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The Vicious Cycle of Psychopathology and Stressful Life Events: A Meta-Analytic Review Testing the Stress Generation Model

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Stress generation theory initially posited that depression elevates risk for some stressful events (i.e., dependent events) but not others (i.e., independent events). This preregistered meta-analytic review examined whether stress generation occurs transdiagnostically by examining 95 longitudinal studies with 38,228 participants (537 total effect sizes) from over 30 years of research. Our multilevel meta-analyses found evidence of stress generation across a broad range of psychopathology, as evidenced by significantly larger prospective effects for dependent (overall psychopathology: $r = .23$) than independent (overall psychopathology: $r = .10$) stress. We also identified unique patterns of effects across specific types of psychopathology. For example, effects were larger for depression than anxiety. Furthermore, effects were sometimes larger in studies with younger participants, shorter time lags between assessments, checklist measures of stress, and for interpersonal stressors. Finally, a multilevel meta-analytic structural equation model suggested that dependent stress exacerbates psychopathology symptoms over time ($\beta = .04$), possibly contributing to chronicity. Interventions targeting the prevention of stress generation may mitigate chronic psychopathology. Conclusions of this study are limited by the predominance of depression effect sizes in the literature and our review of only English language articles. On the other hand, the findings are strengthened by rigorous inclusion criteria, lack of publication bias, and absence of moderating effects by publication year. The latter underscores the replicability of the stress generation effect over the last 30 years. Taken together, the review provides robust evidence that stress generation is a cross-diagnostic phenomenon that contributes to a vicious cycle of increasing stress and psychopathology.

Public Significance Statement

Stress generation theory originally showed that people with depression can create their own stress. This meta-analysis indicates that stress generation is a cross-diagnostic phenomenon that contributes to the chronicity of symptoms of psychopathology. Findings imply that interventions aimed at preventing stress generation may mitigate chronic psychopathology. Stress generation also varied in nuanced ways across various types of psychopathology, thereby indicating for whom and under what conditions stress generation is most prominent. Importantly, a lack of publication bias and absence of moderating effects by publication year and by most demographic variables underscores the replicability of the stress generation effect over time and its universality across groups.

Keywords: psychopathology, mental disorders, stress generation, life events, chronicity

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Mental disorders represent a serious public health and economic issue. Psychologists, psychiatrists, education, and social work scholars have documented the significant personal costs that psychopathology confers, including lower marital satisfaction, educational and occupational attainment, and quality of life, as well as greater disability and higher mortality rates (e.g., Tanner et al., 2019; E. R. Walker et al., 2015; Whisman et al., 2004). Economists have documented the significant direct and indirect costs of psychopathology that result from lost productivity and health care utilization, with a projected global cost of $6 trillion per year by 2030 (The Lancet Global Health, 2020). The considerable personal and economic burden of psychopathology is due in large part to its high chronicity. Mental disorders are persistent, highly recurrent, and have low rates of remission (Burcusa & Iacono, 2007; Scholten et al., 2013; Vuorilehto et al., 2005). Thus, a more nuanced understanding of the mechanisms that contribute to the course and maintenance of mental disorders will have far-reaching conceptual and applied implications.

Stress generation theory represents a promising line of inquiry for understanding the high chronicity and recurrence of psychopathology. Originally formulated to understand recurrence in major depressive disorder (Hammen, 1991), stress generation theory postulates that individuals with elevated psychopathology contribute more than those with lower psychopathology to the greater occurrence of self-generated or "dependent" stressors. In contrast, the original stress generation hypothesis posits that individuals do not differ in exposure to fateful or "independent" stressors, which are outside of the individual's control. Furthermore, given that stress is a robust proximal predictor of mental disorder symptoms and diagnoses (Harkness, 2023), the generation of greater dependent stress may have key implications for maintaining or exacerbating symptoms and ultimately producing a vicious cycle of increasing stress and psychopathology. Thus, a transactional, mediating effect of generated stress on increases in psychopathology over time may be central to understanding the course of mental disorders, including the relapse, recurrence, and chronicity of symptoms and episodes.

Although stress generation theory emerged from the depression literature, recent research has expanded it to apply it to diverse mental disorders. For example, stress generation effects have been documented in bipolar spectrum disorders (e.g., Bender et al., 2010), anxiety disorders (e.g., Uliaszek et al., 2012), personality disorders (e.g., Conway et al., 2018), and externalizing disorders (e.g., Rudolph et al., 2000). However, null and mixed findings have also been reported (e.g., Joiner et al., 2005), making it difficult to draw sound conclusions on the universality of stress generation. Previous narrative reviews and chapters on stress generation have offered discussions of the stress generation literature as it specifically applies to depression (Hammen, 2005, 2006, 2020; Hammen & Shih, 2008; Harkness & Washburn, 2016; Liu, 2013; Liu & Alloy, 2010), anxiety (Meyer & Curry, 2017), or genetic variables (Bahji et al., 2021). Although a subset of these reviews provided brief discussions on whether or not stress generation may be specific to depression (Hammen, 2020; Hammen & Shih, 2008; Liu & Alloy, 2010), no review to date has focused on the broader question of whether stress generation is a transdiagnostic phenomenon. Crucially, no comprehensive meta-analysis has attempted to quantify the magnitude or pattern of stress generation effects across different forms of psychopathology or to investigate among whom and under what conditions stress generation is most pronounced across various disorders. This represents a significant gap in the literature that has impeded advancements in models of psychopathology and intervention.

The current preregistered meta-analytic review is the first to provide a comprehensive synthesis of over 30 years of research on psychopathology and stress generation. A central goal of this study was to quantify this vast literature to determine whether stress generation is a transdiagnostic phenomenon that occurs across types of psychopathology. Moreover, the present study meta-analytically tested, for the first time, (a) whether stress generation effects differ across types of psychopathology, (b) boundaries of stress generation in terms of the moderators that determine for whom and under what conditions stress generation effects are most prominent, as well as (c) to what extent stress generation accounts for the chronicity of mental disorders.

Stress Generation Theory

In contrast to earlier stress exposure (Grant et al., 2014) and diathesis–stress (Ingram & Luxton, 2005; Monroe & Simons, 1991) models that focus on the impacts of life stress in increasing susceptibility to psychopathology, the stress generation model posits that individuals play an active role in causing negative life events. Those stressors that occur at least in part as a result of characteristics or behaviors of the individual are referred to as *dependent* stressors, and include events such as getting into a serious argument or being fired from a job due to poor performance. *Independent* stressors, in contrast, refer to fateful stressors that occur irrespective of the individual's actions and include events such as the death of a relative due to old age. The first study to propose and test the stress generation hypothesis (Hammen, 1991) followed women with recurrent major depression, bipolar disorder, or chronic medical illness and healthy women for 1 year. At follow-up, women with depression reported more dependent stressful life events since baseline than did women in the other groups. Notably, this effect was particularly large for dependent interpersonal stressors. In direct contrast, there were no group differences in independent stressors. Given the pathogenicity of stress, Hammen theorized that the stress generation phenomenon may be a mechanism underlying recurrence in depression. Consistent with this proposition, dependent events are more strongly predictive of mental disorders, such as depression and anxiety, than are independent events (Broeren et al., 2014; Hammen et al., 1985; Kendler et al., 1999), highlighting the potential explanatory importance of stress generation for psychopathology maintenance and recurrence. Thus, whereas testing stress generation theory involves identifying and comparing the longitudinal associations between psychopathology and later dependent and independent stress, consistent with Hammen (1991), a key

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extension of this work includes investigating the role of generated stress in promoting or prolonging psychopathology over time.

The central proposition of stress generation theory—that individuals actively influence their environments and the corresponding stressors they experience—is relevant to a myriad of research areas beyond clinical science. Personality and social psychologists emphasize person–environment transactions in how individuals actively select, alter, and elicit reactions from their physical and social environments (Buss, 1987; Diener et al., 1984; Mischel, 1973). For example, reciprocal determinism (Bandura, 1982, 1986) suggests that individuals' behaviors influence and are influenced by both their personal characteristics and environmental factors. Developmental psychologists similarly examine the role of individuals in contributing to stressful environments over their lifespan (Champion et al., 1995; Rutter, 1986; Rutter et al., 1997). Furthermore, behavioral geneticists investigate active gene–environment correlations, which describe the self-selection of individuals into environments that align with their genetic proclivities (Kendler et al., 1999; Plomin & Bergeman, 1991; Plomin et al., 1977; Scarr & McCartney, 1983), as well as evocative gene–environment correlations, in which genetically mediated traits provoke environmental responses (Jaffee & Price, 2007; Wilkinson et al., 2013).

Stress generation is a specific example of action theory, which explains human behavior in terms of goal-directed, intentional actions by individuals in the context of the current environment. Action theory originated as an area of philosophy and is now comprised of a body of theories spanning psychology, sociology, and neuroscience (Frese & Sabini, 2021; Valach et al., 2002). Importantly, despite overlap with other action theory-based models, stress generation theory is unique in its emphasis on differentiating dependent and independent stressors. This has allowed for the formulation of testable hypotheses that meaningfully distinguish the active generation of stress from overall stress exposure—a distinction that has been crucial for shaping how researchers conceptualize stress and its role in disease and disorder (e.g., Hankin, & Abramson, 2001).

Testing Stress Generation Theory

There are several key points of clarification regarding stress generation theory and its accurate assessment. First, it is important to note that, whereas the stress generation hypothesis proposes that some individuals generate more stress than others in terms of both the number and severity of stressors, all individuals generate stressful life events. Thus, investigations of stress generation examine differences across individuals, based on factors such as diagnostic status or symptomatology, in the *degree* of dependent stress that is generated. That said, it is important to acknowledge that some may interpret stress generation theory as "blaming the victim." However, assigning agency to the generation of dependent stress does not also assign blame. Rather, it acknowledges that the individual's personal characteristics or behaviors contributed to the likelihood of a dependent stressor occurring. It is critical to understand the individual's role in contributing to greater dependent stress and psychopathology, particularly given that ascribing agency emphasizes the individual's capacity to influence their exposure to stressors. As with the literature on revictimization among trauma survivors (e.g., H. E. Walker et al., 2019), elucidating the mechanisms that lead to stressful life events is critical for informing our models of risk, prevention, and intervention.

Second, a true test of stress generation requires an assessment of the degree of independent stress individuals experience, which acts as a control comparison. An assessment of the generation of dependent stress alone can, therefore, only be considered a preliminary investigation of the stress generation hypothesis. It is important to note, however, that although stress generation theory presumes that independent life events occur at a similar rate across individuals (Hammen, 1991), some studies have reported significant prospective associations of psychopathology with independent stress (e.g., Harkness & Stewart, 2009; Shapero et al., 2013). Considering that independent events are largely fateful, these findings may be reflective of the more challenging or entrapping environments that people with psychopathology may be more likely to live in compared to their healthy counterparts (Davies & Sturge-Apple, 2014; C. Hammen, 2003). Given that greater independent stress may be elevated among some samples of individuals with psychopathology for reasons other than stress generation, it is valuable to assess differences in the magnitude of the association of psychopathology with dependent versus independent stress. Using this approach, larger effects detected for dependent as compared to independent stress are considered to be indicative of stress generation. To provide a robust test of stress generation theory, the present study examined the relative strength of associations of psychopathology with dependent and independent stress. Importantly, although many individual studies simply examine associations between psychopathology and *dependent stress*, we can most robustly identify stress generation effects only when *effect sizes predicting dependent stress are significantly stronger than those predicting independent stress*, a question best addressed using moderation in multilevel meta-analysis (MLMA).

In addition to the assessment of independent stressors, there are several key differences in how studies were conducted to test the stress generation hypothesis that warrant careful consideration. First, some studies have used a cross-sectional or retrospective design. Cross-sectional studies that assess the concurrent association of psychopathology with dependent stress cannot be used to draw conclusions about stress generation given that they do not establish the temporal precedence of psychopathology. Similarly, retrospective studies frequently probe overlapping periods of time with reference to the occurrence of psychopathology versus life events, and memory biases limit reporting accuracy in studies that do attempt to assess variables over distinct intervals (Crosswell & Lockwood, 2020). It is also worth noting that distinguishing between dependent and independent stress is a complex endeavor, particularly given that the boundary between these constructs is not always well-defined. However, some studies use participant-rated determinations of the dependence of life events (e.g., Boecking & Barnhofer, 2014). This approach has serious drawbacks—participants may have a limited understanding or insight into the extent to which they contributed to the occurrence of an event. Further, individuals may over- or underestimate their contribution to negative events as a result of underlying cognitive biases (Krackow & Rudolph, 2008), thereby confounding predictor and outcome variables. Finally, some studies have assessed stress generation with reference to chronic stressors. It is challenging to determine the dependence of stressors that are more chronic in nature given a myriad of factors that contribute to these stressors over time. For example, although chronically occurring stressors may be assessed prospectively, given the longer time scale over which they occur, proximal factors directly contributing to these stressors may have transpired outside of the time

period under investigation and thus may be conflated with those factors causing the index episode of psychopathology. Chronic stress, therefore, may require different explanatory models (Hammen, 2006).

Overcoming these limitations, more rigorous studies of stress generation, therefore, (a) use longitudinal designs to assess the association of psychopathology with prospectively collected life event information, (b) use investigator-rated measures of stressor dependence, and (c) assess multiple episodic stressors. By assessing psychopathology and life stress over clearly nonoverlapping time periods, these studies enable a strong determination of the temporal precedence of psychopathology. They also employ a more valid assessment of stressor dependence that is not conflated with participants' cognitive biases. Finally, by examining acute stressors with a clear onset and offset during the follow-up period, the participants' role in contributing to the occurrence of events can be determined. Although other methodological factors certainly may influence the magnitude of stress generation effects, taken together, these three key methodological features are necessary for ensuring a valid and stringent test of stress generation theory. These methodological factors were therefore used to identify studies for inclusion in the current meta-analysis.

Empirical Research on Stress Generation in Psychopathology

Psychopathology as a Predictor of Stress Generation

Hammen's (1991) finding that depression is prospectively associated with greater dependent stress has now been replicated across a wide range of populations. Studies of samples of individuals with depression have found evidence of stress generation in men (e.g., Cui & Vaillant, 1997), ethnically diverse samples (e.g., Daley et al., 2006), and samples from around the world (e.g., China: Auerbach et al., 2010; Israel: Shahar & Priel, 2003; Netherlands: Maciejewski et al., 2021; Turkey: Tuna, 2020). The depression-dependent stress link has also been reported in individuals across the lifespan, including among children (e.g., Chan et al., 2014; Flynn & Rudolph, 2011), adolescents (e.g., Harkness, & Stewart, 2009; Starr et al., 2013; Wetter & Hankin, 2009), and adults (e.g., Chun et al., 2004; Daley et al., 1997). Furthermore, stress generation has been documented among individuals with current depressive diagnoses (e.g., Cummings et al., 2010; Uliaszek et al., 2012), remitted depressive diagnoses (e.g., Hammen, 1991; Shih & Eberhart, 2008), and lifetime depression (Conway et al., 2012; Safford et al., 2007). That stress generation has been documented outside of periods of acute depression, including in Hammen's (1991) original study, suggests that the depression syndrome itself may not directly cause dependent stress. Symptoms also appear to play an important role—Shih and Eberhart (2010) documented that current depressive symptoms mediated the association of remitted depression diagnoses with subsequent dependent stress. Moreover, elevated depressive symptoms across both clinical (e.g., Liu & Spirito, 2019) and nonclinical samples (e.g., Jenness et al., 2019; Liu, Alloy, et al., 2014) are predictive of stress generation. Taken together, existing research provides robust evidence for stress generation in depression.

Borne out of initial studies showing that the stress generation effect in depression is augmented by the presence of other disorders (Connolly et al., 2010; Daley et al., 1997; Harkness & Luther, 2001; Rudolph et al., 2000), more recent research has documented evidence for stress generation in diverse forms of psychopathology. Stress generation has been linked to other mood disorders (bipolar spectrum disorders and symptoms: e.g., Bender et al., 2010; Molz et al., 2013) and personality disorder pathology (Conway et al., 2018; Daley et al., 1998; Powers et al., 2013), as well as internalizing psychopathology, such as general internalizing symptoms (e.g., Jeronimus et al., 2017; Riskind et al., 2013), and anxiety symptoms and disorders (e.g., Judah et al., 2013; Maniates et al., 2018; Uliaszek et al., 2012). Externalizing psychopathology has also been implicated in stress generation, and effects have been documented for general externalizing symptoms (e.g., Little & Garber, 2005), substance use (e.g., Schmied et al., 2016), attention-deficit hyperactivity disorder (e.g., Daviss & Diler, 2012; Rychik et al., 2021), and disruptive symptoms and disorders (e.g., Champion et al., 1995; Conway et al., 2012; Shapero et al., 2013).

Mirroring findings for depression (e.g., Flynn & Rudolph, 2011; Harkness & Stewart, 2009), several studies have reported particularly strong effects of internalizing psychopathology on dependent interpersonal relative to noninterpersonal stress (Conway et al., 2012; Goldstein et al., 2021). Whereas dependent interpersonal stress encompasses social stressors such as arguments and conflicts, dependent noninterpersonal stress refers to stressors occurring in the domains of education, occupation, recreation, finances, the law, housing, and health, among others. Intriguingly, the inverse pattern of associations of externalizing psychopathology with dependent interpersonal and noninterpersonal stress has been documented. In a series of studies of clinic-referred and community youth, Rudolph et al. (2000) reported the expected association of depression with subsequent dependent interpersonal, and not noninterpersonal, stress. However, in direct contrast, externalizing psychopathology predicted dependent noninterpersonal, but not interpersonal, stress (Rudolph, 2008; Rudolph et al., 2000), with some evidence that boys were more likely to display this pattern of associations. Similarly, in a sample of adolescents, Conway et al. (2012) found that a transdiagnostic internalizing dimension predicted dependent interpersonal stress, whereas an externalizing dimension predicted dependent noninterpersonal stress. Diagnostic-level analyses indicated that major depression incrementally predicted dependent interpersonal life events. This finding suggests that depression is a uniquely potent predictor of interpersonal stress generation, a conclusion that is also supported by comparisons of individuals with depression to those with bipolar disorder (Hammen, 1991) and anxiety (Connolly, 2007). Overall, findings suggest that internalizing and externalizing psychopathology evince distinct effects on the generation of dependent interpersonal versus noninterpersonal stress. At the same time, diagnoses may show specificity in the strength of their effects on stress generation, such that depression may be a uniquely strong predictor of dependent interpersonal stress.

Despite compelling evidence for stress generation across diverse mental disorders, inconsistent findings have also been reported, leading to divergent conclusions as to the degree to which stress generation is specific to depression (cf. Hammen & Shih, 2008; Liu & Alloy, 2010). Several studies did not find evidence for associations of anxiety, conduct, or eating disorder pathology with dependent stress (Bodell et al., 2012; Gunthert et al., 2002; Joiner et al., 2005; Uhrlass & Gibb, 2007; Wingate & Joiner, 2004), despite reporting a stress generation effect in depression. Still others have reported null findings for stress generation in bipolar disorder (Grandin et al., 2007; Reilly-Harrington et al., 1999), types of personality pathology (Powers et al., 2013), and alcohol use (Goldstein et al., 2021).

Inconsistent findings in the literature prevent firm conclusions on the boundaries of the stress generation effect and raise important questions as to its ubiquity, pattern, and presentation across mental disorders. Importantly, these questions have not yet been examined meta-analytically. Therefore, a major aim of the present study was to quantify the stress generation effect across types of psychopathology to determine whether stress generation represents a transdiagnostic phenomenon. A second aim was to examine whether types of psychopathology evince distinct patterns of stress generation, both in terms of the magnitude of effects and the type of dependent stress (interpersonal vs. noninterpersonal) that is generated.

