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The Insidious Influence of Stress: An Integrated Model of Stress, Executive Control, and Psychopathology

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Abstract

Although exposure to acute stress undoubtedly contributes to psychopathology, most individuals do not develop psychopathology following stress exposure. To explain this, biological, emotional, and cognitive responses to stress have been implicated, but individual differences in executive control (i.e., top-down control of cognition and behavior) measured in response to stress has only recently emerged as a potential factor contributing to psychopathology. In this review, we introduce a model—the *integrated model of stress, executive control, and psychopathology*—positing how the impairing effects of acute stress on executive control can contribute to psychopathology. We link to research on biological, emotional, and cognitive processes, all of which can be impacted by executive control, to propose a framework for how poorer executive control under conditions of acute stress can contribute to psychopathology. This integrated model is intended to further our understanding of who is more susceptible to the negative consequences of stress.

Keywords

executive control; executive function; stress; psychopathology

Exposure to acute stress is something we all face regularly. Most people emerge from these stressful experiences relatively unscathed, but for some, this common experience carries insidious consequences. Stress exposure is a contributing factor to the development and maintenance of many diseases and disorders, including virtually all forms of psychopathology (Brown & Harris, 1989; Grant et al., 2004; Shields & Slavich, 2017). Although there is a clear connection between stress and psychopathology, only some individuals appear susceptible to the negative consequences of stress exposure (Baratta et al., 2013; Helgeson & Zajdel, 2017; van der Werff et al., 2013). As a result of these individual differences, theories of the etiology of psychopathology have focused on the many predispositional or situational factors that interact with stress exposure to predict who is vulnerable to psychopathology during times of stress. For example, subjective experiences

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of stress, physiological responses to stress, and choice of coping or emotion regulation strategies each partially explain individual differences in the detrimental effects of stress on health (Cohen et al., 2015; Compas et al., 2017; Epel et al., 2004; Slavich & Irwin, 2014). A common aspect of all of these factors is that they can be modulated by *executive control* (i.e., internally driven, top-down, goal-directed control of cognition and behavior; A. Diamond, 2013; Williams et al., 2009). In the present review, we suggest that poor executive control, *specifically within a stressful context*, confers risk for psychopathology through its modulatory functions on many processes, including these stress-relevant processes. This is based on evidence that executive control itself is susceptible to the effects of stress. A notable implication of our model is that measuring executive control in stressful conditions should improve the predictive validity of executive control within the context of psychopathology research.

The goal of the present review is to propose a model demonstrating that the extent to which stress impairs executive control is an important risk factor for, and likely contributes to, the development of psychopathology. To achieve this goal, we first summarize current research on executive control, focusing on its importance in implementing skills and overriding tendencies, and discuss known links between executive control and psychopathology. In this section, we describe the importance of contextual factors in modulating executive control. In other words, executive control assessed in one context may look different when assessed in another context. Second, we survey the effects of acute stress on executive control and theoretical perspectives that seek to explain this effect. Third, we systematically examine evidence that executive control might be most predictive of psychopathology when it is assessed under conditions of acute stress. We also describe evidence for how executive control under acute stress—by facilitating the implementation of skills and overriding of tendencies—may modulate processes including psychological and biological responses to stress and therefore confer resilience or risk for psychopathology. Fourth, based upon this information, we propose an integrated model of stress, executive control, and psychopathology. In this section, we delineate testable predictions of this model and discuss pressing questions before summarizing and concluding. Ultimately, our model implies that understanding the dynamics between stress and executive control can contribute to our understanding of how stress precipitates psychopathology.

Executive Control

What Is Executive Control?

Throughout the literature, a variety of terms exist to describe the same basic construct of interest to this paper. Although there are nuances to each of these terms, the terms cognitive control, executive function, and executive control each describe the same basic thing: top-down, goal-directed control of cognition and behavior (Anderson & Levy, 2009; Botvinick et al., 2001; Persson et al., 2013). Within this review, we opt to use the term “executive control” to describe a broad or overarching control process, and we use the term “executive functions” when describing the individual components (i.e., functions) that support executive control more broadly.

Although the general definition of *executive control* (i.e., internally driven, top-down, goal-directed control of cognition and behavior) is agreed upon by nearly all who study it, the set of processes or functions necessary for that control of cognition and behavior to occur are not universally agreed upon. This disagreement results in discrepancies in what is implied in the term executive control. For example, likely stemming from different methods used to study executive control, cognitive neuroscientists, cognitive psychologists, and clinical neuropsychologists vary in the extent to which they see executive control as a unitary or umbrella construct (compare Koechlin & Summerfield, 2007; Shields, Sazma, & Yonelinas, 2016; Suchy, 2009). As a result, many theoretical models of executive control have been put forward, each attempting to describe the set of processes or functions necessary for internally driven, goal-directed control of cognition and behavior. A detailed discussion of these models is outside the scope of this review, but we refer the interested reader to an excellent comprehensive examination of them elsewhere (Karr, Areshenkoff, et al., 2018).

The most prominent of these models is the "unity/diversity framework" (Miyake & Friedman, 2012), depicted in Figure 1. This model was derived through factor analysis of performance (e.g., reaction time, accuracy) on tasks thought to require executive control in healthy young adults. This model posits that there is a common executive function that underpins performance across all executive control tasks as well as two more specific executive functions. The authors of this model note that there are likely many more specific processes involved, but they chose to focus on a few that have been investigated in more detail. In this model, the common executive function is also the sole executive function supporting performance on tasks that require *inhibition* (i.e., overriding and stopping prepotent or activated responses). The two specific executive functions are *updating* (i.e., monitoring the contents of working memory and adding or deleting information when appropriate; also referred to as working memory) and *shifting* (i.e., flexibly switching between mental sets, response rules, or tasks; also referred to as cognitive flexibility). Other models of executive control have been derived from factor analyses of a different set or greater number of tasks in healthy young adults (e.g., Karr, Hofer, et al., 2018; Testa et al., 2012), neuropsychological control-related deficits (e.g., Barkley, 2012; Suchy, 2009), and neuroimaging data (e.g., Koechlin & Summerfield, 2007; Petersen & Posner, 2012). Despite these other models, the unity/diversity framework is the most widely used or assumed model (e.g., A. Diamond, 2013) and it has shown strong replicability (Karr, Areshenkoff, et al., 2018). Therefore, for pragmatic purposes of this paper, we assume this model as a guide, holding that executive control is underpinned by a common executive function as well as specific processes, including updating-specific and shifting-specific executive functions, which together support the control of cognition and behavior. Additionally, as alluded to above, we also use the term "executive control" throughout this paper when referring to performance on tasks that reflect both the common executive function and a specific executive function. We chose this approach, in contrast to referring to specific executive functions like updating, because most studies linking executive control to other constructs do not demonstrate specificity of functions under the executive control umbrella.

Why is Executive Control Important?

Executive control is essential to daily life. When considering why executive control is important, it is useful to think of executive control in contrast to bottom-up, automatic, reflexive processing. Bottom-up processing is typically driven by subcortical structures (e.g., the amygdala or striatum) and sensory circuitry, and often involves fast responding to the environment based on habit (Buschman & Miller, 2007; Wood & Rünger, 2016). In contrast, executive control depends upon a broader network, with the network's most important nodes in the prefrontal cortex (Tsuchida & Fellows, 2013), and is considered a reflective or deliberative mode of responding to the environment that allows individuals to direct other processes to think or behave in goal-directed ways (A. Diamond, 2013; Fuster, 2015). In this way, executive control is essential for the capacity to effectively respond to a changing environment and for guiding behavior in novel situations (Banich, 2009). In other words, executive control allows individuals to choose how to respond to the environment, rather than simply react to it.

The definition of executive control—goal-directed control of cognition and behavior—is necessarily broad because executive control has been linked to, and thought to support or facilitate, numerous cognitive and behavioral functions. One approach to categorizing the many ways that executive control contributes to goal-directed behavior is the following: executive control allows individuals to (1) implement various skills in the service of a goal, and (2) override automatic responses that are incompatible with a goal. In the next two subsections, using these two complementary categories as a framework, we provide examples of some of the abilities linked to executive control. We then conclude this section with evidence that impairment in executive control may contribute to psychopathology through these various abilities.

Executive Control: Implementing Skills—The breadth of the impact of executive control is evident in the range of skills associated with executive control. What follows is not a comprehensive review of those skills, but rather a sampling of the types of skills that executive control is thought to contribute to, with a particular focus on skills that have been implicated in psychopathology. For example, executive control has been linked to theory of mind (Devine & Hughes, 2014), reading comprehension (Follmer, 2018), and decision making (Hinson et al., 2003). Research has also linked executive control to separate, but related forms of regulatory ability, including emotion regulation (Zelazo & Cunningham, 2007), coping (Campbell et al., 2008), and self-regulation (Hofmann et al., 2012). Although executive control is undoubtedly important to these many abilities, it is important to note that executing any goal-directed behavior involves formulating a goal, having the motivation to work towards that goal, and having the ability to achieve that goal. Executive control contributes specifically to the ability or capacity to achieve a goal (see Hofmann et al., 2012). Therefore, we highlight a few studies indicating that individual differences in level of executive control are associated with the extent to which someone can effectively carry out such skills when they intend to do so.

