

Review

Risk and protective factors for stress generation: A meta-analytic review

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ABSTRACT

The stress generation hypothesis suggests that some individuals contribute more than others to the occurrence of dependent (self-generated), but not independent (fateful), stressful life events. This phenomenon is commonly studied in relation to psychiatric disorders, but effects are also driven by underlying psychological processes that extend beyond the boundaries of DSM-defined entities. This meta-analytic review of modifiable risk and protective factors for stress generation synthesizes findings from 70 studies with 39,693 participants (483 total effect sizes) from over 30 years of research. Findings revealed a range of risk factors that prospectively predict dependent stress with small-to-moderate meta-analytic effects ($r_s = 0.10$ – 0.26). Negligible to small effects were found for independent stress ($r_s = 0.03$ – 0.12), and, in a critical test for stress generation, most effects were significantly stronger for dependent compared to independent stress ($\beta_s = 0.04$ – 0.15). Moderation analyses suggest effects of maladaptive interpersonal emotion regulation behaviors and repetitive negative thinking are stronger for interpersonal (versus non-interpersonal) stress; effects of repetitive negative thinking and excessive standards for self may be inflated by overreliance on self-report measures that fail to isolate psychological distress from objective experience. Findings have key implications for advancing stress generation theory and informing targets for intervention.

1. Introduction

Life is often punctuated by stressful events—discrete episodes of threatening circumstances that challenge our coping resources. The extent to which people experience stressful life events has a profound impact on well-being, with proximal and lasting effects on mental and physical health (Slavich, 2020). It is difficult to overstate the etiological importance of life stress in the onset, worsening, and maintenance of a broad range of health challenges. For example, depressive episodes (Hammen, 2005; Vrshek-Schallhorn et al., 2020) and suicidal behavior (Stewart et al., 2019; Yen et al., 2005) are commonly preceded by stressful life events, and repeated exposure to life stress can alter the functioning of stress response systems and contribute to increased allostatic load (McEwen, 1998; Young et al., 2021). Advancing

understanding of the specific processes that contribute to or protect against the occurrence of life stress therefore has key implications for improving well-being and mitigating risk for physical and mental health problems. Critically, although some stressors are unavoidable and uncontrollable, the stress generation model (Hammen, 1991) holds that people play an active role in shaping their environment, with certain traits, behaviors, and cognitive styles influencing the likelihood that they will experience stressful life events. As a result, stress exposure is malleable: by modifying risk and protective factors, we can promote a more harmonious environment. To inform theory and intervention efforts, the current project provides a comprehensive meta-analytic review of cross-cutting risk and protective factors for stress generation.

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1.1. Theoretical and methodological considerations for stress generation research

The stress generation hypothesis (Hammen, 1991) suggests that some individuals contribute more than others to the occurrence of *dependent* stressors (i.e., stressful life events that occur at least in part due to individuals' behavior or personal characteristics, such as relationship breakups, failing a class, or job loss due to conflict with a coworker), but not *independent* stressors (i.e., fateful events that occur irrespective of individuals' influence, such as the death of a loved one or job loss due to an economic downturn). The stress generation phenomenon was originally examined in the context of depression research. More recently, stress generation has been studied in relation to a broader range of psychological disorders and, in a companion paper to the present meta-analysis, we synthesized the literature examining mental disorders and symptoms as predictors of stress generation (Rnic et al., 2023). We found that diverse forms of psychopathology (e.g., internalizing, externalizing) prospectively predict greater dependent episodic life stress which, in turn, exacerbates symptoms of psychopathology over time, potentially contributing to chronicity. Although this work broadens our understanding of stress generation as a transdiagnostic phenomenon, research and theory suggest that stress generation effects are also driven by personal characteristics or behavioral styles that are present prior to the onset of psychopathology, that endure outside of periods of active symptomatology, and that cut across or extend beyond the boundaries of DSM-defined disorders (e.g., Hammen, 2006). Thus, limiting our examination of predictors of stress generation to psychopathology provides an incomplete picture of the processes that contribute to, or protect against, the generation of stressors. Indeed, mounting evidence suggests a range of psychological processes other than psychopathology contribute to stress generation. A quantitative synthesis of these findings has the potential to significantly advance our understanding of the mechanisms underlying stress generation and to facilitate identification of targets for intervention.

Researchers generally agree upon a few core study features that are required to test the stress generation hypothesis (e.g., Alloy et al., 2010; Liu, 2013; Meyer & Curry, 2017). For instance, researchers are advised to use a longitudinal study design in which a predictor of interest is measured at one wave and stressful life events during an intervening period are measured at a subsequent wave. Longitudinal designs help to establish the temporal precedence of a given risk or protective factor and avoid the pitfalls of retrospective reporting (e.g., memory biases that impact the accuracy of reporting on past events). To allow for a direct test of the stress generation hypothesis, researchers must also distinguish between *dependent* and *independent* stressors. The most robust test of stress generation involves comparing effects of a predictor on dependent versus independent stress; when a particular risk factor is prospectively associated with the occurrence of dependent stress, but not independent stress, this pattern of findings indicates strong support for the stress generation hypothesis. Most of this research focuses on episodic stressors, which are characterized by the occurrence of a precipitating event with a discrete onset and offset and a relatively short duration, rather than chronic stressors. It is comparatively easier to establish the timing and dependence of episodic stressors, whereas chronic stressors are defined by their persistent, long-term course. Moreover, the causes of changes in chronic stress are more challenging to disentangle with regard to dependence. Finally, although not an essential test of stress generation, researchers also commonly distinguish between events that are primarily interpersonal versus non-interpersonal in nature. This distinction stems from the salience of interpersonal life stress in the etiology of depression and the disruptions in interpersonal functioning commonly observed among those with depressive disorders (Hammen, 2006). Although not all dependent stressors are interpersonal in nature, interpersonal stressful life events (e.g., conflict, disrupted relationships) are more likely to be dependent (Hammen, 2006).

1.2. Estimating the impact of risk and protective factors for stress generation

Multiple prior reviews have proposed theoretical models to explain individual differences in stress generation (Harkness & Washburn, 2016; Liu, 2013; Meyer & Curry, 2017). Across these models, authors suggest that fixed characteristics beyond individuals' control (e.g., parental psychopathology, genetics) and early stress exposure (e.g., childhood maltreatment) interact and play a role in shaping personality traits, cognition, and dispositional factors (e.g., neuroticism, negative cognitive styles, attachment style). These *distal* processes are thought to exert their influence on stress generation through *proximal* behavioral tendencies (e.g., excessive reassurance seeking, avoidant coping strategies, aggression). Moreover, to the extent that stress generation is predominantly an interpersonal phenomenon, those processes that contribute to or reflect disruptions in interpersonal functioning are theorized to be particularly potent predictors of self-generated stress. Prior reviews (e.g., Liu, 2013) emphasize the need to identify specific mechanisms underlying stress generation (especially those that may cut across or extend beyond the bounds of individual psychiatric disorders) and examine the complex temporal and interactive relationships among these processes in the pathway to stress generation. The current review takes a critical step in this direction by summarizing the available literature on cross-cutting risk factors for stress generation.

Importantly, our review also builds upon prior work by incorporating consideration of theoretically protective factors for stress generation. Although the identification of risk factors for stress exposure and mental illness are critical goals for improving public health, professionals have also increasingly emphasized the importance of understanding protective factors (Shaffer & Yates, 2010). In this context, we consider protective factors to be variables that do not merely represent the absence/low levels of risk factors, but instead reflect individual characteristics (e.g., secure attachment style, enhancing cognitive style) that actively contribute to well-being (e.g., by facilitating the development of healthy social relationships) and thereby reduce the likelihood of exposure to dependent episodic life stress.

While various environmental factors (e.g., early stress exposure) and fixed factors (e.g., demographics) are also theorized to play a role in the path to stress generation, the present review focuses specifically on synthesizing findings for those risk and protective factors that represent individual characteristics or behavioral styles that contribute to or protect against stress generation and could be targeted for change in intervention efforts. As described in the sections that follow, we also leveraged available data to test whether certain fixed factors (e.g., gender) moderate stress generation effects. To facilitate analyses, we planned from the outset to assign individual predictor constructs to higher-order categories based on their conceptual similarities, then use these higher-order categories throughout analyses. Importantly, predictor categories were defined after the initial literature review but before data analysis; this approach allowed us to determine which constructs had sufficient coverage in the literature to be combined into meaningful, higher-order categories prior to the quantitative synthesis. Details regarding our process for creating predictor categories are provided in the Method.

1.3. Moderators of stress generation effects

Beyond quantifying the magnitude of stress generation effects, theory would benefit from a deeper consideration of for whom and under which conditions these effects are strongest. The present review leverages meta-analytic techniques to test whether stress generation effects systematically differ as a function of selected demographic and methodological features: gender composition, stress outcome domain, and stress assessment method.

1.3.1. Gender

As earlier narrative reviews of this literature have observed, stress generation effects are often more pronounced among women (Liu & Alloy, 2010), and evidence from individual studies suggests that women report higher rates of stressful life events compared to men (Hankin et al., 2007; Harkness et al., 2010; Shih, 2006). At the same time, some researchers suggest that the specific processes driving stress generation effects among men may be different than those driving effects among women (Harkness & Washburn, 2016; Shih, 2006; Shih et al., 2006). For example, it may be that risk factors like interpersonal dependency or maladaptive support seeking behaviors (e.g., co-rumination) are particularly strong predictors of stress generation among girls (Bouchard & Shih, 2013) and women. Whether stress generation effects systematically differ as a function of gender, such that effects are uniformly stronger for women across the board, or whether different patterns of gender moderation emerge for different types of risk factors, has critical implications for the development of stress generation theory. Thus, the present meta-analysis tested whether the gender composition of study samples moderates stress generation effects across a broad range of risk and protective factors.

1.3.2. Stress outcome domain

As reviewed above, much of the existing literature emphasizes the importance of risk factors related to interpersonal functioning and the generation of *interpersonal* life stress (Hammen, 2006). However, the extent to which stress generation is predominantly an interpersonal phenomenon has yet to be tested systematically. Theory would benefit from a direct examination of whether stress generation effects are uniformly and significantly stronger for interpersonal versus non-interpersonal stress, or whether different patterns of moderation emerge across distinct classes of risk factors. For example, it would be instructive to know whether effects are significantly stronger for interpersonal stress only when the predictors in question primarily reflect disruptions in interpersonal functioning. Similarly, it would be important to establish whether certain risk factors are especially strong predictors of non-interpersonal stress. This meta-analysis therefore examined whether stress generation effects differ in magnitude as a function of stress outcome domain (interpersonal or non-interpersonal) across diverse categories of predictors.

1.3.3. Stress assessment method

Researchers commonly use one of three methods to measure episodic life stress within the stress generation literature: interviews, checklists, and “hybrid” approaches. Interview-based methods that adopt a contextual threat approach to determine the occurrence and objective severity of stressful life events are widely regarded as the gold standard in the field (Monroe, 2008). These methods allow researchers to gather important contextual information to facilitate more accurate coding of (a) whether stressors are truly episodic in nature, (b) whether they are dependent or independent, and (c) the level of objective threat associated with each stressor. Self-report checklists typically require participants to review a predetermined list of negative life events (e.g., “major illness or injury”, “major financial difficulty”) and indicate which events they have experienced during a specified period; some checklists also ask individuals to indicate the frequency or severity of each event they experienced. Checklist measures are widely acknowledged to yield lower quality, less objective data than do interviews (see Harkness & Monroe, 2016; Monroe, 2008). Although some of this difference in quality may be attributed to random error, it is likely to represent systematic bias, as self-report checklists may be more likely to conflate the *objective* occurrence of events with individuals’ *subjective* response to stressors due to reporting biases stemming from participants’ current mood, negative attributional style, or inferences they make about the purpose of the stress assessment (Harkness & Monroe, 2016). If life stress checklists tap elements of certain predictors of stress generation, we might expect stronger effect sizes to emerge when life stress is

assessed using checklists rather than interview-based methods. Finally, “hybrid” approaches typically involve the administration of a life event checklist, which is then followed up with an interview to probe for additional details about endorsed events (e.g., to confirm events actually occurred and were independent of one another or to obtain contextual information to facilitate coding of event severity, dependence, etc.). Given important methodological and psychometric differences across methods of life stress assessment, we tested whether stress generation effects systematically differ as a function of stress assessment method.

1.4. Current project

The current study presents a systematic review and meta-analysis of risk and protective factors for stress generation. A systematic literature search identified longitudinal studies examining one or more prospective predictors of dependent or independent episodic life stress. We specifically focused our review on effects for individual characteristics and behavioral styles that cut across or extend beyond the boundaries of single psychiatric diagnoses, that are potentially modifiable, and that therefore may have critical implications for intervention. We excluded environmental factors because they are difficult to disentangle from dependent or independent stressors (e.g., receipt of social support) or are no longer modifiable (e.g., exposure to early life stress). We also excluded other fixed factors (e.g., demographic variables like gender, family history of psychopathology). This meta-analytic review advances stress generation theory by accomplishing two primary goals. First, we aim to summarize what is known about specific risk and protective factors for stress generation. Guided by prior research, we grouped individual predictor constructs into theoretically-linked categories of risk and protective factors and used a three-level random effects meta-analytic approach to compute pooled estimates of effects on dependent and independent life stress. As a more robust test of the stress generation hypothesis, we also directly compared the relative magnitude of effects for dependent versus independent life stress. Second, we address key theoretical questions about when and for whom stress generation effects are strongest by testing whether sample characteristics (e.g., gender) and methodological factors (e.g., stress domain, stress assessment method) moderate risk factor effects. We hypothesized that stronger meta-analytic effects would emerge when (a) samples included a higher proportion of women, (b) the life stress domain was interpersonal (versus non-interpersonal stress), and (c) life stress was measured with self-report checklists (versus interview-based or hybrid approaches). In addition to our main theoretical moderators, we also tested effects of three descriptive moderators: sample race (i.e., percent of sample identifying their race as White), sample age (i.e., mean sample age at baseline), and length of follow-up (i.e., months elapsed between assessment of predictors and assessment of life stress).

2. Method

This meta-analytic review was conducted in line with PRISMA reporting guidelines (Moher et al., 2009), and the protocol was publicly pre-registered via PROSPERO (CRD42020198180). This project represents the joint effort of two research teams led by AS and KR who initially developed systematic reviews with highly overlapping aims, search strategies, and analytic plans independently (“Team USA”: AS, KC, RC, LS and “Team Canada”: KR, DD, JAH, HL, JL). Upon discovering one another’s projects, we agreed to collaborate on one exceptionally comprehensive and rigorous review by merging these projects. Thus, two independent literature searches were conducted, and our teams made joint decisions regarding final eligibility criteria, coding, and analyses.

2.1. Search strategy

Systematic searches of the literature were conducted independently

by each research team, with the final searches complete through July 2022. Details regarding the specific databases, search terms, and parameters used for each search are presented in [Appendix A](#). A flow diagram displaying each step of the search process is depicted in [Fig. 1](#). The combined search spanned three electronic databases: PsycINFO, PubMed, and Web of Science. Manuscripts were identified using search terms relevant to stress generation (e.g., “stress generation”, “episodic stress”, “negative event”, “life event”). Searches were limited to manuscripts written in English and describing human subjects research; results included published journal articles, as well as unpublished dissertations and theses. In the initial database search, Team USA and Team Canada identified 8869 and 3122 records for review, respectively. Further records were identified in the following ways: (a) a backward search was conducted using reference lists for all studies that ultimately met inclusion criteria, as well as any relevant review papers and book chapters identified in the database searches; (b) a forward search was conducted to identify potentially eligible papers citing the seminal stress generation paper ([Hammen, 1991](#)); and (c) manuscript authors were contacted via email to obtain additional information when insufficient statistical information was available to compute a standardized effect but their study otherwise met eligibility criteria. These processes yielded 605 additional records for review. After removing duplicates, Team USA

and Team Canada reviewed 6389 and 2814 abstracts for eligibility, followed by 623 and 983 full text records, respectively. Prior to combining efforts, results from each search were independently screened and coded for reliability by two raters within each research team. Upon merging our datasets, data were checked for consistency by a third rater, and any remaining studies were independently coded by two raters from the combined team. Thus, data were double coded for 100% of included studies, and in many cases studies were triple- or quadruple-coded. Any coding discrepancies were resolved through discussion between the co-first authors (AS and KR).

2.2. Eligibility criteria

Included studies met the following criteria: (a) longitudinal study design, (b) measurement of at least one risk or protective factor at a wave prior to an assessment of life stress, (c) assessment of episodic stressful life events which authors categorized as dependent or independent at follow-up, and (d) authors provided sufficient statistical information to compute standardized bivariate effect sizes for the prospective relationship between individual predictors and later episodic life stress (i.e., effect sizes that represent the association between the predictor and life stress outcome without controlling for other

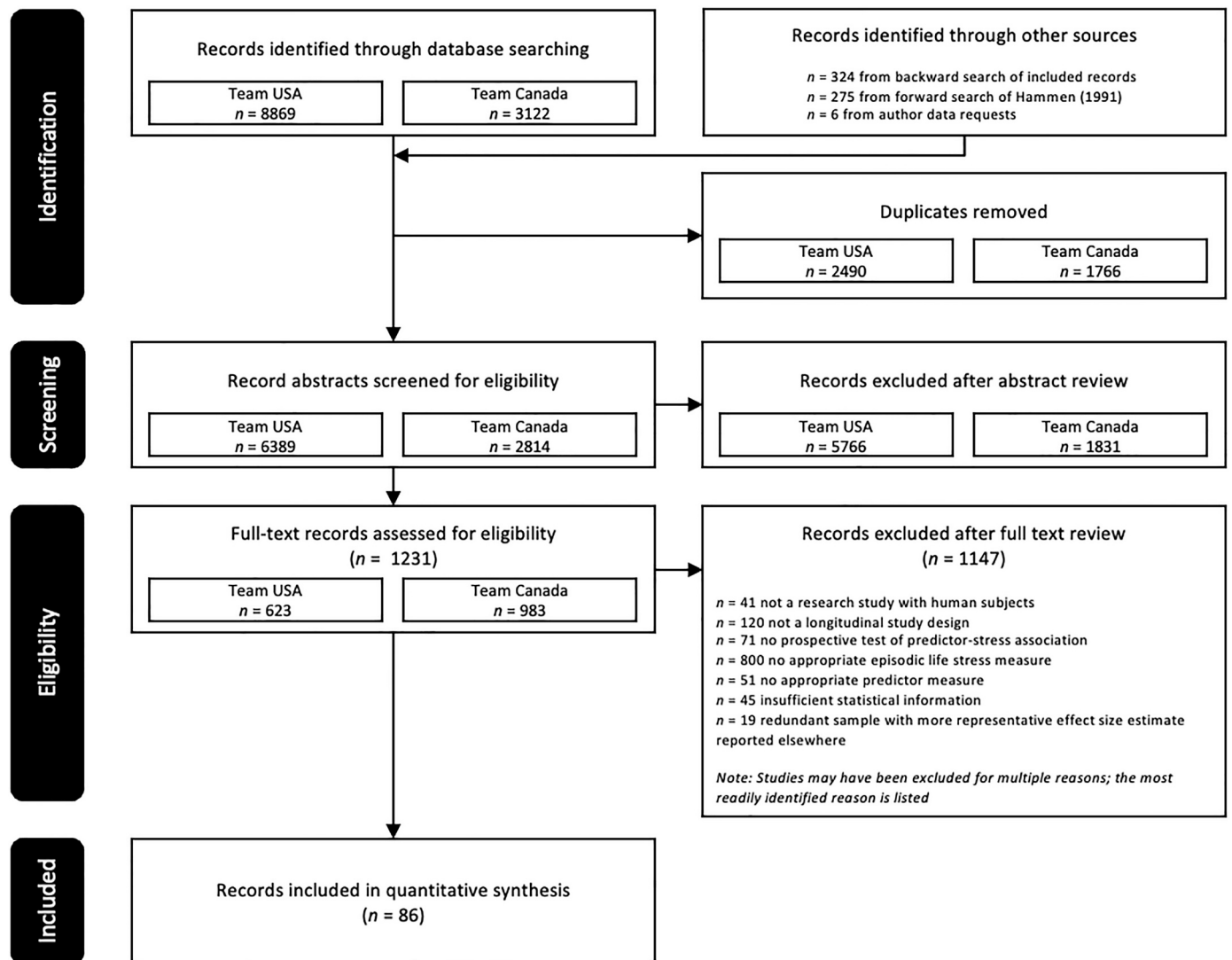


Fig. 1. PRISMA flow diagram for manuscript identification, screening, and inclusion.

Note. Unless otherwise specified, *ns* represent the combined count of records after accounting for duplicates between the two independent searches. *n* = number of published or unpublished manuscripts or unpublished studies with data obtained through author data requests.

variables). Of note, we focused on potentially modifiable risk and protective factors that were endogenous to the individual. Predictors were excluded when they were fixed (e.g., gender), primarily assessed aspects of environmental stress (e.g., childhood adversity, chronic interpersonal stress), or otherwise could not be disentangled from individuals' experience of episodic life stress (e.g., emotional reactivity to daily stressors whereby scores for emotional reactivity were directly influenced by, and confounded with, the occurrence of life stressors). This meta-analysis focuses on cross-cutting risk and protective processes rather than specific DSM-related entities, so psychological disorders and symptoms were excluded as predictors. For a companion meta-analysis of psychopathological constructs (e.g., depression, externalizing) as predictors and outcomes of stress generation, see Rnic et al. (2023). See also Liu et al. (2023), an additional meta-analysis of stress generation research which was under review at time of writing.

