# Intergenerational Transmission of Self-Regulation: A Multidisciplinary Review and Integrative Conceptual Framework

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# Intergenerational Transmission of Self-Regulation: A Multidisciplinary Review and Integrative Conceptual Framework

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This review examines mechanisms contributing to the intergenerational transmission of self-regulation. To provide an integrated account of how self-regulation is transmitted across generations, we draw from over 75 years of accumulated evidence, spanning case studies to experimental approaches, in literatures covering developmental, social, and clinical psychology, and criminology, physiology, genetics, and human and animal neuroscience (among others). First, we present a taxonomy of what self-regulation is and then examine how it develops—overviews that guide the main foci of the review. Next, studies supporting an association between parent and child self-regulation are reviewed. Subsequently, literature that considers potential social mechanisms of transmission, specifically parenting behavior, interparental (i.e., marital) relationship behaviors, and broader rearing influences (e.g., household chaos) is considered. Finally, evidence that prenatal programming may be the starting point of the intergenerational transmission of self-regulation is covered, along with key findings from the behavioral and molecular genetics literatures. To integrate these literatures, we introduce the self-regulation intergenerational transmission model, a framework that brings together prenatal, social/contextual, and neurobiological mechanisms (spanning endocrine, neural, and genetic levels, including gene-environment interplay and epigenetic processes) to explain the intergenerational transmission of self-regulation. This model also incorporates potential transactional processes between generations (e.g., children's self-regulation and parent–child interaction dynamics that may affect parents' self-regulation) that further influence intergenerational processes. In pointing the way forward, we note key future directions and ways to address limitations in existing work throughout the review and in closing. We also conclude by noting several implications for intervention work.

*Keywords:* effortful control, self-control, executive functioning, emotion regulation, impulsivity, family dynamics, genetics

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Self-regulation (i.e., the flexible regulation of cognition, behavior, and emotion; Bandura, 1991; Berger, Kofman, Livneh, & Henik, 2007; Higgins, 1998; Karoly, 1993) has been widely studied from different disciplinary perspectives. Notably, poor self-regulation (SR) has consistently been found to play a critical role in a wide range of outcomes, contributing to substantial costs to society across health care, criminal justice, and educational systems. For instance, across the life span, poorer SR has been associated with greater risk for substance abuse (Fillmore & Rush, 2002), sexual risk taking (e.g., Quinn & Fromme, 2010), physical illness (e.g., obesity, diabetes, and accidental injury; Anzman-Frasca, Stifter, & Birch, 2012; Birch & Fisher, 1998; Graziano, Calkins, & Keane, 2010; Gunstad et al., 2007; Schwebel, 2004), and psychopathology, violence, and criminality (Beauchaine & McNulty, 2013; Carver, Johnson, & Joormann, 2008; DeWall, Baumeister, Stillman, & Gailliot, 2007; Eisenberg, Spinrad, & Eggum, 2010; Gottfredson & Hirschi, 1990; Nigg, 2000). SR also is associated with self-esteem and social and academic functioning (e.g., Blair & Diamond, 2008; Busch & Hofer, 2012; Eisenberg et al., 1997; Spinrad et al., 2006). Difficulties with SR in childhood are even longitudinally related to lower adulthood income and educational attainment (Moffitt et al., 2011; McClelland, Acock, Piccinin, Rhea, & Stallings, 2013).

Despite considerable evidence linking individual differences in SR to a range of outcomes, one area of inquiry has been relatively neglected—the direct association between parent SR and chil-

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dren's SR, and more specifically, the mechanisms by which SR is transmitted across generations. As such, our primary goal is to provide a comprehensive integration of established and emerging evidence of the link between parent and child SR, and the potential causal mechanisms by which such effects operate. Before tackling these subjects as the main foci of the review, we address two important foundational questions: What is SR, and how does it develop over childhood and adolescence into adulthood? With this information as a guide, we then review studies examining whether SR in parents is associated with SR in their children. Our view is that such associations are often assumed to exist (perhaps on the basis of heritability estimates from behavioral genetic studies), but are rarely tested in practice. It is necessary to first consider this basic question as it precedes any subsequent consideration of whether intergenerational transmission occurs and if so, how it occurs.

After reviewing evidence of relations between parent and child SR, we turn to the question of intergenerational transmission mechanisms. We begin by reviewing studies that consider relations between parent SR and key proximal (and potentially stressful) contextual influences (including parenting, interparental relationship/marital adjustment, and elements of the broader rearing context such as home chaos and family socioeconomic status [SES]) that have consistently been associated with children's SR, as we briefly note throughout the review. Included in this aspect of the review are studies demonstrating that these contextual factors can act as mediating social mechanisms of the intergenerational transmission of SR. Then, we selectively draw upon several supporting literatures that encompass studies of human personality (focusing on conscientiousness), human clinical literature (focusing on parents with attention deficit/hyperactivity disorder [ADHD]) and animal neuroscience findings that inform results in human samples. Rounding out coverage of supporting literatures, we briefly examine human neuropsychological case studies that closely parallel findings in the animal neuroscience literature. These supporting literatures provide additional correlational, quasi-experimental, and experimental evidence that bolsters our main conclusions regarding human cross-sectional and longitudinal correlational results. Subsequently, we cover emerging evidence from human and animal neuroscience studies suggesting that the effects of maternal SR on children's subsequent SR may start at the beginning of development in the womb via prenatal programming. We then provide an overview of key behavioral and molecular genetics literature that point to the vital role of genetic factors in the intergenerational transmission process.

Finally, we integrate the relevant, but heretofore separate literatures by introducing the self-regulation intergenerational transmission model. Using this conceptual model, we discuss how contextual factors, which are influenced by parent SR, can affect children's developing neurobiological mechanisms of SR, including the critical role of gene-environment interplay (including passive gene-environment correlation and potential epigenetic processes). We also consider social transaction mechanisms whereby parent SR is affected by family contexts and children's SR. In conclusion, as well as throughout the review, we note major gaps in knowledge and limitations to methods, pointing to promising directions for future work that will lead to new discoveries and further refinement of our understanding of the intergenerational transmission of SR. We end the review by highlighting implications for prevention and intervention efforts aimed at either preventing self-regulatory difficulties or enhancing SR.[1]

## Foundational Considerations

### What is Self-Regulation?

Although diverse terms are used to define SR, in the broadest sense it is a multifaceted aspect of temperament that is biologically based and heritable, but also shaped over time by the confluence of contextual influences and biologically based maturational processes (Eisenberg, Smith, & Spinrad, 2011; Posner & Rothbart, 2000; Rothbart & Bates, 1998; Shiner et al., 2012) as seen in humans and many other species (see Barr, 2012 and McCrae et al., 2000 for discussion). For example, in addition to extensive study in human samples, self-regulatory processes have been studied in nonhuman primates (e.g., Goursaud & Bachevalier, 2007; James et al., 2007; Kalin, Shelton, & Davidson, 2004; Shultz & Dunbar, 2010), rodents (e.g., Afonso, Sison, Lovic, & Fleming, 2007; Dalley, Cardinal, & Robbins, 2004; Davis, Walker, Miles, & Grillon, 2010), canines (e.g., Bray, MacLean, & Hare, 2014), and even fish (Parker, Brock, Walton, & Brennan, 2013), to name just a few.