For example, better executive control has been associated with more effective implementation of reappraisal, an emotion regulation and coping strategy that involves

changing how one thinks about a situation to change the experience of the situation (Malooly et al., 2013). Better executive control also has been linked to better self-reported problem-solving ability in social contexts (Muscara et al., 2008). Better executive control also appears to strengthen the link between intentions to engage in health behaviors and actual engagement in those behaviors; for example, greater intentions to exercise or eat healthy relate more strongly to actually engaging in those actions when levels of executive control are higher (Hall et al., 2008). Although most studies linking executive control to such abilities are correlational, there is some support for a casual relation. For example, one study improved executive control through repeated task practice and found that trained improvement in executive control predicted improvement in the ability to down-regulate negative affect following exposure to aversive film clips (Schweizer, Grahn, Hampshire, Mobbs, & Dalgleish, 2013). Studies such as those reviewed above demonstrate a clear link between executive control and effective implementation of a variety of skills, and they provide preliminary evidence that executive control contributes to the ability to effectively implement such skills.

Executive Control: Overriding Tendencies—Executive control is also thought to contribute to the ability to override tendencies that are not in line with a goal. This means that individuals who have worse executive control are less able to override tendencies compared to others, which results in engaging in these tendencies to a greater extent, leading to undesired outcomes. For example, lower levels of executive control predict subsequent weight gain, but only in individuals with a preference for snack foods (Nederkoorn et al., 2010). The authors interpret these findings to mean that lower levels of executive control resulted in less ability to override the preference for snack foods, resulting in eating more snack foods and subsequent weight gain. In another example, participants whose executive control was improved via training, relative to participants who completed a control task, reported reduced caloric intake and weight loss (Lawrence et al., 2015).

Additional evidence comes from studies on rumination (i.e., a repetitive and passive fixation on negative emotions and thoughts; Nolen-Hoeksema et al, 2008). A number of studies demonstrate that poorer executive control is associated with more rumination (for a meta-analysis, see Yang et al., 2017). Moreover, at least one study has found support for a causal relation between executive control and rumination (Hoorelbeke et al., 2015). In this study, individuals with a tendency to ruminate completed an executive control training paradigm or active control task. After the training period, greater improvements in executive control were associated with greater declines in rumination in the training condition. Together, this work suggests that worse executive control may reduce the ability to override tendencies, leading to increased engagement in those tendencies.

Executive Control and Psychopathology

The research described in the previous subsections indicate that executive control may influence a broad range of behaviors, which is in line with the view that executive control is necessary for effective functioning in nearly all aspects of life (Jurado & Rosselli, 2007; McIntyre et al., 2013). One implication of this broad benefit of executive control is that poorer executive control may lead to significant difficulties in an individual's life. In fact,

impairment in executive control has been associated with numerous negative outcomes, including various forms of psychopathology. For example, meta-analyses show executive control impairments in substance use disorders, depression, bipolar disorder, schizophrenia, obsessive compulsive disorder, and ADHD (for a review of meta-analyses, see Snyder et al., 2015). Thus, there is clear evidence for executive control impairments in various forms of psychopathology.

Although executive control impairments exist in individuals with various forms of psychopathology, the role that these impairments play in disorders is unclear. In some cases, impaired executive control precedes and may contribute to the development of psychopathology (Cannon et al., 2006; Nelson et al., 2018; Parslow & Jorm, 2007). However, evidence for executive control contributing to psychopathology is not conclusive (e.g., Snyder, 2013), and executive control impairments may instead manifest or worsen after the onset of a disorder (e.g., Vita et al., 2012). These possibilities are not mutually exclusive, and each may explain part of the link between executive control impairments and psychopathology. For example, a meta-analysis examining executive control impairments in depression indicated that executive control impairments are sensitive to level of current symptom severity and executive control impairments are observed in remitted depression (Snyder, 2013). Executive control impairments in depression are thus both state-dependent and state-independent. It is possible that this divided role of executive control may be true in other disorders as well.

In order to understand the many ways that executive control may influence and be influenced by psychopathology, we suggest that research needs to move beyond simply observing executive control impairments in individuals with a disorder, and instead implement studies that aim to uncover how deficits in executive control might relate to psychopathology—focusing on potential mechanisms such as a reduced ability to implement skills or an inability to dampen biological stress responses. In this review, we focus on executive control impairment as a factor that contributes to the etiology and maintenance of psychopathology, a view supported by a number of theoretical frameworks. For example, individuals with impaired executive control are less able to effectively reappraise emotional situations and less able to override a tendency to ruminate, which is thought to maintain negative affect and place individuals at risk for depression (De Raedt & Koster, 2010; Joormann, 2010). Impairments in executive control also may contribute to reexperiencing —a key symptom of posttraumatic stress disorder—following exposure to a traumatic event (Parslow & Jorm, 2007). Additionally, poorer executive control could contribute to obsessive compulsive disorder through an inability to inhibit a habitual behavior, resulting in a compulsion (Graybiel & Rauch, 2000; Menzies et al., 2008). Thus, theoretical models and empirical research support the idea that executive control contributes to many forms of psychopathology. Further, lower levels of executive control may contribute to a general factor of psychopathology (Martel et al., 2017; McTeague et al., 2016). In other words, impaired executive control may be one factor that places individuals at risk for developing all forms of psychopathology.

Incorporating Context into the Measurement of Executive Control in Psychopathology Research—When evaluating the predictive ability of measures of

executive control, it is useful to consider that executive control is not something that functions in isolation. The prefrontal cortex, most often considered the region essential for executive control (Buschman & Miller, 2007; Goldman-Rakic, 1996), is part of a neural network, which interacts with other networks (Goulden et al., 2014). This interconnectedness suggests that executive control can be influenced by various other factors. Indeed, research supports the idea that factors like arousal (Kuhbandner & Zehetleitner, 2011), sleep (Killgore, 2010), and stress (Shields, Sazma, & Yonelinas, 2016) can influence executive control. This means that although individuals may have a relatively static capacity for executive control, the extent to which someone can engage in executive control to their full capacity will vary across contexts. For this reason, task-based measures of executive control—as typically administered—may be less than ideal for identifying individuals who are unable to effectively engage in executive control in their daily life, and who are vulnerable to psychopathology as a result. Instead, an alternative measure of executive control that accounts for the context in which executive control is engaged may be needed to better understand the origins of psychopathology and improve our ability to predict who is vulnerable to psychopathology.

One approach for measuring the extent to which individuals can engage executive control across contexts is to simply ask individuals how they behave in a variety of contexts in daily life. Such self-report questionnaire measures of self-control or impulsivity are viewed as alternatives to task-based measures of executive control. Although these self-report measures may better reflect behavior in daily life (Barkley & Fischer, 2011), these measures are subject to the pitfalls of self-report measures, including the potential for social desirability biases (Crutzen & Göritz, 2010) and insight or level of awareness to influence ratings (Nęcka et al., 2012). These self-report measures also capture constructs in addition to individuals' ability to implement executive control. For example, the tendency to set particular behavioral goals also influences the extent to which individuals report engaging in the behaviors probed by these scales (e.g., Toplak et al., 2013). Although self-report measures may better capture some behaviors in daily life, they may not cleanly reflect individuals' ability to use executive control in daily life.

Another approach for measuring executive control in contexts that are more relevant to daily life is to examine "hot" executive control by incorporating emotional content into existing task-based measures of executive control (e.g., using emotional words or images as stimuli). For example, depression and rumination are associated with difficulties removing negative, but not neutral or positive, information from working memory (Joormann & Stanton, 2016). This valence-specific approach suggests that some individuals demonstrate executive control impairments only under certain conditions (e.g., when negative information must be removed from working memory or prevented from entering working memory). Although this research supports moving away from using measures of pure executive control capacity, it may be insufficient for capturing how poorer executive control contributes to psychopathology. One concern is that this approach is too narrow in that it only applies to situations in which executive control is required to manipulate emotional material. Although this formulation may work for explaining how poorer executive control may lead to rumination, which in turn may lead to depression or PTSD, it is not as easily applied to all disorders. Another, perhaps broader, limitation is that including emotional stimuli in tasks

is a small change that remains far from approximating the full context in which executive control is used in daily life.

An alternative approach to measuring executive control in such a way that it predicts psychopathology is to change the context in which the task is administered to a context that reflects life outside of the lab—especially a context that commonly precedes the experience of psychopathology. One condition in life that often precedes psychopathology is exposure to acute stress. Stress has long been seen as an important factor contributing to psychopathology (e.g., Brown & Harris, 1989; Grant et al., 2004). In fact, many models of psychopathology imply that we must consider the ways that stress exposure and vulnerabilities (e.g., cognitive styles, genetic factors) interact in order to understand the emergence of psychopathology (Ingram & Luxton, 2005). Given the important role of acute stress in psychopathology, acute stress is likely an important contextual factor to consider when examining the role of executive control in psychopathology. Indeed, in this review, we propose that examining *executive control under stress* (i.e., executive control measured by standard tasks administered under conditions of acute stress) is a promising approach for understanding stress-related vulnerability to psychopathology.

Stress and Executive Control

Does Stress Affect Executive Control?