2.3. Data extraction and coding

Several pieces of information were systematically extracted from each included study: (a) characteristics of the manuscript (e.g., published or unpublished), (b) sample characteristics, including the overall *N* for participants at baseline, age range, mean, and standard deviation, percent identifying as girls/women, percent reporting their race as White; (c) characteristics of each predictor, including the construct assessed, measure, and whether it was conceptualized as a risk or protective factor; (d) characteristics of the episodic life stress assessment, including the assessment method (i.e., checklist, interview, or "hybrid" assessment), time elapsed between assessment of the predictor and assessment of life stress, measure, dependence (i.e., dependent or independent), and domain of life stress (i.e., interpersonal, non-interpersonal, or combined); when effects were presented for subtypes of dependent interpersonal (e.g., family conflict stress, peer stress) and non-interpersonal (e.g., academic, financial) stress, we re-coded the domain of life stress to fit one of these two categories (e.g., in this example, interpersonal and non-interpersonal, respectively); (e) statistical information reported by the authors for each effect size. To reduce sources of dependency that are not the focus of this review and to improve comparability of effects across studies, some additional rules were employed to select individual effect sizes for inclusion in the quantitative synthesis (see Appendix B). Whenever possible, a bivariate correlation coefficient (*r*) was extracted for the association between a predictor assessed at Time 1 (T1) and episodic life stress assessed at a subsequent wave of data collection. When this information was unavailable in the manuscript or through follow-up contact with authors, the best available information was extracted (e.g., group means and standard deviations) and used to compute the bivariate effect size in the *r* metric. These correlation coefficients were transformed to Fisher's *Z_r* correlations for use in analyses; effects were converted back to the *r* metric for reporting of average meta-analytic effects.

2.3.1. Creation of predictor clusters

Following data extraction, we grouped predictor constructs into theoretically-linked categories guided by prior research. These predictor clusters were created prior to conducting analyses, and assignment of individual constructs to clusters was determined through consensus coding between the co-first authors, with additional input provided by authors LS, JL, KC, and DD. The nine clusters we created are described in turn below, and assignment of individual constructs to clusters is noted in Appendix C. Note that although effect sizes for all individual predictor constructs meeting inclusion criteria are displayed in Appendix C, not all predictors were assigned to higher-order clusters. This is true of any predictors for which effect sizes were obtained from too few studies to compute a pooled meta-analytic effect for either dependent or independent stress (see Analytic Strategy). With the goal of facilitating interpretability and reducing unnecessary heterogeneity within clusters, we prioritized assigning predictors to clusters based on careful

consideration for how they hang together conceptually.

2.3.1.1. Maladaptive interpersonal emotion regulation behaviors. This cluster is composed of interpersonal behaviors that serve an emotion regulation function and that are marked by excessive use of otherwise normative forms of support seeking: co-rumination, excessive reassurance seeking, and negative feedback seeking. Prior research has linked these processes to one another (Dixon-Gordon et al., 2015; Evraire & Dozois, 2011; Starr, 2015) and suggests that each contributes to stress generation, potentially by straining interpersonal relationships and increasing the likelihood of interpersonal rejection (e.g., Evraire et al., 2022).

2.3.1.2. Disinhibition and antagonism. This cluster reflects two interrelated traits that are delineated by the DSM-5 Dimensional Trait Model, and which overlap with the agreeableness and conscientiousness domains of the Five Factor Model (Kotov et al., 2017; Vize et al., 2020). Disinhibition is characterized by risk taking and difficulty with inhibiting automatic impulses, and antagonism is characterized by callousness, difficulties cooperating effectively with others, and aggression. This cluster therefore includes the following predictors: aggression, antagonism, delinquent acts, and impulsivity. Individuals high on antagonism and disinhibition may generate stress due to impulsive, reactive, or aggressive behaviors that deteriorate interpersonal relationships.

2.3.1.3. Avoidance. This cluster describes a range of coping behaviors used to disengage with distress-inducing stimuli, and includes the following predictors: avoidance, avoidant coping strategies (disengagement coping, emotional discharge coping, cognitive avoidance coping), avoidance goals, involuntary disengagement, social disengagement, and behavioral inhibition. Though the avoidance of negative experiences may lead to fewer stressors in the short-term, avoidance behaviors have been shown to generate negative life events in the long-term (Meyer & Curry, 2017). Researchers have hypothesized that avoidance behaviors are inefficient and ineffective coping methods that can induce negative emotional states (such as worry, distraction, or threat) that in turn generate stressful life events (Elliot et al., 2011).

2.3.1.4. Repetitive negative thinking. This cluster describes a range of constructs that share common processes associated with repetitive, passive, difficult-to-control, and negative self-referent thought content (Ehring & Watkins, 2008): rumination, positive affect dampening, and worry. Repetitive negative thinking is often employed as a coping strategy to manage difficult emotions, and some research indicates that repetitive negative thinking offers an ineffective solution to managing distress that can ultimately exacerbate stressors (Lyubomirsky & Nolen-Hoeksema, 1995) and contribute to strain in interpersonal relationships (Bushman et al., 2005; Nolen-Hoeksema & Jackson, 2001).

2.3.1.5. Negative cognitive content. This cluster includes constructs related to negative self-referent cognitions and negative expectations: hopelessness, negative cognitive style, maladaptive schemas, social cognitive distortions, and negative relational self-views. Whereas repetitive negative thinking involves active coping strategies employed in the context of negative events or emotions, negative cognitive content describes a stable tendency towards negative interpretations of the self and the future. Cognitive theories describe depression as characterized by a prolonged bias towards negative thinking patterns (Beck, 1987; Clark et al., 2000) that are both exacerbated by and can cause stressful life experiences (Seeds & Dozois, 2010; Simons et al., 1993). Negative cognitive content, including negative beliefs regarding one's emotions, adequacy, and expectations for the future, has been linked to stress generation in prior research (e.g., Eberhart & Hammen, 2009).

2.3.1.6. Excessive standards for self. This cluster encapsulates several dimensions of perfectionistic concerns and perfectionistic strivings: self-criticism, socially prescribed perfectionism, concern over mistakes, doubts about actions, performance evaluation, and perfectionistic personal standards. Research suggests that perfectionism is a risk factor for depressive symptoms that is conceptually distinct from neuroticism (Hewitt et al., 2022; Smith et al., 2016) and confers risk for stress generation. For example, individuals high on perfectionism may generate stressful experiences by setting unrealistic personal goals and subsequently pursuing potentially stressful circumstances (Smith et al., 2020).

2.3.1.7. Pervasive negative affect. This cluster represents dispositional tendencies towards negative emotions, and includes the following predictors: neuroticism, negative emotionality, and negative affect. Individuals high in trait negative affect may generate life stressors due to a heightened propensity for intense negative emotions and a tendency to perceive stressors as threats rather than challenges (Suls, 2001). Individual studies have indeed demonstrated that high levels of neuroticism prospectively predict the occurrence of negative life events (e.g., Hankin, 2010).

2.3.1.8. Dispositional other-oriented focus. This cluster encapsulates constructs associated with an excessive focus on and/or maladaptive patterns of relating with others, and includes the following predictors: dependency, sociotropy, other-directedness, anxious attachment, rejection sensitivity, unmitigated communion (focusing on the needs or wants of others to the exclusion of oneself), and interpersonal problems characterized by other-oriented focus (e.g., too dependent, too caring). Collectively, these constructs represent stable traits that characterize the formation, meaning, and patterning of emotional bonds. Individuals with a higher degree of other-oriented focus and who base their sense of self-worth on interpersonal relationships may exhibit a range of behaviors aimed at precluding abandonment, but which ultimately strain their social relationships. Individual studies have indeed shown that other-oriented characteristics are associated with the generation of relational conflict (Eberhart & Hammen, 2009; Shih, 2006).

2.3.1.9. Dispositional positive affect & upregulation. We created one cluster to capture theoretically protective factors that represent dispositional positive affect and upregulation: enhancing cognitive style, extraversion, positive emotionality, positive affect, emotion-focused savoring of positive affect, and self-focused savoring of positive affect. Traits associated with maintaining and upregulating positive affect may protect against stress generation by preventing the escalation and occurrence of stressors. Research indeed suggests that positive emotionality moderates the link between rumination and chronic interpersonal stress generation, such that individuals with higher levels of positive emotionality were protected against the negative effects of rumination (Stroud et al., 2015).

2.4. Analytic strategy

2.4.1. Three-level meta-analytic approach

Most studies (97%) reported multiple effect sizes, violating assumptions of independence underlying traditional meta-analytic approaches (i.e., fixed and two-level random effects models). Thus, we used a multi-level meta-analytic approach to allow for inclusion of all effect sizes that met criteria. Beyond avoiding the pitfalls of more common approaches for addressing dependencies among effect sizes (e.g., by choosing one effect size among many using arbitrary decision rules or by averaging across effects), this approach is ideal for directly testing whether the average effect sizes for dependent versus independent stress significantly differ (a critical test of the stress generation hypothesis).

Following established guidelines (Assink & Wibbelink, 2016; Cheung, 2014), we applied a three-level structure to each meta-analytic

model to account for the sampling variance around the estimated population effect size (Level 1), variance between effect sizes within studies (Level 2), and variance between effect sizes across studies (Level 3). Estimation of parameters was performed using restricted maximum likelihood estimation (REML). Across all models, we applied the robust variance estimation (RVE) method with small sample adjustment to correct the meta-analytic estimates of correlation coefficients and their standard errors (Fernández-Castilla et al., 2020; Tipton, 2015). First, when effect sizes were obtained from $k \geq 5$ unique study samples for a given predictor cluster, we estimated an overall effect size for that cluster and later dependent or independent life stress. These meta-analytic effects were computed using separate models for effects on dependent or independent stress. For each predictor cluster, we estimated the distribution of variance across the three levels of the model. We proceeded with tests of candidate moderators of meta-analytic effects when sufficient heterogeneity was present, which we determined to be the case when variance at Level 1 was estimated to be $<75\%$ (Assink & Wibbelink, 2016; Hunter & Schmidt, 2004).

2.4.2. Moderation analyses

For each predictor cluster, we fit a series of three-level mixed effects models to examine whether candidate moderators explain significant variance in stress generation effects between and within studies. First, to directly examine whether average effects for dependent and independent stress significantly differed from one another, we pooled effects across both stress outcomes and tested a model with stress dependence as a moderator. Results are interpreted as supportive of the stress generation hypothesis when effects are stronger for dependent stress compared to independent stress.

Next, we tested whether sample demographics, length of follow-up, and stress outcome characteristics explain significant variance in effect sizes for dependent stress between and within studies. Our primary moderators of interest included gender, stress assessment method, and stress domain. Gender was examined as a continuous moderator based on the proportion of each sample identifying their gender as girls/women. Stress assessment method was tested as a categorical moderator with two levels: checklist versus interview/hybrid. This decision was made because hybrid measures are defined by the inclusion of an interview component, and thus they more closely resemble the “gold standard” interview approaches than pure life event checklist approaches. Stress domain was also tested as a categorical moderator with two levels for interpersonal versus non-interpersonal stress. Effects for which the outcome domain was coded as “combined” were dropped from the model for this analysis, as these reflect a mix of interpersonal and non-interpersonal stressors. Additional moderators tested for descriptive purposes included race (a continuous variable representing percentage of the sample that identified as White), age (a continuous variable based on mean sample age), and follow-up length (a continuous variable computed as months between assessment of the predictor and assessment of life stress); hypotheses were not advanced for these moderators. Separate three-level models were fit for each moderator. For analyses of moderation by follow-up length, we included effect sizes for all available follow-up waves with stress assessment within each study. All other analyses used the effect size of each predictor-outcome association for the first available follow-up lag.

2.4.3. Publication bias

Consistent with other recent three-level meta-analytic reviews (e.g., Cahill et al., 2021; Giletta et al., 2021), we evaluated publication bias using multiple methods. First, we visually inspected funnel plots of effect sizes for each higher-order predictor domain on each life stress outcome. Asymmetry may be present when effect sizes are unequally distributed around their mean and effects with larger standard errors are disproportionately available for one side of the plot, potentially signaling that effects from smaller studies with unfavorable or nonsignificant findings are underreported. Second, based on Egger's test of

asymmetry and using the RVE method, we tested a model in which observed effect sizes were regressed on their study weights and their standard error was examined as a moderator of this association. Finally, we tested whether manuscript publication status moderated effects; publication bias may be present if stronger effects are observed in published compared to unpublished studies.

Analyses were conducted in R (Version 4.0.4; R Core Team, 2018). Multilevel meta-regression models were estimated using the *metafor* package (Version 3.0–2; Viechtbauer, 2010); RVE corrections were applied using the *robumeta* (Version 2.0; Fisher et al., 2017) and

clubSandwich (Version 0.5.5; Pustejovsky, 2022) packages. All materials, including the data, codebook, and R scripts for analyses are available on the Open Science Framework (<https://osf.io/e7mg2/>).

3. Results

3.1. Study characteristics

This systematic search yielded 86 manuscripts, with findings from 70 unique studies including 39,693 participants from over 30 years of

Table 1

Summary of meta-analytic associations between predictor clusters and later life stress.

Predictor Cluster / Stress Outcome	k ESs	k studies	Effect Size Estimates		Heterogeneity Estimates					Tests for Publication Bias		
			<i>r</i> (95% CI)	<i>t</i>	<i>Q</i>	σ^2 L2	σ^2 L3	<i>I</i> ² % L1	<i>I</i> ² % L2	<i>I</i> ² % L3	Funnel Plot Asymmetry	Moderation by Publication Status
Maladaptive interpersonal ER behaviors	28	10										
Dependent stress	21	10	0.17 (0.13, 0.22)	8.72***	33.24*	0.003	0.000	61.96	38.04	<1	$\beta = 0.98$	$t(1.45) = 0.95$
Independent stress	7	6	0.03 (−0.05, 0.10)	1.01	5.47	0.000	0.000	100	<1	<1	$\beta = -0.14$	$t(3.62) = 3.29^*$
Disinhibition and antagonism	30	10										
Dependent stress	18	10	0.18 (0.12, 0.24)	7.28***	35.29**	0.005	0.000	45.13	51.21	3.66	$\beta = -0.69$	$t(1.56) = 0.50$
Independent stress	12	8	0.07 (0.03, 0.11)	4.63**	9.58	0.000	0.000	100	<1	<1	$\beta = 0.15$	$t(1.25) = 0.88$
Avoidance	18	9										
Dependent stress	15	9	0.10 (0.03, 0.18)	3.28*	49.63***	0.003	0.005	27.95	28.51	43.54	$\beta = 0.31$	$t(6.70) = -0.29$
Independent stress	3	3	–	–	–	–	–	–	–	–	–	–
Repetitive negative thinking	42	16										
Dependent stress	29	16	0.26 (0.18, 0.34)	6.82***	209.95***	0.004	0.019	13.70	13.56	72.74	$\beta = -0.99$	$t(1.36) = 5.16$
Independent stress	13	8	0.12 (0.05, 0.19)	4.22**	16.03	0.000	0.003	59.55	<1	40.45	$\beta = 0.49$	$t(1.61) = -1.08$
Negative cognitive content	43	15										
Dependent stress	33	15	0.24 (0.16, 0.31)	6.43***	311.25***	0.004	0.016	10.53	19.15	70.32	$\beta = -0.82$	$t(3.02) = -0.47$
Independent stress	10	7	0.08 (0.01, 0.16)	3.22*	5.76	0.000	0.000	98.12	<1	1.88	$\beta = 0.81$	$t(3.39) = -2.15$
Excessive standards for self	28	6										
Dependent stress	22	6	0.21 (0.09, 0.32)	4.47**	97.69***	0.004	0.009	20.61	24.37	55.01	$\beta = -0.98$	–
Independent stress	6	3	–	–	–	–	–	–	–	–	–	–
Pervasive negative affect	26	14										
Dependent stress	15	14	0.17 (0.11, 0.23)	5.85***	179.42***	0.005	0.005	11.01	43.13	45.86	$\beta = 1.00^*$	$t(11.30) = -4.33^{**}$
Independent stress	11	10	0.11 (0.06, 0.15)	5.50***	23.63**	0.000	0.002	33.58	13.66	52.76	$\beta = 0.80$	$t(6.50) = -4.07^{**}$
Dispositional other-oriented focus	37	11										
Dependent stress	29	11	0.16 (0.06, 0.26)	3.41**	151.18***	0.005	0.020	14.78	16.83	68.38	$\beta = -0.89$	$t(1.15) = 3.42$
Independent stress	8	3	–	–	–	–	–	–	–	–	–	–
Dispositional PA and upregulation	25	11										
Dependent stress	14	11	−0.02 (−0.09, 0.06)	−0.54	50.83***	0.007	0.004	21.94	50.94	27.12	$\beta = -0.47$	$t(1.37) = 0.08$
Independent stress	11	8	0.00 (−0.08, 0.09)	0.14	27.42**	0.000	0.005	35.65	<1	64.35	$\beta = 0.85$	$t(1.59) = -0.58$

Note. Summary effects are only displayed if effect sizes were available for $k \geq 5$ unique samples. For test of moderation by publication status, “unpublished” was the reference group. No effect sizes were obtained from unpublished manuscripts for excessive standards for self on dependent stress, so moderation analyses were not completed. k = number of studies or effect sizes. ES = effect size. r = bivariate correlation coefficient. Q = Q statistic for the heterogeneity of effect sizes. ER = emotion regulation. PA = positive affect. β coefficients are from meta-regression models of the association between effect sizes and their study weights, where standard errors were examined as a moderator (based on Egger’s test for funnel plot asymmetry).

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

research. A summary of sample characteristics and effect sizes for included studies are presented in [Appendix C](#), and references for all manuscripts meeting inclusion criteria are listed in [Appendix D](#). Forest plots of effect sizes for each predictor cluster on dependent and independent stress are depicted in [Appendix E](#) and [Appendix F](#), respectively. Across samples, the overall mean sample age at baseline was 20.66 years ($SD = 12.52$; range 3.55–61.00 years); the overwhelming majority (84%) of studies were conducted with children, adolescents, and adults under 22 years of age. In terms of gender composition, samples were composed of 64.38% ($SD = 20.46\%$) girls/women on average. In terms of life stress assessment methods, 39 studies (56%) used self-report checklists, 19 (27%) used interview-based methods, and 11 (16%) used hybrid approaches; one study provided insufficient information to determine the method used.

Notably, our multi-team process resulted in a more comprehensive review than those previously available. Most manuscripts included in this review ($N = 68$; approximately 79%) were produced or published after the last comprehensive systematic review of stress generation with similar inclusion criteria ([Liu & Alloy, 2010](#)). Seven of the 18 included manuscripts that were published before 2010 were not captured by [Liu and Alloy's \(2010\)](#) review. Eighty-three percent ($N = 71$) of included records were not captured by a recent review that focused specifically on anxiety-related predictors of stress generation ([Meyer & Curry, 2017](#)).

The included studies yielded 353 effect sizes from 70 unique samples ($N = 35,374$) for dependent stress and 130 effect sizes from 39 unique samples ($N = 20,696$) for independent stress. Average meta-analytic effect sizes, heterogeneity estimates, and results from tests of publication bias are presented in [Table 1](#) for each higher-order predictor cluster examined as a predictor of later life stress in $k \geq 5$ unique study samples. Results from moderation analyses are depicted in [Table 2](#) (continuous moderators, including sample demographics and follow-up length) and [Table 3](#) (categorical moderators, including stress assessment method and outcome domain). Findings are addressed in turn below.

3.2. Specific Cluster Analyses

3.2.1. Maladaptive interpersonal emotion regulation behaviors

Overall, 28 effect sizes were extracted from 10 studies for excessive reassurance seeking ($k = 18$), co-rumination ($k = 7$), and negative feedback seeking ($k = 3$) as prospective predictors of episodic life stress. The estimated average effect for maladaptive interpersonal emotion regulation behaviors on dependent stress was small, yet significant ($r = 0.17$, 95% CI: 0.13, 0.22, $p < .001$), whereas the effect on independent stress was nonsignificant ($r = 0.03$, 95% CI: -0.05 , 0.10, $p = .365$). When effects for all stress outcomes were combined in one model to test for moderation by stress outcome dependence, results indicated the average effect for dependent stress is significantly stronger than the average effect for independent stress, $\beta = 0.15$, $t(5.16) = 4.52$, $p = .006$.

The test for heterogeneity in the overall model for dependent stress suggested significant variance across all effects ($Q[df = 20] = 33.24$, $p = .032$). When we examined the distribution of variance across levels, the I^2 values for Level 1 ($I^2 = 61.96\%$), Level 2 ($I^2 = 38.04\%$), and Level 3 ($I^2 < 1\%$) indicated that <75% of the total variance in the model could be attributed to sampling variance at Level 1 and there was substantial variation between effect sizes within studies. Thus, we proceeded with our planned tests of moderation for dependent stress. As depicted in [Tables 2 and 3](#), the only moderator of effects for maladaptive interpersonal emotion regulation behaviors in the current sample of effects was stress outcome domain. Specifically, results from moderation analyses showed that the average effect for interpersonal stress ($r = 0.21$, 95% CI: 0.15, 0.27, $p < .001$) was significantly larger than the average effect for non-interpersonal stress ($r = 0.08$, 95% CI: 0.02, 0.13, $p = .022$), $t(3.80) = 4.77$, $p = .010$.

3.2.2. Disinhibition and antagonism

We extracted 30 bivariate effect sizes from 10 studies for impulsivity ($k = 19$), aggression ($k = 7$), antagonism ($k = 2$), and delinquent acts ($k = 2$). The average meta-analytic effect sizes for this domain were small and significant for both dependent ($r = 0.18$, 95% CI: 0.12, 0.24, $p < .001$) and independent ($r = 0.07$, 95% CI: 0.03, 0.11, $p = .010$) stress. Consistent with the stress generation hypothesis, a moderation analysis for dependence of stress outcome across all effects for disinhibition and antagonism suggests that the average effect for dependent stress is significantly stronger than the effect for independent stress, $\beta = 0.11$, $t(5.11) = 7.10$, $p < .001$.

In the model for effects on dependent stress, tests of heterogeneity indicated significant variance across all effects ($Q[df = 17] = 35.29$, $p = .006$), and examination of the distribution of variance across Level 1 ($I^2 = 45.13\%$), Level 2 ($I^2 = 51.21\%$), and Level 3 ($I^2 = 3.66\%$) suggested substantial variation between effects within studies. Indicators of heterogeneity supported the examination of potential moderators; however, all moderation findings were nonsignificant (all $ps > 0.05$).

