderating role of SNS reactivity in the relation between ELS and symptomatology is unclear. Understanding other formative experiences in relation to skin conductance, such as caregiving, one of the most influential factors in child and adolescent development (Hair et al., 2008; Hoskins, 2014), may help to elucidate these associations.

Caregiving behaviors have been found to either buffer or aggravate the association between ELS and child symptomatology, depending on the valence of the caregiving. For example, positive caregiving behaviors, such as emotionally coaching children, have been found to protect youth who have been exposed to ELS from developing internalizing symptoms (Lobo et al., 2021). Researchers have also found that in children reared in an institution, a positive caregiver relationship was associated with decreases in internalizing symptoms after adoption (Callaghan et al., 2019) or after placement in an experimental foster care intervention (McLaughlin, Zeanah, et al., 2012). Flouri and Midouhas (2016) found that negative caregiving, characterized by harsh discipline, moderated the association between early life adversity and changes in internalizing and externalizing symptoms from the ages of 3–7 years; specifically, they found that either high adversity or high negative caregiving levels were associated with increases in internalizing, and stronger increases in externalizing were observed in children exposed to high levels of both adversity and negative caregiving. Within the same sample, caregiving characterized by warmth toward the child was found to buffer the relation between neighborhood disadvantage and externalizing symptoms, whereas a lack of warmth was associated with high symptoms regardless of neighborhood disadvantage (Flouri et al., 2015). Similarly, a lack of positive caregiving, characterized by emotionally coaching children, has been associated with a stronger link between family stress exposure and internalizing symptoms (Lobo et al. 2021). In sum, these findings suggest that caregiving behavior has the potential to alter associations between ELS and psychopathology symptoms by promoting resiliency in the context of positive caregiving, and increasing susceptibility in the context of negative caregiving.

Biological reactivity and early caregiving behaviors have been shown, independently, to moderate the association between ELS exposure and the development of symptoms of psychopathology. The specific nature of the moderation by biological reactivity is less clear. Whereas high levels of biological reactivity may index sensitivity to ELS (Boyce & Ellis, 2005), low levels of reactivity may reflect insensitivity to reward or punishment (Del Giudice et al., 2012). In contrast, caregiving may influence either resilience or susceptibility, depending on the nature of the caregiving provided. Whether the caregiving environment and biological reactivity interact to predict the development of symptoms following ELS has not yet been explored. It is possible, for example, that negative caregiving behaviors, especially in the context of ELS, are associated with higher levels of symptoms of psychopathology. We do not know, however, whether biological sensitivity to stress comoderates this association, a possibility we explore in the present study.

A few studies have examined the interaction of caregiving and biological reactivity in predicting symptoms in adolescence, but without considering ELS. For example, Erath et al. (2009) found that harsh caregiving was more strongly associated with externalizing symptoms in children with lower skin conductance reactivity. Similarly, Kochanska et al. (2017) found that parental power assertion was associated with externalizing symptoms among children with low SCLs. In both cases, lack of reactivity may reflect an insensitivity to punishment. Conversely, others have found that the association between caregiving and symptoms was stronger in children with higher reactivity. Rousseau et al. (2014) found that negative caregiving, characterized by parental psychological control, was associated with increased internalizing symptoms among adolescents with high SCLs. Further, Wagner and Abaied (2016) found that negative caregiving, also characterized by high parental psychological control, interacted with high skin conductance to predict reactive aggression, and with low stress reactivity to predict proactive aggression in adolescents. Taken together, these studies provide evidence that caregiving and individual stress physiology interact to predict symptoms of psychopathology in young people. However, not only is the direction of these findings inconsistent across studies, but these investigations also often examined only internalizing or externalizing symptoms, and did not consider possible effects of ELS. A comprehensive analysis of these constructs (i.e., both internalizing

and externalizing symptoms as separate outcomes, and ELS exposure as a predictor) will elucidate pathways of susceptibility or resilience to symptoms of psychopathology following exposure to ELS.

