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A Systematic Review and Bayesian Meta-Analysis of 30 Years of Stress Generation Research: Clinical, Psychological, and Sociodemographic Risk and Protective Factors for Prospective Negative Life Events

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Stress generation posits that (a) individuals at-risk for psychopathology may inadvertently experience higher rates of prospective dependent stress (i.e., stressors that are in part influenced by their thoughts and behaviors) but not independent stress (i.e., stressors occurring outside their influence), and (b) this elevated dependent stress, in some measure, is what places these individuals at-risk for future psychopathology. In recognition of 30 years of stress generation research, we conducted a systematic review and meta-analysis using frequentist and Bayesian approaches (102 articles with 104 eligible studies, $N = 31{,}541$). Generally strong support was found for psychopathology predicting dependent stress (e.g., $ds_{\text{Overall psychopathology}} = 0.36–0.52$, $BF_{10} = 946.00$ to 4.65×10^{18}). Moderator analyses for dependent stress revealed larger effects for briefer assessments periods, shorter follow-ups, and self-report measures than for interviews. Among risk factors, depressogenic cognitive styles ($ds = .26–.50$, $BF_{10} = 47.50$ to 1.00×10^{5}) and general interpersonal vulnerability ($ds = .26–.44$, $BF_{10} = 2.72$ to 2708.00) received the strongest support as stress generation mechanisms, and current evidence is modest for protective factors predicting dependent stress. Overall, larger effects were generally found for prospective prediction of dependent stress than independent stress. Evaluations of mediation in the research literature were relatively few, limiting the current review to qualitative analysis of the mediation component of stress generation. General support was found, however, for dependent stress as a mediator for psychopathology and associated risk factors in relation to subsequent psychopathology. The current review ends with recommendations for future research and integration of stress generation within minority stress frameworks.

Public Significance Statement

Stress generation is the process by which individuals' characteristics, behaviors, and psychopathology inadvertently generate stress in their lives. This systematic review and meta-analysis of 102 studies provides a comprehensive evaluation on the prospective association between (a) psychopathology, (b) biopsychosocial risk factors, (c) protective factors, and (d) sociodemographic characteristics in relation to dependent and independent stress. We found robust effects for associations between overall dependent stress and (a) overall psychopathology, particularly depression, and (b) cognitive and interpersonal vulnerabilities. Some supportive evidence was found, however, for small associations with independent stress. In short, there is robust evidence that psychopathology predicts future dependent stressors and that these stressors may in turn lead to further psychopathology. Prevention and intervention efforts aimed at addressing and preventing dependent stressors may be critical to reduce psychopathology. Findings further our understanding of stress generation in the development, maintenance, and recurrence of mental health problems across development, with key future research directions discussed.

Keywords: depression, life stress, meta-analysis, stress generation

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continued

Since the publication 55 years ago of Holmes and Rahe's (1967) life stress measure, the Schedule of Recent Experiences, a substantial and sustained body of research has emerged devoted to evaluating life stress[1] in relation to psychological and health-related outcomes, including psychological well-being. Just as the measurement of life stress has developed considerably since the introduction of this initial measure, so too have the intervening years seen the development of important theoretical models characterizing the nature of stressful life events within the individual's environment and psychopathology.

One of the most prominent theoretical models introduced over the last several decades is the stress generation hypothesis (Hammen, 1991). This conceptual model is notable for articulating a pathway between past and future psychopathology through intervening life stress. It has received considerable empirical interest. Reflective of this, in the 30 years since the publication of Hammen's (1991) initial article introducing the concept of stress generation, there have been six systematic and conceptual reviews devoted entirely to different elements of this topic (Bahji et al., 2021; Hammen, 2006, 2020; Liu, 2013; Liu & Alloy, 2010; A. E. Meyer & Curry, 2017; Rnic et al., 2023; Santee et al., 2023). One of these articles presented a systematic review of the entire stress generation literature (Liu & Alloy, 2010), and the rest have been more circumscribed in their focus, that is, systematic reviews of stress generation in the context of anxiety (A. E. Meyer & Curry, 2017) or genetics research (Bahji et al., 2021), or provided background on the development of and/or conceptual elaborations of stress generation with a focus on depression (Hammen, 2006, 2020; Liu, 2013). The broadest of the five prior systematic reviews (Liu & Alloy, 2010) found in 57 studies support for depression and related constructs (e.g., other psychopathology, cognitive factors, personality, and interpersonal factors) in association with stress generation. Bahji et al. (2021) identified seven studies examining genetic markers of stress generation and found that although genetic variation in serotonin, the hypothalamic-pituitary-adrenal axis, and oxytocin systems did not have direct effects on stress generation, they did serve as moderators of it.[2] A. E. Meyer and Curry (2017) identified 41 studies of anxiety and broadly related constructs (e.g., neuroticism) in relation to stress generation and found that anxiety, as a general construct but not anxiety-specific physiological hyperarousal, was associated with stress generation. Rnic et al. (2023) focused on studies of psychopathology in relation to stress generation, finding support for the transdiagnostic relevance of this phenomenon in 95 studies. Finally, Santee et al. (2023) conducted a meta-analysis of 70 studies on risk and protective factors for stress generation, finding support for a broad band of risk factors as predictors of stress generation.

The current review aims to build on these prior reviews in several notable ways. First, it provides the first Bayesian meta-analysis and most comprehensive review to date of stress generation research. More specifically, given the considerable corpus of research on this topic, the current effort aims to provide a systematic review of all studies that have been published in the 30 years since the introduction of the stress generation hypothesis (Hammen, 1991). It thus builds on two recent meta-analyses that examined psychopathology and nonpsychiatric predictors, respectively, of stress generation (Rnic et al., 2023; Santee et al., 2023), providing a more complete picture of the state of this field of research. Not only does the current review provide a substantive update to the first systematic review—a narrative review—of the entire stress generation literature (Liu & Alloy, 2010), but it does so using a meta-analytical approach, thereby quantifying the strength of the empirical support for different components of the stress generation hypothesis. Of critical importance, it incorporates Bayesian meta-analysis, which, as detailed below, is necessary for adequately and fully evaluating the stress generation hypothesis. In this manner, it extends the findings of prior stress generation reviews relying on frequentist meta-analysis (Rnic et al., 2023; Santee et al., 2023). The current meta-analytic approach also allows for quantitative evaluations of moderators of the strength of stress generation effects in ways that are not possible with narrative reviews and allows for comparison across psychopathology and risk and protective factors. Additionally, given the substantial growth in stress generation research since our prior broad systematic review (Liu & Alloy, 2010), the coverage of candidate stress generation predictors in the current review is notably more comprehensive than was possible in our prior review (e.g., we are now able to conduct evaluations at a greater level of resolution in terms of specific forms of psychopathology, cognitive and psychological factors, and life stress, and we are able to expand evaluations of sociodemographic

[1] Although subjective and physiological experiences of stress are an important construct in their own right (Park, 2010), life stress, as conceptualized within the stress generation hypothesis (Hammen, 1991) and as previously defined by G. W. Brown & Harris (1978), are stressful life events that occur within the individual's external environment. This definitional approach separates out individual differences in subjective and physiological stress response to life events, which are viewed instead as the product of these stressors and the individual's preexisting vulnerabilities (i.e., a diathesis–stress interaction; for discussions of this issue, see Hammen, 2005; Harkness & Monroe, 2016; Monroe & Harkness, 2005).

[2] Although this review is important for providing a comprehensive review of biological predictors of stress generation, it included four cross-sectional studies, which, for reasons articulated below, risks confounding stress exposure with stress generation.

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characteristics beyond gender identity[3]). Finally, and as will be detailed below, this review aims to advance novel considerations of stress generation that we hope may lead to innovative growth in the field.

Contextualizing the Study of Life Stress and Psychopathology: Stress Exposure and Diathesis–Stress

To allow for a proper evaluation of the stress generation hypothesis, particularly its historical significance and underlying rationale, it is necessary to contextualize it within the broader study of life stress and psychopathology. We therefore first briefly describe early research on stress and psychological well-being, with emphasis on the stress exposure model and its subsequent diathesis–stress elaborations.

The earliest conceptual model of life stress in relation to psychopathology is the stress exposure model, which posits that greater experiences of life stress (e.g., severity and frequency) place the individual at increased risk for subsequent negative mental health outcomes. Since its early emergence, this conceptual model of life stress has received further elaborations, particularly in the form of diathesis–stress models (i.e., the notion that life stress interacts with preexisting vulnerabilities to confer increased risk for psychopathology), which are the basis of several psychological theories of psychopathology (e.g., Abramson et al., 1989; Beck, 1967, 1987; S. L. Johnson & Roberts, 1995; Monroe & Simons, 1991; Nock, 2010; Nolen-Hoeksema, 1991; Walker et al., 2008). These stress exposure and diathesis–stress models have received considerable empirical support (Beards et al., 2013; Hyman & Sinha, 2009; Klauke et al., 2010), particularly in the case of depression (Hammen, 2018; Mazure, 1998; Paykel, 2003).

Several aspects of stress exposure and diathesis–stress models of psychopathology are important to mention as context for the stress generation hypothesis. First, early conceptualizations of life stress were informed by the prevailing view that individuals have little influence in shaping events within their lives relevant to psychological well-being (Hammen, 2006). Second, implicit in these early views of the relation between life stress and psychopathology is the assumption that this relationship is unidirectional in nature, with life stress leading to negative mental health outcomes. Third, these views collectively led, in part, to the prevailing focus on independent stress (i.e., "fateful" or stressful life events that occur outside the control of the individual, such as the death of a loved one) in empirical evaluations of life stress in relation to psychopathology. Traditionally, the focus on independent stress resulted in the exclusion of dependent stress (i.e., stressful life events that are at least in part influenced by the individual's behavior, such as the dissolution of a romantic relationship). Any association between certain disorders and dependent stress was largely interpreted as a potential manifestation and confound of these disorders (Cui & Vaillant, 1997; Hammen, 1991, 1992; Liu & Alloy, 2010). Thus, it was viewed as necessary to exclude from analysis or statistically control for dependent stress rigorously to test whether life stress (here conceptualized as independent stress) may confer risk for subsequent psychopathology.

Stress Generation Hypothesis

The stress generation hypothesis was first introduced 30 years ago (Hammen, 1991), originally to provide a theoretical account of depression chronicity. The stress generation hypothesis has expanded considerably from its original formulation, as will be detailed below, but as initially conceived, it consisted of three central predictions: (a) Individuals with depression experience significantly higher levels of prospectively occurring dependent stress, particularly within interpersonal domains;[4] (b) contrastingly, individuals with depression do not differ from others in their experience of prospectively occurring independent stress;[5] and (c) the higher levels of prospective dependent stress are a mechanism underlying risk for future depression, mediating the association between past depression and its subsequent recurrence.

It is important to note briefly here that stress generation should not be taken to imply blame or otherwise hold the individual responsible for the challenges with dependent stress they experience (Hammen, 2006). Rather, the stress generation hypothesis offers an explanation for how individuals may inadvertently maintain or contribute to the emergence of depressive symptoms by engaging in behaviors that confirm negative cognitive styles or unintentionally diminish social support and resources, thereby increasing the risk of the first onset or future recurrence of depression (Joiner, 2000). It also recognizes that symptoms of psychopathology are inherently stressful and may impair or overwhelm the individual's internal resources for navigating and avoiding potentially emerging stressors.

In challenging existing perspectives at the time on the relation between life stress and depression, stress generation drew on the emerging view that rather than being recipients of events occurring within their environment, individuals have agency in shaping the world around them and, in some measure, choosing the environments they inhabit (Bandura, 1982; Hammen, 2006; Mischel, 1973). Additionally, rather than being defined by a unidirectional relationship, life stress and depression were proposed to share a complex reciprocal or transactional relationship. Furthermore, rather than having an exclusive focus on independent stress and viewing dependent stress as a methodological confound to be statistically controlled or excluded entirely from analysis, stress generation gave prominence to understanding the nature of dependent stress. Importantly, others have observed dependent stress to occur in the context of depression, and some have recognized the consequent importance of dependent stress (Miller et al., 1986). The stress generation hypothesis, however, does not simply describe how depression may be related to future life stress. Instead, it frames dependent stress as a mediational pathway in a coherent conceptual model addressing an enduring core clinical question: Why does depression often lead to its future recurrence? Thus, stress generation

[3] The term gender identity describes a broad range of participant-reported gender, whereas sex assigned at birth is typically treated as a binary variable (male vs. female). A limitation within the stress generation literature is that most studies have conflated gender identity with sex assigned at birth and have taken a binary approach to operationalizing gender, despite the wider range of gender identities (e.g., transgender, genderfluid, nonbinary) that exist within the general population. Gender identity within the current review is therefore limited to this binary operationalization of gender in the stress generation literature.

[4] Interpersonal stress is defined as negative life events that occur within interpersonal contexts, including but not limited to family, peer, and romantic relationships.

[5] The implicit underlying logic is that if independent events occur outside the individual's influence, nothing about the individual should be prospectively associated with the occurrence of these events, and thus they are just as likely to occur prospectively for an individual with depression as they are with any other person.

may be a factor in the recurrence of psychopathology (Hammen, 2006), potentially providing insight into one key mechanism to be targeted within treatment and prevention programs.

This last point warrants further consideration because it cannot be automatically assumed that the mediational component of the stress generation hypothesis would hold based solely on empirical support that individuals with depression have higher levels of prospective dependent stress (i.e., the first prediction of the stress generation hypothesis). If depressed individuals also experience higher prospective rates of independent stress (i.e., if the second prediction of the stress generation hypothesis is not supported) in a manner consistent with the harsh environment hypothesis (Grandin et al., 2007), it may be that greater prospective environmental exposure to independent stress is the constant that places depressed individuals at-risk for future depression. Further reason for direct evaluation of the mediational component of the stress generation hypothesis is the possibility that the greater dependent stress prospectively experienced by depressed individuals may not be sufficient in magnitude or duration (i.e., stress generation diminishes too quickly following resolution of a depressive state or episode) to elicit a depressive response.

Conceptual Extensions of the Stress Generation Hypothesis

Although stress generation was originally conceived as occurring within the context of depression, it has since been expanded in several significant ways, each with its own implications: (a) Stress generation may be a transdiagnostic phenomenon; (b) in the case of depression, it may occur outside of depressive episodes (e.g., during periods of remission), and by extension, acute experiences of psychopathology more generally; and (c) risk factors for psychopathology may be potent drivers of stress generation, perhaps even more so than the psychopathology with which they are associated. Indeed, consistent with these three possibilities, Hammen has made the following observation:

> Regarding specificity, perhaps one of the intriguing issues is whether stress generation is about depression at all. Depression certainly may be the outcome of negative interpersonal life events, but perhaps it is the correlates or third variables such as personality pathology (e.g., borderline symptoms) or neuroticism or depressotypic cognitions that are more directly related to occurrence of dependent life events. (Hammen, 2006, p. 1,077)

Stress Generation as a Transdiagnostic Phenomenon

With regards to the first possibility—that stress generation is a transdiagnostic process—although a substantial focus of empirical inquiry with stress generation continues to be in relation to depression, research has since extended the study of stress generation to understand its role in other forms of psychopathology (Conway et al., 2012; A. E. Meyer & Curry, 2017; Rudolph et al., 2000). Some research has included a focus on personality pathology (Cummings et al., 2013; Daley et al., 2006), an intuitive extension of stress generation research. In extending evaluations of stress generation to psychiatric conditions other than depression, it is important to account for depression in some manner (e.g., as a covariate) to ensure that any observed findings with stress generation are not simply a function of comorbidity with depression.

That being said, the relation between stress generation and psychopathology may be a complex one, such that stress generation may manifest differently depending on the form of psychopathology under study. It has been suggested that whereas interpersonal stress generation may be a hallmark of depression, the generation of noninterpersonal dependent stress may be more relevant to externalizing psychopathology (Conway et al., 2012; Rudolph et al., 2000). Empirical evaluation of this possibility has important implications for understanding the etiology and processes underlying maintenance and recurrence of different disorders.

Stress Generation as a Process Occurring Outside of Acute Psychopathology

The second possibility, that stress generation may occur even in the absence of depression in individuals with a history of this disorder (Hammen, 2009b), has conceptually and clinically related implications. Support for this possibility would suggest that stress generation is a potentially persistent process rather than an episodic or intermittent phenomenon, as depression can often be. Given that proximally occurring life stress (i.e., within the prior 1–3 months) appears most etiologically relevant to depressive onset (Hammen, 2005; Harkness et al., 2006), if stress generation were only to occur during depression, stress generation would be a strong candidate mechanism for depressive persistence and relapse (i.e., a slide back into depression before full recovery is achieved; Frank et al., 1991) but not for depressive recurrence (i.e., a new occurrence of depression). Furthermore, although depression is an often-recurring disorder, many cases of depressive recurrence emerge years after an index episode (Richards, 2011). This means that long-term persistence of stress generation would be required for this phenomenon to account for a sizeable proportion of recurrent depression. Even if stress generation is present beyond instances of depression, how long it persists is an important clinically relevant empirical question. This question is also relevant to the broader question of whether stress generation may explain persistence and recurrence of other forms of psychopathology more generally.

A question that naturally follows and directly relates to the third possible extension of stress generation is: What may account for persistence of stress generation outside of depression specifically and psychopathology more generally? Given that depression and many other forms of psychopathology fluctuate in severity over time and the possibility that stress generation may, to some degree, similarly wax and wane (Liu & Spirito, 2019), more long-standing stress generation risk mechanisms may be involved. In fact, stress generation has been hypothesized to be influenced more broadly by an individual's beliefs, expectations, characteristics, and behavioral tendencies (Hammen, 1991, 2006, 2009b).

Moving Beyond Psychopathology in Predicting Stress Generation.

Cognitive Vulnerability Factors. Within this framework—and moving beyond psychopathology as the sole predictor—there have been an increasing number of studies aiming to identify potential stress generation risk factors. Of these candidate risk factors, cognitive vulnerability is among the most studied. Cognitive vulnerability is the focus of several cognitive theories of depression (e.g., the hopelessness theory of depression, Abramson et al., 1989; Beck's, 1967, 1987, the cognitive theory of depression; and the response style theory, Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008)

and anxiety (e.g., Riskind et al., 2013).[6] Underlying much research in this area is the hypothesis that the ways in which individuals think about themselves, the events in their lives, and their relation with others may directly or indirectly lead to behaviors that elicit life stress, particularly dependent stress in their interpersonal relationships. More specifically, drawing on the existing literature on self-fulfilling prophecies (e.g., Buchanan & Hughes, 2009; Jussim, 1986), it may be that individuals with negative self-expectations may engage with (or disengage from) their environment in a manner that ultimately conforms with their negative expectations. Furthermore, depressogenic cognitive styles have been proposed to lead to stress generation through the mediating effects of more proximal behavior risk factors (Liu, 2013).

Other Intrapersonal Psychological and Biological Factors. An assortment of other intrapersonal psychological factors has also been evaluated as potential stress generation mechanisms. These include psychological constructs that have been implicated in depression risk, such as avoidance (Holahan et al., 2005) and hopelessness (Kleiman et al., 2015), which may lead to stress generation by permitting minor issues to worsen through neglect into significant stressors or by inducing social strains when avoidance occurs within interpersonal contexts (Holahan et al., 2005). Also associated with depression and studied in relation to stress generation (Nelson et al., 2001) is the concept of dysfunctional autonomy, the notion that certain individuals place undue weight on independence and goal attainment to feel self-worth (Beck, 1983). Autonomous individuals become vulnerable to depression when they experience loss of control and failure. The high degree of independence valued by autonomous individuals may lead to devaluing the concerns of others, which may result in interpersonal stress generation (Daley et al., 1997). Finally, two externalizing constructs—aggression and impulsivity—have also attracted interest as potential stress generation predictors (Iacovino et al., 2016; Shih & Eberhart, 2010), in part because of their association with negative personal outcomes (Molz et al., 2013). More recently, research has examined biological factors, including genetic and physiological vulnerabilities, as predictors of stress generation. Considering genetic vulnerabilities, the serotonin transporter gene polymorphism (5-HTTLPR), particularly the short allele (*s* allele), has been studied in relation to risk for depression (Delli Colli et al., 2022). Work in this area has been extended to examine whether the *s* allele may predict stress generation (Brinksma et al., 2018; Starr et al., 2012). Similarly, atypical pubertal timing, which has been linked to depression in adolescence (Graber, 2013; Rudolph et al., 2014), has also received interest in relation to stress generation (Hamilton et al., 2014; Stroud et al., 2018).

Interpersonal Vulnerability and Personality Factors. Given that stress generation is an example of an action theory (i.e., based on the view that individuals are active agents in shaping the events in their environment; Hammen, 2006) and the central role that interpersonal dependent stress has in this theory, behavioral risk factors for depression, particularly interpersonal ones, are promising candidates for stress generation research. Excessive reassurance-seeking, as described in the interpersonal theory of depression (Coyne, 1976a), may serve a role in interpersonal stress generation (Eberhart & Hammen, 2009; Liu, 2013; Stroud et al., 2018). According to this theory, depression-prone individuals (i.e., those with a history of depression or vulnerability factors that have been implicated in risk for depression) seek reassurance from others in hopes of confirming their self-worth and the care and interest of others. Depression-prone individuals, however, tend to interpret the initial reassurances they receive as insincere, motivating them to seek further reassurance. A repetitive pattern emerges of seeking and discounting reassurance until others become irritated and frustrated, resulting in a deterioration of the relationship and rejection of the depression-prone individual. Ironically, this pattern then appears to confirm the individual's negative self-perceptions and doubts about the validity of the initial feedback. Indeed, studies have shown excessive reassurance-seeking to be associated with interpersonal rejection (Starr & Davila, 2008). Other behavioral interpersonal vulnerabilities have also been investigated in relation to stress generation, including maladaptive attachment styles (Eberhart & Hammen, 2009), rejection sensitivity (Hernandez et al., 2016), co-rumination (Bouchard & Shih, 2013; Hankin et al., 2010), or the tendency to focus excessively in dyadic relationships on discussions of personal problems (Rose, 2002).

In addition to these interpersonal processes, temperament and personality factors have also drawn interest as drivers of stress generation. Given that neuroticism has been associated with interpersonal difficulties (Kendler et al., 2003), it is perhaps not surprising that it has received particular attention in relation to stress generation (Barker, 2020; Goldstein et al., 2020; Iacovino et al., 2016; Uliaszek et al., 2012). Extraversion and perfectionism have also been subject to study in association with stress generation (Goldstein et al., 2021; La Rocque et al., 2016).

Other Risk and Protective Factors. Finally, a few miscellaneous risk factors and protective factors have also been evaluated in the context of stress generation. Potential risk factors include child maltreatment and adversity (Hernandez et al., 2016; Kushner et al., 2017), as well as maternal depression (Feurer et al., 2017; Gershon et al., 2011). In terms of protective factors, given that maladaptive interpersonal processes have been implicated in stress generation (Bouchard & Shih, 2013; Eberhart & Hammen, 2009; Hankin et al., 2010; Liu, 2013; Stroud et al., 2018), a natural extension has been to assess the potential role of adaptive interpersonal characteristics (i.e., social competence; Cummings et al., 2010). Subjective well-being may be another potential protective factor against stress generation (Elliot et al., 2011; Hamilton et al., 2017).

Important Considerations for Assessing the Stress Generation Hypothesis

In addition to the aforementioned potential predictors of stress generation, it is important to consider how sample, study design, and statistical considerations may influence study findings. Below,

[6] Definitions are provided here of cognitive vulnerability constructs within these theories that have been featured in multiple stress generation studies. Negative inferential styles, as conceptualized within the hopelessness theory (Abramson et al., 1989), are the tendency to respond to a negative event by inferring stable and global causes for the event, negative consequences of the event, and negative self-characteristics as a result of the event. Dysfunctional attitudes within Beck's (1967, 1987) cognitive theory involve rigid schemata relating to perfectionism and concerns about evaluation by others. Rumination is a preservative tendency to focus on feelings of distress and the potential causes and consequences of this distress (Miranda et al., 2008; Nolen-Hoeksema et al., 2008). One particular aspect of rumination—brooding—involves passively comparing oneself to an unachieved standard (Treynor et al., 2003).

we first discuss several demographic characteristics that have been evaluated both as direct predictors of and/or moderators of stress generation. We then present study characteristics that may moderate effect sizes for the relation between psychopathology and stress generation. Finally, we end with statistical considerations of particular relevance for evaluations of stress generation.