3.2.3. Avoidance

Eighteen bivariate effect sizes were obtained from 9 studies for avoidance ($k = 5$), behavioral inhibition ($k = 4$), avoidance goals ($k = 2$), disengagement coping ($k = 2$), involuntary disengagement ($k = 2$), social disengagement ($k = 1$), cognitive avoidance coping ($k = 1$), and emotional discharge coping ($k = 1$). The estimate of the average effect for avoidance on dependent stress was small ($r = 0.10$, 95% CI: 0.03, 0.18, $p = .012$). Given that only 3 studies reported effects from this cluster for independent stress, we did not directly compute a summary effect for avoidance on independent stress.

Examination of estimates for heterogeneity among effects for avoidance on dependent stress suggested significant variance across all effects ($Q[df = 14] = 49.63$, $p < .001$). The distribution of variance across levels indicated substantial variation between effects within studies (Level 2 $I^2 = 28.51\%$) and across studies (Level 3 $I^2 = 43.54\%$), so we proceeded with tests of moderation. None of the candidate moderators for sample demographics, length of follow-up, or stress outcome characteristics emerged as significant moderators of the association between avoidance and dependent stress (all $ps > 0.05$).

3.2.4. Repetitive negative thinking

Forty-two effect sizes were extracted from 16 studies for rumination ($k = 31$), worry ($k = 6$) and positive affect dampening ($k = 5$). The overall estimated effects of repetitive negative thinking on dependent ($r = 0.26$, 95% CI: 0.18, 0.34, $p < .001$) and independent ($r = 0.12$, 95% CI: 0.05, 0.19, $p = .005$) stress were each small and significant. Contrary to hypotheses, the effect on dependent stress was not significantly stronger than the effect on independent stress, $\beta = 0.06$, $t(6.58) = 1.33$, $p = .228$.

Tests of heterogeneity for effects on dependent stress suggested significant variance across all effects ($Q[df = 28] = 209.95$, $p < .001$), and examination of the distribution of variance across Level 1 ($I^2 = 13.70\%$), Level 2 ($I^2 = 13.56\%$), and Level 3 ($I^2 = 72.74\%$) suggested substantial variation between effects within and across studies. Thus, we proceeded with planned tests of moderation. As summarized in [Tables 2 and 3](#), multiple significant moderators emerged for repetitive negative thinking. Specifically, effects were moderated by stress assessment method such that the average effect for checklists ($r = 0.34$, 95% CI: 0.27, 0.41) was significantly stronger than the average effect for hybrid/interview-based measures ($r = 0.12$, 95% CI: 0.03, 0.21), $t(11.05) = 4.69$, $p < .001$. Effects also differed as a function of stress domain such that the average effect for interpersonal stress ($r = 0.25$, 95% CI: 0.15, 0.34) was significantly larger than the effect for non-interpersonal stress ($r = 0.15$, 95% CI: 0.03, 0.26), $t(5.44) = 3.36$, $p = .018$. Effects of repetitive negative thinking on dependent episodic stress did not

Table 2

Summary of moderation effects for continuous moderators.

	Sample Demographic Characteristics											
	Gender (% women)			Race (% White)			Age (mean sample age)			Follow-Up Length (months)		
Predictor Cluster	<i>k</i> <i>ESs</i>	<i>k</i> studies	Moderator Test Statistic	<i>k</i> <i>ESs</i>	<i>k</i> studies	Moderator Test Statistic	<i>k</i> <i>ESs</i>	<i>k</i> studies	Moderator Test Statistic	<i>k</i> <i>ESs</i>	<i>k</i> studies	Moderator Test Statistic
Maladaptive interpersonal ER behaviors	21	10	$t(4.71) = 1.98$	20	9	$t(4.75) = -0.38$	21	10	$t(3.80) = 1.43$	23	10	$t(5.53) = -1.16$
Disinhibition and antagonism	18	10	$t(3.04) = -2.33$ $t(1.63) = 0.54$	17	9	$t(1.79) = -0.31$ $t(2.58) = 0.79$	18	10	$t(1.32) = -0.80$	21	9	$t(1.84) = 1.48$
Avoidance	15	9	$t(4.39) = -0.89$ $t(1.97) = 0.28$	15	9	$t(2.27) = 0.15$ $t(4.29) = 1.19$	15	9	$t(1.51) = 1.10$ $t(10.30) = -0.46$	20	9	$t(1.90) = -1.43$
Repetitive negative thinking	29	16	$t(1.97) = 0.28$	26	13	$t(4.29) = 1.19$	29	16	$t(7.17) = 0.18$	31	15	$t(4.44) = -1.54$
Negative cognitive content	33	15	$t(1.03) = -0.29$ $t(4.97) = 1.02$	25	10	$t(3.69) = -2.20$ $t(4.23) = -1.00$	33	15	$t(2.34) = 0.03$ $t(3.78) = -1.80$	45	15	$t(1.21) = -7.23$
Excessive standards for self	22	6	$t(4.97) = 1.02$ $t(2.89) = 0.38$	12	4	$t(3.69) = -2.20$ $t(4.23) = -1.00$	22	6	$t(2.34) = 0.03$ $t(3.78) = -1.80$	22	6	$t(1.47) = -1.07$
Pervasive negative affect	15	14	$t(2.89) = 0.38$ $t(2.60) = -0.82$	14	13	$t(4.23) = -1.00$ $t(4.50) = 1.67$	15	14	$t(3.16) = 2.04$	18	13	$t(3.45) = -0.82$
Dispositional other-oriented focus	29	11	$t(2.60) = -0.82$	25	8	$t(4.50) = 1.67$	29	11	$t(3.16) = 2.04$	31	11	$t(3.13) = -0.81$
Dispositional PA and upregulation	14	11	$t(2.60) = -0.82$	13	10	$t(4.50) = 1.67$	14	11	$t(1.27) = 0.56$	15	10	$t(1.76) = 1.34$

Note. Moderators were only tested if effect sizes were available from $k \geq 5$ unique samples. For analyses in which follow-up length was examined as a moderator, effect sizes for all available follow-up waves with stress assessment within each study were included. k = number of studies or effect sizes. ES = effect size. ER = emotion regulation. PA = positive affect.

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

Table 3

Summary of moderation effects for categorical moderators.

Predictor Cluster	Stress Outcome Characteristics							
	Stress Assessment Method				Stress Outcome Domain			
	Method	<i>k</i> ESs	<i>k</i> studies	Moderator Test / r (95% CI)	Domain	<i>k</i> ESs	<i>k</i> studies	Moderator Test / r (95% CI)
Maladaptive interpersonal ER behaviors		21	10	$t(4.09) = -0.78$		17	10	$t(3.80) = 4.77^*$
	checklist	6	4	$0.15 (-0.01, 0.31)$	interpersonal	11	10	$0.21 (0.15, 0.27)^{***}$
	interview/hybrid	15	6	$0.19 (0.13, 0.25)^{**}$	non-interpersonal	6	5	$0.08 (0.02, 0.13)^*$
Disinhibition and antagonism		18	10	$t(6.58) = 0.59$		5	3	–
	checklist	10	5	$0.20 (0.10, 0.29)^{**}$	interpersonal	3	3	–
	interview/hybrid	8	5	$0.17 (0.03, 0.29)^*$	non-interpersonal	2	2	–
Avoidance		15	9	$t(4.33) = 0.54$		7	3	–
	checklist	7	6	$0.12 (0.01, 0.22)^*$	interpersonal	4	3	–
	interview/hybrid	8	3	$0.08 (-0.18, 0.33)$	non-interpersonal	3	2	–
Repetitive negative thinking		29	16	$t(11.05) = 4.69^{***}$		20	10	$t(5.44) = 3.36^*$
	checklist	15	10	$0.34 (0.27, 0.41)^{***}$	interpersonal	12	10	$0.25 (0.15, 0.34)^{***}$
	interview/hybrid	14	6	$0.12 (0.03, 0.21)^*$	non-interpersonal	8	7	$0.15 (0.03, 0.26)^*$
Negative cognitive content		33	15	$t(10.50) = 0.41$		17	8	$t(4.15) = 1.83$
	checklist	17	9	$0.25 (0.13, 0.35)^{**}$	interpersonal	11	8	$0.21 (0.10, 0.32)^{**}$
	interview/hybrid	16	6	$0.22 (0.07, 0.35)^*$	non-interpersonal	6	5	$0.14 (0.01, 0.27)^*$
Excessive standards for self		22	6	$t(3.10) = 4.59^*$		13	5	$t(2.79) = 1.17$
	checklist	12	3	$0.28 (0.26, 0.31)^{**}$	interpersonal	7	5	$0.21 (0.10, 0.32)^{**}$
	interview/hybrid	10	3	$0.10 (-0.13, 0.31)$	non-interpersonal	6	4	$0.14 (-0.10, 0.35)$
Pervasive negative affect		14	13	$t(6.15) = -1.00$		4	3	–
	checklist	4	4	$0.12 (-0.09, 0.33)$	interpersonal	3	3	–
	interview/hybrid	10	9	$0.20 (0.12, 0.27)^{***}$	non-interpersonal	1	1	–
Dispositional other-oriented focus		29	11	$t(8.40) = 1.03$		23	10	$t(3.87) = 1.62$
	checklist	11	5	$0.21 (0.03, 0.38)^*$	interpersonal	17	10	$0.14 (0.06, 0.23)^{**}$
	interview/hybrid	18	6	$0.12 (-0.06, 0.29)$	non-interpersonal	6	5	$0.07 (-0.08, 0.21)$
Dispositional PA and upregulation		14	11	$t(5.13) = -1.69$		6	3	–
	checklist	4	4	$-0.10 (-0.33, 0.13)$	interpersonal	5	3	–
	interview/hybrid	10	7	$0.03 (-0.05, 0.11)$	non-interpersonal	1	1	–

Note. Moderators were only tested if effect sizes were available from $k \geq 5$ unique samples. For tests of moderation by stress assessment method, “interview/hybrid” was the reference category. For tests of moderation by stress outcome domain, “non-interpersonal” was the reference category. k = number of studies or effect sizes. ES = effect size. r = bivariate correlation coefficient. ER = emotion regulation. PA = positive affect.

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

significantly differ as a function of sample demographics or follow-up length (all p s > 0.05).

3.2.5. Negative cognitive content

We obtained 43 effect sizes from 15 studies for negative cognitive style ($k = 21$), maladaptive schemas (i.e., impaired autonomy, disconnection and rejection; $k = 13$), social cognitive distortions ($k = 4$), negative relational self-views ($k = 3$), and hopelessness ($k = 2$). The average meta-analytic effects for dependent ($r = 0.24$, 95% CI: 0.16, 0.31, $p < .001$) and independent ($r = 0.08$, 95% CI: 0.010, 0.165, $p = .038$) stress were small and significant. When we tested stress dependence as a moderator across all effects in this cluster, results showed the effect for dependent stress was significantly stronger than the effect for independent stress, $\beta = 0.12$, $t(4.92) = 3.68$, $p = .015$.

Tests of heterogeneity for effects on dependent stress suggested significant variance across all effects ($Q[df = 32] = 311.25$, $p < .001$) and examination of the distribution of variance across Level 1 ($I^2 = 10.53\%$), Level 2 ($I^2 = 19.15\%$), and Level 3 ($I^2 = 70.32\%$) suggested substantial variation between effects within and across studies, so we proceeded with tests of moderation. However, none of the variables we tested emerged as significant moderators of effects in this domain (all p s > 0.05).

3.2.6. Excessive standards for self

Twenty-eight effect sizes were extracted from 6 studies for perfectionism ($k = 18$) and self-criticism ($k = 10$). The estimate of the average effect for excessive standards for self on dependent stress was small ($r = 0.21$, 95% CI: 0.09, 0.32, $p = .007$). Given that only a small number of studies ($k = 3$) provided effects for independent stress, we did not directly compute a summary effect for this cluster of predictors on independent stress.

Examination of estimates for heterogeneity among effects for avoidance on dependent stress suggested significant variance across all effects ($Q[df = 21] = 97.69$, $p < .001$). The distribution of variance across levels indicated substantial variation between effects within (Level 2 $I^2 = 24.37\%$) and across (Level 3 $I^2 = 55.01\%$) studies, so we proceeded with tests of moderation. Results from these analyses suggest effects of excessive standards for self on dependent stress are moderated by stress assessment method. Specifically, the average effect for checklist-based assessments of life stress ($r = 0.28$, 95% CI: 0.26, 0.31) was significantly stronger than the average effect of interview/hybrid measures ($r = 0.10$, 95% CI: -0.13 , 0.31), $t(3.10) = 4.59$, $p = .018$. No other significant moderators emerged from analyses (p s > 0.05).

3.2.7. Pervasive negative affect

Twenty-six bivariate effect sizes were reported in 14 studies for neuroticism ($k = 17$), negative emotionality ($k = 5$), and negative affect ($k = 4$). The average meta-analytic effects for both dependent ($r = 0.17$, 95% CI: 0.11, 0.23, $p < .001$) and independent ($r = 0.11$, 95% CI: 0.06, 0.15, $p < .001$) stress were small and significant. Contrary to hypotheses, effects were not significantly different from one another when we tested stress dependence as a moderator, $\beta = 0.04$, $t(7.90) = 1.64$, $p = .140$.

Examination of estimates for heterogeneity among effects for pervasive negative affect on dependent stress suggested significant variance across all effects ($Q[df = 14] = 179.42$, $p < .001$). Substantial variation was observed between effects within studies (Level 2 $I^2 = 43.13\%$) and across studies (Level 3 $I^2 = 45.86\%$), so we continued with tests of moderation. None of the candidate moderators (sample demographics, follow-up length, or stress outcome characteristics) significantly moderated the association between pervasive negative affect and dependent stress (all p s > 0.05).

3.2.8. Dispositional other-oriented focus

We extracted 37 effect sizes from 11 studies for dependency ($k = 18$), sociotropy ($k = 6$), anxious attachment ($k = 4$), rejection sensitivity ($k = 3$), unmitigated communion ($k = 3$), interpersonal problems involving

being too dependent or caring too much about others ($k = 2$), and maladaptive interpersonal schema involving other-directedness ($k = 1$). There was a small average meta-analytic effect for these predictors on dependent stress ($r = 0.16$, 95% CI: 0.06, 0.26, $p = .007$). Insufficient studies ($k = 3$) provided effects to compute a meta-analytic effect for independent stress.

Tests of heterogeneity for effects on dependent stress suggested significant variance across all effects ($Q[df = 28] = 151.18$, $p < .001$) and examination of the distribution of variance across Level 1 ($I^2 = 14.78\%$), Level 2 ($I^2 = 16.83\%$), and Level 3 ($I^2 = 68.38\%$) suggested substantial variation between effects across studies. However, none of our candidate moderators appeared to predict differences in the magnitude of effects for dispositional other-oriented focus on dependent stress (all p s > 0.05).

3.2.9. Dispositional positive affect and upregulation

Overall, 25 effect sizes were extracted from 11 studies for extraversion ($k = 8$), positive emotionality ($k = 5$), positive affect ($k = 6$), emotion-focused savoring ($k = 2$), enhancing cognitive style ($k = 2$), and self-focused savoring ($k = 2$). Combined, the overall meta-analytic effects for these predictors on both dependent ($r = -0.02$, 95% CI: -0.09 , 0.06, $p = .603$) and independent ($r = 0.00$, 95% CI: -0.08 , 0.09, $p = .890$) stress were not significant. Moreover, the test of moderation by stress outcome dependence when effects across stress outcomes were accounted for in the same model suggests these effects are not significantly different from each other, $\beta = -0.02$, $t(4.27) = -0.62$, $p = .566$.

The test for heterogeneity in the overall model for dependent stress suggested significant variance across all effects ($Q[df = 13] = 50.83$, $p < .001$). The I^2 values for Level 1 ($I^2 = 21.94\%$), Level 2 ($I^2 = 50.94\%$), and Level 3 ($I^2 = 27.12\%$) of the variance components indicated considerable variance between effect sizes within and across studies, so we proceeded to test candidate moderators of stress generation effects. As depicted in Table 2, findings suggest that none of the candidate moderators accounted for a significant portion of heterogeneity in effects (all p s > 0.05). Upon visual inspection of the distribution of effect sizes in this domain, we noted that effects of these putative protective factors ranged widely, from -0.25 to 0.23 for dependent stress and from -0.14 to 0.18 for independent stress.

3.3. Publication bias

Appendix G displays funnel plots of effects for each of the higher-order predictor clusters on each of the two life stress outcomes. In evaluating the presence of publication bias, visual inspection of funnel plots revealed no apparent asymmetry in the distribution of effect sizes around the average meta-analytic effects for each analysis. When we followed up with tests of asymmetry based on Egger's regression, all but one of the tests yielded nonsignificant slopes (p s > 0.05), indicating no support for selective reporting of effects across most predictor-outcome pairings. In the case of pervasive negative affect predicting dependent episodic stress, results indicated asymmetry ($\beta = 1.00$, $p = .039$), but the direction of this effect was not consistent with publication bias (i.e., if asymmetry were due to bias in this case, we would expect a significant negative β value; Card, 2015). Finally, when we tested whether effects were moderated by manuscript publication status, findings generally supported the absence of publication bias, with a few exceptions. Specifically, we found that effects for maladaptive interpersonal emotion regulation behaviors on independent stress were stronger for published versus unpublished manuscripts, $t(3.62) = 3.29$, $p = .035$. However, we note that this pattern does not align with the bias we would expect to see for independent stress (i.e., authors might be incentivized to selectively exclude significant findings for independent stress, as these contradict the stress generation hypothesis). By contrast, for pervasive negative affect, effects obtained from published manuscripts were weaker than effects from unpublished manuscripts for dependent, $t(11.30) = -4.33$, $p = .001$, and independent, $t(6.50) = -4.07$, $p = .006$, stress, partially supporting possible underreporting of significant effects

of pervasive negative affect on episodic stress in the published literature. However, we interpret these results with caution given that all unpublished effect sizes for this cluster ($k = 2$) came from a single manuscript. All other moderation analyses for publication status were nonsignificant ($p > 0.05$).

4. Discussion

For over three decades, the stress generation model has shaped understanding of the intricate interface between individual characteristics and environmental risk. Although early studies focused primarily on depression and other forms of psychopathology, capturing the reciprocal relationship between symptoms and stress, psychological processes that cut across diagnostic categories also play a role in stress generation. This meta-analytic review summarizes the available evidence for cross-cutting risk and protective factors as prospective predictors of episodic life stress. The meta-analytic review employed a rigorous, publicly pre-registered, multi-team study identification approach. Our collaborative team approach allowed for unprecedented thoroughness and accuracy; further, the three-level meta-analytic design permitted us to determine not only whether risk factors predicted dependent or independent stress (as is commonly reported in individual studies), but whether effect sizes were significantly stronger for the prediction of dependent compared to independent stress, enabling a stronger test of the stress generation hypothesis. This review revealed a large and growing literature, with most manuscripts (79%) published since the last comprehensive systematic review (Liu & Alloy, 2010). Clearly, this remains an active area of inquiry, and this review provides a much-needed update on the state of the literature. Analyses yielded several notable findings.

4.1. Estimating the magnitude of stress generation effects

The present review encompasses effect sizes for a wide range of risk and protective processes, including personality factors, cognition, and interpersonal behaviors. Based on the predictors we identified with effect sizes meeting inclusion criteria, we were able to categorize risk factors into eight clusters, including maladaptive interpersonal emotion regulation behaviors (e.g., excessive reassurance seeking, co-rumination), disinhibition and antagonism (e.g., impulsivity, aggression), avoidance (e.g., behavioral inhibition, avoidance coping strategies), repetitive negative thinking (e.g., rumination, worry), negative cognitive content (e.g., negative cognitive style, maladaptive self-schemas), excessive standards for self (e.g., perfectionism, self-criticism), pervasive negative affect (e.g., neuroticism, negative emotionality), and dispositional other-oriented focus (e.g., dependency, sociotropy). We found that all eight of these risk factor clusters predicted subsequent dependent episodic life stress, with small-to-moderate meta-analytic effects ranging from $r = 0.10$ to 0.26 .³

Importantly, we also found small, yet significant, effects on *independent* stress for several risk factors, including disinhibition and antagonism, repetitive negative thinking, negative cognitive content, and pervasive negative affect. Independent stress is, by definition, fateful—it captures experiences that the person does not play a role in generating. Why, then, would individual characteristics make someone more or less likely to experience *independent stress*? We offer a few speculations. First, some risk factors may drive increased reporting of stressful events, resulting in artificially inflated rates of independent stress among those with higher levels of vulnerability (Espejo et al., 2011; Harkness & Monroe, 2016). It is also possible that these stressors are truly independent, but that people with higher levels of certain risk

factors are more likely than others to live in contexts in which independent stressors occur more frequently (Harkness & Washburn, 2016); thus, small-yet-significant meta-analytic effects could reflect the continuity of stressful contexts over time. It is also worth noting that, although events are dichotomized as independent or dependent based on their predominant causes, individual characteristics may play a distal causal role or may influence contextual factors. For example, if a friend gets arrested, it is likely an independent event, but certain individuals may be more likely to self-select into peer groups with rule-breaking peers. If a friend dies of cancer or moves away, clearly this is independent, but it may be rated as more contextually impactful compared to ratings for less vulnerable individuals if the friend is the sole source of social support due to difficulties with creating and maintaining personal relationships. Indeed, truly fateful events—stressors that are essentially randomly assigned, completely isolated from personal characteristics and divorced from other environmental events, such as the proverbial lightning strike—are likely rare.

That said, although some effect sizes emerged as significant in our highly powered meta-analysis, even the strongest effects for prediction of independent stress were quite small ($r_s = 0.03$ to 0.12). Further, results from models that directly compared the relative magnitude of meta-analytic effect sizes for dependent and independent stress suggested that effects are significantly stronger for dependent stress across the board, with the exception of pervasive negative affect and repetitive negative thinking. These findings underscore that testing effects on dependent stress alone, without testing effects on independent stress as a comparison, provides an incomplete test of the stress generation model. Unfortunately, this stands in contrast to the state of the literature. Across the studies included in this review, we obtained effect sizes for dependent stress from 100% of them, but we were only able to extract effect sizes for independent stress from about half (56%) of these studies. Indeed, the relative dearth of reported findings for independent stress precluded our ability to compute average meta-analytic effects on this outcome for three of our predictor clusters.