In sum, there have been limited but relatively consistent findings in which negative versus positive parenting behaviors accentuate versus attenuate the influence of ELS on psychopathology, whereas the role of stress reactivity as a moderator remains unclear. The current study was designed to extend prior work by testing whether negative caregiving and SCL reactivity, considered independently, moderate the association between ELS and internalizing and externalizing symptoms. Specifically, we expected that negative caregiving will strengthen the relations between ELS and symptoms, such that externalizing symptoms (Hypotheses 1a) and internalizing symptoms (Hypothesis 1b) will be higher in individuals who experienced higher levels of both ELS and negative caregiving. Further, we expected that the positive association between ELS and externalizing symptoms will be stronger at low levels of stress reactivity (Hypothesis 2a) and, in contrast, that the positive association between ELS and internalizing symptoms will be stronger at high levels of stress reactivity (Hypothesis 2b). We expected that caregiving will interact with stress reactivity, such that higher levels of negative caregiving will be associated with more severe externalizing symptoms in the context of lower levels of stress reactivity (Hypothesis 3a), and with more severe internalizing symptoms in the context of higher levels of stress reactivity (Hypothesis 3b). Finally, as exploratory analyses we conducted two separate models examining the possible comoderation of the association between ELS and internalizing and externalizing symptoms by negative caregiving and SCL reactivity, testing the three-way interactions of ELS, negative caregiving, and SCL reactivity predicting internalizing and externalizing symptoms.

2 | METHODS

2.1 | Participants

Participants were drawn from an ongoing longitudinal study of ELS conducted in the California San Francisco Bay Area. Participants were recruited via flyers and local media. Exclusion criteria were postpubertal status, nonfluency in English, inability to undergo magnetic resonance imaging, and history of neurological disorder or major medical illness. Participants were invited to return for follow-up assessments approximately every 2 years. All participants and their legal guardians gave informed assent and consent, respectively, and were compensated for their time. Participants were advised that all study activities were voluntary and that they would be compensated for the activities in which they chose to engage. All study procedures were in accordance with the guidelines set forth by the Declaration of Helsinki and were approved by the Stanford University Institutional Review Board.

Participants were assessed three times at approximately 2 year intervals. At the first timepoint (T1; September 2013–August 2016), 224 participants (50.8% female) were recruited with a mean age of 11.34 years (SD = 1.05). 172 participants (55.7% female) completed the assessment conducted at the second timepoint (T2; June 2015–July 2019), with a mean age of 13.35 years (SD = 1.05). 162 participants (58.5% female) completed the assessment at the third timepoint (T3; February 2018–June 2021), with a mean age of 15.47 years (SD = 1.72). In total, 169 participants completed the TSST; 10 participants were excluded for having noisy skin conductance data. Adolescents reported on their exposure to negative caregiving at T1 and T2, and on their internalizing and externalizing symptoms at all timepoints (T1, T2, and T3; see below). A total of 120 participants had complete data available from all assessments.

2.2 | Procedures

All participants completed assessments of ELS exposure at T1. Because participants must be debriefed following the administration of the Trier Social Stress Test (TSST), it can be administered to each participant only once. Therefore, we randomly selected approximately half of the participants to complete the TSST at T1 and the other half at T2. We recorded ANS reactivity while participants completed the TSST. Self-reported negative caregiving and psychopathology symptoms were assessed concurrent with the TSST, and symptoms were also assessed at T3.

2.3 | Measures

2.3.1 | Early life stress

The Traumatic Events Screening Inventory for Children (TESI-C; Ribbe, 1996), administered at T1, is an interview used to assess lifetime history of the occurrence and subjective experiences of over 30 types of stressful life experiences (e.g., domestic violence, abuse, parental separation). Participants provided details about stressful events, including their subjective rating of the severity of each event. Using a modified version of the UCLA Life Stress Interview coding system (Rudolph et al., 2000), a panel of three to four coders blind to the subjective experience of the participants rated the severity of each event endorsed in the interview on a scale from 0 (nonevent/not impactful) to 4 (extremely severe impact), with high interrater reliability (ICC = 0.99). To account for both the number of stressors and the coded severity of each stressor, we summed the panel ratings across the stressors that were endorsed by each participant. The ELS severity scores ranged from 0 to 28 ($M = 6.93$, SD = 5.41). This operationalization of ELS overcomes limitations of approaches that rely on subjective reports or that focus only on the number or severity of stressful events (Hammen, 2016; Jenness & McLaughlin, 2015).