Sociodemographic Factors and Sample Considerations

Age and Development. Age and development are important constructs to consider when examining stress generation. Although stress generation has been documented across the lifespan (Hammen, 2005; Hankin et al., 2010; Liu, 2013), it may be of particular relevance to adolescence (Liu & Alloy, 2010), a period of development marked by increased interpersonal life stressors (Compas et al., 1985; Ge et al., 1994), increased autonomy and individuation, decreased parental monitoring, and increased importance of peer relations and social networks (Blakemore & Mills, 2014; B. B. Brown, 1990; Grotevant & Cooper, 1985; Parke & Bhavnagri, 1989; Steinberg, 2002; Steinberg & Silverberg, 1986; Wray-Lake et al., 2010). A product of this greater role in navigating and shaping the social environment is a greater opportunity for the occurrence of dependent stress (e.g., conflicts with parental authorities as a consequence of striving for greater autonomy). Although opportunities for autonomy and attendant opportunities for dependent stress may continue in adulthood, there is theoretical and empirical reason to believe that stress generation may decrease later in adulthood. According to the socioemotional selectivity theory (Carstensen et al., 1999; Charles & Carstensen, 2010), as adults age, they become more selective with their use of time, with increasing priority being given to positive social interactions. This involves gradually refining their social networks to maximize positive socioemotional experiences. As a consequence, as people age, they tend to experience less interpersonal-dependent stress (Charles & Carstensen, 2010). Furthermore, with age, adults become better at resolving small interpersonal stressors before they become larger ones (Birditt & Fingerman, 2003; Blanchard-Fields, 2007; Charles & Carstensen, 2010). Stress generation may decline in adulthood as impulsivity and risk-taking also naturally decrease (Steinberg, 2010). Although there are benefits to these behavioral tendencies during development (Telzer et al., 2022), they may also increase risk for dependent stress. Thus, it is expected that stronger associations will be present for stress generation in studies with younger (particularly adolescent) samples.

Gender. Although stress generation has been observed in both men and women (Hammen, 2005; Liu & Alloy, 2010), the stress generation hypothesis was originally articulated to explain the recurrence of depression in a sample of women. In part, it was based on the view that depressed women often occupy social roles that place them in broader social networks and, thus, have greater opportunities for stressful interpersonal life events (Hammen, 2005, 2006). Thus, several studies have evaluated gender as a stress generation predictor (Mezulis et al., 2010; Rose et al., 2017; Shih & Auerbach, 2010), and it is possible that gender may moderate the stress generation effect, such that studies with a higher proportion of females may have larger effects.

Race and Ethnicity. Another sociodemographic characteristic that may be important to consider is race and ethnicity. Racially and ethnically minoritized individuals are at a greater risk of experiencing stressful life events—particularly interpersonal ones—as a direct result of their minoritized status. According to a biopsychosocial model of racism (Clark et al., 1999), minoritized individuals experience victimization, discrimination, and prejudicial rejection because of their minority identity. These behaviors are inherently interpersonal in nature. Minoritized individuals may therefore prospectively experience higher rates of dependent stress than nonminoritized individuals. Although this would superficially seem consistent with stress generation, caution would be warranted against such an interpretation. Stress generation is an action theory (Hammen, 2006), meaning experiences of dependent stress are an inadvertent product of a person's attitudes, cognitions, and behavior. It therefore cannot account for dependent stressors that are instead the product of racism. Thus, although stress generation is worth evaluating in the context of race and ethnicity, findings of greater dependent stress would suggest the need for further investigation to make clear the factors driving their occurrence.

Socioeconomic Status. Socioeconomic status (SES) is also of potential relevance to stress generation (Chan et al., 2014). It may be that low SES may deprive the individual of instrumental resources to prevent the occurrence of negative event outcomes. More specifically, SES has been associated with more challenging family environments (Chan et al., 2014). For example, this may be because a parent needing to work multiple jobs has less opportunity to connect with their child. The current review provides an opportunity to evaluate whether SES is inversely related to stress generation.

Sample Type. Finally, sample type may also moderate stress generation. Insofar as stress generation is a transdiagnostic phenomenon (Conway et al., 2012; A. E. Meyer & Curry, 2017), levels of dependent stress may reasonably be expected to be saturated in clinical samples, resulting in a reduced range compared to in community samples. Moreover, comparisons for clinical predictors in clinical samples would be relative to other clinical presentations for which stress generation would also be expected to occur, whereas in community samples, comparisons for those same clinical predictors would more likely be made relative to healthy controls. Stress generation effects may therefore be predicted to be larger in community samples than clinical ones.

Study Design Considerations

Before discussing methodological considerations, a trend that merits attention is the common practice of attempting assessments of stress generation with cross-sectional data (e.g., examining dependent stress in relation to concurrent psychopathology or evaluating dependent stress immediately prior to current psychopathology). Although positive findings from studies adopting this approach provide value in indicating that inherently more intensive longitudinal research is warranted, they cannot be taken as evaluations of stress generation as they unavoidably conflate stress generation with stress exposure. In fact, studies that evaluate dependent stress immediately prior to current psychopathology are more accurately described as studies of stress exposure rather than stress generation (Liu, 2013). This is particularly true if the individual's experience of life stress shortly prior to current psychopathology may differ from what is normally observed at other times (e.g., if the onset or exacerbation of symptoms is more likely to occur shortly following exposure to life stress). An example illustrating this concern at a fine temporal level can be found in a study retrospectively assessing life

stress in the 48 hr immediately prior to a suicide attempt in a sample of adults admitted to acute psychiatric care (Bagge et al., 2013). This study found a significantly higher likelihood of interpersonal dependent stress—but not other forms of life stress—in the 24 hr immediately before their suicide attempt than in Hours 24–48 preceding the attempt. Therefore, for the current review, we only considered studies that ensured clean temporal separation between predictor and outcome (i.e., evaluated candidate risk factors in relation with subsequent life stress).

Measurement of Psychopathology. Beyond this basic requirement of temporal separation between stress generation and its predictors, temporal factors are important in the measurement of psychopathology variables evaluated as stress generation risk factors. That is, whether lifetime versus current psychopathology is assessed may potentially have a moderating effect on stress generation. Inasmuch as clinical experiences and severity may more accurately predict proximal-dependent stress, evaluations of current psychopathology may reasonably be expected to produce larger effects. However, insofar as stress generation may persist long term, even if in attenuated form, assessments restricted to current psychopathology may capture remission states and miss past experiences of psychopathology that contribute to stress generation, potentially leading to smaller effects than if lifetime history of psychopathology were accounted for.

Another relevant study design feature is whether operationalization of psychopathology is based on diagnosis versus symptom severity. Although prior research offers compelling reasons to examine disorders along a dimensional continuum (Bala et al., 2021; Haslam et al., 2020; Kotov et al., 2021; Liu, 2016; Prisciandaro & Tolliver, 2015), categorical approaches have been widely utilized in the stress generation literature and prevalent in clinical settings. To the extent that stress generation is hypothesized to account for chronicity of psychopathology, determining how best to assess psychopathology as it pertains to stress generation, whether larger effects are obtained with diagnostic versus symptom-based assessment approaches may be informative.

Also, an outstanding question is whether the use of self-report versus interview-based measures of psychopathology may have a moderating influence on stress generation. Although we know of no theoretical or empirical support for larger effects to be obtained with one assessment method over the other, evaluating this possibility may inform interpretation of effects generated in individual studies.

Measurement of Life Stress. Of central importance to the study of life stress is the measurement of this construct. In terms of how life stress is measured, available options largely fall into two categories: self-report questionnaires and interviews. Interviews may be further subdivided into general interviews, which may include establishing the dates of events and confirming the validity of endorsed items (e.g., that a rejection had occurred rather than was anticipated or perceived), and contextual threat life stress interviews, which involve generating contextual narratives for endorsed events (G. W. Brown & Harris, 1978).

The choice of measure type is important because there is considerable evidence to indicate that self-report measures, although widely used and convenient in terms of economy, are qualified by certain psychometric limitations. Self-report methods have been found to be prone to inflated life event endorsement rates (Hammen et al., 1985; Lewinsohn et al., 2003; McQuaid et al., 1992; Oei & Zwart, 1986), in part due to endorsement of different aspects of the same event as multiple events (e.g., counting a conflict that ended in a break-up as both a conflict and dissolution of the relationship). There is also evidence that this inflated response tendency with self-report measures is more pronounced in individuals with psychopathology because both they and these measures are more vulnerable to idiosyncratic and subjective interpretative biases. This tendency has been found, for example, in individuals with depression (S. L. Johnson & Roberts, 1995), who appear vulnerable to the mood-congruent interpretative biases that are at the center of cognitive theories of depression (Abramson et al., 1989; Beevers, 2005; Mathews & MacLeod, 2005). This finding extends to vulnerability factors. Neuroticism, for example, produces higher subjective ratings on measures of life stress (Espejo et al., 2011). Also of note, individuals prone to depression appear more physiologically reactive to life stress (Gotlib et al., 2008), which may increase their perception of what is worth endorsing on self-report life stress checklists. Collectively, these findings suggest that responses on self-report checklists may in part reflect underlying personality, cognitive, and physiological vulnerabilities in addition to the actual stressors they are designed to measure. In confounding life stress with related predictor variables, self-report life stress measures may inflate estimates of the relation between stress generation and its predictors.

General life stress interviews may partially address some of these concerns insofar as they confirm the occurrence of the stressors in question. Contextual threat life stress interviews, however, are viewed as the gold standard in the field (Hammen, 2005; Harkness & Monroe, 2016; Monroe, 2008), as they offer unique advantages over general interviews and self-report checklists in a few notable ways. First, these interviews involve ratings generated by individuals unaware of (and thus unbiased by) the participant's condition, providing a more objective measure of life stress. Second, and of particular relevance to stress generation, they allow for precise coding of dependence versus independence. This is important because depressed adolescents have been found to overestimate their role in the occurrence of the life stress they experience (Krackow & Rudolph, 2008), which could lead to inflated endorsement of dependent stress in self-report measures. Even if this issue is addressed in general interviews that include confirming occurrence of endorsed stressors, imprecision on categorizing stressors based on dependence versus independence may still occur without the additional information a context threat interview provides. In the case of a child changing schools, for example, a contextual threat approach is necessary to determine whether it is a dependent event (e.g., because of expulsion from a prior school) or an independent event (e.g., because of the child's parents' job change required moving to a different city). Coding of dependence versus independence is made by the researchers, which in the case of measures that do not employ a contextual threat approach means that events such as in this example must be discarded or have a standard coding applied based on what the researchers believe it likely to be in most cases (e.g., always coded as dependent if they believe most endorsements of the event within the study population would usually be for a behavioral issue). Both solutions are not ideal because of data loss with the first option and validity issues with the latter.

A third unique aspect of contextual threat approaches is that they allow for standardized ratings of the impact of individual stressors, in contrast to the aforementioned subjective influences to which self-report life stress measures are vulnerable. This involves having ratings for all participants generated by individuals unaware of the

participants' conditions. The advantage of this is that it allows for standardized life stress severity ratings that are not possible with other life stress measurement methods. A fourth advantage is the ability to accurately code and distinguish between chronic stress (i.e., stress that has lasted longer than 12 months; Hammen, 2005; McGonagle & Kessler, 1990) and episodic stress (i.e., discretely occurring negative life events that are short-lived in nature, with clearly defined beginnings and endings; Hammen, 2005). Given the role of chronic and episodic stress in mental health outcomes (Grover et al., 2009; Hammen, 2005; Hammen et al., 2010; Harkness et al., 2014; McGonagle & Kessler, 1990; Vrshek-Schallhorn et al., 2015), whether stress generation is involved in both stress types deserves evaluation.

Also important to consider when measuring life stress is the temporal window covered by the assessment. The importance of this lies in findings from prior methodological studies that recollection of life events fades over time, with even recall of major events appearing to decline after a year in adults (G. W. Brown & Harris, 1982; Paykel, 1997) and over brief periods of time in adolescents (Monck & Dobbs, 1985). These findings would suggest that longer temporal windows would reduce documentation of life stress, and thus reduce estimates of stress generation.

Length of Follow-Up Period. Prior meta-analytic work with longitudinal research has found the strength of the relation between psychopathology and other variables of interest diminishes as a function of increasing time (Liu et al., 2020). Insofar as stress generation with clinical predictors may be a similarly state-sensitive process influenced by clinical severity at the time of assessment (Liu & Spirito, 2019), it is possible that studies that evaluate psychopathology as a predictor of stress generation over larger prospective windows would produce smaller effects than those testing this relation within briefer follow-up periods.

Study Production Year. Whether the effect size observed in individual studies varies as a function of time is also an interesting consideration. If effect sizes are not smaller in more recent studies than in older ones, it would suggest that the stress generation hypothesis may be robust to issues of replicability within the field (Nosek et al., 2022; Shrout & Rodgers, 2018; Tackett et al., 2019). If, on the other hand, reduced effect sizes are observed in recent studies, a conservative interpretation would be to give these studies greater weight to the extent that they are more likely to feature rigorous designs and other research practices that enhance replicability.

Statistical Considerations

An important but almost uniformly overlooked consideration is that the independent stress component of stress generation is essentially a null hypothesis (i.e., that psychopathology and related risk factors would not be associated with prospectively occurring independent stress). The standard practice in stress generation research of evaluating this element of stress generation using null hypothesis significance testing (NHST) is problematic because NHST was not designed for testing predictions of a null effect. Rather, with NHST, a *p* value is intended to be used to quantify the probability of the observed data (or more extreme results) occurring if the null hypothesis were true. That is, one can only reject or fail to reject the null hypothesis. Indeed, Ronald Fisher, the statistician who introduced the concept of the null hypothesis and the *p* value, cautioned, "The null hypothesis is never proved or established, but is possibly disproved" (Fisher, 1935, p. 19). Similarly, the American Psychological Association Task Force on Statistical Inference issued this proscription: "Never use the unfortunate expression 'accept the null hypothesis'" (Wilkinson, 1999).

An alternative statistical approach particularly well suited for addressing this limitation of NHST by evaluating the null hypothesis is Bayesian analysis (Dienes, 2011; Jarosz & Wiley, 2014). In contrast to NHST, Bayesian analysis allows for a direct evaluation of the strength of support from the observed data for the study hypothesis (i.e., Hypothesis 1) relative to the null hypothesis (i.e., Hypothesis 0), in the case of the dependent stress component of stress generation (i.e., that psychopathology and related risk factors would be associated with prospectively occurring dependent stress). Moreover, in the case of the independent stress component of stress generation, Bayesian analysis permits directly quantifying the strength from the observed data for Hypothesis 0 relative to Hypothesis 1.

A statistical consideration that relates, in part, to the first methodological issue mentioned above involves testing the mediational component of the stress generation hypothesis. A common practice in the broader psychological literature is to apply cross-sectional statistical tests of mediation for a hypothesized longitudinal association. The problems with this practice have been well characterized in that it can lead to misleading results (e.g., Kazdin, 2007; Maxwell & Cole, 2007; Winer et al., 2016). Just as temporal ordering of stress generation and its predictors, as mentioned above, is important on theoretical grounds, ensuring temporal ordering in the assessment of candidate stress generation risk factors, mediating dependent stress, and mental health outcomes is necessary to conduct valid statistical evaluations of the mediational component of the stress generation hypothesis.

The Present Study

The objective of the current review is to provide a comprehensive and systematic evaluation of the first three decades of published research on stress generation. We applied both traditional frequentist and Bayesian meta-analytic techniques, particularly with the view of facilitating interpretative clarity for data pertaining to the independent stress part of stress generation. Two components of the stress generation hypothesis were submitted to quantitative review: (a) Psychopathology and related risk factors are positively associated with subsequent overall dependent stress and its interpersonal and noninterpersonal subtypes, and (b) psychopathology and related risk factors are not associated with subsequent independent stress. To advance characterization of the stress generation phenomenon, whenever possible, quantitative synthesis was also conducted for protective factors and sociodemographic characteristics in relation to subsequent dependent and independent stress. The third remaining component of the stress generation hypothesis—that dependent stress mediates the association between psychopathology and related risk factors and subsequent psychopathology—was submitted to qualitative review.

We specifically aimed to evaluate the following hypotheses: First, psychopathology (including internalizing and externalizing psychopathology and specific disorders) and associated risk factors would prospectively predict higher dependent stress (including its subtypes) but not be predictive of independent stress. Second, we hypothesized that protective factors would prospectively predict lower dependent stress but also not be predictive of independent

stress. Third, we hypothesized that the association for psychopathology and associated risk factors, respectively, with mental health outcomes would be mediated by dependent stress. Fourth, wherever possible, we examined sociodemographic and sample characteristics as both predictors and moderators of the relation between psychopathology and stress generation. Of these, we hypothesized that younger age (particularly adolescence), being assigned female at birth, lower SES, and clinical (vs. nonclinical) samples would predict greater stress generation. Additional moderator analyses of the association between psychopathology and stress generation included evaluations of whether larger effects would be observed for clinical diagnoses (vs. symptoms), self-report life stress measures (vs. interview-based measures), briefer life stress measure timeframes, and briefer study follow-up periods. Finally, exploratory analyses in relation to stress generation were conducted for race and ethnicity, measure of psychopathology (self-report vs. interview), timeframe for assessing psychopathology (lifetime vs. current), life stress operationalization (i.e., contextual threat impact ratings vs. life stress frequency count, and chronic vs. episodic stress), and year that the study was produced.

Method

Search Strategy and Eligibility Criteria

The present review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement. A systematic search of the literature was conducted in APA PsycInfo and MEDLINE to identify studies of potential relevance to the current review. The following search string was applied: ("stress generation" or "generation of stress" or "generation of stressful" or "generation of life stress" or "generation of life stressor" or "generation of life stressors" or "generation of life event" or "generation of life events" or "generation of interpersonal" or "generation of social" or "generation of dependent" or "generation of positive dependent" or "generation of positive interpersonal" or "generation of positive social" or "generation of positive life" or "generation of positive event" or "generation of positive events" or "event generation" or "events generation" or "dependent stress" or "dependent stressors" or "dependent life" or "dependent interpersonal" or "dependent social")[7] NOT (oxidati* or oxygen or rats or rodent* or mice or receptor). The search results were limited to (a) English-language publications and (b) journal articles in press or in print from 1991 through December 31, 2021, covering the 30 years of published research since Hammen's (1991) original stress generation study.

The study inclusion criteria were as follows: (a) The study directly evaluated at least one component of the stress generation hypothesis (i.e., a predictor variable in relation to some form of dependent stress or independent stress); (b) dependent and independent life events were separated from each other (i.e., analyses that combined both life-event types were ineligible); (c) there was clean temporal separation between predictor variable(s) and life events in the relevant analyses;[8] (d) given that stress generation is about the actual occurrence of stressful events rather than the perception or subjective experience of higher stress, relevant analyses must have been evaluations of the occurrence of observable life events rather than subjective or physiological stress; (e) the assessment of predictor variable(s) was standardized and systematic across study participants; and (f) the assessment of life events was standardized and systematic across study participants (e.g., studies that extracted life events data from medical chart reviews were excluded).

Each unique search result was reviewed independently by at least two of the authors to determine eligibility ($\kappa = .94$). In cases where eligibility could not be ruled out based on the title and abstract, the full text was also examined. In cases where more information was needed to determine study eligibility or for data extraction, as detailed below, every effort was made to obtain additional details regarding the measure or study design (e.g., examining other publications based on the same data set or contacting study authors). Discrepancies in coding article eligibility were resolved by the first author.

For studies meeting eligibility criteria but featuring overlapping samples, determination of which study to include in the meta-analysis was based, in descending order, on (a) inclusion of sufficient reported data for meta-analysis and (b) the largest sample size for the relevant analysis. In cases where two studies used overlapping samples but examined different associations (e.g., different predictors of subsequently occurring dependent stress), however, both studies were retained for the relevant analyses. Whenever it remained unclear after full-text inspection whether two studies reported on overlapping samples, the study authors were contacted to seek clarity on this issue.

Moderator Analysis

We extracted data for 16 study characteristics for consideration for moderator analysis. These included six sample characteristics: (a) percentage of female participants in the study sample, (b) percentage of White participants in the study sample, (c) participants' starting age as a continuous variable (i.e., mean age at study baseline), (d) participants' starting age as a categorical variable (i.e., youth vs. adult sample), (e) participants' age at the end of study as a categorical variable, and (f) sample type (clinical/at-risk vs. community). Data for seven study design characteristics were also extracted, including (a) psychopathology operationalization (i.e., diagnosis vs. symptom severity), (b) psychopathology measure type (i.e., interview vs. self-report questionnaire), (c) psychopathology measure timeframe (i.e., current vs. lifetime), (d) life stress measure type (i.e., interview vs. self-report questionnaire),[9] (e) life stress operationalization (i.e., contextual threat impact ratings

[7] Terms related to generation of positive life events were included in our search terms, as we originally wanted to evaluate the possibility of positive life event generation (Liu & Alloy, 2010). There were too few studies, however, for meta-analysis.

[8] Based on this criterion, studies that employ a longitudinal design are not necessarily eligible. Longitudinal studies that evaluated a predictor over a follow-up window relative to temporally overlapping life events occurring within the same window were ineligible (e.g., a study that assessed current depressive symptoms measured at baseline in relation to life stress measured at a 6-month follow-up but for which the life stress measure covered the past 12 months). In addition to fully prospective longitudinal analyses, retrospective studies that evaluated a temporally defined predictor in relation to more recent life events were eligible (e.g., a study that retrospectively assessed both childhood maltreatment experiences in relation to life stress but excluded from analyses any maltreatment experiences that occurred subsequent to the occurrence of life stress).

[9] As contextual threat interviews and noncontextual threat interviews did not differ in the strength of their effects across all moderator analyses (see notes to Tables 3–5), they were combined in these analyses, thereby ensuring the largest k for each analysis and the largest possible number of feasible analyses.

vs. life stress frequency count),[10] (f) life stress measure timeframe (i.e., ≤6 months vs. >6 months, and <1 year vs. ≥1 year), and (g) length of study follow-up (i.e., ≤6 months vs. >6 months, and <1 year vs. ≥1 year). Additionally, the year of study production was also extracted. For this moderator variable, the earliest date available for each study was extracted, either in the published article or online (i.e., in hierarchical order, date of data collection, date of submission for publication, date of acceptance for publication, date of online publication, and date of print publication). An exception was made for one study, where the online publication date was later than the print publication date, for which the date of print publication was extracted (Chun et al., 2004). Additionally, in studies that were part of a family of overlapping samples, the dates used for these analyses were based on the earliest dates available within the family of studies. Study quality was also assessed as a moderator, with the Newcastle–Ottawa scale (Wells et al., 2011) serving as an index of quality. For this scale, higher scores (from 0 to 8) indicate higher study quality. Finally, a single analysis feature was extracted: fully prospective longitudinal versus retrospective analysis.[11] These study characteristics formed the basis of eight hypothesis-driven and seven exploratory moderator analyses for dependent stress, as detailed below. As the stress generation hypothesis does not posit a difference in independent stress, analyses with these same study characteristics in relation to that stress type, when conducted, were exploratory.

Data Analysis Methods

Quantitative analyses of predictors of negative life events were conducted only when the number of unique effects was ≥3, so as to obtain reliable pooled estimates of effect sizes (Favril et al., 2020; Liu et al., 2022). Data for quantitative analysis were independently extracted by at least two authors, with discrepancies resolved by the first author. Similarly, data for the qualitative analysis section of the current review were independently extracted by two authors, with discrepancies resolved by a third author.

All traditional frequentist analyses were conducted with Comprehensive Meta-Analysis Version 4.0.000 (Biostat, 2022). The standardized mean difference (Cohen's *d*) was used as the primary index of effect size. Cohen's *d* of 0.10 is interpreted as a trivial effect size, 0.20 as small, 0.50 as medium, and 0.80 as large (Hamza et al., 2015; National Academies of Sciences Engineering & Medicine, 2018). All pooled effects were calculated such that values greater than zero reflected a positive association between a predictor of interest and dependent and/or independent stress. Larger positive *d* values for putative risk factors of overall dependent stress and its interpersonal and noninterpersonal subtypes are indicative of greater support for stress generation, as are larger negative *d* values for putative protective factors (i.e., SES, general protective factors, social competence, and subjective well-being) in relation to these outcomes. In contrast, smaller *d* values, regardless of valence, are consistent with the component of the stress generation hypothesis regarding the absence of a predictive relation with independent stress.

Random-effects models were generated in preference to fixed-effects models to account for the expected high heterogeneity across studies resulting from differences in samples, measures, and design (Borenstein et al., 2009). Random-effects models are more appropriate than fixed-effects models in that they account for this heterogeneity by incorporating both sampling and study-level errors, with the pooled effect size representing the mean of a distribution of true effect sizes instead of a single true effect size. In contrast, fixed-effects models assume that a single true effect size exists across all studies and that any variance detected is strictly due to sampling error. It thus estimates only within-study variance.