As highlighted above, stress may be an important contextual factor to consider when evaluating the role of executive control in risk for psychopathology. This raises the question of whether stress affects executive control. In the next section, we review evidence for the effects of stress on executive control, followed by a section examining theoretical perspectives on how stress impacts executive control. Understanding the potential ways that stress influences executive control is important for developing a complete understanding of how executive control may contribute to psychopathology.

Although we all experience stress, there has been debate about how to best define and measure stress in humans (e.g., Epel et al., 2018). For purposes of this paper, we will use the term “stress” as a shorthand for “acute stress,” which refers to a short, time-delimited event that confers a sense of uncontrollability, unpredictability, novelty, and/or threat to an individual (Epel et al., 2018; Koolhaas et al., 2011; Shields & Slavich, 2017). This includes events that can be classified as traumatic as well as relatively milder forms of stress. Subjectively, the experience of stress occurs when challenges to one’s physical or emotional wellbeing exceed one’s ability to cope (e.g., Gunnar & Quevedo, 2007). Our definition of stress omits “chronic stress” (i.e., a persistent, ongoing difficulty without a clearly defined beginning and/or end), as the effects of chronic stress on executive control, the role of executive control in mitigating the effects of chronic stress, and the importance of executive control to psychopathology within the context of chronic stress have not been extensively studied (though see Blair et al., 2011; Mika et al., 2012; Raver et al., 2013).

The question of whether stress influences executive control has been addressed by numerous studies. Some of these studies have found that stress improves individual executive functions (e.g., Schwabe et al., 2013), whereas other studies have found that stress impairs them

(e.g., Shields, Trainor, et al., 2016). A recent meta-analysis found that, when analyzing all types of executive control tasks together, stress impairs executive control (Shields, Sazma, et al., 2016). Further, this meta-analysis (Shields, Sazma, et al., 2016) found that stress, on average, impairs working memory updating, shifting, and cognitive inhibition, whereas it enhances response inhibition, suggesting that some of the discrepancies and mixed findings in the literature can be resolved by careful consideration of which executive function a task utilizes most. Most importantly, this meta-analysis clearly demonstrates that stress impacts executive control, and in most cases—as was the case when all tasks were meta-analyzed concurrently—stress worsens executive control.

How Does Stress Influence Executive Control?

A number of theories have been put forward that help to explain how and why stress influences executive control. Some of these theories were not constructed with the intention of explaining the findings that stress impairs performance on executive control tasks, but each can help make sense of the findings. Although these theories do not always align with one another, they are not mutually exclusive, and each may in fact contribute to our understanding of how and why stress influences executive control.

The most commonly discussed theory of why stress influences executive control is what we term the neurobiological theory, which is often coupled with an evolutionary perspective. In brief, this theory notes that excessive subcortical activity related to threat inhibits prefrontal cortical neuronal activity, resulting in impaired executive control (e.g., Arnsten, 2015; Heatherton & Wagner, 2011; Henckens et al., 2012; Hermans et al., 2014). On a different level of explanation, this theory often contends that an impairment of executive control is adaptive when faced with a severe or life-threatening stressor. Impairing executive control shifts behavior in favor of rapid, unplanned responses to salient environmental stimuli (e.g., the stressor) at the expense of slow, deliberate actions (Henckens et al., 2012). As an extension of the neurobiological theory, the biphasic-reciprocal reallocation of neural resources model (Hermans et al., 2014) posits that the neural tradeoff favoring processing of salient environmental cues at the expense of executive control reverses approximately an hour after stress onset due to the time-dependent effects of stress hormones, which correspondingly facilitates planning during recovery from stress. Although the neurobiological theory explains much of the data on stress and executive control, it cannot easily account for why stress improves response inhibition (Schwabe et al., 2013) without extension. Regardless, this model provides a compelling explanation for why stress-induced impairments in executive control are observed.

A second theoretical framework that is useful for understanding how stress influences executive control is found in limited cognitive resource theories. Rather than taking the view that executive control is uniformly impaired under conditions of stress, these theories highlight that controlled processing is finite. They suggest that attention allocated to a stressor or subsequent rumination compete for—and take—the same resources that would be used to control other aspects of cognition and behavior (Curci et al., 2013; Klein & Boals, 2001; Mather & Sutherland, 2011). An implication of this limited resource framework is that exposure to a stressor impairs executive control only during situations in which

there is insufficient motivation to exert executive control—that is, stress occupies cognitive resources, but with sufficient motivation, one can still summon all available cognitive resources and exert typical levels of executive control (e.g., Plessow et al., 2017). This prediction has been borne out in some work (e.g., Plessow et al., 2017); however, limited resource theories have difficulty accounting for why biological manipulations, which mimic at least some of the biological effects of stress but lack the occurrence of any particularly salient event and do not generate an event to ruminate on, also impair executive control (Butts et al., 2011; Henckens et al., 2012).

The Yerkes-Dodson law (Yerkes & Dodson, 1908) is another framework that may help explain how stress influences executive control. This law states that as arousal increases, performance on simple and complex tasks both increase up to a certain point of arousal, after which performance on complex tasks decreases, leading to an inverted-U function between arousal and performance on complex tasks. This law was developed specifically within the context of arousal, but it has been extended with success to understand the effects of stress on cognitive performance (e.g., D. M. Diamond et al., 2007; Kofman et al., 2006). Although the Yerkes-Dodson law cannot account for data suggesting that moderate-to-severe stressors can enhance response inhibition (e.g., Schwabe et al., 2013), it is a regularity observed in many cognitive processes (e.g., Robbins & Arnsten, 2009), and it successfully predicts the observed decrease in performance of certain executive functions (e.g., updating) as stress severity increases from mild to severe (Shields, Sazma, et al., 2016) or as task difficulty increases (e.g., Oei et al., 2006).

The final theoretical framework we review here that is useful for understanding how stress may influence executive control is the “mood as information” theory. This theory posits that negative mood promotes an analytic style of information processing (as opposed to relying on heuristics), entailing that increases in negative mood should improve most aspects of executive control (Mitchell & Phillips, 2007; Schwarz & Clore, 1983, 2003). This theory provides a good account of most (for review, see Mitchell & Phillips, 2007) but not all (Shields, Moons, et al., 2016) results from emotion induction studies. In the present context, however, there is a clear limitation of this theory, and a discrepancy between it and the previously reviewed theories; whereas exposure to stress—which may induce negative affect among other factors—typically impairs executive control, this theory predicts that most conditions that induce negative affect would enhance executive control. Despite this discrepancy, this theory may contribute to explanations of why stress can enhance executive control under some conditions (i.e., mildly stressful situations that increase negative affect without initiating a strong stress response).

In sum, at least four theoretical frameworks—the neurobiological, limited resource, Yerkes-Dodson, and mood as information frameworks—can be used to understand how and why stress influences executive control. Although these theories were developed to explain distinct areas of research, a common thread through these theories is that moderate-to-severe stress (not discussed by mood as information theory) should, on average, impair executive control.

Individual Differences in the Extent to Which Stress Impairs Executive Control: One Size Does Not Fit All

The theories described above support the idea that stress, on average, impairs executive control. This average effect, in and of itself, does not necessarily entail anything for risk of psychopathology. If stress affected executive control the same way for everyone, relatively worse executive control under stress would be no different from relatively worse executive control in nonstressful conditions in conferring risk for psychopathology. In other words, for executive control under stress to be a useful predictor of or contributor to psychopathology, individual differences in the extent to which stress impacts executive control must exist.

A number of stable, individual difference factors have been identified that appear to alter the extent to which stress impacts executive control (for reviews of moderators, see Plieger & Reuter, 2020; Shields, Sazma, et al., 2016; Tsai et al., 2019). For example, dispositional mindsets and beliefs about stress (Crum et al., 2013, 2017; Newman et al., 2020), traits related to perceptions of stress (Lempert et al., 2012), dispositional mindfulness (Feldman et al., 2016), one's parents' experiences (Roos et al., 2020), genetic variation (Buckert et al., 2012; Qin et al., 2012; Zareyan et al., 2020), and biological sex (Schoofs et al., 2013; Shields, Trainor, et al., 2016) all moderate the extent to which stress impairs executive control. As such, although there is, on average, an impairing effect of stress on executive control, stress does not reduce executive control to the same extent or in the same way across all individuals. Therefore, individual differences in executive control under stress may represent a construct that is distinct from individual differences in executive control under nonstressful conditions. Together, these findings entail that individual differences in executive control under stress may relate to psychopathology differently than individual differences in executive control under nonstressful conditions.

Executive Control Under Acute Stress and Psychopathology

Does Executive Control Under Acute Stress Predict Psychopathology?