The Vicious Cycle of Stress Generation

Although stress generation theory was advanced to explain the recurrence of depression, a relative paucity of research has examined whether stress generation is a mechanism underlying the course of depression, and even less research has examined this question across nondepressive disorders. Yet, as previously noted, a comprehensive test of stress generation theory involves an assessment of the role of dependent stress in predicting prospective changes in psychopathology. Several longitudinal studies have documented that dependent stress is a strong predictor of depression chronicity and recurrence (Bos et al., 2007; Cole et al., 2006; Hammen et al., 2004; Maciejewski et al., 2000; Shapero et al., 2013). Critically, other studies have provided more direct evidence of stress generation's contribution to maintaining psychopathology by documenting transactional associations between stress and symptoms of psychopathology. Specifically, these studies have shown that dependent stress mediates the stability and exacerbation of psychopathology symptoms over time (e.g., depressive symptoms: Davila et al., 1995, 1997; Meiser & Esser, 2019; Rudolph et al., 2009; internalizing symptoms: Goldstein et al., 2021; Hankin et al., 2005). However, although existing findings suggest that stress generation is a mechanism underlying homotypic continuity and chronicity, most longitudinal studies have not tested dependent stress as a mediator of prospective changes in psychopathology, even when data are available to answer this question. It therefore remains unclear to what extent initial findings for mediation are replicated across the literature and across psychopathology. Thus, another objective of the present study was to test whether generated stress maintains and/or exacerbates diverse symptoms of psychopathology over time.

Stress Generation Moderators

Because no prior study has offered a meta-analytic review of stress generation across psychopathology, synthesizing this extensive literature for the first time provides a valuable and unique opportunity to examine among whom and under what conditions stress generation effects are most robust. Therefore, an aim of the present study was to examine whether demographic factors, such as gender and age, and methodological features, including time lag between the assessment of psychopathology and stress, timeframe assessed by diagnostic measure, and stress assessment method influence the magnitude of stress generation effects.

Demographic Moderators

Gender. Several studies examining stress generation in the context of depression have reported larger effects among women and girls compared to men and boys (e.g., Calvete et al., 2013; Davila et al., 1997; Meiser & Esser, 2019; Rudolph et al., 2000; Shih et al., 2006, 2009). Given that stress generation in depression has been shown to be largely an interpersonal process (Hammen, 2020), this finding is potentially due to the greater time and emphasis women and girls place on relationships as a result of gender role socialization (Nolen-Hoeksema, 1987). This may, in turn, increase opportunities for interpersonal stress generation. However, a study on stress generation in depression has reported the opposite pattern of gender differences (e.g., Shih et al., 2009), and other studies have not found gender differences (e.g., Cole et al., 2006; Safford et al., 2007; Shih, 2006). Furthermore, gender did not moderate associations of internalizing and externalizing dimensions of psychopathology and diagnoses with independent, dependent interpersonal, and dependent noninterpersonal stress in a large sample of youth (Conway et al., 2012). Unfortunately, a substantial number of stress generation studies have not tested for gender differences. This is a major omission because differences in stress generation across girls/women and boys/men may, given the pathogenicity of dependent stress, account for some degree of observed gender disparities in the prevalence rates of some mental disorders.

Age. Another moderator that may influence the magnitude of stress generation effects is age. Developmental theories posit that as children transition into adolescence, self-regulated action, and an emphasis on peer relationships increase alongside rapid development of cognitive and social cognitive abilities, physical maturation, and expanding responsibilities (Zimmer-Gembeck & Collins, 2008). Adolescents and adults have greater autonomy and opportunities to generate stress than do children as they form intimate partner relationships and establish families, manage finances, make decisions impacting their education and careers, and so forth. Such decisions and responsibilities become more normative across adolescence and into early adulthood as individuals begin to create the psychosocial environment in which they will interact for years (C. L. Hammen & Shih, 2014). Following this transition, stress generation effects may attenuate across adulthood (e.g., Alloy et al., 2010). Although prior empirical work has documented that stress generation effects for depression increase over time among children and adolescents (Cole et al., 2006), differences in the magnitude of stress generation effects across developmental periods of childhood, adolescence, and adulthood remain largely untested.

Methodological Moderators

Several methodological differences have important theoretical implications not only for determining how study design decisions influence the relative magnitude of stress generation effects, but also for better understanding the role of psychopathology in generating stress.

Length of Follow-Up. Studies with a shorter interval of time between the assessment of psychopathology and life stress may report larger effect sizes because psychopathology is likely to be more predictive of dependent events occurring in close temporal proximity as compared to events occurring after lengthy intervals (Alloy et al., 2010). This is essentially the inverse of the finding that recent events are more predictive of the onset of a depressive episode than events occurring in the more distant past (Hammen et al., 1986). If a shorter length of follow-up is associated with larger stress generation effects, this would suggest that the symptoms or

diagnoses of mental disorders have a proximal influence on the generation of stress. In contrast, if length of follow-up is not a significant moderator, this would suggest that other, related, and possibly more enduring factors may be playing a more direct role in driving stress generation.

Diagnostic Timeframe. We investigated whether diagnostic timeframe moderated results among studies that used diagnostic measures of psychopathology. That is, we examined whether there were differences in effects based on whether current, remitted, or lifetime diagnoses were assessed.

Stress Assessment Method. There are three primary methods of measuring stress: checklist indices, contextual interview rating systems, and hybrid checklist/interview-based measures. Checklists are comprised of lists of negative life events (e.g., "major financial difficulty," "serious illness in family member") and require individuals to check off those events that occurred over a specified interval of time. Checklists are easily administered and scored and are therefore widely used (Harkness, 2008). However, these self-report measures have important limitations. When endorsing events, respondents may have idiosyncratic criteria for whether an experience "counts" as a particular stressful life event, which may differ from the investigator's operationalization (Monroe, 2008). Responses and severity ratings may also be influenced by demand characteristics (i.e., participants recognize that stress is being measured and respond in a manner consistent with their general views about stress; Uher & McGuffin, 2010) or by cognitive and memory biases. Conversely, interview-based measures are time- and labor-intensive, yet yield more precise data. Interviewers use provided probes and follow-up questions to glean important contextual information that an independent rating team later uses to score the dependence and severity of each event for each individual (Harkness, 2008, 2023). Interview-based measures distinguish the objective severity of an event from the participant's perceptions of threat by asking only about objective details associated with life events and by keeping raters blind to the clinical status and subjective reactions of the participant. Finally, studies using hybrid checklist/interview-based measures typically first administer a checklist to participants, which determines the probes to be used in the subsequent interview. Importantly, self-administered checklists are associated with more reported events (Lewinsohn et al., 2003) and inflated severity scores as compared to interview-based stress assessments (e.g., Hammen et al., 1985; McQuaid et al., 2000; Simons et al., 1993), suggesting key psychometric differences across stress assessment methods (Harkness & Monroe, 2016). Taken together, there are important methodological and psychometric differences across checklist, interview, and hybrid (combination of checklist and interview) measures of stress that may have implications on the strength of stress generation effects detected.

The Present Study

The present publicly preregistered study provides the first systematic review and meta-analysis of empirical stress generation studies spanning domains of psychological science and psychiatry. Specifically, by synthesizing the vast literature on psychopathology and stress generation, this study addresses three key theoretical research questions: (1) Does stress generation occur across psychopathology? (2) Do types of psychopathology evince distinct patterns of stress generation, both in terms of the magnitude of effects, the moderators that determine for whom and under what conditions stress generation is most pronounced, and the type of dependent stress generated? And finally, (3) Does stress generation account for the chronicity of various types of psychopathology? To conduct a rigorous test of stress generation theory, we directly compared the magnitude of effects for dependent versus independent stress, and we restricted inclusion to those studies that used a prospective longitudinal design and investigator-defined criteria for determining the dependence of multiple episodic life stressors.

We hypothesized that psychopathology would show a significantly larger prospective association with dependent versus independent stress, indicative of stress generation. We similarly expected that each type of psychopathology, including both internalizing and externalizing psychopathology, as well as more specific disorder groupings (e.g., depression, anxiety), would each evince significantly larger associations with dependent as opposed to independent stress. In terms of whether the magnitude and patterns of effects differ across psychopathology, we hypothesized that internalizing psychopathology (vs. externalizing) and depressive symptoms/disorders (vs. all other types of symptoms/disorders) would show the strongest association with dependent interpersonal stress. Conversely, we also expected that externalizing psychopathology would evince larger effects than internalizing psychopathology on dependent noninterpersonal stress. We did not advance specific hypotheses for overall dependent stress.

We also aimed to determine whether the magnitude of associations with dependent stress varied across types of psychopathology based on gender, age, length of follow-up, diagnostic timeframe, and stress measure. Specifically, we hypothesized that effects would be larger among women and girls as opposed to men and boys. We also anticipated that adolescence and early adulthood would be associated with larger effects for the generation of dependent stressors. In terms of methodological factors, we hypothesized that a longer interval between the assessment of psychopathology and life stress would be negatively associated with the magnitude of effects and that studies assessing current, compared to remitted or lifetime diagnoses, would demonstrate stronger associations with dependent stress given the greater proximal impact current syndromes may have on subsequently assessed stressors. Due to prior evidence suggesting that individuals with psychopathology overreport the occurrence and severity of stressors using self-report checklists (Lewinsohn et al., 2003; Simons et al., 1993), we expected stronger effects for checklist measures of stress compared to interview-based and hybrid checklist/interview measures. In addition to our main theory-based moderators, we also explored the effects of descriptive moderators (i.e., psychopathology measure, race, publication year, and country of data collection).

Finally, with regard to whether stress generation accounts for the chronicity of psychopathology, we hypothesized that dependent stress would mediate the longitudinal association of baseline symptoms of psychopathology with subsequent symptoms, and that this effect would be greater in magnitude than the effect for independent stress.

Method

Collaborative Science

The current meta-analysis represents an international collaboration of two research teams led by KR and AS that had independently

conducted meta-analytic reviews of the stress generation literature (Team Canada: KR, JH, HL, RN, DM, DD, and JL, and Team United States: AS, KC, RC, and LS). We became aware of each others' shared goals during the process of one team contacting authors for unpublished data, and at this time decided to merge projects. As such, two independent literature searches were conducted, with both teams making final inclusion and exclusion decisions jointly. This resulted in an exceedingly comprehensive and complete search of the literature and high coding fidelity.

Literature Search

To identify eligible studies, both teams conducted independent literature searches. Team Canada conducted their search on the PsycInfo and PubMed databases using the following search string for titles and abstracts: ("stress generation" OR "generation of stress*" OR "generated stress*" OR "dependent stress*" OR "dependent life event*" OR "dependent event*"). This search was conducted by two independent reviewers and was limited to journal articles and dissertations published since November 1991, when the first study to propose the stress generation hypothesis (Hammen, 1991) was published. Team United States searched PsycInfo, PubMed, and Web of Science databases using the following search string: (["life event*" OR stressor OR "episodic stress" OR "stressful event*" OR "negative event*"] AND [generate OR generation OR dependent] NOT oxidative). This latter search was limited to studies conducted with humans and published in English, and it also was conducted by two independent reviewers. Boolean terms were used across both literature searches since no Medical Subject Headings subheadings exist for stress generation. Furthermore, reviewers across both teams conducted a backward search by manually searching the reference sections of all included articles as well as book chapters and reviews on stress generation for additional relevant research studies. In addition, a forward search of all articles that cited Hammen's (1991) seminal article was conducted using the PsycInfo database. All titles and abstracts were initially screened for eligibility based on the inclusion and exclusion criteria described below, followed by the full article text when necessary. As shown in the Preferred Reporting Items for Systematic Reviews and Meta-analyses flow diagram in Figure 1, Team Canada identified 2,666 reports through database searching, Team United States identified 8,506 reports, and 603 reports were identified from backward or forward searching or from author contacts (see the Contacting Authors section below). The literature searches were initially completed in July 2020 and were updated in December 2021.

Inclusion and Exclusion Criteria

The aim of this meta-analysis was to provide a valid and rigorous test of stress generation theory across psychopathology by synthesizing data only from studies that (a) used a prospective longitudinal design, (b) assessed stressor dependence using investigators' determinations, and (c) examined multiple episodic stressors. We reviewed all potentially eligible reports based on the inclusion and exclusion criteria below.

Empirical Studies Based on Quantitative Data

Only empirical studies based on quantitative data were included. Narrative reviews and book chapters (e.g., Hammen, 2020) and prior domain-specific systematic literature reviews (e.g., Bahji et al., 2021) were therefore excluded. To reduce publication bias, articles in peer-reviewed journals as well as dissertations and other unpublished research were included.

Published in English

Studies were excluded if they were not published in English.

Longitudinal Study Designs

Only prospective longitudinal studies that assessed psychopathology at a date preceding the measurement of stress were included. Therefore, studies needed to include at least two waves of assessments, and studies using cross-sectional or retrospective designs were excluded. Moreover, longitudinal studies were excluded if the assessment period for stressors overlapped with the prior assessment period for psychopathology. For analyses testing whether stress generation accounts for the chronicity of psychopathology over time, studies needed to also assess psychopathology at the second wave of assessment.

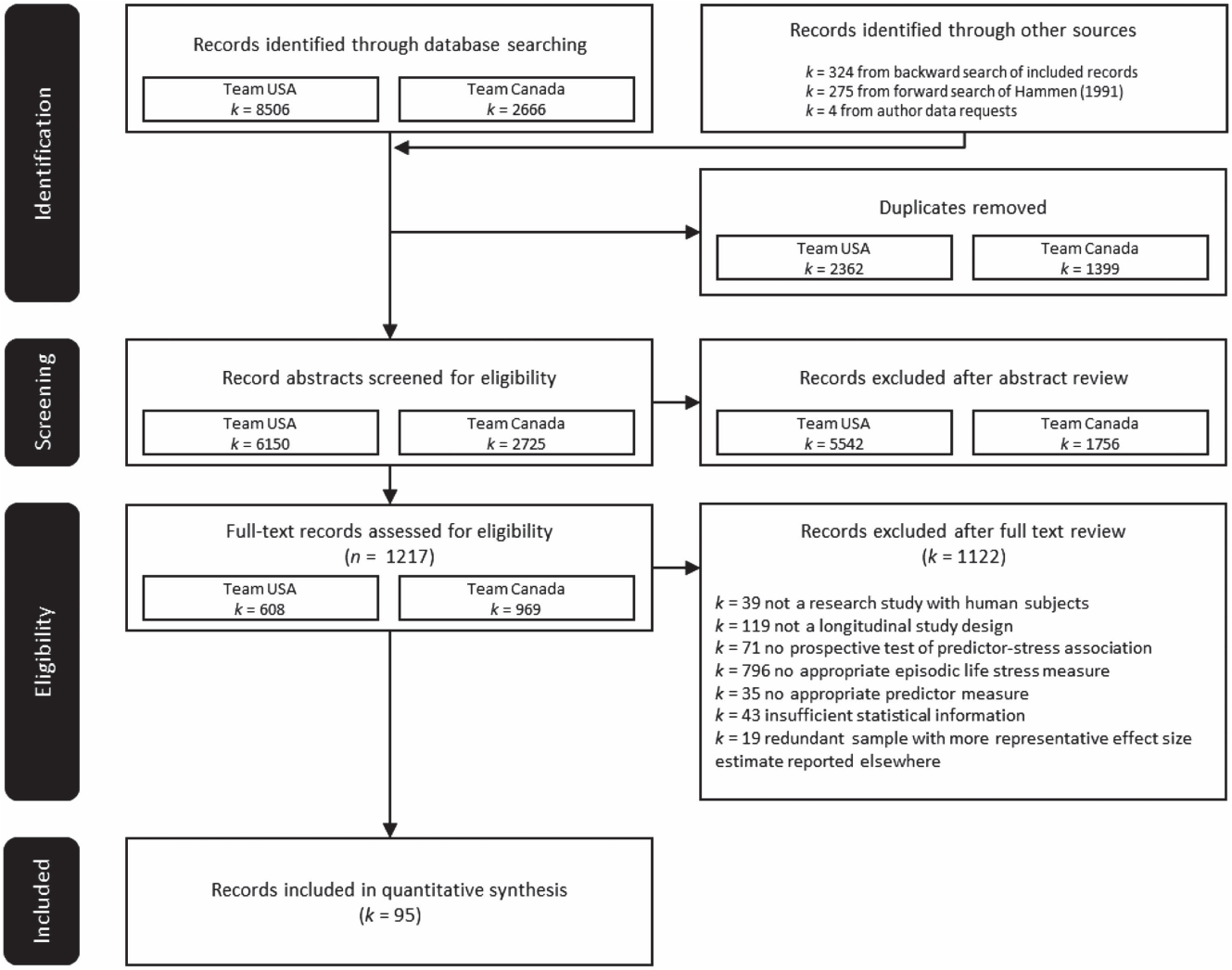
Psychopathology

Given our interest in understanding stress generation across mental disorders and types of psychopathology, studies needed to measure psychopathology using either diagnostic or symptom-based measures. For studies using diagnostic measures of psychopathology, group comparisons were required to be made between a diagnostic group and a healthy control group, such that, similar to effect sizes for continuous measures, effect sizes for diagnostic groups would also reflect the continuum of psychopathology severity. One exception was a study where group differences were based on a group with remitted major depressive disorder compared to a group with past minor/subthreshold depressive disorder (Hamilton, 2017). We included effects from this study given that they represent differences across the spectrum of psychopathology severity. Effects based on comparisons across two different diagnostic groups (e.g., depressive and anxiety disorders) were excluded as these effects would not share the same meaning with effects based on comparisons between diagnostic and control groups. Thus, they could not be meaningfully combined to compute an overall effect size. In terms of symptom-based measures, both specific (e.g., hypomanic symptoms) and general internalizing and externalizing measures were included. Furthermore, self-, parent, and teacher report as well as combined measures (e.g., combined self- and parent report) were eligible for inclusion. Only studies assessing psychopathology symptoms were included in analyses testing whether stress generation mediates the chronicity of psychopathology given that this approach represents the majority of stress generation research conducted to date, with few studies assessing changes in diagnostic status over time (for an exception, see Bos et al., 2007).

Dependent and Independent Stress

Studies were included if they distinguished dependent from independent stressors, thereby ensuring that stress generation, and not general stress exposure, was the phenomenon under investigation. Checklist measures of stress typically categorize events as uniformly dependent or independent based on a priori investigator ratings. In contrast, interviews typically provide a combination of

Figure 1
PRISMA Flow Diagram



Note. Unless otherwise specified, *k*s represent the combined count of records after accounting for duplicates between the two independent searches. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

manualized instructions and example vignettes for the interviewer or a rating team blinded to participants' clinical status to score the dependence of events based on the individuals' unique context. Moreover, studies were required to use investigator-determined, and not participant-scored, ratings of dependence. This was considered necessary for studies to provide a valid test of stress generation given (a) the complexity of the construct of dependence and the fact that participants may lack insight into the extent to which they contributed to events, and (b) because participants' ratings would potentially be influenced by cognitive biases (Krackow & Rudolph, 2008), thereby confounding the assessment of psychopathology with the assessment of dependence.

Episodic Life Stress

Studies were included if they assessed multiple episodic life stressors over time. Studies assessing life events and/or minor daily hassles were included. Studies were excluded if they assessed only a single type of life event or only assessed chronic stress (e.g., Nelson et al., 2001).

Individual-Level Stress Generation

To be included, studies had to test the stress generation hypothesis at the individual, not family, group, or community level. That is, both the independent (i.e., psychopathology) and dependent (i.e., stress) variables needed to assess the same individual. Effects for the intergenerational transmission of stress and psychopathology were therefore excluded (e.g., Hammen et al., 2004).

Necessary Statistics Provided

Studies were included if they provided the zero-order Pearson's correlation coefficient (*r*) of Time 1 psychopathology with Time 2

dependent, dependent interpersonal, dependent noninterpersonal, or independent stress. Time 2 stress assessed stressors that occurred during the interval between Times 1 and 2. For studies reporting results in other metrics, data were transformed to *r* using standard procedures (e.g., Card, 2012). For instance, in several cases, correlations were converted from group means and standard deviations comparing diagnostic and control groups (e.g., Morris et al., 2014) or from the results of significance tests (e.g., *t* tests; Cui & Vaillant, 1997). When information was not provided to compute a zero-order correlation or when studies only included multivariate associations between psychopathology and stress (e.g., partial correlations controlling for a third variable), we contacted the corresponding author to attain the unadjusted bivariate correlation (see the Contacting Authors section below). For studies to be included in analyses assessing whether stress generation accounts for the chronicity of psychopathology, studies needed to provide, or provide information to compute, three zero-order correlations: (a) Time 1 psychopathology with Time 2 dependent or independent stress (to determine the generation of stress over time), (b) Time 2 stress with Time 2 psychopathology (to determine the association of stress over the Time 1 to Time 2 interval with subsequent psychopathology), and (c) Time 1 psychopathology with Time 2 psychopathology (to determine the stability of psychopathology over time).