To supplement the traditional frequentist meta-analytic approach, we also conducted random-effects Bayesian meta-analysis. We used the *metaBMA* package (Heck et al., 2021) to obtain Bayes factors for each analysis. We set weakly informative priors for each analysis based on similar prior work and standard recommendations (Harrer et al., 2021; Williams et al., 2018). This is in line with the idea that studies may vary in level of credibility. For similar reasons, we set half-Cauchy priors for the τ^2 distribution, which are similar to a one-tailed normal distribution but with thicker tails. This allows for a wide range of possible τ^2 values while assigning less probability to more extreme values. The half-Cauchy distribution has been found to have positive properties for psychological research (Williams et al., 2018).

Bayes factors allow for direct quantification of the strength of the existing empirical evidence in favor of the study hypothesis—that a candidate stress generation predictor is associated with subsequent dependent stress—relative to the null hypothesis (i.e., BF_{10} = the likelihood of the data with the stress generation hypothesis [Hypothesis 1] ÷ the likelihood of the data given the null hypothesis [Hypothesis 0]). Bayesian analysis was also used to quantify the empirical support for the null hypothesis (BF_{01}) in the case of independent stress (i.e., BF_{01} = the likelihood of the data given the independent stress component of the stress generation hypothesis [Hypothesis 0] ÷ the likelihood of the data given a difference exists in independent stress [Hypothesis 1]). When the $BF_{10} < 1$, the data are more supportive of Hypothesis 0 than Hypothesis 1. $BF_{10} = 1$ indicates that the data do not favor Hypothesis 1 or Hypothesis 0. As BF_{10} increases, the strength of support for Hypothesis 1 increases, such that values between 1 and 3 provide anecdotal support for Hypothesis 1, values between 3 and 10 indicate substantial evidence in support of Hypothesis 1, values between 10 and 30 reflect strong support, values between 30 and 100 indicate very strong empirical support, and values exceeding 100 provide extreme evidence in support of Hypothesis 1 (Jeffreys, 1961; Wagenmakers et al., 2011).

If, according to the stress generation hypothesis, psychopathology and associated risk factors are prospectively associated with greater dependent stress, interpretation of BF_{10} and *d* for dependent stress is therefore consistent with each other in that support for associations with dependent stress strengthens with increasingly higher values. Additionally, if, according to the stress generation hypothesis, psychopathology and associated risk factors are not prospectively associated with independent stress, BF_{01} and *d* for independent stress

[10] We had originally planned to evaluate stress generation with chronic stress versus episodic stress, but there were too few studies of chronic dependent stress meeting our eligibility criteria for viable analysis. Chronic and episodic stress were therefore combined in all analyses.

[11] This study analysis feature was subsequently determined to be unfeasible for moderator analysis because only three eligible studies were identified as employing fully or partially retrospective analyses. Specifically, one employed a fully retrospective analysis (Kushner et al., 2017), whereas two featured a longitudinal design, but with assessments of the occurrence of the predictor of interest prior to the life stress outcome taking place concurrently with assessments of life stress (Conway et al., 2012; Liu, Alloy, et al., 2014).

should contrast. As BF_{01} increases in magnitude, support increases for the independent stress component of the stress generation hypothesis (and BF_{01} values approaching zero provide increasingly strong support against this part of the stress generation hypothesis). This differs from *d*, for which smaller absolute values are more consistent with the independent stress component of the stress generation hypothesis (and larger *d* values are increasingly inconsistent with this part of the stress generation hypothesis).

Heterogeneity analyses were conducted for clinical predictors of life stress. Heterogeneity was evaluated using (a) the τ^2 statistic, an estimate of the variance of the true effect sizes (i.e., between-study variance due to heterogeneity rather than measurement error); (b) the *Q* statistic and associated *p* value; and (c) I^2, which indicates the percentage of the variance in an effect estimate that is due to heterogeneity across studies rather than sampling error (i.e., chance). Low heterogeneity is indicated by I^2 values of around 25%, and moderate heterogeneity is indicated by I^2 values of 50%. Substantial heterogeneity that is due to real differences in study samples and methodology is indicated by an I^2 value of 75%, which suggests that the observed heterogeneity is more than would be expected with random error (Higgins et al., 2003). High heterogeneity is indicative of the need for moderator analyses to account for potential sources of this heterogeneity. Each potential moderator was assessed individually in frequentist analyses, with an estimate of the effect size at each level of the moderator calculated in the case of categorical moderators.

Meta-analyses allow for an estimation of potential publication bias in the literature. Studies with small effect sizes or nonsignificant findings are less likely to be published and therefore may be more likely not to appear in meta-analyses, resulting in a potentially inflated effect size estimate. To evaluate for potential publication bias, the following publication bias indices were used: Duval and Tweedie's trim-and-fill analysis (Duval & Tweedie, 2000) and Egger's regression intercept (Egger et al., 1997). Duval and Tweedie's trim-and-fill analysis generates an estimate of the number of missing studies based on asymmetry in a funnel plot of the standard error of each study in a meta-analysis (based on the study's sample size) against the study's effect size. This analysis also calculates an estimated effect size and confidence interval, accounting for these missing studies. This procedure assumes homogeneity of effect sizes, and its results must be interpreted with some caution where significant heterogeneity is present. Egger's regression intercept estimates potential publication bias, with *p* values $<.05$ indicating the presence of significant publication bias.

Data Analytic Plan

Psychopathology in Relation to Prospective Stress Types

Both frequentist and Bayesian meta-analyses were first conducted for overall psychopathology, internalizing psychopathology, and externalizing psychopathology, respectively, in relation to prospective overall dependent stress, its interpersonal and noninterpersonal subtypes, and independent stress. For these analyses, overall psychopathology included any diagnostic or symptom variable, as well as general internalizing and externalizing psychopathology. Personality pathology and self-injurious thoughts and behaviors (SITBs) were included as well. Analyses of internalizing psychopathology included anxiety disorders and depression, as well as general internalizing symptoms. Externalizing psychopathology included attention-deficit/hyperactivity disorder, oppositional defiant disorder, conduct disorder, substance use, and general externalizing symptoms. Wherever possible (i.e., $k \geq 3$), meta-analyses were also conducted for specific disorders with prospective life stress types. As stress generation was originally conceptualized as a theory of depression, among individual disorders, it has received the most attention in this body of research. Therefore, it is possible that findings for internalizing psychopathology could be largely driven by a high representation of depression in the relevant analyses. To evaluate this possibility, sensitivity analyses were conducted for internalizing psychopathology with depression variables removed. Furthermore, meta-analyses were viable and conducted for depression in relation to four additional dependent stress subtypes: interpersonal family stress, interpersonal romantic stress, interpersonal peer stress, and noninterpersonal achievement stress.

All frequentist meta-analyses with dependent stress types were complemented by qualitative evaluations of whether any observed significant associations with dependent stress remained after covarying baseline depression (where relevant) and life stress. These evaluations were done qualitatively because meta-analysis is recommended with bivariate relationships rather than associations that have been adjusted for the effects of covariates (Siddaway et al., 2019).[12] Interpreting effect sizes generated by meta-analysis with multivariate relationships is particularly complicated in cases where few studies are included in the analysis and each study differs substantially in the number and types of covariates they included.

If moderator analyses were viable for overall psychopathology, internalizing psychopathology, externalizing psychopathology, and depression, respectively, in relation to prospective dependent and independent stress, heterogeneity analyses were first conducted for these associations. Where significant heterogeneity was observed, moderator analyses were then conducted with the 15 study characteristics (17 moderator variables) described above (see Moderator Analysis section). Following moderator analyses, analyses of publication bias and associated funnel plots were carried out.

Risk Factors, Protective Factors, and Sociodemographic Characteristics in Relation to Prospective Stress Types

Wherever possible (i.e., $k \geq 3$), frequentist and Bayesian meta-analyses were then conducted for risk factors (i.e., cognitive, other intrapersonal psychological, biological, interpersonal, personality, and other risk factors), protective factors, and sociodemographic factors in relation to prospective overall dependent stress, its interpersonal and noninterpersonal subtypes, and independent stress. As before, all frequentist meta-analyses were complemented

[12] Note that the number of studies unique effects (*k*) will almost invariably be lower in qualitative review of multivariate findings than in the corresponding meta-analytic review because the multivariate analysis of interest in the current review may not be central to the aims of the original study. For example, a bivariate finding included as part of descriptive analyses in a correlation matrix of all study variables may have been unrelated to the original study's hypotheses and so never carried forward to the final multivariate model. As another example, a predictor variable of interest in the current review may have been simply a covariate in the original article, and as is often the case, it was included as such in the final multivariate model but not reported in the associated table.

by qualitative evaluations of whether any observed significant associations held after covarying baseline depression and life stress.

Evaluating Stress Generation as a Mediator of Clinical Outcomes

Finally, we conducted a qualitative review evaluating the mediational component of the stress generation hypothesis (i.e., past psychopathology and associated risk factors predict future psychopathology through the mediating effect of dependent stress). We reviewed all identified articles of stress generation for evidence of mediational analyses and coded articles based on the presence of specific study characteristics. These included sample characteristics (e.g., adult or child/adolescent), predictor variables (e.g., type of vulnerability factor or psychopathology), type of primary mediator (e.g., dependent stress, interpersonal dependent stress), and outcome variables (i.e., form of psychopathology). Further, it was evaluated whether studies tested full temporal mediation and whether they covaried baseline psychopathology and life stress in mediational analyses. Two authors independently coded the articles, and data were then synthesized descriptively in the review.

Transparency and Openness

We followed the PRISMA-P checklist when preparing the protocol, and we followed PRISMA reporting guidelines for the final report. The meta-analytic data (Dreier, 2023) are shared at https://osf.io/5vyun/.

Results

Out of the 1,375 unique records identified, we excluded 1,125 based on their titles and abstracts. Following this initial screen, we excluded an additional 146 articles based on full-text review, resulting in 104 articles that satisfied the eligibility criteria. We contacted authors of studies that did not report adequate data for meta-analysis and obtained data for three of these studies (Bart et al., 2019; Bender et al., 2010; Rose et al., 2017). Two articles featuring overlapping samples were excluded at this stage, resulting in a final set of 102 articles featuring 104 studies drawn from 106 samples (66 nonoverlapping samples) included in the current review (see Figure 1).

Study characteristics are summarized in Table 1. Reports were published or completed between 1991 and 2023. These studies were conducted in nine countries across four continents: North America (Canada $k = 11$; the United States $k = 78$; Canada and the United States combined $k = 1$), Europe (Belgium $k = 1$; Germany $k = 1$; the Netherlands $k = 3$; Spain $k = 3$), Australia ($k = 4$), and Asia (China $k = 3$; Japan $k = 1$). In total, 31,541 participants were included in the analyses, with sample sizes from individual studies ranging from $n =$ 44 to 2,064. Samples encompassed individuals across developmental stages, with mean age at baseline ranging from 9.84 to 61.00 years old ($M = 19.25$, $SD = 10.22$, $Mdn = 18.08$). Just under half of the studies ($k = 51$; 49.04%) included youth samples (i.e., mean age at baseline <18), and just over half of the studies included adult samples ($k = 55$; 52.88%). The distribution of sample type was as follows: community $k = 74$, at-risk or mixed $k = 27$, and clinical $k = 5$. The mean proportion of female participants was 66.65% ($SD = 19.6$, $Mdn = 60.17$, range = 12.1%–100%), and the mean proportion of White participants was 65.90% ($SD = 20.92$, $Mdn = 70.84\%$, range = 0%–95%). The majority of studies ($k = 48$; 46.15%) measured life stress using questionnaires, followed by contextual threat interviews ($k = 36$; 34.62%), noncontextual threat interviews ($k = 16$; 15.38%), and daily diary ($k = 8$; 7.69%).[13] The timeframe for assessing life stress ranged in length from 1 week to 30 months ($M = 6.59$ months, $SD =$ 6.47 months, $Mdn = 6$ months). The length of follow-up ranged from 1 week to 6 years ($M = 11.04$ months, $SD = 12.78$ months, $Mdn = 6$ months). Study quality based on the Newcastle–Ottawa scale ranged from 3 to 7 ($M = 4.62$, $SD = .93$, $Mdn = 5$).

Quantitative Synthesis

Psychopathology in Relation to Prospective Stress Types: Main Analyses

Results of frequentist and Bayesian meta-analyses for overall psychopathology, internalizing psychopathology, and externalizing psychopathology, respectively, are presented in Table 2. Specific disorders for which it was also possible to conduct meta-analyses in relation to overall dependent stress and independent stress (i.e., $k \geq 3$) were depression, anxiety, posttraumatic stress disorder (PTSD), alcohol and substance use disorders, and personality disorders. With the exception of PTSD, alcohol and substance use, and personality disorders, it was also possible to conduct meta-analyses for all psychopathology variables in relation to interpersonal dependent stress and noninterpersonal dependent stress.

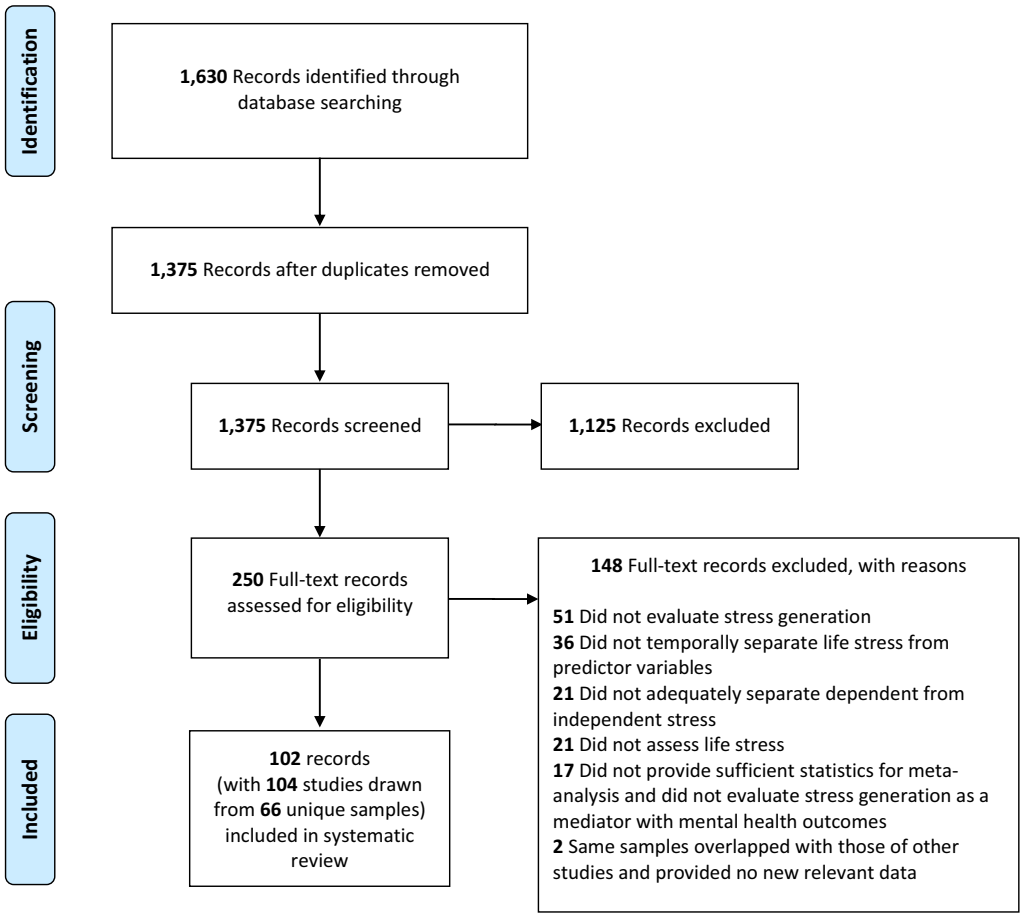
In frequentist meta-analyses, all psychopathology predictors were significantly associated with all stress types, with a few exceptions (i.e., anxiety and PTSD in relation to independent stress and externalizing psychopathology in relation to interpersonal dependent stress). Bayesian meta-analyses were generally consistent with frequentist meta-analyses.

Results were largely consistent across overall psychopathology, internalizing psychopathology, and depression in relation to each stress type, with ds = 0.52–0.54 for overall dependent stress, ds = 0.50–0.53 for interpersonal dependent stress, ds = 0.36–0.40 for noninterpersonal dependent stress, and ds = 0.21–0.24 for independent stress. Overall psychopathology, internalizing psychopathology, depression, and anxiety were more strongly associated with overall dependent stress than independent stress, as indicated by nonoverlapping 95% confidence intervals (CIs) between the two stress types for each psychopathology predictor. Externalizing psychopathology was significantly associated with all stress types except interpersonal dependent stress, with ds ranging from 0.17 for independent stress to 0.43 for overall dependent stress.

In sensitivity analyses, the strength of the associations between internalizing psychopathology and all four stress types remained largely the same after removing depression variables from these analyses (overall dependent stress: $k = 41$, $d = 0.49$, 95% CI [0.42, 0.57], $p < .001$; interpersonal dependent stress: $k = 23$, $d = 0.48$, 95% CI [0.40, 0.57], $p < .001$; noninterpersonal dependent stress: $k = 12$, $d = 0.35$, 95% CI [0.16, 0.53], $p < .001$; independent stress: $k = 17$, $d = 0.20$, 95% CI [0.12, 0.29], $p < .001$). Again, internalizing psychopathology remained significantly more associated with overall dependent stress than with independent stress, as indicated by nonoverlapping 95% CIs.

[13] Note that two studies (Eberhart & Hammen, 2009; Snyder & Hankin, 2016) used multiple life stress methods (see Table 1 for specifics).

Figure 1
PRISMA Flowchart of Literature Search



Note. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses. See the online article for the color version of this figure.

In analyses of depression in relation to additional dependent stress subtypes, significant associations were consistently found in frequentist analyses, and Bayesian analyses produced generally consistent results. That is, depression was associated with prospective interpersonal family stress ($k = 6$, $d = 0.47$, 95% CI [0.33, 0.60], $p < .001$, $BF_{10} = 113.00$), interpersonal romantic stress ($k = 5$, $d = 0.46$, 95% CI [0.37, 0.56], $p < .001$, $BF_{10} = 133.00$), interpersonal peer stress ($k = 5$, $d = 0.61$, 95% CI [0.34, 0.89], $p < .001$, $BF_{10} = 29.90$), and noninterpersonal achievement stress ($k = 11$, $d = 0.51$, 95% CI [0.33, 0.68], $p < .001$, $BF_{10} = 845.00$).

In supplemental qualitative analyses, we evaluated whether each form of psychopathology remained significantly associated with each dependent stress type after covarying life stress. Depression was also considered as a covariate (except in the case of overall psychopathology, internalizing psychopathology, and depression, as it would have been confounded with the predictor variable of interest [or the same as the predictor in the case of depression]). In general, the findings largely held in analyses that covaried baselined dependent stress but with weaker evidence for noninterpersonal dependent stress. It was the case for (a) overall psychopathology in relation to prospective overall dependent stress (four of six studies remained significant), interpersonal dependent stress (one of one study), and noninterpersonal dependent stress (one of one study); (b) internalizing psychopathology in relation to prospective overall dependent stress (five of seven studies), interpersonal dependent stress (two of three studies), and noninterpersonal dependent stress (one of two studies); and (c) depression in relation to overall dependent stress (five of seven studies), interpersonal dependent stress (five of seven studies), and noninterpersonal dependent stress (one of three studies).

Externalizing psychopathology remained significantly associated with overall dependent stress and its subtypes (one of one study in each case) in analyses covarying baseline depression and/or dependent stress. Apart from mixed findings for anxiety, all other specific disorders varied in terms of covarying baseline depression and/or dependent stress, but findings remained significant in almost all cases.

Psychopathology in Relation to Prospective Stress Types: Heterogeneity and Moderator Analyses

Heterogeneity analyses were conducted for overall psychopathology, internalizing psychopathology, and depression, respectively, in relation

Table 1
Study Characteristics

Study author(s) (year)	N[a]	% Female[a]	% White[a]	Mean age[a]	Sample source	Sample type	Life stress			Length of follow-up	Quality[b]
							Measure	Format	Time frame		
Allen et al. (2022)	355	76.90	75.49	28.30	United States	Clinical	RLCQ	Questionnaire	1 year	1 year	4
Auerbach and Ho (2012)$_a$	179	60.34	79.50	13.87	Canada	Community	ALEQ	Questionnaire	1 month	4.5 months	4
Auerbach et al. (2010) Canadian sample$_a$	118	54.00	78.90	15.17	Canada	Community	ALEQ	Questionnaire	1 month	4.5 months	4
Auerbach et al. (2010) Chinese sample$_b$	405	50.20	0.00	16.18	China	Community	ALEQ	Questionnaire	1 month	6 months	4
Auerbach, Bigda-Peyton, et al. (2011)$_a$	258	57.40	79.50	14.48	Canada	Community	ALEQ	Questionnaire	1 month	6 months	4
Auerbach, Webb, et al. (2011) Canadian sample$_a$	255	57.40	75.50	14.48	Canada	Community	ALEQ	Questionnaire	1 month	6 months	4
Auerbach, Webb, et al. (2011) Chinese sample$_b$	405	50.20	0.00	16.18	China	Community	ALEQ	Questionnaire	1 month	6 months	4
Auerbach et al. (2012)$_a$	105	60.00	79.70	15.12	Canada	Community	ALEQ	Questionnaire	1 month	6 months	4
Auerbach et al. (2014)$_a$	157	59.24	81.50	13.99	Canada	Community	ALEQ	Questionnaire	1 month	6 months	4
Bakker et al. (2011)$_c$	2,064	51.34		11.09	Netherlands	Community	SSM	Questionnaire	2 years	2.5 years	3
Barker (2020)	651				United States	Community	SSM	Questionnaire		10–20 weeks	5
Bart et al. (2019)$_d$	347	62.80	56.80	18.43	United States	Mixed	LEI^I, LES	Noncontextual threat interview	1 year	1 year	4
Belmans et al. (2019)	2,011	52.86	93.00	13.73	Belgium	Community	ALEQ	Questionnaire	3 months	1 year	5
Bender et al. (2010)$_e$	163	66.87	56.71	19.71	United States	Mixed	LEI^I, LES	Noncontextual threat interview	4 months	4 months	5
Bouchard and Shih (2013)	364	57.15	89.60	19.66	United States	Community	NLEQ	Questionnaire	1 week	2 months	5
Brinksma et al. (2018)$_c$	1,306	49.62		11.20	Netherlands	Community	LTDQ	Questionnaire	2 years	2 years	3
Calvete (2011)	853	52.29		15.86	Spain	Community	APES	Questionnaire	6 months	6 months	5
Calvete et al. (2013)	1,187	45.91		13.42	Spain	Community	APES	Questionnaire	6 months	6 months	5
Calvete et al. (2019)	1,190	49.33		15.16	Spain	Community	ALEQ	Questionnaire	1 year	2 years	4
Chan et al. (2014)	160	42.11	68.00	10.13	United States	Mixed	UCLA LSI	Contextual threat interview	1 year	1 year	5
Chun et al. (2004)	645	54.88	86.51	39.64	United States	Mixed	SSM	Questionnaire	1 year	1 year	4
Clements et al. (2008)	764	54.00	19.00	11.40	United States	At-risk	DHS, SSM	Questionnaire	1 month and 1 year	1 year	3
Cohen et al. (2013)$_b$	1,150	51.91	0.00	16.26	China	Community	ALEQ	Questionnaire	1 month	6 months	4
Conway et al. (2012)$_f$	705	51.49	92.00	19.00	Australia	At-risk	UCLA LSI	Contextual threat interview	1 year	N/A[c]	5
Conway et al. (2018)$_g$	1,630	55.00	65.00	59.60	United States	Community	LTE-Q	Noncontextual threat interview	6 months	6 months	5
Cox et al. (2009)	552	50.91		44.00	Canada	Community	LEI^Q	Questionnaire	1 year	1 year	5
Cummings et al. (2010)$_h$	310	77.70	86.50	19.67	United States	Community	SSM	Daily diary	1 week	1 Week	4
Cummings et al. (2013)$_h$	305	77.70	86.60	19.66	United States	Community	SSM	Daily diary	1 week	1 week	3
Daley et al. (1997)$_i$	131	100	46.00	18.29	United States	Community	UCLA LSI	Contextual threat interview	6 months	1–2 years	6
Daley et al. (2006)	54	100			United States	At-risk	UCLA LSI	Contextual threat interview		6 months	6
Davila et al. (1995)$_i$	140	100	46.00	18.18	United States	Community	UCLA LSI	Contextual threat interview	6 months	1 year	6
Davila et al. (1997)	154[d]	50.00[d]	64.00[d]	26.80[d]	United States	Community	UCLA LSI	Contextual threat interview	6 months	1 year	6
Eberhart and Hammen (2009)$_j$	104	100	27.90	18.82	United States	Community	UCLA LSI, SSM	Contextual threat interview, daily diary	2 weeks, 1 month	2 weeks, 1 month	5
Eberhart and Hammen (2010)$_j$	104	100	27.90	18.82	United States	Community	SSM	Daily diary	2 weeks	4 weeks	5
Eberhart et al. (2011)	118	100	68.60	21.28	Canada	Community	GHSS	Questionnaire	1 week	5 weeks	5
Elliot et al. (2011) Study 1	260	65.38	76.54	19.54	United States	Community	SSM	Questionnaire	6 weeks	3 months	3
Elliot et al. (2011) Study 2	159	64.78	69.81	19.95	United States	Community	RCSAEF	Questionnaire	1 month	3 months	3
Feurer et al. (2017)	129	52.70	84.50	11.83	United States	At-risk	UCLA LSI	Contextual threat interview	6 months	6 months	5
Flynn and Rudolph (2011)$_k$	156	51.50	77.80	12.41	United States	Mixed	YLSI	Contextual threat interview	1 year	1 year	6
Flynn et al. (2010)$_l$	122	61.48	63.00	19.78	United States	Community	LEI^I, LES	Noncontextual threat interview	6 weeks	9 months	5

