

Ecological, Biological, and Developmental Studies of Self-Regulation and Stress

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INTRODUCTION

In recent decades, important new research in the social and biological sciences has shed light on the negative consequences of social adversity and biological stress. These effects can include physical health outcomes like cardiovascular disease and mental health outcomes like chronic depression. This is highlighted in the work of Felitti et al. [1] who assessed seven types of adverse childhood experiences (ACES), including child maltreatment, domestic violence, and having a family member with mental health problems in a sample of over 13,000 middle-aged adults. Experiencing more ACES (particularly 4 or more) dramatically increased the odds of an individual being a current smoker, being obese, experiencing depression, having a history of attempted suicide, being an alcoholic, and having used illicit drugs. Beyond identifying long-term outcomes like these, research has linked stress—especially stress experienced in childhood and adolescence—to physiological changes in the brain, and to disruptions in development [2]. These findings have increased attention to the connections between a person’s environment in early life (that is, the period through early adulthood), and his or her social, emotional, economic, and physical health and productivity across the life course.

It is important to note that the impact of different types of stressors on any child or youth may vary considerably based upon the context or environment as well as an individual’s biology, genetics, and temperament [3]. As described in the first report in this series entitled *Self-Regulation and Toxic Stress: Foundations for Understanding Self-Regulation from an Applied Developmental Perspective*, the act of self-regulating is dependent on ongoing and specific environmental and contextual supports as well as factors that are individual to a child or youth. In particular, caregiver support including warmth, responsiveness, and scaffolding is believed to serve a protective or buffering role for the impact of stressors on children [4]. Other environmental supports or interventions may serve a similar role. In addition, each child’s individual characteristics and temperament, which are defined to some extent by genetics [5], likely contribute to their responsiveness to stressful conditions. It is also important to consider the impact of stress at different developmental stages, given that self-regulation skills develop across an extended period from birth through early adulthood. Thus, the present review will assess ecological, biological, and developmental studies of self-regulation and stress.

The goal of this report is to clarify how stress may impact the development of self-regulation. We will first operationalize stress and self-regulation as they are relevant to our empirical literature review, drawing from research across several scientific disciplines. Then we will discuss different perspectives of how stress and self-regulation interact and influence each other. From this foundation, we will identify important questions in the literature that we attempt to address with a methodical and comprehensive empirical review of human and nonhuman studies of stress and self-regulation. Following a summary of key findings, our report concludes with limitations of the current literature in this area and implications for interventions, the topic of the next report in this series.

Stress

In considering the link between stress and development, it is important to distinguish between acute and chronic stress. **Acute stress** involves the body's stress system activating for a short period of time in response to a temporary stimulus. Although such stress can have lasting biological or behavioral effects if it is severe enough, the human stress response system is generally well-equipped to manage acute stress. In contrast, **chronic stress**—in which the body's stress system is activated very frequently or for a prolonged period of time or in response to persistent stimuli—may have detrimental effects on the brain and behavior. When a child experiences strong, frequent, and/or prolonged adversity that overwhelms his/her skills or support, the result can create **toxic stress** response [2]. Stressors that may induce toxic stress responses include physical or emotional abuse, chronic neglect, caregiver substance abuse or mental illness, exposure to violence, and/or the accumulated burdens of family economic hardship (i.e., poverty). The word “**trauma**” describes an event or experience where an individual's life or physical wellbeing (or that of someone important to them) is threatened. Trauma can be either acute (such as a natural disaster or robbery) or chronic (such as child maltreatment). In this regard, it can be considered a stressor, which may create toxic stress in those situations where the child or youth's abilities to cope are overwhelmed. In addition, the aftermath of an acute trauma (for example, sustained homelessness or disruption of social networks after a natural disaster) can itself constitute a chronic stressor; in that way, even acute trauma can have chronic effects if consequences are long-lasting.

Understanding the impact of stress also requires consideration of the physiological principle of **allostasis**. Allostasis describes processes by which the body deploys resources to maintain “stability through change,” or in other words to withstand disruptions. One physiological system that plays a central role in allostasis is the **hypothalamic-pituitary-adrenergic (HPA) axis**, which manages the body's “fight or flight” response. When the body encounters negative conditions in the environment—for example, a prolonged deprivation of calories or the presence of perceived threats—this system comes online to try to redirect physiological resources toward enduring these challenges in a way that will minimize the long term biological consequences. Hormones are secreted that drive the release of sugar into the blood stream to provide muscles with a short-term energy boost, even if that comes at the expense of digestion, growth, bone and tissue repair, and other physiological processes that may be important in the long run, but are not immediately necessary to address the pressing challenge. In this sense, the system is an evolutionary analogue to an individual's or family's more conscious response to acute or prolonged economic challenge—when faced with a sudden job loss, for example, individuals or families might redirect their resources to pay their rent and other expenses in order to get through the period of immediate difficulty, even if doing so comes at the expense of future-oriented activities like saving or paying for school.

In a healthy person, levels of stress hormones rise sharply in the face of an acute challenge, which is known as a **stressor**, and then decline sharply shortly after the challenge has passed. However, this sharp up-then-down trajectory can be disrupted if exposure to stressors happens too frequently or goes on too long—in that case, the stress system is **hyper-stimulated**. Chronic exposure to stressors can

mean prolonged hyper-stimulation of the stress system, which eventually leads to high concentrations of stress hormones even without any immediately accompanying stressor or sense of arousal [6-8]. This chronic hyper-stimulation is associated with adverse conditions in childhood and adolescence. Additionally, chronic hyper-stimulation of the stress system has chemically toxic effects on brain regions involved with planning, with the orientation of behavior toward a goal, and with cognitive control of behavior [9]. Conversely, this same stimulation enhances the functioning of brain regions involved with emotional or affective response [9-12]. One characteristic that appears to predict whether a stressor will produce a toxic stress response is its degree of controllability and predictability—adverse conditions that are unpredictable, or over which individuals have insufficient control to ameliorate them, may be especially toxic [13, 14].

Self-Regulation and Its Development

A comprehensive conceptualization of self-regulation from an applied perspective is provided in our first report, *Foundations for Understanding Self-Regulation from an Applied Developmental Perspective*, but it is useful to briefly review our definition and key principles here. The definition of self-regulation used in this series of reports is: the act of managing cognition and emotion, which enables goal-directed actions such as organizing behavior, controlling impulses, and solving problems constructively.

Self-Regulation is the act of managing cognition and emotion, which enables goal-directed actions such as organizing behavior, controlling impulses, and solving problems constructively.

In our integrative model, self-regulation is conceptualized as comprising three overlapping domains: cognitive, emotional, and behavioral. **Cognitive self-regulation** includes effortful attentional control and executive functions (EF) such as working memory, cognitive flexibility or mental shifting, planning, and inhibitory control. **Emotional self-regulation** involves intentional processes to manage strong and unpleasant feelings, which can include cognitive regulatory processes such as attention shifting and re-appraisal. Indeed, the integration of cognitive and emotional self-regulation is an important developmental task necessary for behavioral regulation. **Behavioral self-regulation** includes delay of gratification, persistence, control of impulses, and goal-oriented behaviors. Again, these constructs and their inter-relations are described further in the Foundations report.

There are also physiological underpinnings (e.g., brain structure and function) of self-regulation that warrant addressing briefly as they have been examined in biological studies relevant to this review. Self-regulation may be characterized as a reflection of the interactions between brain regions that process emotion or sensations (the “hotter” aspects of reasoning and behavior), and those that play an executive role managing processes involved in planning (the “cooler” aspects) [15, 16]. Many of the “hot” aspects of cognition, for example, might be coarsely understood to occur primarily in some structures of the brain—for example, the amygdala is an almond shaped structure in the core of the brain that plays a critical role in processing emotions and reading emotional cues in others [17]. By the same coarse mapping, many of the “cooler” aspects of cognition can be characterized as more localized in other regions—for example, the prefrontal cortex, which plays a central role in managing “executive

functions,” which are considered to be an important aspect of cognitive self-regulation. The development of brain regions that drive many of the “hotter” aspects of decision-making (like the amygdala) follows a substantially different trajectory from birth to young adulthood, compared with the developmental trajectory of the parts involved in “cooler” aspects (like the prefrontal cortex). To the extent that self-regulation can be viewed as operating where “hot” and “cool” interact, it is not surprising that these different trajectories have important implications in the development of self-regulation [18].

The development of self-regulation can best be conceptualized from a cross-disciplinary perspective as described further in our Foundations report. To summarize briefly, there is reason to believe that self-regulation develops over an extended period from birth through young adulthood (and beyond). Although self-regulation can look very different at different ages, there is a pattern of development across cognitive, emotional, and behavioral domains in which skills build upon each other and become more complex over time as environmental demands and expectations requiring self-regulation increase. Second, there is evidence that self-regulation can be strengthened and taught like literacy, with continuous structure, support, instruction, and reinforcement or scaffolding provided from the environment. Although skills may develop earlier in environments with stronger foundations of support, all children have the capacity to develop these skills with effective instruction, suggesting multiple opportunities for intervention across development. Finally, self-regulation in children is conceptualized as being dependent on “co-regulation” provided by parents or other caregiving adults. That is, an interactional process in which a caregiver (i.e., parent or teacher) provides support, scaffolding, and modeling that facilitates a child’s ability to understand, express, and modulate feelings, thoughts, and behavior. More specific examples of these principles of self-regulation development are provided in our Foundations report, along with detailed descriptions of self-regulation across different ages.