Data Extraction and Coding

We extracted data necessary for coding effect sizes and moderators from studies. To compute effect sizes, we recorded zero-order correlations of interest as described above, as well as the corresponding sample sizes. Effect sizes were coded for whether they assessed associations of psychopathology with overall dependent, dependent interpersonal, dependent noninterpersonal, or independent stress. Studies examining subtypes of dependent interpersonal (e.g., family conflict stress, peer stress) and noninterpersonal (e.g., academic, financial) stress were coded as examining interpersonal and noninterpersonal stress, respectively. For correlations used in our analyses examining whether stress generation mediates chronicity of psychopathology symptoms, we used a conservative approach by recording the smallest sample size across the three correlations used for each study. Furthermore, we coded for the following moderators: age, gender, type of stress measure, length of follow-up, diagnostic timeframe, stress domain, type of psychopathology measure, race, publication year, publication status, and country where data were collected.

Given that the present study represents the combined contributions of two initially independent research teams, a subset of studies were initially coded by one or both teams. Within each team, two independent raters independently coded data. After merging data sets across teams, the remaining studies were coded. Therefore, data were double-coded for 100% of included studies, and in many cases, studies were triple- or quadruple-coded. We examined interrater agreement for a randomly selected 25% of included reports, consistent with several recent meta-analyses (e.g., Berres & Erdfelder, 2021; Giletta et al., 2021; Imuta et al., 2022). Interrater agreement was high (96%). Discrepancies were discussed by the first authors until 100% agreement was reached.

To provide the most comprehensive and complete test of stress generation theory across the broadest range of psychopathology, we maximized the number of effect sizes included in the meta-analysis, as well as the sample sizes of those effect sizes. This was made possible by use of sophisticated analytic approaches that account for nonindependence of effect sizes (see the Statistical Analyses section below) and was achieved by using several strategies. First, if multiple eligible effect sizes were reported in a single study, we included them all. Second, if multiple waves of data were collected, we included all associations of the first wave of psychopathology data with all subsequent waves of stress data. Third, if multiple reports used data from the same study sample (e.g., Mater University Study of Pregnancy), we included all unique, nonredundant effects. Effects were nonredundant if they were based on different measures of psychopathology, different stress outcomes, and/or different waves of data collection. If reports provided the same effects (i.e., same psychopathology and stress measures assessed in the same sample and across the same waves of data collection), we used the effect that reported on the largest sample and that was therefore considered to be more generalizable, or for which correlations could be extracted (see also the Contacting Authors section). In cases where reports provided the identical effect for the same sample size, we prioritized published reports as compared to unpublished reports.

Contacting Authors

We contacted the corresponding author (as well as the senior author or coauthors in cases where the corresponding author's email address was not deliverable) for studies that did not present necessary information to compute an effect size. When contacting authors, we also queried for unpublished or in press data. Most authors who were unable to provide information for computing effect sizes indicated that they no longer had access to the data.

Main Moderators

We coded six main moderators: age, gender, length of follow-up, diagnostic timeframe, type of stress measure, and stress domain.

Age. We recorded mean age at baseline. In cases where only an age range was provided (e.g., Shahar & Priel, 2003), we imputed mean age as the midpoint value of the upper and lower limits. When information was not provided for age and data were collected from undergraduates in an introductory course (Joiner et al., 2005; Sahl et al., 2009), we imputed mean age as 19 years. Furthermore, we classified samples with a mean age of 12 years and below as children, between 13 and 21 as adolescent/early adult, and 22 and above as adult. Where possible, we examined age both as a continuous variable and, in separate analyses, as a categorical (child, adolescent/early adult, adult) variable in order to test for both linear and nonlinear developmental effects.

Gender. We recorded the proportion of participants that identified as girls or women for each sample.

Length of Follow-Up. Length of follow-up was recorded in months.

Diagnostic Timeframe. For studies using diagnostic measures of psychopathology, we recorded whether effect sizes were based on current, remitted, or lifetime (potentially including both current and remitted) diagnoses.

Stress Assessment Measure. We coded stress measures as checklists, interviews, or hybrid (combined checklist and interview) measures.

Stress Domain. We coded whether effect sizes for dependent stress were based on dependent interpersonal or dependent non-interpersonal stress.

Other Moderators

In addition to our main theoretical moderators, we also coded descriptive moderators, which included psychopathology measure, race, publication year, country where data were collected, and publication status.

Psychopathology Measure. We recorded whether psychopathology measures were based on diagnoses or symptom severity.

Race. Due to reporting differences across studies, proportion of non-White participants was the most reliable measure of race that could be assessed across studies. Proportion of White participants was recorded.

Publication Year. We recorded the year of publication for published journal articles and year of completion for dissertations. This variable was coded as missing for unpublished research.

Country of Data Collection. We recorded the country where data were collected. Due to a small number of effect sizes from several countries, we classified studies by whether or not they were conducted in North America.

Publication Status. We recorded whether reports were published (i.e., peer-reviewed journal articles) or unpublished (i.e., dissertations or unpublished data).

Study Quality

Study quality was assessed using the Newcastle-Ottawa Scale (NOS; Wells et al., 2011), which has been recommended by the Cochrane Collaboration (Higgins & Green, 2011). We modified this scale where necessary to apply it to the stress generation literature. Studies were rated based on NOS criteria, with possible scores ranging from 0 to 8, such that higher scores are indicative of higher study quality.

Effect Sizes Calculation

Effect sizes (Pearson correlation coefficients, r) were extracted from all identified studies. Pearson correlation coefficients of .10 are considered small, .30 moderate, and .50 are large (Cohen, 1969). To adjust for skewed standard errors, Pearson's r correlation coefficients were transformed to Fisher's Z_r correlations with corresponding standard errors computed as the square root of the variance estimates. Following analyses, Fisher's Z_r correlations were converted back to Pearson's r for ease of interpretation using standard formulas (Card, 2012).

Statistical Analyses

The majority of the included studies reported multiple eligible effect sizes. For example, numerous studies reported on several types of psychopathology, used both diagnostic and symptom-based measures, and/or reported on both dependent and independent stress outcomes. Effect sizes were not independent, thereby violating traditional meta-analytic models' assumption of independence. We, therefore, used a multilevel meta-analytic approach to account for dependencies among effect sizes (M. W. L. Cheung, 2014). Multilevel analyses allow for the inclusion of multiple dependent effect sizes from the same study by explicitly accounting for the nested structure of the data. Specifically, we estimated three-level random-effects models using restricted maximum likelihood (ML) estimation, whereby Level 1 corresponds to the random sampling variance, (i.e., the sampling variation of observed effect sizes around the true population value as a function of sample size), Level 2 reflects variance between effect sizes extracted from the same sample (i.e., within-study variance), and Level 3 captures the variance between samples (i.e., between-study variance). We examined heterogeneity by assessing the significance of the Level 2 and Level 3 variance using one-sided log-likelihood ratio tests. We also assessed how the total variance was distributed across the three levels (M. W. L. Cheung, 2014) and reported heterogeneity using the Q statistic and proportion of explained variance using I^2. An I^2 of .25, .50, and .75 reflect small, medium, and large amounts of heterogeneity, respectively (Higgins et al., 2003). We applied an a posteriori robust variance estimation (RVE) correction with small sample adjustment to correct the meta-analytic estimates of correlation coefficients and their standard errors (Fernández-Castilla et al., 2020; Tipton, 2015).

Aim 1: Does Stress Generation Occur Across Psychopathology?

To answer our first question, we first conducted an unconditional three-level random-effects model assessing the overall effect of psychopathology on both dependent and independent stress. After assessing heterogeneity, we tested for the stress generation effect by conducting a moderator analysis comparing the relative magnitude of effects for dependent versus independent stress, whereby a larger effect for dependent stress would be considered indicative of stress generation. Separate unconditional models for the associations of psychopathology with dependent and independent stress were estimated to assess the magnitude of effects for each. Finally, we assessed heterogeneity in the association of psychopathology with dependent stress. When significant heterogeneity was detected, we assessed moderation by conducting a series of conditional models to separately investigate each moderator. Dummy variables were used for all categorical moderators.

Aim 2: Do Types of Psychopathology Evince Distinct Patterns of Stress Generation?

We investigated whether different types of psychopathology display distinct stress generation patterns by examining the magnitude of effects across types of psychopathology, the moderators that determine for whom and under what conditions stress generation is most pronounced, and differences in the type of dependent stress generated. We assessed type of psychopathology in two ways: (a) internalizing versus externalizing symptoms/disorders and (b) using diagnostic and symptom categories for disorder clusters with sufficient coverage in the literature. To test whether types of psychopathology differed in the magnitude of their prospective associations with dependent stress, we examined whether type of psychopathology moderated the overall effect of psychopathology on dependent stress. We then examined each type of psychopathology separately for stress generation effects by estimating unconditional three-level random-effects models of the association of psychopathology with overall stress, and testing whether stressor dependence moderated effects. Separate unconditional models for dependent and independent stress were conducted to assess the magnitude of effects

for each. When significant heterogeneity was detected in the association of psychopathology with dependent stress, patterns of moderating effects were investigated by conducting a series of conditional models to separately investigate each moderator as a predictor. Dummy variables were used for all categorical moderators. When a categorical moderator included more than two levels, we conducted multiple models to provide comparisons between every combination of categories. Finally, we examined differences across types of psychopathology in the domain of stress generated. This was achieved by examining type of psychopathology as a moderator of the association of overall psychopathology with dependent interpersonal stress and, separately, with dependent noninterpersonal stress.

Aim 3: Does Stress Generation Account for the Chronicity of Various Types of Psychopathology?

We employed a two-stage multilevel meta-analytic structural equation modeling (MASEM) approach (Wilson et al., 2016) to test whether dependent, but not independent, stress-mediated increases in psychopathology over time. Only studies in which psychopathology symptoms were measured at one time point (e.g., Time 1), the same psychopathology symptoms were measured again at a second time point (e.g., Time 2), and a measure of stressors that occurred during the intervening period of time (i.e., stress measure administered at Time 2 that assesses Time 1–Time 2 stress) were included. It is important to note that although stress was measured at the same time as Time 2 psychopathology, and that in many cases, there would be some overlap in the time period of assessment of these two measures, this was considered preferable to only including studies with three or more waves of data collection given that (a) it would be less meaningful to assess how Time 1–Time 2 stress predicts psychopathology occurring at Time 3, thereby ignoring the most proximal stressors that occurred during the Time 2–Time 3 interval and (b) a two-wave study design represents the vast majority of stress generation research. Only studies that included all three correlations among these variables were included in analyses. In Stage 1 of the multilevel MASEM, we estimated a three-level random-effects no-intercept model using ML estimation. Pearson's r was used as the input effect size, and rs were weighted using inverse sample sizes (Wilson et al., 2016). This model provides a pooled correlation matrix. In Stage 2, the hypothesized model was fitted to the pooled correlation matrix using weighted least squares (WLS) estimation (Stolwijk et al., 2022), as required for MASEM (M. W.-L. Cheung & Chan, 2005; Jak, 2015; Stolwijk et al., 2022; Wilson et al., 2016), and the indirect effects of dependent and independent stress on the association of Time 1 with Time 2 psychopathology were estimated.

Study Quality

To examine whether findings vary based on study quality, we tested whether study quality moderated the effect of overall psychopathology, as well as each type of psychopathology that demonstrated significant heterogeneity, on dependent stress.

Publication Bias

We used several methods to assess for publication bias. We first examined publication status (published vs. unpublished) as a moderator of the overall effect of psychopathology on dependent stress. Publication bias is indicated if effect sizes systematically differ by publication status, such that larger effect sizes are reported in published studies. Second, we visually inspected funnel plots for asymmetry. In the absence of publication bias, funnel plots are symmetric in shape, such that as sample sizes increase, effect sizes converge around the true mean. Asymmetry, in turn, may suggest an absence of studies with small sample sizes and small effect sizes, such that small studies with significant findings and large studies are preferentially published. We also assessed contour-enhanced funnel plots to assess for lack of nonsignificant findings. Finally, based on recent recommendations by Rodgers and Pustejovsky (2020), we conducted two variants of Egger's regression test which handle dependent effect sizes through the use of RVE (i.e., Egger sandwich test) and MLMA (MLMA Egger test), respectively.

Transparency and Openness

We adhered to the Meta-Analysis Reporting Standards guidelines for meta-analytic reporting (Appelbaum et al., 2018). Our database Rnic et al. (2023), research materials (including coding scheme), and R code are available on the Open Science Framework (https://osf.io/wkve5/?view_only=3afb85e908114c86ac413105f253bba5). All analyses were conducted in R software Version 4.1.2. (R Core Team, 2022). Multilevel metaregression models were conducted using the metafor package (Viechtbauer, 2010), and RVE was conducted through the robumeta package (Version 2.0; Fisher et al., 2017) and the clubSandwich package (Version 0.5.5; Pustejovsky, 2022). Multilevel MASEM models were estimated using the metafor package for Stage 1 and the metasem package (Cheung, 2015) for Stage 2. This meta-analysis was publicly preregistered on the International Prospective Register of Systematic Reviews (see the Supplemental Material).

Results

Sample Description

A total of 95 studies, reported in 82 peer-reviewed articles, 12 dissertations, and 1 unpublished article were included in this meta-analytic review. Reports were published or completed between 1991 and 2021. Overall, 537 effect sizes (k) were extracted from 80 independent study samples ($M = 5.65$ effect sizes per sample, $SD = 12.79$, $Mdn = 2$). These studies were conducted in 11 different countries and across four continents, which included 63 in North America (United States: $k = 53$; Canada: $k = 9$; United States and Canada combined: $k = 1$), 11 in Europe (Germany: $k = 2$; Netherlands: $k = 4$; Norway: $k = 1$; Spain: $k = 3$; United Kingdom: $k = 1$), three in Asia (China: $k = 1$; Israel: $k = 1$; Turkey: $k = 1$), and three in Australia. In total, 38,228 participants were included in the current analyses, and sample sizes ranged from $n = 33$ to 2,858. Participants' mean age at baseline spanned developmental periods from childhood through to older adulthood and ranged from 9.17 to 61.00 years ($M = 22.57$, $SD = 12.47$, $Mdn = 19.00$). The mean proportion of girl/women participants was 62.72% ($SD = 22.53$, range = 0%–100%), and the mean proportion of White participants was 70.69% ($SD = 24.14$, range = 0%–100%). Across all waves of data collection, length of follow-up was 20.76 months on average ($SD = 24.74$, range = 0.23–240.00 months). Disorders were classified into internalizing and externalizing categories based on the Hierarchical

Taxonomy of Psychopathology (Kotov et al., 2017). As expected, depression was the most reported type of psychopathology ($k = 85$ reports). A sizable number of studies also reported on anxiety ($k = 18$) and internalizing symptoms ($k = 90$) which, together with depression, comprised our internalizing psychopathology category. In terms of externalizing psychopathology, studies reported on Cluster B personality disorders and symptoms ($k = 2$), substance-related disorders and symptoms ($k = 3$), childhood disruptive disorders and symptoms ($k = 5$), attention-deficit/hyperactivity ($k = 2$), and general externalizing symptoms ($k = 2$). In addition, a number of studies reported on other disorders and symptoms, including bipolar-related symptoms and disorders ($k = 2$), suicidality ($k = 2$), other personality symptoms and disorders ($k = 5$), and general psychopathology ($k = 1$). See Table 1 for a summary of descriptive characteristics of studies included in the current meta-analytic review. Furthermore, information on stress measures used and the psychometric properties of stressor dependence ratings are provided in the Supplemental Material.

Aim 1: Does Stress Generation Occur Across Psychopathology?

Test of Stress Generation for Overall Psychopathology

We tested the stress generation effect for overall psychopathology by examining differences in the magnitude of the prospective associations of psychopathology with dependent versus independent stress. To do this, we first needed to assess whether there was significant heterogeneity in the mean effect of psychopathology on overall stress (i.e., including both dependent and independent stress). As expected, heterogeneity was significant, $Q(536) = 4029.05$, $p < .001$; $\sigma_{(2)}^2 = .003$, $p < .001$; $\sigma_{(3)}^2 = .013$, $p < .001$; $I_{(1)}^2 = 9.96\%$, $I_{(2)}^2 = 16.33\%$, $I_{(3)}^2 = 73.03\%$, in the prospective association of psychopathology with overall stress ($r = .21$, $SE = .01$, 95% CI [.18, .23], $p < .001$). We then tested the stress generation hypothesis by examining stressor dependence as a moderator. As hypothesized, the effect of stressor dependence was significant, $\beta = .054$, $p = .008$, such that, consistent with the stress generation hypothesis, the prospective association of psychopathology with stress was significantly greater for dependent than for independent stress. Separate unconditional random-effects multilevel models indicated that the mean estimate of psychopathology on dependent stress was small-to-moderate and significant ($r = .23$, $SE = .01$, 95% CI [.20, .26], $p < .001$), whereas the effect on independent stress was small and significant ($r = .10$, $SE = .01$, 95% CI [.08, .12], $p < .001$). Figure 2A depicts all of the 371 effect sizes extracted for dependent stress, which ranged from −.12 to .58, and Figure 2B depicts the 166 effect sizes for independent stress, which ranged from −.23 to .40.

Moderators of the Association of Overall Psychopathology With Dependent Stress

After confirming a stress generation effect, we next examined moderators of the generation of dependent stress. Overall heterogeneity was significant in the model for the association of psychopathology with dependent stress, $Q(370) = 3265.13$, $p < .001$. There was also significant heterogeneity across levels, as indicated by log-likelihood ratio tests comparing the full multilevel model to two-level models with the Level 2 or Level 3 variance constrained to zero, $\sigma_{(2)}^2 = .003$, $p < .001$; $\sigma_{(3)}^2 = .013$, $p < .001$. This indicates that a three-level model best fit the data. The I^2 values indicated that 10.14% of the total variance was accounted for by Level 1 (i.e., sampling variance), 16.05% by Level 2 (i.e., within-study variability), and 73.81% by Level 3 (i.e., between-study variability). Given substantial heterogeneity across levels, we proceeded with moderator analyses.

Results for moderator analyses are shown in Table 2. There was a significant linear effect of age, $\beta = -.003$, $p = .023$, such that older age was associated with a smaller effect of psychopathology on dependent stress, see Figure 3. Furthermore, studies of adolescents/early adults ($r = .26$, $SE = .02$, 95% CI [.23, .30]) showed significantly larger effects than studies of adults ($r = .17$, $SE = .03$, 95% CI [.12, .23]), $\beta = -.09$, $p = .007$. There were no other significant age group differences ($|\beta|$s $\leq .06$, ps $\geq .127$). Together, findings indicate that the effect of psychopathology on dependent stress is substantial in adolescence and then declines with older age. There was also a significant effect of length of follow-up ($\beta = -.001$, $p = .021$), indicating that longer intervals of time between assessments were associated with smaller effects of psychopathology on dependent stress, see Figure 4. There were no significant moderator effects for gender ($\beta = .0001$, $p = .813$), diagnostic timeframe ($|\beta|$s $\leq .16$, ps $\geq .118$), stress assessment method ($|\beta|$s $\leq .120$, ps $\geq .057$), or stress domain ($\beta = -.02$, $p = .348$). In terms of descriptive moderators, there was a significant effect of psychopathology measure ($\beta = -.07$, $p = .014$), whereby symptom-based measures demonstrated larger effects ($r = .24$, $SE = .02$, 95% CI [.21, .27]) than diagnoses ($r = .17$, $SE = .02$, 95% CI [.13, .22]). No other descriptive moderators influenced effects. That is, neither race ($\beta = -.001$, $p = .205$), publication year ($\beta = .000006$, $p = .998$), nor continent ($\beta = .06$, $p = .198$), influenced the magnitude of the link between psychopathology and dependent stress.

Aim 2: Do Types of Psychopathology Evince Distinct Patterns of Stress Generation?

We investigated whether types of psychopathology display distinct patterns of stress generation by examining similarities and differences in (a) the magnitude of stress generation effects on dependent stress, (b) the moderators that determine for whom and under what conditions stress generation effects are strongest, and (c) the type of stress (i.e., dependent interpersonal vs. dependent noninterpersonal) generated.