(*table continues*)

Table 1 (*continued*)

Study author(s) (year)	N^a	% Female[a]	% White[a]	Mean age[a]	Sample source	Sample type	Life stress: Measure	Life stress: Format	Life stress: Time frame	Length of follow-up	Quality[b]
Gershon et al. (2011)	44	100	70.46	11.53	United States	At-risk	YLSI	Contextual threat interview	30 months	30 months	4
Goldstein et al. (2020)$_m$	550	100	80.50	14.38	United States	Community	SLES	Contextual threat interview	9 months	9 months	5
Goldstein et al. (2021)	917	53.90	86.70	19.10	United States	Community	UCLA LSI	Contextual threat interview	1 year	5 years	4
Hamilton and Alloy (2017)	102	79.00	74.00	19.86	United States	At-risk	SSM	Daily diary	1 month	2 weeks	6
Hamilton et al. (2013)$_n$	301	56.00	48.00	12.82	United States	Community	ALEQ, LEI^I	Noncontextual threat interview	9 months	9 months	6
Hamilton et al. (2014)$_n$	310	55.00	47.00	12.83	United States	Community	ALEQ, LEI^I	Noncontextual threat interview	9 months	9 months	4
Hamilton et al. (2017)$_d$	304	68.00	58.00	18.20	United States	Mixed	LEI^I, LES	Noncontextual threat interview	1 year	1 year	4
Hammen (1991)	60	100	80.00	38.22	United States	Mixed	UCLA LSI	Contextual threat interview	6 months	1 year	5
Hankin et al. (2005) Study 3	233	69.96	90.00	18.60	United States	Community	NLEQ	Questionnaire	2 years	2 years	5
Hankin et al. (2010)$_o$	350	57.00	53.00	14.50	United States	Community	ALEQ	Questionnaire	5 weeks	5 months	5
Harkness and Stewart(2009)$_p$	88	64.77		15.35	Canada	Mixed	LEDS	Contextual threat interview	6 months	1 year	6
Hasegawa et al. (2018)	198	58.45		20.07	Japan	Community	ISE	Questionnaire	1 month	1 month	4
Hernandez et al. (2016)$_q$	185	75.10	55.70	19.65	United States	Community	LEI^I, LES	Noncontextual threat interview	4 months	4 months	5
Herres and Kobak (2015)	132	53.00	21.00	13.00	United States	At-risk	DES	Daily diary	2 weeks	1 year	3
Herzberg et al. (1998)$_i$	137	100	45.16	17.98	United States	Community	UCLA LSI	Contextual threat interview	6 months	1 year	6
Holahan et al. (2005)	1,211	41.29	92.00	61.00	United States	Community	LISRES	Questionnaire	1 year	4 years	3
Iacovino et al. (2016)$_g$	998	55.40	71.60	59.60	United States	Community	LTE-Q	Noncontextual threat interview	6 months	6 months	5
Jenness et al. (2019)	382	59.00	70.68	13.04	Canada and United States	Community	ALEQ	Questionnaire	3 months	3 months	4
Jeronimus et al. (2017)$_c$	957	55.00		19.10	Netherlands	Mixed	SLEI	Contextual threat interview	2 years	2 years	5
Joiner et al. (2005)	169	53.85	68.00	19.00	United States	Community	NLEQ	Questionnaire	5 weeks	5 weeks	3
Judah et al. (2013)	112	74.11	87.50	19.40	United States	Community	NLEQ	Questionnaire		1 month	4
Kercher and Rapee (2009)	710	49.40	75.00	12.80	Australia	Community	ALEQ	Questionnaire	6 months	6 months	3
Kercher et al. (2009)	801	100	80.00	12.30	Australia	Community	CASE	Questionnaire	6 months	1 year	4
Kleiman and Riskind (2014)	304	82.87	50.00	21.27	United States	Community	LES	Questionnaire	6 weeks	6 weeks	5
Kleiman et al. (2013)$_r$	167	100	58.00	20.50	United States	Community	CLSI	Questionnaire	1 month	1 month	4
Kleiman et al. (2015)$_r$	209	84.20	54.00	20.58	United States	Community	CLSI	Questionnaire	1 month	1 month	5
Kushner et al. (2017)$_p$	110	74.55	89.09	16.24	United States	Clinical	LEDS	Contextual threat interview	6 months	N/A[b]	6
La Rocque et al. (2016)	503	84.49			Canada	Community	NLEQ	Questionnaire	4 months	4 months	5
Liu and Kleiman (2012)$_r$	201	84.10	53.20	20.47	United States	Community	CLSI	Questionnaire	1 month	1 month	4
Liu and Spirito (2019)	99	79.80	78.40	14.68	United States	Clinical	UCLA LSI	Contextual threat interview	6 months	6 months	7
Liu et al. (2013)$_q$	66	77.27	62.12	19.86	United States	Clinical	LEI^I, LES	Noncontextual threat interview	4 months	4 months	5
Liu, Alloy, et al. (2014)$_q$	185	75.10	55.70	19.65	United States	Community	LEI^I, LES	Noncontextual threat interview	4 months	4 months	5
Liu, Kraines, et al. (2014)$_q$	66	77.27	62.12	19.86	United States	Clinical	LEI^I, LES	Noncontextual threat interview	4 months	4 months	5
Long et al. (2022)$_s$	680	55.00	67.94	11.80	United States	Community	ALEQ	Questionnaire	3 months	3 months	5
Mackin et al. (2019)$_m$	467	100	88.00	14.39	United States	Community	SLES	Contextual threat interview	9 months	18 months	6
Mandel et al. (2018)	145	68.97	76.00	41.20	Canada	Community	UCLA LSI	Contextual threat interview	1 year	1 year, 4 years	5
Maniates et al. (2018)	116	12.10	76.72	56.23	United States	At-risk	SLEI	Contextual threat interview	2 years	2 years	4
Meiser and Esser (2019)	924	48.20		12.08	Germany	Community	SSM	Noncontextual threat interview	1 year	20 months	5
Mezulis et al. (2010)	366	50.55	90.00	11.20	United States	Community	APES	Questionnaire	1 year	2 years	5
Molz et al. (2013)$_e$	200		71.00		United States	mixed	LEI^I, LES	Noncontextual threat interview	4 months	variable	4
Morris et al. (2014) Study 1	230	54.20	82.00	11.86	United States	At-risk	LEIA	Contextual threat interview	1 year	6 years	6
Morris et al. (2014) Study 2	68	63.24		23.39	United States	Mixed	APES	Noncontextual threat interview	8 months	8 months	4
Nelson et al. (2001)$_i$	115	100	46.00	18.20	United States	Community	UCLA LSI	Contextual threat interview	6 months	1–2 years	6
Potthoff et al. (1995)	267	61.80		18.90	United States	Community	NLEQ	Questionnaire	3 weeks	3 weeks	4
Riskind et al. (2013)$_r$	99	100	50.00	21.25	United States	Community	LES	Questionnaire	1I haved ed	1 month	4
Rose et al. (2017)	429	51.95	62.76	14.52	United States	Community	DHQ	Questionnaire	1 month	9 months	5
Rudolph (2008)$_k$	148	52.70	76.60	12.41	United States	Mixed	YLSI	Contextual threat interview	1 year	1 year	6

(*table continues*)

Table 1 (*continued*)

Study author(s) (year)	*N*[a]	% Female[a]	% White[a]	Mean age[a]	Sample source	Sample type	Life stress: Measure	Life stress: Format	Life stress: Time frame	Length of follow-up	Quality[b]
Rudolph and Klein (2009)$_k$	140	53.85	78.30	12.37	United States	Mixed	YLSI	Contextual threat interview	1 year	1 year	6
Rudolph et al. (2009)$_k$	167	51.50	77.80	12.41	United States	Mixed	YLSI	Contextual threat interview	1 year	1 year	6
Rychik et al. (2021)	217	70.10		19.33	United States	Community	ALEQ-R	Questionnaire	1 week	1–10 weeks	4
Safford et al. (2007)$_l$	157	66.88		19.31	United States	Community	LEI^I, LES	Noncontextual threat interview	6 weeks	9 months	4
Sahl et al. (2009)	127	67.00	80.00		United States	Community	SSM	Daily diary	1 week	1 week	4
Schmied et al. (2016)	1,599	14.00	66.00	29.91	United States	At-risk	DRRI stressors subscale	Questionnaire	1 year	1 year	3
Shapero et al. (2013)$_o$	356	57.00	53.00	14.50	United States	Community	ALEQ	Questionnaire	5 weeks	5 months	5
Shih (2006)$_t$	99	50.51	44.00	19.08	United States	Community	UCLA LSI	Contextual threat interview	6 weeks	6 weeks	4
Shih and Auerbach (2010)	206	67.48	72.60		United States	Community	SSM	Daily diary	2 weeks	2 weeks	4
Shih and Eberhart(2008)$_t$	51	100	47.00	19.08	United States	At-risk	UCLA LSI	Contextual threat interview	6 weeks	6 weeks	4
Shih and Eberhart(2010)$_t$	99	50.51	44.00	19.08	United States	Community	UCLA LSI	Contextual threat interview	6 weeks	6 weeks	4
Shih et al. (2009)	140	50.71	84.30	9.84	United States	At-risk	CLES, LEI^I	Contextual threat interview	1 year	1 year	6
Snyder and Hankin (2016) Study 1$_s$	360	52.70	75.00	12.06	United States	Community	ALEQ, YLSI	Contextual threat interview, Questionnaire	3 months, 1.5 years	1.25 years, 1.5 years	4
Starr et al. (2012)$_f$	381	60.89	95.00	15.00	Australia	At-risk	UCLA LSI	Contextual threat interview	1 year	5 years	6
Stroud et al. (2015)$_u$	126	100	82.60	12.39	United States	Community	UCLA LSI	Contextual threat interview	1 year	1 year	5
Stroud et al. (2018)$_u$	126	100	82.60	12.39	United States	Community	UCLA LSI	Contextual threat interview	1 year	1 year	5
Stroud et al. (2023)$_u$	106	100	80.90	12.35	United States	Community	UCLA LSI	Contextual threat interview	1 year	1 year	6
Uliaszek et al. (2012)	497	69.42	48.17	16.91	United States	At-risk	UCLA LSI	Contextual threat interview	1 year	1 year	4
Wetter and Hankin (2009)$_o$	345	57.00	53.00	14.50	United States	Community	ALEQ	Questionnaire	5 weeks	5 months	4

Note. Studies with identical subscripts were drawn from the same or overlapping samples but presented unique data included in this review. ALEQ = Adolescent Life Events Questionnaire; ALEQ-R = Adolescent/Adult Life Events Questionnaire–Revised; APES = Adolescent Perceived Events Scale; CASE = Child and Adolescent Survey of Experiences; CLES = Children's Life Events Scale; CLSI = College Life Stress Inventory; DES = Daily Event Scale; DHQ = Daily Hassles Questionnaire; DHS = Daily Hassles Scale; DRRI = Deployment Risk and Resilience Inventory; GHSS = General Hassles Scale for Students; ISE = Interpersonal Stress Event Scale; LEDS = Life Events and Difficulties Schedule; LEI^I = Life Events Interview (^{I} is to differentiate from the questionnaire with the same acronym); LEI^Q = Life Events Inventory (^{Q} is to differentiate from the interview with the same acronym); LEIA = Life Events Interview for Adolescents; LES = Life Events Scale; LISRES = Life Stressors and Social Resources Inventory; LTDQ = Long-Term Difficulties Questionnaire; LTE-Q = Life Threatening Experiences Questionnaire; N/A = not applicable; NLEQ = Negative Life Events Questionnaire; RCSAEF = Revised College Students' Activity and Events Form; RLCQ = Recent Life Changes Questionnaire; SLEI = Stressful Life Events Interview; SLES = Stressful Life Events Schedule for Children and Adolescents; SSM = study-specific measure; UCLA LSI = University of California, Los Angeles Life Stress Interview; YLSI = Youth Life Stress Interview.

[a] The sample size, percentage female, percentage White, and mean age for participants included in relevant analyses, rather than of the entire study sample, are presented and incorporated in moderator analyses whenever available. For ease of presentation, whenever the sample size, mean age, or percentage female varied across multiple relevant analyses within a study, data for the cumulative number of unique participants across these analyses are presented here, and the sample size used in each analysis was retained in the relevant meta-analysis for purposes of obtaining weighted effect sizes. [b] Quality was indexed using the Newcastle–Ottawa scale. [c] For stress generation analyses, life stress and its candidate predictors of interest were assessed concurrently, with temporal separation between candidate predictors and life stress achieved by analyzing predictors with onset dates preceding life stress onset dates. [d] Here, the sample size, percentage female, percentage White, and mean age are for both members of romantic dyads combined to adhere to assumptions of independent samples in meta-analysis.

Table 2
Psychopathology in Relation to Prospective Stress Types

Psychopathology	Overall dependent stress					Interpersonal dependent stress					Noninterpersonal dependent stress					Independent stress				
	k	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{01}
Overall psychopathology[a]	58	25,432	0.52***	[0.46, 0.58]	4.65×10^{18}	32	12,150	0.50***	[0.42, 0.57]	1.55×10^{11}	18	6,733	0.36***	[0.23, 0.48]	946.00	24	11,060	0.21***	[0.15, 0.26]	2.92×10^{-5}
Internalizing psychopathology[a]	57	23,788	0.52***	[0.45, 0.59]	1.47×10^{17}	32	12,136	0.50***	[0.43, 0.58]	3.29×10^{11}	18	6,733	0.36***	[0.23, 0.50]	592.00	24	9,553	0.21***	[0.15, 0.28]	6.57×10^{-5}
Depression[a]	54	22,825	0.54***	[0.47, 0.61]	1.38×10^{17}	32	12,066	0.53***	[0.46, 0.61]	2.62×10^{12}	19	6,848	0.40***	[0.27, 0.54]	2,172.00	22	8,681	0.24***	[0.17, 0.30]	2.59×10^{-5}
Anxiety[a]	14	8,043	0.46***	[0.33, 0.59][b]	2,098.00	10	5,871	0.46***	[0.32, 0.59]	314.00	7	2,972	0.29*	[0.04, 0.53]	1.80	6	3,994	0.15	[−0.03, 0.33][c]	1.37
Posttraumatic stress disorder	4	1,599	0.38**	[0.15, 0.60]	5.39											4	1,599	0.21	[−0.01, 0.42]	.96
Externalizing psychopathology	7	4,751	0.43***	[0.22, 0.64]	16.00	3	1,978	0.27	[−0.07, 0.61]	.91	3	1,978	0.36*	[0.02, 0.70]	1.59	6	4,534	0.17**	[0.05, 0.29]	.55
Alcohol/substance use	3	3,221	0.16*	[0.02, 0.30]	.81											3	3,221	0.10**	[0.03, 0.17]	1.88
Personality disorders	3	1,989	0.52***	[0.30, 0.74]	9.94															

Note. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. Bayesian analyses: For dependent stress analyses, larger BF_{10} reflects greater support for the alternative hypothesis compared to the null hypothesis (i.e., support for stress generation). For independent stress analyses, larger BF_{01} reflect greater support for the null hypothesis compared to the alternative hypothesis (i.e., support for stress generation). Meta-analyses of predictors of life events subtypes were conducted only when the number of unique effects ≥3, so as to obtain reliable pooled estimates of effect sizes. BF = Bayes factor; CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses.
[a] Nonoverlapping confidence intervals for frequentist pooled effects between overall dependent stress and independent stress were observed, indicating a significant difference in pooled effect size. Overlapping confidence intervals do not necessarily indicate nonsignificance. [b] The lower end of the confidence interval was rounded down but exceeded .33. [c] The upper end of the confidence interval was rounded up but did not exceed .33.
* $p < .05$. ** $p < .01$. *** $p < .001$.

to subsequent dependent and independent stress. Externalizing psychopathology was not included in heterogeneity analyses due to insufficient number of unique effects ($k_{\text{Overall dependent stress}} = 7$; $k_{\text{Independent stress}} = 6$) for follow-up moderator analyses in the event that significant heterogeneity is detected. This was similarly the case for specific disorders other than depression. Across all analyses (see Table 3), significant heterogeneity was detected, regardless of the form of stress evaluated, indicating that moderator analyses were warranted. A notable pattern emerged, however, with heterogeneity being low-to-medium in all analyses of independent stress (I^2 = 44.31–52.78) and generally in the high range in analyses of dependent stress and its subtypes (I^2 = 81.04–82.74 for overall dependent stress, I^2 = 71.02 to 72.59 for interpersonal dependent stress, I^2 = 82.26–85.43 for noninterpersonal dependent stress).

Moderator analyses were first conducted for overall psychopathology in relation to overall dependent stress, interpersonal dependent stress, noninterpersonal dependent stress, and independent stress, respectively (see Table 4). These analyses were repeated for internalizing psychopathology and depression, respectively (see Tables 5 and 6).

Results of moderator analyses were mostly consistent across overall psychopathology, internalizing psychopathology, and depression, as well as across stress types. Life stress measure type, life stress measure timeframe, and study follow-up period were significant moderators in all analyses except for independent stress. That is, associations between psychopathology variables and overall dependent stress were significantly stronger for self-report questionnaires than for life stress interviews (ds = 0.60 vs. 0.40 for overall psychopathology, ds = 0.61 vs. 0.38 for internalizing psychopathology, ds = 0.61 vs. 0.41 for depression). Shorter life stress measure timeframes and study follow-up periods yielded stronger associations between psychopathology variables and overall dependent stress and its subtypes. For example, associations between psychopathology variables and overall dependent stress were significantly stronger for life stress measure timeframes ≤6 months than >6 months (ds = 0.60 vs. 0.39 for overall psychopathology, ds = 0.61 vs. 0.38 for internalizing psychopathology, ds = 0.61 vs. 0.41 for depression). Similarly, psychopathology variables were more strongly associated with overall dependent stress for study follow-up periods ≤6 months than >6 months (ds = 0.64 vs. 0.44 for overall psychopathology, ds = 0.65 vs. 0.44 for internalizing psychopathology, ds = 0.64 vs. 0.47 for depression). These patterns of findings mostly held for dependent stress subtypes.

Of note, in moderator analyses of life stress measure timeframe and length of study follow-ups, the psychopathology variables were still significantly associated with dependent stress when life stress timeframes and study follow-up periods were extended past a year, suggesting that stress generation effects are persistent, even if they diminished with time. To evaluate the long-term persistence of stress generation more directly and thereby inform the viability of stress generation as a mechanism underlying recurrence of psychopathology, we conducted sensitivity analyses for overall dependent stress, restricted to studies featuring follow-ups of 4 years or longer. Even in these analyses, significant associations between psychopathology variables and prospective dependent stress were observed (overall psychopathology: $k = 3$, $d = 0.27$, 95% CI [0.04, 0.50], $p = .02$; internalizing psychopathology: $k = 3$, $d = 0.28$, 95% CI [0.07, 0.50], $p < .01$; depression: $k = 4$, $d = 0.27$, 95% CI [0.10, 0.45], $p < .01$).

Table 3
Heterogeneity Analyses for Overall Psychopathology, Internalizing Psychopathology, and Depression in Relation to Prospective Stress Types

	Overall dependent stress				Interpersonal dependent stress				Noninterpersonal dependent stress				Independent stress			
Psychopathology	τ^2	Q	p	I^2	τ^2	Q	p	I^2	τ^2	Q	p	I^2	τ^2	Q	p	I^2
Overall psychopathology	.04	300.61	<.001	81.04	.03	113.09	<.001	72.59	.06	95.83	<.001	82.26	.01	41.30	.01	44.31
Internalizing psychopathology	.05	324.44	<.001	82.74	.03	108.27	<.001	71.37	.07	110.66	<.001	84.64	.01	48.71	<.01	52.78
Depression	.05	298.81	<.001	82.26	.03	106.99	<.001	71.02	.07	123.53	<.001	85.43	.01	38.54	.01	45.51

Note. τ^2 is an estimate of the variance of the true effect sizes (i.e., between-study variance), with larger values reflecting greater heterogeneity. Q serves as another index of heterogeneity, with its associated p value indicating whether heterogeneity is significant based on this statistic. I^2 indicates the percentage of the variance in an effect estimate that is due to heterogeneity across studies rather than sampling error, with higher values indicating greater heterogeneity.

In contrast to the aforementioned significant findings, moderator analyses were mostly nonsignificant for study sample characteristics (i.e., percentage of White participants in the study sample,[14] sample age regardless of how it was operationalized, and sample type), except in the case of gender. Here, four of nine moderator analyses were significant, with samples with more participants assigned female at birth having stronger associations between psychopathology variables and dependent stress subtypes. Finally, the three psychopathology predictor variables (i.e., operationalization, measure type, and timeframe), one life stress measure variable (i.e., operationalization of life stress), study year, and study quality were largely not significant in moderator analyses.

Psychopathology in Relation to Prospective Stress Types: Publication Bias Analyses

Analyses of publication bias were also conducted for overall psychopathology, internalizing psychopathology, and depression, respectively, in relation to subsequent dependent and independent stress (see Table 7 and Supplemental Figures S1–S3). Consistent evidence of publication bias was found for overall dependent stress and interpersonal dependent stress across all forms of psychopathology, as indicated by asymmetrical funnel plots, significant Egger's regression tests, and reduced adjusted effects size estimates in trim-and-fill analyses. Although the estimated ds were reduced from their corresponding values in Table 2 (e.g., for overall dependent stress, $d_{\text{Overall psychopathology}}$ was adjusted from 0.52 to 0.43, $d_{\text{Internalizing psychopathology}}$ was adjusted from 0.52 to 0.43, and $d_{\text{Depression}}$ was adjusted from 0.54 to 0.45), these effects remained close to medium in size.

Analyses for noninterpersonal dependent stress and independent stress were consistent across all forms of psychopathology in yielding little to no presence of publication bias. This was indicated by largely symmetrical funnel plots, nonsignificant Egger's regression tests, and trim-and-fill analyses that yielded mostly unchanged effect size estimates.

Cognitive, Other Intrapersonal, and Biological Factors in Relation to Prospective Stress Types

Results from frequentist and Bayesian meta-analyses were largely consistent with respect to evaluations of cognitive, other intrapersonal psychological, and biological predictors of dependent stress (see Table 8). Frequentist meta-analyses yielded significant associations with all dependent stress types (e.g., in the case of overall dependent stress, ds = 0.42–0.53 for cognitive vulnerability to depression variables, ds = 0.30–0.62 for other intrapersonal psychological factors, and ds = 0.21–0.34 for biological factors) but also mixed results for predictions of independent stress, with small significant associations for three of the seven predictor variables (i.e., overall cognitive vulnerability to depression, rumination, and impulsivity). Additionally, for five out of seven predictor variables (i.e., overall cognitive vulnerability to depression, negative inferential styles and/or dysfunctional attitudes, rumination, aggression/irritability, and impulsivity), associations with overall dependent stress were stronger than with independent stress, as reflected by nonoverlapping 95% CIs. Here, Bayesian meta-analyses were generally more consistent than frequentist meta-analyses in yielding mostly anecdotal support for associations with prospective independent stress (BF_{01} = 1.04–3.29).