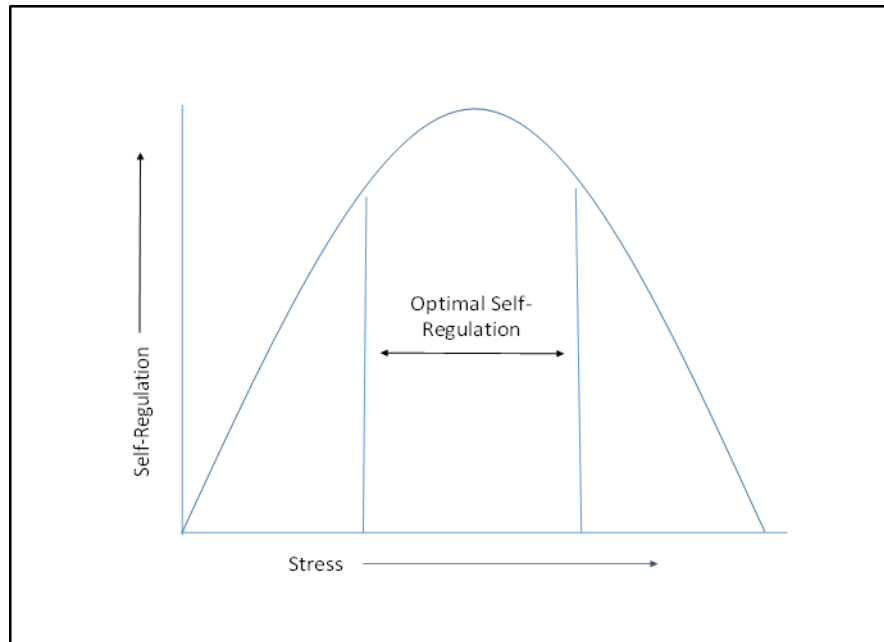
Relationship between Stress and Self-Regulation

Stress and self-regulation have been studied at length by scientists across such diverse disciplines such as neuroscience, psychology, economics, sociology, epidemiology, and population science. More recently, studies have begun to examine the intersection of these different theoretical and empirical approaches, enriching our understanding of how stress and self-regulation interact. Chronic exposure to stressful events, such as is typically experienced when children live in poverty, has been linked to difficulties in self-regulation development [19]. In addition, adverse childhood experiences such as abuse and neglect have been associated with impaired inhibitory control and delay of gratification, excessive or blunted emotional reactions, and impulsive, disorganized thinking [20, 21], which reflect self-regulation difficulties. In sum, stress is believed to impact underlying neurobiological processes of self-regulation as well as cognitive, affective, and behavioral aspects of self-regulation.

Some suggest that stress depletes self-regulation capacity as exertion depletes the function of a muscle [22]. However, there are data that contradict this claim and suggest that depletion may have more limited effects than initially thought [23]. A model that is well-accepted in the child development literature is that stress and self-regulation have a curvilinear relationship (like an inverted U). As can be seen in Figure 1 below, some stress may increase arousal, focus, and goal-orientation in a way that

enhances self-regulation, while too much stress may decrease it. Indeed, mild and intermittent stress within a child's abilities to cope might actually support the development of self-regulation skills. However, as the level, intensity, or duration of stressors exceeds a child's coping skills and support, self-regulation is expected to decrease. In addition, environmental factors are believed to play an important role in an individual's response to stress. That is, stress reactivity could enhance development in supportive contexts [24, 25] but in higher risk environments without support, could lead to negative developmental outcomes [25].

Figure 1. A Curvilinear Relationship between Stress and Self-Regulation



In this model, stress could enhance development in supportive contexts, but may be more likely to lead to negative developmental outcomes in higher risk environments without support. Another view suggests that being more reactive and less reflective (which we might think of as poorer self-regulation) may actually be adaptive in impoverished or conflictual environments [26]. These varied conceptual views highlight the need for the kind of critical review of empirical data on the relationship between stress and self-regulation that we provide in the present report.

Chronic and acute stressors may impact children's development and particularly their development of self-regulation in several ways. It may be that there are particular periods of development during which such stress has stronger or more lasting effects due to the concurrent development of brain circuits that are critical for self-regulation. Although it is now recognized that this remodeling of brain architecture, often referred to as **developmental plasticity**, occurs well into a person's 20s

Developmental Plasticity

refers to changes in neural connections during development as a result of environmental factors as well as learning.

[27], it is possible that one's developmental trajectory may be disrupted to a greater extent by toxic stress if it occurs during a time when developmental plasticity is at its greatest (i.e., preschool, adolescence). This question will be examined in our literature review.

Another way that stress may impact self-regulation development is through what is known as the **psychology of scarcity** [28,29]. In this line of inquiry at the intersection of psychology and economics, conditions of scarcity (which could be a lack of time, money, food, or even companionship) interfere with self-regulation because of the "mental bandwidth" (i.e., the mental energy required to deal with a situation) consumed by these pressing concerns [28]. When preoccupied with scarcity, individuals have fewer cognitive resources for planning and problem-solving, which results in a lack of attention to other important things and short-sighted decision-making [29]. Indeed, performance on cognitive tests declines under conditions of scarcity that are manipulated experimentally as well as under natural experiments where individuals' wealth varies at different times of the year [29]. One implication of this research is that individuals who appear to struggle with self-regulation under conditions of scarcity (e.g., living in poverty) may be just as capable as others when they are not distracted by scarcity [28]. For children, this also implies that self-regulation development may be delayed under conditions of scarcity or chronic stress.

Important Unanswered Questions

Several questions related to the impact of toxic stress on self-regulation warrant greater empirical scrutiny:

- How does stress impact self-regulation development and capacity? Are these effects long-term? Can these effects be reversed?
- Are there particular periods of development when self-regulation development is more or less vulnerable to stress?
- How do individual and environmental differences moderate the impact of stress on self-regulation?
- What contextual protective factors may mitigate the impact of stress on self-regulation?

Although our ability to answer these questions in our review is limited by the existing literature, identifying strengths and gaps in the evidence base is important for setting a focused research agenda for the future. What we do learn can also help identify promising intervention approaches and inform policy to interrupt the life-cycle effects of social and economic adversity.

PROCEDURE FOR EMPIRICAL LITERATURE REVIEW

We indexed published peer-reviewed studies that include search terms corresponding to stress-related constructs and to self-regulation-related constructs. In identifying what counts as stress-related and self-regulation-related constructs, we used the conceptual framework laid out in the first report in this series. Our review of the empirical literature includes terms aimed at uncovering studies that explore

self-regulation's relationships with stressors like socioeconomic deprivation, but also with physiological indicators of stress itself, like the hormones managed by the hypothalamic-pituitary-adrenergic axis.

The specific search terms, and the publication databases to which we took those terms, were chosen so that empirical studies with the proper constructs would be indexed across ecological, biological, and developmental studies. Our search terms were defined very broadly in order to reduce the risk of missing important studies that might use slightly different terms for relevant constructs. In order to manage the enormous volume of hits, we also included explicit terms identifying irrelevant applications of the search terms, and specifically excluded papers containing such applications from our indexed results.

Search Terms

We made two lists of search terms (see Appendix A) —the first list comprises stress-related predictors, outcomes and markers; the second, self-regulation. Examples of terms on the stress list include:

- Direct references to stress. These include “stress,” “stressor,” “allostasis,” and “allostatic.”
- Physiological correlates of stress. These include the names of specific stress hormones (including cortisol, adrenocorticotropic hormone or ACTH,) and other terms that refer to the biochemical stress response (e.g., hypothalamic, adrenergic, HPA).
- Family factors related to potential stressors. These include terms such as “maltreatment,” approaches to “discipline,” and socioeconomic variation in “parenting” styles, and so on.
- Ecological factors related to potential stressors. These include “adverse childhood experiences,” “psychology of scarcity,” “neighborhood” characteristics, “poverty,” or “exposure to violence,” for example.

On the self-regulation list, we included:

- Direct references to self-regulation. These include the terms “self-regulation,” “self-control,” “emotion regulation,” “behavior regulation,” “effortful control,” “executive function,” “cognitive flexibility,” “cognitive control,” “working memory,” and “behavioral inhibition.”
- Psychological or neurocognitive indicators of self-regulation such as “impulsivity,” “sensation seeking,” and “attentional control”.
- Behavioral correlates of self-regulation or of deficits in self-regulation. These include terms such as “problem behaviors,” “time inconsistency,” and “delayed reward”.
- Physiological correlates of self-regulation. These include specific brain regions involved in emotional responses, and regions involved in controlling those responses (e.g., amygdala, orbitofrontal cortex, prefrontal cortex). They also include references to neural architecture used when these regions interact (e.g., white matter).

Search Procedure

We took these search terms to databases that cover the English-language research literature from a number of the relevant academic disciplines, and indexed all recent articles during the five year period between 2009 and 2013 that included at least one term from our stress list and one term from our self-

regulation list. We established a 5 year time horizon because the literature is cumulative; the important insights from longer than 5 years ago are likely to have been built into more recent research. There were also practical reasons for this decision; over this time horizon, about 7500 peer-reviewed articles have appeared in print.

A detailed inclusion/exclusion list is also provided in Appendix A. In order not to distort the picture presented by the literature, we tried to use algorithms—rather than case-by-case judgments—to exclude any articles that had inappropriate uses of our search terms (e.g., papers about “oxidative stress,” which is an entirely separate thing from toxic stress in the sense we are investigating, were excluded). We also excluded studies that were not empirical, and studies relevant only to adult populations, to very specific subpopulations, or very specific stressful circumstances (e.g., traumatized combat veterans, or those living with a terminal illness). Due to human resource constraints, we also excluded articles that were not written in English. We also excluded dissertations. We did not allow our own personal judgments of research quality to affect whether a study was included in this review, although we will comment on methodological limitations of the studies in our discussion.

HOW AND WHY WE INCLUDED PSYCHIATRIC DISORDERS DEFINED BY STRESS AND DYSREGULATION

It is also worth noting how we handled two important, specific clinical conditions that were encountered in studies identified in our initial search for this report: Post-Traumatic Stress Disorder (PTSD) and Attention-Deficit/Hyperactivity Disorder (ADHD). Although these psychiatric disorders are defined to a large extent by difficulties in self-regulation, much of the vast clinical literature is not directly relevant to our questions about the impact of stress on the development of self-regulation. **Post-Traumatic Stress Disorder (PTSD)** is a psychiatric condition in which an individual experiences a constellation of impairing clinical symptoms related to an experience of traumatic stress. Our literature search uncovered many studies of this condition, but given our research questions for this paper, we included only those studies that investigated how cognitive and emotional processes interact in individuals with PTSD, especially as compared with the general population. We excluded studies that investigated physiological or genetic predictors of PTSD and studies of interventions for PTSD, since these do not directly address the impact of stress on the development of self-regulation.