For executive control under stress to be a useful predictor of or contributor to psychopathology, it must be a better predictor of psychopathology than standard measures of executive control. To determine whether executive control under stress predicts psychopathology more strongly than executive control under nonstressful conditions, we conducted a systematic review of all human studies that examined any aspect of mental health or psychopathology in relation to executive control and included an experimental manipulation of acute stress. We did not include studies that manipulated emotion or arousal (e.g., Schweizer & Dalgleish, 2011; Zhang et al., 2013) because these types of manipulations produce effects that are different from stress manipulations. For example, compared to emotion and/or arousal manipulations, stress manipulations modulate more biological processes and exert different effects on executive control (Mitchell & Phillips, 2007; Shields, 2020; Shields, Moons, et al., 2016; Shields, Sazma, et al., 2016). Relatedly, we only included studies that experimentally manipulated stress in this literature review because without experimental manipulation, we cannot know whether a stress induction was effective or whether other influences on executive control can be ruled out. Most notably, when stress was not experimentally manipulated, studies typically relied on

assessing executive control pre- and then again post-stressor, which resulted in stress being confounded with practice effects, potentially obscuring the effects of stress on the association between executive control and psychopathology (Shields, 2020). For studies that employed a within-subjects crossover design, we only included studies that administered the stress and control tasks on separate days given that the effects of stress and glucocorticoids can last for hours (e.g., Henckens et al., 2011; Schwabe & Wolf, 2014; Shields et al., 2016). Although this strict inclusion criteria led to the exclusion of studies that were central in early formulations of our model (e.g., Quinn & Joormann, 2015a), this approach allowed us to more accurately assess whether executive control under stress is a distinct predictor of psychopathology.

We exhaustively searched PubMed, PsycINFO, the ProQuest Psychology Database, and Web of Science using the search strings given in Supplemental Material for studies published until July 31, 2022. This search returned 952 results. After removing duplicates and irrelevant articles, 104 full-text articles were assessed for inclusion, and 10 met our inclusion criteria (see Figure 2 for exclusions).

Of articles that met our inclusion criteria (Table 1), 6/10 found that the association between executive control and poor mental health was stronger when executive control was assessed under acute stress, relative to when it was assessed under control conditions; 2/10 did not report whether the association significantly differed between conditions; and 2/10 reported that the association was not significantly stronger in the stress condition than in the control condition. Therefore, the majority of studies found that executive control under stress is a stronger predictor of psychopathology or other symptoms of poor mental health than executive control when assessed under nonstressful conditions.

When viewing the details of the reviewed studies, the reported effects do not appear specific to particular forms of psychopathology or particular types of executive control. Compared to a standard measure of executive control, executive control under stress was a significantly stronger predictor of the following outcomes in at least one study: depression, disordered eating, borderline personality disorder, attention deficit hyperactivity disorder, and general physical and mental health complaints. Similarly, all types of executive control assessed (e.g., updating, shifting, inhibition) showed the same trend of being more strongly related to psychopathology when measured under stress versus control conditions, suggesting that this increased strength of association is robust across different component executive processes.

How Might Worse Executive Control Under Acute Stress Contribute to Psychopathology?

The work described above offers support for the idea that relatively worse executive control under stress predicts higher levels of psychopathology. This work naturally leads to a question of mechanisms. Given the many ways that executive control can facilitate implementing skills and overriding tendencies, it may be that worse executive control under stress contributes to psychopathology in a variety of ways. A small but growing number of studies have examined correlates of executive control under stress with the goal of better understanding how executive control under stress may contribute to psychopathology, and we describe these studies below.

Executive Control Under Acute Stress and Reduced Ability to Carry Out Skills—Executive control contributes to the ability to implement a variety of skills. Worse executive control under conditions of stress may therefore contribute to less effective implementation of various skills, some of which are important to stress regulation within stressful contexts, which then contributes to the experience of psychopathology. As an example, a recent study found that relatively worse executive control under acute stress was associated with worse reappraisal ability (Quinn & Joormann, 2020). Similarly, relatively worse executive control under acute stress has been associated with greater stress-induced increases in negative affect, potentially reflecting poorer emotion regulation under stress (Shields et al., 2017). Thus, poorer executive control may contribute to reduced regulatory ability.

In addition, poorer executive control under stress may also contribute to psychopathology by impairing non-regulatory abilities. For example, a recent study (Bernstein et al., 2019) examined mnemonic discriminability (i.e., the ability to differentiate similar representations), which is thought to depend upon executive control and contribute to worry and anxiety via overgeneralized fear learning. When mnemonic discriminability was measured under conditions of stress, it was associated with higher levels of self-reported worry. Although speculative, when viewed in the context of the present review, this finding raises the possibility that poorer executive control under stress may impair mnemonic discriminability, leading to increased worry, thereby increasing vulnerability to psychopathology. Each of the reviewed studies suggests that acute stress-induced impairment in executive control may impair the ability to implement a relevant skill (e.g., reappraisal, mnemonic discriminability), which in turn could contribute to psychopathology.

Executive Control Under Acute Stress and Reduced Ability to Override Tendencies—In addition to reduced ability to implement skills, poorer executive control also contributes to reduced ability to override habitual responses or tendencies. It follows from this that poorer executive control under stress may contribute to reduced ability to override tendencies in stressful contexts, which then contributes to the experience of psychopathology. In support of this idea, we found that the relation between executive control under acute stress and depressive symptoms was moderated by the tendency to engage in rumination (Quinn & Joormann, 2015b). We interpret these results to mean that, for individuals with a tendency to ruminate, impairment in executive control under acute stress may result in an inability to override the tendency to ruminate, leading to high levels of rumination and associated risk for experiencing depression. Similarly, acute stress alters neural activity in ways consistent with a reduction in executive control (i.e., reducing BOLD activity within the dorsolateral prefrontal cortex, ventromedial prefrontal cortex, and anterior cingulate cortex) during presentation of food cues in individuals with binge eating disorder, and those changes in neural activity predict binge eating behavior in individuals' daily life (Fischer et al., 2017; Lyu & Jackson, 2016). Based on these findings, it may be that reduced executive control under stress impairs the ability to override the tendency or desire to eat when exposed to food cues. Each of the reviewed studies allows for the possibility that executive control under stress may contribute to an inability to effectively override a variety

of habits, like rumination or binge eating, that are opposed to one's long-term goals, which may then contribute to a variety of disorders.

Executive Control Under Stress and Intermediate Outcomes Related to Psychopathology—In addition to examining links between executive control under stress and the ability to implement skills or override tendencies, investigating potential consequences of worse executive control under stress that are intermediate to psychopathology may also provide information on the role of executive control under stress in psychopathology. For example, the finding reported previously that individuals with relatively worse executive control under acute stress had a stronger link between recent life stress exposure and poor health was mediated by perceptions of severity of recent life stressors (Shields et al., 2017). In other words, individuals with relatively worse executive control under stress perceived their recent life stress as more severe than others with better executive control who had experienced a similar number of stressors, and this explained the stronger association between stress exposure and health symptoms in those individuals.

In addition to perceptions of stress, acute stress-induced inflammatory response is another mechanism in the link between stress and psychopathology (Slavich, 2019; Slavich & Irwin, 2014) that demonstrates how executive control under stress may contribute to psychopathology. Some studies have investigated the possibility that relatively worse executive control under stress may contribute to relatively greater inflammatory responses to stress. For example, one study found that better executive control of emotional information following an acute emotional stressor was associated with smaller salivary cytokine (i.e., interleukin [IL]-1β, IL-6, and IL-8) responses to that emotional stressor (i.e., an aversive vs. control film (Shields, Kuchenbecker, et al., 2016). Another study found mixed results in associations between levels of cytokines (e.g., tumor necrosis factor [TNF]-α, IL-2) and a task measuring various components of attention, each of which were measured following a social evaluative stress induction (Maydych et al., 2018). Although the authors found relations between some cytokines and either "orienting efficiency" or "alerting efficiency" across both conditions, they did not find any associations with "executive efficiency," suggesting that executive control under stress was not related to cytokine response to stress in their study. Finally, a third study found that lower levels of executive control under stress were associated with greater increases in IL-6, although this effect was not found for two other proinflammatory cytokines, IL-1β and TNF-α (Quinn et al., 2020). Although these results are not conclusive, they highlight additional ways in which potential downstream effects of poorer executive control under stress may be examined in future research.

In sum, existing research is consistent with the possibility that poorer executive control under stress may contribute to phenomena like increased perceived stress and greater inflammatory response to stress, which can be considered outcomes intermediate to psychopathology. When combined with other evidence, we propose that executive control under stress leads to reduced ability to regulate initial stress responses (e.g., cognitive, physiological), leading to more pronounced or prolonged threat responses (e.g., increased inflammation or increased perceptions of stress levels), which could then contribute to psychopathology.

Tying It All Together: The Integrated Model of Stress, Executive Control, and Psychopathology

Based on the findings reviewed above, we propose what we term the *integrated model of stress, executive control, and psychopathology*, which indicates that the extent to which acute stress impairs executive control influences psychopathology risk, such that poorer executive control under stress confers relatively greater risk for developing psychopathology in response to stress. We suggest that executive control under stress is an observable trait. An important implication of our model is that individual differences in executive control under stress is a stronger, and therefore more useful, predictor of stress-related psychopathology than individual differences in executive control when it is assessed under non-stressful conditions. This model proposes not only that executive control under stress predicts psychopathology, but also that individual differences in the extent to which stress impairs executive control confers vulnerability to psychopathology. This model also explains, based on the many functions of executive control, how executive control under stress may contribute to psychopathology. In short, this model indicates that for some people, exposure to acute stress leads to greater executive control impairment, leading to impaired ability to override habits and impaired ability to carry out a variety of skills, which may then contribute to the emergence of psychopathology during or following periods of stress exposure.