In supplemental qualitative analyses evaluating whether significant associations with dependent stress held after covarying baseline depression and/or dependent stress, findings were very mixed for overall cognitive vulnerability to depression (support in one of three studies for overall dependent stress). When analyses are decomposed into specific cognitive vulnerabilities, rumination was no longer associated with dependent stress in almost all analyses, whereas negative inferential styles, either alone (positive findings from two of three studies) or in combination with dysfunctional attitudes (positive findings from two of three studies), mostly received support in association with prospective overall dependent stress. For other intrapersonal psychological factors, aggression/irritability (one of one study), hopelessness (two of three studies), and impulsivity (one of one study) remained significantly associated with overall dependent stress in multivariate analyses, whereas dysfunctional autonomy (one of one study) did not. None of the studies with biological factors yielded analyses covarying for baseline depression and/or dependent stress.

Interpersonal and Personality Factors in Relation to Prospective Stress Types

Frequentist and Bayesian meta-analytic results are broadly consistent but generally weaker in Bayesian meta-analyses with

[14] Median values in moderator analyses ranged from 62.76% in the case of depression and noninterpersonal dependent stress to 77.5% in the case of overall psychopathology and independent stress.

Table 4
Moderator Analyses for Overall Psychopathology in Relation to Prospective Stress Types

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Overall dependent stress							
Percentage female	54	23,814	<.01	<.01			.23
Percentage White	38	17,151	<.01	<.01			.15
Age at start of study (continuous)	46	21,778	<.01	<.01			.27
Age at start of study (categorical)							.86
Adult	31	12,291			0.52	[0.43, 0.61]	<.001
Child and adolescent	25	12,900			0.51	[0.41, 0.61]	<.001
Age at end of study (categorical)							.76
Adult	32	12,428			0.53	[0.44, 0.62]	<.001
Child and adolescent	25	12,900			0.51	[0.41, 0.61]	<.001
Sample type							.05
Clinical/at-risk	10	4,461			0.39	[0.24, 0.55]	<.001
Community	39	18,775			0.56	[0.49, 0.64]	<.001
Psychopathology measure operationalization							<.01
Categorical (diagnosis)	8	2,392			0.35	[0.21, 0.48]	<.001
Continuous (symptoms)	43	20,826			0.55	[0.48, 0.63]	<.001
Psychopathology measure type							.25
Interview	14	2,347			0.45	[0.28, 0.61]	<.001
Questionnaire	41	20,548			0.55	[0.48, 0.63]	<.001
Psychopathology measure timeframe							.34
Current	48	22,235			0.53	[0.46, 0.61]	<.001
Lifetime	5	1,214			0.40	[0.15, 0.66]	<.01
Life stress measure type[a]							<.01
Interview	27	8,356			0.40	[0.31, 0.49]	<.001
Questionnaire	28	16,507			0.60	[0.51, 0.69]	<.001
Life stress operationalization							.12
Life stress impact ratings	21	5,148			0.44	[0.33, 0.56]	<.001
Life stress frequency count	37	20,284			0.55	[0.48, 0.63]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.001
≤6 months	31	13,292			0.60	[0.52, 0.68]	<.001
>6 months	24	11,323			0.39	[0.30, 0.48]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	34	14,137			0.59	[0.52, 0.67]	<.001
≥1 year	21	10,478			0.37	[0.28, 0.47]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.01
≤6 months	24	9,143			0.64	[0.54, 0.74]	<.001
>6 months	33	15,584			0.44	[0.37, 0.52]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.01
<1 year	28	10,107			0.61	[0.52, 0.70]	<.001
≥1 year	29	14,620			0.45	[0.36, 0.53]	<.001
Study year	58	25,432	−.01	<.01			.04
Study quality	56	24,812	−.05	.04			.16
Interpersonal dependent stress							
Percentage female	29	10,842	<.01	<.01			<.05
Percentage white	24	8,869	<.01	<.01			<.05
Age at start of study (continuous)	27	10,752	<.01	<.01			.47
Age at start of study (categorical)							.87
Adult	16	5,540			0.50	[0.39, 0.61]	<.001
Child and adolescent	14	6,369			0.48	[0.37, 0.60]	<.001
Age at end of study (categorical)							.70
Adult	17	5,677			0.52	[0.40, 0.63]	<.001
Child and adolescent	14	6,369			0.48	[0.37, 0.60]	<.001
Sample type							.11
Clinical/at-risk	5	1,528			0.38	[0.19, 0.57]	<.001
Community	22	9,462			0.55	[0.46, 0.65]	<.001
Psychopathology measure operationalization							<.01
Categorical (diagnosis)	6	2,167			0.33	[0.24, 0.42]	<.001
Continuous (symptoms)	23	9,180			0.52	[0.43, 0.61]	<.001
Psychopathology measure type							.74
Interview	10	2,158			0.48	[0.34, 0.63]	<.001
Questionnaire	21	9,347			0.51	[0.42, 0.61]	<.001
Psychopathology measure timeframe							.26
Current	28	10,998			0.51	[0.43, 0.60]	<.001
Lifetime	3	989			0.38	[0.16, 0.60]	<.001

(*table continues*)

Table 4 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Life stress measure type[a]							<.01
Interview	18	4,851			0.39	[0.29, 0.49]	<.001
Questionnaire	13	7,167			0.59	[0.48, 0.70]	<.001
Life stress operationalization							.11
Life stress impact ratings	14	3,430			0.41	[0.29, 0.54]	<.001
Life stress frequency count	18	8,720			0.54	[0.45, 0.64]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.001
≤6 months	18	6,648			0.61	[0.51, 0.71]	<.001
>6 months	12	4,797			0.30	[0.23, 0.38]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	19	6,958			0.61	[0.51, 0.70]	<.001
≥1 year	11	4,487			0.28	[0.21, 0.35]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.01
≤6 months	14	4,454			0.62	[0.51, 0.72]	<.001
>6 months	17	6,991			0.41	[0.32, 0.50]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.001
<1 year	16	5,193			0.61	[0.52, 0.71]	<.001
≥1 year	15	6,252			0.37	[0.28, 0.46]	<.001
Study year	32	12,150	−.01	.01			.20
Study quality	32	12,150	−.04	.06			.46
Noninterpersonal dependent stress							
Percentage female	16	5,579	<.01	<.01			<.05
Percentage White	14	4,805	<.01	<.01			.24
Age at start of study (continuous)	16	5,579	<.01	<.01			.59
Age at start of study (categorical)/age at end of study (categorical)[b]							.76
Adult	10	4,451			0.34	[0.18, 0.49]	<.001
Child and adolescent	8	2,282			0.38	[0.15, 0.61]	<.01
Sample type							.62
Clinical/at-risk	2	845			0.29	[−0.02, 0.61]	.07
Community	12	4,913			0.38	[0.22, 0.54]	<.001
Psychopathology measure operationalization							.48
Categorical (diagnosis)	3	1,535			0.28	[0.11, 0.46]	<.01
Continuous (symptoms)	13	4,532			0.37	[0.20, 0.54]	<.001
Psychopathology measure type							.35
Interview	6	1,346			0.26	[0.04, 0.48]	<.05
Questionnaire	11	4,742			0.39	[0.22, 0.56]	<.001
Psychopathology measure timeframe							.06
Current	15	5,680			0.38	[0.23, 0.52]	<.001
Lifetime	2	890			0.18	[0.05, 0.32]	<.01
Life stress measure type[a]							<.001
Interview	11	3,612			0.21	[0.10, 0.32]	<.001
Questionnaire	7	3,121			0.54	[0.38, 0.69]	<.001
Life stress operationalization							.05
Life stress impact ratings	7	2,200			0.21	[0.02, 0.39]	.03
Life stress frequency count	11	4,533			0.43	[0.29, 0.57]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.05
≤6 months	6	1,940			0.48	[0.29, 0.66]	<.001
>6 months	11	4,142			0.24	[0.12, 0.36]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							.10
<1 year	7	2,241			0.43	[0.26, 0.61]	<.001
≥1 year	10	3,841			0.25	[0.11, 0.38]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.05
≤6 months	6	2,162			0.54	[0.34, 0.74]	<.001
>6 months	11	3,866			0.27	[0.14, 0.40]	<.001
Study follow-up period (<1 year vs. ≥1 year)							.07
<1 year	8	2,892			0.48	[0.30, 0.65]	<.001
≥1 year	9	3,136			0.26	[0.11, 0.42]	<.01
Study year	18	6,733	<.01	.01			.88
Study quality	18	6,733	−.01	.12			.91
Independent stress							
Percentage female	24	11,060	<.01	<.01			.11
Percentage White	20	9,999	<.01	<.01			.11
Age at start of study (continuous)	20	9,940	<.01	<.01			.09
Age at start of study (categorical)/age at end of study (categorical)[b]							<.05

(*table continues*)

Table 4 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Adult	14	7,049			0.16	[0.08, 0.23]	<.001
Child and adolescent	10	4,011			0.28	[0.22, 0.35]	<.001
Sample type							.22
Clinical/at-risk	7	3,778			0.26	[0.19, 0.33]	<.001
Community	12	5,576			0.20	[0.12, 0.27]	<.001
Psychopathology measure operationalization							.22
Categorical (diagnosis)	6	1,796			0.13	[0.00, 0.25][c]	<.05
Continuous (symptoms)	15	7,952			0.22	[0.15, 0.28]	<.001
Psychopathology measure type							.56
Interview	7	1,393			0.19	[0.09, 0.30]	<.001
Questionnaire	14	7,293			0.23	[0.16, 0.30]	<.001
Psychopathology measure timeframe							.96
Current	16	8,125			0.19	[0.11, 0.26]	<.001
Lifetime	4	1,115			0.19	[0.07, 0.31]	<.01
Life stress measure type[a]							.62
Interview	16	6,049			0.19	[0.12, 0.26]	<.001
Questionnaire	7	4,884			0.22	[0.12, 0.32]	<.001
Life stress operationalization							.78
Life stress impact ratings	12	3,776			0.20	[0.10, 0.29]	<.001
Life stress frequency count	12	7,284			0.21	[0.14, 0.29]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							.94
≤6 months	9	3,755			0.20	[0.14, 0.27]	<.001
>6 months	15	7,305			0.21	[0.12, 0.29]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							.60
<1 year	12	4,591			0.22	[0.16, 0.28]	<.001
≥1 year	12	6,469			0.19	[0.10, 0.28]	<.001
Study follow-up period (≤6 months vs. >6 months)							.46
≤6 months	6	2,761			0.18	[0.10, 0.25]	<.001
>6 months	17	7,594			0.22	[0.14, 0.29]	<.001
Study follow-up period (<1 year vs. ≥1 year)							.51
<1 year	9	3,287			0.18	[0.11, 0.25]	<.001
≥1 year	14	7,068			0.22	[0.13, 0.30]	<.001
Study year	24	11,060	<.01	.01			.68
Study quality	24	11,060	−.02	.03			.49

Note. CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses; *SE* = standard error. *b* and *SE* are reported for continuous moderator variables, and *d* and 95% CI are reported for categorical moderator variables. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis.

[a] Contextual threat interviews and noncontextual threat interviews were combined for the purpose of these analyses because they were found not to differ in the strength of their effects (overall dependent stress $p = .33$; dependent interpersonal stress $p = .44$; dependent noninterpersonal stress $p = .94$; independent stress $p = .88$). [b] Age as a categorical variable did not differ between baseline and follow-up for any of the studies, and the associated moderator analyses are therefore identical. [c] The lower end of the confidence interval was rounded down but exceeded 0.00.

less data (e.g., co-rumination, excessive reassurance-seeking, and perfectionism; see Table 9). With the exception of extraversion, all interpersonal and personality factors were significantly associated with all stress types in frequentist meta-analyses, with *d*s for overall dependent stress ranging from 0.28 for co-rumination as a predictor to 0.53 for excessive reassurance-seeking as a predictor. Again, in cases where analyses of independent stress were viable, the predictor variables (i.e., overall interpersonal vulnerability and neuroticism) were significantly more associated with overall dependent stress than with independent stress, as reflected by nonoverlapping 95% CIs.

In supplemental qualitative analyses, overall interpersonal vulnerability received mixed support in relation to overall dependent stress after covarying baseline depression and dependent stress (significant association was maintained in one of two studies). For all other interpersonal and personality factors, positive associations held in one of one study for co-rumination, one of two studies for excessive reassurance-seeking, one of four studies for rejection sensitivity, and three of three studies for neuroticism. None of the studies with negative attachment styles or perfectionism included multivariate analyses covarying baseline depression and/or dependent stress.

Other Risk Factors and Protective Factors in Relation to Prospective Stress Types

For the remaining risk factors—childhood maltreatment/adversity and maternal depression—frequentist meta-analyses yielded significant associations with overall dependent stress and independent stress (except for childhood maltreatment/adversity in relation to independent stress). *d*s were 0.35 for childhood maltreatment/adversity and 0.33 for maternal depression in relation to overall dependent stress. As for protective factors, frequentist meta-analyses produced a significant association with dependent stress only in the case of overall protective factors ($d = -0.29$). This variable was also significantly more associated with overall dependent stress than with independent stress, as indicated by nonoverlapping 95% CIs.

Table 5
Moderator Analyses for Internalizing Psychopathology in Relation to Prospective Stress Types

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Overall dependent stress							
Percentage female	53	22,170	<.01	<.01			.19
Percentage White	39	15,853	<.01	<.01			.09
Age at start of study (continuous)	47	20,480	<.01	<.01			.45
Age at start of study (categorical)/age at end of study (categorical)[a]							.36
Adult	31	10,784			0.55	[0.46, 0.64]	<.001
Child and adolescent	25	12,900			0.48	[0.38, 0.59]	<.001
Sample type							.06
Clinical/at-risk	10	4,461			0.39	[0.22, 0.56]	<.001
Community	38	17,131			0.57	[0.49, 0.66]	<.001
Psychopathology measure operationalization							.14
Categorical (diagnosis)	9	2,515			0.42	[0.26, 0.59]	<.001
Continuous (symptoms)	43	19,359			0.56	[0.48, 0.64]	<.001
Psychopathology measure type							.24
Interview	15	2,595			0.44	[0.28, 0.60]	<.001
Questionnaire	41	20,548			0.55	[0.47, 0.62]	<.001
Psychopathology measure timeframe							.35
Current	48	20,730			0.53	[0.46, 0.60]	<.001
Lifetime	5	1,214			0.40	[0.14, 0.66]	<.01
Life stress measure type[b]							<.001
Interview	26	6,712			0.38	[0.28, 0.47]	<.001
Questionnaire	28	16,507			0.61	[0.52, 0.70]	<.001
Life stress operationalization							.03
Life stress impact ratings	21	5,134			0.41	[0.29, 0.52]	<.001
Life stress frequency count	36	18,654			0.57	[0.49, 0.65]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.001
≤6 months	30	11,648			0.61	[0.52, 0.70]	<.001
>6 months	24	11,323			0.38	[0.28, 0.47]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	33	12,493			0.61	[0.52, 0.69]	<.001
≥1 year	21	10,478			0.36	[0.25, 0.46]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.01
≤6 months	23	7,513			0.65	[0.55, 0.76]	<.001
>6 months	33	15,570			0.44	[0.36, 0.52]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.01
<1 year	27	8,477			0.62	[0.52, 0.72]	<.001
≥1 year	29	14,606			0.44	[0.35, 0.52]	<.001
Study year	57	23,788	−.01	.01			<.05
Study quality	56	23,478	−.07	.04			.08
Interpersonal dependent stress							
Percentage female	29	10,828	<.01	<.01			.11
Percentage White	25	8,891	<.01	<.01			<.05
Age at start of study (continuous)	28	10,774	<.01	<.01			.67
Age at start of study (categorical)/age at end of study (categorical)[a]							.72
Adult	17	5,663			0.52	[0.42, 0.62]	<.001
Child and adolescent	14	6,369			0.49	[0.37, 0.61]	<.001
Sample type							.07
Clinical/at-risk	5	1,528			0.38	[0.21, 0.54]	<.001
Community	22	9,448			0.55	[0.46, 0.65]	<.001
Psychopathology measure operationalization							<.05
Categorical (diagnosis)	7	2,290			0.37	[0.27, 0.47]	<.001
Continuous (symptoms)	24	9,343			0.53	[0.43, 0.62]	<.001
Psychopathology measure type							.44
Interview	10	2,144			0.46	[0.34, 0.58]	<.001
Questionnaire	21	9,347			0.52	[0.43, 0.62]	<.001
Psychopathology measure timeframe							.25
Current	28	11,024			0.51	[0.43, 0.60]	<.001
Lifetime	3	989			0.39	[0.20, 0.58]	<.001
Life stress measure type[b]							<.01
Interview	18	4,837			0.39	[0.30, 0.47]	<.001
Questionnaire	13	7,167			0.60	[0.48, 0.72]	<.001
Life stress operationalization							.02
Life stress impact ratings	14	3,416			0.39	[0.29, 0.50]	<.001
Life stress frequency count	18	8,720			0.56	[0.46, 0.66]	<.001

(*table continues*)

Table 5 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Life stress measure timeframe (≤6 months vs. >6 months)							<.001
≤6 months	18	6,634			0.62	[0.52, 0.73]	<.001
>6 months	12	4,797			0.32	[0.25, 0.38]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	19	6,944			0.61	[0.52, 0.71]	<.001
≥1 year	11	4,487			0.30	[0.23, 0.36]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.001
≤6 months	14	4,454			0.64	[0.52, 0.75]	<.001
>6 months	17	6,977			0.40	[0.32, 0.48]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.001
<1 year	16	5,193			0.63	[0.53, 0.73]	<.001
≥1 year	15	6,238			0.36	[0.28, 0.44]	<.001
Study year	32	12,136	<.01	.01			.39
Study quality	32	12,136	−.06	.06			.28
Noninterpersonal dependent stress							
Percentage female	16	5,579	<.01	<.01			<.05
Percentage White	14	4,805	<.01	<.01			.14
Age at start of study (continuous)	16	5,579	<.01	<.01			.58
Age at start of study (categorical)/age at end of study (categorical)[a]							.74
Adult	10	4,451			0.34	[0.18, 0.50]	<.001
Child and adolescent	8	2,282			0.39	[0.14, 0.64]	<.01
Sample type							.55
Clinical/at-risk	2	845			0.25	[−0.16, 0.66]	.23
Community	12	4,913			0.39	[0.22, 0.55]	<.001
Psychopathology measure operationalization							.35
Categorical (diagnosis)	3	1,535			0.25	[0.01, 0.49]	<.05
Continuous (symptoms)	14	4,695			0.39	[0.22, 0.56]	<.001
Psychopathology measure type							.40
Interview	6	1,346			0.27	[0.01, 0.52]	<.05
Questionnaire	11	4,742			0.40	[0.22, 0.58]	<.001
Psychopathology measure timeframe							<.05
Current	16	5,843			0.39	[0.25, 0.53]	<.001
Lifetime	2	890			0.12	[−0.04, 0.29]	.14
Life stress measure type[b]							<.01
Interview	11	3,612			0.21	[0.09, 0.33]	<.001
Questionnaire	7	3,121			0.55	[0.38, 0.72]	<.001
Life stress operationalization							.03
Life stress impact ratings	7	2,200			0.19	[<0.01, 0.37]	<.05
Life stress frequency count	11	4,533			0.45	[0.30, 0.60]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.05
≤6 months	6	1,940			0.51	[0.30, 0.72]	<.001
>6 months	11	4,142			0.23	[0.11, 0.35]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							.06
<1 year	7	2,241			0.46	[0.27, 0.66]	<.001
≥1 year	10	3,841			0.24	[0.10, 0.37]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.05
≤6 months	6	2,162			0.57	[0.36, 0.79]	<.001
>6 months	11	3,866			0.27	[0.14, 0.40]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.05
<1 year	8	2,892			0.50	[0.32, 0.69]	<.001
≥1 year	9	3,136			0.26	[0.11, 0.42]	<.001
Study year	18	6,733	<.01	.01			.85
Study quality	18	6,733	−.02	.13			.88
Independent stress							
Percentage female	24	9,553	<.01	<.01			.33
Percentage White	21	8,528	<.01	<.01			.14
Age at start of study (continuous)	21	8,469	<.01	<.01			.09
Age at start of study (categorical)/age at end of study (categorical)[a]							.21
Adult	14	5,542			0.18	[0.08, 0.29]	<.001
Child and adolescent	10	4,011			0.26	[0.20, 0.33]	<.001
Sample type							.46
Clinical/at-risk	7	3,778			0.26	[0.15, 0.37]	<.001
Community	12	4,069			0.21	[0.12, 0.30]	<.001
Psychopathology measure operationalization							.40
Categorical (diagnosis)	7	1,919			0.16	[0.03, 0.29]	<.05
Continuous (symptoms)	14	6,322			0.23	[0.14, 0.32]	<.001

(*table continues*)

Table 5 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Psychopathology measure type							.72
Interview	9	1,615			0.21	[0.11, 0.30]	<.001
Questionnaire	14	7,293			0.23	[0.15, 0.31]	<.001
Psychopathology measure timeframe							.98
Current	16	6,594			0.19	[0.10, 0.28]	<.001
Lifetime	4	1,115			0.19	[0.07, 0.31]	<.01
Life stress measure type[b]							.59
Interview	16	4,542			0.19	[0.12, 0.25]	<.001
Questionnaire	7	4,884			0.23	[0.10, 0.35]	<.001
Life stress operationalization							.54
Life stress impact ratings	13	3,899			0.19	[0.11, 0.27]	<.001
Life stress frequency count	11	5,654			0.23	[0.13, 0.33]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							.76
≤6 months	9	2,248			0.23	[0.14, 0.31]	<.001
>6 months	15	7,305			0.21	[0.12, 0.30]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							.42
<1 year	12	3,084			0.24	[0.17, 0.32]	<.001
≥1 year	12	6,469			0.19	[0.09, 0.29]	<.001
Study follow-up period (≤6 months vs. >6 months)							.47
≤6 months	5	1,131			0.17	[0.05, 0.29]	<.01
>6 months	18	7,717			0.22	[0.14, 0.31]	<.001
Study follow-up period (<1 year vs. ≥1 year)							.50
<1 year	8	1,657			0.18	[0.08, 0.28]	<.001
≥1 year	15	7,191			0.23	[0.13, 0.32]	<.001
Study year	24	9,553	<.01	.01			.88
Study quality	24	9,553	−.03	.03			.30

Note. CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses; *SE* = standard error. *b* and *SE* are reported for continuous moderator variables, and *d* and 95% CI are reported for categorical moderator variables. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis.
[a] Age as a categorical variable did not differ between baseline and follow-up for any of the studies, and the associated moderator analyses are therefore identical. [b] Contextual threat interviews and noncontextual threat interviews were combined for the purpose of these analyses because they were found not to differ in the strength of their effects (overall dependent stress $p = .78$; dependent interpersonal stress $p = .97$; dependent noninterpersonal stress $p = .65$; independent stress $p = .70$).

All frequentist and Bayesian analyses for these other risk and protective factors are presented in Table 10.

As for supplemental findings, none of the childhood maltreatment/adversity studies included analyses covarying baseline depression and/or dependent stress. Of the three for maternal depression, one reported a significant association with overall dependent stress in an analysis with covariates. In terms of protective factors, four of five relevant studies yielded significant associations with overall dependent stress in multivariate analyses.

Sociodemographic Characteristics in Relation to Prospective Stress Types

Frequentist and Bayesian meta-analyses of sociodemographic characteristics in association with subsequent life stress are detailed in Table 11. Regarding age, with the exception of a significant small-to-medium ($d = 0.28$) relationship with noninterpersonal stress in frequentist meta-analyses, there was little evidence of a predictive relationship with stress types, and Bayesian meta-analyses produced consistent findings. Although gender identity was significantly associated with most stress types in frequentist meta-analyses, the strength of these associations was small (*d*s = 0.08–0.20). No associations between race and prospective stress types were found in any analyses. Finally, SES was negatively associated with both dependent and independent stress (*d*s = −0.38), indicating higher SES was protective against these outcomes.[15]

In supplemental qualitative analyses, no studies covaried baseline depression and/or dependent stress in analyses of gender in relation to overall dependent stress. In the two studies of SES that did include at least one of these covariates, SES was not predictive of overall dependent stress in the relevant multivariate models.