We were similarly focused in addressing studies of **Attention-Deficit/Hyperactivity Disorder (ADHD)**, a psychiatric disorder beginning in childhood which is characterized by developmentally-inappropriate levels of inattention, hyperactivity, and impulsivity. We included only studies of ADHD that focused on relationships between stressors or the body’s stress system and the symptoms or severity of this condition because this information may have application beyond a specific clinical sample. Including carefully selected studies of individuals with what may be considered disorders of self-regulation is also helpful in expanding our understanding of extreme effects of stress (in the case of PTSD) and of individual differences in stress reactivity (in the case of ADHD).

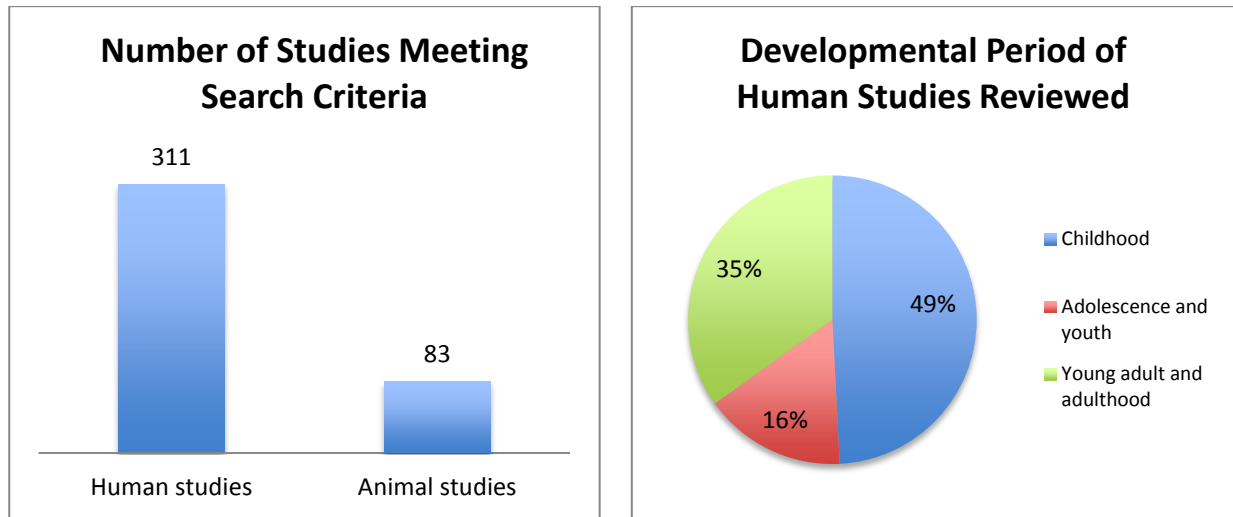
HOW AND WHY WE INCLUDED ANIMAL STUDIES

Animal studies offer great promise in identifying the mechanisms linking stress to self-regulation outcomes, since stress can be experimentally manipulated more extensively in laboratory animals than in humans, and more invasive biological measurement can be done in animals. In fact, many of the empirical studies of stress and self-regulation from cognitive psychology and neuroscience literatures have been conducted in rodents or non-human primates. It is possible to learn a great deal from these experiments, because mammals share very similar stress response systems. Furthermore, while rodent brain development moves much faster and rodent brains are much simpler, it is possible to map important parts of rats' developmental pathway onto stages of human development. For example, the developmental changes in brain and biology during the second month in the life of a standard lab rat are similar in many important ways to developmental changes experienced over a much longer period by human adolescents. Similarly, repeated or prolonged application of stressors in rats can lead to chronic hyperstimulation of their stress systems in the same way that the human stress system can become dysregulated from chronic exposure to stressors. Since laboratory animals live shorter lives and have simpler brains and stress systems, it does not take as long to generate toxic effects of chronic stress in an animal, compared with a human. For example, exposing a rat to repeated "footshock stress" (see Box 1.1) for just a month would be roughly equivalent to repeated stressor-exposure in a human for all the years of adolescence. We recognize that there are many limitations to generalizing these findings to humans, which we will address in discussing our findings and drawing conclusions.

FINDINGS

Of the original 7500 studies identified, 394 empirical studies met our full inclusion criteria, i.e. both a term reflecting a stress-related predictor, marker, or outcome and a term reflecting self-regulation with no irrelevant application of these terms or irrelevant application in clinical disorders. As seen in Figure 2 below, 311 of these studies (79%) were human studies. Of those, approximately half were studies focused primarily on childhood (any age below adolescence), over a third included adults, and only 16% were focused on adolescence (see Figure 3). It should be noted that these periods were defined by the age at which self-regulation was measured, and many studies actually assessed self-regulation across development. No specific age thresholds were used for categorization; rather we relied upon the authors' descriptions of their participants.

Figures 2 and 3. Number of Studies and Developmental Period of Human Studies Reviewed

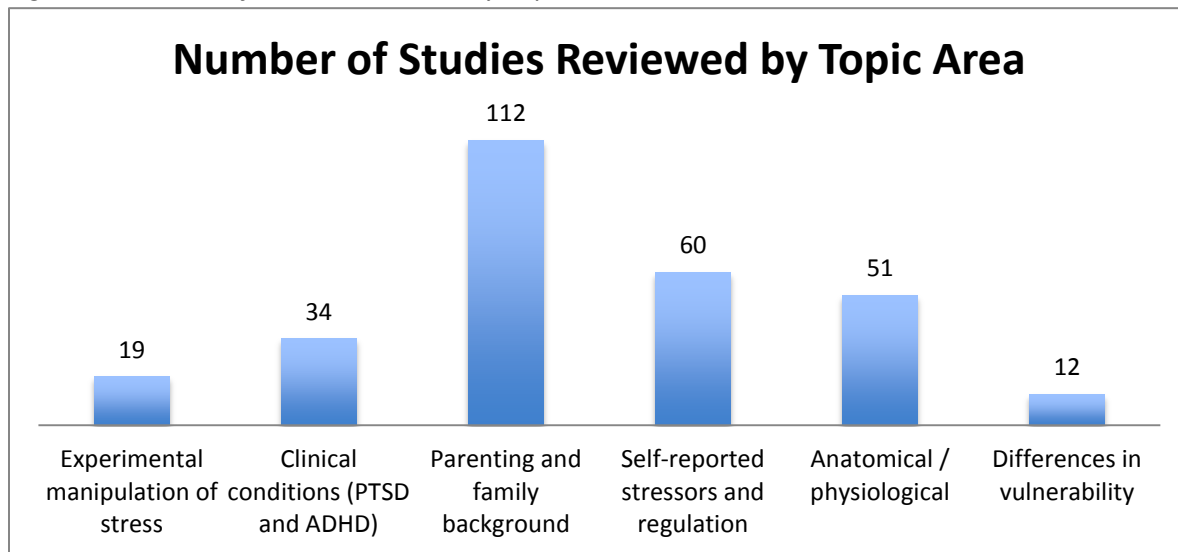


As depicted in Figure 4 and described below, the human studies clustered into several broad categories:

- **Experimental manipulation of stress:** includes laboratory studies where volunteers are exposed to stressors and their reactions assessed
- **Clinical conditions:** PTSD and ADHD
- **Parenting and family background:** includes developmental studies, many of which are longitudinal
- **Self-reported stressors and self-regulation:** includes a wide variety of studies, most in which participants report on their experience of negative or stressful life events, and then are either tested or asked about self-regulation
- **Anatomical /physiological:** includes cortisol studies and brain scan studies that also measure stress
- **Differences in vulnerability:** includes moderation studies examining whether the stress-self-regulation relationship is different among different groups of individuals (gender, genotype, etc.)

Of note the largest number of these (28%) is studies that examine parenting and family background characteristics that are related to stress and self-regulation.

Figure 4. Number of Studies Reviewed by Topic Area



Another way to categorize the studies is by the approaches used to define self-regulation. Approximately 30% of the studies made direct reference to “self-regulation,” “self-control,” or “executive function.” Another 40% involved the analysis of neurocognitive correlates of self-regulation, like the control of attention. Approximately one-fifth involved physiological measures of brain activity relating to self-regulation, like activity of the prefrontal cortex. Only a tiny fraction made reference to behavioral manifestations of self-regulation (like impulsivity, sensation seeking, or social competence). In terms of stress constructs, the most frequently observed were stressors related to family environment (maltreatment, adverse childhood events, and so on), although a substantial fraction of the studies made explicit mention of the terms “stress” or “allostasis.”

Animal Research Methods

Applying concepts of self-regulation to animals requires a careful mapping of relevant emotional, cognitive, and behavioral outcomes. Emotions in animals are difficult to measure directly, but researchers can systematically and rigorously measure **affective displays** (which are behaviors that reflect the experience of an emotion—for example, freezing or crouching, which reflects the animal’s experience of fear). Therefore, in discussing animal studies we will refer to “affect” which is the closest research gets to measuring emotion in animals. Some of the studies assessing the cognitive or affective consequences of toxic stress in animals involve direct comparison of the anatomy and physiology of the brains of experimentally stressed versus unstressed animals. We treat these outcomes as relevant to self-regulation if the comparisons involve brain structures or functions that have been established as directly involved in regulation of affect or behavior (that is, the amygdala, the prefrontal cortex, or neural connections between these two).

It is also possible to use an animal’s behavior to learn about its capacity for self-regulation, in a way that is also relevant for humans. Examples of how stress has been manipulated and specific indicators of

self-regulation in laboratory animals (attention, impulsivity, and anxiety) are provided in Boxes 1.1-1.4 below.

Box 1.1. Experimental Manipulation of Stress in Laboratory Animals

In order to study the impacts of acute or chronic stress in animals on their cognition, affect, and behavior, researchers have converged on a handful of standard ways to produce conditions of acute or chronic toxic stress. One common approach is to confine lab rats for an extended period of time in plastic cages that are slightly smaller than the animals themselves (so their bodies are mildly compressed). This “restraint” procedure activates their biological stress response, since they are not used to being confined in this manner. Another common procedure involves electrifying a metal grid in the floor of an animal’s cage, and sending mild levels of electrical current through the grid, shocking its feet, at irregular and unpredictable times. Other common procedures include exposing them to startling noises, separating young animals from their mothers, depriving them of sleep, or exposing them to hostile animals or predators. Others studies administer stress hormones directly into an animal, rather than subjecting the animal to stressful conditions [30].

