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Reciprocal effects of neuroticism and life stress in adolescence

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Abstract

Background: Stressful life experiences and personality can influence one another. Personality may contribute to the amount and type of stress individuals experience, which is referred to as a selection effect. Life stress may also impact one's personality, which is referred to as a socialization effect. It was hypothesized that neuroticism would predict increased chronic and episodic stress (selection effect) and that chronic and episodic stress would predict increased neuroticism (socialization effect).

Methods: The current study investigated selection and socialization effects of neuroticism and life stress over a three-year period in 627 adolescents. Life stress data were examined in terms of duration (chronic versus episodic) and type (interpersonal versus non-interpersonal). Episodic stress data were examined as dependent or independent.

Results: The results from ten cross-lagged panel models provided some evidence for significant selection and socialization effects depending on stress type. Over three years, we observed that neuroticism increases interpersonal chronic stress and non-interpersonal stressful events (selection effects) and that dependent non-interpersonal stressful events and chronic stress increase neuroticism (socialization effects).

Limitations: Study limitations include a lack of a lifespan perspective and a statistical approach that does not differentiate between- from within-person variance.

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Conclusions: Findings suggest the value of attending to stress response as well as targeting neuroticism in prevention and intervention approaches in adolescents.

Keywords

neuroticism; chronic stress; episodic stress; selection effect; socialization effect

Introduction

Personality—which reflects differences in magnitude or likelihood of reactions to trait-relevant challenges (Caspi et al., 2005)—can influence one's life experiences; similarly, life experiences can shape one's personality. Personality predicting occurrence of subsequent stress is referred to as a selection effect, whereas personality change in response to stress is referred to as a socialization effect (Specht et al., 2011). Consequently, the impact of life experiences cannot be viewed independently from personality (Magnus et al., 1993), in part due to genetic influences (Clarke et al., 2018). Individual differences are likely to alter the amount and content of stress to which individuals are exposed, which in turn may change individuals' views of their surroundings, relationships, and actions. Neuroticism has been identified as a particularly important personality construct, given its associations with increased life stress and negative life outcomes (Jeronimus et al., 2015). The purpose of this longitudinal study is to systematically examine selection and socialization effects between neuroticism and life stress in adolescents. This will enable proper identification of intervention targets in the context of high neuroticism and/or stress-exposed individuals at risk for developing psychopathology.

Neuroticism

Neuroticism refers to individual differences in the tendency to experience and express negative emotions that are intense and enduring (Shackman et al., 2016). Individuals with high neuroticism are more negatively reactive and prone to unpleasant emotions compared to individuals with low neuroticism (Eysenck, 1967). Neuroticism is relatively stable but also susceptible to change (Shackman et al., 2016). There is evidence to suggest that neuroticism changes over the lifespan (Roberts & DelVecchio, 2000; Specht et al., 2011). Given that neuroticism is a robust risk factor for psychopathology (Bucher et al., 2019; Hur et al., 2019; Michelini et al., 2020; Ormel et al., 2013; Zinbarg et al., 2016), negative physical health outcomes (Charles et al., 2008; Hintsanen et al., 2014), and negative life circumstances, such as premature mortality (Graham et al., 2017; Puterman et al., 2020) and economic burden (Cuijpers et al., 2010; ten Have et al., 2005), examining factors that modify neuroticism has theoretical and practical importance.

Life Stress

Life stress acts together with underlying vulnerabilities to contribute to psychopathology (diathesis-stress models; Beck, 1987) and continued experience of stress (stress generation; Hammen, 1991). Whereas the vast majority of research on stress and negative outcomes has focused on acute predictors ("episodic stress"), prolonged exposure to stress ("chronic stress") is another predictor of negative outcomes (Hammen, 2005). We have previously

shown in this sample that both acute and chronic stress prospectively predict depression outcomes over one- and five-year periods (Mineka et al., 2020; Vrshek-Schallhorn et al., 2015), and that episodic stress predicted depression and anxiety onsets over a one-year period (Uliaszek et al., 2012). Together, this work supports prospective associations between life stress and internalizing psychopathology and suggests that investigating predictors of life stress is crucial to better understanding disorder onset and maintenance.

Selection and Socialization Effects

Selection Effects—As mentioned above, selection effects are those in which personality influences stress. In accord with selection effects, individuals with high neuroticism tend to self-select into stressful environments and conduct their lives in ways that encourage increased stress (e.g., negatively interpreting environmental cues; Jeronimus et al., 2015). Neuroticism can dispose individuals to more interpersonal stress (e.g., relationship instability) and non-interpersonal stress (e.g., unemployment; see Jeronimus et al., 2015 for review). Associations between neuroticism and stressful life events have been supported through longitudinal investigations (Boals et al., 2015; Jeronimus et al., 2014; Kendler et al., 2003; Lüdtke et al., 2011; Magnus et al., 1993; Specht et al., 2011; Uliaszek et al., 2012; van Os & Jones, 1999, 2001). Limited work specifically examines selection effects with a focus on interpersonal stressful life events (Gunthert et al., 1999; Kendler et al., 2002; Poulton & Andrews, 1992) or chronic stress (Jeronimus et al., 2014). We previously showed that neuroticism predicted greater contextual threat of total episodic life stress and interpersonal chronic stress over one-year (Uliaszek et al., 2012). Research therefore supports selection effects between neuroticism and life stress, but extant literature is skewed toward stressful life events, neglecting chronic stress and lacking stressor characteristic specification (e.g., dependent versus independent).