2.3.2 | SNS reactivity

Participants completed the TSST (Kirschbaum et al., 1993) while we recorded SCL from electrodes on the nondominant hand to measure changes in SNS activity through changes in the electrical potential of

the skin. Electrodes were attached to BioNomadix transducers connected to a Biopac MP150 system that transmitted data wirelessly to a computer with AcqKnowledge data acquisition software at a sample rate of 500 Hz (Biopac Systems, Goleta, CA).

Following a 5-min baseline segment, participants were read a story and told to prepare an exciting ending to the story that would be judged and videotaped. Participants were given 5 min to prepare and 5 min to present. During the presentation, a confederate "judge" maintained a neutral expression and took notes. If participants ended before the 5-min period, they were prompted to continue. If they declined, the participant and judge sat in silence. In the next 5-min period, participants were administered a mental math subtraction task. If they made a mistake, they were asked to start over. Following this task, participants watched a 15-min calming nature scene. Participants then were debriefed about the nature of the test, including that they were not actually judged.

Data were preprocessed and extracted using Autonomic Nervous System Laboratory (ANSLAB; Blechert et al., 2016) software. Signals were visually inspected by trained research staff for drops in the signal or movement artifacts. These anomalies were corrected using the "high smooth" function in ANSlab. The average SCL during the baseline of the TSST was subtracted from the average SCL during each participant's peak SCL period, which was either during the performance or the math portion of the TSST, to index SCL reactivity.

2.3.3 | Negative caregiving

At T1 and T2, participants completed a self-report measure of negative caregiving, defined as parental psychological control and harshness, or lack of warmth toward the child. The 11-item parenting control subscale is derived from the Maternal Psychological Control measure (Olsen et al., 2002) and assesses the degree of control and harshness the parent displays (e.g., "My parent tells me that he/she gets embarrassed when I do not meet his/her expectations"). Participants were asked to rate the caregiving behaviors of the parent or guardian who accompanied them to the assessment. Participants rated the frequency with which the parent displays these behaviors on a scale from 1 (almost never) to 5 (very often). In our sample, Cronbach's alpha was .81 for this scale, indicating good internal consistency.

2.3.4 | Internalizing and externalizing symptoms

At each timepoint participants completed the Youth Self-Report (YSR; Achenbach, 1991), a questionnaire assessing emotional and behavioral problems. Participants are asked to rate 112 items on a three-point scale from 0 (not true) to 2 (very true). The YSR has eight syndrome scales, of which the anxious/depressed, somatic complaints, and withdrawn scales comprise an internalizing score, and aggressive behavior and rule-breaking behavior scales comprise an externalizing score. T-scores adjusted for sex and age were used for all analyses.

2.4 | Analytic plan

All analyses were conducted using the "psych" (Revelle, 2022) and "interactions" (Long, 2021) packages in conjunction with the PROCESS macro (Hayes, 2012) in R Studio v. 2022.2.0.443 (R core team, 2022). We first computed correlations among the variables of interest. We created dummy coded variables for each racial identity (African American/Black, Hispanic, Asian American/Asian, biracial, or other) except for White, which was used as the reference category. ELS measured at T1 was the independent variable, SCL reactivity measured at either T1 or T2 (assigned randomly) and concurrent negative caregiving were entered as moderators, and internalizing and externalizing symptoms measured at T3 were the dependent variables in two separate models. We used the PROCESS macro for R, model number 3, a regression-based procedure that allows for analysis of two moderator variables. We estimated two separate models predicting externalizing and internalizing symptoms with the following interactions: ELS severity × negative caregiving, ELS severity × SCL reactivity, negative caregiving × SCL reactivity, and ELS severity × negative caregiving × SCL reactivity. Models were adjusted for race (which has been linked to skin conductance, see Boucsein, 1992), age at the TSST assessment (i.e., at T1 or T2), and internalizing or externalizing symptoms concurrent with the TSST assessment. Continuous variables were centered, 5000 bootstrap samples were used, and Huber-White heteroscedasticity consistent standard errors were estimated. Finally, we probed significant interactions with simple slopes.