It is important to note that each of these stressors has been confirmed to activate the animal’s biological stress response, but if it is applied only a limited number of times or not too frequently, then after each application, the animal’s stress response generally returns to pre-task levels. Therefore, experiments that involve short, limited applications of these stressors can shed light on effects of **acute** stress. Such experiments, however, may not be comparable to situations of **chronic** stress that can create a toxic response. In experiments where researchers aim to produce chronic stress, they do so by frequently repeating and/or prolonging application of stressors like these. In many of the studies we will highlight, stressor application is deliberately and carefully timed within the animal’s life cycle to occur during periods when the animal’s brain development is analogous to the development that normatively occurs in the brains of humans during childhood, or during adolescence. As we will discuss, one line of inquiry in this research has been to investigate the different effects of different *dosages* (whether in terms of severity, frequency, or duration) of stress on brain and behavior—in effect, asking, “when does stress *turn toxic* in laboratory animals?”

Box 1.2. Measuring Attention and Impulsivity in Laboratory Animals

Researchers have studied two behaviors in rodents that are directly relevant to self-regulation—namely, attention and impulsiveness, by giving the animals tasks in which they earn rewards by focusing attention and restraining themselves [31]. One standard protocol used with rodents is the Five Choice Serial Reaction Time Task (5-CSRTT), in which a rodent must distribute its attention across five apertures. Every few seconds, a light will flash in one of the apertures, and the rodent’s task is to react by approaching the aperture where the light appeared. If the rodent goes to the correct opening, it is rewarded with food; if it acts too impulsively (e.g., approaches openings prematurely) or pays insufficient attention (e.g., does not react at all after the light flashes, or reacts incorrectly), then food is

withheld. Without too much training, rodents learn how the task works, and perform reasonably well. Impulsiveness and poor control of attention are measured by their performance on the task. A few studies have investigated the effects of stress on aggression in laboratory animals, which may be considered an indicator of behavioral self-regulation related to impulsivity. The brain processes that are engaged when an animal is trying not to engage in impulsive aggression are similar to those involved in other forms of self-regulation [32], indicating that lessons learned from observing aggressive behavior in lab animals may apply to other forms of self-regulation. More generally, pathological levels of social aggression have been linked to disruptions in biological stress systems [33].

Box 1.3. Measuring Anxiety and Novelty Aversion in Laboratory Animals

A substantial fraction of studies explores whether high levels of acute and chronic stress increases animals' risk of anxiety-like behaviors or of developing a pathological aversion to novel situations; we treat those two outcomes as relevant to self-regulation because they can be considered a manifestation of emotional or behavioral dysregulation. One way of measuring these outcomes is by placing animals in conditions to which they are poorly adapted, and systematically measuring their reactions. For example, the "elevated plus maze" is a tool that takes advantage of the fact that rodents are naturally averse to open spaces and to heights; it is a structure shaped like a "+" sign, placed a few feet off the ground, on which two arms have cliff-edges and two are enclosed by high walls. In order to measure in rodents something that is akin to anxiety, researchers will place an animal in the center of one of these structures, and keep track of the amount of time it spends exploring the sections that have cliff-edges, before retreating to the more comfortable enclosed sections. Rodents who retreat very quickly are identified as experiencing a higher degree of anxiety. Other common procedures to measure anxiety or aversion to novelty include giving young primates a maternal effigy (an inanimate object that is shaped and smells like their mother) to which they can retreat and then tracking how often they retreat to it, or placing rodents on a wide, brightly lit floor and keeping track of the amount of time they spend exploring versus drawing back into corners or against walls.

Box 1.4. Measuring Goal Orientation under Stress

Finally, some studies investigate animals' ability to successfully orient their behavior toward a goal by placing them in conditions where they have an unambiguous problem to solve, and observing how good they are at solving it. One example of this is the "Radial Arm Water Maze" (RAWM) procedure [34], which takes advantage of the fact that rodents, when placed in water, instinctively seek an escape. In the RAWM procedure, the rodent is placed underwater in a central chamber which has several arms extending in different directions. Only one of these arms has an escape platform at the end, onto which the animal can climb out of the water. A rodent's capacity to orient behavior toward a goal is measured in this procedure by the efficiency with which it learns which arm has the escape hatch, and how successful it is, when placed under the same conditions a second time, in going directly for the correct arm.

Findings from Studies of Laboratory Animals

In the next several sections, results of laboratory animal studies are described in a way that addresses questions of interest in this report. We start by considering how early stressors may generally impact later development, then we look at the long-term impact of acute but severe stressors and the impact of early stress on vulnerability to later stress. Finally, we address moderators of the effects of stress on self-regulation and whether the impact of early stress experiences may be reversed by changes in the environment.

IMPACT OF EARLY STRESSORS ON LATER DEVELOPMENT

Another series of studies has identified links between stress administered early in an animal's life or during its gestation and the way its brain processes the chemical dopamine, which plays an important role in the control of attention [35-38]. In one study [39], fear extinction (i.e., a reduction in fear response over time, described further below) was observed to be impaired for rats whose mothers were exposed to stressful conditions while they were gestating (prenatal stress). Similar studies have reported that rodents exposed to stress during gestation or in childhood (which in rodents, comprises the first several weeks of life) displayed hyperactive and reckless behavior, together with evidence of structural or chemical changes in the brain that parallel these behavioral changes [40-43]. Stress in rodents' childhood has also been observed to affect the structure and function of brain regions involved in planning or in emotional processing [44-50], and may even affect the mother-child bond [51]. In contrast to these findings, other studies have found evidence of maternal deprivation and being reared in isolation also on other areas of the brain, but not in the areas that are involved in self-regulation [52, 53]. In rats, stress in adolescence leads to physiological and behavioral changes in adulthood that are consistent with impaired stress response and increased activity in areas of the brain that are involved in emotion [54, 55]. In one study, exposing rodents to severe childhood social stress (by separating them from their mothers) did increase some kinds of impulsive behavior [56]. Similarly, when early adolescent rats were exposed to this same attachment stressor, they became more aggressive and there was evidence of changes in their neurochemical response to conflict with others [57].

LONG-TERM IMPACT OF ACUTE BUT SEVERE STRESSORS

Recent studies have begun to shed light on how acute but severe stressors may have lasting effects in animals. In one study, for example, rats were exposed to the scent of cinnamon and then submerged in water. Even many weeks later, in a nonthreatening environment, these rats still showed behavioral and neurophysiological evidence of debilitating fear when they smelled cinnamon [58]. Several other studies also suggest that a relatively short burst of stress, if traumatic enough, can have long lasting effects on the anatomy, physiology, and biochemistry of the brain [59-70]. Long after an original stressor is taken away, exposure to high levels of stress can distort rodents' approach to problem-solving, interfere with their ability to successfully orient their behavior toward a goal, and reduce their ability to engage with new or uncertain conditions [71-75]. It is also important to note that some of these effects may be evidenced simply when the stressor is *anticipated* [76]. Exposure to acute but severe stress (e.g., restraint) also shifts rats away from undertaking a difficult behavior that would yield a large reward, in favor of easier behaviors that yield small rewards [77].

IMPACT OF EARLY STRESS ON VULNERABILITY TO LATER STRESS

In addition to the long-term impact of acute but severe stressors on self-regulation described above, there is also considerable evidence that early stress experiences and exposure in laboratory animals impact their vulnerability to later stressors, in a process referred to as *stress priming*. We first describe the impact of chronic early stress experiences and then address exposure to stress hormones and toxic chemicals like nicotine.

Chronic Stress Experiences. Chronic stress, especially if experienced early in life, may leave an individual more vulnerable to debilitating effects of stressors experienced later on [39, 51, 78-88]. For example, in one study [78], researchers put adult rats in an easily recognizable box many times over a span of two days; each time a rat was placed in the box, the floor of the box was electrified for a few seconds. In time, the rats learned to fear the box because they associated it with the painful footshock sensation. As a result, when researchers placed them in the box even *without* electrifying the floor, the rats still exhibited debilitating effects of stress (specifically, they froze in place—a phenomenon in rodents that is sometimes viewed as comparable to severe depression in humans). Then, for several days, the rats were placed repeatedly in the same box without electrification; after many uneventful spells in the box, control rats that had been raised in normal laboratory conditions gradually lost their fear. Researchers could place them in the box and they would behave normally rather than freezing; this is a normal process which psychologists call “fear extinction.” By comparison, this fear extinction was severely impaired in the group of rats who had been exposed to stressful conditions in their adolescence. Thus, adolescent stress made it more difficult for rats to overcome debilitating effects of stress experienced in adulthood.

Other Stress Exposures. In addition, there is evidence that the long-lasting effects of acute stress are even worse in animals whose brain regions were primed with a long period of low-level exposure to stress hormones before an acute event [89]. Another provocative study [78, 90] found that rodents’ exposure to nicotine in adolescence and to prolonged stress each had a negative impact on their goal-oriented behavior; more striking, however, was the fact that the *combined* effects of nicotine and stress were *even more* harmful than the sum of the two independent effects. This raises interesting questions as to whether stress may be more harmful to the self-regulation of a young smoker than a young non-smoker. As noted, however, adolescence may not be the only sensitive period for such stress priming.

MODERATORS OF THE IMPACT OF STRESS ON SELF-REGULATION

Two types of moderators have been examined in relation to stress and self-regulation in laboratory animals: characteristics of the stressors themselves (severity, predictability, and controllability) and individual differences in the animals (both biological and experiential).