Socialization Effects—In line with socialization effects, wherein stress impacts personality, negative life events and major stressors may intensify propensities that originally contributed to stressors (Jeronimus et al., 2014). It is theorized that stressful events place a burden on individuals that tax their abilities to respond adaptively to stress (Jeronimus et al., 2015). Prospective research supports the contribution of stressful life events to increased neuroticism (Boals et al., 2015; Goldstein et al., 2019; Jeronimus et al., 2013; Lüdtke et al., 2011; Riese et al., 2014). The socialization effect between neuroticism and chronic stress is less studied, but existing work suggests that chronic stress contributes to small, persistent changes in neuroticism (Jeronimus et al., 2014).

Gaps in The Literature

Together, the aforementioned research supports bidirectional associations between neuroticism and life stress. Yet, there are a number of gaps in the literature. Existing research has focused on the number of acute stressful life events over a specified time period but often fails to consider the impact of chronic stress in selection and socialization. Stressful life events have been studied generically as opposed to separately examining interpersonal versus non-interpersonal and dependent versus independent stressors. The interpersonal nature of an event is important to examine because it informs whether difficulties with family, peers, or significant others as opposed to occupational, educational,

or health-related difficulties are contributors to negative outcomes. Further, the dependence of stressors is also crucial as it can distinguish whether or not stressors resulting from one's actions—as opposed to one beyond the individual's control—have adverse effects. It is important to evaluate specific types because identification of stress types that can impact personality, and vice versa, allows for more informed interventions. There is also limited work focusing on selection and socialization effects in adolescents. Adolescence is a developmental period marked by pervasive stress (Compas et al., 1993), increased stress-response activity and emotional reactivity (Dahl & Gunnar, 2009), and psychopathology onset linked to stress (Hammen, 2005). Further clarification of neuroticism-stress relationships could therefore elucidate how to prevent negative outcomes in adolescents.

The Present Study

The present study aims to better understand the longitudinal selection and socialization pathways between neuroticism [operationalized here as a general neuroticism factor (GNF)] and life stress, and to evaluate the impact of stress that varies in duration and content in an adolescent sample. A better understanding of these relationships can inform prevention strategies to curb the onset of psychopathology associated with high neuroticism. The goals of the present study were to longitudinally examine whether (1) neuroticism predicts future stress beyond the effect of prior stress (selection effect) and (2) life stress predicts future neuroticism beyond the effect of prior neuroticism (socialization effect) in adolescents. We also examined the contribution of interpersonal versus non-interpersonal stress within the chronic and episodic models and dependent versus independent stress within the episodic models. First, consistent with a selection effect, we hypothesized that neuroticism would predict increased chronic and episodic stress over three years. Consistent with a socialization effect, we also hypothesized that chronic and episodic stress would predict increased neuroticism levels over three years. We explored the impact of chronic versus episodic, interpersonal versus non-interpersonal, and dependent versus independent stress upon the selection and socialization effects.

Methods

Participants

627 adolescents aged 15-17 years old enrolled in a two-site, 8-to-10-year longitudinal study who completed structured life stress interviews and self-report measures over a three-year study period were eligible for inclusion in the analyses. The umbrella study aimed to examine risk factors for psychopathology during the transition into early adulthood (Youth Emotion Project; Zinbarg et al., 2010) in three cohorts of high school juniors from two diverse public high schools in Chicago and Los Angeles. To increase the likelihood of psychopathology over the study course, individuals exhibiting high neuroticism levels (i.e., top tertile) as measured by the Neuroticism subscale of the revised 23-item Eysenck Personality Questionnaire Neuroticism Scale (Eysenck & Eysenck, 1975) were oversampled (Clark et al., 1994; Hayward et al., 2000). Detailed sampling procedures can be found in Zinbarg et al. (2010). The resulting sample was 68.9% female and 48.2% White, 15.3% Hispanic/Latino, 13.1% African American, 4.3% Asian, 0.6% Pacific Islander, 13.1% Multiracial, and 5.4% "Other." Participant socioeconomic status was coded based on

participants' report of their parents' educational attainment and occupational status (Hollingshead, 1975). This scale is designed to range from 0-66, with scores 40 indicating trained/professional parental employment and scores <20 indicating unskilled parental employment. Our data suggest that many participants reported parental occupations requiring minimal formal training (*M*=48.08, *Mdn*=51.00, *SD*= 12.94).

Procedure

Participants in the final sample completed an annual battery of self-report inventories measuring neuroticism and related cognitive vulnerabilities as well as life stress interviews. Supplementary Table 1 shows the number of participants who completed questionnaires and life stress interviews at each wave over three years. These data highlight substantial attrition.

We examined potential associations between missing data, demographic characteristics, and key study variables. First, we computed the total number of missing observations for each participant regardless of timepoint. Missing data at each of the four waves was coded dichotomously (0=not missing, 1=missing) and summed for each key study variable (neuroticism (GNF), chronic stress, episodic stress) separately. Pearson correlations between the total number of missing observations on GNF, chronic stress, and episodic stress and SES measured at baseline were computed. There were no significant associations between missing data on GNF, chronic stress, or episodic stress and SES (*p*s>.05). Independent samples *t*-tests comparing missing data on GNF, chronic life stress, and episodic life stress showed no differences between participants of different gender (male=0, female=1) or ethnic minority status (Caucasian=0, ethnic minority=1) (*p*s>.19).