3 | RESULTS

3.1 | Descriptive characteristics and correlations among the variables

Table 1 presents descriptive data for the sample for relevant study variables and results from *t*-tests comparing those participants with T1/T2 TSST data who participated at T3 with those individuals who did not participate at T3. These two groups differed significantly only in Asian racial identity: those who participated at T3 were more likely to identify as Asian than were those who did not participate at T3. Table 2 presents the correlation matrix for the variables assessed in this study. Briefly, ELS severity was positively correlated with internalizing and externalizing symptoms, which were positively correlated as well.

Hypothesis 1: Negative caregiving moderates the relation between ELS severity and symptoms

We hypothesized that ELS severity will be associated with increased externalizing symptoms, and that negative caregiving will moderate this association, such that externalizing symptoms will be most severe in the context of greater ELS and more negative caregiving (Hypothesis 1a). ELS did not significantly predict externalizing symptoms, although negative caregiving did ($B = 0.43$, $p = .004$) (see Table 3 for full results). The interaction between ELS severity and negative caregiving was significant ($B = -0.08$, $p = .017$); however, the direction of the associations

TABLE 1 Demographic characteristics and means and standard deviations of study variables

Demographic characteristics	*M* (SD)	Range	*T*-tests	
Race				
White	44.6%	–	$t(222) = 1.14$,	$p = .257$
Black/African American	8.3%	–	$t(222) = -0.09$,	$p = .926$
Hispanic/Latin	8.9%	–	$t(222) = 0.82$,	$p = .416$
Asian	10.7%	–	$t(222) = -2.21$,	$p = .028$
Biracial	21.0%	–	$t(222) = -0.29$,	$p = .770$
Other	6.3%	–	$t(222) = 0.12$,	$p = .908$
Sex (% female)	50.8%	–	$t(222) = 1.01$,	$p = .313$
Age at TSST	12.24 (1.42)	12.3–15.9	$t(168) = 0.77$,	$p = .444$
Age at T3	15.48 (1.88)	15.5–19.2	$t(160) = 1.00$,	$p = .316$
Early life stress				
ELS severity score	6. 72 (5.10)	0–28.1	$t(213) = 0.72$	$p = .235$
Child-report parenting (T1/2)				
Negative caregiving	21.14 (5.78)	21.1–55.0	$t(206) = -0.70$,	$p = .484$
Skin conductance level (T1/T2)				
Baseline SCL	8.74 (5.57)	8.9–31.6	$t(157) = 0.80$,	$p = .424$
Peak SCL	10.37 (6.14)	10.5–32.4	$t(162) = 0.51$,	$p = .608$
SCL reactivity	1.71 (1.42)	1.7–5.6	$t(157) = -0.70$,	$p = .483$
Youth-self report				
Externalizing symptoms	50.09 (10.44)	29–87	–	
Internalizing symptoms	55.09 (11.37)	27–80	–	

Note: *B*, unstandardized regression coefficients; TSST, Trier Social Stress Test; *T*-tests, results comparing characteristics of those who did versus those who did not participate at T3.

TABLE 2 Correlations among variables of interest

Race	1	2	3	4	5	6	7	8	9	10	11
1. Black/African American	–										
2. Hispanic/Latin	−.10	–									
3. Asian	−.11	−.1	–								
4. Biracial	−.16*	−.16*	−.18**	–							
5. Other	−.08	−.08	−.09	−.13*	–						
6. Age at TSST	.01	−.15	−.10	−.01	.06	–					
7. Negative caregiving at TSST	.04	−.09	.06	.07	.18*	−.02	–				
8. SCL reactivity at TSST	−.02	.04	−.08	.05	−.03	−.09	−.01	–			
9. ELS severity at T1	−.09	.18**	−.10	.05	.00	−.09	.08	.03	–		
10. Externalizing symptoms at T3	−.07	.02	−.06	.04	.01	−.03	.12	.01	.25**	–	
11. Internalizing symptoms at T3	−.12	−.01	.05	.14	.08	−.12	.06	−.02	.25**	.49**	–

Note: $^{**}p < .01$, $^{*}p < .05$. TSST, Trier Social Stress Test; ELS, early life stress; T1, Time 1; T3, Time 3.