Differences in the Magnitude of Prospective Associations With Dependent Stress

To test whether types of psychopathology differed in the magnitude of their prospective associations with dependent stress, we examined whether type of psychopathology moderated the overall effect of psychopathology on dependent stress. As described above, the model for the effect of psychopathology on dependent stress demonstrated significant heterogeneity. We examined differences between internalizing and externalizing psychopathology, with internalizing coded as the reference category, as well as differences across disorder clusters. Disorder clusters were only included as a category when they contained at least three independent study samples. This resulted in the inclusion of depressive symptoms and disorders ($k = 74$ independent study samples), anxiety symptoms and disorders ($k = 17$), personality symptoms and disorders ($k = 3$), substance use symptoms and disorders ($k = 3$), and disruptive

Table 1
Summary of Included Studies

Study				Age (years)									
ID	Name [*Subgroup*]	*N*	Country	Range	*M* (*SD*)	% Female	% White	Follow-up (months)[a]	*N* effect sizes	Sub-construct	Dx time frame	Method	Quality
1	Shih et al. (2009)	140	Canada	6–14	9.8 (2.4)	51	84	12.0	3	Depression Sx	—	H	6
2	Auerbach, Bigda-Peyton, et al. (2011)	258	Canada	12–18	14.5 (1.5)	57	80	1.4	4	Anxiety Sx	—	C	4
2	Auerbach, Bigda-Peyton, et al. (2011)								4	Depression Sx	—	C	4
3	Hamilton et al. (2013)	301	United States	12–13	12.8 (0.6)	56	48	9.0	2	Anxiety Sx	—	H	6
3	Hamilton et al. (2013)								1	Depression Sx	—	H	6
3	Hamilton et al. (2014)	310	United States	12–13	12.8 (0.6)	55	47	9.0	2	Anxiety Sx	—	H	4
3	Hamilton et al. (2015)	382	United States	12–13	12.9 (0.6)	53	49	7.0	3	Depression Sx	—	H	5
4	Safford et al. (2007)	157	United States	—	19.3 (2.2)	67	61	6.0	2	Depression Dx	Lifetime	H	5
5	Hamilton (2017)	105	United States	18–22	19.8 (1.2)	76	71	0.5	2	Depression Dx	Past	C	4
6	Bender et al. (2010)	164	United States	18–24	19.7 (1.9)	67	57	4.0	3	Bipolar disorder Dx	Current	H	5
6	Bender et al. (2010)								3	Depression Sx	—	H	5
6	Bender et al. (2010)								3	Hypomania Sx	—	H	5
7	Holahan et al. (2005)	1,211	United States	55–65	61 (3.2)	41	92	48.0	1	Depression Sx	—	C	3
8	Calvete et al. (2013)	1,187	Spain	13–17	13.4 (1.3)	46	—	6.0	1	Depression Sx	—	C	5
8	Calvete et al. (2015)	1,000	Spain	—	13.4 (1.3)	46	—	12.0	1	Depression Sx	—	C	5
9	Calvete (2011)	853	United States	14–17	15.9 (1.0)	52	—	6.0	1	Depression Sx	—	C	5
10	Alba and Calvete (2019)	584	Spain	14–19	16 (1.1)	45	—	4.0	1	Depression Sx	—	C	4
11	Calvete et al. (2019)	1,190	Spain	13–18	15.2 (1.0)	49	—	24.0	1	Depression Sx	—	C	4
12	Chun et al. (2004)	848	United States	18+	39.7	55	87	12.0	6	Depression Dx	Current	C	4
13	Sahl et al. (2009)	127	United States	—	19.0[b]	67	80	0.2	2	Depression Sx	—	C	4
14	Cox et al. (2009) [*Males*]	271	Canada	—	44.0 (16.0)	0	—	12.0	1	Depression Sx	—	C	5
14	Cox et al. (2009) [*Females*]	281	Canada	—	44.0 (16.0)	100	—	12.0	1	Depression Sx	—	C	5
15	Cummings et al. (2010)	310	United States	—	19.7 (1.2)	78	87	0.7	2	Depression Dx	Current	C	4
15	Cummings et al. (2010)								2	Depression Sx	—	C	4
15	Cummings et al. (2013)	305	United States	—	19.7 (1.2)	78	87	0.7	2	Avoidant personality disorder Sx	—	C	3
16	Daviss and Diler (2012)	79	United States	11–18	13.8 (1.8)	39	78	8.0	2	ADHD Dx	Current	C	4
16	Daviss and Diler (2012)								2	ADHD Sx	—	C	4
16	Daviss and Diler (2012)								2	Anxiety disorder Dx	Current	C	4
16	Daviss and Diler (2012)								2	Depression Sx	—	C	4
16	Daviss and Diler (2012)								2	Externalizing disorder Dx	Current	C	4
17	Driscoll (2011)	98	United States	18–67	33.4 (11.0)	81	0	6.0	2	Depression Sx	—	C	5
18	Dudeck (2007)	183	Canada	—	13.0 (0.7)	52	—	12.0	4	Depression Sx	—	C	4
19	Mandel et al. (2018)	145	Canada	18–65	41.2 (12.3)	69	76	12.0	8	Depression Sx	—	I	5
20	Eberhart and Hammen (2009)	104	United States	17–23	18.8 (1.2)	100	28	0.9	1	Depression Sx	—	H	5
21	Feurer et al. (2022)	180	United States	8–14	11.4 (1.9)	52	100	6.0	2	Anxiety Sx	—	I	5
21	Feurer et al. (2022)								2	Depression Dx	Lifetime	I	5
21	Feurer et al. (2022)								2	Depression Sx	—	I	5
22	Freedman (2000)	125	United States	20–81	48.0	57	96	18.0	2	Depression Sx	—	I	5
23	Conway et al. (2012)	705	Australia	—	20.0 (0.0)	51	92	60.0	3	Alcohol abuse/ dependence Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Conduct disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								6	Depression Dx	Lifetime	I	5

(*table continues*)

Table 1 (*continued*)

Study				Age (years)									
ID	Name [*Subgroup*]	*N*	Country	Range	*M* (*SD*)	% Female	% White	Follow-up (months)[a]	*N* effect sizes	Sub-construct	Dx time frame	Method	Quality
23	Conway et al. (2012)								3	Drug abuse/ dependence Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Generalized anxiety disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Obsessive–compulsive disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Oppositional defiant disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Panic disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Posttraumatic stress disorder Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Social phobia Dx	Lifetime	I	5
23	Conway et al. (2012)								3	Specific phobia Dx	Lifetime	I	5
23	Starr et al. (2012)	381	Australia	15	15.0	61	95	60.0	2	Depression Sx	—	I	6
23	Starr et al. (2013)	354	Australia	15	15.1	61	100	60.0	1	Depression Dx	Lifetime	I	5
23	Starr et al. (2013)								1	Depression Sx	—	I	5
24	Daley (1996)	155	United States	16–19	18.2	100	46	12.0	4	Personality disorder Sx	—	I	5
24	Daley et al. (1997)	155	United States	16–19	18.3 (0.5)	100	46	12.0	3	Depression Dx	Current	I	6
24	Daley et al. (1998)	155	United States	16–19	18.3 (0.5)	100	46	3.0	1	Cluster A personality disorder Sx	—	I	5
24	Daley et al. (1998)								1	Cluster B personality disorder Sx	—	I	5
24	Daley et al. (1998)								1	Depression Sx	—	I	5
25	Hammen (1991) [*Bipolar Dx versus Healthy*]	33	United States	—	37.4 (5.1)	100	76	12.0	3	Bipolar disorder Dx	Past	I	5
25	Hammen (1991) [*MDD Dx versus Healthy*]	36	United States	—	37.6 (4.9)	100	72	12.0	3	Depression Dx	Past	I	5
26	Wetter and Hankin (2009)	350	United States	11–17	14.5 (1.4)	57	53	5.0	2	Depression Sx	—	C	4
27	Jenness et al. (2019)	382	United States, Canada	11–15	13.0 (1.1)	59	71	3.0	7	Anxiety Sx	—	C	4
27	Jenness et al. (2019)								7	Depression Sx	—	C	4
28	Harkness and Stewart (2009)	88	Canada	13–18	15.4 (1.3)	65	96	12.0	3	Depression Dx	Current	I	5
28	Harkness and Stewart (2009)								4	Depression Sx	—	I	5
29	Harkness et al. (2014)	68	Canada	18–60	42.7 (12.7)	62	—	12.0	2	Depression Sx	—	I	5
30	Drieling et al. (2006) [*High Risk Group*]	89	Germany	—	29.5 (7.9)	66	—	80.0	1	Depression Sx	—	H	4
30	Drieling et al. (2006) [*Comparison Group*]	49	Germany	—	30.0 (7.4)	53	—	77.0	1	Depression Sx	—	H	4
31	Maciejewski et al. (2000)	2,858	United States	25+	52.9 (17)	64	67	36.0	2	Depression Sx	—	C	5
32	Joiner et al. (2005)	178	United States	—	19.0[b]	44	—	1.2	2	Depression Sx	—	C	3
33	Kendler et al. (2002)	1942	United States	—	35.8 (8.2)	100	100	36.0	2	Depression Dx	Lifetime		3
34	Kercher and Rapee (2009)	756	Australia	—	12.8 (0.4)	49	86	6.0	1	Depression Sx	—	C	3
35	Kercher et al. (2009)	896	Australia	10–13	12.3 (0.4)	100	80	12.0	2	Depression Sx	—	C	4
36	Kindt et al. (2015)	1,343	Netherlands	—	13.4 (0.8)	52	—	6.0	3	Depression Sx	—	C	4
37	Kleiman (2013)	193	United States	17–44	20.7 (3.9)	70	45	1.4	2	Depression Sx	—	H	4
37	Kleiman (2013)								2	Suicide ideation	—	H	4
38	Goldstein et al. (2020)	550	United States	13–15	14.4 (0.6)	100	81	9.0	4	Depression Sx	—	I	5
38	Mackin et al. (2019)	467	United States	13–15	14.4 (0.6)	100	88	18.0	1	Depression Sx	—	I	6

(*table continues*)

Table 1 (*continued*)

Study				Age (years)									
ID	Name [*Subgroup*]	*N*	Country	Range	*M* (*SD*)	% Female	% White	Follow-up (months)[a]	*N* effect sizes	Sub-construct	Dx time frame	Method	Quality
39	Mumper et al. (2020)	392	United States	9	9.2 (0.4)	47	95	36.0	2	Anxiety Sx	—	I	5
40	Little (2000)	240	United States	—	11.9 (0.6)	54	82	12.0	4	Depression Sx	—	I	6
40	Little (2000)								4	Externalizing Sx	—	I	6
41	Liu, Kraines, et al. (2014)	185	United States	—	19.7 (1.5)	76	56	4.0	4	Depression Dx	Lifetime	H	5
41	Liu, Kraines, et al. (2014)								4	Depression Sx	—	H	5
42	Liu and Spirito (2019)	99	United States	13–17	14.7 (1.5)	80	78	6.0	2	Depression Sx	—	I	7
42	Liu and Spirito (2019)								2	Suicide attempts	—	I	7
42	Liu and Spirito (2019)								2	Suicide ideation	—	I	7
43	Maciejewski et al. (2021)	1,771	Netherlands	18–65	42.0 (13.2)	64	—	24.0	8	Depression Sx	—	C	3
44	Meiser and Esser (2019)	924	Germany	9–13	12.1 (1.1)	48	—	19.0	2	Depression Sx	—	I	5
45	Aldrich (2020)	150	United States	11–14	13.0 (0.9)	51	78	4.0	6	Depression Sx	—	I	5
46	Morris et al. (2014)	68	United States	18–31	23.4 (3.9)	63	—	8.0	2	Depression Dx	Past	H	4
46	Morris et al. (2014)								14	Depression Sx	—	H	4
47	Conway et al. (2018)	1,630	United States	55–64	59.6 (2.7)	55	65	6.0	6	Borderline personality disorder Sx	—	H	4
47	Powers et al. (2013)	1,294	United States	55–64	59.6 (2.8)	54	69	6.0	12	Antisocial personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Avoidant personality disorder Sx	—	C	3
47	Powers et al. (2013)								10	Borderline personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Dependent personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Histrionic personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Narcissistic personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Obsessive compulsive personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Paranoid personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Schizoid personality disorder Sx	—	C	3
47	Powers et al. (2013)								12	Schizotypal personality disorder Sx	—	C	3
48	Jeronimus et al. (2017)	1816	Netherlands	—	16.3 (0.7)	55	100	24.0	2	Externalizing Sx	—	I	5
48	Jeronimus et al. (2017)								2	Internalizing Sx	—	I	5
48	Jeronimus et al. (2017)								2	Psychopathology	—	I	5
49	Birgenheir et al. (2010)	110	United States	18–40	19.4 (2.7)	73	92	1.4	1	Depression Sx	—	C	3
50	Pettineo (2011)	177	United States	14–22	17.3 (1.6)	100	0	24.0	4	Depression Sx	—	C	3
51	Kleiman et al. (2015)	209	United States	17–50	20.6 (4.1)	84	54	0.9	1	Depression Sx	—	C	3
51	Liu and Kleiman (2012)	201	United States	—	20.5 (0.3)	84	53	0.9	1	Depression Sx	—	C	5
52	Riskind et al. (2013)	99	United States	18–48	21.3 (5.1)	100	50	1.4	1	Internalizing Sx	—	C	3
53	Rnic, (2014)	151	Canada	18–28	19.7 (2.2)	100	51	3.0	4	Anxiety Sx	—	I	4
53	Rnic (2014)								4	Depression Sx	—	I	4
54	Bottonari et al. (2007)	68	United States	—	40.1 (11.3)	71	91	3.0	2	Depression Sx	—	H	4
55	Rose et al. (2017)	628	United States	—	14.5	52	63	9.0	2	Depression Sx	—	C	5

(*table continues*)

Table 1 (*continued*)

Study				Age (years)									
ID	Name [*Subgroup*]	*N*	Country	Range	*M* (*SD*)	% Female	% White	Follow-up (months)[a]	*N* effect sizes	Sub-construct	Dx time frame	Method	Quality
56	Flynn and Rudolph (2011)	167	United States	9–14	12.4 (1.2)	52	78	12.0	4	Anxiety Sx	—	I	6
56	Flynn and Rudolph (2011)								4	Depression Sx	—	I	6
56	Rudolph and Klein (2009)	143	United States	—	12.4 (1.3)	54	78	12.0	3	Depression Sx	—	I	5
57	Champion et al. (1995)	192	United Kingdom.	10	10.0 (0.0)	40	100	240.0	2	Behavior problems	—	I	3
58	Rychik et al. (2021)	224	United States	18–23	19.3	70	—	—	1	ADHD Sx	—	C	4
58	Rychik et al. (2021)								1	Depression Sx	—	C	4
59	Clements et al. (2008)	907	United States	—	11.4 (0.9)	54	19	12.0	4	Depression Sx	—	C	3
60	Shahar and Priel (2003)	603	Israel	14–16	15.0[b]	54	—	4.0	2	Anxiety Sx	—	C	4
60	Shahar and Priel (2003)								2	Depression Sx	—	C	4
61	Bouchard and Shih (2013)	364	United States	18–25	19.7 (1.3)	57	90	1.8	2	Depression Sx	—	C	5
61	Shih et al. (2018)	364	United States	18–25	19.7 (1.3)	57	90	1.8	1	Depression Sx	—	C	4
62	Shih and Eberhart (2010)	99	United States	—	19.1 (1.1)	51	44	1.4	1	Depression Dx	Lifetime	H	4
62	Shih and Eberhart (2010)								1	Depression Sx	—	H	4
62	Shih (2003)	99	United States	—	19.1 (1.2)	51	44	1.4	1	Depression Sx	—	H	4
63	Snyder and Hankin (2016)	360	United States	8–16	12.1 (2.4)	57	75	15.0	1	Anxiety Sx	—	C	4
63	Snyder and Hankin (2016)								1	Depression Sx	—	C	4
64	Stroud et al. (2015)	126	United States	10–13	12.4 (0.8)	100	83	12.0	2	Depression Sx	—	I	5
64	Stroud et al. (2018)	126	United States	10–13	12.4 (0.8)	100	83	12.0	4	Current depression Sx	—	I	5
64	Stroud et al. (2018)								4	Lifetime depression Sx	—	I	5
65	Goldstein et al. (2021)	917	United States	18+	19.2 (1.3)	54	87	60.0	4	Alcohol use Sx	—	I	4
65	Goldstein et al. (2021)								4	Anxiety Sx	—	I	4
65	Goldstein et al. (2021)								4	Depression Sx	—	I	4
65	Goldstein et al. (2021)								4	Posttraumatic stress disorder Sx	—	I	4
66	Schmied et al. (2016)	1,599	United States	—	29.9 (8.9)	14	66	12.0	2	Depression Sx	—	C	3
66	Schmied et al. (2016)								2	Posttraumatic stress disorder Sx	—	C	3
66	Schmied et al. (2016)								2	Substance abuse Sx	—	C	3
67	Chan et al. (2014)	171	United States	8–12	10.1 (1.4)	42	68	12.0	1	Depression Sx	—	I	5
68	Trombello et al. (2011)	344	United States	—	27.0 (3.7)	50	64	6.0	28	Depression Sx	—	C	4
69	Tsai (2016) [*European Americans*]	304	United States	—	15.6 (0.6)	53	100	6.0	1	Depression Sx	—	C	4
69	Tsai (2016) [*Vietnamese Americans*]	372	United States	—	15.6 (0.6)	52	0	6.0	1	Depression Sx	—	C	4
70	Tuna (2020)	162	Turkey	18–29	21.1 (1.7)	93	—	5.0	1	Depression Sx	—	C	4
71	Cui and Vaillant (1997)	113	United States	—	—	0	—	—	2	Indicators of possible depression Dx	—	C	3
72	Waaktaar et al. (2004)	163	Norway	12–16	13.8 (0.8)	52	—	13.0	2	Depression Sx	—	C	4
73	Wals et al. (2005)	132	Netherlands	12–21	16.1 (2.7)	30	—	14.0	2	Internalizing Sx	—	I	5
74	Judah et al. (2013)	112	United States	—	19.4 (2.3)	74	88	0.9	2	Anxiety Sx	—	C	4
74	Judah et al. (2013)								2	Depression Sx	—	C	4
75	Maniates et al. (2018)	116	United States	27–70	56.2 (9.5)	12	77	24.0	2	Depression Sx	—	I	4
75	Maniates et al. (2018)								2	Posttraumatic stress disorder Sx	—	I	4
76	Uliaszek et al. (2012)	627	United States	15–18	16.9 (0.4)	69	48	12.0	2	Anxiety disorder Dx	Current	I	4
76	Uliaszek et al. (2012)								2	Depression Dx	Current	I	4
77	Allen et al. (2022)	355	United States	18–50	28.3 (7.6)	77	75	12.0	6	Depression Sx	—	C	4
77	Allen et al. (2022)								6	Internalizing Sx	—	C	4

(*table continues*)

Table 1 (*continued*)

Study				Age (years)				Follow-up (months)[a]	*N* effect sizes		Dx time frame		
ID	Name [*Subgroup*]	*N*	Country	Range	*M* (*SD*)	% Female	% White			Sub-construct		Method	Quality
78	Barker (2020)	645	United States	17–41	18.7	59	90	—	1	Depression Sx	—	C	5
79	La Rocque et al. (2016)	301	Canada	—	18.3 (2.0)	86	70	4.0	2	Depression Sx	—	I	5
80	Auerbach, Webb, et al. (2011)	405	China	14–19	16.2 (1.0)	50	0	1.0	6	Anxiety Sx	—	C	4
80	Auerbach, Webb, et al. (2011)								6	Depression Sx	—	C	4

Note. Each ID number corresponds to an independent study sample. Quality was assessed using the Newcastle-Ottawa Scale (Wells et al., 2011). ID = identification number; Dx = Diagnosis; Sx = Symptoms; C = Checklist; H = Hybrid Checklist/Interview; I = Interview; ADHD = attention-deficit/hyperactivity disorder; MDD = major depressive disorder.
[a]Where multiple follow-ups were conducted, the shortest length of follow-up is reported. [b]Value imputed from another article reporting on the same study sample.

symptoms and disorders ($k = 5$). Given that stress generation theory originated, and is relatively more established, in the depression literature, coupled with the relatively small number of studies in some disorder clusters, we only conducted analyses examining depression as the reference category.