Measures

Neuroticism and its Facets.—Factor scores were derived from a general neuroticism factor (GNF), originally developed as part of a hierarchical neuroticism model validated in Zinbarg et al. (2016). In this model, neuroticism is defined by traditional scales measuring neuroticism as well as other measures of cognitive risk for depression and anxiety, such as cognitive style and anxiety sensitivity. This approach is consistent with the premise that these cognitive risk constructs are facets of neuroticism (e.g., pessimism; Scheier, et al., 1994).

The hierarchical model was derived from eight vulnerability questionnaires completed by participants at baseline: Eysenck Personality Questionnaire-Revised, Neuroticism Scale (Eysenck & Eysenck, 1975); International Personality Item Pool-NEO-PI-R (Goldberg, 1999); Behavioral Inhibition Scale (Carver & White, 1994); Big Five Mini-Markers N Scale (Saucier, 1994); Cognitive Style Questionnaire (Alloy et al., 2000; Hankin et al., 2004); Dysfunctional Attitudes Scale (Weissman & Beck, 1978); Personal Style Inventory (Robins et al., 1994); Anxiety Sensitivity Index–Expanded Form (Li & Zinbarg, 2007; Reiss et al., 1986). Each of these measures contains some variance attributable to the GNF which is the factor general to all vulnerability indicators (Zinbarg et al., 2016). Modeling specifics can be found in Zinbarg et al. (2016). Factor scores previously generated from this model were used in the current analyses.

As factor scores correspond to the GNF, they reflect variance that is common to depression and anxiety items. One of the advantages of operationalizing neuroticism using the hierarchical factor model, as used here, is that it mitigates item overlap problems with criterion variables such as anxiety or depression (Uliaszek et al., 2009). Specifically, the Uliaszek et al. (2009) hierarchical factor model approach features group factors which cleanly parse the variance of the general neuroticism factor from group factors which reflect more specific facets of neuroticism. Further, the general and group factors are specified as being orthogonal in the hierarchical factor model (e.g., McDonald, 1999). Thus, the variance associated with the content specific to a particular facet of neuroticism—such as depression-proneness or anxiety-proneness—is apportioned to its group factor and is parsed from the variance common to all neuroticism facets which is apportioned to the general factor.

Chronic Stress.—The UCLA Life Stress Interview (LSI; Hammen, 1991; Hammen et al., 1987), a semi-structured interview of factual information about ongoing, typical conditions in ten life domains, was used as the measure of life stress at baseline and annual follow-up timepoints. The LSI administered at each follow-up interview assessed stress types occurring in the interim since the previous interview, unless an interview had been missed, in which case only the previous 12 months were assessed. Each LSI domain was rated by trained interviewers to indicate the severity of chronic stress on a behaviorally anchored scale ranging from 1 (*minimal stress*) to 5 (*very stressful circumstances*), using half-point increments. Scores of 1 and 5 were considered rare and indexed relatively extreme cases. Interviewer ratings were based on objective information about each domain.

To assess chronic interpersonal and non-interpersonal stress separately, role domains of (a) close friendships, (b) social life, (c) romantic relationships, and (d) family relationships were categorized as interpersonal, whereas domains of (e) neighborhood/dorm environment, (f) academic performance, (g) work environment, (h) financial status, (i) personal health, and (j) family member health were considered non-interpersonal. To determine baseline reliability of chronic life stress in the present study, intraclass correlation coefficients (ICCs) were calculated using 76 intersite- and intrasite-rated audiotaped interviews. The cross-site ICCs ranged from .57-.91 for each domain and averaged .73 across domains. The average ICC for the interpersonal domain was .71 (Doane et al., 2013). For current analyses, we explored selection and socialization effects with chronic stress with three models: total chronic stress, interpersonal chronic stress, and non-interpersonal chronic stress. Composite scores for chronic interpersonal and non-interpersonal stress were calculated by averaging category-relevant domain scores. Total chronic stress composite scores were calculated by averaging across all domains.

Episodic Stress.—Acute stressful life events were probed within each chronic life stress domain. Interpersonal and non-interpersonal stress were examined separately. The rating team assigned a code to describe each event from a modified Paykel and Mangen (1980) event list. Events were coded a priori by content (primarily interpersonal or not). Interpersonal content referred to situations primarily involving or affecting participant relationships. Around 10% of events at each timepoint were unspecified and coded as non-interpersonal.

Interviewers obtained factual details concerning the description and date of the event, the degree, duration, and impact of its consequences, the participan's prior experience with the event, and availability of social support. This information was later presented by the interviewer to an independent team of two raters who evaluated the event on contextual threat severity. Contextual threat was assessed by objectively rating how much impact a particular episodic event would have for the average person in those exact circumstances. Ratings for episodic events were made on a 1-5 scale: 1 (*minimal or no threat*), 2 (*mild threat*), 3 (*moderate threat*), 4 (*marked impact with many consequences*), and 5 (*severe and catastrophic negative impact*). Any subjective impressions the participants offered about event stressfulness were not presented to raters. Time 1 reliability was assessed by rating 208 audio recordings of life events across sites. The ICC for event threat ratings was .84 (Doane et al., 2013).