TABLE 3 Negative caregiving and SCL reactivity moderate the relation between ELS severity and T3 externalizing symptoms

Race	*B*	SE	*p*	95% CI
Black/African American	−2.66	2.12	.213	−6.86, 1.55
Hispanic/Latin	3.92	3.32	.240	−2.65, 10.49
Asian	−4.49	2.43	.068	−9.32, 0.33
Biracial	−0.16	1.66	.922	−3.46, 3.13
Other	−1.58	3.82	.680	−9.15, 5.99
Age at TSST	−0.07	0.56	.904	−1.17, 1.03
Externalizing at TSST	0.62	0.08	<.001	0.46, 0.79
ELS severity	−0.01	0.13	.911	−0.28, 0.25
Negative caregiving	0.24	0.13	.071	−0.02, 0.50
SCL reactivity	−0.35	0.55	.535	−1.44, 0.75
ELS severity × negative caregiving	−0.07	0.03	.007	−0.13, −0.02
ELS severity × SCL reactivity	0.19	0.09	.031	0.02, 0.36
Negative caregiving × SCL reactivity	−0.12	0.12	.329	−0.36, 0.12
ELS severity × negative caregiving × SCL reactivity	−0.02	0.02	.449	−0.07, 0.03
R^2				0.449
p				<.0001

Note: *B*, unstandardized regression coefficients; TSST, Trier Social Stress Test.

was contrary to our hypothesis. Simple slopes analysis indicated that the relation between ELS severity and externalizing symptoms was positive for participants with low levels (1 SD below the mean) of negative caregiving ($B = 0.88$, $p = .004$), and not significant for those with high levels (1 SD above the mean) of negative caregiving (see Figure 1).

We also expected that negative caregiving will moderate the association between ELS and internalizing symptoms (Hypothesis 1b). Although ELS was positively associated with internalizing symptoms ($B = 0.47$, $p = .02$), the interaction term of ELS severity and negative caregiving was not statistically significant (see Table 4 for full results).

Hypothesis 2: SCL reactivity moderates the relation between ELS severity and symptoms

We hypothesized that SCL reactivity will moderate the relation between ELS severity and externalizing symptoms, such that higher ELS will be associated with more severe symptoms for youth with lower SCL reactivity (Hypothesis 2a). The interaction term of ELS severity and SCL reactivity was significant ($B = 0.23$, $p = .048$); however, the direction of the interaction was not as we predicted. There was a positive association between ELS severity and externalizing symptoms in participants with greater reactivity levels ($B = 0.75$, $p = .01$), but not in participants with lower reactivity levels (see Figure 2).

We also hypothesized that SCL reactivity will moderate the association between ELS and internalizing symptoms, such that higher levels of both ELS and SCL reactivity will predict more severe internalizing symptoms (Hypothesis 2b), but the interaction term was not statistically significant.

Hypothesis 3: SCL reactivity moderates the association between negative caregiving and symptoms

We hypothesized that higher levels of negative caregiving and lower levels of SCL reactivity will predict more severe externalizing symptoms (Hypothesis 3a), and that higher levels of negative caregiving and higher levels of SCL reactivity will predict more severe internalizing symptoms (Hypothesis 3b). Neither interaction term was statistically significant (see Tables 3 and 4, respectively).