The effect of internalizing/externalizing was nonsignificant, $\beta = .01$, $p = .785$, indicating that the effect of psychopathology on dependent stress does not differ as a function of internalizing or externalizing psychopathology. In contrast, there was a significant effect of disorder cluster, whereby the association with dependent stress was significantly larger for depression ($r = .24$, $SE = .02$, 95% CI [.20, .27]) than for anxiety ($r = .18$, $SE = .02$, 95% CI [.14, .21]), $\beta = -.06$, $p = .002$. Effect sizes for the anxiety disorder cluster were predominantly based on general anxiety symptoms ($k = 35$ effects) and disorders ($k = 3$). Additional effects were based on generalized anxiety disorder ($k = 2$), obsessive–compulsive disorder ($k = 2$), specific phobia disorder ($k = 2$), social anxiety disorder ($k = 2$), panic disorder ($k = 2$), and posttraumatic stress disorder symptoms ($k = 4$) or disorder ($k = 2$). No other disorder clusters differed significantly from depression in their association with dependent stress ($|\beta|$s $\leq .07$, ps $\geq .252$).

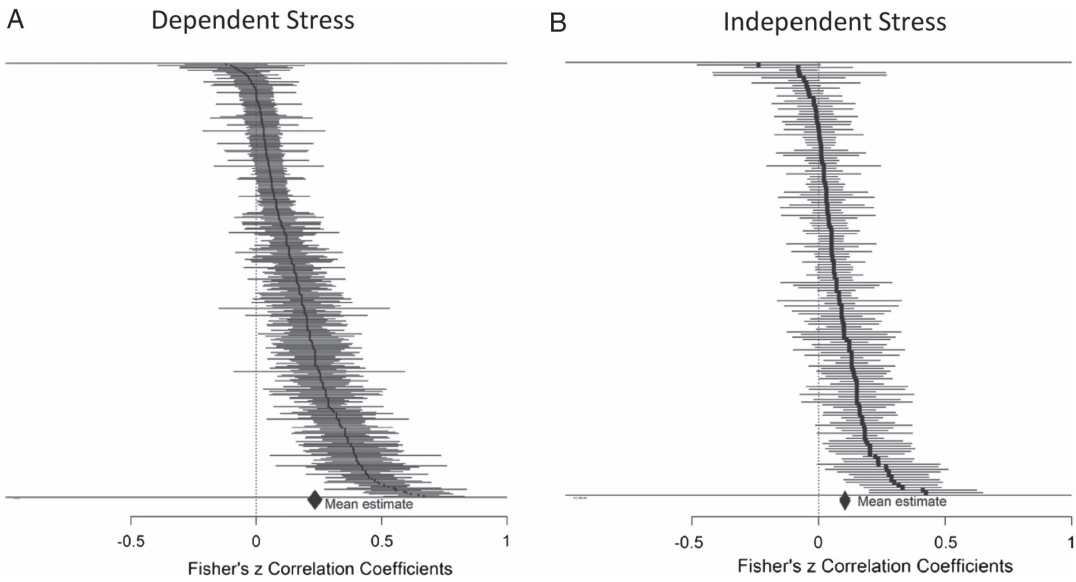
Tests of the Stress Generation Effect for Types of Psychopathology

Before assessing patterns of moderation in the generation of dependent stress, we tested the stress generation effect for each type of psychopathology. This was achieved by examining the moderating effect of stressor dependence on random-effects multilevel models of the association of psychopathology with overall stress. To ensure reliable parameter estimates of random-effects metaregression models, we only examined types of psychopathology for which effect sizes had been extracted from $k \geq 5$ unique studies. This resulted in the following psychopathology clusters: internalizing psychopathology ($k = 78$), externalizing psychopathology ($k = 10$), depressive symptoms and disorders, ($k = 74$), anxiety symptoms and disorders ($k = 17$), and childhood disruptive disorders ($k = 5$). Models for the effect of each type of psychopathology on overall stress all yielded significant mean estimates, rs $\geq .16$, ps $\leq .014$, significant total heterogeneity, Qs ≥ 69.94, ps $< .001$, as well as significant heterogeneity across levels (see the Supplemental Material, for σ^2 and I^2 values). Consistent with the stress generation hypothesis, there was a significant moderating effect of stressor dependence for internalizing, externalizing, depression, and anxiety, βs $\geq .05$, ps $\leq .031$, such that the association of types of psychopathology was larger for dependent than for independent stress. The effect of stressor dependence was nonsignificant in the model of the association of disruptive disorders with overall stress, $\beta = .08$, $p = .085$, though summary effects for dependent ($r = .20$, $SE = .05$, 95% CI [.05, .34]) and independent stress ($r = .12$, $SE = .03$, 95% CI [.02, .22]) fell in the expected direction.

Analyses of moderation by stressor dependence were followed up by estimating separate unconditional random-effects multilevel models for the association of each type of psychopathology with dependent and independent stress (see Figure 5, for mean estimates). We confirmed significant heterogeneity across levels in random-effects multilevel models for dependent stress before proceeding with analyses of main and descriptive moderators. All models showed significant heterogeneity, Qs ≥ 52.61, ps $< .001$ (see the

Figure 2

Caterpillar Plots Displaying Observed Effect Sizes Reflecting Prospective Associations of Psychopathology With Stress



Note. Horizontal bars represent 95% confidence intervals of observed effect sizes. Panel A represents the effects of psychopathology on dependent stress ($k = 371$ effect sizes; mean estimate: $r = .23$, $p < .011$), and Panel B represents the effects of psychopathology on independent stress ($k = 166$ effect sizes; mean estimate: $r = .10$, $p < .001$). Consistent with the stress generation hypothesis, the prospective association of psychopathology with stress was significantly greater for dependent than for independent stress, $\beta = .054$, $p = .008$.

Supplemental Material, for all σ^2 and I^2 values). Though heterogeneity was not significant at Level 3 in the model for disruptive disorders on dependent stress, $\sigma_{(3)}{}^2 = .005$, $p = .142$, given that less than 75% of the variance was accounted for by the sampling variance at Level 1 ($I_{(1)}{}^2 = 16.79\%$), heterogeneity was still considered substantial enough to warrant assessment of moderators (Assink & Wibbelink, 2016; Hunter & Schmidt, 2004). See Table 3 for a summary of findings.

In sum, evidence was found for the stress generation phenomenon across all types of psychopathology examined, with the exception of disruptive disorders. Models of the association of psychopathology on dependent stress evinced substantial heterogeneity, allowing us to proceed with our planned moderator analyses.

Differences in Moderators of Associations of Psychopathology With Dependent Stress

Main Moderators.

Age at Baseline. There were significant moderating effects of age at baseline on the associations of internalizing psychopathology and depression with dependent stress. Studies of adolescents/early adults reported larger associations of internalizing with dependent stress ($r = .26$, $SE = .02$, 95% CI [.22, .30]) than did studies of adults ($r = .19$, $SE = .03$, 95% CI [.13, .24]), $\beta = -.07$, $p = .037$. There were no other significant age group differences for internalizing psychopathology ($|\beta|$s $\leq .06$, ps $\geq .157$), and the linear effect of age was nonsignificant, $\beta = -.002$, $p = .106$. Age similarly moderated the association of depression with dependent stress such that effects were larger for samples of adolescents and early adults ($r = .27$, $SE = .02$, 95% CI [.23, .31]) than for adults ($r = .19$, $SE = .03$, 95% CI [.13, .24]), $\beta = -.09$, $p = .020$. There was also a significant linear effect of age on the depression-dependent stress link ($\beta = -.003$, $p = .032$), such that younger age was associated with larger effects. Together with the significant difference by age group, these findings suggest that the association of depression with dependent stress is strongest among adolescents and early adults, and then declines with older age. In contrast, there were no effects of age on associations of externalizing, anxiety, or disruptive disorders with dependent stress, $|\beta|$s $\leq .02$, ps $\geq .149$, suggesting that effects do not significantly vary by age for these disorder clusters.

Length of Follow-Up. Length of follow-up was a significant moderator for the associations of internalizing psychopathology and anxiety with dependent stress. The significant moderating effect of length of follow-up for internalizing, $\beta = -.002$, $p = .046$, indicated that a longer time lag between the assessment of internalizing symptoms and disorders and the assessment of dependent stress was associated with smaller effects. The effect of length of follow-up on anxiety evinced a similar pattern: a longer interval between assessments was associated with a smaller effect of anxiety on dependent stress, $\beta = -.004$, $p = .034$. Together, these findings suggest that internalizing and anxiety likely play a proximal role in the generation of dependent stress. In contrast, length of follow-up did not moderate associations of externalizing, depression, or disruptive disorders with dependent stress, $|\beta|$s $\leq .001$, ps $\geq .067$.

Stress Assessment Method. Stress assessment moderated associations of internalizing psychopathology, depression, and anxiety with dependent stress. Specifically, stress assessment method moderated effects for internalizing such that studies using checklist

Table 2

Results of Multilevel Metaregression Assessing Moderators of Stress Generation Effects

Moderator and levels	*k* studies	*k* ESs	*r*	β	*SE*	[95% CI]	*p*	*Q*	$\sigma_{(2)}^2$	$\sigma_{(3)}^2$
Overall psychopathology and dependent stress										
Main moderators										
Age—continuous	79	370		−.003	.001	[−.0048, −.0004]	.023	2039.664***	.003	.012
Age—categorical	80	371						2394.109***	.003	.012
Child	14	43	.204		.033	[.135, .271]				
Adolescent/early adult	45	172	.261		.019	[.225, .297]				
Adult	21	156	.174		.025	[.122, .225]				
Child versus adolescent/ early adult				.060	.038	[−.019, .139]	.127			
Child versus adult				−.031	.042	[−.116, .054]	.460			
Adolescent/early adult versus adult				−.091	.032	[−.155, −.027]	.007			
Gender (% girls/women)	80	371		.0001	.0004	[−.001, .001]	.813	3239.919***	.003	.013
Length of follow-up (months)	77	367		−.001	.0003	[−.0019, −.0002]	.021	3192.932***	.003	.012
Diagnostic timeframe	16	56						199.33***	.006	.001
Current	7	19	.157		.024	[.094, .220]				
Remitted	3	6	.288		.061	[.005, .529]				
Lifetime	6	31	.132		.027	[.008, .252]				
Current versus remitted				.137	.065	[−.108, .367]	.150			
Current versus lifetime				−.026	.036	[−.0116, .065]	.501			
Remitted versus lifetime				−.163	.066	[−.399, .094]	.118			
Stress assessment method	79	370						3210.293***	.003	.013
Checklist	43	219	.244		.028	[.190, .296]				
Interview	25	115	.181		.018	[.144, .218]				
Checklist/interview hybrid	12	36	.295		.064	[.139, .436]				
Checklist versus interview				−.065	.034	[−.132, .002]	.057			
Checklist versus hybrid				.055	.081	[−.261, .360]	.564			
Interview versus hybrid				.120	.066	[−.022, .256]	.091			
Stress domain (*interpersonal vs. noninterpersonal*)	35	220		−.024	.022	[−.089, .041]	.348	1844.758***	.003	.012
Interpersonal	35	137	.226		.023	[.180, .270]				
Noninterpersonal	20	83	.203		.022	[.159, .246]				
Other moderators										
Psychopathology measure (*symptom vs. diagnosis*)	80	371		−.068	.022	[−.119, −.018]	.014	3265.133***	.003	.013
Symptom-based	74	315	.238		.015	[.210, .267]				
Diagnosis	16	56	.173		.022	[.125, .219]				
Race (% White)	61	337		−.001	.0005	[−.0018, .0005]	.205	2493.931***	.003	.010
Publication year	79	368		.000	.003	[−.007, .007]	.998	3264.244***	.003	.013
Continent (*North America vs. outside North America*)	80	371		.057	.043	[−.032, .144]	.198	2942.097***	.003	.013
North America	63	305	.218		.015	[.189, .246]				
Outside North America	17	66	.271		.040	[.190, .349]				
Study quality	80	371		−.017	.032	[.084, .050]	.592	2891.535***	.003	.013
Internalizing and dependent stress										
Main moderators										
Age—continuous	77	242		−.002	.001	[−.0043, .0005]	.106	1568.817***	.004	.013
Age—categorical	78	243						1609.552***	.004	.012
Child	13	40	.201		.035	[.126, .274]				
Adolescent/early adult	45	142	.258		.020	[.219, .296]				
Adult	20	61	.188		.027	[.132, .242]				
Child versus adolescent/ early adult				.060	.041	[−.025, .144]	.157			
Child versus adult				−.014	.045	[−.105, .078]	.762			
Adolescent/early adult versus adult				−.073	.034	[−.141, −.005]	.037			
Gender (% girls/women)	78	243		−.0001	.0005	[−.001, .001]	.822	1652.305***	.004	.013
Length of follow-up (months)	75	240		−.002	.001	[−.00309, −.00004]	.046	1207.136***	.004	.011
Diagnostic timeframe	15	41						160.949***	.006	.003
Current	6	14	.153		.039	[.043, .260]				
Remitted	3	4	.313		.094	[−.086, .626]				
Lifetime	6	23	.144		.032	[.049, .236]				

(*table continues*)

Table 2 (*continued*)

Moderator and levels	*k* studies	*k* ESs	*r*	β	*SE*	[95% CI]	*p*	*Q*	$\sigma_{(2)}^2$	$\sigma_{(3)}^2$
Current versus remitted				.169	.102	[−.168, .470]	.201			
Current versus lifetime				−.009	.050	[−.127, .109]	.863			
Remitted versus lifetime				−.177	.099	[−.472, .152]	.177			
Stress assessment method	77	242						1336.083***	.004	.011
Checklist	42	124	.271		.022	[.229, .312]				
Interview	24	92	.169		.019	[.130, .208]				
Checklist/interview hybrid	11	26	.209		.033	[.136, .280]				
Checklist versus interview				−.107	.030	[−.165, −.047]	<.001			
Checklist versus hybrid				−.065	.040	[−.151, .021]	.125			
Interview versus hybrid				.041	.039	[−.040, .122]	.299			
Stress Domain (*interpersonal vs. noninterpersonal*)	34	143		−.076	.020	[−.121, −.031]	.004	784.601***	.004	.009
Interpersonal	34	98	.247		.021	[.207, .287]				
Noninterpersonal	19	45	.174		.023	[.128, .221]				
Other moderators										
Psychopathology measure (*symptom vs. diagnosis*)	78	243		−.056	.025	[−.113, .002]	.056	1498.04***	.004	.013
Symptom-based	72	202	.238		.016	[.208, .267]				
Diagnosis	15	41	.185		.025	[.130, .238]				
Race (% White)	59	210		−.001	.001	[−.002, .001]	.229	1047.655***	.004	.011
Publication year	77	240		−.001	.002	[−.006, .004]	.636	1652.982***	.004	.013
Continent (*North America vs. outside North America*)	78	243		.052	.046	[−.043, .146]	.272	1655.124***	.004	.013
North America	62	188	.220		.015	[.191, .250]				
Outside North America	16	55	.269		.043	[.181, .353]				
Study quality	78	243		−.053	.014	[−.082, −.024]	<.001	1531.008***	.004	.012
Externalizing and dependent stress										
Main moderators										
Age—continuous	10	29		−.005	.002	[−.015, .005]	.149	126.000***	.002	.011
Gender (% girls/women)	10	29		.003	.001	[−.001, .008]	.089	170.899***	.002	.013
Length of follow-up	9	28		−.0003	.001	[−.006, .006]	.817	146.794***	.002	.013
Stress assessment method	10	29		.006	.099	[−.228, .239]	.954	148.601***	.002	.017
Checklist	4	14	.186		.085	[−.085, .431]				
Interview	6	15	.192		.051	[.063, .314]				
Other moderators										
Stress domain (*interpersonal vs. noninterpersonal*)	4	18		.048	.019	[−.039, .135]	.134	73.463***	.002	.008
Interpersonal	4	9	.075		.048	[−.080, .226]				
Noninterpersonal	4	9	.123		.049	[−.034, .273]				
Race (% White)	9	28		−.0002	.004	[−.011, .010]	.954	121.306***	.002	.013
Publication year	10	29		−.004	.005	[−.018, .010]	.471	162.277***	.002	.015
Continent (*North America vs. outside North America*)	10	29		.012	.079	[−.209, .233]	.886	139.411***	.002	.017
North America	7	19	.186		.060	[.041, .322]				
Outside North America	3	10	.197		.052	[−.030, .406]				
Study quality	10	29		.057	.027	[−.016, .129]	.098	122.136***	.002	.013
Depression and dependent stress										
Main moderators										
Age—continuous	73	182		−.003	.001	[−.0054, −.0003]	.032	1046.718***	.003	.015
Age—categorical	74	183						1042.982***	.003	.015
Child	12	31	.235		.038	[.154, .314]				
Adolescent/early adult	42	96	.268		.023	[.225, .310]				
Adult	20	56	.185		.028	[.128, .241]				
Child versus adolescent/early adult				.035	.045	[−.059, .128]	.445			
Child versus adult				−.053	.048	[−.150, .045]	.275			
Adolescent/early adult versus adult				−.088	.036	[−.159, −.015]	.020			
Gender (% girls/women)	74	183		−.0003	.001	[−.002, .001]	.650	1163.313***	.003	.016
Length of follow-up (months)	71	180		−.001	.0005	[−.0027, .0002]	.067	928.067***	.003	.014
Diagnostic timeframe	14	26						63.977***	.005	.002

(*table continues*)

Table 2 (*continued*)

Moderator and levels	*k* studies	*k* ESs	*r*	β	*SE*	[95% CI]	*p*	*Q*	$\sigma_{(2)}^2$	$\sigma_{(3)}^2$
Current	5	11	.158		.041	[.034, .278]				
Remitted	3	4	.311		.096	[−.096, .629]				
Lifetime	6	11	.166		.023	[.101, .229]				
Current versus remitted				.161	.104	[−.184, .471]	.225			
Current versus lifetime				.008	.047	[−.104, .119]	.877			
Remitted versus lifetime				−.153	.098	[−.452, .176]	.223			
Stress assessment method	73	182						1071.602***	.003	.014
Checklist	41	96	.281		.025	[.235, .326]				
Interview	21	62	.174		.020	[.135, .214]				
Checklist/interview hybrid	11	24	.219		.035	[.144, .292]				
Checklist versus interview				−.112	.031	[−.174, −.049]	<.001			
Checklist versus hybrid				−.066	.043	[−.156, .025]	.142			
Interview versus hybrid				.046	.040	[−.037, .129]	.259			
Stress Domain *(interpersonal vs. noninterpersonal)*	34	100		−.060	.023	[−.110, −.009]	.024	508.68***	.003	.013
Interpersonal	34	68	.257		.024	[.211, .303]				
Noninterpersonal	19	32	.201		.025	[.151, .249]				
Other moderators										
Psychopathology measure (*symptom vs. diagnosis*)	74	183		−.034	.027	[−.097, .029]	.248	1118.949***	.003	.015
Symptom-based	68	157	.246		.017	[.213, .277]				
Diagnosis	14	26	.213		.028	[.155, .271]				
Race (% White)	56	153		−.0004	.0005	[−.002, .001]	.414	737.499***	.002	.014
Publication year	73	181		−.001	.002	[−.005, .004]	.774	1161.561***	.003	.016
Continent (*North America vs. outside North America*)	74	183		.057	.050	[−.048, .160]	.273	1153.475***	.003	.015
North America	60	150	.230		.017	[.198, .262]				
Outside North America	14	33	.283		.047	[.187, .375]				
Study quality	74	183		−.048	.013	[−.076, −.020]	.002	1133.271***	.003	.014
Anxiety and dependent stress										
Main moderators										
Age—continuous	17	54		.003	.003	[−.010, .016]	.362	408.284***	.004	.014
Gender (% girls/women)	17	54		.0001	.002	[−.005, .005]	.951	405.076***	.004	.015
Length of follow-up (months)	17	54		−.004	.001	[−.0072, −.0005]	.034	213.118***	.004	.009
Stress assessment method (*checklist vs. interview*)	17	54		−.189	.036	[−.265, −.110]	<.001	171.485***	.004	.002
Checklist	8	28	.292		.032	[.216, .364]				
Interview	8	24	.109		.017	[.066, .152]				
Checklist/interview hybrid	1	2	—		—	—				
Stress domain (*interpersonal vs. noninterpersonal*)	9	43		−.106	.018	[−.170, −.042]	.016	209.014***	.003	.009
Interpersonal	9	30	.218		.036	[.138, .295]				
Noninterpersonal	6	13	.114		.034	[.032, .194]				
Other moderators										
Psychopathology measure (*symptom vs. diagnosis*)	17	54		−.123	.068	[−.346, .113]	.181	327.412***	.004	.013
Symptom-based	14	39	.212		.036	[.137, .284]				
Diagnosis	3	15	.091		.058	[−.187, .356]				
Race (% White)	16	52		−.002	.001	[−.006, .002]	.235	256.773***	.004	.014
Publication year	16	53		−.002	.006	[−.020, .016]	.736	408.092***	.004	.015
Continent (*North America vs. outside North America*)	17	54		.026	.094	[−.259, .306]	.802	401.896***	.004	.015
North America	14	34	.186		.036	[.109, .260]				
Outside North America	3	20	.210		.086	[−.158, .527]				
Study quality	17	54		−.111	.025	[−.186, −.035]	.018	278.221***	.003	.009
Disruptive disorders and dependent stress										
Main moderators										
Age—continuous	5	9		−.016	.010	[−.055, .022]	.219	33.802***	.007	.0002
Gender (% girls/women)	5	9		−.0001	.005	[−.022, .021]	.979	50.758***	.078	.007
Length of follow-up (months)	5	9		−.0003	.001	[−.006, .005]	.789	49.077***	.006	.006