4.2. Specific cluster findings

Most of the specific clusters of risk or protective factors predicted stress generation in the expected direction. That is to say, there was not one overarching category of mechanistic factors that appear to uniquely drive stress generation effects. We interpret this to mean that stress generation has multifarious contributors; there are many pathways that leave some people more at risk than others for generating or selecting into stressful environments. This is not particularly surprising, as dependent stressful events are themselves remarkably heterogeneous, encompassing a wide diversity of human experiences that are undoubtedly influenced by equally varied individual factors.

An unexpected finding was a lack of support for stress generation effects via the repetitive negative thinking (e.g., rumination, worry, positive affect dampening) and pervasive negative affect (e.g., neuroticism, negative emotionality) clusters. Although these clusters significantly predicted dependent stress, effect sizes did not differ from those predicting independent stress, leaving us unable to conclude that these factors are uniquely predictive of stress generation, versus predictive of elevated reports of stress in general. Perhaps because the stress generation literature originated with a focus on depression (Hammen, 1991), there has long been an assumption among researchers that persistently elevated negative affect at least in part drive stress generation effects (e.g., Hammen, 2006; Meyer & Curry, 2017). Notably, the pervasive negative affect cluster is distinct from depression; while the former encompasses measures of neuroticism, negative emotionality, and self-reported experiences of negative emotion in everyday life, the latter is a heterogeneous construct that encompasses a broad range of symptoms and experiences beyond negative affect (e.g., anhedonia, irritability) that may contribute to the larger effect size observed for depression. Negative affect also has adaptive functions in certain contexts (e.g., by

³ For context, our companion meta-analysis on psychopathology and stress generation (Rnic et al., 2023) found that depression, the most commonly studied predictor of stress generation, had a small-to-moderate and significant effect on dependent stress, $r = 0.24$, 95% CI: 0.21, 0.27, $p < .001$.

orienting towards threat, enabling problem solving, and facilitating interpersonal effectiveness; Coifman et al., 2016; Forgas, 2013), and it is possible that these protective elements may somewhat offset challenges that pervasive negative affect may introduce.

In addition to risk factors, an important goal of this meta-analysis was to explore *protective* factors for stress generation. Notably, the breadth of existing literature supported the creation of only one cluster of protective factors: dispositional positive affect and upregulation (e.g., extraversion, enhancing cognitive style, positive affect savoring). Findings suggest that this cluster was not protective against stress generation; meta-analytic effects were negligible for both dependent and independent stress ($r_s = -0.02$ and 0.00 , respectively), and estimates were not significantly different from one another. Alongside findings discussed above for pervasive negative affect, these results undermine the notion that trait-level affective experiences play a fundamental role in stress generation. Alternatively, just as negative affect can be both adaptive and corrosive, it may be that trait-level positive affect could have both benefits and drawbacks. For instance, it may be that high levels of positive affect increase the degree to which people engage with their social environment, which represents a double-edged sword in that it facilitates social connection while also increasing the likelihood that one will experience interpersonal stress (Hamilton et al., 2017).

The relatively limited depth of the literature on specific protective processes for stress generation is noteworthy, as it is almost certainly the case that some individual characteristics and behavioral styles function to reduce risk and buffer against the generation of life stress. However, the investigation of factors that are potentially protective against stress generation is an emerging area of inquiry. As a result, there were not enough eligible studies for us to examine other putative protective factor clusters, such as adaptive emotion regulation and coping, competent interpersonal behaviors, or positive cognitive content. Furthermore, a number of specific variables that have already been shown to attenuate the effects of stress have not yet been examined with reference to stress generation. These represent promising directions for future research on resilience to stress generation, and include adaptive emotion regulation strategies such as reappraisal and reflection (Rnic et al., 2022), emotion regulation flexibility (Battaglini et al., 2022), positive schemas (Lumley & McArthur, 2016), and optimism (Hamilton et al., 2017), among others. Moreover, social support, a documented protective factor for stress generation (Auerbach et al., 2011), was excluded from the current review because it is not an individual characteristic or behavioral style. Rather, social support represents part of individuals' environmental context (Bronfenbrenner, 1979) and is therefore difficult to disentangle from experiences of dependent stress. In sum, broadening and deepening our understanding of protective factors beyond dispositional positive affect is a critical avenue for future research.

4.3. Examination of moderators

4.3.1. Stress assessment method

We found mixed support for our hypothesis that stress generation effects would be significantly stronger when life stress was assessed using checklist-based measures compared to interview-based methods. Specifically, two predictor clusters—repetitive negative thinking and excessive standards for self—showed significantly stronger effects on dependent stress when stress was assessed using self-report checklists. These differences were pronounced: effect sizes nearly tripled in size for self-report measures compared to interview or hybrid approaches. Bias in self-report measures of stress have been documented for decades (Harkness & Monroe, 2016; Monroe, 2008), with some researchers describing self-report versus interview-based stress assessments as tapping essentially different constructs with distinct implications for depression and health risk (McQuaid et al., 2000; Monroe, 2008). The current results suggest specific psychological processes may be associated with bias. For example, people with excessively high standards for themselves may be more prone to self-label events as failures or conflicts

(Simons et al., 1993). Repetitive negative thinking leads to negative bias in autobiographical memory (Lyubomirsky et al., 1998), which may affect responses on measures that rely entirely on self-report to quantify the frequency or severity of negative life events. Taken together, these results underscore the importance of higher quality stress measures in this line of research. Unfortunately, our review also reveals that reliance on self-report stress measures is widespread. Most of the included studies (56%) used self-report checklists. Although this pattern is unsurprising given the relative ease with which checklists can be administered compared to more resource-intensive interview-based methods, our results illustrate the bias they introduce into the literature.

4.3.2. Stress outcome domain

A second set of analyses for moderation by stress outcome characteristics provided some support for our hypothesis that stress generation effects would be stronger for interpersonal stress compared to non-interpersonal dependent stress. Specifically, we found that effects of maladaptive interpersonal emotion regulation behaviors and repetitive negative thinking were stronger for interpersonal stress compared to non-interpersonal stress. In the case of maladaptive interpersonal emotion regulation behaviors, (e.g., excessive reassurance seeking, co-rumination, negative feedback seeking), the explanation for why effects are significantly stronger for interpersonal stress is straightforward. This cluster is composed of support-seeking behaviors that are employed in interpersonal contexts and that could theoretically play a direct, proximal role in eroding the quality of interpersonal relationships and generating conflict. For effects of repetitive negative thinking, the story is perhaps less intuitive, as the psychological processes in this cluster (e.g., rumination, worry, positive affect dampening) are not explicitly characterized by their connection to interpersonal functioning and may not be directly visible to others. However, results of the current study dovetail with findings from prior work connecting rumination to poorer interpersonal problem solving skills (Lyubomirsky & Nolen-Hoeksema, 1995; Nezu, Nezu, Damico, & Gerber, 2023). We also note that, although not all meta-analytic differences rose to the level of significance in this sample of effects, the relative magnitude of effect sizes was consistent with hypotheses such that effects were larger for interpersonal stress compared to non-interpersonal stress across the board. This trend is consistent with assertions in prior work regarding the centrality of disrupted interpersonal functioning and interpersonal stress to the stress generation phenomenon (e.g., Hammen, 2006). However, present findings suggest that some constructs show a higher degree of specificity than others in the prediction of interpersonal stress.

4.3.3. Additional moderators

Additional moderation analyses revealed no significant differences in meta-analytic effects for dependent stress as a function of gender (percentage of sample identifying as girls/women), race (percentage of sample reporting their race as White), mean sample age, or length of follow-up (i.e., months elapsed between assessment of predictors and assessment of life stress).

Contrary to hypotheses, results suggest that stress generation effects are comparable regardless of sample gender composition. These findings do not support the commonly held assumption that stress generation effects are significantly stronger among women and girls compared to men and boys (e.g., Liu & Alloy, 2010). Of note, because a very small minority of studies meeting inclusion criteria presented effect sizes stratified by gender, the present study relied upon sample-wide estimates (i.e., percentage of sample identifying as girls/women as a moderator of effect size estimates that collapsed across all genders within a sample) in conducting these analyses. This provides a coarser and more indirect test of moderation by gender that may have potentially obscured important differences between subsamples of girls/women and boys/men. That is, although this study provides the most comprehensive test of systematic differences in stress generation by gender across the literature to date, the nature of the available data

precluded a more direct test of gender as a categorical moderator of stress generation. Null findings ought to be interpreted in light of this limitation. While true moderation effects of gender may exist (and future research and broad data sharing efforts may enable more fine-grained analyses that examine differences among subgroups), current results indicate that gender moderation effects may not be as strong as researchers have previously concluded.

Although we proposed no specific hypotheses regarding effects of sample race or mean sample age on the magnitude of stress generation effects, and although null findings for these descriptive moderators may be interpreted to suggest that there are no systematic differences as a function of sample demographics, these results should be interpreted in light of certain observations about the available literature. For example, we found a striking lack of variation in age across studies, with just 11 of the 70 included studies reporting a mean sample age of 22 years or older at baseline, signifying that most research has been conducted with children, adolescents, and young adults. This may have contributed to ceiling effects in our analyses, and generally suggests that stress generation remains an understudied phenomenon for samples across the lifespan. It will be important for future studies in this area to enroll participants across a broader range of ages. Because of the lack of findings from adult samples, the current data were inadequate to test more nuanced hypotheses about shifts in stress generation processes from a lifespan developmental perspective. Insights from research with participants across developmental epochs will be crucial in driving further theory and informing the creation of developmentally informed interventions to address stress generation.

Finally, results of the present analyses suggest length of follow-up is not a significant moderator of stress generation effects. Null findings could suggest that the predictors captured in the present meta-analysis generally represent more enduring traits or behavioral tendencies that contribute to stress generation in an ongoing, rather than temporally limited, manner. However, it is also possible that follow-up length effects exist for at least some clusters, but that our ability to detect these effects is hampered by the fact that it is uncommon for researchers to report effects from multiple follow-up lags *within* a study. Effect sizes from multi-lag studies provide the best information for directly testing this question while holding participant and study method factors constant. As research continues to accumulate in this area and more multi-lag studies are published, it could be fruitful to revisit the question of whether the strength of stress generation effects vary as a function of time.

4.4. Extending research on psychopathology and stress generation

The present results complement and extend findings from our companion meta-analysis, which synthesized research on mental disorders and symptoms as predictors and outcomes of stress generation. For instance, findings from Rnic et al. (2023) suggest that stress generation is a transdiagnostic phenomenon, with significantly larger effects emerging for dependent compared to independent stress across internalizing and externalizing disorders and symptoms. Taken together, results across both reviews underscore the notion that a broad range of psychological processes appear to be involved in the self-generation of life stress. Further, results of moderation analyses in Rnic et al. (2023) suggest that different patterns of stress generation emerge across forms of psychopathology. For example, we found that effects of internalizing disorders on dependent stress were significantly stronger when life stress was captured using checklist-based measures compared to interview-based measures. The current results add texture to these findings by suggesting specific risk factors (e.g., repetitive negative thinking and excessive standards for self) are associated with stress measurement bias. In Rnic et al. (2023), we also showed that effects of internalizing disorders were stronger for interpersonal stress compared to non-interpersonal stress. The present findings take this a step further by highlighting specific constructs that may be particularly potent

predictors of interpersonal stress generation (e.g., maladaptive interpersonal emotion regulation behaviors, repetitive negative thinking).

4.5. Limitations and future directions

Despite the many strengths of this review, findings ought to be considered in light of some important study limitations. First, as noted in our Method, we were only able to incorporate effect sizes for predictors with sufficient coverage in the literature (i.e., constructs for which effect sizes were available from five or more unique study samples) in the meta-analytic portion of this review. We purposefully assigned predictors to higher-order clusters based on careful consideration of how they hang together conceptually. However, a consequence of this process is that some risk and protective factors were excluded from the quantitative synthesis solely because they remain under-examined in the available literature (e.g., potential risk factors like emotion suppression or potential protective factors such as mindfulness). We include a full accounting of these effect sizes in Appendix C, and we encourage researchers to continue investigating the full range of risk and protective processes captured in the present review. Second, power was relatively limited for fine-grained examination of moderators due to relatively low numbers of studies for certain predictor clusters. Third, it is important to note that risk factors here are very likely interrelated and overlapping. The present large-scale review specifically synthesizes bivariate effect sizes of risk and protective factors for subsequent episodic life stress. Although the literature on stress generation is vast and continually growing, the current state of the literature is such that it is not yet feasible to obtain the data required to model more complex meta-analytic associations (e.g., using multivariate meta-analysis) that account for the overlapping nature of the risk and protective factors included in this review and that highlight their unique contributions to stress generation. Nevertheless, advancing understanding of the complex interplay among processes that confer risk for and protect against stress generation remains an important question to be addressed as this literature develops. For now, this may be a task best met by large-scale data-sharing efforts rather than by meta-analysis. Fourth, this meta-analytic review cannot directly test theoretical questions regarding the temporal relationships among risk or protective factors in the prediction of stress generation (e.g., by evaluating whether interpersonal behaviors serve as more “proximal” predictors that mediate the association between cognitive or personality factors and dependent episodic stressors; Liu, 2013). Further research that directly evaluates the complex interplay of multiple predictor processes in a temporally sensitive manner is warranted, and we hope this meta-analytic review will prove useful to those who conduct these types of studies by identifying and quantifying the impact of specific constructs that may be part of the path to stress generation.

4.6. Clinical implications

The assumption upon which the stress generation hypothesis rests—that people play an active role in constructing their relationships and environments—can cut two ways. That is, assuming individuals actively contribute to the generation of stressors in their lives, they can also play an active role in changing the cognitive and behavioral patterns that contribute to the generation of stressors. A unifying feature of the individual predictors reviewed in this meta-analysis is that they are all, at least in theory, modifiable. As such, they may be useful targets for intervention to prevent or reduce the occurrence of stress generation. Indeed, targeting cross-cutting risk and protective factors may empower people to shape their own worlds, reduce their life stress, and improve their well-being.

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Angela C. Santee: Conceptualization, Methodology, Investigation, Formal analysis, Writing – original draft. **Katerina Rnic:** Conceptualization, Methodology, Investigation, Formal analysis, Writing – review & editing. **Katharine K. Chang:** Investigation, Visualization, Writing – review & editing. **Rachel X. Chen:** Investigation, Writing – review & editing. **Jennifer-Ashley Hoffmeister:** Investigation. **Hallie Liu:** Investigation. **Joelle LeMoult:** Conceptualization, Methodology,

Writing – review & editing, Supervision. **David J.A. Dozois:** Conceptualization, Methodology, Writing – review & editing, Supervision. **Lisa R. Starr:** Conceptualization, Methodology, Writing – review & editing, Supervision.

Declaration of Competing Interest

None

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Appendix A. Search strategy details for independent research teams

	Team USA Search Strategy	Team Canada Search Strategy
Databases	PsycInfo PubMed Web of Science	PsycInfo PubMed
Search terms	("stress generation") OR ("life event" OR "life events" OR stressor OR "episodic stress" OR "stressful event" OR "stressful events" OR "negative event" OR "negative events") AND (generate OR generation OR dependent)) NOT oxidative ^a	"stress generation" OR "generation of stress*" OR "generated stress*" OR "dependent stress*" OR "dependent life event*" OR "dependent event"
Added parameters	For all databases, results were restricted to manuscripts in the English language. For PsycInfo and PubMed, specified human subjects. For Web of Science, specified research areas as psychology, psychiatry, and neuroscience neurology	Search limited to titles and abstracts in PsychInfo and PubMed for articles published after Hammen (1991) was published in November 1991

^a The phrase “stress generation” is commonly used in research for fields well outside the scope of psychology, most notably materials science and engineering. The term “oxidative” was identified in initial searches as a term that frequently co-occurs with “stress generation” and can be used to efficiently weed out false positives in database searches where results are not restricted to the field of psychology (i.e., Web of Science, PubMed).

Appendix B. Decision rules to select effect sizes and reduce sources of dependency

1. When the same effect with the same sample was reported in multiple manuscripts, the effect size for the more representative portion of the sample (i.e., with the largest *N*) was selected.
2. When effects were reported separately for subgroups that are relevant for our planned moderation analyses (e.g., effects stratified by gender, race), the stratified effects were retained.
3. When the same effect with the same sample size was reported in unpublished and published manuscripts (e.g., for an author’s dissertation and the resulting publication), the effect from the published manuscript was selected.
4. When multiple effects were available for the same predictor-outcome pairing across multiple waves, the effect for the shortest lag between the assessment of the predictor at Time 1 and the assessment of episodic stress at a subsequent wave was selected. As an exception to this rule, effect sizes for predictors on life stress at all available waves of follow-up assessment were included in the analysis of follow-up length as a moderator of stress generation effects.
5. When multiple effects were presented for nested measures of a predictor (e.g., total score for negative cognitive style, subscale scores for academic-, social-, and appearance-related negative cognitive styles), the effect for the higher-order construct was selected (e.g., total score for negative cognitive style).
6. When multiple effects were presented for overlapping measures of a predictor (e.g., one predictor construct measured using three different self-report measures), the effect for the measure with the highest level of internal consistency for that sample or, if measures of internal consistency are unavailable or equal across measures, the effect for the measure that was most commonly used to assess that construct across included studies was selected. This rule was applied to facilitate interpretability and comparability across studies.
7. When multiple effects were presented for an outcome domain (e.g., separate effects reported for achievement- and appearance-related stress, which are each specific subdomains of non-interpersonal stress), we selected the effect for the subdomain that is more commonly represented across the literature (e.g., achievement-related stress would be selected because it is more common for studies to use academic or achievement stress to index non-interpersonal stress; by contrast, appearance-related stress is scarcely the focus of non-interpersonal stress measures).

Appendix C. Summary of included studies

		Baseline Sample Information					ES by Stress Outcome											
		Age (years)																
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES N	Co	In	NI	Co	In	NI
1	Shih et al. (2009)	140	6–14	9.84 (2.37)	50.71	84.3	H	excessive reassurance seeking	ERB	R	12 mos	140	–	0.210	0.030	0.090	–	–
								self-criticism	ESS	R	12 mos	140	–	0.170	0.260	0.160	–	–
								negative cognitive style	NCC	R	12 mos	140	–	0.190	0.150	0.110	–	–
								dependency	OOF	R	12 mos	140	–	–0.100	–0.250	0.080	–	–
2	Allen et al., (2020)	355	18–50	28.3 (7.62)	77	75.49	C	antagonism	DIA	R	12 mos	135	0.250	–	–	0.000	–	–
								antagonism	DIA	R	24 mos	96	0.260	–	–	0.060	–	–
								antagonism	DIA	R	36 mos	87	0.300	–	–	0.080	–	–
								impulsivity	DIA	R	12 mos	135	0.160	–	–	0.060	–	–
								impulsivity	DIA	R	24 mos	96	0.180	–	–	0.040	–	–
								impulsivity	DIA	R	36 mos	87	0.190	–	–	–0.070	–	–
3	Hamilton et al. (2013)	301	12–13	12.82 (0.61)	56	48	H	negative cognitive style	NCC	R	9 mos	301	–	–	–	0.050	–	–
								rumination	RNT	R	9 mos	301	–	–	–	0.070	–	–
	Hamilton et al. (2015)	382	12–13	12.87 (0.61)	53	49	H	negative cognitive style	NCC	R	7 mos	366	–	0.230	0.040	–	0.070	–
								rumination	RNT	R	7 mos	366	–	0.210	0.050	–	0.100	–
	Mac et al. (2018)	173	12–13	12.5 ^b	56	47	H	hopelessness	NCC	R	7 mos	173	–	–	–	–0.030	–	–
	Stange et al. (2014)	256	12–13	12.32 (0.61)	54	49	H	distraction & problem-solving	–	P	9 mos	118 ^d	–	–0.110	–0.040	–	0.020	–
								distraction & problem-solving	–	P	9 mos	138 ^c	–	–0.210	–0.190	–	–0.110	–
4	Flynn et al. (2010)	122	–	19.78 (3.54)	61.48	63	H	rumination	RNT	R	9 mos	122	–	0.180	0.250	–	0.080	0.110
	Safford et al. (2007)	157	–	19.31 (2.19)	66.88	61.15	H	negative cognitive style	NCC	R	6 mos	157	–	–	–	0.016	–	–
								negative cognitive style	NCC	R	6 mos	105 ^c	0.229	–	–	–	–	–
								negative cognitive style	NCC	R	6 mos	52 ¹	0.021	–	–	–	–	–
5	Hamilton (2018) ^a	105	18–22	19.84 (1.17)	76	71	C	resting state stress regulation	–	R	2 wks	105	–	0.050	–	–0.050	–	–
								stress reactivity	–	R	2 wks	105	–	–0.180	–	0.130	–	–
6	Bart et al. (2019)	347	14–19	18.43 (1.4)	62.8	56.8	H	behavioral activation	–	R	11 mos	347	0.158	–	–	0.079	–	–
								impulsivity	DIA	R	11 mos	347	0.190	–	–	0.090	–	–
	Hamilton et al. (2017)	304	14–19	18.2 (1.39)	68	58	H	emotion-focused savoring	DPA	P	6 mos	304	–	0.230	–	–	0.180	–
								positive affect	DPA	P	6 mos	304	–	–0.020	–	–	0.070	–
								self-focused savoring	DPA	P	6 mos	304	–	0.070	–	–	0.130	–
								negative affect	PNA	R	6 mos	304	–	0.240	–	–	0.120	–
								positive affect dampening	RNT	R	6 mos	304	–	0.000	–	–	0.070	–
								rumination	RNT	R	6 mos	304	–	0.190	–	–	0.020	–
7	Molz et al. (2013)	200	–	19.65 (1.55)	66.87 ^b	68.9	H	aggression	DIA	R	varied	200	0.173	–	–	–	–	–
								impulsivity	DIA	R	varied	200	0.388	–	–	–	–	–
8	Barker, 2020	645	17–41	18.7	59.1	89.9	C	agreeableness	–	P	varied	572	–0.080	–	–	–	–	–
								conscientiousness	–	P	varied	572	–0.130	–	–	–	–	–
								mastery	–	P	varied	572	–0.120	–	–	–	–	–
								openness	–	P	varied	572	0.030	–	–	–	–	–
								avoidance coping	AVD	R	varied	572	0.120	–	–	–	–	–
								extraversion	DPA	P	varied	572	0.060	–	–	–	–	–
								neuroticism	PNA	R	varied	572	0.100	–	–	–	–	–
9	Holahan et al. (2005)	1211	55–65	61 (3.2)	41	92	C	cognitive avoidance coping	AVD	R	48 mos	1211	0.150	–	–	–	–	–
								emotional discharge coping	AVD	R	48 mos	1211	0.210	–	–	–	–	–
10	Calvete et al. (2013)	1187	13–17	13.42 (1.3)	45.91	–	C	maladaptive schema - disconnection and rejection	NCC	R	6 mos	1187	0.370	–	–	–	–	–
								maladaptive schema - impaired autonomy	NCC	R	6 mos	1187	0.320	–	–	–	–	–
								negative cognitive style	NCC	R	6 mos	1187	0.240	–	–	–	–	–
	Calvete et al. (2015)	1000	–	13.42 (1.3)	45.5	–	C	rumination	RNT	R	6 mos	1000	0.230	–	–	–	–	–
								rumination	RNT	R	12 mos	1000	0.110	–	–	–	–	–
11	Calvete (2011)	853	14–17	15.86 (0.96)	52.29	–	C	negative cognitive style	NCC	R	6 mos	853	–	0.170	–	–	–	–
								sociotropy	OOF	R	6 mos	853	–	0.100	–	–	–	–
12	Alba & Calvete (2019)	584	14–19	15.99 (1.1)	44.86	–	C	maladaptive schema - disconnection and rejection	NCC	R	4 mos	584	0.520	–	–	–	–	–
								maladaptive schema - disconnection and rejection	NCC	R	8 mos	584	0.490	–	–	–	–	–
								maladaptive schema - disconnection and rejection	NCC	R	12 mos	584	0.460	–	–	–	–	–