3.2 | Exploratory analysis: negative caregiving and SCL reactivity as comoderators of the association between ELS and externalizing and internalizing symptoms

We examined the possibility that negative caregiving and SCL reactivity comoderate the association between ELS and externalizing and internalizing symptoms. The three-way interaction of ELS severity, negative caregiving, and SCL reactivity three-way interaction was not significant when predicting either externalizing or internalizing symptoms (see Tables 3 and 4, respectively), indicating that negative caregiving and SCL reactivity do not interact to comoderate the association between ELS and symptoms of psychopathology.

4 | CONCLUSIONS

The goal of this study was to examine the relations among ELS severity, negative caregiving, SCL reactivity, and externalizing and internalizing symptoms in adolescence. We found that negative caregiving and SCL reactivity each moderated the relation between ELS severity and externalizing, but not internalizing symptoms. Contrary to our hypotheses, the association between ELS severity and externalizing symptoms was positive at low levels of negative caregiving and at

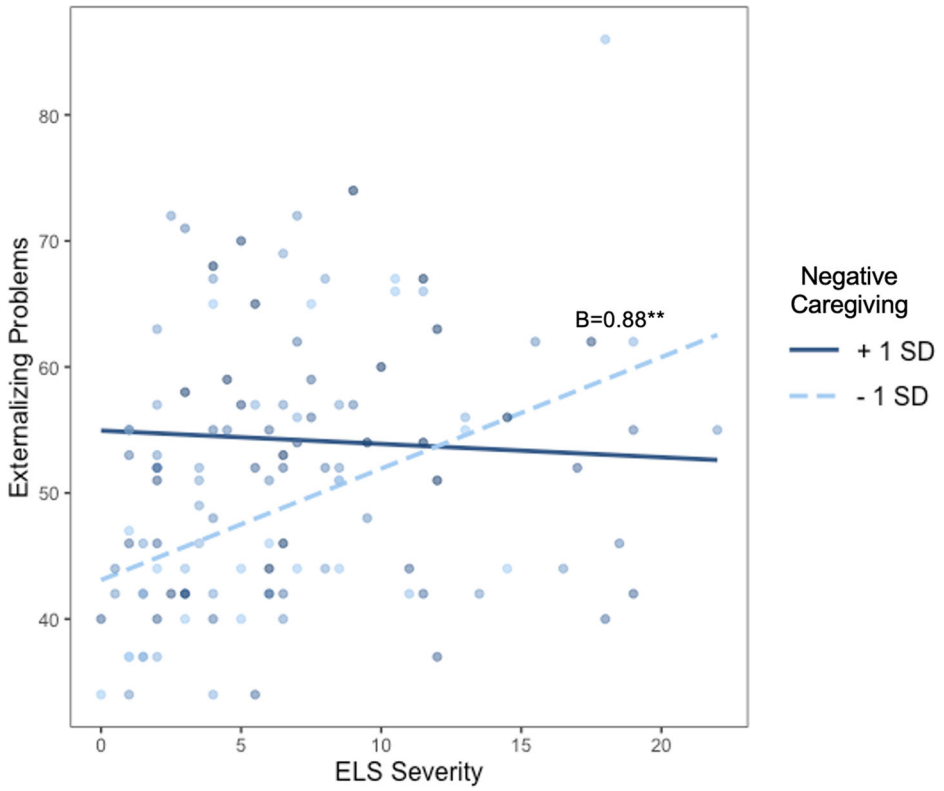

FIGURE 1 Negative caregiving at Time 2 moderates the relation between ELS severity at Time 1 and externalizing problems at Time 3. Note: *B* represents unstandardized regression coefficient, $^{**}p < .01$

TABLE 4 SCL reactivity and negative caregiving do not moderate the relation between ELS severity and T3 internalizing symptoms