Qualitative Synthesis

Evaluating Stress Generation as a Mediator of Clinical Symptom Outcomes

Of the 102 studies included in the meta-analysis, 44 studies (43.14%) evaluated whether stress generation mediated the relationship between psychopathology or associated vulnerabilities and subsequent psychopathology (see Table 12). All but one study provided evaluations of this component of stress generation within full-temporal mediation analyses (i.e., the predictor temporally

[15] SES was operationalized in different ways across these studies. Four studies based SES on family income (Chan et al., 2014; Feurer et al., 2017; Iacovino et al., 2016; Stroud et al., 2018), one used eligibility for free lunch (Hamilton et al., 2013), and one used an income-to-needs ratio (Herres & Kobak, 2015).

Table 6
Moderator Analyses for Depression in Relation to Prospective Stress Types

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Overall dependent stress							
Percentage female	50	21,207	<.01	<.01			.43
Percentage White	38	15,847	<.01	<.01			.19
Age at start of study (continuous)	46	18,013	<.01	<.01			.33
Age at start of study (categorical)/age at end of study (categorical)[a]							.78
Adult	30	10,778			0.55	[0.46, 0.64]	<.001
Child and adolescent	23	11,943			0.53	[0.41, 0.64]	<.001
Sample type							<.05
Clinical/at-risk	9	4,291			0.40	[0.24, 0.56]	<.001
Community	38	17,241			0.58	[0.50, 0.66]	<.001
Psychopathology measure operationalization							.16
Categorical (diagnosis)	9	2,515			0.43	[0.27, 0.59]	<.001
Continuous (symptoms)	44	19,807			0.56	[0.48, 0.63]	<.001
Psychopathology measure type							.12
Interview	14	2,479			0.44	[0.27, 0.61]	<.001
Questionnaire	38	19,346			0.58	[0.51, 0.66]	<.001
Psychopathology measure timeframe							.38
Current	47	20,724			0.54	[0.46, 0.61]	<.001
Lifetime	5	1,214			0.43	[0.19, 0.66]	<.001
Life stress measure type[b]							<.01
Interview	23	5,639			0.41	[0.30, 0.52]	<.001
Questionnaire	28	16,617			0.61	[0.53, 0.70]	<.001
Life stress operationalization							.08
Life stress impact ratings	18	4,061			0.44	[0.30, 0.57]	<.001
Life stress frequency count	36	18,764			0.58	[0.50, 0.66]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.01
≤6 months	30	11,758			0.61	[0.52, 0.70]	<.001
>6 months	21	10,250			0.41	[0.31, 0.51]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	33	12,603			0.61	[0.53, 0.70]	<.001
≥1 year	18	9,405			0.38	[0.27, 0.49]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.05
≤6 months	23	7,623			0.64	[0.54, 0.74]	<.001
>6 months	30	14,497			0.47	[0.38, 0.56]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.05
<1 year	27	8,587			0.62	[0.52, 0.71]	<.001
≥1 year	26	13,533			0.47	[0.37, 0.56]	<.001
Study year	54	22,825	−.01	.01			.06
Study quality	53	22,515	−.06	.04			.12
Interpersonal dependent stress							
Percentage female	29	10,758	<.01	<.01			.18
Percentage White	25	8,891	<.01	<.01			.11
Age at start of study (continuous)	28	10,704	<.01	<.01			.50
Age at start of study (categorical)/age at end of study (categorical)[a]							.82
Adult	17	5,663			0.55	[0.45, 0.65]	<.001
Child and adolescent	14	6,299			0.53	[0.40, 0.65]	<.001
Sample type							.44
Clinical/at-risk	5	1,528			0.48	[0.30, 0.67]	<.001
Community	22	9,448			0.56	[0.47, 0.66]	<.001
Psychopathology measure operationalization							.34
Categorical (diagnosis)	7	2,290			0.47	[0.34, 0.60]	<.001
Continuous (symptoms)	24	9,273			0.55	[0.45, 0.64]	<.001
Psychopathology measure type							.82
Interview	10	2,074			0.55	[0.46, 0.64]	<.001
Questionnaire	21	9,347			0.53	[0.44, 0.63]	<.001
Psychopathology measure timeframe							.95
Current	28	10,954			0.53	[0.45, 0.62]	<.001
Lifetime	3	989			0.54	[0.37, 0.71]	<.001
Life stress measure type[b]							<.05
Interview	18	4,767			0.45	[0.35, 0.55]	<.001
Questionnaire	13	7,167			0.61	[0.49, 0.72]	<.001
Life stress operationalization							.18
Life stress impact ratings	14	3,346			0.47	[0.34, 0.59]	<.001
Life stress frequency count	18	8,720			0.57	[0.47, 0.67]	<.001

(*table continues*)

Table 6 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Life stress measure timeframe (≤6 months vs. >6 months)							<.01
≤6 months	18	6,634			0.63	[0.53, 0.73]	<.001
>6 months	12	4,727			0.39	[0.28, 0.49]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.001
<1 year	19	6,944			0.63	[0.53, 0.72]	<.001
≥1 year	11	4,417			0.37	[0.26, 0.47]	<.001
Study follow-up period (≤6 months vs. >6 months)							<.01
≤6 months	14	4,454			0.64	[0.53, 0.75]	<.001
>6 months	17	6,907			0.43	[0.34, 0.52]	<.001
Study follow-up period (<1 year vs. ≥1 year)							<.001
<1 year	16	5,193			0.64	[0.54, 0.74]	<.001
≥1 year	15	6,168			0.39	[0.30, 0.47]	<.001
Study year	32	12,066	−.01	.01			.36
Study quality	32	12,066	−.04	.06			.45
Noninterpersonal dependent stress							
Percentage female	17	5,694	<.01	<.01			<.01
Percentage White	15	4,920	<.01	<.01			<.05
Age at start of study (continuous)	17	5,694	<.01	<.01			.47
Age at start of study (categorical)							.55
Adult	10	4,451			0.34	[0.17, 0.51]	<.001
Child and adolescent	8	2,282			0.44	[0.18, 0.69]	<.001
Age at end of study (categorical)							.70
Adult	11	4,566			0.38	[0.21, 0.54]	<.001
Child and adolescent	8	2,282			0.44	[0.18, 0.69]	<.001
Sample type							.39
Clinical/at-risk	2	845			0.25	[−0.17, 0.67]	.25
Community	13	5,028			0.45	[0.28, 0.61]	<.001
Psychopathology measure operationalization							.20
Categorical (diagnosis)	3	1,535			0.24	[0.00, 0.49][c]	.05
Continuous (symptoms)	15	4,810			0.44	[0.27, 0.61]	<.001
Psychopathology measure type							.55
Interview	7	1,461			0.34	[0.07, 0.61]	<.05
Questionnaire	11	4,742			0.44	[0.25, 0.62]	<.001
Psychopathology measure timeframe							<.01
Current	17	5,958			0.43	[0.29, 0.58]	<.001
Lifetime	2	890			0.12	[−0.06, 0.30]	.19
Life stress measure type[b]							<.05
Interview	12	3,727			0.29	[0.14, 0.43]	<.001
Questionnaire	7	3,121			0.56	[0.39, 0.73]	<.001
Life stress operationalization							.07
Life stress impact ratings	8	2.315			0.26	[0.05, 0.47]	.02
Life stress frequency count	11	4,533			0.49	[0.35, 0.64]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							<.05
≤6 months	7	2,055			0.55	[0.35, 0.75]	<.001
>6 months	11	4,142			0.27	[0.13, 0.41]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							<.05
<1 year	8	2,356			0.54	[0.37, 0.72]	<.001
≥1 year	10	3,841			0.24	[0.10, 0.39]	<.01
Study follow-up period (≤6 months vs. >6 months)							.08
≤6 months	6	2,162			0.58	[0.35, 0.80]	<.001
>6 months	12	3,981			0.34	[0.20, 0.49]	<.001
Study follow-up period (<1 year vs. ≥1 year)							.06
<1 year	8	2,892			0.55	[0.38, 0.71]	<.001
≥1 year	10	3,251			0.32	[0.15, 0.48]	<.001
Study year	19	6,848	−.01	.01			.24
Study quality	19	6,848	.08	.12			.48
Independent stress							
Percentage female	22	8,681	<.01	<.01			.72
Percentage White	21	8,613	<.01	<.01			.17
Age at start of study (continuous)	21	8,554	<.01	<.01			<.05
Age at start of study (categorical)/age at end of study (categorical)[a]							.13
Adult	14	5,627			0.21	0.11, 0.30]	<.001
Child and adolescent	8	3,054			0.30	0.22, 0.37]	<.001
Sample type							<.05
Clinical/at-risk	6	3,662			0.33	[0.26, 0.40]	<.001
Community	13	4,270			0.23	[0.15, 0.30]	<.001

(*table continues*)

Table 6 (*continued*)

Moderator	*k*	*N*	*b*	*SE*	*d*	95% CI	*p*
Psychopathology measure operationalization							.70
Categorical (diagnosis)	7	1,919			0.22	[0.06, 0.37]	<.01
Continuous (symptoms)	15	6,762			0.25	[0.18, 0.32]	<.001
Psychopathology measure type							.40
Interview	8	1,499			0.30	[0.19, 0.40]	<.001
Questionnaire	12	6,182			0.24	[0.16, 0.32]	<.001
Psychopathology measure timeframe							.24
Current	16	6,679			0.21	[0.13, 0.29]	<.001
Lifetime	4	1,115			0.30	[0.18, 0.42]	<.001
Life stress measure type[b]							.86
Interview	13	3,469			0.24	[0.15, 0.32]	<.001
Questionnaire	8	5,085			0.23	[0.12, 0.33]	<.001
Life stress operationalization							.91
Life stress impact ratings	10	2,826			0.23	[0.12, 0.35]	<.001
Life stress frequency count	12	5,855			0.24	[0.16, 0.32]	<.001
Life stress measure timeframe (≤6 months vs. >6 months)							.75
≤6 months	10	2,449			0.23	[0.15, 0.31]	<.001
>6 months	12	6,232			0.25	[0.15, 0.34]	<.001
Life stress measure timeframe (<1 year vs. ≥1 year)							.59
<1 year	13	3,285			0.26	[0.19, 0.33]	<.001
≥1 year	9	5,396			0.22	[0.11, 0.34]	<.001
Study follow-up period (≤6 months vs. >6 months)							.35
≤6 months	6	1,332			0.18	[0.07, 0.29]	<.01
>6 months	15	6,644			0.25	[0.16, 0.33]	<.001
Study follow-up period (<1 year vs. ≥1 year)							.69
<1 year	9	1,858			0.21	[0.12, 0.30]	<.001
≥1 year	12	6,118			0.24	[0.14, 0.34]	<.001
Study year	22	8,681	<.01	.01			.79
Study quality	22	8,681	−.02	.03			.65

Note. *b* and *SE* are reported for continuous moderator variables, and *d* and 95% CI are reported for categorical moderator variables. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses; *SE* = standard error.
[a] Age as a categorical variable did not differ between baseline and follow-up for any of the studies, and the associated moderator analyses are therefore identical. [b] Contextual threat interviews and noncontextual threat interviews were combined for the purpose of these analyses because they were found not to differ in the strength of their effects (overall dependent stress $p = .94$; dependent interpersonal stress $p = .73$; dependent noninterpersonal stress $p = .56$; independent stress $p = .51$). [c] The lower end of the confidence interval was rounded up but did not exceed 0.00.

preceding the mediator, which in turn temporally precedes the outcome), the one exception being a study that employed a half-longitudinal analytical design, with the association between life stress and depressive symptom outcome evaluated contemporaneously with each other (Hamilton & Alloy, 2017). Of note, however, only 18 studies included stress at an earlier timepoint (baseline) when examining stress generation as a mediator, and only 11 studies covaried for baseline psychopathology in mediational analyses where clinical symptoms were the outcomes.

The majority of mediational studies focused on clinical symptoms such as depression or anxiety as the primary predictor ($k = 18$), dependent interpersonal stress as the primary mediator ($k = 28$), and depression symptoms as the primary outcome of the stress generation relationship ($k = 37$). Anxiety symptoms were the secondary clinical outcome of interest ($k = 8$), with most studies finding that stress generation also predicted anxiety symptoms. All studies focused on symptoms rather than clinical disorders as both the independent predictor and dependent outcomes. The overwhelming number of studies ($k = 30$) also focused on children and adolescents. Notably, most studies tested multiple pathways of mediation, which included several independent predictors, types of life stress (e.g., dependent and independent stress, interpersonal dependent and achievement-based stress), and outcomes (depression symptoms, anxiety symptoms). Overall, 95% of studies ($k = 41$) found evidence of at least one significant mediational pathway in which overall dependent stress or dependent interpersonal stress, but not independent stress, mediated the relationship between psychopathology or biopsychosocial vulnerabilities and subsequent psychopathology.

When this is broken down, clinical symptoms were the primary predictor of stress generation in mediational analyses, consistent with the stress generation hypothesis as originally conceived (Hammen, 1991). Indeed, 12 separate studies had depression symptoms as a predictor. Several studies also examined social anxiety ($k = 1$), ADHD ($k = 3$), PTSD ($k = 1$), and personality disorders ($k = 2$) as predictors in the stress generation mediational models, with nearly all finding significant support for mediation by stress generation. Of note, only one study evaluated the full mediational study with externalizing symptoms (i.e., ADHD) as the outcome.

Many studies ($k = 29$) also evaluated various cognitive, interpersonal, biological, and personality/temperament vulnerability

Table 7
Egger's Regression Test and Trim-and-Fill Analyses for Overall Psychopathology, Internalizing Psychopathology, and Depression in Relation to Prospective Stress Types

Psychopathology	Overall dependent stress			Interpersonal dependent stress			Noninterpersonal dependent stress			Independent stress		
	Egger's regression test *p*	*d*	95% CI	Egger's regression test *p*	*d*	95% CI	Egger's regression test *p*	*d*	95% CI	Egger's regression test *p*	*d*	95% CI
Overall psychopathology	.01	0.43	[0.36, 0.50]	<.01	0.43	[0.35, 0.50]	.55	0.36	[0.23, 0.48]	>.99	0.21	[0.15, 0.26]
Internalizing psychopathology	.04	0.43	[0.36, 0.50]	.01	0.44	[0.36, 0.52]	.43	0.36	[0.23, 0.50]	.46	0.21	[0.15, 0.28]
Depression	.04	0.45	[0.38, 0.52]	.02	0.48	[0.40, 0.56]	.23	0.34	[0.20, 0.49]	.61	0.23	[0.17, 30]

Note. CI = confidence interval. For Egger's regression test, $p < .05$ indicate significant publication bias. For dependent stress analyses, larger *d* indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values are traditionally interpreted as giving greater support for the stress generation hypothesis. *d* and associated 95% CI are for adjusted effect size estimates of stress generation after accounting for publication bias based on trim-and-fill analyses.

factors as predictors of stress generation in the mediational models. Most studies ($k = 21$) on vulnerability factors primarily included cognitive and interpersonal vulnerabilities, though there was not substantial overlap in the specific cognitive or interpersonal vulnerability included, with most studies examining unique cognitive (e.g., negative inferential/cognitive style, dysfunctional attitudes, rumination, self-criticism) or interpersonal factors (e.g., dependency, reassurance seeking, social support). Nearly all 29 of the identified studies with vulnerability factors as the independent predictors found evidence of stress generation to subsequent symptoms through overall dependent stress or dependent interpersonal stress.

In summary, most ($k = 28$) of the mediational studies examined the mediational pathway through subtypes of dependent stress (interpersonal or achievement/noninterpersonal) as a mediator between psychopathology or vulnerabilities and subsequent symptoms of psychopathology. The majority of studies examined interpersonal dependent stress ($k = 28$), a smaller number examined noninterpersonal or achievement dependent stress ($k = 6$), and a total of 17 studies examined total dependent stress as a mediator. Overall, most mediational studies demonstrated significance of pathways through overall dependent and dependent interpersonal stress but not dependent noninterpersonal stress.

Discussion

The past 30 years have seen sustained and significant empirical interest in the concept of stress generation. In the current review, we provided frequentist and Bayesian meta-analyses of these 30 years of studies on the stress generation phenomenon. Quantitative analyses were conducted for psychopathology, related risk factors, protective factors, and sociodemographic characteristics in relation to prospective overall dependent stress, its subtypes, and independent stress. We evaluated sample and study design characteristics as potential moderators of overall psychopathology, internalizing psychopathology, and depression in relation to prospective dependent and independent stress. Quantitative analyses were supplemented by qualitative analyses evaluating whether significant associations with prospective dependent stress remained after covarying baseline depression and/or dependent stress. Finally, we conducted qualitative reviews of studies investigating the mediational component of stress generation. This study critically builds upon frequentist meta-anlaytic reviews (e.g., Rnic et al., 2023; Santee et al., 2023) by providing the most comprehensive review of the stress generation literature to date and advancing the field through the use of Bayesian approaches.

Primary Findings

Across our analyses, there were several notable patterns of findings. Most of the focus of the existing research has been on overall dependent stress, followed by interpersonal dependent stress. This means that the independent stress component of stress generation (i.e., psychopathology and related risk factors are hypothesized not to predict subsequent independent stress) has been relatively understudied. In addition, studies of specific forms of psychopathology have disproportionately focused on internalizing psychopathology, especially depression, with externalizing psychopathology being comparably understudied. This pattern is likely

Table 8

Cognitive Factors, Other Intrapersonal Factors, and Biological Factors in Relation to Prospective Stress Types

Cognitive, other intrapersonal, and biological factor	Overall dependent stress					Interpersonal dependent stress					Noninterpersonal dependent stress					Independent stress				
	k	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{01}
Cognitive factors																				
Cognitive vulnerability to depression[a]	19	7,009	0.50***	[0.37, 0.62]	1.00×10^5	10	3,707	0.39***	[0.25, 0.52]	419.00	8	2,541	0.26***	[0.15, 0.36]	47.50	7	1,450	0.11*	[0.02, 0.21]	1.09
Negative inferential styles and/or dysfunctional attitudes[a]	12	5,055	0.42***	[0.26, 0.57]	176.00	6	2,726	0.33***	0.16, 0.50]	11.40	5	1,864	0.22**	[0.08, 0.36]	3.08	5	1,020	0.10	[−0.02, 0.23]	2.32
Negative inferential styles	8	3,779	0.47***	[0.29, 0.65]	40.00	4	1,607	0.36***	[0.22, 0.49]	10.10	3	745	0.17*	[0.03, 0.32]	.88	3	507	0.17	[<0.01, 0.35]	1.24
Rumination[a]	9	2,900	0.53***	[0.32, 0.75]	238.00	6	1,647	0.45***	[0.29, 0.62]	56.10	5	1,334	0.28***	[0.15, 0.41]	8.11	5	1,209	0.12*	[<0.01, 0.23]	1.68
Ruminative brooding	3	628	0.48***	[0.32, 0.65]	12.30															
Cognitive vulnerability to anxiety	3	515	0.24**	[0.06, 0.41]	1.62															
Other intrapersonal factors																				
Aggression/irritability[a]	7	1,883	0.30***	[0.20, 0.41]	35.00											5	1,584	0.09	[−0.01, 0.19]	3.29
Avoidance	6	2,784	0.34***	[0.21, 0.46]	38.60															
Dysfunctional autonomy	3	354	0.35**	[0.14, 0.56]	3.15															
Hopelessness	3	510	0.62*	[0.06, 10.18]	3.29															
Impulsivity[a]	6	2,299	0.38***	[0.23, 0.52]	23.00											4	1,901	0.12**	[0.03, 0.21]	1.04
Biological factors																				
Biological vulnerability	6	2,357	0.21**	[0.19, 0.52]	2.67	4	584	0.25***	[0.19, 0.52]	9.18						4	2,209	0.13	[−0.03, 0.29]	1.90
Pubertal timing/status	4	584	0.34***	[0.17, 0.50]	6.94															

Note. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. Bayesian analyses: For dependent stress analyses, larger BF_{10} reflects greater support for the alternative hypothesis compared to the null hypothesis (i.e., support for stress generation). For independent stress analyses, larger BF_{01} reflects greater support for the null hypothesis compared to the alternative hypothesis (i.e., support for stress generation). Meta-analyses of predictors of life events subtypes were conducted only when the number of unique effects ≥3, so as to obtain reliable pooled estimates of effect sizes. BF = Bayes factor; CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses.

[a] Nonoverlapping confidence intervals for frequentist pooled effects between overall dependent stress and independent stress were observed, indicating a significant difference in pooled effect size. Overlapping confidence intervals do not necessarily indicate nonsignificance.

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

Table 9

Interpersonal and Personality Factors in Relation to Prospective Stress Types

Interpersonal and personality factor	Overall dependent stress					Interpersonal dependent stress					Noninterpersonal dependent stress					Independent stress				
	k	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{01}
Interpersonal factors																				
Interpersonal vulnerability[a]	15	6,029	0.40***	[0.27, 0.54]	2708.00	13	3,652	0.44***	[0.31, 0.58]	2369.00	7	1,584	0.26**	[0.06, 0.47]	2.72	6	1,433	0.16**	[0.05, 0.26]	.40
Co-rumination	3	1,149	0.28***	[0.16, 0.40]	3.88															
Excessive reassurance-seeking	5	822	0.53***	[0.39, 0.68]	3.88															
Negative attachment styles	4	1,074	0.52**	[0.20, 0.83]	7.64															
Rejection sensitivity	6	3,629	0.50***	[0.23, 0.76]	19.50															
Personality factors																				
Extraversion	4	2,421	−0.12	[−0.27, 0.03]	.06															
Neuroticism[a]	8	4,896	0.40***	[0.28, 0.53]	134.00											4	3,072	0.12*	[<0.01, 0.23]	1.68
Perfectionism	4	998	0.37***	[0.15, 0.59]	4.82															

Note. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. Bayesian analyses: For dependent stress analyses, larger BF_{10} reflects greater support for the alternative hypothesis compared to the null hypothesis (i.e., support for stress generation). For independent stress analyses, larger BF_{01} reflects greater support for the null hypothesis compared to the alternative hypothesis (i.e., support for stress generation). Meta-analyses of predictors of life events subtypes were conducted only when the number of unique effects ≥3, so as to obtain reliable pooled estimates of effect sizes. BF = Bayes factor; CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses.

[a] Nonoverlapping confidence intervals for frequentist pooled effects between overall dependent stress and independent stress were observed, indicating a significant difference in pooled effect size. Overlapping confidence intervals do not necessarily indicate nonsignificance.

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

Table 10
Other Risk Factors and Protective Factors in Relation to Prospective Stress Types

Other risk factor and protective factor	Overall dependent stress					Independent stress				
	k	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{01}
Other risk factors										
Childhood maltreatment/adversity	4	533	0.35*	[0.08, 0.62]	2.94	3	401	0.41	[−0.06, 0.88]	.58
Maternal depression	5	703	0.33*	[0.03, 0.63]	2.12	4	543	0.51*	[0.04, 0.99]	.31
Protective factors										
Overall protective factors[a]	16	4,604	−0.29***	[−0.43, −0.15]	.03	4	783	0.04	[−0.10, 0.19]	7.11
Social competence	4	762	−0.12	[−0.32, 0.08]	.08					
Subjective well-being	4	849	−0.24	[−0.65, 0.17]	.13					

Note. Frequentist analyses: For dependent stress analyses, larger *d* (with $p<.05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. Bayesian analyses: For dependent stress analyses, larger BF_{10} reflects greater support for the alternative hypothesis compared to the null hypothesis (i.e., support for stress generation). For independent stress analyses, larger BF_{01} reflects greater support for the null hypothesis compared to the alternative hypothesis (i.e., support for stress generation). Meta-analyses of predictors of life events subtypes were conducted only when the number of unique effects ≥3, so as to obtain reliable pooled estimates of effect sizes. BF = Bayes factor; CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses.
[a] Nonoverlapping confidence intervals for frequentist pooled effects between overall dependent stress and independent stress were observed, indicating a significant difference in pooled effect size. Overlapping confidence intervals do not necessarily indicate nonsignificance.
* $p < .05$. ** $p < .01$. *** $p < .001$.

a reflection of the origins of stress generation as theory of depression and is consistent with the findings reported by Rnic et al. (2023).

Even so, there was generally clear support for psychopathology and risk factors in relation to dependent stress, based on both frequentist and Bayesian analyses. An important qualification is worth noting here. General support from Bayesian analyses (i.e., size of Bayes factors) was appreciably weaker in cases where few unique effects are available (i.e., *k*), whereas frequentist analyses (i.e., magnitude of effect sizes) appeared relatively unaffected by this consideration. This is to be expected, in that for frequentist analyses, significance (i.e., *p*) and corresponding confidence intervals for *d* rather than magnitude of pooled effects are what are most affected by the amount of available data, whereas for Bayesian analyses, the amount of data on which to evaluate Hypothesis 1 directly against Hypothesis 0 has a direct bearing on the strength of the evidence in support of one hypothesis versus the other and thus the size of the resulting Bayes factor. Therefore, if future studies were to find comparable effects to ones found in the current review, *d* in frequentist analyses would reasonably be expected to remain largely unchanged, whereas the corresponding Bayes factor would be expected to increase.