In addition to a severity rating for episodic events, dependence was assessed by objectively rating how much the respondent was responsible for causing or contributing to the event. Ratings for dependence were made on a 1-5 scale: 1 (*almost completely independent*), 3 (*mixed*), 5 (*almost completely dependent*). Most interpersonal events were rated a 3 based on information that the event was at least partially the result of the respondent. If raters could not reach consensus, the episode was presented to a third rater who helped raters reach consensus. The ICC was for dependence ratings was .90 (Doane et al., 2013).

For current analyses, we explored selection and socialization effects of episodic stress with seven models: total stressful events (severity ≥ 2.5), interpersonal and non-interpersonal stressful events (severity ≥ 2.5), and independent (dependence <2) and dependent events (dependence ≥ 3) within the interpersonal and non-interpersonal domains.

All study procedures were approved by Institutional Review Boards at Northwestern University (protocol #00007246) and University of California, Los Angeles (protocol #10-001607).

Data Analysis

Analyses were conducted using Mplus Version 8 (Muthén & Muthén, 1998-2017). We constructed ten cross-lagged panel models (CLPM; Campbell, 1963; Kenny, 1975) to examine relationships between neuroticism and life stress over a three-year period (four timepoints).

Model selection was performed in a series of steps. First, we estimated saturated models (i.e., all possible paths, freely estimated). Next, we assumed stationarity in six relationships across time; thus, we fixed regression coefficients of the same path type to be equal. Stationarity was imposed because it is believed that the degree to which neuroticism produces change in stress, and vice versa, remains the same over time (Cole & Maxwell, 2003). The six regression coefficients constrained to the same value at each wave were: (1) autoregressive paths from stress at wave t-1 to stress at wave t, (2) selection effects represented in the path from GNF at wave t-1 to the stress at wave t, (3) socialization effects represented in the path from the stress at wave t-1 to GNF at wave t, (4) from GNF at wave t-1 to the GNF at wave t, (5) gender effect of stress, and (6) gender effect of GNF. Although

there was no theoretical reason to suspect deviations from stationarity, we compared saturated CLPMs to CLPMs with constrained paths for each stress type to determine whether the stationarity assumption was justified. We then removed lagged paths between neuroticism and stress (i.e., GNF(t)-stress(t+2; t+3); stress(t)-GNF(t+2; t+3)). Because further removal of paths introduced model misfit, the model selection process stopped. We examined objective fit indices of models using conventional cut-offs for root mean square error of approximation (RMSEA) of 0.06, standardized root mean square residual (SRMR) of .08, and comparative fit index (CFI) 0.95 (Hooper et al., 2008). Fit indices for selected models are shown in Table 1. Fit indices for all models tested in the model fitting sequence are available in Supplementary Table 2.

Multiple paths were estimated in the final structural equation models: the six coefficients constrained to equality as well as six freely estimated paths to capture lagged stress effects (stress at wave t to stress at wave t+2; stress at wave t to stress at wave t+3) and lagged GNF effects (GNF at wave t to GNF at wave t+2; GNF at wave t to GNF at wave t+3). The covariance and residual covariances between the GNF and stress were also estimated (see Figure 1). A structural equation model of the same path types was arranged to examine selection and socialization effects for ten stress types: chronic stress, chronic interpersonal stress, chronic non-interpersonal stress, episodic stress, episodic interpersonal stress, episodic non-interpersonal stress, dependent episodic interpersonal stress, independent episodic interpersonal stress, dependent episodic non-interpersonal stress, and independent episodic non-interpersonal stress.

For CLPM analyses, we used maximum likelihood estimation to accommodate missing data and robust standard errors to mitigate the impact of skewed stress variables on significance tests. All stress variables were converted to *z*-scores prior to analyses for ease of comparison across indices. Given research demonstrating gender differences in neuroticism (Costa et al., 2001) and other neuroticism facets (e.g., Hankin & Abramson, 2001), gender was included as a covariate in all regressions. The Holm correction factor was applied to a $p < .05$ threshold within each model for paths with corresponding hypotheses (selection effect; socialization effect) (Holm, 1979).

Results

Stress Descriptive Statistics

Table 2 contains descriptive statistics for stress variables in our sample. Across chronic stress variables, participants experienced between mild to moderate stressors on average. Table 3 contains frequencies and descriptions of the most commonly endorsed stressful life events in our sample. Common interpersonal events included the end of dating relationships and serious arguments or problems with friends. Common specified non-interpersonal events included traffic accidents and minor physical illness or injuries.

Stationarity Assumption

Fit indices for saturated models and models assuming stationarity for six regression paths can be found in Supplementary Table 2. We found that the stationarity assumption was

violated only in the independent episodic non-interpersonal stress model. Therefore, model fit improved as a result of freeing all paths in this model. Parameters were examined to localize the source of misfit. The paths capturing the selection effect led to enhanced model fit when they were freed, suggesting that this path is not as stable as assumed. Therefore, the selection effect paths in the independent episodic non-interpersonal stress model were freely estimated for the present analyses.

Stress and Neuroticism Effects

Table 4 summarizes study findings. Table 5 displays estimates for selection and socialization effects, as well as R^2 values, for ten (three chronic; seven episodic) models.