Severity, Predictability, and Controllability of the Stressor. As noted previously, studies suggest that the level of the intensity of an acute stressor may impact its effects, with more severe stressors having more significant and potentially long-term effects. Another example of this is seen in a study that injected stress hormones directly into rodents’ brains and found that at low dosages, the stress hormones could improve their performance on an attention task (see Box 1.2), but at higher doses that

effect disappeared [91]. Similarly, the controllability and predictability of the stressor may also impact its effects. As expected, conditions in which animals have less control or in which stressors are less predictable have more severe anatomical and physiological effects, compared with conditions with more control or predictability [92, 93].

Individual Differences. Stress vulnerability in laboratory animals also varies with individual differences, both biological and experiential. Several studies indicate that biochemical and physiological characteristics are associated with stress vulnerability in laboratory animals [66, 94-102]. In addition, there is evidence that stress may turn toxic for male rodents more quickly than for females [103], although additional genetic and physiological characteristics may also be involved [73, 104-111]. However, there is also reason to consider such biological vulnerabilities in the context of parenting and environmental stressors. For example, there is evidence that a *mother's* vulnerability to the toxic effects of stress can interfere with the ability of her *young offspring* to engage successfully with new or uncertain situations [112]. Other studies have provided evidence of important feedback effects; experimentally manipulating a specific brain region involved in self-regulation—the medial prefrontal cortex—impacts the behavioral and biochemical effects of environmental stressors [79], and in turn, stress also affects the function of this same brain region [51]. Finally, there is indication that individual differences may interact with the characteristics of stressors. For example, the effects of different doses of stress hormones has been shown to vary based on underlying genetic differences in brain chemistry [113].

POTENTIAL FOR REVERSING EARLY STRESS EXPERIENCES WITH SUPPORTIVE ENVIRONMENTAL CONDITIONS

A handful of experiments indicate that many of the long lasting anatomical, physiological, biochemical, and behavioral effects of stress experienced in early life and adolescence may be reversible through improved environmental conditions [54, 60, 80, 114]. For example, in one of these [54], researchers set up three groups of adolescent rats—one group was exposed to powerful stressors every day for three consecutive days, and then returned to their cages to be housed under normal laboratory conditions until they became adults (which for a rat takes one month); the second group was exposed to the same powerful stressors, but while they were growing to be adults, they were placed in an “enriched environment,” which included conditions more appealing to a rat than normal laboratory conditions—for example, they were housed in social settings with stimulating colorful objects hanging in their cages and exercise wheels and higher quality food; the last group was a control group which was not exposed to stressors at all. When researchers compared the adolescent-stress-then-standard-cage rats with the control group, they found that the brains of the adolescent-stressed rats had been rewired, that the biochemistry of their stress systems had been altered, and that their behavior was affected in a manner that might be viewed as homologous with depression in humans. For example, they were less likely than control animals to try to find ways to escape painful footshocks (“learned helplessness”), or to explore novel settings. However, the adolescent-stress-then-enriched-environment rats performed at least as well as control rats on all these measures. Their stress systems were normal, and in some cases in fact they did a better job than the control rats at solving problems or exploring novel settings.

SUMMARY OF ANIMAL STUDIES

In laboratory animals, experimental administration of stressors has been shown to induce measurable change in brain anatomy, physiology, and biochemistry relevant to self-regulation, and in the cognitive, affective, and behavioral processes that are defined as self-regulation in laboratory animals. In particular, rodent studies link stress to aggression, hyperactivity, and impulsivity, which may reflect a lack of behavioral self-regulation, as well as to attention, planning, and emotional regulation. There is also evidence that acute, severe stress may have long-lasting negative effects including decreased goal-orientation, perseverance, and problem-solving. Of particular interest in this review is research suggesting that early chronic stress in childhood may leave animals more vulnerable to the effects of stressors experienced later on but that effects of stress early on may be reversible with certain environmental conditions. It is important to note, however, that rodent experiments are limited in their ability to replicate conditions of chronic stress that may become toxic for children.

Evidence for a Relationship between Stress and Self-Regulation in Humans

The next section summarizes findings on self-regulation and stress from the 311 empirical studies reviewed that include human participants. As noted in the overview, a range of methodologies and populations have been examined that have a different set of strengths and limitations than do animal research. Some of the strengths of this literature include culturally and ethnically diverse samples, a large body of studies of early child development, and a range of methodologies that show similar findings. Limitations and constraints will be identified throughout each section, particularly the small body of experimental research, frequent use of volunteer or other non-random samples as well as self-report methods which are subject to bias.

This section begins with experimental studies that manipulate stress in humans, which is a relatively small subset of the literature in this area. Then we present evidence of biological effects of stress in humans including brain differences related to traumatic stress exposure and the relationship between stress hormones and behavioral dysregulation. Next we turn to the relationship between stress and self-regulation across development, with particular focus on parenting studies, which comprise approximately 1/3 of the human studies we identified. These studies examine harsh parenting and maltreatment as well as positive parenting. Finally, we examine self-regulation and broader environmental factors including family context, co-parenting, and other adversity such as poverty and food insecurity. This section concludes with a discussion of potential moderators of the impact of stress on self-regulation including both individual and environmental factors, which may have implications for intervention.

EFFECTS OF ACUTE STRESS MANIPULATED IN VOLUNTEERS

A series of studies has experimentally manipulated the effects of acute stress on volunteers in laboratory tasks and measured self-regulation outcomes. These studies asked human volunteers (including adults, adolescents, and in a few cases children) to engage in activities that required self-regulation or executive functioning skills, while exposing them to stressors of varying strength using tools like the Trier Social Stress Test (see Box 2). It is important to note that experimental manipulations of stress in volunteers identify effects of acute stress on volunteers' current capacity to self-regulate.

They do not generate a chronic stress response; the stress induced by these manipulations is unlikely to prove toxic because a participant's stress response usually winds itself down according to the normal, healthy pattern. They also do not measure effects more than a few hours after the stress exposure. Therefore, studies of this kind can provide only limited information about the effects of toxic stress on long-term development of self-regulation.

Box 2. Experimental Manipulation of Stress in Humans

The "Trier Social Stress Test" is a way to reliably generate a physiological stress response in a volunteer; it involves asking the volunteer to prepare a 5 minute oral presentation, and then to present it in front of a small panel of judges (and sometimes a video camera), and then immediately after the speech, to solve mental arithmetic problems under the gaze of these same judges. Researchers will often directly measure the level of stress hormones in the volunteer's blood, to ensure that the experience "worked" in terms of being stressful. In a small number of studies, stress effects have also been examined in response to injecting volunteers with stress hormones.

Such experimental manipulations have been observed to affect volunteers' performance on self-regulation tasks completed in the laboratory including assessments of working memory and cognitive flexibility [115-119], although some people may be more vulnerable to this effect than others [120, 121]. Another way to manipulate stress in participants is to show them aversive films or film scenes. For example, after watching the first 30 minutes of "Saving Private Ryan" (a war movie generally considered aversive), young adults performed more poorly on an executive functioning tasks than after watching the first 30 minutes of "Shrek"(a humorous child's film) [122].

Instead of asking them to engage in stressful tasks, researchers can also manipulate stress in volunteers by injecting them with stress hormones. In one study, young adults injected with low levels of stress hormones performed better on an emotional regulation task than those injected with placebo, but those injected with higher levels performed worse [123]. In two studies, direct injection of stress hormones affected adults' ability to control their attention [124, 125]. These results suggest that the relationship between stress and self-regulation is not simple or linear.

EFFECTS OF SLEEP DEPRIVATION ON SELF-REGULATION

Similar to experimental studies of acute stress manipulation, a series of studies has explored the impacts of short-term sleep deprivation on the capacity of children, adolescents, and adults to sustain attention. The evidence from these studies indicates that insufficient sleep can severely hamper performance on tasks that require sustained attention [126-137]. In preschoolers and elementary school children, poor sleep has been found to correlate with higher levels of stress hormones [138, 139]. In a few studies, acute sleep deprivation has also been found to predict impulsive responses to negative stimuli and reduced ability to recognize emotions in others [140-142], to react to the sight of emotional faces [143], and to modulate emotional responses [144]; on the other hand, one study presents evidence that the relationship may be cyclical, in that exposure to negative emotional stimuli affects subsequent sleep

quality [145]. In one study, sleep deprived participants showed lower willingness than well-rested participants to invest more effort in a task in return for more rewards [146]; sleep deprivation is also associated with a greater willingness to take risk [147]. Our literature search, however, uncovered no studies which specifically measured links between chronic sleep deprivation and the long-term development of self-regulation.

BIOLOGICAL EFFECTS OF STRESS ON SELF-REGULATION

In this next short section, we describe two set of studies that provide evidence for biological effects of stress on self-regulation in humans. The first group of studies describes differences in brain structure and function in individuals with a history of stress experiences as compared to those without. The next set reviews studies examining the relationship between stress hormones and behavioral dysregulation. Although these studies do not randomly assign individuals to stressful conditions (which would be unethical), their findings parallel the animal research previously reviewed that demonstrates similar biological effects of experimentally manipulated stress.

Brain Differences Associated with Traumatic Stress Exposure. Using relatively new technologies to observe aspects of brain anatomy and physiology, researchers have been able to observe relationships between stressful conditions and brain structure and function. Some of these studies suggest that brain regions involved in planning are structurally different in children who have experienced traumatic stress compared with those who have not [148]. A series of important studies has examined a sample of children who were raised in orphanage institutions in Romania who were severely physically, socially, and emotionally deprived. These studies have indicated poorer planning and goal-oriented behavior in these children, relative to normatively raised controls [149, 150], and higher risk of problem behavior in late childhood [151]. In parallel with these behavioral findings, researchers have observed differences in brain structure and function relevant to self-regulation in these children, relative to normatively raised controls [152-156]. In addition, structural and functional differences in brain regions involved in self-regulation have been observed when comparing young adult volunteers who were maltreated in childhood against young adult volunteers who were not [157-165]. The relationship between childhood stress or trauma and brain structure and functioning have been observed in older adults [161, 166-170] and in children, where such findings also correlate with social problems in everyday life [171-178].