At a more precise level, SR can be differentiated into two more specific behaviorally and neurobiologically separable, but interacting components: "top-down" SR, reflecting more effortful/executive control processes served by cortical structures and the anterior cingulate cortex (ACC), and "bottom-up" SR reflecting more automatic (reactive) processes served primarily by subcortical structures. At an even finer-grained level, "top-down" SR can be further differentiated into two subcomponents, behavioral and emotional SR, based on some separable neurobiological mechanisms (although they also share certain neurobiological components). Likewise, "bottom-up" reactive SR differentiates further into behavioral inhibition/fear (i.e., behavioral overcontrol) and impulsivity (i.e., behavioral undercontrol). Having broadly described and introduced SR, we now turn to overviews of each aspect of top-down and bottom-up regulatory processes, focusing on conceptual definitions, and operational definitions at the level of behavior and neurobiology.

#### Top-down self-regulatory processes.

***Behavioral SR.*** Behavioral SR constructs usually are assessed with questionnaires, laboratory and computerized tasks, or neuropsychological assessments. These constructs include effortful control (Eisenberg et al., 2011; Rothbart, Ellis, Rueda, & Posner, 2003), self-control (Gottfredson & Hirschi, 1990), and executive functioning, with the latter consisting of three core interrelated processes—working memory, inhibitory control, and attention shifting/cognitive flexibility (Miyake & Friedman, 2012; Miyake et al., 2000). Effortful control and the underlying executive attention network (Petersen & Posner, 2012; Posner, 2012) are frequently discussed within the developmental literature as concepts stemming from the psychobiological model of temperament (Roth-

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[1] Because of the extensive ground we need to cover to support a conceptual framework of the complex processes involved in the intergenerational transmission of self-regulation that we propose, when available, we point to important (mostly recent) reviews that provide more depth and breadth of coverage for selected topics throughout our review.

bart & Derryberry, 1981) and the even earlier concepts of ego resiliency and ego control in theories of childhood personality (Block, 1950; Block & Block, 1980). In contrast, executive functioning is most often discussed within the confines of the neuroscience and neuropsychology literatures, but increasingly it is being considered within the developmental literature. Finally, self-control, particularly as defined in the criminology literature, reflects adequate impulse control and attention (e.g., Boutwell & Beaver, 2010; Nofziger, 2008).

Although the behavioral SR constructs noted here are often discussed in the literature as potentially distinct entities, there is growing consensus of considerable similarity if not actual overlap between them at conceptual and behavioral levels (see Bridgett, Oddi, Laake, Murdock, & Bachmann, 2013, and Beaver, Wright, & Delisi, 2007). In addition, there is overlap among behavioral SR constructs at the neurobiological level. For example, the dorsal ACC (dACC) and the dorsolateral prefrontal cortex (dlPFC) have been identified as playing notable roles in effortful control and executive attention (Fan, McCandliss, Fossella, Flombaum, & Posner, 2005; Posner, 2012) as well as working memory (e.g., Chein, Moore, & Conway, 2011; Wager, Spicer, Insler, & Smith, 2014). The ventrolateral prefrontal cortex (vlPFC; Spitzer, Goltz, Wacker, Auksztulewicz, & Blankenburg, 2014; Wager et al., 2014) and the orbitofrontal cortex (OFC; e.g., Barbey, Koenigs, & Grafman, 2011) are also implicated in working memory.

Unlike effortful control and working memory, inhibitory control (i.e., impulse control) has been associated with the right inferior frontal cortex (e.g., Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013; Simmonds, Pekar, & Mostofsky, 2008; Tabibnia et al., 2011). However, like effortful control and working memory, inhibitory control has been associated with activation in the dlPFC, the ACC, and the ventral PFC (Borst et al., 2014; Criaud & Boulinguez, 2013; Hart et al., 2013). Neurobiological mechanisms of cognitive flexibility also have been articulated, with studies pointing to the dlPFC and medial PFC (mPFC) in attention shifting (e.g., Konishi et al., 2010)—areas also implicated in other behavioral SR processes. However, additional areas such as the inferior frontal junction and the superior frontal sulcus also have been noted to play roles in cognitive flexibility (e.g., Kim, Cilles, Johnson, & Gold, 2012; Kim, Johnson, Cilles, & Gold, 2011; Stelzel, Basten, & Fiebach, 2011).

***Emotional SR.*** In contrast to behavioral SR, emotional SR typically has been examined in the context of specific emotion regulatory strategies such as reappraisal or suppression (Gross & Thompson, 2007) via questionnaires or laboratory based assessments (e.g., Troy, Shallcross, & Mauss, 2013), or with the use of cardiac biomarkers such as heart rate variability (HRV), respiratory sinus arrhythmia (RSA) and vagal tone (Beauchaine, 2012; Beauchaine, Gatzke-Kopp, & Mead, 2007; Calkins, 1997; Porges, 1995, 2007), which are indices of parasympathetic nervous system function. Reappraisal allows one to modulate emotional experience through cognitive reframing (see Gross, 2001, 2002 for more discussion). Suppression also modulates the effects of emotion on behavior by reducing an individual's outward expression of emotion, but not the internal experience of emotion (Gross & John, 2003). In regard to RSA, "resting" RSA reflects individual differences in emotion-related physiological arousal, with higher resting RSA (as well as other cardiac markers, such as HRV and vagal tone) being associated with better emotional SR. Beyond the resting state, changes in RSA in response to emotional challenges reflect active regulation of emotional arousal, which also is a marker of better emotional SR (Beauchaine, 2001; Thayer, Hansen, Saus-Rose, & Johnsen, 2009; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009). Although we have highlighted reappraisal, suppression, and cardiac dynamics in this review because they are the most extensively studied aspects of emotional SR in adults, there are other forms of emotional SR such as distraction, gaze aversion, and physical self-soothing that are frequently (but not exclusively) studied in children (e.g., Grolnick, Bridges, & Connell, 1996; Stifter & Spinrad, 2002).

Just as with behavioral SR, there is a rich understanding of the neurobiological underpinnings of emotional SR. A recent meta-analysis (Buhle et al., 2014) of 48 neuroimaging studies associated activation in the dlPFC, the vlPFC, and the dorsal medial prefrontal cortex (dmPFC) with reappraisal. An earlier meta-analysis also reported activation of the ventral medial prefrontal cortex (vmPFC) during reappraisal (Diekhof, Geier, Falkai, & Gruber, 2011). Additional areas important for reappraisal also have been noted, such as the ACC and OFC, though these areas have been less consistently mentioned in the literature (e.g., Giuliani, Drabant, & Gross, 2011; Kanske, Heissler, Schonfelder, Bongers, & Wessa, 2011).

Other aspects of emotional SR also have received some attention. McRae et al. (2010) found that reappraisal and distraction activated the left PFC, right inferior frontal cortex and dACC, but that distraction also led to activation of areas important for attention allocation and to relatively greater down regulation of amygdala activity. Regarding suppression, Kuhn, Gallinat, and Brass (2011) linked gray matter volume in the dmPFC to suppression, and another study reported activation of the right inferior frontal gyrus when participants engaged in suppression (Vanderhasselt, Kuhn, & De Raedt, 2013). Finally, although an overview of the complex physiological underpinnings of how top-down emotional SR mechanisms influence RSA is beyond the scope of the current review (see Thayer & Lane, 2009), top-down areas implicated in RSA include the superior PFC, the ACC, right dlPFC, as well as areas in the parietal cortex (Lane et al., 2009; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Thus, although at first glance RSA may seem unrelated to top-down mechanisms of SR, RSA is connected via physiological pathways to top-down structures implicated in the use of specific emotional SR strategies (e.g., reappraisal). In sum, the neurobiological origins of behavioral and emotional SR appear to be heavily concentrated in the frontal lobes, and include the ACC.