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Baseline Sample Information										ES by Stress Outcome									
		Age (years)												Dependent			Independent		
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES N	Co	In	NI	Co	In	NI	
								maladaptive schema - impaired autonomy	NCC	R	4 mos	584	0.440	–	–	–	–	–	–
								maladaptive schema - impaired autonomy	NCC	R	8 mos	584	0.410	–	–	–	–	–	–
								maladaptive schema - impaired autonomy	NCC	R	12 mos	584	0.390	–	–	–	–	–	–
								maladaptive schema - other-directedness	OOF	R	4 mos	584	0.440	–	–	–	–	–	–
								maladaptive schema - other-directedness	OOF	R	8 mos	584	0.430	–	–	–	–	–	–
								maladaptive schema - other-directedness	OOF	R	12 mos	584	0.420	–	–	–	–	–	–
								rumination	RNT	R	4 mos	584	0.470	–	–	–	–	–	–
								rumination	RNT	R	8 mos	584	0.460	–	–	–	–	–	–
								rumination	RNT	R	12 mos	584	0.420	–	–	–	–	–	–
								mindfulness	–	P	24 mos	1190	–0.150	–	–	–	–	–	–
13	Calvete, et al. (2019)	1190	13–18	15.16 (0.95)	49.33	–	C	maladaptive schema - disconnection and rejection	NCC	R	24 mos	1190	0.130	–	–	–	–	–	–
14	Clarke et al. (2018)	9173	–	56.4	62.5	–	C	neuroticism	PNA	R	6.6 yrs	7599	–0.031	–	–	0.096	–	–	–
15	Sahl et al. (2009)	127	–	19 ^b	67	80	C	interpersonal competence - conflict management	–	P	1 wk	127	0.100	–	–	–	–	–	0.090
								interpersonal competence - disclosure	–	P	1 wk	127	0.100	–	–	–	–	–	0.090
								interpersonal competence - emotional support	–	P	1 wk	127	0.130	–	–	–	–	–	0.060
								interpersonal competence - initiation	–	P	1 wk	127	0.100	–	–	–	–	–	0.110
								interpersonal competence - negative assertion	–	P	1 wk	127	–0.050	–	–	–	–	–	–0.020
								aggression	DIA	R	1 wk	127	0.130	–	–	–	–	–	0.050
								perfectionism - concern over mistakes	ESS	R	12 mos	271 ^d	0.160	–	–	–	–	–	–
								perfectionism - concern over mistakes	ESS	R	12 mos	281 ^c	0.120	–	–	–	–	–	–
								perfectionism - doubts about actions	ESS	R	12 mos	271 ^d	0.290	–	–	–	–	–	–
								perfectionism - doubts about actions	ESS	R	12 mos	281 ^c	0.320	–	–	–	–	–	–
								self-criticism	ESS	R	12 mos	271 ^d	0.340	–	–	–	–	–	–
self-criticism	ESS	R	12 mos	281 ^c	0.370	–	–	–	–	–	–								
socially-prescribed perfectionism	ESS	R	12 mos	271 ^d	0.360	–	–	–	–	–	–								
socially-prescribed perfectionism	ESS	R	12 mos	281 ^c	0.310	–	–	–	–	–	–								
17	Cummings et al. (2010)	310	–	19.67 (1.24)	77.7	86.5	C	interpersonal competence	–	P	3 wks	310	–0.113	–	–	–0.077	–	–	–
18	Driscoll (2012) ^a	98	18–67	33.35 (11.04)	80.6	0	C	intercultural competence	–	P	6 mos	98	0.070	–	–	0.010	–	–	–
19	Dudeck (2008) ^a	183	–	13 (0.72)	52.46	–	C	responsive caring	–	P	12 mos	183	0.150	–	–	0.220	–	–	–
								responsive caring	–	P	24 mos	183	0.160	–	–	0.220	–	–	–
								delinquent acts	DIA	R	12 mos	183	0.290	–	–	0.070	–	–	–
								delinquent acts	DIA	R	24 mos	183	0.360	–	–	0.020	–	–	–
								positive affect	DPA	P	12 mos	183	–0.100	–	–	0.020	–	–	–
								positive affect	DPA	P	24 mos	183	–0.010	–	–	–0.020	–	–	–
								personal standards perfectionism	ESS	R	48 mos	145	–	0.220	0.020	–	–0.010	0.070	
								self-critical perfectionism	ESS	R	48 mos	145	–	0.240	–0.040	–	–0.050	0.020	
								insecure attachment - avoidant	–	R	4 wks	104	–	0.160	–	–	–	–	–
								excessive reassurance seeking	ERB	R	4 wks	104	–	0.300	–	–	–	–	–
								dependency - exploitable	OOF	R	4 wks	104	–	0.150	–	–	–	–	–
dependency - love	OOF	R	4 wks	104	–	–0.080	–	–	–	–	–								
insecure attachment - anxious	OOF	R	4 wks	104	–	0.320	–	–	–	–	–								
22	Elliot et al. (2011)	260	18–39	19.54	65.38	76.54	C	avoidance goals	AVD	R	15 wks	260	0.180	–	–	–	–	–	
23	Elliot et al. (2011)	159	17–40	19.95	64.78	69.81	C	avoidance goals	AVD	R	15 wks	159	0.230	–	–	–	–	–	
24	Freedman (2001) ^a	125	20–81	48	57	96	I	perceived control	–	P	18 mos	125	–0.020	–	–	0.020	–	–	–
25	Starr et al. (2012)	381	15	15	60.89	95	I	secure attachment	–	P	60 mos	381	–0.090	–0.120	–	–0.040	–	–	–
	Starr et al. (2013)	354	15	15	61.3	100	I	secure attachment	–	P	60 mos	354	–	–	–	–	–	–0.050	
26	Daley et al. (1997)	155	16–19	18.26 (0.48)	100	46	I	autonomy	–	R	12 mos	134	0.280	0.250	–	–	–	–	–
								sociotropy	OOF	R	12 mos	134	0.180	0.200	–	–	–	–	–
								positive emotionality	DPA	P	5 mos	345	–0.170	–	–	–0.140	–	–	–
27	Wetter & Hankin (2009)	350	11–17	14.5 (1.4)	57	53	C	negative emotionality	PNA	R	5 mos	345	0.270	–	–	0.210	–	–	–
28	La Rocque et al. (2016)	301	–	18.26 (2.03)	86	70	I	perfectionism - self-oriented	ESS	R	4 mos	301	–	0.015	–0.039	–	–	–	–

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Baseline Sample Information										ES by Stress Outcome									
		Age (years)												Dependent			Independent		
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES N	Co	In	NI	Co	In	NI	
29	Hochwalder & Jacek (2015)	1012	30–64	46.9 (8.9)	100	–	C	perfectionism - socially prescribed	ESS	R	4 mos	301	–	0.149	0.057	–	–	–	
30	Broeren et al. (2014)	202	3–4	4.02 (0.35)	50	18.9	C	sense of coherence	–	P	15 mos	664	–0.191	–0.133	–0.140	–0.011	–	–	
31	Kendler et al. (2002)	1942	–	35.8 (8.2)	100	100	–	behavioral inhibition	AVD	R	24 mos	178	0.009	–	–	–0.489	–	–	
32	Kercher & Rapee (2009)	756	–	12.8 (0.39)	49.4	86	C	self-esteem	–	R	36 mos	1942	0.110	–	–	0.090	–	–	
								neuroticism	PNA	R	36 mos	1942	0.120	–	–	0.120	–	–	
								negative cognitive style	NCC	R	6 mos	756	0.375	–	–	–	–	–	
								rumination	RNT	R	6 mos	756	0.469	–	–	–	–	–	
33	Kercher et al. (2009)	896	10–13	12.3 (0.4)	100	80	C	neuroticism	PNA	R	12 mos	896	0.186	–	–	0.136	–	–	
34	Kindt et al. (2015)	1343	–	13.4 (0.77)	52.3	–	C	negative cognitive style	NCC	R	6 mos	1152	–	0.250	–	–	–	–	
35	Kleiman (2014) ^a	193	17–44	20.66 (3.88)	70	45	H	negative cognitive style	NCC	R	12 mos	974	–	0.260	–	–	–	–	
								negative cognitive style	NCC	R	18 mos	1000	–	0.190	–	–	–	–	
								negative cognitive style	NCC	R	6 wks	193	0.260	–	–	0.130	–	–	
								neuroticism	PNA	R	9 mos	528	0.170	–	–	–	–	–	
36	Goldstein et al. (2020)	550	13–15	14.38 (0.63)	100	80.5	I	neuroticism	PNA	R	18 mos	513	0.140	–	–	–	–		
	Mackin et al. (2019)	467	13–15	14.39 (0.63)	100	88	I	neuroticism	PNA	R	27 mos	496	0.190	–	–	–	–	–	
								neuroticism	PNA	R	36 mos	491	0.100	–	–	–	–	–	
								reward processing	–	P	18 mos	467	–0.080	–	–	0.020	–	–	
								behavioral inhibition	AVD	R	9 yrs	392	–0.060	–	–	0.010	–	–	
37	Mumper et al. (2020)	392	3	3.55 (0.26)	46.94	94.9	I	dependency - connectedness	OOF	R	12 mos	213	–	0.030	0.080	–	0.120	–0.070	
38	Little (2001) ^a	240	–	11.86 (0.57)	54.2	82	I	dependency - neediness	OOF	R	12 mos	213	–	0.070	0.100	–	0.110	0.000	
39	Hernandez et al. (2016)	185	–	19.65 (1.48)	75.1	55.7	H	excessive reassurance seeking	ERB	R	4 mos	185	0.260	0.290	0.110	–	–	–	
								negative feedback seeking	ERB	R	4 mos	185	0.200	0.200	0.130	–	–	–	
								rejection sensitivity	OOF	R	4 mos	185	0.400	0.400	0.180	–	–	–	
								excessive reassurance seeking	ERB	R	4 mos	185	–	–	–	–0.044	–	–	
	Liu (2012) ^a	185	–	19.65 (1.49)	75.68	56.22	H	negative cognitive style	NCC	R	4 mos	185	0.491	0.467	0.324	0.147	–	–	
								self-perceived academic competence	–	P	4 mos	185	–0.218	–0.173	–0.287	–0.067	–	–	
								self-perceived appearance competence	–	P	4 mos	185	–0.003	–0.035	–0.064	–0.037	–	–	
								self-perceived social competence	–	P	4 mos	185	–0.011	–0.029	–0.075	–0.056	–	–	
40	Shiner et al. (2017)	205	8–12	9.96	55.61	73	H	agreeableness	–	P	120 mos	205	–0.360	–	–	–0.230	–	–	
								conscientiousness	–	P	120 mos	205	–0.280	–	–	–0.080	–	–	
								openness	–	P	120 mos	205	–0.160	–	–	–0.190	–	–	
								extraversion	DPA	P	120 mos	205	0.040	–	–	0.050	–	–	
41	Meiser & Esser (2019)	924	9–13	12.08 (1.09)	48.2	–	I	neuroticism	PNA	R	120 mos	205	0.260	–	–	0.170	–	–	
42	Aldrich (2021) ^a	150	11–14	13.03 (0.93)	51.3	77.5	I	dysfunctional attitudes	–	R	19 mos	763	–	0.030	0.060	–	–	–	
								co-rumination	ERB	R	4 mos	138	–	0.070	–	–	–0.110	–	
								co-rumination	ERB	R	8 mos	127	–	0.210	–	–	0.160	–	
								co-rumination	ERB	R	12 mos	127	–	0.040	–	–	0.020	–	
43	Murphy et al. (2013)	133	15–19	17.04 (1.39)	100	48	I	conscientiousness	–	P	30 mos	133	–0.050	–	–	0.060	–	–	
44	Iacovino et al. (2016)	998	–	59.6 (2.8)	55.4	71.6	H	agreeableness	–	P	6 mos	998	–0.100	–	–	0.010	–	–	
								impulsivity	DIA	R	6 mos	998	0.160	–	–	0.050	–	–	
								neuroticism	PNA	R	6 mos	998	0.170	–	–	0.050	–	–	
								frustration	–	R	24 mos	957	0.100	–	–	0.060	–	–	
45	Jeronimus et al. (2017)	1816	–	16.3 (0.7)	54.6	–	I	excessive reassurance seeking	ERB	R	6 wks	110	–	0.290	–	–	–	–	
46	Birgenheir et al. (2010)	110	18–40	19.4 (2.7)	72.7	92	C	sociotropy	OOF	R	6 wks	110	–	0.200	–	–	–	–	
47	Kleiman et al. (2013)	167	17–50	20.5 (4.1)	100	58	C	enhancing cognitive style	DPA	P	4 wks	167	–0.250	–	–	–0.030	–	–	
								hopelessness	NCC	R	4 wks	209	0.100	–	–	–	–	–	
								negative cognitive style	NCC	R	4 wks	209	0.140	–	–	–	–	–	
								impulsivity - lack of perseverance	DIA	R	4 wks	201	0.099	–	–	0.023	–	–	
	Kleiman et al. (2015)	209	17–50	20.58 (4.08)	84.2	54	C	impulsivity - lack of premeditation	DIA	R	4 wks	201	0.144	–	–	0.026	–	–	
								impulsivity - negative urgency	DIA	R	4 wks	201	0.242	–	–	0.081	–	–	
								impulsivity - sensation seeking	DIA	R	4 wks	201	0.108	–	–	0.140	–	–	
								impulsivity - sensation seeking	DIA	R	4 wks	201	0.108	–	–	0.140	–	–	

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		Baseline Sample Information						ES by Stress Outcome										
		Age (years)											Dependent			Independent		
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES N	Co	In	NI	Co	In	NI
48	Riskind et al. (2013)	99	18–48	21.25 (5.06)	100	50	C	anxiety sensitivity - mental impairment	–	R	6 wks	99	0.230	–	–	–	–	–
								anxiety sensitivity - physical	–	R	6 wks	99	0.180	–	–	–	–	–
								anxiety sensitivity - social	–	R	6 wks	99	0.080	–	–	–	–	–
								looming cognitive style	–	R	6 wks	99	0.130	–	–	–	–	–
49	Rnic (2014) ^a	151	18–28	19.69 (2.15)	100	51	I	avoidance	AVD	R	3 mos	151	0.160	0.200	–0.030	0.100	–	–
								impulsivity - negative urgency	DIA	R	3 mos	151	0.144	0.210	–0.089	–0.019	–	–
								excessive reassurance seeking	ERB	R	3 mos	151	0.220	0.250	0.010	0.020	–	–
								maladaptive schema - disconnection and rejection	NCC	R	3 mos	151	0.206	0.189	0.111	0.070	–	–
								maladaptive schema - impaired autonomy	NCC	R	3 mos	151	0.111	0.107	0.049	0.132	–	–
								worry	RNT	R	3 mos	151	0.027	0.052	–0.044	0.092	–	–
50	Rose et al. (2017)	628	–	14.52	51.95	62.76	C	co-rumination	ERB	R	9 mos	429	–	0.120	0.060	–	–	–
								rumination	RNT	R	9 mos	429	–	0.330	0.190	–	–	–
51	Caldwell et al. (2004)	605	10–12	11.7 (0.68)	50.41	60.8	C	social disengagement - social helplessness	–	R	6 mos	551	–	0.160	–	–	–	–
								social disengagement - social helplessness	–	R	12 mos	490	–	0.150	–	–	–	–
								social engagement - prosocial behavior	–	P	6 mos	551	–	–0.240	–	–	–	–
								social engagement - prosocial behavior	–	P	12 mos	490	–	–0.150	–	–	–	–
								social disengagement - social withdrawal	AVD	R	6 mos	551	–	–0.030	–	–	–	–
								social disengagement - social withdrawal	AVD	R	12 mos	490	–	–0.040	–	–	–	–
								negative relational self-view - perceived control	NCC	P	6 mos	551	–	–0.080	–	–	–	–
								negative relational self-view - perceived control	NCC	P	12 mos	490	–	–0.130	–	–	–	–
								negative relational self-view - social self-competence	NCC	R	6 mos	551	–	0.080	–	–	–	–
								negative relational self-view - social self-competence	NCC	R	12 mos	490	–	0.060	–	–	–	–
								negative relational self-view - social self-worth	NCC	R	6 mos	551	–	0.160	–	–	–	–
								negative relational self-view - social self-worth	NCC	R	12 mos	490	–	0.120	–	–	–	–
52	Flynn & Rudolph (2011)	167	9–14	12.41 (1.19)	51.5	77.8	I	engagement coping	–	P	12 mos	156	–	–0.250	–0.220	–	–	–
								engagement coping	–	P	24 mos	158	–	–0.190	–0.270	–	–	–
								involuntary engagement	–	R	12 mos	156	–	0.250	0.160	–	–	–
								involuntary engagement	–	R	24 mos	158	–	0.240	0.240	–	–	–
								disengagement coping	AVD	R	12 mos	156	–	0.050	0.140	–	–	–
								disengagement coping	AVD	R	24 mos	158	–	0.000	0.170	–	–	–
								involuntary disengagement	AVD	R	12 mos	156	–	0.230	0.180	–	–	–
								involuntary disengagement	AVD	R	24 mos	158	–	0.150	0.190	–	–	–
53	Shahar & Priel (2003)	603	14–16	15 ^b	53.9	–	C	self-criticism	ESS	R	4 mos	603	–	0.250	0.290	–	–	–
								dependency	OOF	R	4 mos	603	–	0.190	0.130	–	–	–
54	Bouchard & Shih (2013)	364	18–25	19.66 (1.25)	57.14	89.6	C	co-rumination	ERB	R	8 wks	364	–	0.180	–	0.060	–	–
								co-rumination	ERB	R	8 wks	276	0.152	–	–	–	–	–
								self-criticism	ESS	R	8 wks	276	0.302	0.300	–	0.093	–	–
								dependency	OOF	R	8 wks	276	0.051	0.082	–	–0.052	–	–
								rumination	RNT	R	8 wks	276	0.296	0.313	–	0.126	–	–
	Shih et al. (2018)	364	18–25	19.66 (1.25)	57.14	89.6	C	attachment - avoidant	–	R	8 wks	276	0.194	0.188	–	0.031	–	–
								attachment - anxious	OOF	R	8 wks	276	0.268	0.277	–	0.046	–	–
								unmitigated communion	OOF	R	8 wks	276	0.115	0.140	–	0.055	–	–
55	Shih (2004) ^a	99	–	19.08 (1.12)	50.51	44	H	autonomy	–	R	6 wks	99	–	–	0.140	–	–	–
								sociotropy	OOF	R	6 wks	99	–	–	0.060	–	–	–
	Shih (2006)	99	–	19.08 (1.12)	50.51	44	H	autonomy	–	R	6 wks	99	–	0.120	–	–	–	–
								sociotropy	OOF	R	6 wks	99	–	0.210	–	–	–	–
	Shih & Eberhart (2010)	99	–	19.08 (1.12)	50.51	44	H	interpersonal problems - hard to be supportive	–	R	6 wks	99	–	–0.030	–	–	–	–
								interpersonal problems - too open	–	R	6 wks	99	–	–0.040	–	–	–	–
								aggression	DIA	R	6 wks	99	–	0.070	–	–	–	–
								interpersonal problems - too caring	OOF	R	6 wks	99	–	0.190	–	–	–	–
								interpersonal problems - too dependent	OOF	R	6 wks	99	–	0.050	–	–	–	–