Brain Differences Associated with Socio-Economic Status. Structural differences have been observed in the brains of children whose parents report lower socioeconomic status (SES). In those studies, parent SES was measured through a combination of parent education, income and occupation variables. As has been described earlier in this report, low SES of parents is associated with environmental adversities and stressors such as economic hardship, food insecurity, daily hassles, and exposure to dangerous or unhealthy living situations. Relative to those whose parents report higher SES, children of lower SES parents demonstrate lower self-regulatory capacity. For example, in one study, children of lower SES demonstrated large differences in volume in areas of the brain involved in social-emotional processing and cognitive control, with some of these differences increasing with age [179]. Given variations in the way people respond to adversity and how factors such as parenting and other supports moderate outcomes, these findings should not be interpreted as deterministic. Instead, these findings highlight the importance of addressing environmental factors when considering interventions.

Stress Hormones and Behavioral Dysregulation. A handful of studies have examined stress hormone levels in volunteers with and without behavioral regulation problems (for example, problem gamblers), and have found evidence that adults who are prone to risky behavior tend to display the kind of blunted stress response that is consistent with the toxic effects of chronic stress exposure [180-182]. A similar pattern has also been observed among individuals with more neurotic personalities [183], with those who have more trouble interpreting the emotional content of facial expressions [184], and with those who have more trouble with anger regulation [185]. In one study, a blunted stress response was observed in adults who reported having been abused as children, and these same adults were observed to perform more aggressively toward strangers in a laboratory task [186].

THE RELATIONSHIP BETWEEN STRESS AND SELF-REGULATION ACROSS DEVELOPMENT

Several studies using different methodologies highlight the relationship between early stress experiences and self-regulation across development. With regard to the prenatal period, there is evidence that distress experienced by a mother during pregnancy may predict difficult temperament in her infant [187]. Infants whose prenatal and neonatal experiences indicated higher levels of physiological or environmental stress (low birth weight, preterm birth, mother's history of substance use during pregnancy) reacted more emotionally to frustrating conditions, while those whose experiences were more normative were better able to manage their emotions and plan a way to ameliorate their situation [188-191]. It has been noted, however, that some of these differences could be related to factors besides stress [192].

Illustrating how stress exposure early in development can affect stress vulnerability much later, one study injected stress hormones and placebo into two groups of older adolescents—one whose mothers had experienced a “negative life event” while they were gestating, and another whose mothers had not—and found that the stress hormones disrupted executive functioning in the former group and not the latter [193]. These groups likely differed in other important ways; however, so prenatal conditions may not be the only or even the best explanation for these results.

Another set of studies has examined the relationship between stress and self-regulation using self-report and laboratory assessments. Respondents are asked about current or past experiences of negative or stressful life events, and then provide information about or complete tests of cognitive, emotional, or behavioral regulation. In studies like these, adolescents who report more stressful life events are generally observed to have more trouble regulating their emotions than those who report fewer stressful life events [194, 195]. Those with a history of maltreatment or exposure to a parent's intimate partner violence have been observed to be less effective at controlling their attention, regulating their emotions, and planning [196-204], and these deficits are apparent into adulthood [205-209]. For example, college students with self-reported histories of maltreatment appear more likely to engage in deliberate self-harm, perhaps as a way to reduce negative affect [210]. Adults who report more adversity in their environments also report higher levels of impulsivity [211], and those seeking treatment for stress-related exhaustion perform poorly on executive functioning tasks [212].

It is important to note that all of these findings are correlational and subject to recall bias; they should not be interpreted as evidence that stress causes these difficulties. They are nonetheless consistent with the experimental data reviewed previously.

Parenting, Child Maltreatment, and Self-Regulation

As indicated in the introduction to findings, the largest number of studies identified in this review described relationships between parenting behaviors and self-regulation in children and adolescents (although there are notably fewer adolescent than early childhood studies). We review first the studies of negative effects of harsh parenting and child maltreatment, and then studies examining the effects of positive parenting, warmth, and responsiveness.

DELETERIOUS EFFECTS OF PARENTING AND CHILD MALTREATMENT

Parenting style has been linked to physiological indicators of stress in children [213-215]. For example, maternal punishment has been shown to predict stress hormone levels in preschoolers encountering strangers, and this is also related to their behavioral responses in that situation [216]. Relatedly, a few studies have shown that children punished corporally function worse in terms of self-regulation, whereas those whose parents adopt authoritative but non-corporal styles perform better [217-220]. Such an authoritative style involves setting limits, reasoning with children, and being responsive to their emotional needs. Similar effects are seen into young adulthood, where young adults whose parents are assessed by external observers to be more “controlling” and less “warm” have been observed to be less effective at emotion regulation [221-224]. In addition, anatomical characteristics of young adult volunteers’ planning and emotion processing brain regions were also associated with their mothers’ propensity to display punishing behavior toward them in laboratory tasks [225] and with attachment security [226]. In contrast to the majority of studies, two studies identified in this review did not find any correlation between parenting style and self-regulation [227, 228].

Several studies indicate that child maltreatment predicts behaviors that may indicate self-regulation difficulties across developmental periods. In infants and toddlers, neglect and maltreatment predict impairment in independent play behaviors and socially competent behavior [229]. In school-aged children, abuse and neglect have been found to predict bullying and dominance behaviors [230]. Child maltreatment also predicts specific self-regulation difficulties in young adulthood such as unprotected sex and impulsive personality traits [231, 232], although this relationship appears to depend on other factors in the environment as well as individual characteristics. In adults, a self-reported history of abuse or neglect predicts mental health difficulties, particularly depression and anxiety, although again such data are subject to recall bias. Of particular interest is that maltreatment appears related to stress-related biomarkers and differences in brain structure and function that appear to mediate later psychiatric symptoms [233-237].

BUFFERING EFFECTS OF POSITIVE PARENTING

Considerable evidence [191, 238-250] links a range of parenting factors (discipline, sensitivity, family routines, and parent-child relations) to self-regulation [251-255], with associations observed as early as infancy and toddler-hood [256-258]. Among low-income children, parental warmth, emotional

responsiveness, and support have been found to predict emotion regulation and delay of gratification [238, 247], which may also have a buffering effect against other stress. In one study of preschool children exposed to intimate partner violence, better parenting and fewer maternal mental health problems predicted greater resilience in the children's social-emotional functioning [239]. Another study found that while poverty predicts slower growth in children's development of self-control during the preschool years, positive parenting was associated with greater growth [242]. Also of interest, low-income children living with two parents were found to have higher levels of executive functioning than those living with a single parent [246], although this was mediated by parent responsivity, enrichment activities and family companionship. Socially disadvantaged parents may shield their children from negative behavioral effects of stress if they have more social support, lower risk of depression, or display more positive parenting behaviors [259-263].

Among children with a history of maltreatment who have been adopted, correlational evidence suggests that self-regulation may improve [21, 264, 265]. There is also evidence of a causal effect from a randomized controlled study of children living in orphanages in Romania. Going into foster family care ameliorated some of the negative effects on their self-regulation [266, 267] as compared with a randomized control group. Unfortunately, in severe cases of maltreatment, the impact of early adversities may remain stable into adulthood [268]. For young adults, mentoring by other adults or young adult peers may also help to offset the effects of low parental support [269-273]. This finding suggests that caregivers other than parents can provide co-regulation (described in our Foundations report) in a way that supports self-regulation development.

This body of research identifies parenting as an important moderator of children's self-regulation, however, it is not without its limitations. First, only a tiny fraction of these studies are experimental, limiting our ability to apply causal interpretations to the results. That is, one cannot say much about whether parenting either causes or prevents self-regulation difficulties; rather, that an association is clearly established. Moreover, the association between parenting and self-regulation could reflect reverse causation, where children with certain temperaments elicit certain parenting behaviors. Also, in some of these studies, parenting styles and children's self-regulation are both assessed by parent report which is a methodological approach subject to bias. And although some parenting studies have been conducted in different countries and with some minority samples, the pathways between parenting and self-regulation remain under-explored across cultures [274-284]. It is also important to note that the relationship between parenting and self-regulation may reflect more general ecological factors related to stress in the child's environment that are not often assessed in these studies—including, for example, poverty or other environmental stressors that the family encounters, or even parents' own self-regulation. Thus, an examination of broader ecological influences such as is provided in the next section is useful.

Broader Family and Environmental Stressors and Self-Regulation

Beyond specific parenting behaviors, there are other stressors associated with parent characteristics, parent-child interactions, and family context or conflict that may impact self-regulation. In addition, other environmental adversity beyond the family like poverty and food insecurity appears to play a role.

PARENT CHARACTERISTICS

With regard to parent characteristics, several studies have shown that a mother's negative emotionality correlates with reduced emotion regulatory capacity in children, adolescents, and adults [285-291]. Children whose mothers displayed more depressive symptoms also had larger fear and emotion processing regions than children whose mothers displayed fewer symptoms [292]. Maternal depression is also strongly associated with problem behaviors in children and adolescents [293-297]. Potential mechanisms for this effect are suggested by a study showing that if a mother's stress response is activated while she is with her infant, the infant's stress system activates in parallel [298]. Finally, adult antisocial characteristics predict maladaptive parenting and family conflict, which is associated with child disruptive disorders [299].

PARENT-CHILD INTERACTIONS

Several studies also suggest a bidirectional relationship between parenting style or practices that may generate a stress response in children or adolescents and behaviors that may indicate deficits in self-regulation, like disruptive behavior or delinquency. For example, parents appear more coercive or less attached—which may generate stress—when their children exhibit more oppositional behavior or greater levels of delinquent behavior—which may be an indicator of behavioral dysregulation [300-303]. In one study, children's externalizing behavior was found to predict parents' sense of competence, while parents' sense of competence did not predict child behavior [304]. In particular during adolescence, over-reactive parenting and parent-child conflict may contribute to adjustment problems and antisocial behavior [305, 306], although the link between these parenting practices and children's stress response has only been indirectly established. On the other hand, parental warmth, monitoring, and rule making and support may moderate increases in problem behaviors over time during adolescence [307].