**Bottom-up self-regulatory processes.**

***Behavioral inhibition/fear.*** In contrast to top-down effortful/executive mechanisms, behavioral inhibition/fear is a reactive "overcontrolled" type of SR (Aksan & Kochanska, 2004; Eisenberg et al., 2013). Individuals higher in this aspect of SR tend to be more cautious and reserved, particularly when confronted with novel situations. Such individuals also are characterized as being more fearful and shy (Calkins, Fox, & Marshall, 1996; Kagan, 1997; Kagan & Snidman, 1999). When behavioral inhibition/fear is elevated, individuals are at greater risk of developing anxiety, and particularly social anxiety (e.g., Clauss & Blackford, 2012). As with behavioral and emotional SR, this aspect of SR has its roots in the temperament and personality literatures, particularly the work of Gray (Gray, 1970, 1987; Gray & McNaughton, 2000).

In children, behavioral inhibition/fear is often assessed on the basis of behavioral reactions during laboratory tasks (e.g., Gartstein et al., 2010), physiologically (e.g., Fox, Henderson, Marshall, Nichols, & Ghera, 2005), and with questionnaires completed by parents, teachers, or other caregivers or via self-report (Gartstein, Bridgett, & Low, 2012). Similarly, in adults, behavioral inhibition/fear is assessed using physiological approaches (e.g., Balconi & Mazza, 2009) and self-report measures (e.g., Carver & White, 1994).

Like other aspects of SR, the neurobiological underpinnings of behavioral inhibition/fear have been studied extensively. The subcortical structures most consistently implicated include the amygdala—particularly the central nucleus—and hippocampus (Beaton et al., 2008; Davidson, Jackson, & Kalin, 2000; Kalin et al., 2004; Kennis, Rademaker, & Geuze, 2013; Oler et al., 2010). Although these structures have received the greatest attention, there are other subcortical areas that have been implicated in behavioral inhibition/fear. For example, some work has identified the bed nuclei of the stria terminalis (e.g., Oler et al., 2012) and the caudate and basal ganglia (e.g., Clauss et al., 2014). For a recent review that covers the breadth of subcortical structures involved in behavioral inhibition/fear, see Kennis, Rademaker, and Gueze (2013).

***Impulsivity.*** Whereas behavioral inhibition/fear is a reactive, overcontrolled type of SR, impulsivity can be characterized as a reactive, undercontrolled type of SR (e.g., Eisenberg et al., 2004; Eisenberg et al., 2013; Kagan, Reznick, & Gibbons, 1989) that also has long-standing roots in the temperament and personality literatures (e.g., Barratt & Patton, 1983; Carver, 2005; Strelau, 1987). Individuals high in impulsivity tend to react without much if any forethought or consideration of the potential longer-term implications of their behaviors. As such, when described in the literature, impulsivity in the sense described here also is referred to as disinhibition, dysfunctional impulsivity, or impulsive decision making (Dickman, 1990; Sharma, Markon, & Clark, 2014),[2] and in a more extreme form, is characteristic of hyperactivity-impulsivity symptoms seen in those diagnosed ADHD and as a component of other externalizing difficulties (e.g., Beauchaine & McNulty, 2013; Martel & Nigg, 2006; Winstanley, Eagle, & Robbins, 2006). Like other aspects of SR, impulsivity can be assessed using laboratory and computerized tasks as well as a variety of self- and other-report measures (Sharma et al., 2014). Studies that have examined the neurobiological mechanisms of impulsivity have consistently identified the ventral striatum, and within this area the caudate nucleus, nucleus accumbens, and putamen (Besson et al., 2010; Buckholtz et al., 2010; del Campo et al., 2013; Plichta & Scheres, 2014; van Duijvenvoorde et al., 2014) as neural areas contributing to impulsive behaviors. Other subcortical areas also have been implicated in impulsive behavior, including the ventral tegmental area and the substantia nigra (Bourdy & Barrot, 2012; Gatzke-Kopp & Beauchaine, 2007; Tomasi & Volkow, 2014; Whelan et al., 2012).

***Summary, future directions, and foundational considerations for review.*** We have described a taxonomy for understanding SR broadly and at a finer-grained level. At the most specific level, there are two distinct but related top-down SR processes (i.e., behavioral and emotional SR) originating from neural structures within the frontal lobes and the ACC. We also identified two aspects of bottom-up SR (i.e., behavioral inhibition/fear and impulsivity) served by subcortical structures. On the basis of this information, within the main foci of our review we emphasize literature examining behavioral inhibition/fear, impulsivity, and behavioral (e.g., effortful control and executive functioning) and emotional SR (e.g., reappraisal and RSA). However, there is room for further refinement. Specifically, there have been recent calls for theoretical and empirical integration across aspects of behavioral SR that traditionally have been considered as distinct components (e.g., Bridgett, Oddi et al., 2013; Zhou, Chen, & Main, 2012). Our overview also points to similarity (e.g., common neural structures) between behavioral and emotional SR. In light of recent calls for integration, and our observation of some overlap between behavioral and emotional SR, future work should continue to focus on points of convergence and divergence, across levels of analysis, among aspects of top-down SR. Finally, within this section we presented each aspect of SR separately for purposes of clarity, representing a limitation to our overview. However, next we turn to how SR processes develop and interact and the implications such information has for our review.

## Development of and Interplay Between Self-Regulatory Systems

**Development of bottom-up self-regulation.** Bottom-up self-regulatory processes emerge early in life and reach moderate to high rank-order stability (across studies, ranging from .32 to .72 for impulsivity and from .20 to .66 for behavioral inhibition/fear) by the time children are approximately 3 years of age, if not sooner (e.g., Aksan & Kochanska, 2004; Eisenberg et al., 2013; Garcia-Coll, Kagan, & Reznick, 1984; Karevold, Ystrom, Coplan, Sanson, & Mathiesen, 2012; Pfeifer, Goldsmith, Davidson, & Rickman, 2002; Putnam, Rothbart, & Gartstein, 2008; Putnam & Stifter, 2005). Fairly high (ranging from .55 to .77) levels of stability of behavioral inhibition/fear (e.g., Eggum et al., 2012) and impulsivity (e.g., Eisenberg et al., 2004) are typical by the time children are 6 to 10 years of age. By late adolescence to early adulthood, behavioral inhibition/fear and impulsivity exhibit high rank-order stability (across studies, ranging from .58 to .84 for impulsivity and .57 to .81 for behavioral inhibition/fear; Arneklev, Cochran, & Gainey, 1998; Gest, 1997; Hopwood et al., 2013; Meldrum, Young, & Weerman, 2012; Windsor, Pearson, & Butterworth, 2012). Although both show the same pattern of rank-order stability in individual differences, behavioral inhibition/fear and impulsivity show distinct patterns of typical mean-level change (i.e., ontogeny). Mean levels of impulsivity rise over late infancy and toddlerhood, peak by 3 years of age, then decline (e.g., Graziano, Keane, & Calkins, 2010; Shaw, Lacourse, & Nagin, 2005) over childhood and adolescence (e.g., Collado, Felton, MacPherson, & Lejuez, 2014; Eisenberg et al., 2007; Eisenberg et al., 2004; Pedersen, Molina, Belendiuk, & Donovan, 2012) into early adulthood before mean stability is attained. For instance, in a sample ($N = 7{,}640$) followed from 12 to 24 years of age, average declines

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[2] Impulsivity does not appear to be a unitary construct as the colloquial usage of the term implies. Rather, existing work suggests that impulsivity is multifaceted (see Sharma, Markon, & Clark, 2014 for a review). Our decision to focus on impulsivity as described in this section was driven by the descriptions of impulsivity within the literature that is the focus of our review. We note this as a limitation and point to future directions later in the review.

in impulsivity were found from 12 to 22 years, with mean-level stability appearing by 24 years (Harden & Tucker-Drob, 2011; also see Quinn & Harden, 2013).