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		Baseline Sample Information						ES by Stress Outcome											
		Age (years)												Dependent			Independent		
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES N	Co	In	NI	Co	In	NI	
56	Snyder & Hankin (2016)	360	8–16	12.06 (2.35)	57.2	75	C	effortful control	–	P	15 mos	360	–0.345	–	–	–	–	–	
								rumination	RNT	R	15 mos	360	0.373	–	–	–	–	–	
57	Stroud et al. (2015)	126	10–13	12.39 (0.76)	100	82.6	I	constraint	–	P	12 mos	105	–	–0.160	–	–	–	–	
								positive emotionality	DPA	P	12 mos	105	–	0.020	–	–	–	–	
								negative emotionality	PNA	R	12 mos	105	–	0.420	–	–	–	–	
	Stroud et al. (2018)	126	10–13	12.39 (0.76)	100	82.6	I	excessive reassurance seeking	ERB	R	12 mos	110	–	0.400	0.120	–	0.120	0.060	
								rumination	RNT	R	12 mos	111	–	0.360	0.150	–	0.130	–0.010	
58	Goldstein et al. (2021)	917	18+	19.19 (1.28)	53.9	86.7	I	extraversion	DPA	P	60 mos	917	–	–0.030	0.040	–	–0.030	–0.050	
								neuroticism	PNA	R	60 mos	917	–	0.130	0.020	–	0.060	–0.010	
59	Tsai (2016) ^a	676	–	15.6 (0.63)	52.6	52.1	C	emotion suppression	–	R	6 mos	304 ^e	–	0.240	–	–	–	–	
								emotion suppression	–	R	6 mos	372 ^f	–	–0.010	–	–	–	–	
60	Tuna (2020)	162	18–29	21.11 (1.7)	92.6	–	C	excessive reassurance seeking	ERB	R	5 mos	162	–	0.220	–	–	–	–	
								rumination	RNT	R	5 mos	162	–	0.450	–	–	–	–	
61	Judah et al. (2013)	112	–	19.4 (2.3)	74.1	87.5	C	worry	RNT	R	4 wks	112	0.150	–	–	–	–	–	
								worry	RNT	R	8 wks	112	0.060	–	–	–	–	–	
62	Maniates et al. (2018)	116	27–70	56.23 (9.54)	12.1	76.7	I	constraint	–	R	24 mos	108	–0.224	–	–	–0.130	–	–	
								positive emotionality	DPA	P	24 mos	108	0.008	–	–	0.023	–	–	
								negative emotionality	PNA	R	24 mos	108	0.098	–	–	0.085	–	–	
63	Uliaszek et al. (2012)	627	15–18	16.91 (0.39)	68.9	48.2	I	behavioral inhibition	AVD	R	24 mos	488	0.068	–	–	–	–	–	
								extraversion	DPA	P	12 mos	627	–0.090	–	–	–	–	–	
								extraversion	DPA	P	24 mos	480	–0.066	–	–	–	–	–	
								neuroticism	PNA	R	12 mos	627	0.180	–	–	–	–	–	
								neuroticism	PNA	R	24 mos	497	0.156	–	–	–	–	–	
64	Auerbach et al. (2011)	405	14–19	16.18 (0.95)	50.2	0	C	extrinsic aspirations	–	R	1 mo	383	–	0.135	–	–	–	–	
								extrinsic aspirations	–	R	2 mos	376	–	0.115	–	–	–	–	
								extrinsic aspirations	–	R	3 mos	372	–	0.062	–	–	–	–	
								extrinsic aspirations	–	R	4 mos	379	–	0.092	–	–	–	–	
								extrinsic aspirations	–	R	5 mos	371	–	0.081	–	–	–	–	
								extrinsic aspirations	–	R	6 mos	341	–	0.089	–	–	–	–	
								intrinsic aspirations	–	P	1 mo	383	–	–0.070	–	–	–	–	
								intrinsic aspirations	–	P	2 mos	375	–	–0.100	–	–	–	–	
								intrinsic aspirations	–	P	3 mos	370	–	–0.114	–	–	–	–	
								intrinsic aspirations	–	P	4 mos	377	–	–0.114	–	–	–	–	
								intrinsic aspirations	–	P	5 mos	371	–	–0.127	–	–	–	–	
								intrinsic aspirations	–	P	6 mos	342	–	–0.102	–	–	–	–	
								physical health aspirations	–	–	1 mo	391	–	–0.108	–	–	–	–	
								physical health aspirations	–	–	2 mos	383	–	–0.107	–	–	–	–	
								physical health aspirations	–	–	3 mos	378	–	–0.126	–	–	–	–	
								physical health aspirations	–	–	4 mos	385	–	–0.086	–	–	–	–	
								physical health aspirations	–	–	5 mos	377	–	–0.111	–	–	–	–	
								physical health aspirations	–	–	6 mos	347	–	–0.040	–	–	–	–	
65	Auerbach et al. (2011)	255	12–18	14.48 (1.47)	57.4	79.5	C	extrinsic aspirations	–	R	6 wks	81	–	–0.061	–	–	–	–	
								extrinsic aspirations	–	R	12 wks	72	–	–0.039	–	–	–	–	
								extrinsic aspirations	–	R	18 wks	71	–	–0.128	–	–	–	–	
								extrinsic aspirations	–	R	24 wks	23	–	–0.150	–	–	–	–	
								intrinsic aspirations	–	P	6 wks	81	–	–0.226	–	–	–	–	
								intrinsic aspirations	–	P	12 wks	72	–	–0.213	–	–	–	–	
								intrinsic aspirations	–	P	18 wks	71	–	–0.199	–	–	–	–	
								intrinsic aspirations	–	P	24 wks	23	–	–0.053	–	–	–	–	
								physical health aspirations	–	–	6 wks	82	–	–0.237	–	–	–	–	
								physical health aspirations	–	–	12 wks	73	–	–0.156	–	–	–	–	

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Baseline Sample Information										ES by Stress Outcome									
		Age (years)											Dependent			Independent			
ID	Manuscript	N	Range	M (SD)	% Women	% White	Stress Assess Type	Predictor Construct	Cluster	R or P	Follow-Up Length	ES	N	Co	In	NI	Co	In	NI
66	Felton et al. (2022)	213	12–17	15.02 (0.95)	45	53	C	physical health aspirations	–	–	18 wks	71	–	–0.149	–	–	–	–	–
								physical health aspirations	–	–	24 wks	23	–	–0.036	–	–	–	–	–
								delay discounting	–	R	12 mos	193	0.030	–	–	–0.100	–	–	–
								delay discounting	–	R	24 mos	154	0.090	–	–	–0.010	–	–	–
67	Hasegawa et al. (2022)	201	18–51	20.2 (2.83)	58.21	0	C	delay discounting	–	R	36 mos	152	0.130	–	–	–0.070	–	–	–
								aggression	DIA	R	8 wks	201	–	0.330	0.180	0.230	–	–	–
68	Harrison et al., (2022)	136	6–13	8.69 (1.81)	55.9	84.6	C	rumination	RNT	R	8 wks	201	–	0.350	0.370	0.270	–	–	–
								social cognitive distortions	NCC	R	10 mos	136	0.320	0.240	0.280	0.160	–	–	–
69	Taylor & Snyder (2021)	224	18–23	19.3 (1.27)	70.09	59.4	C	social cognitive distortions	NCC	R	70 mos	113	0.010	0.090	–0.040	0.060	–	–	–
								attentional control	–	P	varied	209	–0.260	–	–	–	–	–	–
								executive functioning	–	P	varied	209	–0.110	–	–	–	–	–	–
								IQ	–	–	varied	209	0.020	–	–	–	–	–	–
70	Chen et al. (in prep) ^a	241	14–17	15.9 (1.09)	54	73.9	I	psychomotor speed	–	–	varied	209	–0.010	–	–	–	–	–	
								working memory	–	P	varied	209	–0.060	–	–	–	–	–	–
								rumination	RNT	R	varied	209	0.260	–	–	–	–	–	–
								worry	RNT	R	varied	209	0.290	–	–	–	–	–	–
								positive affect dampening	RNT	R	19 mos	190	–	0.110	0.100	0.230	–	–	–
								rumination	RNT	R	19 mos	190	–	0.140	–0.040	0.220	–	–	–
								negative emotion differentiation	–	P	19 mos	189	–0.210	–	–	–0.140	–	–	–
								negative emotion variability	–	R	19 mos	189	0.200	–	–	0.190	–	–	–
								positive emotion differentiation	–	P	19 mos	189	–0.190	–	–	–0.070	–	–	–
								positive emotion variability	–	R	19 mos	189	0.160	–	–	0.150	–	–	–
Santee & Starr (in prep) ^a	232	14–17	15.93 (1.09)	53.02	74.57	I	positive affect	DPA	P	19 mos	189	0.050	–	–	0.030	–	–	–	
							negative affect	PNA	R	19 mos	189	0.280	–	–	0.180	–	–	–	

Note. ID = sample ID. *N* = number of participants. *M* = mean. *SD* = standard deviation. R = theoretical risk factor. P = theoretical protective factor. ES = effect size. Co = combined stress (i.e., stressful life event measure which includes interpersonal and non-interpersonal stressors). In = interpersonal stress. mo = month. wk = week. yr = year. C = checklist. I = interview. H = hybrid approach (i.e., stressful life event measure which incorporates the use of a self-report checklist followed up by a life stress interview). NI = non-interpersonal stress. ERB = maladaptive interpersonal emotion regulation behaviors. DIA = disinhibition and antagonism. AVD = avoidance. RNT = repetitive negative thinking. NCC = negative cognitive content. ESS = excessive standards for self. PNA = pervasive negative affect. OOF = dispositional other-oriented focus. DPA = dispositional positive affect and upregulation.

^a unpublished manuscript.

^b imputed value for analyses. In cases where only an age range was provided (e.g., Shahar & Priel, 2003), we imputed mean age as the midpoint value of the upper and lower limits. When information was not provided for age and data were collected from undergraduates in an introductory course (Sahl et al., 2009), we imputed mean age as 19 years.

^c female subsample.

^d male subsample.

^e European American subsample.

^f Vietnamese American subsample.

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Appendix E. Forest plots of effect sizes for predictor clusters on dependent stress

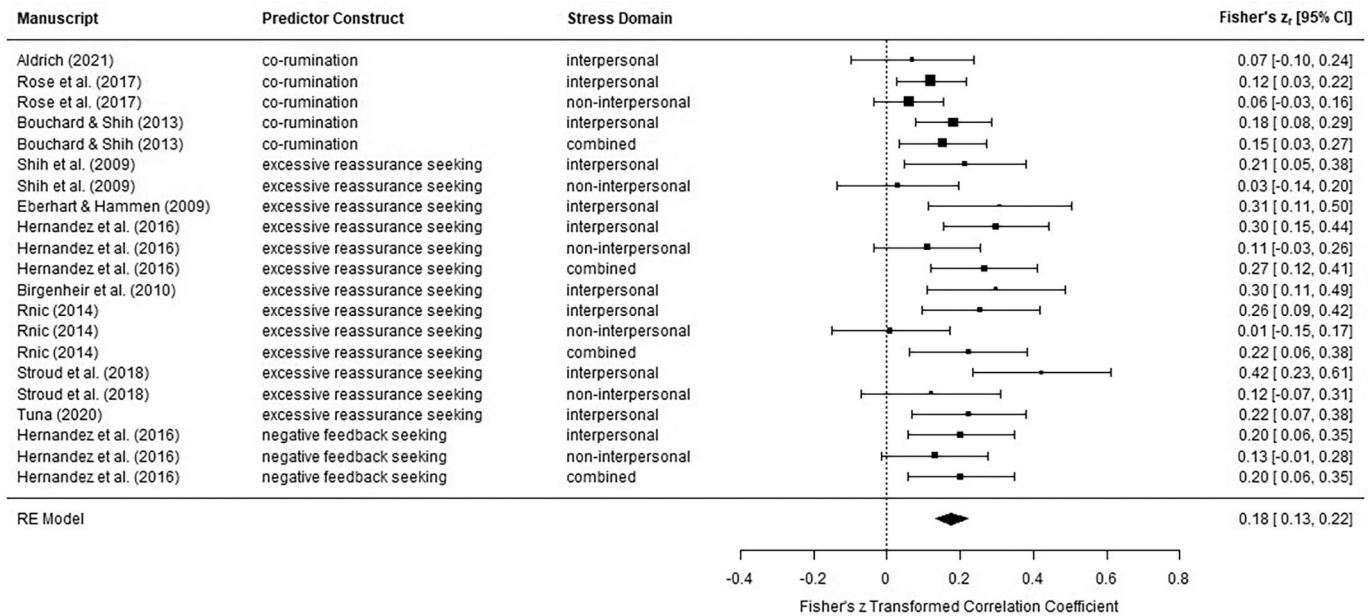


Fig. E.1. Maladaptive interpersonal emotion regulation behaviors.

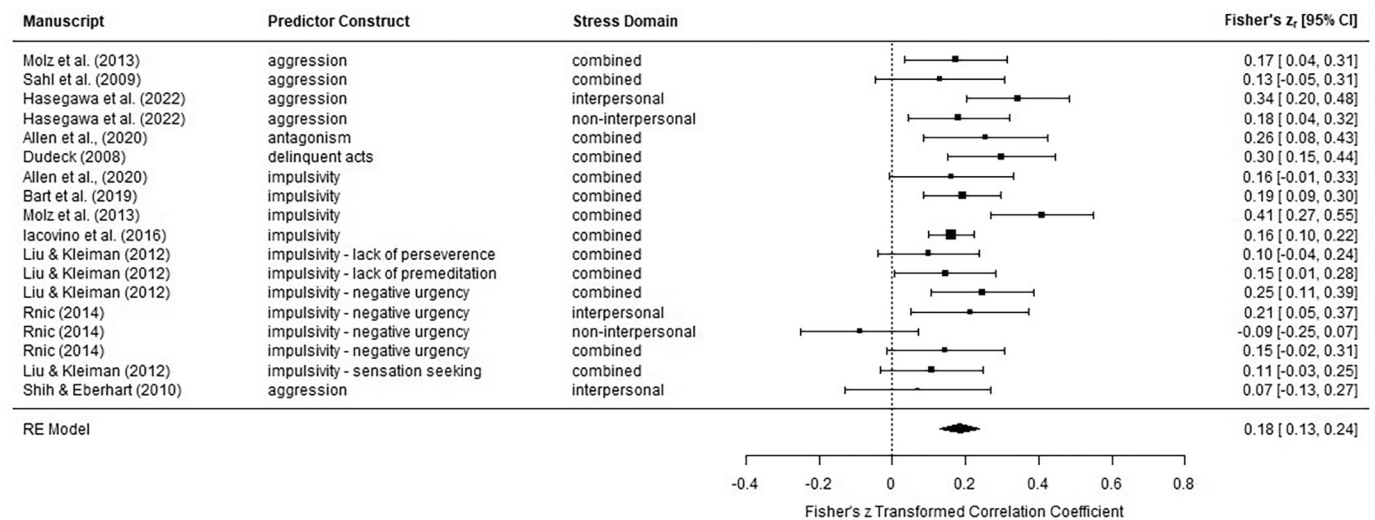


Fig. E.2. Disinhibition and antagonism.

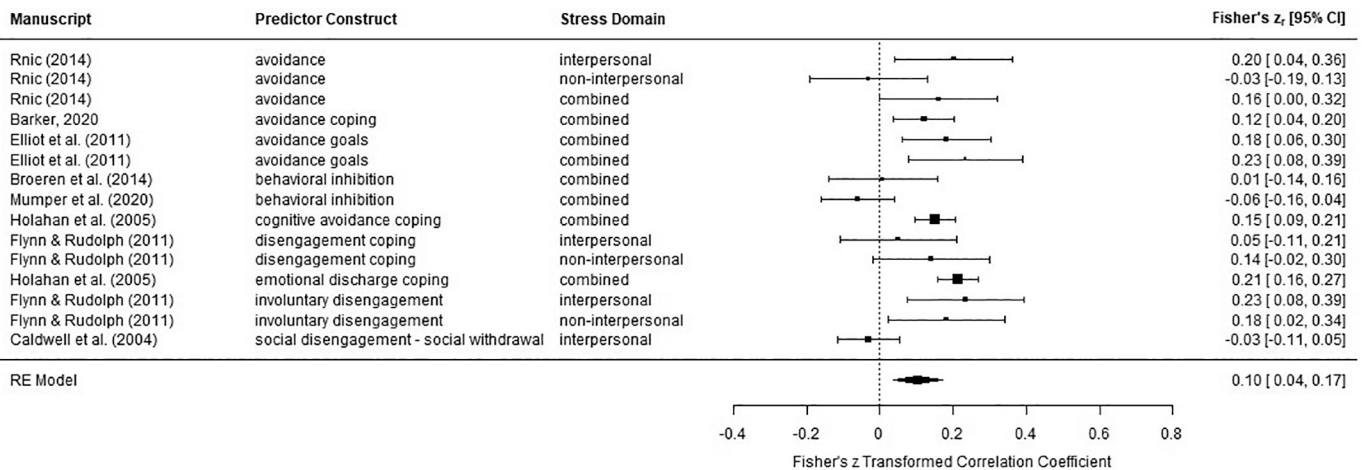


Fig. E.3. Avoidance.

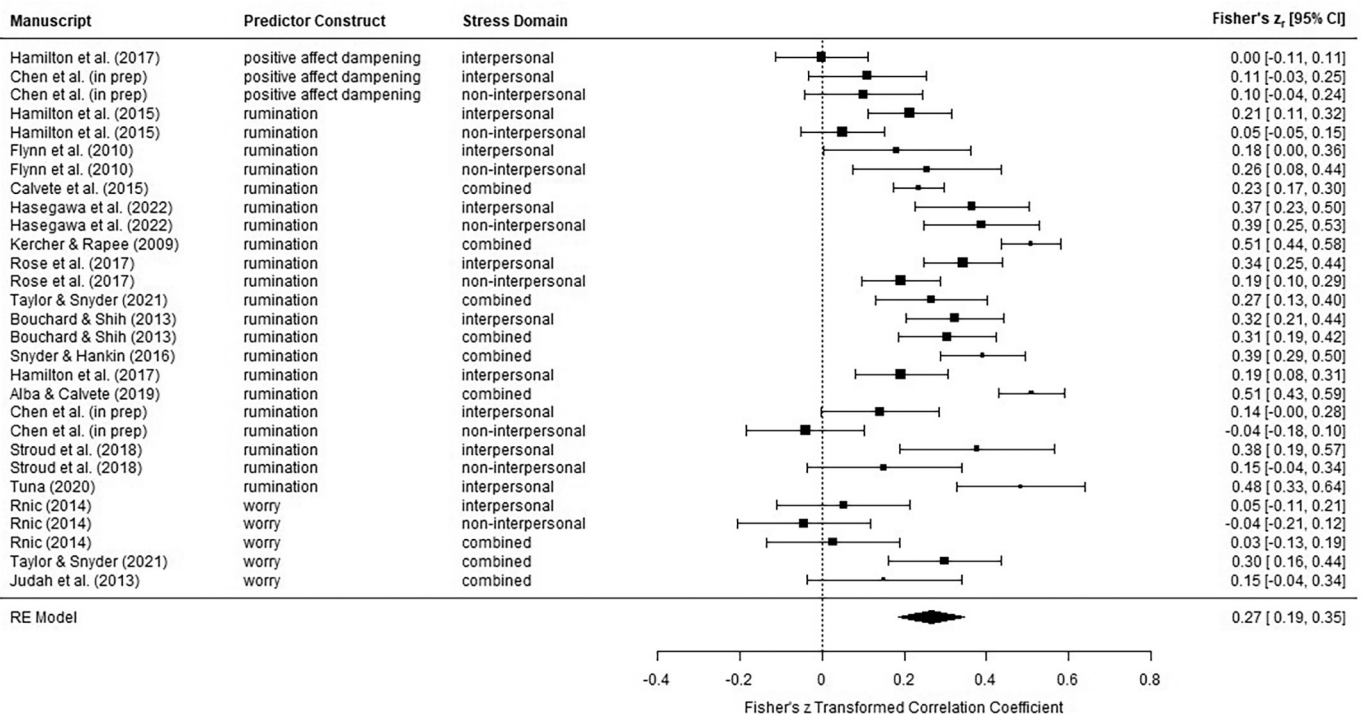


Fig. E.4. Repetitive negative thinking.

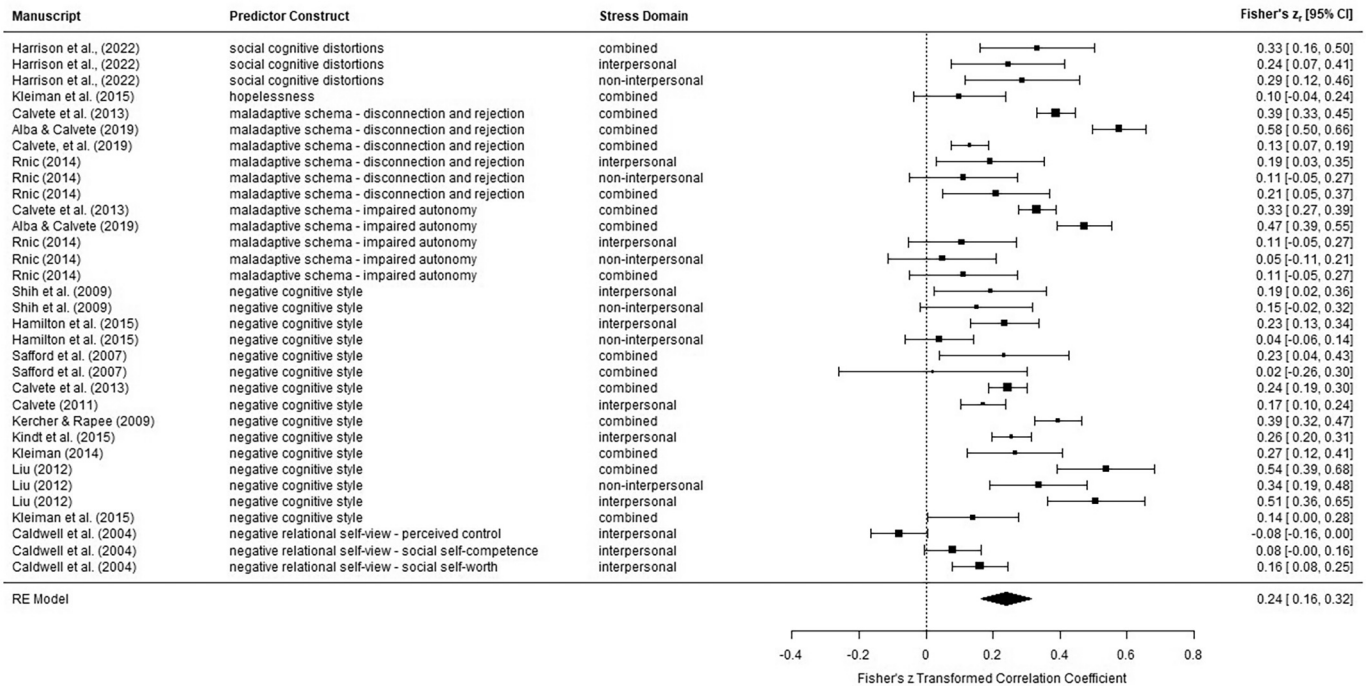


Fig. E.5. Negative cognitive content.