CO-PARENTING AND SIBLINGS

With regard to other family factors that may be related to children's self-regulation development, there is evidence that co-parenting cooperation and conflict predict child adjustment. For example, among preschoolers born to teen mothers, greater co-parenting conflict was associated with increased behavior problems, particularly for boys [215]. Similarly, when parents of preschoolers are supportive of each other's parenting efforts, self-regulation difficulties do not predict behavior problems as they typically do [308], suggesting something of a buffering effect. Child emotional and behavioral difficulties may also be related to conflict between a single mother and others in a co-parenting role including grandmothers as well as fathers [309]. These findings are consistent with results of a meta-analysis which found that parenting cooperation, agreement, and conflict predict changes in child adjustment, although these effects are generally small [310].

A few studies also suggest that siblings may have an influence on behavioral regulation and problem behaviors. For example, younger siblings of pregnant teens were found to have higher rates of behavior problems, which appeared related to harsh parenting and mother-sibling conflict during the period before and after the sibling's pregnancy [311]. Of particular interest for adolescents, an older sibling's problem and risk behaviors appear to decrease the influence of parenting on the younger sibling's risk behaviors [312]. Thus, considering family influences on self-regulation beyond parenting alone appears important.

OTHER ENVIRONMENTAL ADVERSITY

Children whose parents report more adversity in their environment have stronger links between physiological stress and emotion regulation than those whose parents report less adversity [313]. Poverty and neighborhood disadvantage are also associated with failures in behavioral regulation [314, 315] including impulsivity and risk-taking [316, 317], with effects seen as early as 6 months of age [318]. Similarly, poverty is associated with math achievement, but this is largely accounted for by attention and task persistence, which can be effectively targeted to reduce these disparities [319]. Other studies indicate that young children who witness or experience violence are at greater risk of problem behaviors including aggression [320-322], although this relationship is multi-determined [323]. Neighborhood dangerousness may also moderate effects of parenting on children's behavior [324].

Food insecurity is one specific and powerful stressor that has been linked to self-regulation. In a large, longitudinal study of school-aged children, parent-reported food insecurity (i.e., inadequate access to sufficient, safe, and nutritious food that meets children's dietary needs) predicted lower self-control, attention and task persistence as rated by teachers, but not interpersonal skills or disruptive behavior [325]. In another large, prospective birth-cohort study, food insecurity predicted symptoms of hyperactivity and inattention three years later, even after controlling for a number of other risk factors [326].

Despite these links, it is important to note that the specific biological mechanisms through which environmental adversity influences self-regulation development and how individual differences contribute to different effects are poorly understood at this time.

Stress and Self-Regulation in Studies of PTSD and ADHD

As noted, we investigated the relevant literatures on two mental health disorders characterized by difficulties in self-regulation—attention deficit/hyperactivity disorder (ADHD) and post-traumatic stress disorder (PTSD). We utilized specific inclusion criteria that allowed us to identify studies of these populations that focused on relationships between stressors or the body's stress system and the symptoms or severity of these disorders so as to expand our understanding of stress and stress reactivity.

STRESS AND SELF-REGULATION IN STUDIES OF PTSD

As noted earlier, young children exposed to a traumatic event may be at substantial risk of impaired behavioral functioning and post-traumatic stress [327]. Adolescents may be particularly vulnerable to

PTSD, with one review suggesting their risk of experiencing trauma is higher than that of either children or adults [328]. A handful of studies investigated how individuals diagnosed with post-traumatic stress disorder differ in terms of cognitive, affective, and behavioral aspects of decision-making, compared with those who have not been diagnosed. One study found evidence that individuals with this condition react to stress triggers with enhanced activity of brain regions involved in emotional response, and reduced activity in regions involved with planning [329-331]; in contrast, other studies suggest that *anticipating* a trigger affects brain functioning primarily in the planning regions [76, 332, 333] for individuals with PTSD. However, important questions remain unanswered about differences in the physiology of self-regulation in those with PTSD compared with those who had similarly traumatic experiences but did not develop PTSD [334].

STRESS AND SELF-REGULATION IN STUDIES OF ADHD

About 25 studies examined links between ADHD risk and social or biological markers of stress. A few of these studies compared the physiological stress response of children with and without diagnoses of ADHD and other clinical conditions related to behavioral regulation, like oppositional defiant disorder (ODD) and conduct disorder. In some studies, individuals diagnosed with these conditions have been found to display evidence of dysregulation in their stress system, which would be consistent with the toxic effects of chronic stress [335-344], although the most recent studies suggest that the relationship between diagnosed behavior disorders and the dynamics of the stress response may be more complicated than was previously appreciated [345-352]; important questions remain unanswered in this area. Parents of children diagnosed with ADHD of various subtypes report less secure attachment than parents of children without a diagnosis [353-360], and diagnosed children are more likely to indicate evidence of maltreatment [361-364]. However, it is important to note that most of these studies are not designed to distinguish the direction of the effects of this relationship. That is, there may be negative effects of parenting on the behavioral disorder or negative effects of child behavior on parenting, or both.

Negative life experiences and low family socioeconomic status are associated with diagnoses of ADHD or ODD [365-368], as are adverse neighborhood conditions like violence, low quality physical infrastructure, and weak social support [367-371], and adverse conditions in pregnancy [367, 368, 372-376]. There may also be important variability within these associations; for example, risk of ADHD appears to be higher for boys who are maltreated compared to those who are not, whereas the opposite may be true for girls [377]. Sex may not be the only dimension on which vulnerability varies [356, 378-380]. For example, certain genotypes may interact with parenting to predict levels of inattention and hyperactivity [356], and peer acceptance may reduce levels of oppositional behavior associated with harsh parenting [379]. Finally, an interesting and underexplored link connects infant atopic eczema—a skin disorder—with later ADHD risk; this may reflect links between ADHD and dysregulation of the body’s physiological stress response, or it may indicate effects of sleep deprivation (caused by skin discomfort) on brain development, or both, or neither [381, 382].

Factors that may moderate the Impact of Stress on Self-Regulation

An important question for this review is how individual characteristics and experiences may make some children and youth more vulnerable to stress than others. Both individual and environmental factors have been examined in the literature [173, 383-392], including underlying anxiety levels, sex, age, other biological and genetic factors and prior stress experiences. Understanding these factors can help inform how interventions are targeted. Similarly, some children and youth may be more resilient in the face of stressors or with the support of certain protective factors, suggesting particular intervention approaches.

UNDERLYING ANXIETY AND STRESS EXPERIENCES

A few studies have suggested that underlying anxiety levels may predict stress reactivity and a handful of studies identify prior stress experiences as a risk for having a worse response to new stressors. In one experiment researchers used a questionnaire to assess volunteers' risk for anxiety, then observed brain functioning in those same volunteers while they made decisions in a context where specific colors were paired with stressful conditions, and then finally again in a context where they saw the same colors but this time under normal conditions. Among the volunteers who had been at higher risk of anxiety, neurons involved in fear and emotion remained much more responsive to the sight of stress-paired colors even when those colors were shown under normal conditions [393]. Similarly, a handful of studies provide evidence that experimentally administered acute stress can be more disruptive to attention if adults have experienced traumatic stress in the past [394-397] or if they suffer from an anxiety disorder [398]. Underlying anxiety levels could be related to earlier stress experiences as well as temperament, thus these results are likely intertwined. Overall, these findings are consistent with the stress priming effects seen in animal data. Consider, however, that these data are derived from laboratory volunteers and measure response to acute stressors.

Other studies suggest that vulnerability to the negative impact of stress on self-regulation may be impacted by biological or genetic factors. For example, chemicals involved in inflammation appear to be centrally involved in vulnerability, as is the neurotransmitter gamma-aminobutyric acid (GABA). Genes acting within the dopamine system may also interact with stress experiences to impact regulated behaviors such as eating [399]. Specific genotypes have also been shown to predict the relation between negative parenting behaviors and inattention [356] and between childhood adversity and psychotic experiences [400]. Such findings suggest individual biological sensitivity to context and open the way for researchers to begin linking stress and self-regulation to the rich research into social, genetic, and environmental drivers of variation in these physiological characteristics [401-403].

SEX AND AGE AS STRESS VULNERABILITY FACTORS

A number of studies suggest differential vulnerability of boys and men as compared to girls and women to the impact of stress on self-regulation. Behavioral effects of harsh parenting also appear to vary by sex [404, 405], with girls who experience harsh parenting at higher risk of internalizing problem behaviors and boys who experience harsh parenting at higher risk of externalizing [257]. A large longitudinal study found that child maltreatment significantly predicted delinquency for males but not for females [406], although this did not hold true for violent offending [407], where both males and

females are at higher risk[407]. One study suggested differential sex effects for prenatal parental characteristics like locus of control or reinforcement [408]. However, sex may not be the only important driver of this heterogeneity; personality and underlying stress-responsiveness also appear to be involved [409-411]. Moreover, gender includes social and cultural factors as well as genetic and physiological factors. Thus, reasons for some of these differences are likely related to many different and inter-related factors.

One study found that reactivity to laboratory stress tasks was higher among older versus younger adolescents [412], which may reflect the fact that social stressors become more salient during this period. In general, however, there was limited evidence that certain developmental ages are more or less vulnerable than others. Rather, stress effects are seen across development.

PROTECTIVE FACTORS

In this review, the strongest data regarding factors that may protect or “buffer” a child from the negative impact of stress are positive parenting practices including warmth, responsiveness and effective behavior management, which are consistent with the model of self-regulation development in context described in Report 1 in this series (*Foundations for Understanding Self-Regulation from an Applied Developmental Perspective*). In addition to parenting, informal mentoring by other adults may be protective in young adults who have low parental support [269], as previously noted. A few additional studies suggest other possible protective factors including a high level of participation in extra-curricular activities among adolescents [413] and having higher initial levels of self-regulation [414]. In those studies, manipulation of stress in laboratory tasks generated a smaller stress response in adults and children who had higher executive function capacity *before* engaging in them [415, 416].

It is important to note that the present review did not examine intervention studies, as that is the topic for the third report in this series, entitled *A Comprehensive Review of Self-Regulation Interventions from Birth through Young Adulthood*. By definition, interventions may be considered protective factors, and as such, those findings will complement the present results.