The integrated model of stress, executive control, and psychopathology can be viewed as a moderated mediation model, wherein acute stress leads to reductions in executive control, which contributes to psychopathology through a variety of pathways (see Figure 3). Importantly, this stress-induced impairment in executive control occurs to a greater degree in some individuals than others, meaning that the extent to which stress contributes to psychopathology is dependent on factors that influence the extent to which stress impairs executive control. These moderating factors are not yet clearly delineated but may include factors such as genetic variation and dispositional beliefs (Plieger & Reuter, 2020; Shields, Sazma, et al., 2016; Tsai et al., 2019).

One mechanism by which poorer executive control under stress can contribute to psychopathology is a reduced ability to implement a variety of skills. This reduced ability can contribute to psychopathology by allowing acute stress responses to spiral into prolonged or sustained stress responses. For example, poorer executive control under stress could contribute to a reduced ability to implement reappraisal, causing the initial sadness elicited by the stressor to maintain, ultimately contributing to a depressive episode. A reduced ability to implement skills could also contribute to psychopathology via means that are unrelated to other aspects of the individual's stress response. For example, drawing from Bernstein and colleagues (2019), poorer executive control under stress could contribute to reduced mnemonic discriminability, leading to overgeneralized fear learning and a subsequent increase in worry and other symptoms of generalized anxiety disorder.

Poorer executive control under stress may also contribute to psychopathology via reduced ability to effectively override tendencies or habits. This pathway could also involve acute stress responses spiraling into sustained stress responses. For example, poorer

executive control under stress could contribute to an inability to stop ruminating, causing initial physiological activation to result in a prolonged inflammatory response, contributing to depression. A reduced ability to override tendencies could also contribute to psychopathology via means that are unrelated to other aspects of the individual's stress response. For example, poorer executive control under stress could contribute to a reduced ability to override the urge to engage in compulsions, leading to obsessive compulsive disorder. As these examples indicate, a goal of delineating this model is to demonstrate the many potential pathways to psychopathology resulting from poorer executive control under stress.

In addition to delineating pathways to psychopathology, this model suggests how poorer executive control under stress may serve as a transdiagnostic risk factor, and at the same time allows for the emergence of distinct disorders. The explanatory power of this model is possible because executive control influences such a wide array of abilities, meaning that individual differences in executive control can manifest in many different ways. How executive control manifests in a given individual is dependent on other factors, including an individual's environment, predispositions, and habits. Thus, if an individual experiences poorer executive control under stress, the particular effects of that deficit will also be dependent on other factors. For example, an individual's attributional style may influence whether a person responds to a stressful event with negative cognitions, whereas an individual's level of executive control under stress will influence the extent to which the negative cognitive response emerges and is maintained. In this way, individuals with poorer executive control under stress may be vulnerable to all forms of psychopathology, but whether a disorder emerges, which disorder emerges, and how many disorders emerge may be dependent on a host of other risk factors.

A New Measure for Psychopathology Research: Executive Control Under Stress

As described in detail above, our integrated model of stress, executive control, and psychopathology proposes that poorer executive control under stress confers risk for psychopathology. A notable methodological consequence of this proposal is that executive control under stress is a measurable individual difference that is distinct from standard measures of executive control and should be a better predictor of psychopathology than executive control measured under non-stressful conditions. In particular, our model suggests that by measuring executive control under conditions of stress, we can better understand how executive control impairments may contribute to psychopathology and better predict which individuals are vulnerable to psychopathology.

Based on our model, we propose an approach of using performance-based executive control tasks measured under conditions of acute stress in the lab in order to predict psychopathology. We believe this approach has a number of benefits. For example, this approach retains the benefits of controlled experimental designs (e.g., using validated stress inductions), rather than relying on individuals' assessment of their ability to use executive control in such contexts (e.g., using self-report questionnaires). Additionally, this approach entails using existing executive control tasks, which allows us to draw from research using these same executive control tasks. For example, we can draw from models

such as the unity/diversity framework (Miyake & Friedman, 2012; Miyake et al., 2000) to select validated tasks as well as to conceptualize the structure of executive control. Similarly, we can draw from knowledge of the neural correlates of executive control that have been gained using these same tasks (Perone et al., 2018) to better understand distinctions among components of executive control in relation to psychopathology. At the same time, our proposed approach differs from previous work examining the role of executive control in psychopathology by changing the context in which these tasks are administered, which improves ecological validity. This is because we propose administering the tasks in conditions (e.g., following an acute stress induction) that better approximate the conditions in real life that often precede the onset of psychopathology. To effectively measure executive control under stress, there are a number of methodological factors to consider (e.g., type of stressor and cognitive task); these considerations are the focus of a recent review (Shields, 2020). In short, our model suggests that poor executive control under stress may be a useful predictor of psychopathology, and this paradigm may thus be a useful methodological tool in clinical research.

Testable Predictions

Using the measures of executive control under stress described above, several testable predictions can be derived from our integrated model of stress, executive control, and psychopathology. The first prediction is that executive control under stress is a transdiagnostic risk factor. There is preliminary evidence for the proposed model; depression, anxiety, disordered eating, borderline personality disorder, attention deficit hyperactivity disorder, and general mental health complaints have all been linked to poorer executive control under stress (see Table 1). Attempts are needed to both replicate these studies and extend these studies by using measures of other disorders as outcomes. For example, does poorer executive control under stress increase vulnerability to obsessive compulsive disorder or bipolar disorder? Studies answering questions like this would be important tests of the proposed model.

A second prediction is that poorer executive control under stress plays a causal role in psychopathology through the many functions of executive control. Although tests of causal relations have not yet been conducted, correlational research examining constructs ranging from perceived stress severity (Shields et al., 2017) to binge eating behavior (Fischer et al., 2017; Lyu & Jackson, 2016) to rumination (Quinn & Joormann, 2015b), support the varied pathways of the model. Additional research is needed to examine both the breadth and direction of the pathways illustrating how poorer executive control under stress may contribute to psychopathology.

It should be noted that there are alternative interpretations to the existing findings that support our model, and that testing those alternative interpretations could furnish support for or against this model. For example, our model indicates that poorer executive control under stress contributes to an inability to override the tendency to ruminate, which contributes to psychopathology. It is also possible that frequent rumination in response to stress could both lead to increased risk for psychopathology and impair executive control due to the attentional demands of rumination. This perspective would then see greater impairments

in executive control as epiphenomenal, or somewhat of a side effect, of rumination. Importantly, though, this idea is testable. Rumination can be induced independently of an acute stressor (Ehring et al., 2009), and if executive control assessed following a rumination induction related to symptoms of psychopathology more strongly than executive control under stress, this could suggest that our model might be incorrectly or insufficiently conceptualizing the relations between stress, executive control, and rumination. Ultimately, such alternative theoretical conceptualizations provide fruitful opportunities for future research.

A third testable prediction is that this model can reconcile both the existence of high rates of comorbidity and the appearance of distinct disorders. Based on our model, we expect that poor executive control under stress contributes to the emergence of multiple disorders; thus, poor executive control under stress could contribute to the occurrence of comorbid diagnoses. At the same time, which disorder(s) emerges for a given individual is dependent on additional factors. In other words, poor executive control under stress can lead to distinct outcomes when other factors interact with executive control under stress to contribute to risk for specific disorders. This implies not only the existence of varied moderators but also varied mediators in the link between executive control under stress and psychopathology. For example, we expect that only individuals with a tendency to worry will experience high levels of worry when executive control is impaired by stress. For these individuals, impaired executive control under stress may contribute to high levels of worry, which then contributes to generalized anxiety disorder. Future research examining this model, will want to evaluate moderators (e.g., a tendency to worry) as well as distinct intermediate outcomes resulting from the interaction between the moderator and executive control under stress (e.g., worrying to a high degree), in order to understand how distinct disorders can emerge from a single transdiagnostic risk factor, executive control under stress.

Pressing Issues and Future Directions

We believe that the integrated model of stress, executive control, and psychopathology makes important strides in understanding why poor executive control represents risk for psychopathology as well as why executive control tends to be a stronger predictor of psychopathology when it is assessed under stressful conditions. To better understand this model and its implications for psychopathology research, several pressing issues merit discussion and future clarification.

Impairment in Which Aspect of Executive Control Contributes to Which Disorders?—Perhaps the most important of these pressing issues is to more closely examine which components of executive control are responsible for the link between executive control under stress and psychopathology. For example, is a relatively greater impairment in the common executive function under stress is a general risk factor for psychopathology? Do relatively greater impairments in particular executive functions predispose risk for distinct types of psychopathology? Snyder et al. (2015) suggested that a parsimonious explanation for the range of executive control impairments seen in psychopathology is that the common component of executive control may be a general psychopathology vulnerability factor, which has been proposed to explain high rates of

comorbidity among disorders (Caspi et al., 2014; Lahey et al., 2012). Extending this idea to our model raises the possibility that stress impairs the common component of executive control, which may predispose individuals to a range of disorders. Alternatively, individual predispositions to a relatively greater impairment in one executive function (e.g., updating) under stress but not another (e.g., shifting) may help to explain why the same experience of stress can lead to differing forms of psychopathology (e.g., disorders of impulse control vs. rigidity) in different individuals. Including multiple measures of executive control under stress and examining their association with symptoms of a range of disorders would help to answer this question.