(*table continues*)

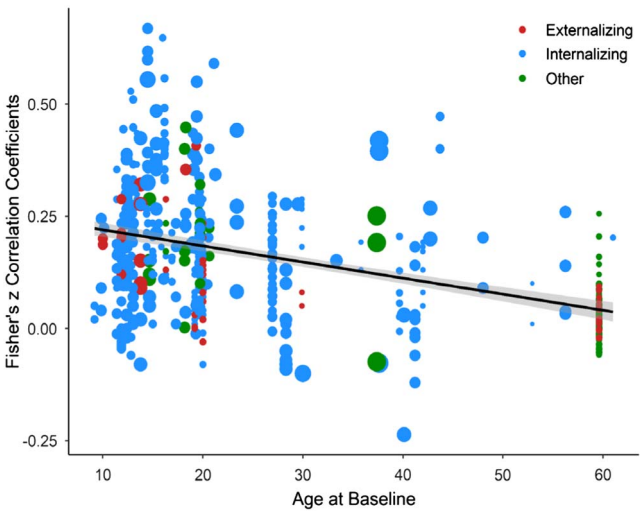
Table 2 (*continued*)

Moderator and levels	*k* studies	*k* ESs	*r*	β	*SE*	[95% CI]	*p*	*Q*	$\sigma_{(2)}^2$	$\sigma_{(3)}^2$
Other moderators										
Race (% White)	4	8		−.007	.006	[−.033, .020]	.387	27.277***	.006	.003
Publication year	5	9		−.0004	.005	[−.022, .021]	.943	52.573***	.006	.007
Study quality	5	9		.004	.023	[−.136, .144]	.875	52.437***	.006	.007

Note. Values are only displayed if effect sizes were available for $k \geq 3$ unique studies for categories of a given moderator. Specific comparisons between categories are labeled in italics. The first variable listed in comparisons represents the reference category (i.e., dummy code = 0). Note that a negative β value indicates that the first category has a larger association with dependent stress than the second category. When a categorical moderator included more than two levels, multiple models were run to provide comparisons between all combinations of categories. Separate analyses were conducted for age as a continuous or a categorical variable. CI = confidence interval; ES = effect size; *r* = Pearson's correlation coefficient; *SE* = Standard errors; *Q* = *Q* statistic for the residual heterogeneity of effect sizes; $\sigma_{(2)}^2$ = heterogeneity of effects at Level 2 (within-study variance); $\sigma_{(3)}^2$ = heterogeneity of effects at Level 3 (between-study variance). β coefficients and their corresponding *SE*s and *p* values are from metaregression analyses where moderators were entered as predictors. All categorical moderators were dummy coded using a series of dummy variables such that for each variable, the category corresponding to the variable name received a dummy code of 1, and all other categories received a dummy code of 0.
*** $p < .001$.

indices of stress ($r = .27$, $SE = .02$, 95% CI [.23, .31]) reported larger effects than studies that used interviews ($r = .17$, $SE = .02$, 95% CI [.13, .21]), $\beta = -.11$, $p < .001$. Other differences between stress assessment measures were nonsignificant ($|\beta|s \leq .07$, $ps \geq .125$). The same pattern emerged for both depression and anxiety. For depression, studies using checklist measures ($r = .28$, $SE = .03$, 95% CI [.24, .33]) reported larger effects than studies that used interviews ($r = .17$, $SE = .02$, 95% CI [.14, .21]), $\beta = -.11$, $p < .001$. Furthermore, in the model for anxiety, checklist indices were again associated with greater effects ($r = .29$, $SE = .03$, 95% CI [.22, .36]) than interviews ($r = .11$, $SE = .02$, 95% CI [.07, .15]), $\beta = -.19$, $p < .001$. No other stress assessment measure differences were significant for depression or anxiety ($|\beta|s \leq .07$, $ps \geq .142$). Furthermore, stress assessment method did not moderate effects for externalizing disorders ($\beta = .01$, $p = .954$). We were unable to examine stress assessment method as a moderator of disruptive disorders given insufficient studies across categories of this moderator. In sum, findings suggest that stress assessment method influences the magnitude of effects on dependent stress for internalizing types of psychopathology (including depression and anxiety), whereby checklist measures display the largest effects.

Figure 3
Scatterplot of the Association of Participant Age With the Effect of Overall Psychopathology on Dependent Stress



Note. Older age was associated with a smaller effect of psychopathology on dependent stress, $\beta = -.003$, $p = .023$. See the online article for the color version of this figure.

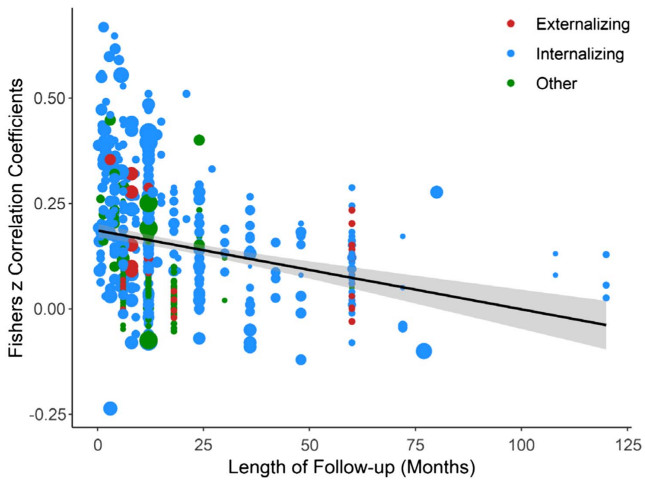
Stress Domain. There was a significant moderating effect of stress domain on associations of internalizing psychopathology, depression, and anxiety with dependent stress. As expected, in the model for the association of internalizing with dependent stress, effects were significantly larger for dependent interpersonal stress ($r = .25$, $SE = .02$, 95% CI [.21, .29]) than for dependent noninterpersonal stress ($r = .17$, $SE = .02$, 95% CI [.13, .22]), $\beta = -.08$, $p = .004$). Likewise, dependent interpersonal stress was also associated with a greater magnitude of effects for depression ($r = .26$, $SE = .02$, 95% CI [.21, .30]) compared to noninterpersonal stress ($r = .20$, $SE = .03$, 95% CI [.15, .25]), $\beta = -.06$, $p = .024$. Similarly, in the model for the association of anxiety with dependent stress, effects were larger for dependent interpersonal ($r = .22$, $SE = .04$, 95% CI [.14, .30]) than for dependent noninterpersonal stress, ($r = .11$, $SE = .03$, 95% CI [.03, .19]), $\beta = -.11$, $p = .016$. In contrast, the magnitude of effects was not influenced by stressor domain for externalizing psychopathology, $\beta = .05$, $p = .134$. We were not able to assess stress domain for disruptive disorders due to the small number of studies examining levels of this moderator. Thus, whereas internalizing disorders (including depression and anxiety) are associated with larger effects for dependent interpersonal stress, effects for externalizing psychopathology do not differ as a function of type of dependent stress.

Nonsignificant Main Moderators. Neither gender, $|\beta|s \leq .003$, $ps \geq .089$, nor diagnostic timeframe, $|\beta|s < .18$, $ps \geq .177$, moderated effects for any type of psychopathology. Diagnostic timeframe was only examined for internalizing and depressive psychopathology given the small number of studies across categories of this moderator for the other disorder clusters and psychopathology types.

Descriptive Moderators. There were no differences in effects related to descriptive moderators, which included psychopathology measure ($|\beta|s \leq .12$, $ps \geq .056$), race ($|\beta|s \leq .01$, $ps \geq .229$), publication year ($|\beta|s \leq .004$, $ps \geq .471$), and continent ($|\beta|s \leq .06$, $ps \geq .272$). Thus, the magnitude of effects for each type of psychopathology was similar across studies irrespective of whether

Figure 4

Scatterplot of the Association of Length of Follow-Up With the Effect of Overall Psychopathology on Dependent Stress



Note. Longer intervals of time between assessments were associated with smaller effects of psychopathology on dependent stress, $\beta = -.001$, $p = .021$. One study with a mean follow-up of 240 months was excluded from the current figure for clarity of presentation. See the online article for the color version of this figure.

a symptom versus diagnostic measure was used, the study's racial distribution, or when or where the study was conducted.

Differences in Type of Stress Generated

To investigate differences across psychopathology in the type of stress generated, we examined type of psychopathology as a moderator of the generation of dependent interpersonal and of dependent noninterpersonal stress. Before testing moderation, we first estimated unconditional random-effects multilevel models for each of dependent interpersonal and dependent noninterpersonal stress and confirmed their heterogeneity across levels.

Dependent Interpersonal Stress. The model for dependent interpersonal stress demonstrated substantial heterogeneity, $Q(136) = 1547.82$, $p < .001$; $\sigma_{(2)}^2 = .003$, $p < .001$; $\sigma_{(3)}^2 = .013$, $p < .001$; $I_{(1)}^2 = 11.10\%$, $I_{(2)}^2 = 14.26\%$, $I_{(3)}^2 = 74.64\%$, in the prospective association of psychopathology with dependent interpersonal stress ($r = .24$, $SE = .02$, 95% CI [.19, .28], $p < .001$). The moderating effect of internalizing versus externalizing was nonsignificant ($\beta = .03$, $p = .096$), though the estimated average effects fell in the expected direction, with internalizing ($r = .24$, $SE = .02$, 95% CI [.20, .28]) evincing a marginally larger association with dependent interpersonal stress than did externalizing ($r = .16$, $SE = .03$, 95% CI [.05, .26]). Furthermore, there was a significant moderating effect of disorder cluster, such that depression ($r = .26$, $SE = .02$, 95% CI [.21, .30]) was associated with larger effects on dependent interpersonal stress than anxiety ($r = .19$, $SE = .02$, 95% CI [.14, .22]), $\beta = -.07$, $p = .010$. No other disorder cluster differences were significant ($|\beta|$s $\leq .22$, ps $\geq .268$).

Dependent Noninterpersonal Stress. The model for dependent noninterpersonal stress also showed significant heterogeneity, $Q(82) = 296.94$, $p < .001$; $\sigma_{(2)}^2 = .002$, $p < .001$; $\sigma_{(3)}^2 = .004$, $p < .001$; $I_{(1)}^2 = 20.02\%$, $I_{(2)}^2 = 26.75\%$, $I_{(3)}^2 = 53.23\%$, in the association of psychopathology with dependent noninterpersonal stress ($r = .13$, $SE = .02$, 95% CI [.09, .17], $p < .001$). The magnitude of effects on dependent noninterpersonal stress did not differ by internalizing versus externalizing psychopathology, $\beta = .05$, $p = .475$. Similar to findings for dependent interpersonal stress; however, there was a significant effect for psychopathology cluster, such that effects were larger for depression ($r = .15$, $SE = .02$, 95% CI [.11, .19]) than for anxiety ($r = .08$, $SE = .02$, 95% CI [.03, .14]), $\beta = -.08$, $p = .026$. Furthermore, depression also evinced larger effects compared to personality disorders and symptoms ($r = .13$, $SE = .06$, 95% CI [−.14, .38]), $\beta = -.11$, $p = .029$. There were no other significant differences between disorder clusters and depression, $|\beta|$s $\leq .03$, ps $\geq .308$.

In sum, whereas effects did not significantly differ as a function of internalizing versus externalizing psychopathology, depression was associated with larger effects than anxiety across both dependent interpersonal and noninterpersonal stress and was also associated with a larger effect than personality for noninterpersonal stress. All other effects were similar in magnitude.

Aim 3: Does Stress Generation Account for the Chronicity of Various Types of Psychopathology?

We used a multilevel MASEM approach (Wilson et al., 2016) to examine whether stress generation mediates the chronicity of symptoms of psychopathology over time. This approach is the gold standard method for accounting for dependencies among effect sizes extracted from the same studies when modeling structural paths, including indirect effects (Stolwijk et al., 2022). Specifically, we used a two-stage approach in which a random-effects no-intercept three-level model was first estimated to provide a pooled correlation matrix, which the hypothesized correlation model was fit to in Stage 2. Included in analyses were 40 independent samples, from 47 reports, and reporting a total of 318 effect sizes spanning internalizing, externalizing, depression, bipolar, anxiety, personality, and general psychopathology.

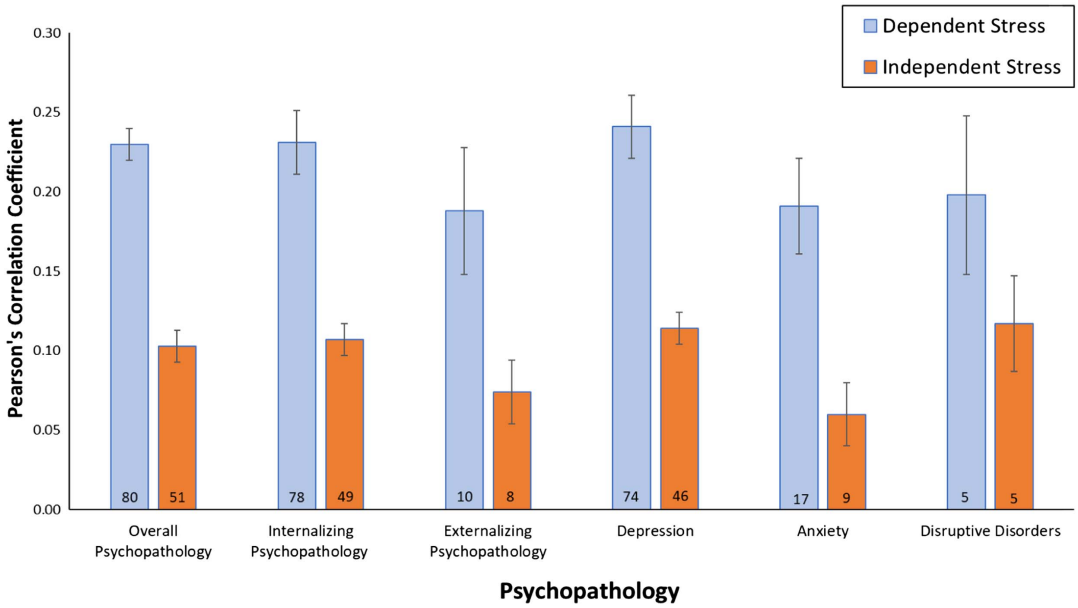
The Stage 1 model displayed significant heterogeneity across levels, with 11.74% of the total variance accounted for by Level 1, 51.26% accounted for by Level 2, and 37.00% accounted for by Level 3. In Stage 2, the hypothesized mediation model was fit to the pooled correlation matrix using WLS estimation. Given that the model was saturated ($df = 0$), fit indices were not examined. We assessed parameter estimates and indirect effects for each of dependent and independent stress on the association of psychopathology symptoms at Time 1 with Time 2. See Figure 6 for a plot of the parameter estimates and their 95% confidence intervals. Both the indirect effects of dependent stress ($\beta = .04$, 95% CI [.03, .05]), and independent stress ($\beta = .01$, 95% CI [.003, .017]) were significant. However, the indirect effect for dependent stress was significantly greater in magnitude than the effect for independent stress ($\beta = .03$, 95% CI [.02, .04]), indicating that dependent stress plays a more central role than independent stress in the maintenance and/or exacerbation of symptoms of psychopathology.

Study Quality

We examined study quality as a moderator of effects of overall psychopathology and types of psychopathology on dependent stress.

Figure 5

Overall Effects From Unconditional Random-Effects Multilevel Models for the Association of Psychopathology With Dependent and Independent Stress



Note. Multilevel meta-analyses indicated significant stress generation effects for overall psychopathology, internalizing, externalizing, depression, and anxiety, as evidenced by significantly larger effects for dependent compared to independent stress, βs ≥ .05, *p*s < .001. Numbers shown within the bars represent the total number of independent study samples included within that category. See the online article for the color version of this figure.

Effects were nonsignificant for overall psychopathology, externalizing psychopathology, and disruptive disorders, *p*s > .098. However, higher study quality was negatively associated with effects of internalizing psychopathology, depression, and anxiety disorders on dependent stress, |β|s ≥ −.05, *p*s < .002. Stress assessment method was integrated into quality scores, such that studies that used interview or hybrid measures received higher scores than those that used checklists. To examine whether stress assessment method accounted for the significant effects of study quality, we conducted models that included *both* quality scores and stress assessment method as moderators. Indeed, after controlling for stress assessment, effects for study quality were no longer significant for internalizing, depression, or anxiety, *p*s ≥ .065.

Publication Bias

We assessed publication bias using four approaches. We first tested publication type (published vs. unpublished) as a moderator of the summary effect of overall psychopathology on each of dependent and independent stress, as well as of the overall effects of each type of psychopathology on dependent and independent stress when there was a sufficient number of both published and unpublished studies. Results are reported for dependent and independent stress in the Supplemental Material. Across all types of psychopathology, effect sizes did not differ by publication status, |β|s ≤ .07, *p*s ≥ .198. Second, we visually inspected standard funnel plots centered around the mean effect for asymmetry. We also inspected contour-enhanced funnel plots, which were centered at 0 (i.e., a null effect), to examine whether there was evidence of the suppression of nonsignificant findings. See Figure 7 for funnel plots for overall psychopathology and Supplemental Material for funnel plots for all other types of psychopathology. There was no clear evidence of asymmetry in standard funnel plots. In contour-enhanced funnel plots, publication bias would be indicated by a relative lack of studies in the white zone of the plot, which represents nonsignificant findings. Given that there were numerous studies reporting nonsignificant findings, including a number of published studies across all funnel plots, there was no evidence of publication bias. Finally, we conducted analyses using two variants of Egger's test that specifically account for dependent effect sizes—the Egger sandwich test, which uses RVE, and the MLMA Egger test. Only the effect of externalizing psychopathology on dependent stress indicated asymmetry (Egger's sandwich test: β = 1.00, *p* = .022; Egger's MLMA test: β = .99, *p* = .021). All other tests yielded nonsignificant slopes |β|s ≤ .93, *p*s ≥ .064, indicating no evidence of selective reporting of effects (see the Supplemental Material).