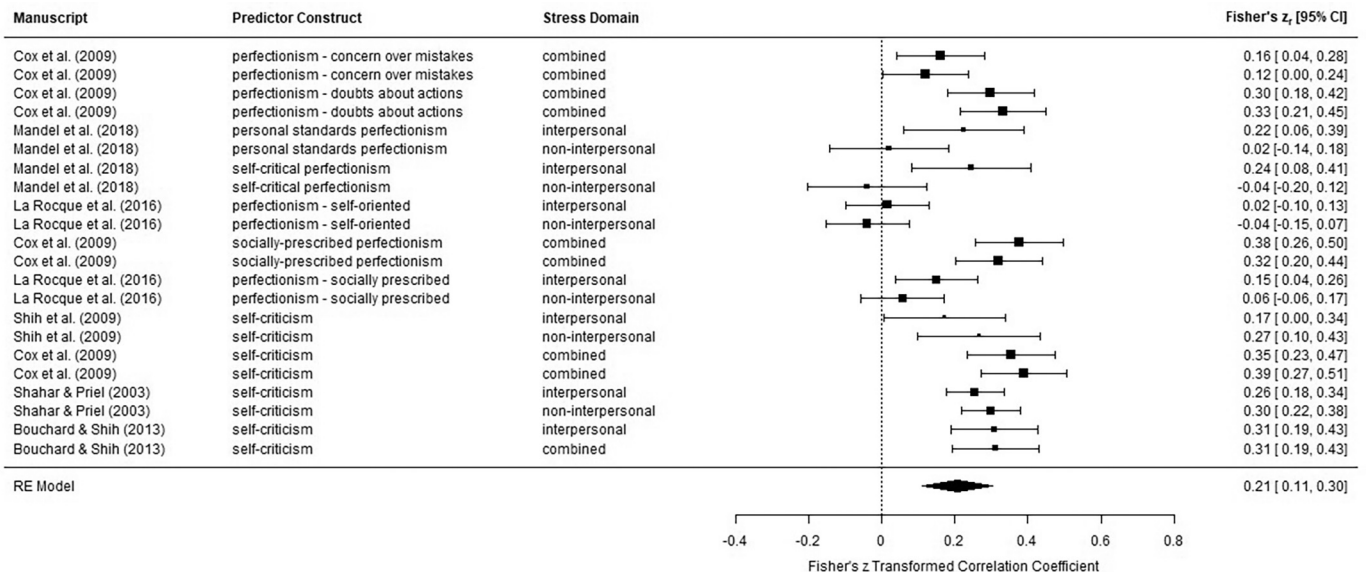


Fig. E.6. Excessive standards for self.

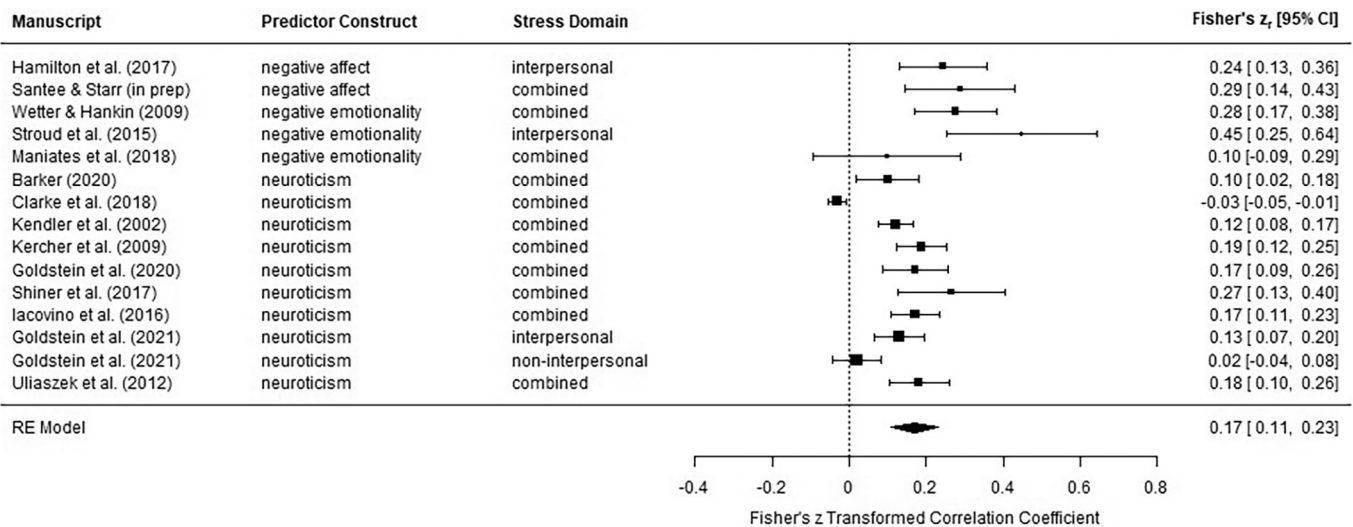


Fig. E.7. Pervasive negative affect.

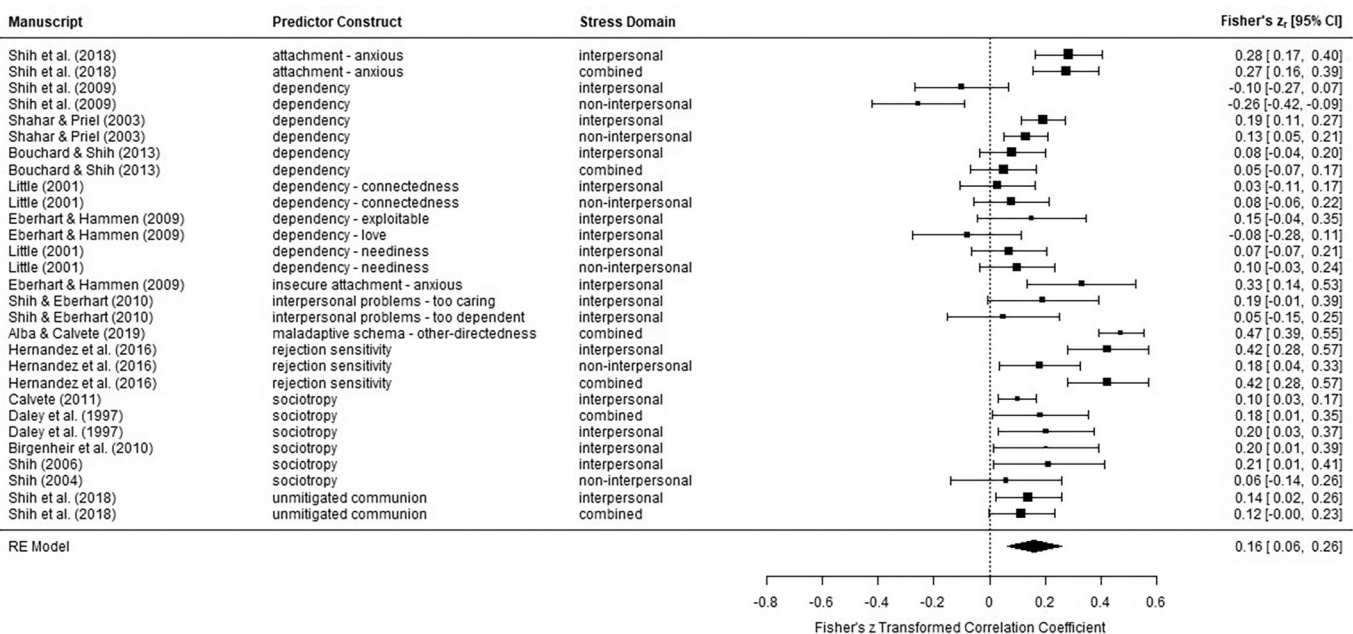


Fig. E.8. Dispositional other-oriented focus.

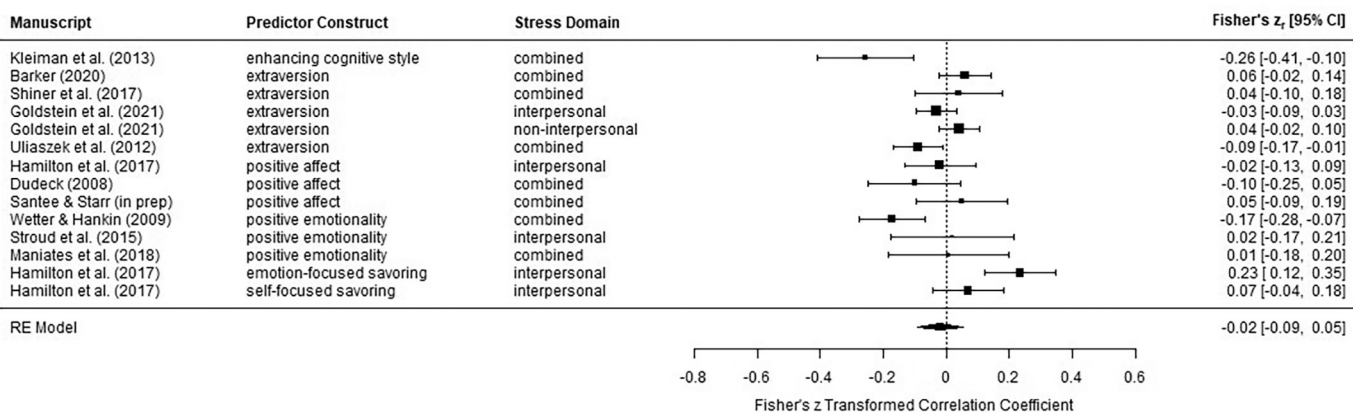


Fig. E.9. Dispositional positive affect and upregulation.

Appendix F. Forest plots of effect sizes for predictor clusters on independent stress

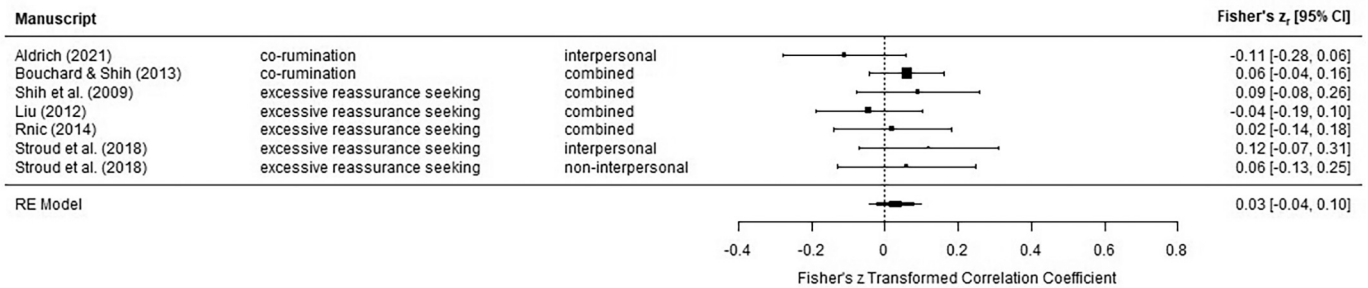


Fig. F.1. Maladaptive interpersonal emotion regulation behaviors.

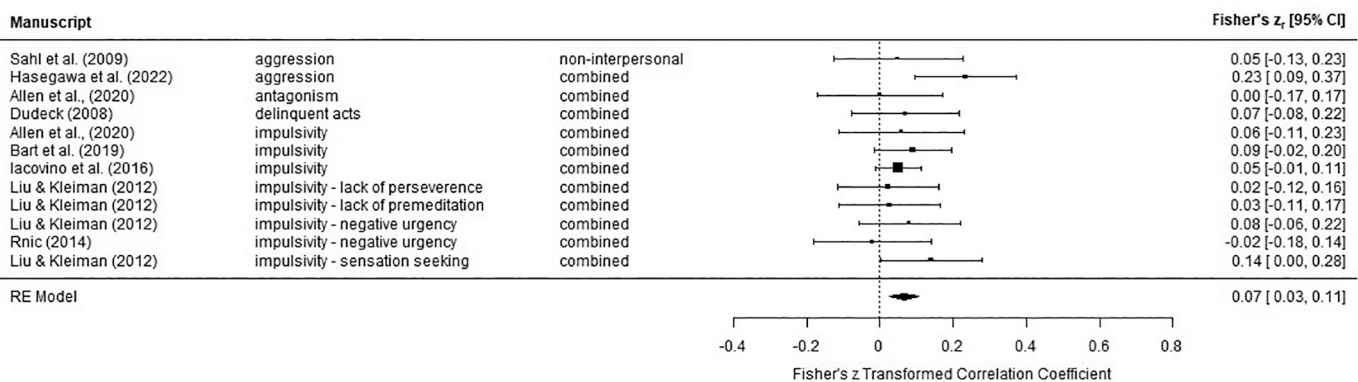


Fig. F.2. Disinhibition and antagonism.

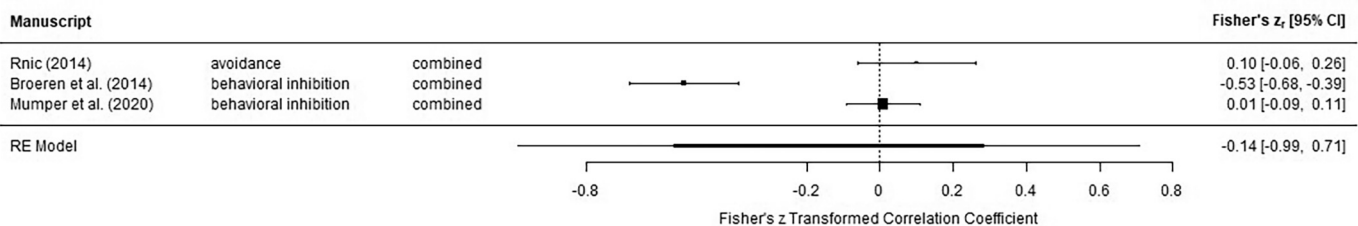


Fig. F.3. Avoidance.

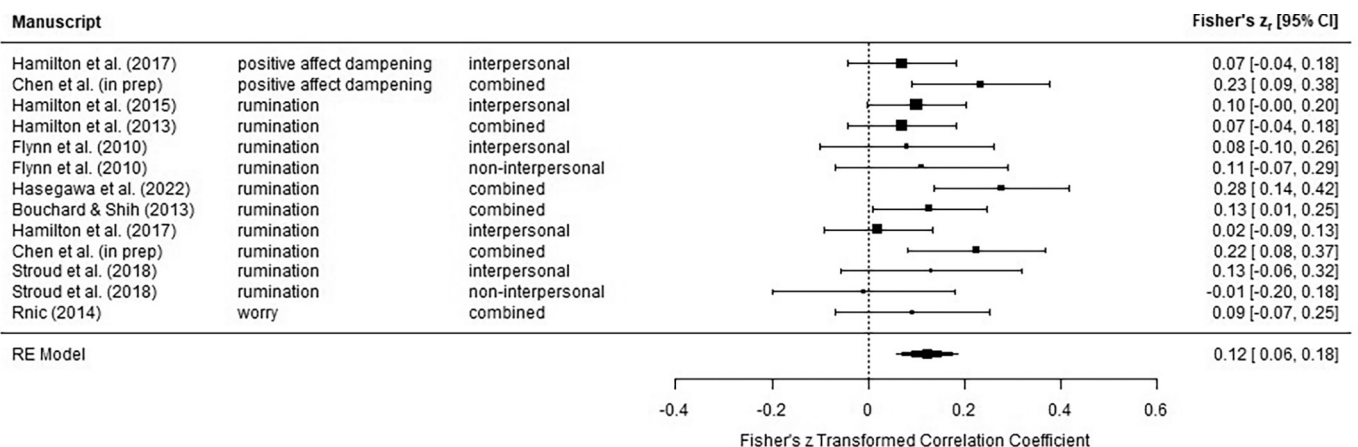


Fig. F.4. Repetitive negative thinking.

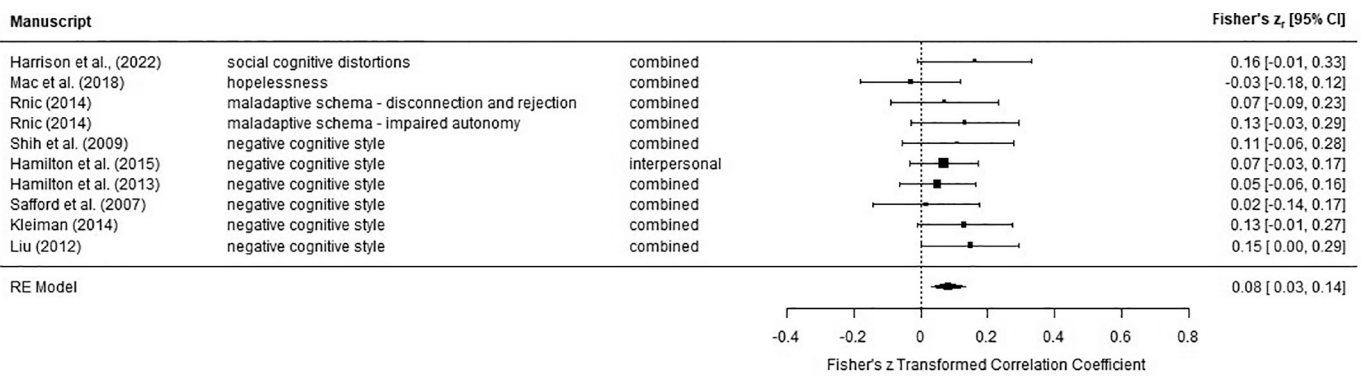


Fig. F.5. Negative cognitive content.

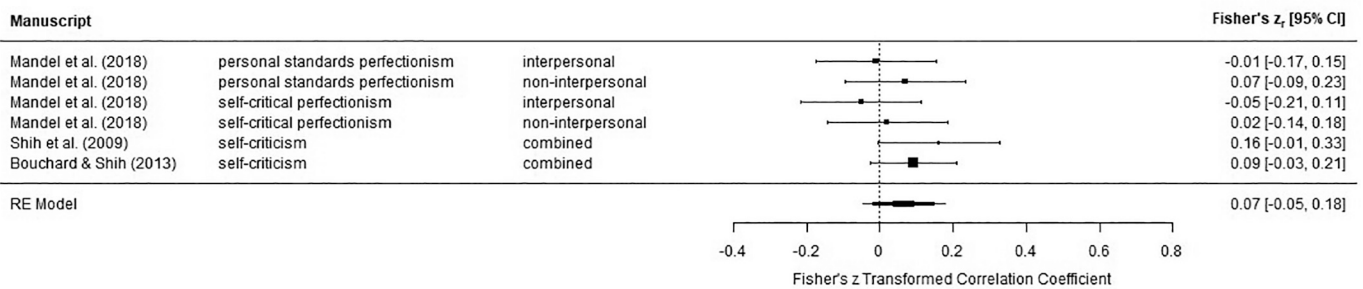


Fig. F.6. Excessive standards for self.

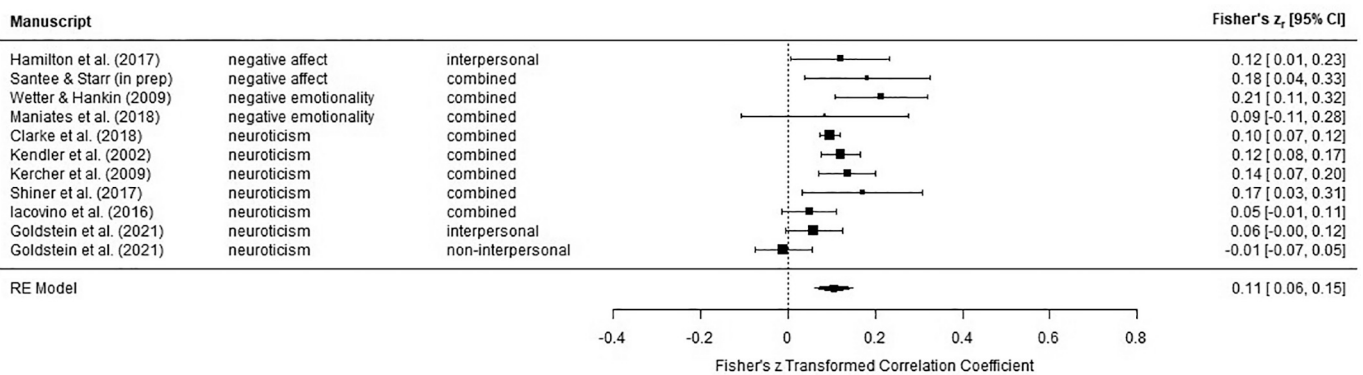


Fig. F.7. Pervasive negative affect.

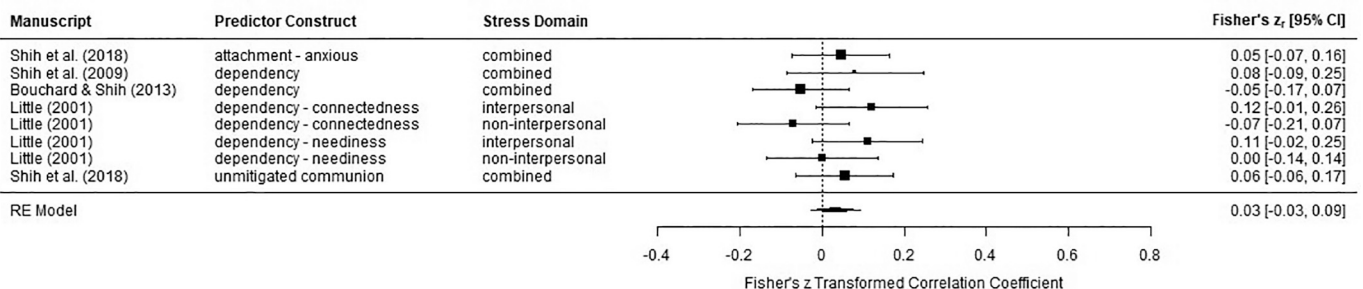


Fig. F.8. Dispositional other-oriented focus.