Similar to impulsivity, there are increases in average behavioral inhibition/fear during infancy (e.g., Gartstein et al., 2010) and over early childhood until 4 to 6 years of age (e.g., Karevold et al., 2012). Findings generally converge on an age between 6 and 12 years as the time frame during which behavior inhibition/fear stabilizes and reaches horizontal asymptote (e.g., Côté, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002; Karevold et al., 2012). Although there are individual differences in mean-level changes, and variability across studies (e.g., see Lengua, 2006 who reported significant mean-level change in behavioral inhibition/fear), mean-level change is typically small when there are significant increases or decreases in behavioral inhibition/fear and most children exhibit mean-level stability by late childhood to early adolescence. For example, Oh et al. (2008) followed a large sample of children from 10 to 14 years of age and found that 85% exhibited stable mean levels of behavioral inhibition/fear; only 15% exhibited increasing (7%) or decreasing (8%) mean-levels (also see Dennissen, Asendorpf, & van Aken, 2008 for similar findings). By early adulthood, mean level behavioral inhibition/fear appears to be quite stable. For instance, in a large longitudinal study ($N = 7{,}468$) of three age cohorts (young, middle-age, and older adults), Windsor, Pearson, and Butterworth (2012) concluded that behavioral inhibition/fear exhibits mean-level stability throughout adulthood though with very small perturbations (i.e., slight increase in early adulthood, slight decrease in old age).

Like the behavioral literature, studies examining the development of neural structures and functions in areas important for bottom-up regulatory processes show evidence of rapid changes early in development followed by *relative* stability by middle childhood to early adolescence (see Krogsrud et al., 2014; Pagliaccio et al., 2013; and Payne, Machado, Bliwise, & Bachevalier, 2010 regarding hippocampus and amygdala). Nevertheless, findings regarding maturation of bottom-up structures are not always consistent. Specifically, some work notes changes across development in the striatum, including the nucleus accumbens, amygdala, and hippocampus—but particularly before and again during adolescence through early adulthood (e.g., Dennison et al., 2013; Raznahan et al., 2014; Wierenga et al., 2014). However, it is equally important to add that structural and functional changes may only be modestly related (see Pine, 2007). For example, there is evidence that the amygdala has adult-like functionality fairly early in life (see Pine, 2003, 2007 for overviews), which is consistent with behavioral evidence of early maturation of bottom-up behavioral inhibition/fear. In contrast, bottom-up areas implicated in impulsivity demonstrate functional and structural changes as late as adolescence (for a review, see Smith, Chein, & Steinberg, 2013)—findings that are consistent with behavioral evidence of impulsivity maturing later than behavioral inhibition/fear.

**Development of top-down self-regulation.** Similar to bottom-up processes, top-down SR emerges and can be assessed in the first year of life. For example, the executive attention network underlying effective effortful control is functional by the time children are 12 months of age (Derryberry & Rothbart, 1997; Rothbart, Sheese, & Posner, 2007), with even earlier developing attentional processes preceding and supporting its emergence (e.g., Gartstein, Bridgett, Young, Panksepp, & Power, 2013; Papageorgiou et al., 2014). Similarly, executive functioning emerges in the first year and can be assessed as early as 6 to 12 months of age using measures such as the classic A-not-B task (Best & Miller, 2010; Best, Miller, & Jones, 2009; Diamond & Doar, 1989; Sun, Mohay, & O'Callaghan, 2009). Emotional SR also can be reliably assessed in the first year of life. Although children are heavily reliant on caregivers for external support in regulating emotional states during infancy and toddlerhood (Calkins & Hill, 2007; Kopp, 1982, 1989), infants begin utilizing self-initiated emotional SR (e.g., self-distraction, gaze aversion, and physical self-soothing) by 6 months of age, and increase their effective and flexible use of these strategies thereafter (Calkins & Hill, 2007; Grolnick et al., 1996; Kopp, 1982, 1989; Stifter & Spinrad, 2002). Subsequently, as children develop, they are increasingly able to employ more complex emotional SR strategies (e.g., reappraisal; McRae et al., 2012). In addition, RSA and related cardiac indices of emotional SR can be measured in infancy and are related to reactive and regulatory aspects of temperament (e.g., Brooker & Buss, 2010; Poehlmann et al., 2011) and to subsequent behavioral and emotional difficulties (e.g., Conradt, Measelle, & Ablow, 2013; Dale et al., 2011; El-Sheikh & Erath, 2011).

Although top-down SR processes emerge and can be reliably assessed at approximately the same time as bottom-up processes, relative to the more rapidly maturing bottom-up processes, top-down SR undergoes a more protracted period of development. In a study that assessed executive functioning five times between 5-months and 4-years of age, rank-order stability of individual differences was modest, ranging from .03 to .19 (Wolfe, Zhang, Kim-Spoon, & Bell, 2014). Larger but still modest rank-order stability of executive functioning, ranging from .24 to .28, have been reported from 18 to 26 months of age (Bernier, Carlson, & Whipple, 2010). Higher still, parent report of infant regulation-related processes that precede effortful control have ranged from .33 over an 8-month interval to .63 to .78 over 2-month intervals in a sample followed from 4 to 12 months of age (Bridgett et al., 2011). Anchoring the upper end of estimates, Eisenberg et al. (2013) followed children from 2.5 to 4.5 years of age and used a multimethod latent variable approach to assess effortful control. They reported stability coefficients ranging from .67 to .82 (see Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996 for similar findings regarding effortful control, Fuhs & Day, 2011, for similar findings in relation to executive functioning, and Deater-Deckard, 2014, for a summary regarding executive function and attention).

Emotional SR also exhibits modest to moderate rank-order stability early in development. The rank order stability of RSA and vagal tone ranges from .20 to .47 across intervals as short as 6 months to as long as 4.5 years in samples spanning 6 months to 5 years of age (e.g., Alkon, Boyce, Davis, & Eskenazi, 2011; Perry, Mackler, Calkins, & Keane, 2014). Nevertheless, there is substantial variability across studies of young children. For instance, Porter, Bryan, and Hsu (1995) assessed vagal tone three times when children were 1 to 6 months of age and found that rank order stability ranged from .12 to .39, whereas Propper et al. (2008) reported the stability of resting RSA to be .68 between 6 and 12 months of age (also see Porges, Doussard-Roosevelt, Portales, & Suess, 1994 who reported similar levels of stability over 3 years in early childhood).

Although less common, studies have considered the stability of behavioral and emotional SR in older children, adolescents, and young adults. In a study of 9- to 15-year-olds' wherein emotional SR was assessed three times annually, the rank order stability of reappraisal ranged from .28 to .42, and for suppression ranged from .35 to .50 (Gullone, Hughes, King, & Tonge, 2010). Lengua's (2006) study of effortful control from 8 to 12 years of age showed stability coefficients ranging from .68 to .80 for child–parent report composites. These findings are consistent with the stability of adolescent self-control, which ranged from .44 (5-year interval) to .68 (yearly intervals) in a study that followed adolescents from 12 to 17 years of age (Winfree, Taylor, He, & Esbensen, 2006). However, although rank-order stability typically increases with age, there is variability across studies. For instance, in a sample assessed at 8 and 12 years of age, the stability of inhibitory control was modest, ranging from .28 to .38 (Harms, Zayas, Meltzoff, & Carlson, 2014; see Biederman et al., 2007 for an example of modest stability of executive functioning in samples of adolescent girls).