Discussion

Over the past 3 decades, stress generation theory has informed our understanding of the active role individuals have in shaping their environments and the stressors they experience. Initially advanced to understand depressive recurrence, stress generation theory is fundamental to contemporary models of depression (Hammen, 2005). As a compelling theoretical framework for delineating links between psychopathology and stress, researchers have also examined whether

Table 3

Results of Random-Effects Multilevel Meta-Analyses for Each Type of Psychopathology

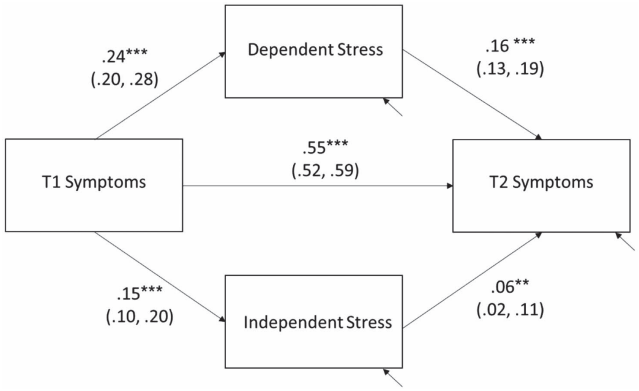
Type of psychopathology	*k* studies	*k* ESs	*r*	[95% CI]	*Q*	Stress generation effect (β)	Significant moderators
Overall psychopathology	80	537	.205***	[.177, .232]	4029.045***	.054**	—
Dependent stress	80	371	.230***	[.202, .257]	3265.126***	—	Age, length of follow-up, psychopathology measure, type of psychopathology
Independent stress	51	166	.103***	[.082, .124]	479.976***	—	—
Internalizing psychopathology	78	355	.207***	[.179, .235]	2378.009***	.069***	—
Dependent stress	78	243	.231***	[.202, .260]	1656.393***	—	Age, length of follow-up, stress assessment method, stress domain
Independent stress	49	112	.107***	[.084, .130]	291.827***	—	—
Externalizing psychopathology	10	46	.160**	[.077, .240]	211.71***	.052*	—
Dependent stress	10	29	.188**	[.093, .279]	172.509***	—	None
Independent stress	8	17	.074*	[.024, .125]	28.275*	—	—
Depression	74	272	.217***	[.186, .248]	1747.403***	.081***	—
Dependent stress	74	183	.241***	[.210, .272]	1170.788***	—	Age, stress assessment method, stress domain
Independent stress	46	89	.114***	[.089, .139]	202.054***	—	—
Anxiety	17	72	.176***	[.109, .241]	560.162***	.049*	—
Dependent stress	17	54	.191***	[.124, .256]	409.459***	—	Length of follow-up, stress assessment method, stress domain
Independent stress	9	18	.060*	[.010, .110]	78.156***	—	—
Disruptive disorders	5	16	.165*	[.060, .266]	69.938***	.076†	—
Dependent stress	5	9	.198*	[.050, .338]	52.605***	—	None
Independent stress	5	7	.117*	[.015, .216]	10.365	—	—

Note. Summary effects are only displayed if effect sizes were available for $k \geq 5$ unique samples. *k* = number of studies or effect sizes. CI = confidence interval; ES = effect size; *r* = Pearson's correlation coefficient; *Q* = *Q* statistic for the heterogeneity of effect sizes. β coefficients are from metaregression models of the association of psychopathology with overall stress, where stressor dependence (dependent vs. independent stress) was examined as a moderator. All significant β coefficients represent moderation by stressor dependence such that the magnitude of effects was greater for dependent than for independent stress, indicative of the stress generation effect. All other moderators were tested only for the association of psychopathology with dependent stress.

* $p < .05$. ** $p < .01$. *** $p < .001$. † $p < .10$.

Figure 6

Multilevel Meta-Analytic Structural Equation Model of the Indirect Effects of Dependent and Independent Stress on Prospective Changes in Symptoms of Psychopathology



Note. CI = confidence interval. The indirect effect of dependent stress (β = .04, 95% CI [.03, .05]) was significantly greater than the indirect effect for independent stress (β = .01, 95% CI [.003, .017]), β = .03, 95% CI [.02, .04], suggesting that dependent stress plays a more central role in the maintenance or exacerbation of symptoms of psychopathology. Parameter estimates are shown with 95% confidence intervals in brackets. T1 = Time 1; T2 = Time 2. ** $p < .01$. *** $p < .001$.

stress generation is relevant to other mental disorders, albeit with inconsistent results (e.g., Hammen & Shih, 2008; Liu & Alloy, 2010). Given the broad implications stress generation theory may have for understanding the course and recurrence of psychopathology, we aimed to advance the stress generation literature by conducting a comprehensive meta-analysis to test the broad applicability of stress generation across psychopathology. The present publicly preregistered study systematically reviewed and meta-analyzed over 30 years of psychopathology and stress generation research. In doing so, we sought to answer three central questions: (1) Does stress generation occur across psychopathology? (2) Do types of psychopathology evince distinct patterns of stress generation, both in terms of the magnitude of effects, the moderators that determine for whom and under what conditions stress generation is most pronounced, and the type of dependent stress generated? And finally, (3) Does stress generation account for the chronicity of various types of psychopathology? The present study used rigorous analytic approaches to test the boundaries of stress generation across psychopathology, including a three-level random-effects meta-analytic approach in combination with an a posteriori RVE correction (Fernández-Castilla et al., 2020; Tipton, 2015) that allowed for the inclusion of multiple effect sizes from each study, and a state-of-the-art two-stage, three-level MASEM (Wilson et al., 2016) that enabled an assessment of a structural path model while accounting for the nested structure of the data. In offering the most comprehensive investigation of psychopathology and stress generation to date, the present study makes several novel contributions. Findings indicate that stress generation occurs across several disorders beyond depression. Specifically, stress generation effects were found for the broader construct of internalizing, externalizing, and anxiety (see Table 3 and Figure 5), although it is important to note the small number of studies in some of these categories. Stress generation also demonstrates a nuanced presentation across types of psychopathology. Critically, results also indicate that psychopathology and stress generation contribute to a vicious cycle of increasing dependent stress and symptomatology that likely plays a central role in the chronicity of psychopathology. Findings and their implications are discussed in detail below.

Does Stress Generation Occur Across Psychopathology?

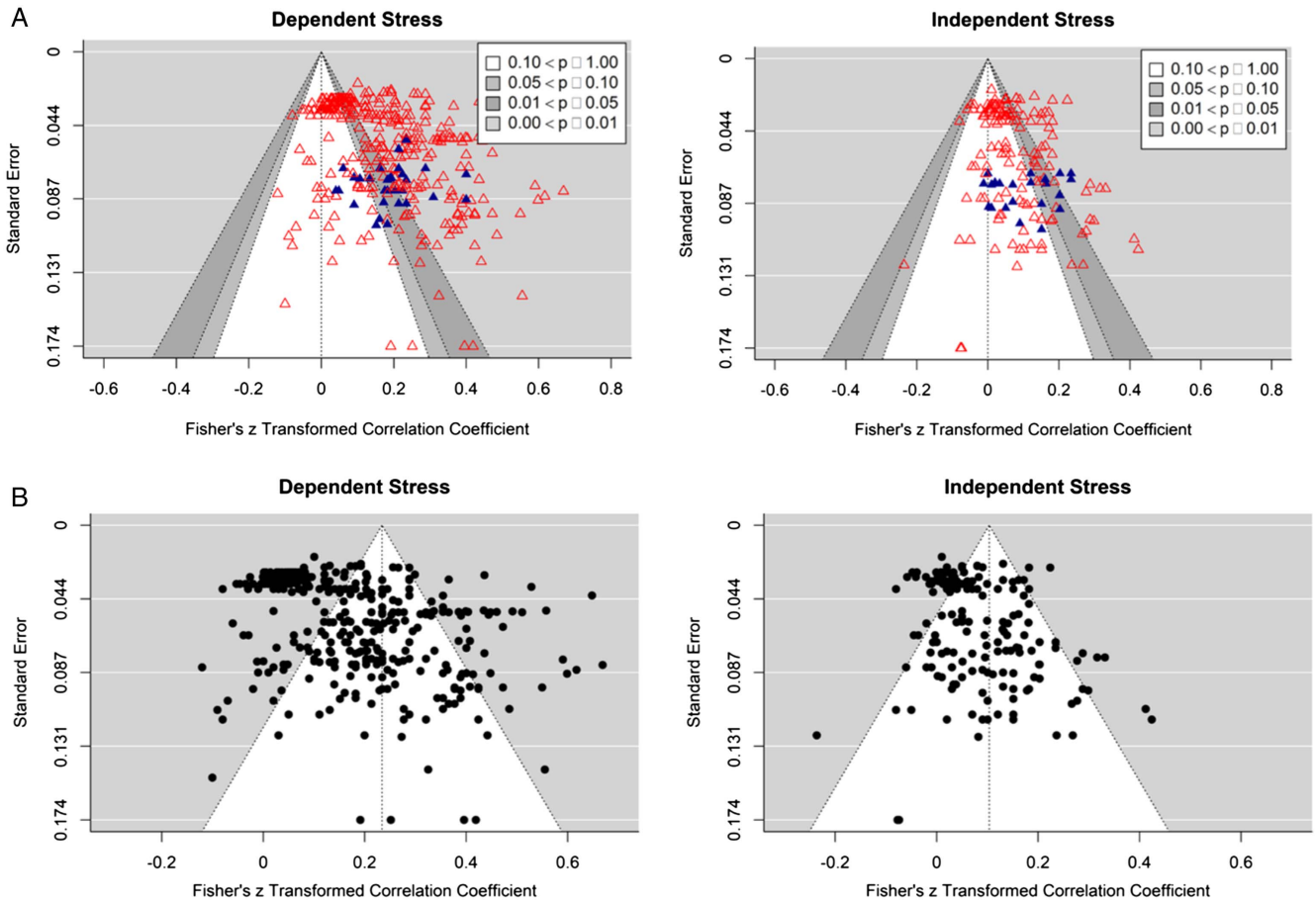
A central aim of the present study was to examine whether there is evidence of stress generation across psychopathology symptoms and disorders. Inconsistent findings in the literature (e.g., Hammen & Shih, 2008; Liu & Alloy, 2010) have made it challenging for researchers to draw firm conclusions as to the ubiquity of the stress generation phenomenon, thereby limiting advancements in our models of psychopathology. Critically, the stress generation hypothesis posits that some individuals contribute to the greater occurrence of dependent stressors relative to their experiences of independent, or fateful, stressors. Thus, to provide a true test of the stress generation hypothesis, it is necessary to compare the magnitude of associations of psychopathology with dependent versus independent stress, the latter of which serves as a control comparison. Results from 537 effect sizes extracted from 80 independent studies that were reported in 95 published and unpublished articles and dissertations revealed a significant moderating effect of stressor dependence (i.e., dependent vs. independent stress) on the longitudinal association of psychopathology with episodic life stress. Consistent with the key tenet of stress generation theory, the association of psychopathology was significantly greater for dependent ($r = .23$) than independent ($r = .10$) stress (see Figure 2). Critically, this finding indicates that psychopathology, defined broadly, prospectively predicts stress generation.

Findings are particularly noteworthy given that they are based on studies that used rigorous methodological approaches—studies were required to employ prospective longitudinal designs, investigator-rated determinations of stressor dependence, and assessment of multiple episodic stressors. Moreover, this overall effect was based on numerous forms of psychopathology, including, but not limited to, depressive, bipolar, personality, anxiety (including trauma-related and obsessive–compulsive), substance use, disruptive, and attention-deficit/hyperactivity symptoms and disorders; however, it is important to note the abundance of effect sizes for depression, which may have augmented effects. It is also interesting to consider other factors beyond the direct effect of psychopathology that may have accounted for the larger association of psychopathology with dependent than independent stress. For example, underlying risk factors for psychopathology (e.g., cognitive styles, interpersonal behaviors) may play a causal role in the generation of stress, and may, at least in part, explain the psychopathology-dependent stress link. See Santee et al. (2023) and Liu et al. (2023) for meta-analyses of risk and protective factors for stress generation.

As expected, significant heterogeneity emerged in the observed effect of overall psychopathology on subsequent dependent stress. We, therefore, sought to examine conceptual and descriptive moderators that influence the strength of the stress generation effect and that define its boundaries. The strength of the prospective association of psychopathology with dependent stress varied as a function of age, length of follow-up, and psychopathology measure

Figure 7

Contour-Enhanced and Standard Funnel Plots of Effect Sizes for the Association of Overall Psychopathology With Dependent and Independent Stress



Note. In Panel A, hollow triangles represent published studies, and solid triangles represent unpublished studies. Effects in the white zone represent statistically nonsignificant findings ($p < .05$), and the contour represent points at which effect sizes reach statistical significance. Contours are centered around the null value. Standard funnel plots are presented in Panel B. See the online article for the color version of this figure.

(see Table 2). Specifically, younger mean sample age at baseline and adolescent/early adult (as compared to adult) samples were associated with larger effects on dependent stress (see Figure 3), as were shorter length of follow-up (see Figure 4) and use of symptom-based (as compared to diagnostic) measures of psychopathology. The finding that effects are larger in magnitude for symptom- compared to diagnostic-based assessments is consistent with evidence that continuous measures of psychopathology have superior psychometric properties compared to discrete measures (Markon et al., 2011). It also supports conclusions that subthreshold symptoms impact functioning (e.g., Haller et al., 2014; Karsten et al., 2013). Importantly, this finding indicates that stress generation is not specific to clinical disorders, but also occurs among nonclinical samples of individuals, many of whom report subthreshold symptom elevations. Given that moderating effects of age and length of follow-up may have been disproportionately influenced by specific types of psychopathology, these effects are described in more detail below in our discussion of specific patterns of moderation across types of psychopathology.

Do Types of Psychopathology Evince Distinct Patterns of Stress Generation?

Whereas findings for overall psychopathology provided a robust assessment of the link between psychopathology and the generation of stress, collapsing across all symptoms and disorders may have obscured important nuances in the phenomenology and boundaries of stress generation. We therefore investigated whether types of psychopathology demonstrate distinct patterns of stress generation. This was achieved by examining similarities and differences in (a) the magnitude of associations with dependent stress, (b) moderators that determine for whom and under what conditions stress generation is most pronounced, and (c) the type of stress generated.

Tests of the Stress Generation Effect for Types of Psychopathology

We examined types of psychopathology both in terms of the broad-based categories of internalizing and externalizing, and by

specific disorder clusters that had sufficient coverage in the literature. The generation of dependent stress did not differ as a function of internalizing versus externalizing psychopathology. In terms of disorder clusters, however, depression exhibited a larger prospective association with dependent stress than did anxiety. No other comparisons with depression were significant, indicating that stress generation effects for personality, substance use, and disruptive symptoms and disorders were similar in magnitude to depression. The smaller prospective association of anxiety with the generation of dependent stress might reflect the possibility that anxiety promotes avoidance and withdrawal away from potentially stressful situations (Meyer & Curry, 2017). In some cases, and for some anxiety disorders (e.g., panic disorder; Conway et al., 2012), this may go so far as not only dampening, but *inhibiting,* stress generation. Unfortunately, we could not investigate differences across specific categories of anxiety disorders because the small number of independent studies investigating each diagnostic cluster precluded a separate analysis. Future research is therefore needed to delineate the magnitude and direction of effects for specific anxiety disorders, and to determine what symptoms (e.g., avoidance) or associated features distinguish the stress generation effect in anxiety from that observed in depression.

It is important to note that we did not include effect sizes in our meta-analysis that were based on comparisons between two diagnostic groups given that these effects do not share the same meaning with the effects included. However, such comparisons provide valuable information about differences in the magnitude of stress generation effects. Though there were not enough independent studies reporting such comparisons to meta-analyze, we provide these effects in the Supplemental Material.

Beyond assessing differences in the magnitude of effects of psychopathology on dependent stress, we also confirmed the stress generation effect for each type of psychopathology. We employed the same rigorous test we had previously applied to overall psychopathology and tested for a significant difference in the association of each type of psychopathology with dependent versus independent stress. As hypothesized, internalizing, externalizing, depression, and anxiety displayed significant stress generation effects, whereby, consistent with stress generation theory, larger associations were observed for dependent compared to independent stress (see Figure 5). The only exception was disruptive disorders, which demonstrated a nonsignificant trend in which effects for dependent stress were only marginally larger than for independent stress. However, given that a relatively fewer number of studies reported on disruptive disorders, future research is needed to continue to characterize the disruptive disorder–dependent stress link.

It is also interesting to consider the ways in which the ubiquity of depression, and comorbidity more generally, may have influenced our results. The high prevalence of depression across mental disorders (D. J. A. Dozois et al., 2020; Rohde et al., 1991) may have amplified psychopathology-dependent stress associations for the broader construct of internalizing or for other types of psychopathology (e.g., externalizing, anxiety, and disruptive disorders). Thus, one possibility is that stress generation may have appeared more transdiagnostic due to the ubiquity of comorbid depression. Other comorbidities may have also had similar effects. Alternatively, other comorbid disorders may have attenuated specific psychopathology-stress associations, which may have resulted in reduced or nonsignificant effect sizes. Given that comorbid mental disorders were not assessed or reported on in a consistent or comparable manner across studies, we were unable to account for comorbidity in our analyses. It is, therefore, critical that future research carefully examines the influence of comorbidity on stress generation across types of psychopathology.

Although past work characterized stress generation as a feature of depression with relatively high specificity (Alloy et al., 2010; Liu & Alloy, 2010), converging evidence across results of the present study indicates that stress generation occurs across several forms of psychopathology and may represent a transdiagnostic phenomenon. The perspective that stress generation is a depression-specific process appears to be largely a historical artifact of the way in which stress generation was first studied and documented. Moreover, a cross-diagnostic conceptualization of stress generation is consistent with the very high levels of comorbidity documented across mental disorders (Kessler et al., 2005; Plana-Ripoll et al., 2019). Indeed, high rates of comorbidity are observed in the vast majority of clinical presentations (Daré et al., 2019; Valderas et al., 2009). Characterizing stress generation as a feature of depression and keeping it siloed within the depression literature is detrimental to theoretical and empirical advancements in other areas of clinical science.

Differences in Moderators of Associations of Psychopathology With Dependent Stress

Demographic, methodological, and descriptive moderators that could elucidate nuances in the phenomenology of stress generation across psychopathological symptoms and disorders were examined (see Table 2). Similar to findings for overall psychopathology, participant age at baseline moderated effects of internalizing and depression on dependent stress. Specifically, larger effects were observed among adolescents/early adults than adults. Furthermore, older mean sample age was linearly associated with smaller effects of depression on dependent stress. Together, this indicates that for internalizing and depression, adolescence and early adulthood are developmental periods associated with greater stress generation than adulthood. Adolescence and early adulthood correspond to periods of transition and upheaval as individuals gain autonomy and independence from their families, form new friendships and romantic relationships, and make major decisions about their education, jobs, and careers, thereby establishing the interpersonal, socioeconomic, geographic, and physical context in which they will engage for years, if not throughout much of adulthood (Meeus, 2016; Wood et al., 2018). The development of these new relationships and responsibilities likely creates abundant opportunities for the generation of stressful life events. However, as individuals become established in their roles, identities, and relationships and achieve developmental milestones in executive functioning and emotion regulation (Gullone et al., 2010; Zimmermann & Iwanski, 2014), their ability to navigate challenging contexts in a manner that reduces the generation of stress may improve. The decline of stress generation effects with age is also consistent with Charles and Carstensen's socioemotional selectivity theory (see Charles & Carstensen, 2014), which posits that with increasing age and a shrinking time horizon, individuals experience motivational shifts toward emotionally meaningful goals, such as experiencing gratifying social interactions with close others. This, in turn, leads older adults to preferentially select themselves into situations that are

likely to enhance experiences of positive emotions, and to mitigate their exposure to potentially distressing events, both of which may translate to reduced stress generation over time. In contrast, no effects of age were observed for anxiety, externalizing, or disruptive disorders, suggesting that for these types of psychopathology, stress generation is more stable across development. It is important to note, however, that we were only able to test the linear effect of age for these disorders given the insufficient number of studies assessing each of the age group categories.