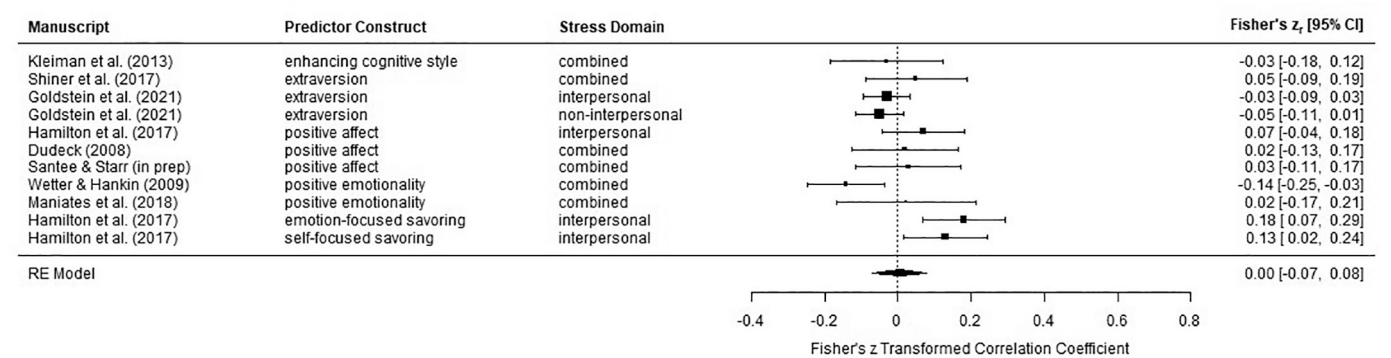


Fig. F.9. Dispositional positive affect and upregulation.

Appendix G. Funnel plots of effect sizes for predictor clusters on dependent stress and independent stress

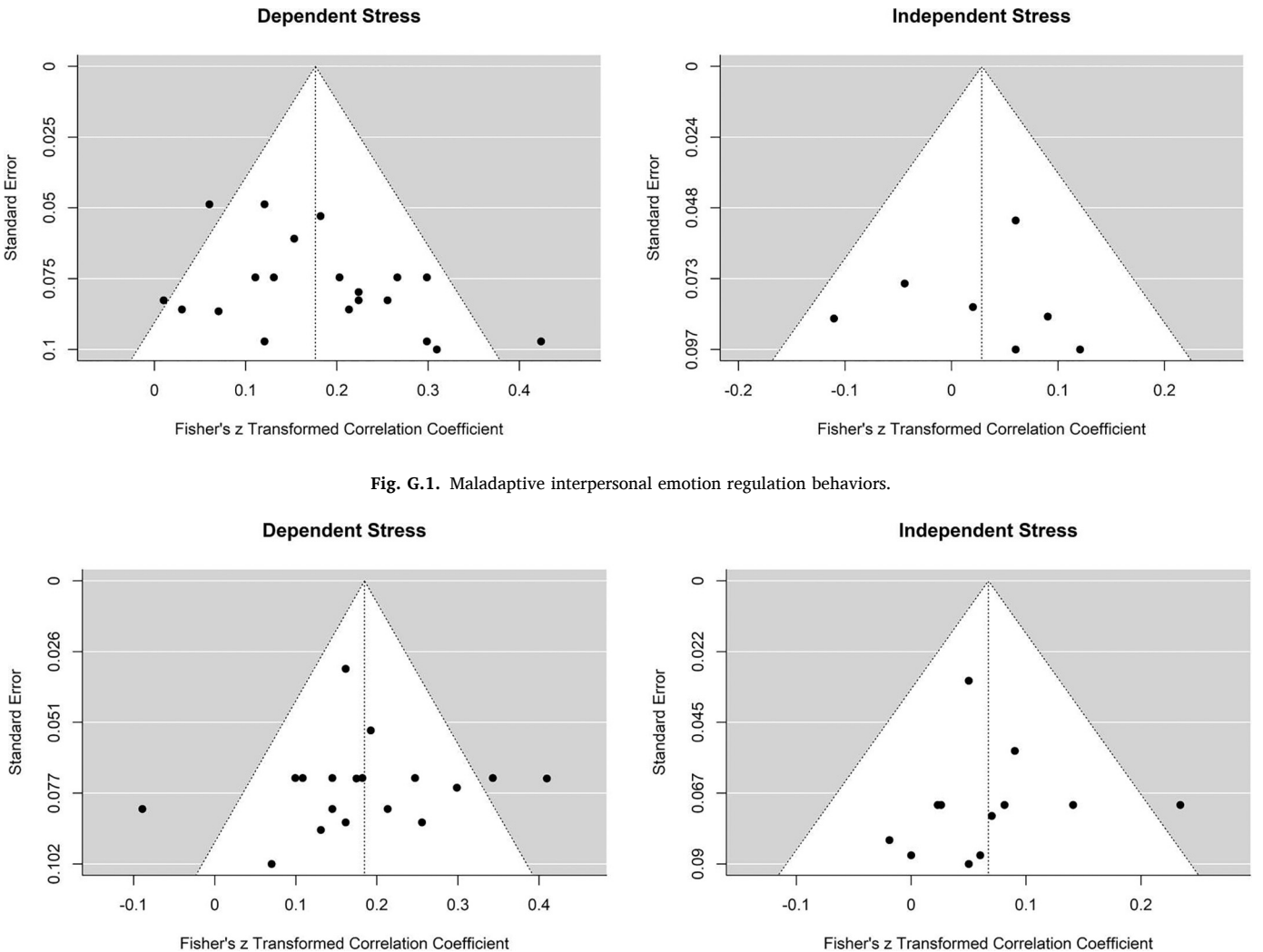


Fig. G.1. Maladaptive interpersonal emotion regulation behaviors.

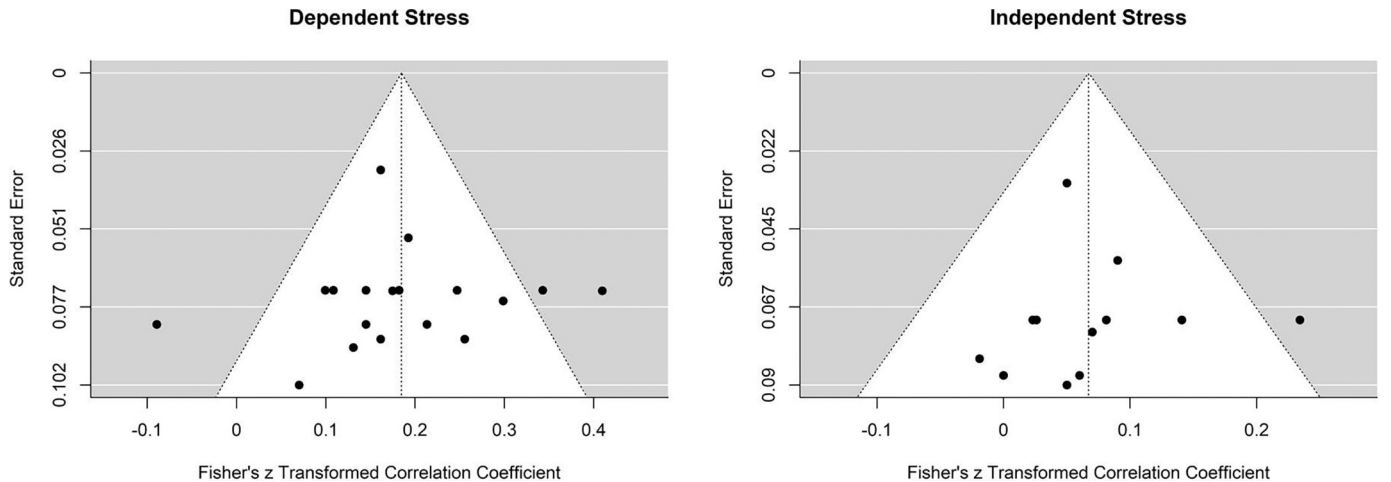


Fig. G.2. Disinhibition and antagonism.

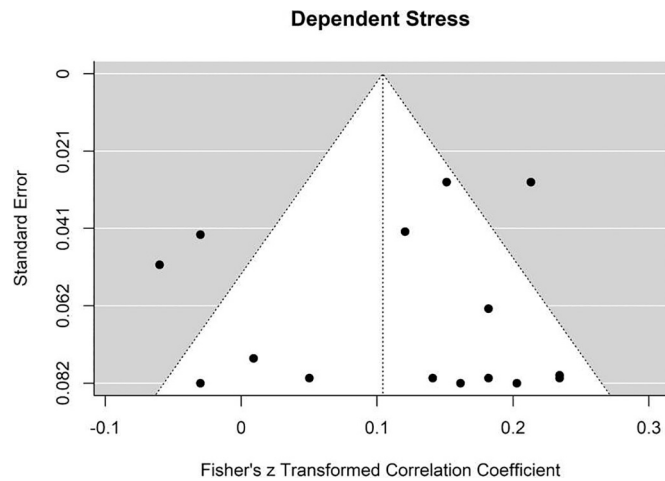


Fig. G.3. Avoidance.

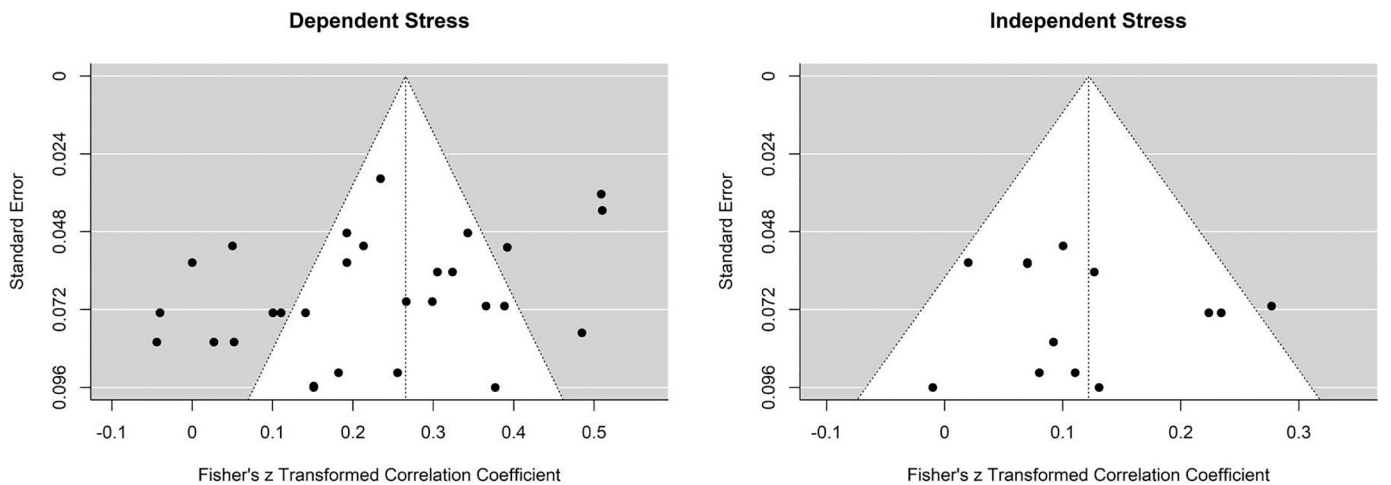


Fig. G.4. Repetitive negative thinking.

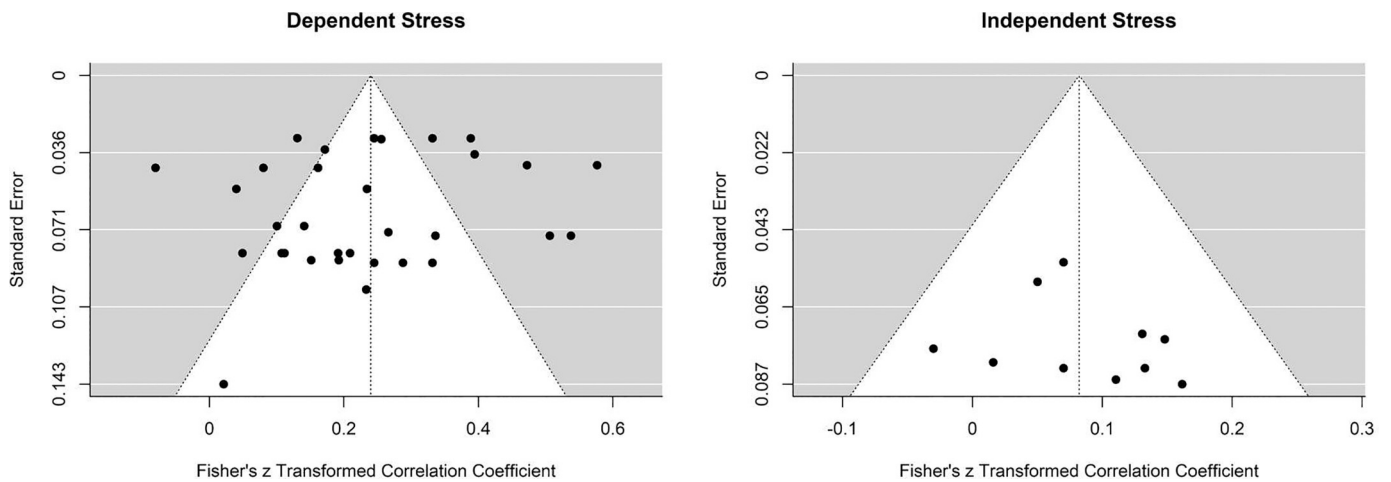


Fig. G.5. Negative cognitive content.

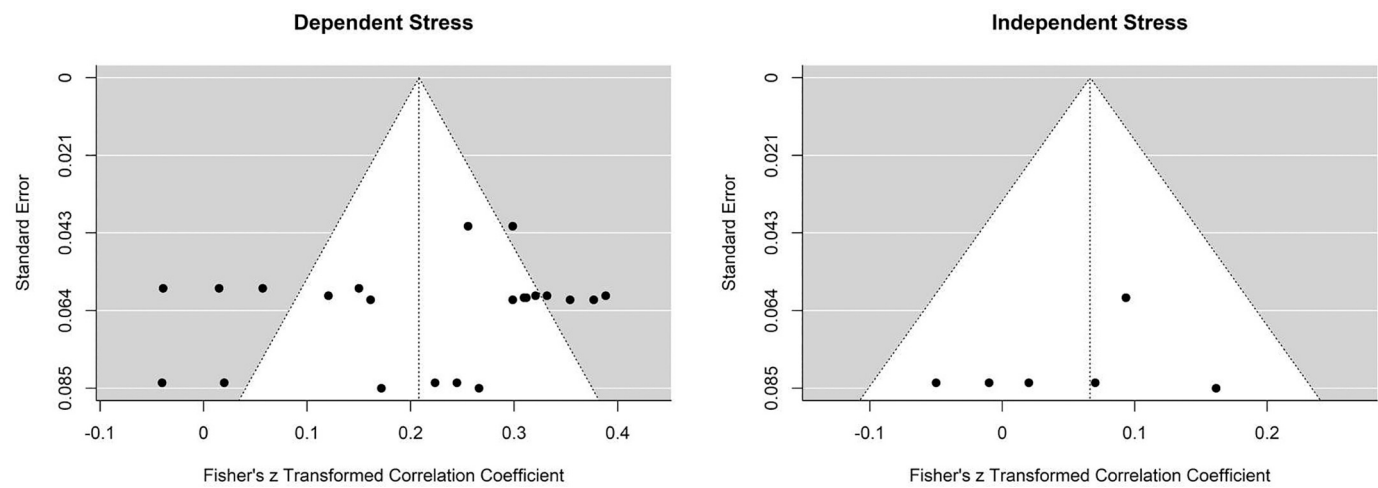


Fig. G.6. Excessive standards for self.

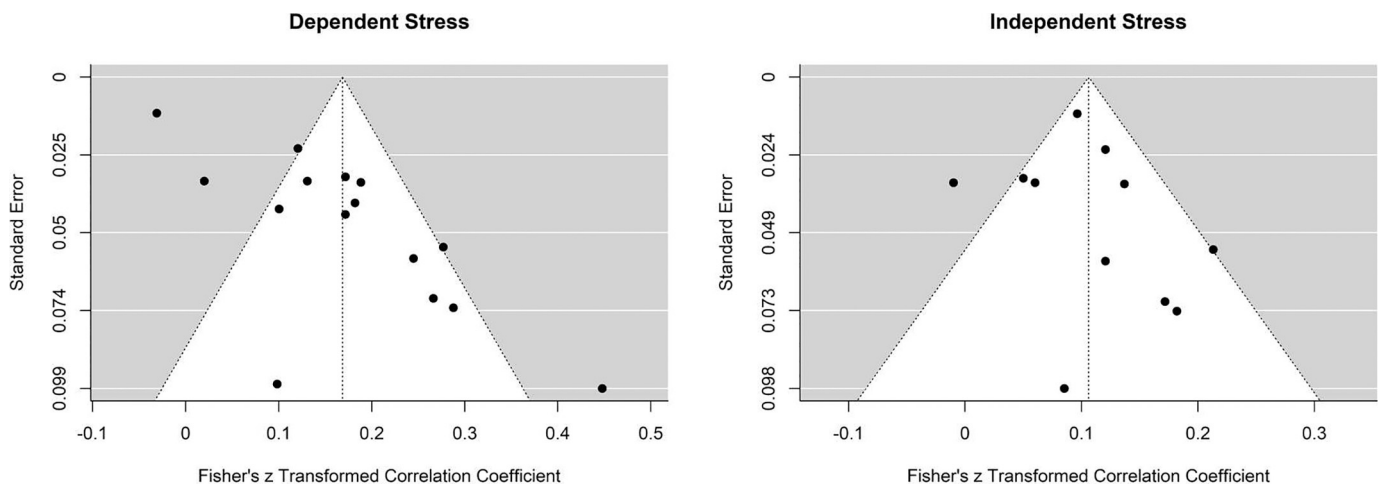


Fig. G.7. Pervasive negative affect.

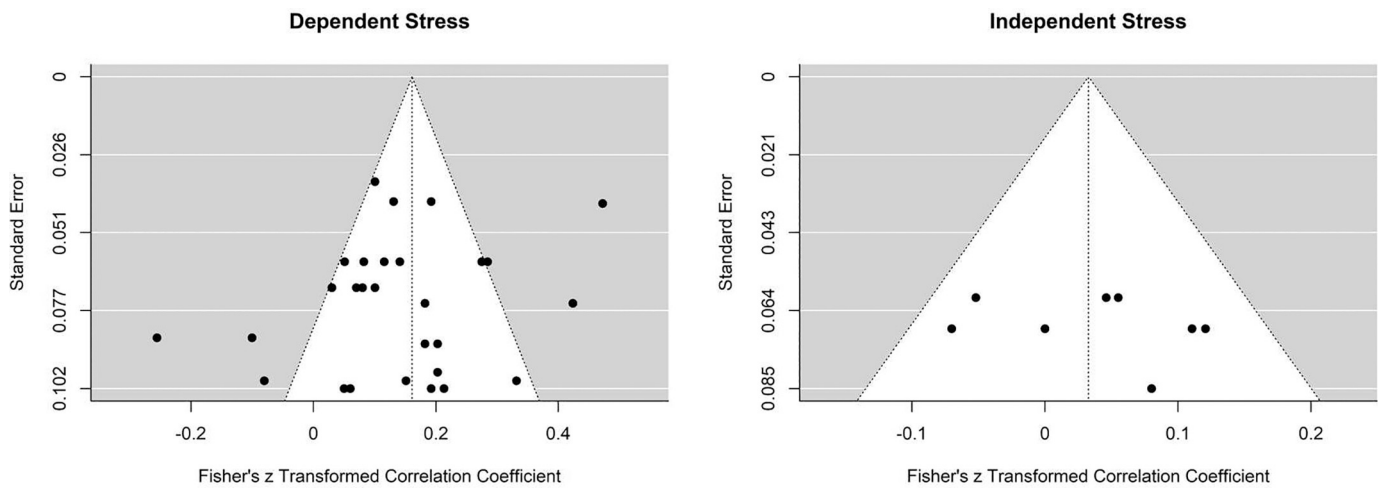


Fig. G.8. Dispositional other-oriented focus.

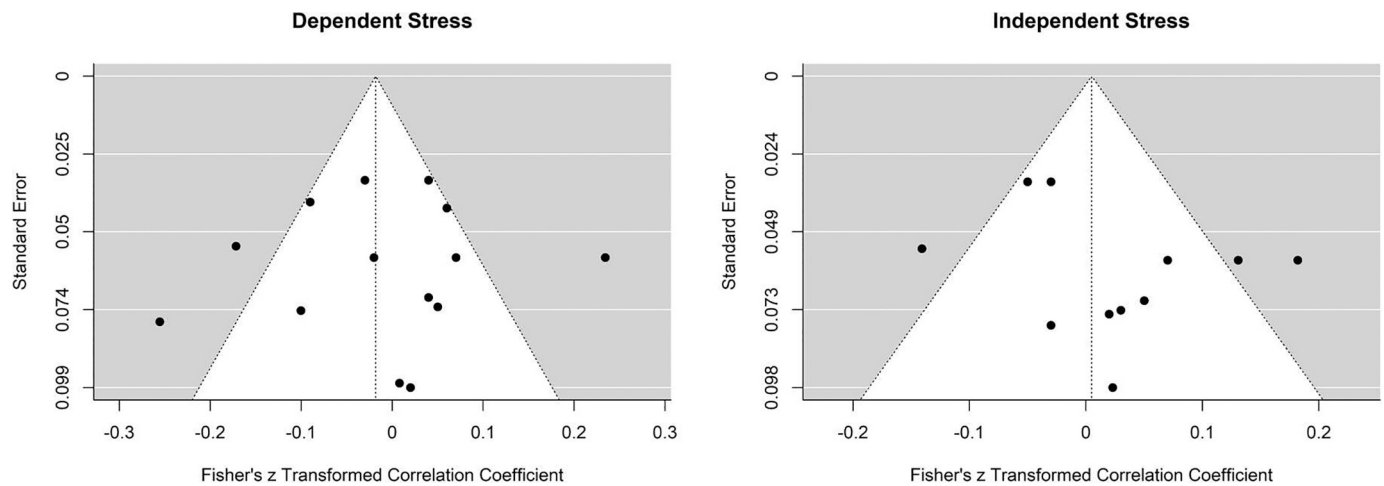


Fig. G.9. Dispositional positive affect and upregulation.

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