Psychopathology in Relation to Prospective Dependent Stress

There are several indications that the stress generation effect—at least as it pertains to overall psychopathology, internalizing psychopathology, and depression in relation to overall dependent stress and its interpersonal and noninterpersonal subtypes—is reasonably robust. First, it is for these relationships that both frequentist and Bayesian analyses give their strongest support. In frequentist meta-analyses, this was evident in the generally medium-sized effects (i.e., strength of the associations; National Academies of Sciences Engineering and Medicine, 2018) for these forms of psychopathology in relation to prospective overall dependent stress and its interpersonal subtype, as well as the small-to-medium effects for noninterpersonal dependent stress. These findings are consistent with those reported by Rnic et al. (2023). In Bayesian meta-analyses, BF_{10} consistently exceeded 500, indicating extreme support for these associations (Jeffreys, 1961; Wagenmakers et al., 2011).[16] Of note, too, given that stress generation originated as a theory of depression, sensitivity analyses for internalizing psychopathology with depression variables removed are supportive of the transdiagnostic relevance of stress generation. With the exception of depression in relation to prospective noninterpersonal dependent stress, these findings received were also supported, albeit with a more modest body of available research, in our qualitative analyses of studies that took the relatively more stringent steps of covarying baseline depression and/or dependent stress.

Second, in particularly stringent tests of long-term persistence of stress generation (i.e., meta-analytically evaluating associations with prospective overall dependent stress over follow-up periods of 4 years or longer), overall psychopathology, internalizing psychopathology, and depression all yielded significant effects in the small-to-medium range. These tests are especially remarkable in the case of depression, an often episodic (thus temporally delimited) disorder that has significant predictive value years into the future. This latter set of evaluations also provides clarity on some of the parameters of the question of whether stress generation may be a viable mechanism underlying onset and recurrence of psychopathology. It answers the question of how long does stress generation persist and if it therefore has potential to influence long-term risk for negative mental health outcomes.

Two additional patterns of findings warrant mention. First, for overall psychopathology and all examined forms of internalizing psychopathology, associations with prospective dependent stress were uniformly stronger than corresponding associations with prospective independent stress, as reflected by nonoverlapping 95% confidence intervals in frequentist meta-analyses. Although this

[16] As described above, unlike frequentist effect sizes, Bayes factors reflect the strength of the evidence that a relation exists; they do not indicate the magnitude of the relation itself.

Table 11
Sociodemographic Characteristics in Relation to Prospective Stress Types

Sociodemographic characteristic	Overall dependent stress					Interpersonal dependent stress					Noninterpersonal dependent stress					Independent stress				
	k	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{10}	*k*	*N*	*d*	95% CI	BF_{01}
Age	17	7,751	0.14**	[0.05, 0.23]	2.50	8	4,229	0.17	[−0.01, 0.35]	.70	5	1,817	0.28**	[0.07, 0.49]	2.63	8	2,482	<0.001	[−0.09, 0.09]	20.10
Sex (female)	26	11,730	0.08*	[0.01, 0.14]	2.01	19	8,833	0.17***	[0.08, 0.25]	19.40	13	5,273	−0.08	[−0.20, 0.05]	.08	14	5,108	0.20***	[0.14, 0.27]	3×10^{-3}
Race (White)	7	2,650	−0.04	[−0.27, 0.19]	.11											5	2,220	−0.12	[−0.29, 0.06]	15.30
SES	5	1,717	−0.38**	[−0.62, −0.13]	.11											3	1,253	−0.38*	[−0.73, −0.03]	6.79

Note. Frequentist analyses: For dependent stress analyses, larger *d* (with $p < .05$) indicates greater support for the stress generation hypothesis. For independent stress analyses, smaller absolute *d* values (with $p \geq .05$) are traditionally interpreted as giving greater support for the stress generation hypothesis. Bayesian analyses: For dependent stress analyses, larger BF_{10} reflects greater support for the alternative hypothesis compared to the null hypothesis (i.e., support for stress generation). For independent stress analyses, larger BF_{01} reflects greater support for the null hypothesis compared to the alternative hypothesis (i.e., support for stress generation). Meta-analyses of predictors of life events subtypes were conducted only when the number of unique effects was ≥3, so as to obtain reliable pooled estimates of effect sizes. BF = Bayes factor; CI = confidence interval; *k* = number of unique effects; *N* = total number of participants included in pooled analyses; SES = socioeconomic status.
* $p < .05$. ** $p < .01$. *** $p < .001$.

finding is not explicitly part of the stress generation hypothesis, it is nonetheless consistent. Second, among dependent stress subtypes, interpersonal dependent stress consistently yielded the largest *d*s, with values falling in the medium range (except for anxiety), as opposed to small-to-medium range in the case of noninterpersonal stress. Additionally, as mentioned above, in analyses covarying baseline depression and/or dependent stress, depression was poorly associated with noninterpersonal stress, with significant findings being reported in only one of three studies. This pattern of findings is consistent with the view that for internalizing disorders, and depression in particular, stress generation is very much an interpersonal phenomenon (Conway et al., 2012; Hammen, 2018; Hankin et al., 2005; Rudolph et al., 2000).

This latter finding has broader relevance. If individuals with depression and closely related psychopathology are significantly more reactive to independent stress, for example, this would potentially diminish the clinical importance of the stress generation hypothesis. Available evidence suggests that this is not the case. Indeed, as views of the importance of dependent stress have shifted, such that it has been increasingly included in studies of stress exposure in the last several decades, a finding that has emerged across various forms of psychopathology is that interpersonal dependent stress appears to be particularly pathogenic (Bagge et al., 2013; Hammen, 2009a; Hammen et al., 2004; Liu & Miller, 2014; Rudolph et al., 2016; Van Nieuwenhove & Meganck, 2019). Indeed, at least in the case of depression, there is direct evidence to suggest that interpersonal dependent stress, compared to independent stress, is more predictive of this disorder (Kessler, 1997; Vrshek-Schallhorn et al., 2015).

When findings of psychopathology are broadened to include externalizing presentations, both frequentist and Bayesian analyses provided strong support for stress generation as a transdiagnostic process but differed in their support for the aforementioned view of how stress generation manifests based on psychopathology (Conway et al., 2012; Rudolph et al., 2000). Whereas the notion that depression is well characterized by interpersonal stress generation appears supported by both analytical approaches, the view that noninterpersonal stress generation is contrastingly more characteristic of externalizing psychopathology received mixed support. That is, frequentist analyses yielded a significant association for externalizing psychopathology with noninterpersonal dependent stress and a nonsignificant association with interpersonal dependent stress. Bayesian analyses, however, did not provide support for an association with either dependent stress subtype, possibly because there were few stress generation studies that have included externalizing psychopathology. Based on the amount of data currently available for testing this extension of the stress generation hypothesis, the most reasonable conclusion that can be currently drawn is that more studies are required before a more certain verdict can be reached regarding the pattern of dependent stress generation that may be occurring with externalizing psychopathology.

Psychopathology in Relation to Prospective Independent Stress

Perhaps one of the most notable sets of findings to emerge relates to the independent stress component of the stress generation hypothesis, according to which psychopathology and related risk

Table 12
Evaluating Stress Generation as a Mediator of Clinical Symptom Outcomes

Independent variable	Mediator	Dependent variable	Study
Psychopathology			
Depression	Dependent	Depression	Clements et al. (2008)[a], *Goldstein et al. (2020)*, *Kercher and Rapee (2009)*, Kercher et al. (2009), Maniates et al. (2018), Stroud et al. (2023)
	Interpersonal dependent	Depression	*Davila et al. (1995)*, *Herres and Kobak (2015)*[b], *Meiser and Esser (2019)*, Rudolph et al. (2009)[a], Stroud et al. (2023)
	Noninterpersonal dependent	Depression	Meiser and Esser (2019), Rudolph et al. (2009)[a], Stroud et al. (2023)
	Independent	Depression	*Kercher et al. (2009)*, Maniates et al. (2018), Stroud et al. (2023)
	Interpersonal dependent	Anxiety	Belmans et al. (2019)[a], *Long et al. (2022)*[a]
Internalizing	Interpersonal dependent	Internalizing	*Goldstein et al. (2021)*[c]
Social anxiety	Interpersonal dependent	Depression	Belmans et al. (2019)[a,c]
PTSD	Dependent	Depression	*Maniates et al. (2018)*[c]
	Independent	Depression	Maniates et al. (2018)[c]
ADHD	Dependent	Depression	*Rychik et al. (2021)*[a,c]
	Dependent	ADHD	*Brinksma et al. (2018)*[c]
	Independent	ADHD	Brinksma et al. (2018)[c]
Personality	Interpersonal dependent	Internalizing	*Daley et al. (2006)*[a,c]
	Dependent/independent	Borderline	Conway et al. (2018)[a,c]
Cognitive factors			
Negative inferential styles/dysfunctional beliefs, rumination, perceived control	Interpersonal dependent	Depression	*Auerbach et al. (2012; boys subsample only)*, *Auerbach et al. (2014)*, *Calvete (2011)*[c], *Hamilton et al. (2013)*[c], *Flynn et al. (2010)*[a,c], Meiser and Esser (2019)
Rumination, perceived control	Interpersonal dependent	Anxiety	*Auerbach et al. (2012, girls subsample only)*, Flynn et al. (2010)
Psychological factors			
Avoidance coping self-criticism, reward sensitivity	Dependent	Depression	*Bart et al. (2019)*, *Cox et al. (2009)*, *Mackin et al. (2019)*
Reward sensitivity	Independent	Depression	Bart et al. (2019); Mackin et al. (2019)
Extrinsic goals, defectiveness, ineffective stress response, failure	Interpersonal dependent	Depression	*Auerbach, Webb, et al. (2011)*, Eberhart et al. (2011)[c], Flynn and Rudolph (2011)[a,c], *Holahan et al. (2005)*[c]
Extrinsic goals, ineffective stress response	Noninterpersonal dependent	Depression	*Auerbach, Webb, et al. (2011, Chinese subsample only)*, Flynn and Rudolph (2011)[a,c]
Biological factors			
Respiratory sinus arrhythmia	Interpersonal dependent	Depression	*Hamilton and Alloy (2017)*
	Independent	Depression	Hamilton and Alloy (2017)
Interpersonal factors			
Co-rumination, anxious or avoidant attachment, social support, social isolation, dependency	Interpersonal dependent	Depression	*Auerbach, Bigda-Peyton, et al. (2011)*, *Eberhart and Hammen (2010)*[a,c], *Hankin et al. (2005)*[a], *Hankin et al. (2010)*[a,c], *Eberhart et al. (2011)*[c]
Reassurance-seeking	Interpersonal dependent	Depression	*Eberhart and Hammen (2010)*[a,c], *Potthoff et al. (1995)*
Sociotropy	Interpersonal dependent	Depression	*Calvete (2011)*[c], *Shih (2006)*[c]
Personality factors			
Rejection sensitivity, negative emotionality, doubts about actions, Socially prescribed perfectionism	Dependent	Depression	*Cox et al. (2009)*[c], *Liu, Alloy, et al. (2014)*[c], *Wetter and Hankin (2009)*[c]
Other factors			
Family conflict, mistrust/abuse, subjugation, emotional deprivation	Interpersonal dependent	Depression	*Auerbach and Ho (2012)*, *Eberhart et al. (2011)*[c]

Note. ADHD = attention-deficit/hyperactivity disorder; PTSD = posttraumatic stress disorder. Italic studies indicate significant mediation. When there are not two studies examining the same mediational pathway, example factors are grouped by independent variable category based on life stress domain. One example factor was included when there were not two studies in the category (e.g., biological). Studies that included moderated mediation were included in the review but not included in this table, given that there was no overlap across studies.
[a] Covaried baseline dependent stress. [b] Interpersonal stress evaluated separately as parental, peer, and teacher events. [c] Covaried baseline depression.

factors should not prospectively predict independent stress. We found tempered support for this aspect of stress generation. In frequentist meta-analyses for overall psychopathology and its subtypes, *d*s were trivial to small, although they were significant in most cases. Here, the unique complementary value of Bayesian analyses becomes most apparent, lending greater clarity to the data. In cases where there were a sizeable number of unique effects—for overall psychopathology, internalizing psychopathology, and

depression—BF_{01} approached zero,[17] indicating robust support for associations between these forms of psychopathology and prospective independent stress. When these frequentist and Bayesian findings are interpreted together with the earlier findings comparing associations for dependent stress and independent stress, they suggest that psychopathology, particularly internalizing presentations and depression, are in fact associated—albeit modestly—with prospective independent stress but less so than with prospective dependent stress. Of note, this finding is consistent with that reported by the recent meta-analytic review conducted by Rnic et al. (2023). A question for future consideration is whether these associations with prospective independent stress are of sufficient magnitude to lead to negative mental health outcomes.

As for what may account for these associations with independent stress, one possible explanation may be found in research on gene–environment correlation (rGE; Kendler, 2010; Liu & Spirito, 2019; Rutter et al., 2006). rGE refers to genetic influences on exposure to environmental risks through various processes. A process of potential relevance is passive rGE, or differential exposure to an environment for reasons apart from the individual's direct involvement (i.e., independent stress). According to this process, the individual's environment is shaped by other influences driven by shared genetic variance (i.e., family members). It is therefore reasonably possible that certain individuals are at prospectively greater risk for independent stress because they are exposed to stress generation in their familial network (e.g., a child whose parents experience a job loss), especially to the extent that psychopathology aggregates within families and social networks (Brent et al., 1996; Cero & Witte, 2020; Harkness et al., 2006; Kim et al., 2005; McGirr et al., 2009; Rosenquist et al., 2011; Wyman et al., 2019).

Psychopathology in Relation to Prospective Stress Types: Moderator Effects

In moderator analyses, the study features that consistently had significant associations were life stress measure type, life stress timeframe, and study follow-up period. Consistent with our hypothesis and prior reviews (Rnic et al., 2023), uniformly stronger associations were found between psychopathology variables and all forms of dependent stress for studies that used self-report measures of life stress than for ones that featured interview-based measures. That is, it is consistent with the view that self-report measures of life stress are more vulnerable to biases in responding due to underlying risk factors for psychopathology (e.g., mood-congruent response bias; S. L. Johnson & Roberts, 1995) and thus highlights the importance of using life stress interviews to arrive at accurate estimates of associations with life stress (Hammen, 2018; Harkness & Monroe, 2016). This concern is magnified in evaluations of the mediational component of the stress generation hypothesis (i.e., psychopathology and its risk factors are associated with future psychopathology through the mediating effect of stress generation). Inasmuch as self-report life stress measures confound life stress with symptom presentation, tests of stress generation as a mediator of the relation between past and future psychopathology run a substantially higher risk of Type I errors, insofar as life stress is confounded with both the predictor and the outcome (Liu et al., 2016).

As for life stress measure timeframe, consistent with our hypothesis related to issues of faded recall with longer timeframes mentioned above (G. W. Brown & Harris, 1982; Monck & Dobbs, 1985; Paykel, 1997), briefer timeframes produced larger effects than did longer timeframes. These findings speak to the importance of ensuring appropriate timeframes in assessments of stress generation. In our meta-analytic review, a substantial proportion of studies (36.56%) included life stress assessments of 1 year or longer, which underscores the degree to which this is currently an issue in the field.

Results from moderator analyses were consistent with our hypothesis that brief follow-ups would yield stronger effects, consistent with other meta-anyltic reviews (Rnic et al., 2023). When considered with the previously mentioned finding of significant stress generation effects persisting for over 4 years, these findings provide insight into the temporal phenomenology of stress generation. They suggest that stress generation is a state-sensitive phenomenon but also a long-lasting one. Additionally, the diminished strength of stress generation over longer temporal intervals leaves open the question of whether long-term stress generation is of sufficient magnitude to elicit clinical outcomes, a question that is, in some measure, more directly addressed in mediational findings discussed below.

Finally, although not a moderator that was found to be consistently significant in our analyses, the year of study production was of particular interest. We evaluated whether the strength of the association between different forms of psychopathology and prospective dependent stress diminished as a function of the year the study was produced. If effect sizes in individual studies were time-invariant, it may suggest that stress generation is fairly robust and relatively immune to the replication crisis that has been reported for other findings in the field (Nosek et al., 2022; Shrout & Rodgers, 2018; Tackett et al., 2019). For the most part, we did not see evidence of reduced effect sizes in more recent studies. The two exceptions were for overall psychopathology and internalizing psychopathology, respectively, in relation to overall dependent stress. However, we believe these two findings alone do not suggest there is an issue of replicability in the field. Given that stress generation originated as a theory of depression, this disorder has understandably received the most empirical attention in the field, and thus, if we were to observe an issue of replicability, it would most likely be for this psychopathology variable, especially in relation to prospective interpersonal dependent stress. As can be seen in moderator analyses, this was not the case.

Psychopathology in Relation to Prospective Stress Types: Accounting for Publication Bias

When evaluating the robustness of our meta-analytic findings, consideration should be given to the potential impact of publication bias. No evidence of publication bias inflating pooled effects was detected for noninterpersonal stress. Even though evidence of publication bias was observed for overall dependent stress and interpersonal dependent stress, adjusting for bias resulted in reasonably modest decreases in their estimated effect sizes to just below medium-sized effects.

As for what may account for this publication bias and what implication it has on our stress generation findings, one possibility is that this is a function of the decision in the current review to exclude

[17] BF can only take values between zero and infinity. When $BF_{01} < 1$, it suggests the data do not support the stress generation hypothesis with regards to independent stress.

unpublished studies (e.g., theses and dissertations). If so, the hypothetical missing studies are more likely to feature self-report life stress measures. This is because of the marked contrast between self-report and interview-based life stress measures in terms of time to train, administer, and process into analyzable form. Conducting life stress interviews, especially ones featuring the gold standard contextual threat approach (Hammen, 2005; Harkness & Monroe, 2016; Monroe, 2008), requires considerable training, participant time, and personnel resources (i.e., having multiple individuals to be trained on and to provide ratings for the narratives for stressors experienced by each participant). In contrast, life events checklists are a much more commonly used alternative in life stress research because they are quick and economical to administer and require no prior training.

Seemingly consistent with this possibility, 34.62% of the studies included in the current review featured the gold standard for measuring life stress, which is much higher than in other fields of life stress research. For example, just 7.69% of studies in a meta-analysis on life stress and nonsuicidal self-injury used contextual threat interviews (Liu et al., 2016), and only 3.16% in a systematic review of life stress in relation to suicidal thoughts and behaviors (Liu & Miller, 2014). Yet, these two prior reviews similarly excluded non-peer-reviewed articles.[18] Additionally, moderator analyses consistently yielded significantly smaller effects for interview-based measures of life stress, a finding also reported by Rnic et al. (2023). Instead, the overrepresentation of contextual threat studies with smaller effects suggests that the publication bias findings likely involve an underrepresentation of studies with self-report life stress measures and small effects relative to studies with self-report life stress measures and larger effects. Furthermore, this potential bias in studies with self-report life stress measures may be largely counterbalanced by the overrepresentation of studies with contextual threat interviews. Indeed, a comparison of *d*s for interview-based life stress measures in the moderator analyses with corresponding adjusted *d*s estimates in the trim-and-fill analyses uniformly yields numerically lower values for the former than the latter with overall dependent stress and its interpersonal subtype.

That being said, it is certainly possible that our focus on peer-reviewed studies may nonetheless have resulted in greater exclusion of studies with contextual threat interviews that had null results, as a function of selection bias among peer-reviewed journals for studies with significant findings. This, in turn, may contribute to overestimates of some of the calculated effect sizes.

Psychopathology-Related Risk Factors in Relation to Prospective Stress Types

Cognitive Factors

Turning now to related risk factors, in general, the strongest meta-analytic support was observed for cognitive vulnerability to depression based on several different theoretical conceptualizations and general interpersonal vulnerability factors. This is particularly the case in both frequentist and Bayesian meta-analyses for associations with overall dependent stress. When factoring in findings from studies that covaried baseline depression and/or dependent stress, however, the picture becomes more complicated, with negative inferential styles emerging as the most robust predictor of dependent stress. Given that cognitive vulnerabilities are central to current understandings of depression etiology and recurrence, additional research is warranted to resolve some of the inconsistencies between meta-analytic and qualitative reviews. Furthermore, cognitive vulnerabilities tend to be more traitlike in nature in adulthood (Beevers et al., 2007), which contrasts with the often-episodic nature of depression. Thus, these cognitive vulnerabilities may have more potential to explain long-term stress generation and, thus, how this phenomenon may in turn account for depressive recurrence.

How these different stress generation mechanisms may interrelate deserves more empirical attention. It may be that cognitive factors function as relatively more distal risk factors for stress generation and that interpersonal behavioral factors mediate this association (Liu, 2013). For example, as noted above, negative schemata of the self and expectations of others may motivate behaviors that lead to self-fulfilling prophecies (e.g., Buchanan & Hughes, 2009; Jussim, 1986), in which vulnerable individuals engage with their social world in ways that ultimately drive the interpersonal stress that they feared (Coyne, 1976a; Swann, 1983, 1990, 2012). Delineating the interrelationship between these risk factors is important to provide a more complete picture of the stress generation phenomenon and thereby identify multiple points for potential intervention efforts.

Other Intrapersonal Psychological Factors

The current body of research focused on other general psychological factors is more modest. Several of these hold particular interest (i.e., aggression/irritability and impulsivity) because they are behavioral in nature and have therefore been hypothesized to serve as more proximal stress generation mechanisms than the aforementioned cognitive processes (Liu, 2013), fitting with the conceptualization of stress generation as a form of action theory (Hammen, 2006). If this is the case, these risk factors should be more strongly associated with prospective dependent stress. However, based on a comparison of meta-analytic effect sizes in frequentist analyses for the cognitive risk factors and other intrapersonal ones, this does not appear to be so. It may be that different behavioral processes (e.g., interpersonal ones) are more relevant proximal mediators between cognitive risk and dependent stress, and that the other intrapersonal processes studied here operate through relatively distinct and independent pathways.

Biological Factors

Evaluations of potential biological drivers of stress generation are modestly represented in the existing literature, with there being just six unique effects on associations with prospective dependent stress.

[18] What may account for this difference in proportion of studies using contextual threat interviews is that the stress generation literature, in contrast to the literature on life stress in relation to nonsuicidal self-injury and suicide, respectively, was developed by a contextual threat researcher (i.e., Hammen). This aspect of the origins of the stress generation hypothesis may likely have influenced how it has been studied. Indeed, a noticeable proportion of stress generation studies have been from her former trainees and collaborators. Rather than potentially introducing a bias toward confirmatory findings, however, this may have resulted in more stringent tests of stress generation than would otherwise be the case, given the theoretical and empirical support for smaller sized effects from these interviews than from self-report life stress checklists.

The significant effect from frequentist meta-analysis is small and balanced by lack of strong support from Bayesian meta-analysis. Also suggesting that the current findings should be viewed tentatively, no analyses from the original studies covaried baseline depression and/or dependent stress. Collectively, these findings indicate that the current literature in this area is preliminary and awaiting further investigation.

One unique aspect of adolescence that may influence the stress generation process is the emergence of puberty. With four unique effects across three studies included in the current review (Hamilton et al., 2014; Rudolph, 2008; Stroud et al., 2018), it is the most studied biological process. Researchers have found pubertal status (Cyranowski et al., 2000) and pubertal timing (Copeland et al., 2010; Mendle et al., 2010; Teunissen et al., 2011), especially among girls, to be related to the pronounced increase in prevalence of depression that emerges in early adolescence (Cohen et al., 1993; Kessler et al., 2001). In particular, timing of pubertal development has been linked with risk for depression (Natsuaki et al., 2009; Teunissen et al., 2011). Early pubertal onset has similarly been implicated in the stress generation process, with higher dependent stress in early-maturing, but not late-maturing, depressed youth (Rudolph, 2008). The preliminary findings in this area suggest that additional research is warranted. For example, if a stress generation effect is supported for pubertal timing, a natural next question is: why? What are the underlying processes that relate early pubertal onset to stress generation? One possibility for future investigation may be the tendency for early-maturing individuals to engage in risky relationships (Rudolph, 2008; Weichold et al., 2003). Alternatively, it may be that early pubertal onset is more a marker for preexisting vulnerabilities and challenges (e.g., maladaptive coping, behavioral problems, and prior psychopathology; Caspi & Moffitt, 1991; Hamilton et al., 2014; Rudolph, 2008).