By early adulthood, the rank-order stability of emotional SR is moderate to high. Among adults, RSA at baseline as well as during emotionally evocative tasks showed stability coefficients from .51 to .63 over 10 months in one study (Uchino, Holt-Lunstad, Bloor, & Campo, 2005), and a 5-year stability coefficient of .74 in another (Bornstein & Suess, 2000). Reappraisal shows similarly strong rank-order stability over 3 months (.70; Gross & John, 2003) and 1 year (.57; Garnefski & Kraaij, 2007). The stability of emotional SR constructs is greatest (>.70) when multi-informant or method-latent variable constructs are used (Berking, Wirtz, Svaldi, & Hofmann, 2014). Although studies of rank-order stability of behavioral SR in adults are rarer than studies of emotional SR, the 1 to 2 month stability of a latent variable of executive functioning in adults ages 50 to 80 was .95 in one study (Ettenhofer, Hambrick, & Abeles, 2006), providing support for very high rank order stability of executive functioning in adults.

Turning to mean-level change (i.e., ontogeny), increases in behavioral SR (including executive functioning, effortful control, and self-control) have been noted across samples of young children (e.g., Fuhs & Day, 2011; Hongwanishkul, Happaney, Lee, & Zelazo, 2005; Li-Grining, 2007), including infants (e.g., Cuevas & Bell, 2010), school-age children (e.g., Bridgett & Mayes, 2011; Lee, Bull, & Ho, 2013; Lengua, 2006; Valiente et al., 2006; Vazsonyi & Huang, 2010), and adolescents (e.g., Luciana, Conklin, Hooper, & Yarger, 2005; Turner & Piquero, 2002). Although gradual, improvements in behavioral SR persist through the mid-20s (e.g., Cepeda, Kramer, & Gonzalez de Sather, 2001; Chevalier, Huber, Wiebe, & Espy, 2013; Fosco, Caruthers, & Dishion, 2012; Huizinga, Dolan, & van der Molen, 2006). Similarly, aspects of emotional SR, including RSA and reappraisal, also exhibit changes across development, showing a pattern of mean level increases over time (Alkon et al., 2011; El-Sheikh, 2005; Gentzler, Rottenberg, Kovacs, George, & Morey, 2012; Hinnant, Elmore-Staton, & El-Sheikh, 2011; Hollenstein, McNeely, Eastabrook, Mackey, & Flynn, 2012; McRae et al., 2012; Sala, Pons, & Molina, 2014; Sang, Deng, & Luan, 2014; Silvers et al., 2012) until mean-level stability is attained at approximately the same time in early adulthood as behavioral SR (Mathewson et al., 2010; Silvers et al., 2012; Zimmermann & Iwanski, 2014; but, see Gullone et al., 2010 who noted mean-level stability of reappraisal in adolescents and young adults), if not slightly earlier (i.e., by mid- to late adolescence) in the case of RSA (Salomon, 2005).

At the neurobiological level, mean increases in top-down SR generally map onto developmental changes to top-down neurobiological structures. In short, brain maturation in areas important for top-down SR occurs most rapidly between birth and 5–8 years of age (Brown & Jernigan, 2012). Although some continued maturation occurs throughout childhood, another major period of development occurs during adolescence (Brain Development Cooperative Group, 2012; Petanjek, Judas, Kostovic, & Uylings, 2008). These changes in early childhood and adolescence occur at cellular, structural, and functional levels. As such, over time there is a complex pattern of increases and decreases in gray and white matter (e.g., increased gray matter early in development followed by decreases in adolescence) that affect the size and functionality of top-down structures implicated in SR (Gogtay & Thompson, 2010; Lenroot & Giedd, 2006; McRae et al., 2012; Petanjek et al., 2011; Tamnes et al., 2013; Vijayakumar et al., 2014; Zhong et al., 2014; for more in depth reviews of this topic see Best & Miller, 2010; Dubois et al., 2014; Brown & Jernigan, 2012).

**Developmental interplay between bottom-up and top-down SR.** Across development, there also is a complex, shifting interplay between bottom-up and top-down processes. In regard to neurobiological structures and functions, improvements in the connectivity between bottom-up and top-down structures result in improvements in SR (see Alexander-Bloch, Giedd, & Bullmore, 2013; Gabard-Durnam et al., 2014; Khundrakpam et al., 2013; Lebel & Beaulieu, 2011; Luna, Padmanabhan, & O'Hearn, 2010; Zhong et al., 2014 for further discussion). Such changes are associated with a shift from relatively greater (but not exclusive) influence of bottom-up SR on behavior earlier in development, toward greater (but again, not exclusive) influence of top-down SR on bottom-up reactive SR and behavior later in development (see Rubia, 2013 for more discussion). Thus, by the time individuals have transitioned into adulthood, frontal structures play an important role in the top-down regulation of striatal function (e.g., van Schouwenburg, O'Shea, Mars, Rushworth, & Cools, 2012), including the nucleus accumbens (e.g., Cohen et al., 2012; Diekhof & Gruber, 2010), the ventral tegmental area (e.g., Ballard et al., 2011; Patton, Bizup, & Grace, 2013), and the amygdala (e.g., Diekhof et al., 2011; Motzkin, Philippi, Wolf, Baskaya, & Koenigs, 2014), as well as the regulation of other subcortical areas (e.g., dorsal raphe nucleus; Amat et al., 2005).

The shifting interplay between top-down and bottom-up neurobiological mechanisms also can be seen at the behavioral level. Across age groups, poorer top-down behavioral and emotional SR have been consistently associated with more impulsivity (Carranza, Gonzalez-Salinas, & Ato, 2013; Eisenberg et al., 2004; Enticott, Ogloff, & Bradshaw, 2006; Gagne, Saudino, & Asherson, 2011; Romer et al., 2009; Schreiber, Grant, & Odlaug, 2012; Schwebel, 2004). However, the interplay between top-down and bottom-up processes involving behavioral inhibition/fear is more complex. Some studies have reported positive relations between behavioral inhibition/fear and top-down SR in young children (e.g., Eisenberg et al., 2013; Thorell, Bohlin, & Rydell, 2004), suggesting that behavioral inhibition/fear may play a role in the emergence of top-down SR (see Aksan & Kochanska, 2004 or Kochanska & Knaack, 2003 for discussion). However, some investigators have not found such associations (e.g., Hastings et al., 2008; Sulik, Eisenberg, Silva, Spinrad, & Kupfer, 2013; Volbrecht & Goldsmith, 2010) and others have reported negative relations (e.g., Blankson, O'Brien, Leerkes, Marcovitch, & Calkins, 2011). In contrast to some inconsistency in findings within the early childhood literature, relations, such that top-down SR is inversely related to behavioral inhibition/fear, are more consistent in older children, adolescents, and adults (e.g., Affrunti & Woodruff-Borden, in press; Muris & Meesters, 2009; Schmidt & Fox, 1994; Wilson, Lengua, Tininenko, Taylor, & Trancik, 2009; Wolgast, Lundh, & Viborg, 2011). These behavioral level findings are consistent with the neurobiological shift from relatively more bottom-up to greater top-down influence on behavior and emotion across development.