Magnitude of effects.—R^2 effect sizes for chronic stress types ranged from .27 to .35, indicating medium to large effects (Cohen, 1988). R^2 effect sizes for episodic stress types ranged from .02 to .09, indicating small effects (Cohen, 1988). R^2 effect sizes for neuroticism ranged from .08 to .09, indicating small effects (Cohen, 1988).

Selection effects.—Neuroticism significantly predicted increased chronic interpersonal stress, β=.06, SE=.02, z=2.83, p=.005. Selection effects were not apparent for total chronic stress or chronic non-interpersonal stress. Neuroticism significantly predicted increased total non-interpersonal episodic stress (β=.08, SE=.03, z=2.41, p=.016), dependent non-interpersonal episodic stress (β=.08, SE=.03, z=2.59, p=.01). In the independent non-interpersonal episodic stress model, neuroticism at 2-year follow-up predicted stress at 3-year follow-up ($β$=.25, SE=.06, z=4.06, p<.001), but there were no significant relationships between neuroticism and stress at other timepoints in this model. Selection effects were not apparent for total episodic stress or episodic interpersonal stress models.

Socialization effects.—Total chronic stress (β=.11, SE=.03, z=3.50, p<.001), chronic interpersonal stress (β=.10, SE=.03, z=3.36, p=.001), and chronic non-interpersonal stress (β=.09, SE=.03, z=2.91, p=.004) significantly predicted increased neuroticism. Total episodic non-interpersonal stress (β=.07, SE=.03, z=2.40, p=.017) and dependent episodic non-interpersonal stress (β=.06, SE=.02, z=2.27, p=.02) significantly predicted increased neuroticism. Socialization effects were not apparent for total episodic stress, episodic interpersonal stress, or independent non-interpersonal stress models.

Discussion

Our results provide evidence for selection (neuroticism predicting stress) and socialization (stress predicting neuroticism) effects (Specht et al., 2011), which were dependent on stress type. Specifically, our results demonstrate that chronic interpersonal stress and dependent episodic non-interpersonal stress have reciprocal stress-neuroticism relationships. Overall, effects of chronic stress ranged from medium to large in magnitude, whereas effects of episodic and neuroticism ranged from small to medium in magnitude. Selection and socialization effects found in the literature are characterized primarily by small to medium effects (e.g., Jeronimus et al., 2013; Jeronimus et al., 2014; Lüdtke et al., 2011). The magnitude of observed effects suggests that these effects in the population may be more modest than expected.

Only socialization effects were observed in the total chronic stress model, suggesting that the experience of overall chronic stressors may be a particularly potent predictor of higher levels of neuroticism. When chronic stress was assessed apart from acute stress, socialization effects were apparent in interpersonal and non-interpersonal domains, indicating that both stress types influence neuroticism. This suggests that experiences of ongoing interpersonal strain, instability in one's environment, or longstanding difficulties with finances or health may strengthen an individual's tendency to experience and express negative emotions.

This finding is consistent with previous reports of neuroticism changes in response to interpersonal relationships as well as in response to non-interpersonal properties of one's situation (e.g., occupation; Jeronimus et al., 2015). In contrast, the selection effect was evident for interpersonal but not for non-interpersonal chronic stress, suggesting that neuroticism contributes to only interpersonal chronic stress. This implies that individuals with high neuroticism are more likely to experience poor quality friendships, social lives, romantic and family relationships and does not necessarily increase one's likelihood of experiencing environmental instability or longstanding financial or health problems. This finding concords with previous work demonstrating that high neuroticism predicts low social support (Kendler et al., 2002), low relational satisfaction (O'Meara & South, 2019), and poor marital quality (Barelds, 2005).

Neither selection nor socialization effects were observed in the total episodic stress model. When parsing episodic stress, only selection effects were observed with non-interpersonal episodic stress. Neither selection nor socialization effects were observed with interpersonal episodic stress. With further parsing of episodic non-interpersonal stress, both socialization and selection effects were evident in the dependent model. Our finding suggests that neuroticism contributes to episodic stressors that are non-interpersonal in nature during adolescence—including personal injuries, illnesses, and academic failures—and that such events resulting from an adolescent's direct contribution in turn contribute to neuroticism. As such, our results align with the proposal that neuroticism is particularly affected by experiences that likely impact one's identity and status as well as major undesirable events (Jeronimus et al., 2015). The selection effect finding in the independent non-interpersonal stress model was only evident at one-timepoint, and therefore should be replicated in future studies before interpreting.

Social functioning and longstanding deficits in the domain are inherently tied to life stressors, suggesting that one's own social functioning may play a role in the stress one experiences. In our comprehensive approach, we disentangled interpersonal and non-interpersonal stress within stress models as well as separated dependent from independent in episodic stress models. Given this separation, the role of social functioning may play a role in the interpersonal and dependent stress types but have less of an effect in other stress types.

The absence of selection or socialization effects of interpersonal episodic stress was unexpected. However, focusing on adolescents could have precluded major interpersonal life events that have been found to have considerable impact on personality such as separation and divorce (Specht et al., 2011). Our adolescents had a fairly narrow range of "typical"

events (e.g., academic failures) and very few high impact events. The narrow range may have contributed to small effects of episodic stress observed. It is likely effects would be amplified with more impactful events. Therefore, results may not generalize.