Race	*B*	SE	*p*	95% CI
Black/African American	−2.53	2.58	.330	−7.64, 2.59
Hispanic/Latin	−1.79	5.48	.744	−12.66, 9.07
Asian	3.12	3.62	.390	−4.05, 10.29
Biracial	3.45	2.13	.108	−0.78, 7.68
Other	3.96	3.55	.267	−3.08, 11.01
Age at TSST	−0.83	0.70	.244	−2.22, 0.57
Internalizing at TSST	0.43	0.10	<.001	0.24, 0.63
ELS severity	0.24	0.20	.234	−0.16, 0.63
Negative caregiving	0.004	0.15	.981	−0.30, 0.31
SCL reactivity	−0.41	0.63	.516	−1.65, 0.83
ELS severity × negative caregiving	−0.03	0.04	.332	−0.10, 0.04
ELS severity × SCL reactivity	−0.08	0.12	.510	−0.32, 0.16
Negative caregiving × SCL reactivity	0.07	0.14	.600	−0.21, 0.36
ELS severity × negative caregiving × SCL reactivity	−0.03	0.03	.389	−0.09, 0.04
R^2				0.331
p				<.0001

Note: *B*, unstandardized regression coefficients; TSST, Trier Social Stress Test.

high levels of reactivity. Negative caregiving and SCL reactivity did not interact to predict externalizing or internalizing symptoms. These findings underscore the importance of considering both environmental factors and individual psychobiological variability when examining emotional development following exposure to ELS.

We had expected to find that higher ELS severity in the context of higher negative caregiving would be associated with more severe internalizing and externalizing symptoms, as others have found (e.g., Shaw & Starr, 2019). Instead, we found that ELS was positively associated with externalizing at lower levels of negative caregiving, and the

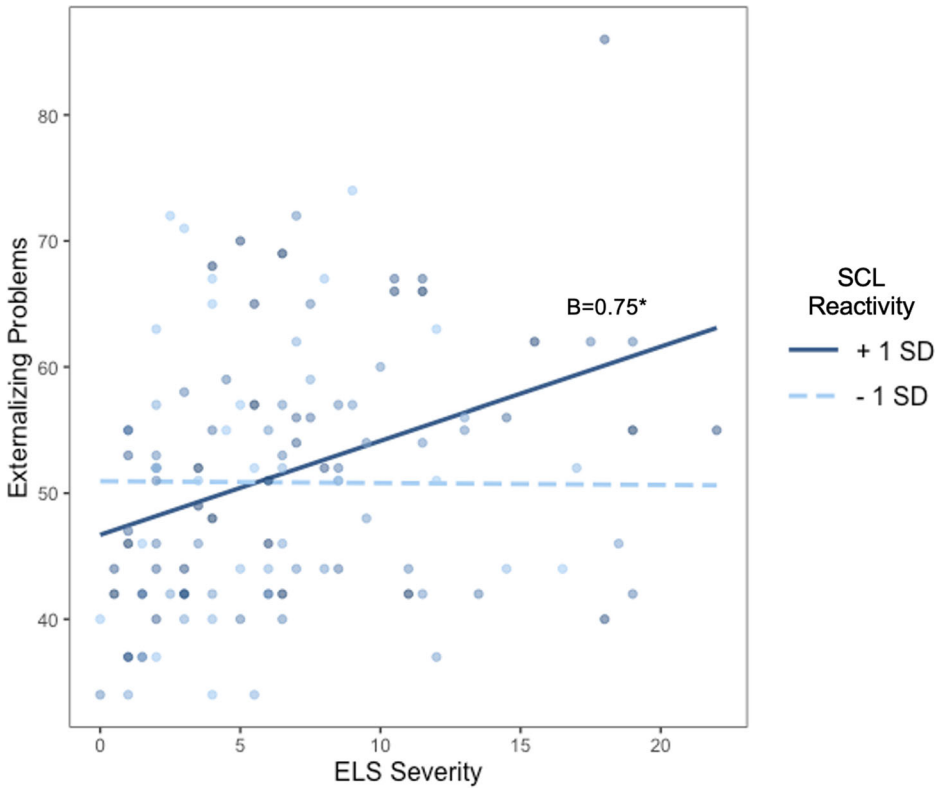

FIGURE 2 SCL reactivity at Time 2 moderates the relation between ELS severity at Time 1 and externalizing problems and Time 3. Note: *B* represents unstandardized regression coefficient, $^{*}p < .05$

interaction predicting internalizing was not significant. Further, externalizing symptoms were lowest at lower levels of both ELS and negative caregiving, which is supported by prior work (e.g., Flouri & Midouhas, 2016). It is possible that our findings would have been in the expected direction had we measured positive caregiving, which may be different from a lack of negative caregiving. That is, a lack of negative caregiving may not necessarily imply the presence of caregiving warmth or support; rather, it may imply the presence of permissive or neglectful caregiving.