Longer length of follow-up was associated with smaller stress generation effects for internalizing psychopathology and anxiety. That is, as the lag between the assessment of psychopathology and subsequent stress increased, stress generation effects decreased, suggesting that mental disorders and symptoms play a more proximal role in the generation of stress, with effects decaying over time. Given the small magnitude of these effects, it is likely, however, that both state (current symptoms and syndromes) as well as trait (stable underlying) factors contribute to the stress generation phenomenon. An alternative explanation for this finding is that longer follow-ups may have resulted in reduced recall of life events, thereby decreasing the magnitude of effects assessed over longer intervals. However, recall likely had a limited impact on findings given that many effect sizes associated with relatively longer follow-ups were extracted from multiwave studies, in which stress was typically assessed only since the most recent wave of data collection (e.g., Jenness et al., 2019; Kindt et al., 2015). Length of follow-up did not predict effects for depression, externalizing, or disruptive disorders, implying that for these types of psychopathology, more enduring underlying factors such as personality traits, cognitive vulnerabilities, and interpersonal styles may play a more significant role in psychopathology-dependent stress associations. Indeed, this proposition is consistent with evidence we offered in a recent systematic review and meta-analysis on key risk and protective factors (Santee et al., 2023). In sum, although anxiety is associated with a smaller stress generation effect compared to depression, it may also have a more proximal, or direct, impact on the generation of stressors.

Critically, stress assessment method significantly predicted the magnitude of effects for internalizing psychopathology, depression, and anxiety. Checklist indices of stress were associated with notably larger effect sizes than were interview-based measures, whereas hybrid checklist/interview-based measures did not differ from either, perhaps unsurprisingly given their overlap with both other approaches. This moderating effect was specific to internalizing disorders (including depression and anxiety), given that stress generation effects did not differ as a function of stress assessment method for externalizing and disruptive disorders. Stress scores, as determined by checklists of participants with internalizing psychopathology, may be unduly influenced by participants' cognitive biases which, compared to those with externalizing forms of psychopathology, may be particularly negatively biased (Harkness & Monroe, 2016). For example, prior research comparing scores obtained using checklist versus interview-based measures of stress have documented that individuals with depression tend to interpret, recall, and report life events as more negative on checklists (Simons et al., 1993). In contrast to checklists, interviews allow the investigator to provide clarifications, examples, and additional probes to ensure that a life event has in fact occurred, confirm that events meet investigator-defined criteria, and determine stressor severity based on contextual information about the circumstances and objective impact of events (Harkness & Monroe, 2016; McQuaid et al., 2000). The current findings suggest that among individuals with internalizing psychopathology, checklist indices may be particularly prone to overinflated stress scores. Interview-based measures, which are generally recommended as the gold standard method of assessing life stress (Harkness, 2023), are particularly important in this context.

Similar to findings for stress assessment method, study quality was only a significant moderator of effects for internalizing psychopathology, depression, and anxiety disorders. However, we found that after accounting for stress assessment method—one of our indicators of study quality—study quality was no longer a significant moderator. These findings underscore the importance of stress assessment in stress generation research, as well as its particular importance for internalizing psychopathology.

Given prior evidence that internalizing and externalizing disorders may be differentially associated with the generation of dependent interpersonal versus noninterpersonal stress, we also examined dependent stress domain as a moderator. As predicted, stress generation effects were larger for dependent interpersonal compared to dependent noninterpersonal stress for each of internalizing psychopathology, depression, and anxiety. This is consistent with past research documenting pronounced effects for interpersonal stress generation among individuals with depression (e.g., Meiser & Esser, 2019; Rudolph, 2008), anxiety (see Meyer & Curry, 2017), or internalizing symptoms more generally (e.g., Conway et al., 2012). Thus, among those with internalizing disorders, stress generation may be predominantly an interpersonal phenomenon, concerning stressors such as arguments, breakups, and rejection. Interpersonal impairments are well-documented among internalizing disorders (e.g., poor social skills, social cognitive dysfunction, maladaptive interpersonal behaviors; Evraire & Dozois, 2011; Rnic et al., 2018) and could account for relative specificity of the stress generation phenomenon in internalizing to the interpersonal sphere. Unexpectedly, effects for externalizing and disruptive disorders, on the other hand, did not vary as a function of dependent stress domain, indicating that interpersonal areas of life were as affected as noninterpersonal domains.

Contrary to hypotheses, we also did not find evidence for gender differences. This finding is counter to prior narrative reviews that have suggested that stress generation tends to be more pervasive among girls and women (e.g., Liu & Alloy, 2010). Since the vast majority of studies reported effect sizes for the total sample rather than providing effects stratified by gender, more fine-grained subgroup analyses were not possible. Thus, whereas the present study suggests that the psychopathology–dependent stress link does not differ as a function of gender, these findings are limited by their reliance on a relatively coarse indicator of gender (i.e., percentage of women/girls in each sample). There was also no evidence of moderation by race, psychopathology measure (symptom vs. diagnosis), diagnostic timeframe (i.e., current, remitted, or lifetime diagnosis), continent where the study was conducted, or publication/completion year. That psychopathology was prospectively associated with stress generation across a range of demographic variables, as well as across the year and location in which the study was conducted, speaks to the universality of the stress generation phenomenon. The null finding for publication year indicates that this literature does not evince the "decline effect," in which the statistical significance of effects in a given field declines over time

(see Schooler, 2011), or the "law of initial results," in which a highly cited initial finding is followed by contradictory results (Ioannidis, 2005a, 2005b). Moreover, the fact that there have not been significant changes in effect sizes for stress generation over the past 3 decades suggests that the stress generation effect is replicable, enduring, and robust to psychology's "replication crisis" (see Shrout & Rodgers, 2018).

Differences in Type of Stress Generated

Finally, we investigated whether types of psychopathology differed in the magnitude with which they were associated with the generation of dependent interpersonal versus noninterpersonal stress. There was no difference in the magnitude of effects for internalizing and externalizing on either dependent interpersonal or noninterpersonal stress. In assessing differences across disorder clusters, depression exhibited larger effects than anxiety on both dependent interpersonal and noninterpersonal stress, suggesting that depression is associated with more pronounced stress generation effects, irrespective of the type of stress generated. Depression was also associated with larger effects than personality psychopathology for the generation of dependent noninterpersonal stress.

Thus, when examining differences in the generation of interpersonal and noninterpersonal stress *within* disorders, as described above, internalizing disorders, including both depression and anxiety, were associated with greater generation of interpersonal as opposed to noninterpersonal stress. However, when comparing the generation of interpersonal and noninterpersonal stress *across* disorders, there were no differences in internalizing versus externalizing. Findings indicate that depression was associated with larger stress generation effects than anxiety across both interpersonal and noninterpersonal dependent stress, and a larger effect than personality psychopathology for noninterpersonal stress. These results suggest that, although stress generation occurs across several types of psychopathology, depression may have a particularly robust effect on the generation of dependent stress. This finding aligns with prior work documenting uniquely robust effects for depression. For example, Conway et al. (2012) found that major depressive disorder incrementally predicted dependent interpersonal stress above and beyond the influence of internalizing. Research comparing depression to other types of psychopathology has also reported particularly large stress generation effects for depression (e.g., bipolar disorder; Hammen, 1991; anxiety and conduct disorder; Wingate & Joiner, 2004). It is possible that symptoms or features associated with depression may be particularly conducive to the generation of stressful life events compared to anxiety or personality, though future cross-diagnostic research is needed to elucidate these factors.

Does Stress Generation Account for the Chronicity of Various Types of Psychopathology?

A multilevel meta-analytic structural equation model indicated that, while both dependent and independent stress mediated positive associations of symptoms of psychopathology with later symptoms, the indirect effect of dependent stress was significantly greater than that for independent stress (see Figure 6). Thus, while both dependent and independent stress contribute to the maintenance or exacerbation of symptoms of psychopathology, dependent stress may play a particularly prominent role. Findings align with past research that has documented that dependent stressors are particularly depressogenic (Kendler et al., 1999; Kendler & Gardner, 2010) and with prior studies that have found evidence of transactional, mediating associations of dependent stress with depression (e.g., Hankin et al., 2005; Rudolph et al., 2009). Our meta-analysis extended these findings to psychopathology more broadly. However, it is important to take into account that other factors beyond stress generation (e.g., cognitive schemas, D. J. Dozois & Rnic, 2015) certainly contribute to chronicity, and that it is unlikely that stress generation fully accounts for symptom stability, particularly given the still significant direct path of baseline symptoms to later symptoms after accounting for indirect effects, $\beta = .55$, $p < .001$. This study was the first to test and find evidence for the role of stress generation in contributing to psychopathology chronicity across disorders, thereby highlighting the broad implications of the stress generation phenomenon and the important need for it to be incorporated into models of mental disorder.

It is important to note that our mediation models may oversimplify the dynamic relationship between stress and psychopathology. For example, research in depression has previously documented that stress generation may be progressive with repeated episodes. Harkness et al. (1999) found that recurrent episodes of depression are associated with greater dependent events in the 12 months preceding an index episode than first onset episodes of depression. In combination with stress sensitization—the process through which less stress is needed to trigger each successive depressive episode (Stroud et al., 2011)—stress generation and sensitization may together produce a particularly vicious cycle of stress and psychopathology from which it may be increasingly difficult for individuals to extricate. Future research is needed to examine to what degree stress generation may be progressive for other forms of psychopathology, as well as to investigate stress generation in conjunction with stress sensitization. Likewise, quantitative modeling of stress, coping, and psychopathology based on nonlinear systems theory (Levy et al., 2012; R. W. J. Neufeld, 1999; Nicholson & Neufeld, 1992; see R. W. Neufeld & Grant, 2020) enables researchers to examine complex associations between stress and related phenomena over time. Integrating this approach into the stress generation literature may allow researchers to better capture rich longitudinal dynamics between stress and psychopathology.

Prospective Associations of Psychopathology With Independent Stress

Intriguingly, we found small, yet significant, prospective associations of psychopathology with elevated independent stress. This finding replicated across analyses for overall psychopathology and psychopathology types, except for disruptive disorders. Given that independent life events are fateful by definition, this finding may appear counterintuitive. There are, however, a number of processes that may account for significant associations of psychopathology with independent stress.

First, many independent stressors may be reflective of ongoing challenging environmental contexts that may have predisposed an individual to develop psychopathology in the first place, consistent with the harsh environment hypothesis (Ellenbogen & Hodgins, 2004). Examples include living in a disadvantaged neighborhood with high crime rates and poor educational and occupational

opportunities, or living with parents or other family members with high behavioral dysregulation. Second, individuals with psychopathology may be more likely to actively select themselves into challenging or entrapping contexts where the occurrence of independent stressors is higher (Hammen et al., 2011; Keenan-Miller et al., 2007). For example, patterns of assortative mating and peer selection are well-documented among individuals with psychopathology (Hammen, 2006; Harkness & Washburn, 2016), who tend to self-select themselves into relationships where they are more likely to be exposed to elevated stressors. For better or worse, selection of family, living, and working environments will affect most aspects of that individual's life for years to come (Hammen, 2009), cascading into an ongoing stream of relationships, socioeconomic contexts, and physical settings that may be more or less stressful (Hammen & Shih, 2010). Thus, the occurrence of many independent events are likely shaped by the individual's distal choices, potentially representing a process of *chronic stress generation* (Hammen, 2020). This proposition implies that the boundary between dependent and independent stress is less distinct than is frequently represented in the stress literature. It is therefore important to acknowledge that there is a great deal of ambiguity in distinguishing between dependent and independent stress, as well as between other stress-related phenomena (e.g., episodic and chronic stress) when conducting stress research.

Third, individuals with psychopathology may be less skilled in actively *avoiding* the occurrence of independent stressors as a result of factors such as poor decisional control (Averill, 1973; Morrison et al., 1988; Shanahan & Neufeld, 2010). That is, individuals with psychopathology may be less planful or proactive than healthy individuals in avoiding daily hassles (e.g., avoiding a traffic jam by planning a route ahead of time) and life events (e.g., reducing the impact of a health issue by attending regular screening appointments; avoiding major financial losses in an economic downturn by maintaining a diversified financial portfolio). Fourth, even when a life event is truly independent, measures using a contextual threat rating approach (i.e., one that considers both the frequency and contextual severity of events in computing total stress scores) may result in higher independent stress ratings among individuals with mental disorders due to psychopathology-related factors. For example, the loss of a close friend would be rated more severe for an individual with psychopathology who is highly isolated as a result of factors such as avoidance or poor social skills.

It is important to note that summary model estimates for independent stress were small (r = .06–.12). Meta-analyses tend to be well-powered, thereby enabling weak effects to emerge as statistically significant. Critically, however, we consistently found stronger effects for dependent than independent stress (with the exception of disruptive disorders), consistent with the central proposition of stress generation theory. Findings underscore the importance of examining both dependent and independent stress in order to assess the relative association of psychopathology with each. Without testing effects for independent stress, researchers are not able to rule out a general stress exposure model. Given that 100% of studies included in our quantitative synthesis examined dependent stress, whereas only 63% reported associations for independent stress, it is critical that future research investigates and reports effects for independent stress, thereby providing a more rigorous test of stress generation theory.

Strengths of the Current Review

This study has several strengths that bolster the robustness of our findings and the strength with which we draw our conclusions. Initially two independent research teams, we initiated a collaboration that resulted in an extraordinarily extensive review of the literature, comprising two independent systematic literature reviews. Comparisons of the reports included in our quantitative synthesis with the most recent systematic reviews indicate that stress generation continues to be a proliferative area of inquiry, with most reports (85%) not included in the most recent overlapping systematic reviews (Liu & Alloy, 2010; Meyer & Curry, 2017). Our inclusion of only studies that met our methodological requirements (i.e., prospective longitudinal design, investigator-determined dependence, assessment of episodic stressors in which dependence can be more accurately determined than for chronic stress) ensured findings were based on data meeting quality thresholds and that effect sizes would share a common meaning. Furthermore, the synthesis of our two teams' data sets resulted in all data and effect sizes being not only double-coded within a team, but in many cases triple- and quadruple-coded across both teams. These considerable efforts resulted in high coding fidelity.

In terms of our analytic approach, we used cutting-edge statistical methods that allowed us to answer novel questions about the ubiquity of stress generation across psychopathology, moderators that influenced the strength of effects, and the mediating role of generated stress on symptom chronicity. Estimation of multilevel models with an a posteriori RVE correction enabled us to include multiple effect sizes from each study while ensuring unbiased parameter estimates. These multiple nested effects included effect sizes for each type of psychopathology reported in a given study, for multiple measures of psychopathology (symptom and diagnostic), for multiple stress outcomes (e.g., dependent, independent), and for multiple waves of data collection, when available. Moreover, this multilevel approach allowed us to directly test the key proposition of stress generation theory by comparing effects for both dependent and independent stress in the same model—a major strength. By enabling us to retain all desired effect sizes, we were also able to examine broad categories such as overall psychopathology, internalizing, and externalizing, which necessitated the inclusion of sometimes several effect sizes from a given study. Finally, the use of a three-level, two-stage MASEM incorporated benefits of multilevel modeling while also employing the state-of-the-art two-stage MASEM approach (Wilson et al., 2016), which together have recently been recommended as the gold standard method for handling effect size dependencies in a MASEM context (Jak & Cheung, 2020; Stolwijk et al., 2022). Finally, we integrated multiple tests for publication bias, including new approaches that specifically handle dependent effect sizes (Rodgers & Pustejovsky, 2020). Across findings, only the association of externalizing psychopathology with dependent stress indicated some evidence of funnel plot asymmetry. Notwithstanding the fact that asymmetry does not necessarily indicate selective reporting (Rodgers & Pustejovsky, 2020), future research examining the stress generation effect for externalizing psychopathology is needed.

Limitations and Future Directions

Findings should be interpreted in the context of limitations of the current meta-analysis and the literature on which findings are based.

Our restriction to studies written in English likely limited our inclusion of research conducted in non-Westernized samples and thus reduced the generalizability of findings. Although study location (North America vs. outside North America) was not a significant moderator, this represents a very coarse examination of possible cross-cultural differences. As evidenced in Table 1, the vast majority of included studies examined Westernized samples (North America: $k = 63$; Europe: $k = 11$; Australia: $k = 3$). Very few were conducted in Asia ($k = 3$), and none were conducted in the Middle East, South America, or Africa. An overreliance of psychological research on Western, Educated, Industrialized, Rich, and Democratic (WEIRD) societies represents a major problem for the discipline given that WEIRD samples are a particularly atypical subpopulation across numerous domains (e.g., moral reasoning, self-concepts; Henrich et al., 2010). We therefore cannot make universal generalizations of our findings to non-WEIRD populations, particularly given documented cross-cultural differences in mental disorder symptom profiles (Juhasz et al., 2012; Kohrt et al., 2014) and societal perspectives on psychopathology (Abdullah & Brown, 2011). Such differences in clinical presentations and cultural views on mental health may translate to differences in the psychopathology-stress generation link.

Another limitation is that we were unable to examine the role of comorbidity in our analyses given that studies were inconsistent in their assessment and report of comorbid psychopathology. Detailed assessment of comorbid disorders and their possible influence on stress generation effects therefore represents a key future direction. Similarly, given that few articles provided effect sizes stratified by gender, we relied on coarser, sample-wide estimates in our moderator tests for gender. Our synthesis of the stress generation literature also highlighted several understudied disorders and populations that may have limited the generalizability of findings to some groups. As exemplified by the relative number of independent studies that examined internalizing ($k = 78$) versus externalizing ($k = 10$) symptoms and disorders, much more work has been done on internalizing, and in depression specifically, than in other areas. We therefore were not able to examine overall effects and moderators for a number of disorder clusters that lacked sufficient coverage in the literature, such as personality, substance use disorders, and bipolar disorders, among others. Furthermore, with the majority of research being conducted among young samples of participants ($M = 21.95$, $SD = 12.32$, range = 9–61 years) and relatively more studies being conducted with adolescent/early adult samples ($k = 45$) as opposed to adults ($k = 21$) or children ($k = 14$), our ability to generalize findings to young children or to older adult populations was limited. Finally, we were unable to test finer grained differences across different gender (e.g., trans, nonbinary) and racial or ethnic groups, as we were limited by the information provided in reports that could be quantified across the literature. However, with data sets increasingly being made available as part of the open science movement, future investigations using raw data may allow for more detailed analyses of specific gender, racial, and cultural groups.

Results from this study highlight several additional key directions for future work. Given the finding that dependent stress mediates the chronicity of symptoms of psychopathology, future work examining changes in diagnostic status over time is needed to assess whether stress generation also mediates episode recurrence or episode/disorder duration. While some work in this area has been conducted for depression (Starr et al., 2012), research is needed for other forms of psychopathology. There is also a need for work that establishes the specific clinical factors most closely associated with stress generation beyond symptom severity or presence of a diagnosis. These could include symptom profiles, number of prior episodes, age of first onset, and episode or disorder duration. As noted above, we were limited in our ability to test the influence of diagnostic timeframe, and this remains an area in need of further investigation.

Practical Implications and Conclusions

Our findings underscore the need for researchers to integrate stress generation theory into conceptual and empirical models of disorders beyond only depression. Given that stress generation theory posits that individuals actively generate stressors, the current findings also have exciting implications for intervention models. Interventions could be adapted to explicitly assess and target stress generation as a way to reduce symptoms, prevent recurrence, and extricate individuals from a vicious cycle of increasing symptoms and stress (see Dobson et al., 2014). Given that stress generation occurs across disorders and symptoms of psychopathology, it may be a particularly attractive target for transdiagnostic intervention protocols aimed at treating individuals with any of a number of disorders (e.g., Barlow et al., 2020). Integrating stress generation into models of diverse disorders and interventions will be important for advancing our understanding and treatment of various forms of psychopathology. These efforts will be highly relevant for psychologists, psychiatrists, social workers, educators, and others who work with individuals with mental disorders.

In examining over 30 years of psychopathology and stress generation research, the current systematic review and meta-analysis offers the most comprehensive and robust test of stress generation and psychopathology to date. Our collaborative team approach resulted in an exceptionally comprehensive systematic literature review, and our state-of-the-art analytic approaches enabled us to address central questions that a mixed literature had been unable to firmly answer. Findings revealed a significant stress generation effect for psychopathology, defined broadly, as well as for more specific types of psychopathology. Prospective associations of psychopathology with dependent stress were small-to-moderate, and various psychopathology clusters evinced distinct patterns in terms of the strength of effects, moderators that determined for whom and under what conditions stress generation was most pronounced, and type of stress generated. Critically, stress generation accounted, at least in part, for the maintenance of symptoms over time, highlighting the centrality of stress generation for understanding the chronicity of psychopathology. Findings offer a valuable opportunity for clinical scientists to advance theoretical models, empirical investigations, and applied interventions across diverse mental disorders.

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