Finally, there is a developmentally complex interplay between bottom-up processes at the neurobiological level. There is growing interest in connectivity between the amygdala (particularly the basolateral amygdala) and the nucleus accumbens, with evidence emerging that the amygdala assists in regulating impulsive reward-seeking behaviors in addition to its role in behavioral inhibition/fear (e.g., Gill & Grace, 2011, 2013; Wassum et al., 2012; Zeeb & Winstanley, 2011; also see Abraham, Neve, & Lattal, 2014 for related discussion). However, much of the work on subcortical circuitry (e.g., amygdala-striatal connectivity) is based on animal models, and advances in technology have only recently allowed finer-grained study (e.g., examination of specific nuclei or parts of subcortical structures) of human subcortical structures and their interconnectivity (see Gopal et al., 2013 and van Honk, Eisenegger, Terburg, Stein, & Morgan, 2013 for examples). Given that work in this area in humans is just now emerging, investigation of the interplay between the various aspects of bottom-up SR, and what the implications of interconnectivity among subcortical structures might mean for top-down processes, is an important direction for work in future human studies.

**Summary, implications for review, and recommendations for future work.** There are similarities and differences in the development of the various aspects of SR. Bottom-up and top-down SR emerge in the first year of life. However, across several levels of analysis (e.g., behavioral and neurobiological), behavioral inhibition/fear completes development earlier than impulsivity. Relative to bottom-up processes, behaviorally based findings of rank-order and mean level stability, along with developmentally sensitive neuroimaging studies converge on a time frame around the mid-20s as the approximate point in development when behavioral and emotional SR reach maturity. Our conclusions about the developmental unfolding of SR have two major implications for the main foci of our review. Most importantly, by the time many (though clearly not all) adults begin raising their own children,[3] they have mature or nearly mature self-regulatory mechanisms that can be employed to meet their children's needs in ways that support the development of SR in the next generation. That is, to be effective caregivers, we would assert that parents must be able to flexibly employ different self-regulatory processes to inhibit impulsive decision making, regulate their affect and behavior, and effectively structure and support the child rearing context in ways that promote, and not hinder, their child's development of SR. Second, thoughtful consideration of the development of SR conveys essential information about developmental windows of opportunity during which intergenerational transmission of SR may be most powerful. On the basis of behavioral and neurobiological evidence, these windows may be during early childhood and adolescence.

Although our conclusions about the development SR processes are well-supported and the implications for our review clear, our overview points to future directions and areas in need of further empirical consideration. In particular, when only considering rank-order stability, it appears that behavioral inhibition/fear and impulsivity reach high levels of stability in early childhood. In contrast, if one only considers mean-level stability, development continues through early adulthood for impulsivity and top-down processes. It is only by considering both types of stability/change—individual differences and average levels—that the more nuanced pattern of development across top-down and bottom-up SR processes become clear. This has major implications for future work regarding decisions about the timing and frequency of the measurement of SR. Specifically, relying on single assessments (rather than longitudinal assessments appropriately timed for the SR constructs in question) is ill advised in developmental studies.

Finally, it is clear that estimates of rank-order stability may be attenuated and difficult to replicate when based on single informants and/or methods. All too rarely used, approaches yielding the highest and arguably most replicable estimates of stability are those accounting for measurement error (see Hopwood et al., 2013 for an example in a sample of adults), or those that use either latent or manifest variable approaches involving multiple measures and/or informants (see Eisenberg et al., 2013; Fuhs & Day, 2011; Kochanska et al., 1996 for examples in samples of young children). When such approaches are employed, even the rank-order stability of young children's top-down SR (e.g., effortful control) can show adult-like levels. Because many existing studies rely on single method or informant measurement, estimates of rank-order stability for bottom-up and top-down regulatory processes are suspect, making it hard if not impossible to make comparisons between bottom-up and top-down estimates of stability. Consequently, the field needs to revisit important questions about the rank-order stability of SR by employing more rigorous measurement modeling (e.g., using multiple methods, measures and informants for each aspect of SR) that will permit more reliable estimation of and conclusions about the rank-order stability and change over development of SR.

## Associations Between Parent and Child Self-Regulation

Having considered important foundation points for our review, we now turn to evidence that SR is transmitted across generations. Herein, we start building toward a framework for understanding the intergenerational transmission of SR by reviewing studies that report relations between parent and child SR. Although the question of whether parent and child SR are related is seemingly basic and innocuous in some regards, it is of paramount importance to

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[3] Given evidence that the major development of neurobiological mechanisms of SR are largely completed by the mid-20s, and that the average age of childbearing has increased (e.g., in the US, the mean age of mothers at the time of having their first child in 2012 was 25.8 years; Martin et al., 2013), at least in Western, industrialized countries, it seems that neurobiological mechanisms of SR are optimal, in most, but certainly not all cases (e.g., teen parents), for adults to utilize them in the service of child rearing activities.

determine whether such relations exist before considering potential explanations (i.e., mechanisms) for such associations.

## Review of Studies Reporting Direct Relations

Consistent with the notion that parent SR should be associated with children's SR, we identified a number of studies that reported such relations (see Online Supplemental Table 1). For instance, Bridgett et al. (2011) reported a concurrent positive relation between maternal effortful control and infant orienting/regulation and a longitudinal positive association with toddler effortful control. Similarly, Bridgett, Burt, Laake, and Oddi (2013) reported a positive association between maternal behavioral SR, assessed using a multimethod approach, and infant falling reactivity, an early manifestation of emotional SR, 2 months later. Likewise, Cumberland-Li, Eisenberg, Champion, Gershoff, and Fabes (2003) reported a negative relation between maternal effortful control and children's cheating behaviors (i.e., lower behavioral SR) during a lab task. In regards to executive functioning, Cuevas et al. (2014a) noted positive associations between mother and child executive functioning, which was longitudinally assessed multiple times using laboratory tasks. Although studies of younger children are more common, findings from studies of school-aged and adolescent samples that have considered executive functioning or effortful control (Jester et al., 2009; Valiente, Lemery-Chalfant, & Reiser, 2007) are consistent with findings in studies of younger children in showing anticipated relations between parent and child behavioral SR.

With regard to emotional SR, Bornstein and Suess (2000) reported positive relations between maternal and child vagal tone when children were 2 months and 5 years of age. However, two later studies did not find significant relations between parent and child cardiac bio-markers of emotional SR (Moore et al., 2009; Perlman, Camras, & Pelphrey, 2008). Nevertheless, studies using other methods to assess emotional SR have consistently reported expected relations. Buckholdt, Parra, and Jobe-Shields (2013) reported a positive association between parent and adolescent self-reported emotional dysregulation (also see Sarıtaş & Gençöz, 2012, who reported similar findings). Likewise, in a study of school-age children, positive relations between maternal and child emotional SR were reported (Han & Shaffer, 2013; also see Samuelson, Krueger, & Wilson, 2012) and Gunzenhauser, Fasche, Friedlmeier, and von Suchodoletz (2014), using a mixed sample of mothers and fathers, reported significant relations between parent and child reappraisal and parent and child suppression (also see Bariola, Hughes, & Gullone, 2012). Finally, using a longitudinal design and a multimethod approach, Kim, Pears, Capaldi, and Owen (2009) also reported anticipated relations between parent and child emotional SR.

Studies also have reported relations between parent impulsivity and child impulsivity and top-down SR. Under the rubric of self-control, Verhoeven, Junger, Van Aken, Dekovic, and Van Aken (2007) reported anticipated associations between low parent impulsivity and better child inhibitory control. Other studies have considered parent and child impulsivity and have identified anticipated positive associations (Brodsky et al., 2008). Additionally, one study reported a positive relation between parent and child SR using a measure reflecting a combination of impulsivity and inhibitory control processes (e.g., Boutwell & Beaver, 2010). Although this study supports anticipated relations, it is ambiguous in terms of whether top-down or bottom-up processes (though, likely both) were driving the association. Despite fairly consistent associations between parent and child impulsivity in the existing literature, we identified two studies that did not find significant relations (Epstein, Dearing, Temple, & Cavanaugh, 2008; Henschel, de Bruin, & Mohler, 2014).