How Is Executive Control Under Stress Related to Emotion-Related Impulsivity?—Executive control under stress appears similar to the construct of emotion-related impulsivity, which is often measured by self-report questionnaire and is described as the tendency to react reflexively in the face of negative or positive affect (Carver & Johnson, 2018). Recent studies have found that emotion-related impulsivity is associated with executive control, particularly measures of response inhibition measured under positive (Johnson et al., 2016) or negative mood inductions (Dekker & Johnson, 2018). Emotion-related impulsivity has been considered something that may contribute to many forms of psychopathology, and may actually predict a general factor of psychopathology (Berg et al., 2015; Carver et al., 2017). What emotion-related impulsivity and executive control under stress have in common is they appear to tap into the idea that contextual factors (i.e., stress or emotion) influence how people think and act. Of course, these constructs also differ in important ways. For example, emotion-related impulsivity focuses on the impact of emotional states and emphasizes the tendency to respond reflexively, whereas executive control under stress focuses on the influence of stress and emphasizes the extent to which individuals are able to override automatic responses. Despite these differences, we view these constructs as more similar than different, and we view the evidence for emotion-related impulsivity predicting psychopathology as consistent with our model that worse executive control under stress contributes to psychopathology. Additional research is needed to better understand how these two constructs overlap and diverge. In particular, examining how the common and unique elements of these constructs influence their ability to predict psychopathology will help refine our understanding of psychopathology vulnerability.

Why Do Certain Individuals Experience Greater Decline in Executive Control Under Conditions of Acute Stress?—Central to the proposed model is the idea that individuals differ in the extent to which executive control is impacted by stress exposure. A useful extension to this model would be to further investigate what contributes to these individual differences. Aspects of individual's emotional or physiological responses to stress may be a promising area to start, particularly where there are stable individual differences in these responses. For example, is the intensity of negative affect elicited by stress important in determining level of executive control under stress? Is level of arousal, regardless of valence, the key contributor to stress-induced impairment in executive control, and would high-arousal positive states impair executive control in the same way as stress? The various models explaining how executive control is impaired by stress offer promising starting points to answer questions such as these.

The neurobiological explanation for how stress impacts executive control indicates that increased subcortical activity resulting from stress exposure can inhibit prefrontal neuronal activity (e.g., through increased noradrenergic activity), manifesting as impairment in executive control (e.g., Arnsten, 2015; Heatherton & Wagner, 2011; Henckens et al., 2012; Hermans et al., 2014). The Yerkes Dodson law indicates that as arousal increases from moderate to high levels, performance on tasks that require executive control decreases. Both of these models suggest that as the physiological consequences of stress increases, executive control declines. This focus on physiological activation or arousal, rather than the valence of emotion experienced aligns with findings from emotion-related impulsivity indicating that impulsivity can be observed in both positive and negative affective states. It is important to keep in mind that these models provide explanations for how stress impairs executive control on average. Limited evidence exists on whether individual differences in physiological activation to a stressor may explain who experiences poorer executive control under conditions of stress. A recent study found that changes in arousal level during a response inhibition task did not uniformly predict trial-to-trial performance; instead, the relation between arousal and response inhibition varied as a function of emotion-related impulsivity (Pearlstein et al., 2019). This suggests that level of executive control does not merely reflect individual differences in arousal level. Instead, certain individuals may show more executive control impairment in the face of arousal.

Other theoretical perspectives on stress and executive control also may be useful for deriving hypotheses about when or in whom stress would impair executive control most. For example, the mood as information theory suggests that distinct types of negative affect may exert unique cognitive effects (Shields, Moons, et al., 2016) due to the information inherent in that affective state; however, individual differences in affective reactions have not been examined as predictors of the effects of stress on executive control. Limited resource theories of executive control suggest that individuals with greater extant cognitive load might show worse executive control under stress, and this prediction has been supported by some studies (e.g., Luettgau et al., 2018). Although these theories offer useful predictions about whom stress would impair most, additional research is needed to test such predictions and identify factors that predict individual differences in executive control under stress.

How Do Early, Chronic, or Cumulative Adversity Relate to This Model?— Exposure to forms of stress other than acute stress, such as chronic stress or early adversity, predict atypical acute stress responses (Carpenter et al., 2010; Chiang et al., 2017; Hostinar et al., 2015; Lam et al., 2019; McLaughlin et al., 2015; Sandner et al., 2020) and poorer executive control (Hunter & Shields, 2022; Hostinar et al., 2012; Johnson et al., 2021; Kim et al., 2013; Kira et al., 2020; Mani et al., 2013; Peters et al., 2019; Slavich & Shields, 2018). How these findings relate to our model, however, has not yet been appropriately addressed.

Although one study to date has found that recent perceived stress was associated with greater detrimental effects of stress on executive control (Luettgau et al., 2018), this result has not been observed in all studies on the topic (e.g., Shields et al., 2017, unpublished secondary analysis). Relatedly, one study has tested whether early adversity moderated links between psychopathology and executive control under stress (Kuehl et al., 2020); this study

found that depression and early adversity each moderated the effect of stress on executive control, but there was no interaction between depression and early adversity, indicating that the stronger link between executive control and depression as a function of stress was not further moderated by early adversity. In short, existing literature does not provide strong support for the idea that our proposed model dynamics are moderated by additional forms of life stress.

We suggest that an appropriate way to integrate prior work documenting links between life stressor exposure and psychopathology with our model may be to consider altered acute stressor effects on executive control as a potential mediator between prior life stressor exposure and poor mental health. For example, life stressors, especially threatening stressors experienced during childhood, may exacerbate some forms of acute stress reactivity (Lam et al., 2019; McLaughlin et al., 2014; Weissman et al., 2022). If stressor exposure during sensitive periods or cumulative stressor exposure exacerbates the detrimental effects of acute stress on executive control, our model would suggest that these stressor exposures may be linked to psychopathology in part through their modulatory effects on executive control under stress. This pattern of results would also help to clarify why some people experience greater declines in executive control under stress than others.

What Is the Role of Stress Recovery?—Although much of our model discussion has directly or indirectly focused on reactivity to acute stress, it is likely that aberrant stress recovery plays an important role in links between poorer executive control under stress and the development or maintenance of symptoms of psychopathology. The biological and cognitive effects of acute stress are time-dependent, but most nongenomic effects (e.g., cortisol levels) typically resolve within one to two hours, with many processes returning to pre-stress levels much sooner (Joëls et al., 2011; Shields, Sazma, et al., 2016)—though some genomic effects of stress or its biological sequelae on cognition can take multiple hours or more to resolve (Joëls et al., 2011; Henckens et al., 2011, 2012; Hermans et al., 2017).

Importantly, the length of time that it takes nongenomic effects of stress to resolve (i.e., stress recovery) is moderated by a number of factors (e.g., Maeda et al., 2017; Raymond et al., 2019), and these factors are likely to influence or contribute to our model dynamics. For example, as described above, poorer executive control can lead to greater rumination, and stress-induced increases in negative affect. Experimental work has further shown that both rumination and greater negative affect during stress in turn prolong and slow physiological recovery from stress (Capobianco et al., 2018; Radstaak et al., 2011). Slower recovery from stress entails that components of the stress response have more time to exert detrimental effects on biological systems and contribute to glucocorticoid receptor resistance (Silverman & Sternberg, 2012), which may then lead to psychopathology (Quinn et al., 2018; Slavich & Irwin, 2014). In short, worse stress recovery confers greater detrimental effects of each stressor exposure on mental and physical health. Because we expect poorer executive control under stress to confer poorer stress recovery (e.g., due to poorer emotion regulation and greater rumination), we expect poorer stress recovery to be one pathway through which poorer executive control under stress leads to the development or maintenance of psychopathology (see Figure 3). Directly testing these hypotheses, however, is a subject for future research.

How Does the Present Model Inform Intervention?—In addition to advancing our understanding of how poorer executive control may contribute to psychopathology, the present model also has implications for interventions intended to relieve or prevent psychopathology symptoms. This model suggests that rather than focusing on improving executive control measured under standard laboratory conditions, it may make sense to focus on improving the stability of executive control, such that it is less influenced by stressful conditions. There are a few ways that stability in executive control could be targeted.

First, it is worth examining whether existing training techniques of repeated practice of executive control tasks may make it easier to maintain executive control in a variety of conditions, including conditions of stress. Along these lines, one training study demonstrated reductions in emotion-related impulsivity following an adaptive cognitive training paradigm (Peckham & Johnson, 2020). However, another study found no evidence for cognitive training influencing emotion-related impulsivity (Peckham et al., 2021) and there is generally limited evidence for such cognitive training paradigms producing changes in nontrained targeted abilities (Simon et al., 2016). Alternatively, methods explicitly aimed at improving executive control under stress may be examined, such as training executive control within a stressful context. For example, executive control tasks could be paired with aversive stimuli or laboratory stress inductions, requiring individuals to engage executive control in a stressful context. Examining whether stability in executive control may be trained in these ways could provide new avenues for intervention.