SUMMARY AND IMPLICATIONS

The present review reflects the current state of knowledge on relationships between toxic stress and self-regulation across ecological, biological, and developmental studies. Our study takes a broad perspective on this topic, using conceptual frameworks from several scientific disciplines and looking beyond human studies. Overall, we found 394 studies that met our inclusion criteria of having a search term referring to stress and a term referring to self-regulation (with no irrelevant application of terms). Approximately 80% of these involved human subjects. Studies include a range of methodologies from laboratory experiments with volunteers to observational analyses. Self-regulation was measured in these studies not only with behavioral correlates like the control of attention, but also using physiological measures of relevant brain activity like neural function in the prefrontal cortex. The largest number of studies was focused on parenting or family context factors. Although studies spanned ages from birth to adulthood, many more included children and adults than adolescents.

In considering this body of literature and conclusions that may be drawn, it is important to note that there are **important distinctions between the likely impacts of acute and chronic stressors**. The human stress system is very effective in responding to acute stressors, except when they are very severe or traumatic. However, frequent and prolonged activation of the stress system can have chronic, toxic effects on brain and behavior in ways that may not be as widely appreciated. In laboratory animals, experimental administration of chronic stressors during childhood and adolescence—especially in situations where the animal has little or no capacity to react in a way that can ameliorate the situation—induces measurable change in brain anatomy, physiology, and biochemistry relevant to self-regulation, and to cognitive, affective, and behavioral domains of self-regulation. In humans, observational studies and less invasive experiments parallel many of the patterns observed in the animal models. This suggests that *self-regulation interventions should attend to chronic stressors that can add up to produce toxic effects such as living in poverty, experiencing maltreatment or having multiple adverse childhood experiences*.

An important finding from this review relates to the **well-established link between parenting and self-regulation development in childhood**. Over 100 studies demonstrate that parental warmth, supportiveness, and sensitivity predict growth in self-regulation while harsh discipline techniques and maltreatment are associated with self-regulation difficulties such as emotional dysregulation, social competence, and impulse control problems including disruptive behavior. Such effects can be seen in children's stress hormone levels as well as on cognitive and behavioral measures. In addition, child abuse and neglect are associated with difficulties across the lifespan including mental health disorders in adulthood. For children living in poverty, positive parenting and co-parenting may serve to buffer the effects of other stressors in the family and environment. Mothers' mental health was also identified as an important factor influencing children's self-regulation abilities. How and when different parenting behaviors lead to differential development of self-regulation has significant implications for intervention approaches and provides an empirical basis for our examination of intervention research in the next report in this series (*A Comprehensive Review of Self-Regulation Interventions from Birth through Young Adulthood*). More specifically, this report will evaluate how parenting behaviors can improve children's development of self-regulation (or prevent dysregulation).

Another key finding is that **there are many factors that appear to moderate the effects of stress on self-regulation, including individual biological characteristics and environmental factors including parenting**. These factors likely interact in complex ways to influence self-regulation in different contexts. Take for example, the study in adolescent rodents that found that exposure to nicotine in combination with prolonged experimental stress had more harmful effects than the sum of the two separate negative effects [90]. If this finding is one that is applicable to humans, there may be a cascading negative cycle given that we know young people in lower socio-economic families who report higher perceived stress are more likely to take up smoking [417]. Unfortunately, most of the studies identified in this review do not consider such eco-biological interactions, which can lead to misinterpretations of the findings. Along these lines, genetic and epigenetic studies have begun to explore gene-environment interactions and to identify specific biological predictors of vulnerability to

toxic stress. Unfortunately, the evidence base in this area is just emerging. Research into non-genetic predictors of vulnerability is at an even earlier stage. For example, do the effects of stress on the development of boys' self-regulation differ from its effects on girls—and if so, why? How does neighborhood or other aspects of physical environment affect vulnerability? Understanding variation in vulnerability is critical to inform where and how intervention resources can be deployed to maximum effect. Our recommendation is that future research take an inter-disciplinary approach to examining these complex links.

Scientific research on stress and self-regulation is also complicated by many important feedback loops and dynamic links between exposures and outcomes that can sometimes span many years. The process by which chronic stress turns toxic (that is, begins to have long-term effects on brain and behavior) is likely to be gradual and cumulative, and an important set of toxic effects is on the stress system itself; a child with a history of stress experiences will react differently to stressors than a child with a healthy stress response. Thus, previous exposure to stressors may sensitize or “prime” a child to have more difficulties self-regulating when faced with acute stress later. Therefore, while it is likely true that providing the most vulnerable children and families with additional supports to cope with chronic stressors earlier may help to prevent problems with self-regulation later, there are also likely to be interventions that can help even after problems have emerged by providing support in coping with the kinds of acute stressors that less disadvantaged people can manage effectively. In other words, both universal and targeted interventions may be important - a question that our next report will examine.

There are also several limitations to the current literature base which should be considered in interpreting the findings. Importantly, most human studies linking stress and self-regulation are observational or correlational, limiting conclusions that can be drawn about causality. Here is where non-human animal experiments contribute value to our understanding of these connections, and provide evidence that stress causes self-regulation difficulties under certain conditions. The challenge, of course, is that although rodents share a similar stress response system to humans, many measures of self-regulation in non-human animals are indirect, and must be mapped onto the behavior of humans, a significant challenge acknowledged by researchers in the field. In addition, many studies in humans have been conducted in volunteers who respond to advertisements and may differ substantially in terms of openness to novel situations, stress responsiveness, or past exposure to stressors, compared with those who do not volunteer for studies or those who are likely to participate in human service programs. Conducting studies of stress and self-regulation with population-based representative samples [418] would be helpful in identifying the real world “dosage” when chronic stress begins to turn toxic to self-regulation.

In sum, examining stress and self-regulation across ecological, biological, and developmental studies confirms important links between parenting, family context, environmental adversity and individual differences in the biological capacity to self-regulate. However, there are important gaps in the basic science knowledge base including 1) the causes and extent of variation in stress responsiveness across individuals, 2) whether particular developmental periods are more or less sensitive to stress, and 3) what protective factors may buffer an individual's responsiveness to stress. Increased research in these

areas within an ecological-biological-developmental model of stress may suggest directions for new intervention in the future. Such empirical studies can complement what is already known about the effectiveness of self-regulation interventions as will be reviewed in the next report in this series.

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APPENDIX A: SEARCH TERMS AND CRITERIA

1.1 Search Approach

Searches were conducted in the following databases: Thomson Reuters Web of Science (formerly ISI Web of Knowledge), PubMed, PsycInfo, EconLit, UMI Digital Dissertations, and JSTOR. Any article that met the search criteria and was published between 2009 and 2013 was included in the review. Finally, any papers cited in an article that met all necessary criteria was also extracted and included in the review as long as it also met the criteria.

1.2 Search Terms

Search terms are shown below. All articles included in the review included at least one element from List 1 (self-regulation) and one element from List 2 (stress-related outcomes and markers). (Note: “*” denotes a Boolean wildcard character).

List 1: Self-regulation related behavioral cognitive or mental health outcomes

- Direct References
 - Self regulation
 - Self control
 - Emotio* regulation
 - Behavio* regulation
 - Executive funct*
 - Cognitive flexibility
 - Cognitive control
 - Behav* inhib*
 - Inhib* behav*
 - Effortful control
 - Working memory
- Psychological Correlates
 - Social skills
 - Impulsiv*
 - Sensation seeking
 - Mindfulness
 - Social cognit*
 - Soci* emot*
 - Social competenc*
 - Emot* processing
- Neurocognitive Correlates
 - Attentio* control
 - Attention deficit
 - Visual attention
 - Spatial attention
 - Atten* bias
- Behavioral Correlates
 - Atten* task
 - Probl* behavior*
 - Delay AND (reward or gratification)
 - Intertemporal choice
 - Intertemporal pref*
 - Intertemporal discount*
 - Time pref*
 - Tempta*
 - Hyperbolic discounting
 - Time inconsistency
 - Dynam* inconsisten*
- Physiological Correlates
 - Prefrontal cort*
 - Frontal lobe
 - Frontal cort*
 - Amygdala
 - Limbic AND cort* AND develop*
 - White matter AND develop*
 - Reward system AND brain AND develop*
 - Function* connect* AND brain AND develop*
 - Neurotoxic*
 - Neuroendocrine

List 2: Stress-related outcomes and markers

- Direct References
 - Stress

-
- Stressor
 - Allostasi*
 - Physiological Correlates
 - Glucocorti*
 - Cortis*
 - Corticosteroid
 - Adrenocortic*
 - ACTH
 - Corticotropin
 - CRH
 - Hypothal*
 - Pituitary
 - Adren*
 - HPA
 - Inflammat*
 - Cytokine
 - Interleukin
 - Inflamm*
 - Family Correlates
 - Maltreatment
 - Insecur*
 - Disciplin*
 - Parenting
 - Adverse childhood experience
 - Negative life even*
 - Traum*
 - Socioeconomic Correlates
 - Depriv*
 - Psychology of scarcity
 - Poverty
 - Neighborhood
 - Violence
 - Under*resource*

1.3 Exclusion Criteria

Articles that included any of the following terms were excluded from the review.

- Irrelevant Stress Constructs
 - Oxidative stress
 - Stress fract*
 - Abiotic stress
 - Drought stress
 - Salinity stress
 - Mitochondrial stress
 - Osmotic stress
 - Shear stress
 - Apoptosis
 - Endoplasmic reticulum stress
 - Parenting stress
- Irrelevant Conditions
 - Schizophren*
 - Bipolar
 - Autism
 - Traumatic brain injur*
 - Traumatic injur*
 - Lesion
 - Multiple sclerosis
 - Epilep*
 - Alzheimer*
 - Closed head injur*
 - Borderline personality disorder
- Irrelevant Populations
 - Combat AND vetera*
 - Soldier*
 - Conscript* AND military
 - Termina* ill*

Also excluded from the review were:

- All papers that were not written in English
- Dissertations
- Any papers that were not empirical studies (i.e. editorials, lectures, commentaries)
- Any studies that are relevant exclusively to outcomes at stages of the life-course beyond late adolescence/early adulthood
- Any studies involving animal experiments that did not link stress to a specific neurocognitive function
- Any papers whose self-regulation-relevant constructs are restricted only to behaviors or whose stress-relevant constructs are restricted to only potential stressors