This is the first systematic investigation of both selection and socialization effects with different stress types. Existing work overlooks specific stress types and fails to consider both selection and the socialization effects (Jeronimus et al., 2015). The current study also examines these effects during adolescence, a key developmental period. Furthermore, our measure of neuroticism is a general neuroticism factor—rather than a score derived from a single self-report—and therefore captures a broader vulnerability construct. Our semi-structured life stress interview conducted by independent raters with consensus ratings overcomes the risk of self-report stress measures being obscured by a respondent's personality and lack of objectivity (Hammen, 2018). Lastly, our data is longitudinal with repeated measures, enabling observation of temporal order of experiences (Jeronimus et al., 2015).

This study is not without limitations. First, this study lacks a lifespan perspective (Jeronimus et al., 2015). It is crucial for future work to explore selection and socialization effects across more extended developmental periods to further examine the plasticity of neuroticism over time in reaction to stress. A longer follow-up period may also allow detection of more robust selection effects, as there is greater opportunity for exposure to life stress. Separately, the narrow age range of our sample limits generalizability of the results to other age groups. Further, traditional CLPM does not differentiate between-from within-person variance, which limits the interpretation of significant prospective paths as the lagged parameters do not represent within-person relationships over time (Hamaker et al., 2015). Future work could use alternative statistical approaches, such as random intercept CLPM. However, this approach would remove the stable variance associated with the stable trait of neuroticism (E. L. Hamaker, personal communication, August 18, 2019; Williams et al., in press). Future work interested primarily in within-person change, as opposed to lagged effects between constructs between specific adjacent timepoints, may also consider traditional multilevel modeling approaches.

Future research should also examine the lasting nature of the effects that stress types have on neuroticism. Long lasting effects may indicate a set-point change—as opposed to a short-term fluctuation—in neuroticism (Jeronimus et al., 2013). Separately, it remains unclear why there was a discrepancy between chronic and episodic models in terms of the observed selection and socialization effects in relation to stress content (interpersonal versus non-interpersonal). It could be a matter of the nature of assessing chronic stressors (i.e., prolonged impact) versus episodic stressors (i.e., occurrence vs. non-occurrence) that led to the discrepancy. However, this reasoning remains speculative and should be the topic of future research.

Our findings suggest that neuroticism is an important target for adolescents in order to curtail detrimental effects of stress. First, because psychopathology is linked to neuroticism and life stress independently (Mineka et al., 2020), identification of specific stress types that can increase neuroticism, and vice versa, could inform intervention and prevention

strategies. Given the observed socialization effects, adolescents could benefit from interventions such as positive reappraisal of stress, which would teach them to interpret stress positively and attach meaning in terms of personal growth (Helgeson et al., 2006). To address observed selection effects, treatments such as mindfulness-based and cognitive behavioral therapies, could target elevated neuroticism (Armstrong & Rimes, 2016; Sauer-Zavala et al., 2020), which may decrease exposure to further chronic interpersonal stress or dependent non-interpersonal stressful life events. The period before adulthood, when personality traits grow more stable (Roberts et al., 2006), is optimal for targeting personality to maximize chances of adaptive outcomes.

Over three years, we observed that neuroticism increases interpersonal chronic stress and non-interpersonal stressful events and that dependent non-interpersonal stressful events and chronic stress increases neuroticism. Our results support the notion that selection and socialization effects are evident but depend on the stress type examined. Thus, our systematic investigation clarifies specific stress types that shape one's personality and vice versa. Life experience and personality therefore cannot be considered separately.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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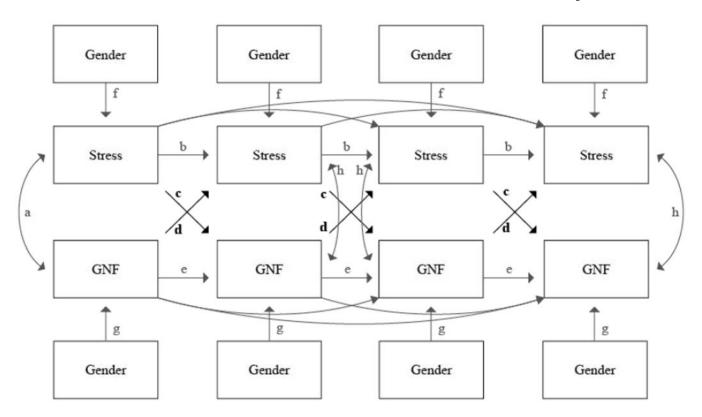
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Highlights

- Selection effects are evident between neuroticism and interpersonal chronic stress, total non-interpersonal episodic stress, and dependent non-interpersonal episodic stress.

- Socialization effects are evident between total chronic stress, interpersonal chronic stress, non-interpersonal chronic stress, and dependent non-interpersonal episodic stress and neuroticism.

- Life stress and neuroticism do not operate independently and can serve as key intervention targets for adolescents.