Second, our model is compatible with existing practices in treatments like Cognitive Behavioral Therapy. Such treatments emphasize the need for clients to practice their therapy skills in their daily life. Drawing from the present model, we suggest that practice may make these skills more habitual and therefore less vulnerable to the impairing effects of stress on executive control. Alternatively, it is possible that attempts to improve executive control under stress—and/or the skills dependent on executive control—may be less effective for individuals who have relatively worse executive control under stress. It may be more beneficial to instead focus on improving beneficial skills that rely less on executive control. For example, if individuals experience greater declines in executive control under stress, it may be more beneficial to encourage them to seek social support during times of heightened stress, rather than attempting to improve their ability to use a cognitively demanding strategy like reappraisal during a stressful situation. In this case, knowing the extent to which an individual’s executive control is impacted by stress would be helpful in determining which strategies for dealing with stress are likely the most effective for that individual. Indeed, a new online skills-based behavioral intervention for individuals with high levels of emotion-related impulsivity demonstrates that strategies requiring low cognitive effort are effective at improving outcomes. This intervention has reduced aggression in individuals with bipolar disorder and high levels of emotion-related impulsivity (Johnson, Sandal, Zisser et al., 2020) as well as in individuals with high levels of both aggression and emotion-related impulsivity (Johnson, Zisser, Sandal et al., 2020). These studies demonstrate the feasibility of intervening to reduce impulsive behavior in high-emotion situations. Interventions like this one that focus on intervening to reduce impulsive behavior specifically in high-emotion

situations may be necessary given evidence of poorer treatment outcomes in individuals with high levels of emotion-related impulsivity (Peckham et al., 2019).

Limitations and Challenges

Although we believe the present model provides a useful framework for guiding research, there are limitations to this model that pose challenges for future research in this area. Our model is based on the premise that incorporating relevant context (i.e., stress) into measures is essential for understanding psychopathology vulnerability. We suggest measuring executive control under conditions of acute stress in a laboratory setting. This improves the ecological validity of our measures, and there is evidence that this provides a better measure of psychopathology vulnerability, compared to standard non-stress measures of executive control. However, laboratory stress inductions are not the same as stress encountered in daily life. This approach is still limited by the constraints of the laboratory setting, including lower ecological validity than would be obtained if measures were assessed in response to stressors naturally occurring in individuals' lives. As ambulatory assessment continues to advance, we hope that new methods can be used to test our model to improve assessments of executive control under stress.

Another challenge to research on executive control under stress is deciding which measures of executive control to use. Existing recommendations when studying psychopathology vulnerability are to use multiple validated measures of executive control in order to extract a common factor of executive control (Snyder et al., 2015). This approach is a challenge when measuring executive control under stress. The effects of stress on executive control decline in magnitude as the number of executive control tasks previously administered in a study increases (Shields, Sazma, et al., 2016), and stress appears to exert different effects on executive control as a function of cognitive fatigue (Shields, 2020). Possibly as a result of these findings, most studies examining the link between executive control under stress and psychopathology have relied on a single measure of executive control, making the time course of impairment in executive control under stress as it relates to psychopathology not fully delineated. Thus, we do not currently know the number of tasks nor the time frame in which these tasks could be administered and still be considered psychopathology-relevant measures of executive control under stress.

Finally, it is worth highlighting a few aspects of our model that could use clarification by future studies. First, the evidence for executive control under stress as a transdiagnostic factor is preliminary. Additional research is needed to fully support this claim. Second, we focus on level of executive control under stress, but it is unclear whether absolute level of executive control under stress or greater within-person decline in executive control under stress is more predictive of psychopathology. Third, there is ambiguity on whether individual differences in level of executive control under stress (e.g., greater stress reactivity) or individual differences in maintenance of executive control impairment under stress (e.g., poorer stress recovery) is most relevant. For example, it is possible that rather than experiencing greater executive control impairment, vulnerable individuals experience a typical level of impairment that lasts for a longer period of time and is thus observed as greater impairment in executive control when it is measured at a single time point following

a stress induction. As research in this area advances, it will become useful to resolve these ambiguities.

Summary and Conclusion

Executive control (i.e., internally driven, top-down control of cognition and behavior) has long been known as important for daily life. Thought to be composed of common and unique components, executive control has been associated with outcomes as varied as career success, marital satisfaction, longevity, and psychopathology (Amirian et al., 2010; A. Diamond, 2013). To better understand how executive control may contribute to psychopathology, we surveyed evidence that stress impairs executive control, as well as emerging evidence that (1) executive control assessed under stressful conditions is a better predictor of psychopathology than executive control assessed under non-stressful conditions, and (2) poorer executive control under stress may exert these effects through a reduced ability to carry out skills and a reduced ability to override tendencies. Based upon these findings, we proposed the *integrated model of stress, executive control, and psychopathology*, which sees poorer executive control under stress contributing to stress-related psychopathology via reduced abilities to execute skills (e.g., reappraisal) and override tendencies (e.g., rumination and other maladaptive habits). Although there is evidence supporting this model, more research is needed to test its predictions and compare the predictive power of executive control under stress to other measures like emotion-related impulsivity. In short, the research reviewed here provides an important theoretical account of how stress and poor executive control jointly confer risk for psychopathology.

Our model extends prior theory on the role of executive control in psychopathology by considering executive control within the context of stress. This specific contextual element is crucial because there is no doubt that stress can proximally contribute to psychopathology. Despite the wealth of excellent research on the topic, why two people can experience the same stressor and yet only one develops psychopathology remains a vexing question. Part of the difficulty in answering this question is that there are many factors that can contribute to divergent outcomes following stress exposure. Our integrated model of stress, executive control, and psychopathology addresses that difficulty and, in doing so, helps answer the question of who is vulnerable to psychopathology following stress exposure. Our model can do this because executive control plays a critical role in so many abilities, including those that shape stress reactivity and recovery. Poor executive control under stress may thus confer a global vulnerability to stress—more so than individual abilities or components of stress reactivity or recovery. In presenting this model, our hope is to enhance understanding of stress-related vulnerability to psychopathology, improve identification of individuals vulnerable to developing psychopathology, and provide key action points for future research in this area.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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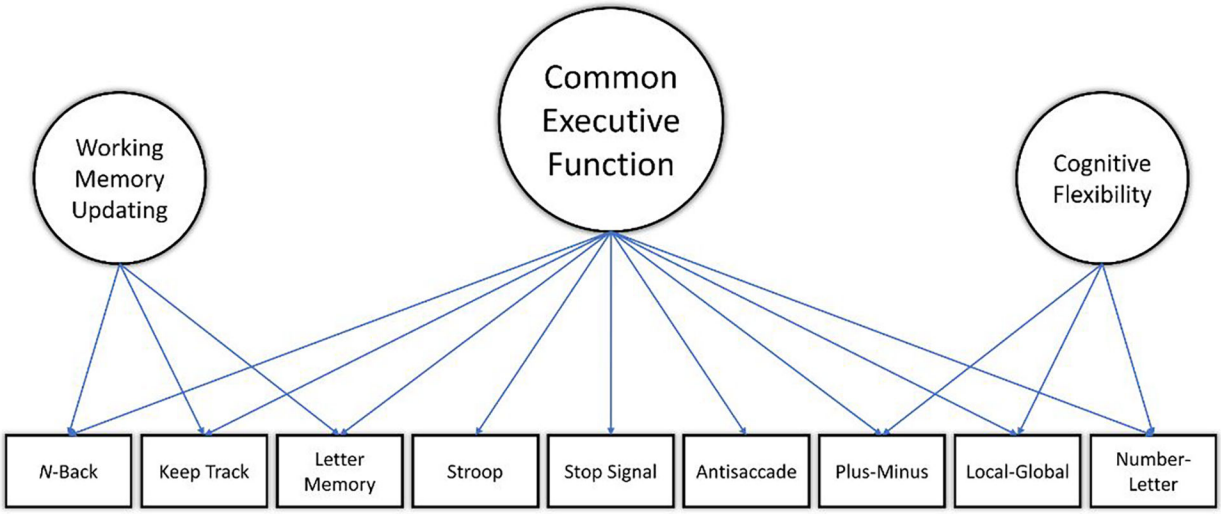

Figure 1.
The unity/diversity framework of executive control (Conceptually adapted from Friedman & Miyake, 2017).