## Section Summary, Recommendations, and Implications for Remainder of Review

On the basis of the studies reviewed in this section, it is clear that parent top-down SR and impulsivity are associated with children's top-down SR and impulsivity, respectively. Nevertheless, there are striking observations regarding this literature that have implications for future work and for the remainder of our review. Notably, we identified only a few studies that considered relations between parent and child behavioral inhibition/fear (e.g., Arroyo, Nevarez, Segrin, & Harwood, 2012; Coplan, Arbeau, & Armer, 2008; Daniels & Plomin, 1985; Degnan, Henderson, Fox, & Rubin, 2008; Gartstein et al., 2010; Kiel & Buss, 2011; Rickman & Davidson, 1994), and these offered mixed support for parent–child similarity. Fewer still reported relations between parent behavioral inhibition/fear and potential mechanisms linking parent and child behavioral inhibition/fear (e.g., Kiel & Maack, 2012; Tackett, Nelson, & Busby, 2013). As such, more work is needed that considers parent and child behavioral inhibition/fear and the mechanisms that may mediate such relations. Because of limited existing work regarding the intergenerational transmission of behavioral inhibition/fear of relevance for much of our review, we return to a discussion of future directions regarding behavioral inhibition/fear in closing.[4] Finally, although studies of relations between the other aspects parent and child SR support the conclusion that SR is transmitted across generations, such studies do not test how transmission occurs. We now turn to evidence of the integral role that social/contextual mechanisms play in the intergenerational transmission of SR.

# Family Dynamics and Rearing Context as Mechanisms of Intergenerational Transmission

## Parenting

**Overview of conceptual/empirical relations with children's self-regulation.** In order for parenting to act as a social mechanism in the intergenerational transmission of SR, it must be related to children's SR and to parent SR. With regard to the first requirement, Kopp (1982) was one of the first to note the important role of parenting behavior in promoting children's self-regulatory development. Similarly, Eisenberg, Cumberland, and Spinrad (1998) noted the role of emotion-related parenting practices for supporting the development of children's emotion-related regulation. Patterson's coercion theory also has been influential in showing the role of harsh parenting in the escalation of dysregulated affect and

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[4] To limit redundancies throughout the remainder of our review (e.g., noting the limited, if any, information for behavioral inhibition/fear in regards to mechanisms of intergenerational transmission), we return to recommendations for future work in this area in closing.

conduct problems in children and adolescents arising from escape conditioning (Patterson, Reid, & Dishion, 1992; Snyder, Schrepferman & St. Peter, 1997). Theoretical work by Dix (1991) also provides a foundation for examination of parenting in the socialization of emotional SR. More recently, the role parental behavior in supporting children's emotional SR was discussed by Morris, Silk, Steinberg, Myers, and Robinson (2007).

Consistent with theoretical work, relations between caregiving and children's executive functioning (e.g., Bernier, Carlson, & Whipple, 2010; Blair, Raver, & Berry, 2014; Conway & Stifter, 2012), effortful control (e.g., Eisenberg et al., 2005; Kochanska & Knaack, 2003; Lee, Zhou, Eisenberg, & Wang, 2013; Spinrad et al., 2007; Zalewski et al., 2012), self-control (e.g., Vazsonyi & Huang, 2010), impulsivity (e.g., King, Lengua, & Monahan, 2013; Olson, Bates, & Bayles, 1990), and behavioral inhibition/fear (e.g., Grady, Karraker, & Metzger, 2012) have regularly been reported. Parenting, including maltreatment, also has demonstrated relations with children's emotional SR (e.g., Cole, Dennis, Smith-Simon, & Cohen, 2009; Lilly, London, & Bridgett, 2014; Roth & Assor, 2012), including vagal withdrawal and RSA (e.g., Perry, Mackler, Calkins, & Keane, 2014; Skowron et al., 2011). Evidence also is accumulating that shows relations linking caregiving with the structural/functional development of neural areas essential for children's top-down and bottom-up SR (see Belsky & de Haan, 2011 for a review). Thus, this long-standing literature makes clear that parenting behavior is an important social process that can support or hinder children's SR.

**Review of studies examining parent self-regulation and parenting.** In order for parenting to operate as a social influence in the intergenerational transmission of SR, it also must be related to parent SR. Consistent with this requirement, work has established relations between parent SR and the very parenting behaviors theoretically and empirically known to contribute to or hamper children's SR (see Online Supplemental Table 2). In the first study to consider parent executive functioning, Deater-Deckard, Sewell, Petrill, and Thompson (2010) reported that mothers with lower working memory reacted more negatively to challenging child behavior than mothers with better working memory. Subsequent work has shown that mothers with poor executive functioning are more likely to harshly respond to child conduct problems (Deater-Deckard, Wang, Chen, & Bell, 2012), and mothers with poor spatial working memory have been found to be less sensitive while interacting with their infants (Gonzalez, Jenkins, Steiner, & Fleming, 2012; also see Chico, Gonzalez, Ali, Steiner, & Fleming, 2014). Although these studies were cross-sectional, Cuevas et al. (2014a, 2014b) reported longitudinal relations between better maternal executive functioning and fewer negative caregiving behaviors, with such caregiving also showing relations with children's executive functioning at 36 and 48 months, but not at 24 months.

Existing work also provides evidence of relations between other aspects of parent behavioral SR and parenting behavior. Bridgett et al. (2011) noted a longitudinal association between maternal effortful control and the amount of time mothers spent in caregiving activities (e.g., play) with infants, which was longitudinally related to better toddler effortful control. Likewise, Bridgett, Laake, Gartstein, and Dorn (2013) reported a negative association between maternal effortful control and a different measure of negative parenting behavior when children in the Bridgett et al. (2011) sample reached 18 months of age. In older samples, higher parent effortful control has been related to more positive and fewer negative reactions to children's negative affect (Valiente et al., 2007) and to more positive interactions and fewer maternal displays of dysphoric behaviors (but not aggressiveness) while interacting with adolescents (Davenport et al., 2011). Likewise, in an earlier study, Cumberland-Li et al. (2003) reported relations between maternal behavioral SR and parenting behaviors, some of which mediated the association between maternal behavioral SR and children's regulation-related behavior and teacher reported behavior problems. Next, Crouch et al. (2012) had parents retrospectively report on their childhood experiences of negative parenting, their current attentional control, and complete a measure assessing risk of child physical abuse perpetration. They found that lower attentional control was related to greater risk of engaging in hostile/aggressive parenting, and that attentional control partially mediated the relation between the experience of negative parenting as children and risk of engaging in hostile/aggressive parenting as adults. Finally, Pears, Capaldi, and Owen (2007) reported more direct evidence of intergenerational relations between inhibitory control and parenting. The effect of generation one's harsh/inconsistent discipline on generation two's poor discipline was mediated by generation two's inhibitory control. In turn, generation two's poor discipline mediated the association between generation two's inhibitory control and generation three's inhibitory control.