Figure 1. Selection and Socialization Effects: Cross-Lagged Panel Model

Note. The structural equation model of stress and the General Neuroticism Factor (GNF) included annual assessments from T1 to T7 which allowed precise estimation of the paths of interest in the present study for the developmental transition from late adolescence to early adulthood. Cross-sectional correlations and residual correlations between stress and the GNF (at baseline) or their residuals (1-Year Follow-Up, 2-Year Follow-Up, 3-Year Follow-Up) are represented by paths a and h, respectively. Paths b and e represent autoregressive paths relevant to stress and the GNF. Path c represents the socialization effect from stress to the GNF. Path d represents the selection effect from the GNF to stress. These paths are bolded because they are the focal paths of study. Paths f and g represent the gender effects for stress and the GNF. In the independent non-interpersonal episodic stress model, path c was freely estimated.

Table 1

Model Fit Information including Results from Chi-square Test of Model Fit and Key Indices for Selected Models

	χ^2	df	*p*	CFI	SRMR	RMSEA
Chronic Stress	29.64	22	.13	.99	.03	.02
Chronic Interpersonal Stress	20.63	22	.54	1.00	.03	.00
Chronic Non-Interpersonal Stress	34.31	22	.05	.98	.03	.03
Episodic Stress	17.60	22	.73	1.00	.03	.00
Episodic Interpersonal Stress	19.56	22	.61	1.00	.03	.00
Dependent Episodic Interpersonal Stress	14.89	22	.87	1.00	.03	.00
Independent Episodic Interpersonal Stress	21.53	22	.49	1.00	.03	.00
Episodic Non-Interpersonal Stress	17.07	22	.76	1.00	.03	.00
Dependent Episodic Non-Interpersonal Stress	24.92	22	.30	.97	.03	.02
Independent Episodic Non-Interpersonal Stress	20.66	20	.42	1.00	.03	.01

Note. CFI = comparative fit index; SRMR = standardized root mean square residual; RMSEA = root mean square error of approximation. In the independent episodic non-interpersonal stress model, results are displayed for the model in which the selection path (neuroticism to stress) is freely estimated and remaining constraints present in other models.

Table 2

Descriptive Statistics of Stress Variables

Measure	Baseline	1-Year Follow-Up	2-Year Follow-Up	3-Year Follow-Up
Chronic (total)	2.28 ± 0.36 (1.45 - 3.50)	2.21 ± 0.33 (1.45 - 3.45)	2.26 ± 0.34 (1.50 - 3.75)	2.24 ± 0.33 (1.55 - 3.50)
Chronic, interpersonal	2.39 ± 0.47 (1.25 - 4.00)	2.31 ± 0.44 (1.25 - 4.13)	2.31 ± 0.46 (1.50 - 4.25)	2.36 ± 0.46 (1.38 - 4.13)
Chronic, non-interpersonal	2.20 ± 0.38 (1.33 - 3.50)	2.14 ± 0.34 (1.42 - 3.42)	2.23 ± 0.35 (1.50 - 4.00)	2.22 ± 0.37 (1.50 - 3.83)
Episodic (total)	0.64 ± 0.97 (0 - 6)	0.60 ± 0.92 (0 - 6)	0.79 ± 1.16 (0 - 8)	0.86 ± 1.07 (0 - 6)
Episodic, interpersonal	0.41 ± 0.77 (0 - 5)	0.38 ± 0.70 (0 - 5)	0.42 ± 0.74 (0 - 4)	0.48 ± 0.80 (0 - 4)
Dependent episodic, interpersonal	0.16 ± 0.47 (0 - 3)	0.16 ± 0.50 (0 - 4)	0.21 ± 0.48 (0 - 2)	0.21 ± 0.51 (0 - 3)
Independent episodic, interpersonal	0.22 ± 0.48 (0 - 3)	1.73 ± 1.41 (0 - 9)	2.03 ± 1.43 (0 - 7)	1.41 ± 1.45 (0 - 7)
Episodic, non-interpersonal	0.23 ± 0.51 (0 - 3)	0.22 ± 0.56 (0 - 5)	0.28 ± 0.63 (0 - 4)	0.32 ± 0.61 (0 - 3)
Dependent episodic, non-interpersonal	0.08 ± 0.29 (0 - 2)	0.07 ± 0.28 (0 - 2)	0.09 ± 0.35 (0 - 3)	0.16 ± 0.42 (0 - 3)
Independent episodic, non-interpersonal	0.14 ± 0.38 (0 - 2)	1.46 ± 1.40 (0 - 8)	1.23 ± 1.25 (0 - 8)	1.35 ± 1.41 (0 - 8)

Note. M ± SD (range).

Table 3

Frequencies and Descriptions of Commonly Endorsed Stressful Life Events per Timepoint

Baseline	1-Year Follow-Up	2-Year Follow-Up	3-Year Follow-Up
Event (%)	Event (%)	Event (%)	Event (%)
Interpersonal			
End dating relationship (6.9)	End dating relationship (9.2)	Move out of home for first time (14.7)	End dating relationship (9.4)
Serious argument or problem with friend (6.6)	Physical or emotional illness, injury or accident to close family member, friend, romantic partner (not leading to death) (6.3)	End dating relationship (9.1)	Physical or emotional illness, injury or accident to close family member, friend, romantic partner (not leading to death) (7.8)
Physical or emotional illness, injury or accident to close family member (6.3)	Serious argument or problem with friend (5.4)	Physical or emotional illness, injury or accident to close family member, friend, romantic partner (not leading to death) (6.2)	Serious argument or problem with friend (5.2)
Non-Interpersonal			
Generic other (13.8)	Generic other (11.5)	Generic other (7.3)	Generic other (9.3)
Traffic accident (4.2)	Traffic accident (5.5)	Minor personal physical illness (4.5)	Minor personal physical illness, injury or accident (5.6)
Minor personal physical illness, injury or accident (3.2)	Minor personal physical illness, injury or accident (4.1)	Injury or accident, traffic accident (4.4)	Important academic failure (3.6)

Note. % refers to the frequency of the event type out of all reported events.