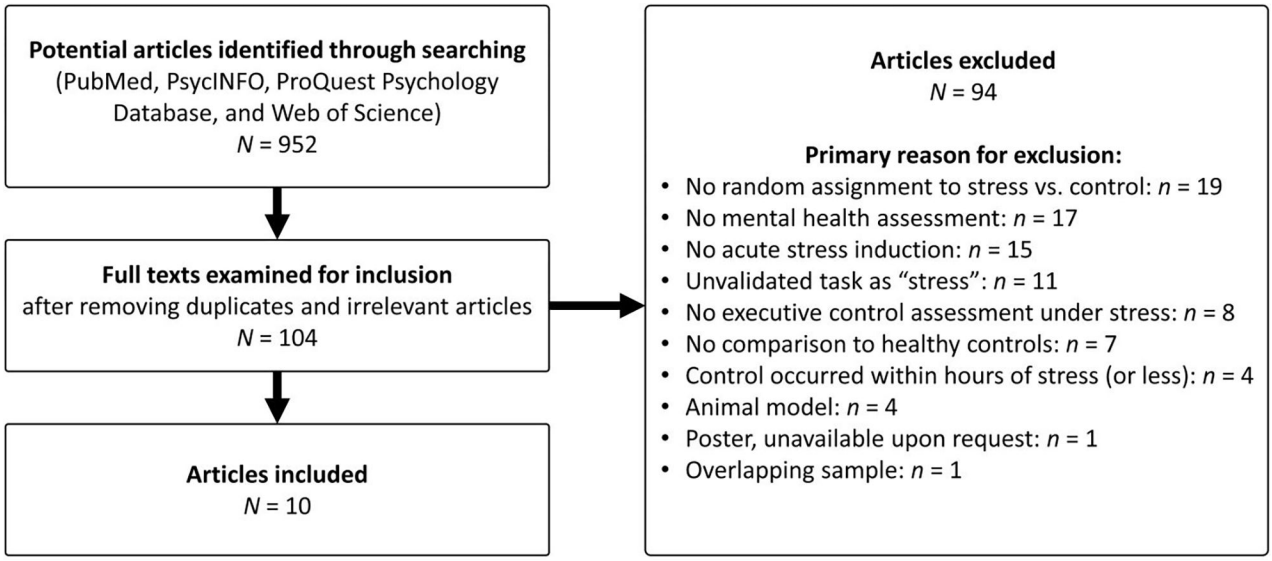

Figure 2.

Results of the systematic literature review. Ten studies met our inclusion criteria and are described in Table 1.

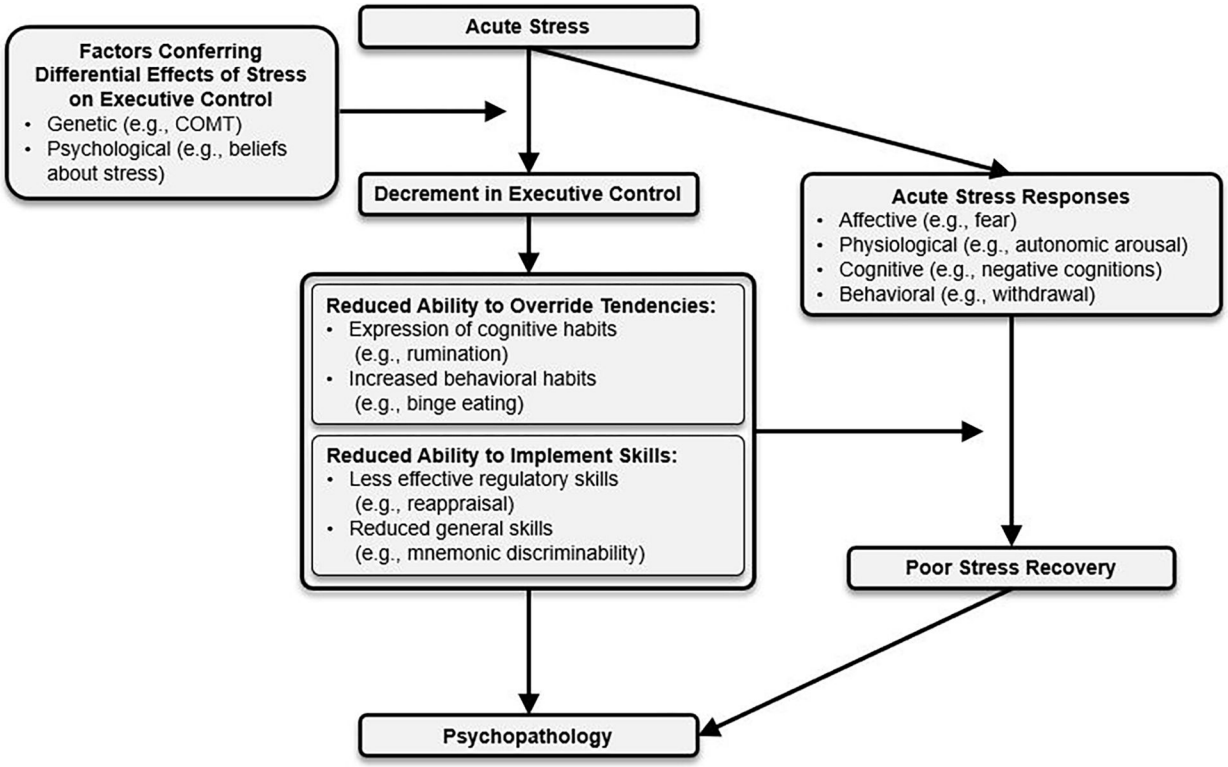

Figure 3.

The integrated model of stress, executive control, and psychopathology.

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Table 1.

Studies that met inclusion criteria for consideration in our systematic review.

Study	Mental Health Focus	Sample Size	Stressor	Stressor Study Design	Executive Control Task(s)	Executive Control Type	Sample Type	Prospective Link to Health?	Effect	Significantly Stronger Link Between EC and Mental Health Under Stress vs. Control?
Quinn & Joormann (2020)	Depression	UN, *n*=66	TSST	Between-Subjects	Emotional *N*-Back	Updating	Nonclinical	Prospective & Cross-Sectional	Poorer EC under stress predicted higher future depressive symptoms; EC assessed in a control condition did not predict depressive symptoms; the effect significantly differed by condition	**Yes**
Kuehl et al. (2020)	Depression	MDD, *n*=84 HC, *n*=59	TSST	Within-Subjects Crossover	Trail-Making Test, Digit Span (back), d2 Test of Attention	Multiple	Clinical	Cross-Sectional	In MDD, EC under stress was sig. worse than EC assessed in a control condition; in HC, EC under stress and EC did not differ; these effects sig. differed by condition in 3/4 EC outcomes	**Yes**
Barthel et al. (2022)	Anxiety	UN, *n*=112	TSST	Between-Subjects	Task-Switching	Shifting	Nonclinical	Cross-Sectional	No report of whether stress condition moderated the relation between task switching and trait anxiety	Unreported
Hood et al. (2015)	Anxiety	UN, *n*=103	CPT (forehead)	Between-Subjects	Letter-Number Sequencing, Digit Span	Updating	Nonclinical	Cross-Sectional	EC was significantly related to anxiety symptoms in the stress condition but not in the control condition; the test of difference between conditions not reported	Unreported
Westwater et al. (2021)	Disordered Eating	AN-BP, *n*=22 BN, *n*=33 HC, *n*=30	Mental arithmetic	Within-Subjects Crossover	Anticipatory Stop Signal	Inhibition	Clinical	Cross-Sectional	Group differences in EC were not sig. impacted by stress, but prefrontal regions showed differential modulation by stress between groups	**No**
Lyu & Jackson (2016)	Disordered Eating	BED, *n*=18 HC, *n*=26	CPT	Between-Subjects	Chocolate Consumption	Global	Clinical	Cross-Sectional	Hot EC (chocolate consumption) was sig. worse for BED under stress compared to hot EC for HC under stress and both groups in control conditions	**Yes**
Krause-Utz et al. (2016)	BPD; ADHD	BPD, *n*=30 ADHD, *n*=28 HC, *n*=30	MMST	Within-Subjects Crossover	IMT, Go/Stop	Inhibition	Clinical	Cross-Sectional	Relative to HC, BPD and ADHD had greater IMT impairment under stress; Relative to HC, ADHD had greater IMT impairment under control conditions; sig. group by	**Yes**

Study	Mental Health Focus	Sample Size	Stressor	Stressor Study Design	Executive Control Task(s)	Executive Control Type	Sample Type	Prospective Link to Health?	Effect	Significantly Stronger Link Between EC and Mental Health Under Stress vs. Control?
									condition interaction for IMT; no group, condition, or interaction effects for Go/Stop	
Cackowski et al. (2014)	BPD	BPD, *n*=31 HC, *n*=30	MMST	Within-Subjects Crossover	Go/Stop	Inhibition	Clinical	Cross-Sectional	Relative to HC, BPD had sig. worse EC under stress; EC under control conditions did not differ between BPD and HC; sig. group by condition interaction	**Yes**
Duesenberg et al. (2019)	BPD	BPD, *n*=49 HC *n*=49	TSST	Within-Subjects Crossover	Word Suppression Test	Updating/ Inhibition	Clinical	Cross-Sectional	EC was worse in BPD than HC; the effect did not differ by stress vs. control condition	**No**
Shields et al. (2017)	Mental and Physical Health Complaints	UN, *n*=110	TSST-G	Between-Subjects	Wisconsin Card Sorting Test	Shifting	Nonclinical	Cross-Sectional	Better EC under stress was associated with attenuated link between life stress & health complaints; no relation in the control condition; this relation sig. differed across stress/control conditions	**Yes**

Note: EC = Executive Control; UN = Unselected for psychopathology, not necessarily healthy (e.g., undergraduates); HC = Healthy Controls; MDD = Major Depressive Disorder; AN-BP = Anorexia Nervosa, Binge-Purge Subtype; BN = Bulimia Nervosa; BED = Binge Eating Disorder; BPD = Borderline Personality Disorder; ADHD = Attention-Deficit/Hyperactivity Disorder; TSST = Trier Social Stress Test; CPT = Cold Pressor Test; MMST = Mannheim Multimodal Stress Test; -G = for Groups; IMT = Immediate Memory Task; sig. = significant or significantly.