These considerations have added relevance for some youth of minoritized identities (e.g., Latine[19] youth, as well as transgender and gender diverse youth). On average, Latine youth experience puberty earlier than their White peers (Deardorff et al., 2021), and this earlier onset may place Latine youth at greater risk to interpersonal stress in the form of peer victimization (Haltigan & Vaillancourt, 2018). In the case of transgender and gender diverse youth, puberty may heighten incongruence between the individual's gender identity and how they are treated by others in their social environment (i.e., being treated increasingly in a cisnormative manner based on their sex assigned at birth; Steensma et al., 2013). This emergence of greater incongruence at puberty may place gender minority youth at-risk for increased interpersonal gender minority stress (e.g., gender-identity-based victimization and discrimination; Hendricks & Testa, 2012). Risk for interpersonal stress may therefore be compounded with the onset of puberty in the case of youth with multiple minoritized identities (e.g., gender minority youth who are Latine).

Interpersonal and Personality Factors

As mentioned above, a number of interpersonal styles and personality traits (e.g., neuroticism) may be strong candidates for behavioral risk factors serving as proximal mediators between cognitive vulnerabilities for depression and prospective dependent stress. Lending promise to this possibility is that several of these interpersonal vulnerabilities and traits are central to current etiological conceptualizations of depression (e.g., excessive reassurance-seeking within Coyne's 1976b interpersonal theory of depression) and have also been specifically identified as candidate mediational pathways between cognitive vulnerabilities and prospective dependent stress.

Although there is general support for the role of these interpersonal styles and personality traits in stress generation based on frequentist meta-analysis, there were few studies for most of these vulnerabilities. This may be what accounts for the generally lower support from Bayesian meta-analyses and suggests this area of research is still underdeveloped. The notable exception is neuroticism, which received extreme support from Bayesian meta-analysis and remained significant in supplemental qualitative analysis of studies covarying for baseline depression and dependent stress. Indeed, outside of psychopathology variables, neuroticism has perhaps the strongest evidence supporting its role in stress generation.

Even so, there is a notable lack of studies evaluating individual interpersonal and personality constructs specifically in relation to interpersonal dependent stress. It stands to reason that interpersonal styles and personality factors may be more strongly related to prospective interpersonal dependent stress than other forms of dependent stress. Whether these interpersonal and personality factors mediate the effect of cognitive vulnerabilities on interpersonal stress generation may therefore warrant future investigation.

Protective Factors

Relatively absent from the stress generation literature is a consideration of possible protective factors. Such mechanisms may operate directly on the stress generation process in preventing the occurrence or reducing the severity of dependent stress (e.g., through active and adaptive problem-solving). Although observed effects in frequentist meta-analyses were in the expected direction, overall protective factors were the only predictor variable found to be significant, with a small-to-medium effect size, and Bayesian analyses did not offer corresponding support for this association. Protective factors unexpectedly received some of the greatest support in multivariate analyses, covarying baseline depression and dependent stress. The findings in this area must be viewed as preliminary, but they suggest further study is warranted.

Evaluating and identifying specific protective mechanisms may offer more promise, and such an approach may be of clinical importance to the degree that it informs treatment and preventive efforts. Drawing on the depression literature, one possibility is dispositional optimism. There is evidence that optimism and social networks may influence each other in a reciprocal manner. Not surprisingly, optimists tend to be more agreeable company than their pessimistic counterparts, with the former more likely to experience social acceptance in contrast to social rejection with the latter (Carver et al., 1994). Optimists have been found to have better relationships with their romantic partners, to experience less intense

[19] The use of the word Latine is used as a gender-neutral word to describe individuals who identify as Hispanic/Latino/Latina/Latinx/Latin*. Latin*, Latinx, and Latine are all used with the goal of being inclusive of the gender diversity within this population. Latine was chosen here given mounting evidence that the term Latinx is not used by many members of the community it describes given incongruence with the Spanish language and pronunciation (Del Real, 2020; Noe-Bustamante et al., 2020).

conflicts, and to be better at resolving disagreements when they do arise (Brissette et al., 2002). Furthermore, optimists' positive focus during conflict negotiation may reduce the potential for a pattern of negative reciprocity, thus increasing the likelihood of a more productive resolution to the interaction (Srivastava et al., 2006).

Sociodemographic Characteristics in Relation to Prospective Stress Types

Age and Development

As for sociodemographic characteristics that may be associated with stress generation, several findings are of note. Overall, we did not find strong support for our hypothesis of a stress generation effect. This was consistent with moderator analyses that indicated that the association between different forms of psychopathology and prospective dependent stress types did not vary as a function of age, in contrast to findings reported by Rnic et al. (2023). Differences in reported findings could be due to categorization differences, including combination of child and adolescent studies in our analyses versus adolescents being combined with early adult samples in Rnic and colleagues'. Importantly, adolescence is a developmental period unique to both childhood and early adulthood and warrants separate examination in future studies as the literature becomes more robust in this area. It also may be that stress generation limitedly or does not vary as a function of age. Alternatively, it may be that sample considerations significantly limited our ability to detect age differences in stress generation. Or if stress generation increases in adolescence and decreases in adulthood, perhaps a study design that would ensure the greatest possible contrast would be one comparing preadolescent children, adolescents, and older adults. Only five studies (with four nonoverlapping samples; Bakker et al., 2011; Brinksma et al., 2018; Chan et al., 2014; Clements et al., 2008; Mezulis et al., 2010) had entirely preadolescent samples at baseline (only two if age at the end of study follow-up is used instead), as well as minimally overlapping predictor variables. Additionally, the highest mean baseline age of any sample in our meta-analysis was 61.0 (Holahan et al., 2005), as most studies skewed toward adolescence.

Gender Identity

Findings relating to differences in stress generation based on gender identity were very mixed. Moderator analyses were very inconsistent in the degree to which they supported this hypothesis. Similarly, direct meta-analyses with prospective dependent stress generated strong support only for interpersonal dependent stress, but with generally small effects in frequentist analyses. Bayesian meta-analyses only provided strong support for interpersonal dependent stress. Collectively, these findings suggest that if a gender difference in stress generation exists, its effect is small, and the best evidence of it is specifically for interpersonal dependent stress. This latter finding regarding specificity to interpersonal dependent stress, however, would be consistent with findings that women tend to have larger social networks, which in turn provide greater opportunities for stressful interpersonal interactions (Hammen, 2005, 2006).

Race and Ethnicity

As for analyses of race, direct analyses and moderator analyses were generally not supportive of the existence of racial differences in experiences of dependent stress. However, this is inconsistent with minority stress models. For example, racial minority (e.g., the biopsychosocial model of racism; Clark et al., 1999), sexual minority (Brooks, 1981; I. H. Meyer, 2003), and gender minority stress models (Hendricks & Testa, 2012) all posit that minoritized individuals are at-risk for experiencing greater interpersonal stress, particularly in the form of discrimination, prejudice, rejection, and victimization based on their identity. Given that life stress measures always need to be calibrated sensitively to the population with which they are used, one potential explanation for the discrepancy between minority stress theory and our current findings is that the life stress measures used in many stress generation studies may not be adequately calibrated and sensitive to the unique stressors experienced by members of specific minoritized communities, which is unfortunately consistent with the longstanding underrepresentation of minoritized populations in psychological research (Adams & Miller, 2022; Barnett et al., 2019; Cha et al., 2018). Discrimination, which has been linked with psychopathology in minoritized populations (Chou et al., 2012), is a form of interpersonal stress that is not often captured in life events measures. Finally, it is also important to note that any observation of higher levels of dependent and independent life stress may be more reflective of systemic factors that exist at multiple ecological levels (i.e., policy, community, and organizational levels) than actual stress generation. This means that despite the same outcome, it is important to be thoughtful of the different processes (stress generation vs. institutional bias and discrimination) that contributed to elevated stress.

Evaluating Stress Generation as a Mediator of Clinical Symptom Outcomes

As for the mediational aspect of stress generation, if psychopathology and related risk factors are found to predict prospectively occurring dependent stress, but this dependent stress does not account for risk for subsequent mental health outcomes, stress generation would be phenomenologically interesting but of limited clinical value. Therefore, tests of the mediational component of stress generation are especially informative.

Overall, there were 44 studies that examined the full mediational pathway in which stress generation mediated the link between psychopathology or associated vulnerability factors and subsequent psychopathology symptoms (mainly depression). Most studies that demonstrated a significant stress generation mediational pathway found evidence for the specific role of interpersonal dependent stress but not noninterpersonal dependent stress or independent stress. Yet, only six mediation studies actually examined noninterpersonal dependent stress, limiting conclusions that can be drawn regarding this type of dependent stress. Noninterpersonal dependent stress can include multiple domains, such as achievement, financial, or health-related stress, which may lead to the onset and maintenance of psychopathology for some individuals.

Thus, future research is needed to examine the role of noninterpersonal stress through the mediational framework and in relation to more diverse psychopathology and to provide a more

fine-grained evaluation of the specific domains of interpersonal dependent stress. For example, an interesting possibility from a developmental perspective is whether the generation of interpersonal dependent stress within peer domains, relative to family domains, is more likely to mediate risk for subsequent psychopathology in adolescents, given the greater importance and sensitivity to peer relationships and evaluation during this developmental period (Blakemore, 2008; Choudhury et al., 2006; Somerville, 2013). Further, few studies covaried for baseline psychopathology or stress in their mediational models, which is critical for establishing temporal precedence rather than stress or psychopathology continuation.

Despite the transdiagnostic nature of stress generation, few studies have examined the full mediational model of stress generation with symptoms of psychopathology other than depression as the primary predictor or outcome. To date, only one study examined the full mediational pathway with externalizing symptoms as both the predictor and outcome, finding supporting evidence for ADHD symptoms in youth (Brinksma et al., 2018). Extending the full mediational model of stress generation to other forms of psychopathology is needed to better shed light on stress generation as a transdiagnostic pathway and mechanism that exacerbates or maintains psychopathology. Furthermore, no studies evaluated whether the full mediational pathway link contributes to the first onset of disorder, which would be critical in establishing potential causality and the importance of stress generation as a key target of intervention.

Future Directions

Externalizing Psychopathology

Given that externalizing psychopathology is comparably understudied, its role in stress generation is less clear other than the existence of a strong small-to-medium effect for general externalizing psychopathology in relation to overall dependent stress. Additional research is required in this area, and there are some interesting possibilities to explore. Transactions between the individual and their social environment are baked into etiological frameworks of externalizing problems, particularly in the context of parent–child interactions and deviant peer group affiliation. Despite forms of interpersonal dependent stress being well-established correlates of externalizing behaviors (Hames et al., 2013), externalizing problems are often recognized and reported in achievement and academic contexts (Baker et al., 2008). Moreover, externalizing behavior is fairly heterogeneous in its presentation. Thus, different forms of externalizing problems may elicit different forms of stress from an individual's social environment. For example, rule-breaking behavior may be more salient in eliciting noninterpersonal dependent stress (academic stress), whereas aggression may be more salient in eliciting interpersonal dependent stress. Future research should not only evaluate whether different dimensions of externalizing psychopathology are predictive of subsequent dependent stress but also advance more fine-grained analyses of the types of stress that externalizing psychopathology generates, which could provide refinement in the conceptualization of stress generation as a transdiagnostic phenomenon across internalizing and externalizing dimensions.

Self-Injurious Thoughts and Behaviors

Another important future direction of research is the examination of SITBs, including suicide and nonsuicidal self-injury, within the context of stress generation. To date, only one relevant study has been conducted on this topic, and findings showed that suicide attempt history, but not suicidal ideation, predicted the prospective occurrence of interpersonal dependent stress (Liu & Spirito, 2019). In examining these dynamics, it is important to understand how stress, particularly interpersonal dependent stress, may play a role in both the onset and maintenance of suicidal thoughts, as well as in the transition from suicidal thoughts to suicidal behaviors (Stewart et al., 2019). Interpersonal dependent stress may be an especially important factor contributing to self-injurious behaviors among individuals at-risk for these behaviors, since etiological models of self-injury emphasize the role of interpersonal stress (Nock & Prinstein, 2004; Van Orden et al., 2010). Indeed, studies have found interpersonal dependent stress, relative to other forms of stress, to be particularly implicated in risk for suicidal thoughts and behaviors (Bagge et al., 2013; Liu & Miller, 2014).

If future support is found for SITBs generating dependent stress, a question that follows is: What are the mechanisms driving stress generation in individuals at-risk for these clinical outcomes? Potential mechanism candidates include deficits in decision-making and interpersonal problem-solving, as well as difficulties navigating interpersonal relationships, all of which have been associated with suicidal behavior (Jollant et al., 2007; Pollock & Williams, 2004). Impulsivity is another potential candidate that has been implicated in SITBs (Liu et al., 2017; McGirr et al., 2008; Wenzel & Beck, 2008) and found to have significant small-to-medium (frequentist) and strong (Bayesian) support in relation to dependent stress in the current review.

Positive Life Events

An interesting consideration for future research is to evaluate prospective predictors of positive dependent (and independent) life events. Research on this topic has clinical implications insofar as positive events may buffer against risk for negative mental health outcomes (G. W. Brown, 1993; J. G. Johnson et al., 1996; Needles & Abramson, 1990). Dispositional optimism may be a promising candidate in this regard. Just as optimists tend to be proactive in safeguarding themselves against significant threats to their well-being and preventing negative outcomes, they may adopt a similar approach-orientation in pursuing more positive outcomes. In a manner not incongruent with the extensive literature on self-fulfilling prophecies (e.g., Buchanan & Hughes, 2009; Jussim, 1986; Jussim & Harber, 2005; Merton, 1948), individuals who expect positive outcomes may take measures that help to ensure their success (e.g., seeking opportunities for interpersonal or professional advancement). In contrast, pessimists may decline opportunities for social engagement or professional attainment given their low expectations of success (e.g., anticipating rejection when trying to form friendships or to date), with these self-defeating behaviors serving as a mechanism seemingly to protect themselves against probable disappointment. Thus, not only may pessimism confer heightened risk for subsequent depression through the generation of dependent stress (Harkness & Stewart, 2009), but this negative future

orientation may be associated with lower rates of positive dependent events.

Methodological Considerations and Limitations

Despite the numerous strengths of this comprehensive meta-analysis, discussion of the limitations pertaining to both the source studies as well as this review is warranted. Indeed, the current review allows for a characterization of the scale to which several aforementioned methodological issues are prevalent in the literature. A substantial number of studies ($k = 36$) were identified that aimed to evaluate stress generation but were excluded from the current review because they did not temporally separate life stress from the predictor variable(s) of interest, thus conflating stress generation with stress exposure. Another sizeable number of studies ($k = 21$) intended to assess stress generation but were also excluded on account of not adequately distinguishing between dependent and independent stress in their analyses. When taken together and considered relative to the number of studies retained in the current review ($k = 102$), it appears that a significant proportion of studies intended to examine the stress generation hypothesis were inadequately designed to evaluate this topic. Studies conflating stress generation with stress exposure may result in inflated effects, and studies that combine dependent and independent stress may report diminished effects.

Second, studies of the mediational component of stress generation were limited in number, and given their heterogeneity, those studies were therefore not subjected to quantitative synthesis and were reviewed via qualitative analysis instead. This allowed for a careful discussion of the various mediational pathways but precluded an estimate of effect size. There were also relatively few studies that measured noninterpersonal dependent stress both in mediation and bivariate analyses; depression was the only risk factor with a sufficient number of effect sizes to be examined in association with specific types of dependent interpersonal stress (i.e., family, romantic, peer), limiting our ability to draw conclusions about whether interpersonal stress in specific domains is more relevant to the stress generation process and whether this differs by developmental period. Keeping with best practices (Siddaway et al., 2019), this meta-analysis included bivariate, not multivariate, effect sizes, but this did not allow for accounting for the possibility that the various risk factors identified may be overlapping by nature. Third, when evaluating articles for inclusion, the current meta-analytic review only used English terms to identify relevant articles and restricted search results to studies published in English-language journals. These factors may impact the generalizability of the findings insofar as they may introduce a monolanguage or monocultural bias (B. T. Johnson, 2021). Indeed, the vast majority of the 104 studies meeting inclusion criteria sampled Western populations ($k = 102$; 98.08%). Even Asian is rarely sampled in these studies ($k = 4$; 3.85%). African and East European countries are altogether absent in this literature. Seeing as the stress generation phenomenon is a product of the transactional relationship an individual has with their environment, it is possible, if not probable, that patterns of findings may differ between individualistic and collectivist cultures. The lack of globally representative perspectives, as indicated by the limited number of non-Western, non-U.S. samples included in this meta-analysis, also reflects the historical context of social science research, wherein studies have traditionally centered the experiences of individuals from Western, educated, industrialized, rich, and democratic societies (i.e., Henrich et al., 2010). It is important to recognize this limitation when interpreting the findings and consider that the evidence base for stress generation outside of Western countries is limited and may manifest differently. For example, the etiological chain between past psychopathology and its future recurrence through the mediating effect of stress generation may be potentially stronger in traditionally more collectivist cultures because of the primacy of interpersonal dependent stress in stress generation and the greater importance of interpersonal relationships in collectivist cultures, respectively. Future research should investigate stress generation among more diverse, non-Western samples to more fully evaluate this possibility.

Furthermore, this meta-analysis included only studies published in peer-reviewed journals, thereby excluding "gray literature," such as unpublished studies, theses, and dissertations, which may have allowed for a greater possibility of publication bias and thus potentially inflating effect size estimates. As mentioned above, to address this possibility, several indices of publication bias were evaluated, and although results found evidence of publication bias, the adjusted effect was only modestly smaller than the unadjusted estimates, suggesting the decision to exclude this literature is not significantly impacting the findings. Consistent with this interpretation, we did not find study quality to be a significant moderator of effect size. Additionally, a recent meta-analysis of psychopathology and stress generation that did include gray literature (Rnic et al., 2023) found largely consistent results.

We now turn to a discussion of important considerations in the measurement of life stress in future studies. In particular, we focus on providing basic guidelines for designing future stress generation studies and new opportunities with technologies for the assessment of interpersonal dependent stress, especially in youth.

Design Recommendations for Stress Generation Research

Research in this field may be strengthened by studies that consistently utilize the following design features: (a) of particular importance, gold standard contextual threat life stress interviews (Hammen, 2005; Harkness & Monroe, 2016; Monroe, 2008); (b) relatively briefer life stress assessment timeframes (i.e., 6 months or fewer); (c) longitudinal designs allowing for clean temporal separation between stress generation and its predictors in analyses; (d) a measure of baseline depression as a covariate to ensure any observed association between a risk factor of interest and prospective dependent stress is not better accounted for by depression; (e) a measure of baseline dependent stress as a covariate to ensure any observed association between a risk factor of interest and prospective dependent stress is not a function of baseline dependent stress; (f) in analyses predicting prospective independent stress, include a measure of baseline independent stress as a covariate; and (g) employ Bayesian analyses, particularly for predictions of independent stress, to complement frequentist analyses for predictions of dependent stress.

Social Media and Stress Generation

In recent years, there have been massive changes in the ways that humans utilize digital technology. Advances in technologies such as computers and smartphones—and, more specifically, social media—have changed the ways that people interact with one

another, as well as with the larger world around them. Little research has examined these new technologies and social platforms within the context of stress generation. To date, there exists a larger body of work examining how social media use and specific social media experiences (e.g., cyberbullying) are associated with psychopathology (Nesi et al., 2021). However, there has been limited (if any) integration of social media within existing life events measures or interviews. It is important to consider the ways in which cognitive vulnerabilities and clinical symptoms that manifest in an individual may affect social media experiences. Social media has the potential to contribute additional layers of stress, particularly at the interpersonal level (Hampton et al., 2015; Murdock, 2013), which may be especially important for adolescents (Steele et al., 2020). Individuals who have cognitive vulnerabilities or engage in problematic interpersonal behaviors such as excessive reassurance-seeking may use social media as a means through which to receive feedback, interpersonal reassurance, and validation (Forchuk et al., 2021; Nesi & Prinstein, 2015; Sheldon & Newman, 2019), which may contribute to subsequent interpersonal dependent stress. Social media provides a platform with novel avenues for cognitive vulnerabilities to be expressed and contribute to subsequent risk for psychopathology. Yet, more research is needed to understand the nuanced ways in which new digital technologies may be implicated in the relationship between stress and psychopathology (Wolfers & Utz, 2022), which may help partially to explain rising rates of psychopathology in young people.

Technology to Improve Stress Generation Research

However, advanced technologies not only impact how life stress is experienced but also provide new opportunities to further explore and assess life events and the temporal sequelae that elicit stress generation and subsequent psychopathology. For instance, it remains unclear how vulnerability factors or psychopathology might influence the occurrence of dependent stress in real time. Understanding how these factors unfold in individuals' day-to-day lives would provide critical information about optimal points of intervention. Given that most stress generation measures cover periods ranging from several months to years, repeated and shorter time frames for assessment could inform just-in-time interventions targeting these factors to prevent the occurrence of more harmful forms of dependent stress. Thus, ecological momentary assessment (EMA) may be one promising methodological approach to improve future research into the stress generation hypothesis. Indeed, only six studies reflecting five unique samples have evaluated stress generation at the daily level (Cummings et al., 2010, 2013; Eberhart & Hammen, 2009; Herres & Kobak, 2015; Sahl et al., 2009; Shih & Auerbach, 2010). While EMA tools would likely be self-reported daily life stress, this method could be paired with the gold-standard approach of using interviews to qualify and contextualize the occurrence, dependency, and severity of these events (Harkness & Monroe, 2016). Using EMA or other ambulatory monitoring approaches that assess vulnerability factors and psychopathology could further enhance our knowledge of state-like factors and behaviors that contribute to dependent stressors, particularly those that are interpersonal in nature and how they contribute to subsequent psychopathology unfolding over time at the daily level.

Furthermore, new methodological and computational approaches now make it possible to collect additional information about the occurrence of life stress from individuals in an ecologically valid way without much participant burden, such as through passive data collection of social media. As noted above, the prevalence and importance of social media can make this a rich source of information about individuals' social interactions and help gauge life stressors through social media posts, comments, and updates. Active engagement in social media (e.g., posts, likes, comments, and shares) can be readily obtained, retrieved, and donated by participants for some platforms such as Facebook or Instagram (most popular for older adults and youth, respectively) or collected by the research team using publicly available data through APIs (e.g., Twitter, TikTok).

Natural language processing and machine learning techniques can then be used to analyze and extract coded information from social media posts (text-based or audio language) to further measure life stress as it unfolds in participants' lives. Indeed, there have been notable advances in research aiming to detect life stress through social media posts (Lin et al., 2016). However, caution is warranted in applying these methods due to potential ethical considerations and potential bias due to differences in individuals who do and do not post life events on social media. For example, it should be carefully considered whether information reported by youth and other vulnerable populations should be extracted from publicly available social media data sets, especially if those individuals did not directly consent to data sharing. This consideration is especially important to weigh for data that contain sensitive information (e.g., sexually explicit content, alcohol or substance use, communication of mental health crisis) from youth or vulnerable populations. Further, even if one individual does consent to sharing data, it raises concerns about identifying information (e.g., names, photos) of others with whom the individual is interacting on social media, especially for those using private accounts or messaging platforms. It would therefore be important for studies with social media data to develop and employ programming scripts to scrub out identifying information to mitigate these concerns.

Still, evidence-based life stress measures paired with these innovative approaches may further aid in our understanding of stress generation by providing new insight into the occurrence and nature of life stress across domains. Thus, multimethod approaches that use both established methods of life event interviews and newer real-time approaches to assessing life events, vulnerabilities, and psychopathology may provide new insights into our understanding of the stress generation process and clinical implications.

Conclusions

In our systematic review and meta-analysis of 30 years of published research on stress generation, we found strong and consistent support for stress generation effects for overall psychopathology, internalizing psychopathology, and depression. They remained reasonably robust to statistical adjustments for potential publication biases. Support was also observed for the mediational component of the stress generation hypothesis. Analyses of independent stress yielded a more complex picture, with general evidence emerging for modest but clear association between psychopathology and prospective independent stress. At the same time, associations with dependent stress were, for the most part, significantly stronger than associations with independent stress. This set of findings and proposed refinements to stress generation warrant further consideration. Finally, future

developments adopting new technological opportunities for studying life stress and efforts to develop sensitive assessments of this construct within minority stress and social–ecological frameworks have potential to add further insights into our understanding of stress generation.

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