Table 4

Summary of Study Findings

	Selection Effect (N → Increase in Stress)	Socialization Effect (Stress → Increase in N)
Chronic Stress		
Chronic Interpersonal Stress		
Chronic Non-Interpersonal Stress		
Episodic Stress		
Episodic Interpersonal Stress		
Dependent Episodic Interpersonal Stress		
Independent Episodic Interpersonal Stress		
Episodic Non-Interpersonal Stress		
Dependent Episodic Non-Interpersonal Stress		
Independent Episodic Non-Interpersonal Stress	*	

Note.

*
selection effect was evident at one timepoint (neuroticism at 2-year follow-up predicting stress at 3-year follow-up) in this model.

Table 5

Selection and Socialization Path Results and Variable R^2 Values from Cross-lagged Panel Models

	Predictor	Outcome	Est.	*SE*	*z*	*p*	Holm threshold
Chronic Stress							
Selection	GNF(t-1)	CS(t)	0.04	0.02	1.76	.08	.05
		R^2	.35				
Socialization	CS(t-1)	GNF(t)	0.11	0.03	3.50	.001*	.025
		R^2	.09				
Chronic Interpersonal Stress							
Selection	GNF(t-1)	CIS(t)	0.06	0.02	2.83	.005*	.05
		R^2	.27				
Socialization	CIS(t-1)	GNF(t)	0.10	0.03	3.36	.001*	.025
		R^2	.08				
Chronic Non-Interpersonal Stress							
Selection	GNF(t-1)	CNIS(t)	0.02	0.02	1.06	.29	.05
		R^2	.35				
Socialization	CNIS(t-1)	GNF(t)	0.09	0.03	2.91	.004*	.025
		R^2	.08				
Episodic Stress							
Selection	GNF(t-1)	ES(t)	0.04	0.03	1.63	.103	.025
		R^2	.09				
Socialization	ES(t-1)	GNF(t)	0.05	0.03	1.62	.104	.05
		R^2	.08				
Episodic Interpersonal Stress							
Selection	GNF(t-1)	EIS(t)	0.01	0.03	0.54	.59	.05
		R^2	.08				
Socialization	EIS(t-1)	GNF(t)	0.03	0.03	0.96	.33	.025
		R^2	.08				
Dependent Episodic Interpersonal Stress							
Selection	GNF(t-1)	dEIS(t)	0.01	0.03	0.36	.72	.05
		R^2	.06				
Socialization	dEIS(t-1)	GNF(t)	0.04	0.03	1.61	.11	.025
		R^2	.08				
Independent Episodic Interpersonal Stress							

	Predictor	Outcome	Est.	*SE*	*z*	*p*	Holm threshold
Selection	GNF[(t-1)]	iEIS[(t)]	0.05	0.028	1.71	.09	.025
		R^2	.04				
Socialization	iEIS[(t-1)]	GNF[(t)]	−0.04	0.04	−1.04	.30	.05
		R^2	.08				
Episodic Non-Interpersonal Stress							
Selection	GNF[(t-1)]	ENIS[(t)]	0.08	0.03	2.41	.016*	.025
		R^2	.02				
Socialization	ENIS[(t-1)]	GNF[(t)]	0.07	0.03	2.40	.017*	.05
		R^2	.09				
Dependent Episodic Non-Interpersonal Stress							
Selection	GNF[(t-1)]	dENIS[(t)]	0.08	0.03	2.59	.01*	.025
		R^2	.02				
Socialization	dENIS[(t-1)]	GNF[(t)]	0.06	0.02	2.27	.02*	.05
		R^2	.08				
Independent Episodic Non-Interpersonal Stress							
Selection	GNF[(0)]	iENIS[(1)]	−0.01	0.06	−0.08	.93	.05
	GNF[(1)]	iENIS[(2)]	0.07	0.04	1.59	.11	.017
	GNF[(2)]	iENIS[(3)]	0.25	0.06	4.06	<.001*	.013
		R^2	0.05				
Socialization	iENIS[(t-1)]	GNF[(t)]	0.01	0.03	0.33	.74	.025
		R^2	.08				

Note. GNF = general neuroticism factor; CS = total chronic stress; CIS = chronic interpersonal stress; CNIS = chronic non-interpersonal stress; ES = total episodic stress; EIS = episodic interpersonal stress; ENIS = episodic non-interpersonal stress; dEIS = dependent episodic interpersonal stress; iEIS = independent episodic interpersonal stress; dENIS = dependent episodic non-interpersonal stress; iENIS = independent episodic non-interpersonal stress. Superscripts indicate timepoint: t indicates a given timepoint; 0 indicates baseline timepoint; 1 indicates 1-year follow-up timepoint, 2 indicates 2-year follow-up timepoint, 3 indicates 3-year follow-up timepoint.

*
denotes significance at Holm threshold.