We also hypothesized that ELS severity would be associated with more severe internalizing symptoms in the context of higher SCL reactivity, and with more severe externalizing in the context of lower SCL reactivity. The interaction predicting internalizing was not significant; however, the significant interaction predicting externalizing was not in the expected direction. The association between ELS and externalizing symptoms was positive in the context of higher SCL reactivity. We hypothesized that lower reactivity would strengthen the association because of a body of literature linking externalizing with lower reactivity, particularly skin conductance (El-Sheikh et al., 2007; Gregson et al., 2014). However, prior work has had mixed findings in this domain, with some finding that the stress-externalizing relation is stronger in those with greater reactivity (Winiarski et al., 2018), and others finding the relation is stronger in those with lower reactivity (Busso et al., 2017). We also found that at lower levels of reactivity, the relation between ELS and externalizing symptoms was relatively flat. This may suggest that decreased reactivity promotes resilience, or decreased sensitivity, to ELS exposure, whereas those with higher levels of reactivity may be more sensitive to ELS exposure (Boyce & Ellis, 2005). Notably, internalizing and externalizing were highly correlated in this sample ($r = .49$), which may have conflated the symptom domain-reactivity relation often reported in the literature.

Our final hypothesis was that higher negative caregiving would predict internalizing symptoms in the context of higher reactivity, and externalizing symptoms in the context of lower reactivity. These interaction terms were not significant. Prior work has had conflicting results, with some finding that lower reactivity moderated the association between negative caregiving and symptoms (Erath et al., 2009; Kochanska et al., 2017), and others findings that higher reactivity moderated the association between negative caregiving and symptoms (Rousseau et al., 2014). Future work examining different aspects of caregiving, such as harshness, affection, support, and neglect, may be able to elucidate these associations.

We should note five limitations of this study. First, although the study design was longitudinal, we cannot make strong inferences about causality. We also acknowledge the likely bidirectional relation between parenting behaviors and child symptoms (Rothenberg et al., 2020), and recognize that we asked about the caregiving behavior of only one parent. Second, the sample was relatively affluent, perhaps limiting the effects of ELS in this sample and possibly constraining the generalizability of our findings to a wider range of SES. Third, we relied on a single measure of SNS reactivity, and the reliability of this measure may be limited by directional fractionation of the ANS (Jänig &

Häbler, 2000; Lacey, 1967). Fourth, we relied on adolescent-reported measures of symptoms and caregiving, which may be biased. We also used the UCLA coding system to create objective ratings blind to the subjective reports of participants assessed with the TESI. Although this procedure has the advantage of yielding ratings of the objective severity of experienced events, it does deviate from the intended use of the TESI, which was designed, in part, to assess subjective responses to stressors. Lastly, our small sample size may have been underpowered to detect all the tested interactions, and prevented us from analyzing potential sex differences.

Despite these limitations, in this study we did elucidate the interplay of ELS severity with negative caregiving and with stress reactivity in predicting externalizing symptoms. Specifically, higher sympathetic reactivity and lower negative caregiving moderated the positive association between ELS and externalizing symptoms. Our finding that ELS in the context of greater reactivity is associated with more severe externalizing symptoms may reflect a biological sensitivity to environmental stressors. Future work in a larger sample that examines more facets of caregiving, including warmth and structure, may shed more light on the interaction we found between ELS and negative caregiving. Notable strengths of this study include the longitudinal design, the use of a structured interview to assess ELS exposure, and child—rather than parent—report of caregiving. Our findings underscore the value of considering the roles of both environmental and individual factors in examining pathways to susceptibility or resilience to psychopathology symptoms, particularly in the domain of externalizing, following ELS exposure.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

DATA AVAILABILITY STATEMENT

Data will be made available upon request.

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