Consistent with findings in studies of behavioral SR, studies have noted associations between parent emotional SR and parenting. For example, Kim, Teti, and Cole (2012) reported negative relations between mothers affect dysregulation and observed behavioral markers of emotional availability during mother-infant interactions. Using a multireporter/method approach, Kim et al. (2009) reported a link between parent emotion dysregulation and more use of problematic discipline strategies. Moreover, they reported an indirect effect through parenting, between parent emotion dysregulation and sons' emotion dysregulation. Similarly, Buckholdt et al. (2013), using cross-sectional data, a notable limitation, reported an indirect effect between parent and adolescent emotion dysregulation via parent invalidation of adolescent emotion (see Sarıtaş, Grusec, & Gençöz, 2013, who reported similar findings). Next, Smith, Cross, Winkler, Jovanovi, and Bradley (2014) noted relations between mother's emotion dysregulation and their higher scores on a measure of child abuse potential. These authors also reported that the relation between maternal maltreatment history and child abuse potential was mediated by emotion dysregulation and negative affect. However, in contrast to most studies, one study that we identified did not report a significant relation between maternal emotional SR and children's perceptions of their mother's parenting (Samuelson et al., 2012). Finally, two studies have examined specific parent emotional SR strategies (e.g., Lorber, 2012; Lorber & O'Leary, 2005), finding that use of reappraisal was related to more supportive and less harsh parenting, whereas greater use of suppression was related to more reliance on harsh/negative discipline strategies.

In addition to studies that have employed behavioral assessments of emotional SR, biomarkers of parent emotional SR also have been considered. Joosen, Mesman, Bakermans-Kranenburg, and van IJzendoorn (2013a) examined maternal baseline HR and RSA and change in these cardiac indices during exposure to an audio recording of an infant crying. They found that greater maternal sensitivity (rated from observed parent–child interactions) was associated with lower maternal HR and higher RSA during baseline and infant cry conditions. Along similar lines, Moore et al. (2009) reported that more sensitive mothers had greater RSA suppression when reunited with their infants after the *still face procedure*, which is potentially indicative of these mothers being more flexible in employing emotional SR to cope with and assist infants in distress regulation. Although the findings reported by Joosen et al. and Moore et al. are consistent with results reported in several other studies (Connell, Hughes-Scalise, Klostermann, & Azem, 2011; Musser, Ablow, & Measelle, 2012; Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013), we identified one study did not find anticipated relations (Joosen et al., 2013b). Furthermore, Mills-Koonce et al. (2009) reported that mothers with lower RSA suppression in response to reunions with their children following a stressful task were more intrusive only if they also exhibited high baseline salivary cortisol, indicating the potential interactive effects of discernible physiological subsystems that should continue to be examined in future research.

Investigators also have considered relations between parent impulsivity and various adaptive and maladaptive parenting behaviors. In the first to do so, mothers who had perpetrated child abuse were found to be more impulsive and to have poorer inhibitory control than nonmaltreating mothers; however, there were no differences between mothers who had neglected their children and nonmaltreating mothers (Rohrbeck & Twentyman, 1986). Verhoeven et al. (2007), referring to impulsivity as self-control, reported that higher parent impulsivity was related to more use of psychological control and physical punishment, but not to lack of structure and less support and positive discipline, in a sample of mothers and fathers. Similar findings, such that higher maternal impulsivity was related to more dysfunctional parenting, were reported by Loney, Huntenburg, Counts-Allan, and Schmeelk (2007). Latzman, Elkovitch, and Clark (2009) also found that high maternal impulsivity was related to poorer monitoring/supervision and more inconsistent discipline, but not less involvement, positive parenting, or more corporal punishment in a sample of adolescents. Likewise, Boutwell and Beaver (2010) reported anticipated relations between mother's and father's impulsivity, referred to as low self-control, and measures of parental involvement; however, parental involvement did not mediate relations between parent and child self-control. Lastly, Henschel, de Bruin, and Mohler (2014) examined mother and father self-control using a measure that assessed behavioral and emotional SR, as well as impulsivity, and found that better maternal self-control was related to lower child abuse potential. Henschel et al. also reported a trend such that mother's child abuse potential partially mediated the relation between maternal self-control and children's delay of gratification. Although the relation between father's self-control and child abuse potential was in the anticipated direction, it was not significant.

Finally, work is beginning to consider relations between parent neural functioning, assessed using neuroimaging, and parenting behavior. Musser, Kaiser-Laurent, and Ablow (2012) exposed mothers to their own infant's cry and to the cry of an unfamiliar infant during fMRI and separately observed them interacting with their infants. Mothers who were more sensitive during interactions with their children had greater activation in the inferior frontal gyrus and right frontal pole when listening to their own child's cry compared with that of the unfamiliar infant. Musser et al. also noted a relationship between greater activation of the left hippocampus to own infant cry compared with the cry of an unfamiliar infant and more harmonious mother-infant interactions. Using a similar fMRI cry procedure, Kim, Feldman, et al. (2011) reported relations between more maternal activation in the right superior frontal gyrus and amygdala and more sensitive interactions with infants who were 3 to 4 months of age. However, in a third study, using a very small sample of fathers ($N = 10$), Kuo, Carp, Light, and Grewen (2012), employing procedures similar to those of Musser et al. and Kim et al., did not find expected relations between neural correlates of SR and observed parenting behavior. Nevertheless, across two of three available studies, neural correlates of SR were related to parenting behavior.

**Section summary and recommendations for future work.** In this section, we provided a brief overview of the long-standing theoretical and empirical work supporting the notion that parent behavior is a key social influence on children's SR. Subsequently, we reviewed work showing relations between parent SR and caregiving behaviors. Although at times findings were mixed (e.g., anticipated effects were present between parent SR and some but not all aspects of parenting behavior), most studies demonstrated anticipated associations between parent behavioral SR, emotional SR, or impulsivity and parenting behavior, including two studies that considered neural correlates of parent SR. Moreover, five studies tested intergenerational effects. Although several were not longitudinal, four of the five studies reported that parenting behavior mediated associations between parent and child SR.[5] Thus, based on the available literature, there is strong evidence that parenting behavior is a likely, and key social mechanism in the intergenerational transmission of SR.

Despite solid evidence in support of our conclusions regarding the material covered in this section, the studies we reviewed have limitations that need to be addressed in future work. Many are cross-sectional, which limits inferences regarding temporal ordering of effects. However, this limitation is tempered by findings from the smaller pool of longitudinal studies that converge with the results from cross-sectional work. Still, additional longitudinal studies are warranted, and particularly those that use complete full-panel designs to repeatedly assess parenting, and parent and child SR. The absence of such designs is a critical shortcoming in the existing body of evidence because concurrent associations can be incorrectly specified as longitudinal effects in incomplete longitudinal designs. Studies considering parent SR also have not simultaneously considered multiple aspects of top-down and bottom-up SR. This is a necessary next step that will permit consideration of unique, additive, and interactive effects among aspects of parent SR in relation to parenting behavior and intergenerational processes. Finally, three studies extended work that has relied on behavioral and questionnaire measurement of parent SR by examining neural correlates of parent SR in relation to observed parenting behaviors. Two of these studies provided pre-

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[5] Given that our review provides strong evidence that parent SR is associated with caregiving, studies that have examined the intergenerational transmission of parenting practices characterized by dysregulated behavior and emotion (see Conger, Schofield, Neppl, & Merrick, 2013; Neppl, Conger, Scaramella, & Ontai, 2009; Simons et al., 1991), provide additional, indirect support (due to the absence of direct assessment parent SR) for the contribution of caregiving to the intergenerational transmission of SR.

liminary evidence of relations between activation of neural structures implicated in top-down and bottom-up SR and parenting. However, findings in all three studies must be viewed cautiously. All were limited by small samples and none assessed parent neural functioning during interactions with infants (i.e., neural functioning and parent-infant interactions were examined separately), a limitation that may be difficult to overcome until new technologies are developed.

## Inter-Parental Relationship Adjustment

**Overview of conceptual/empirical relations with children's self-regulation.** Conceptual work indicates that high levels of marital conflict disrupt children's emotional security (Cummings & Davies, 1996; Davies & Cummings, 1994) contributing to their dysregulated emotion and behavior. Moreover, it is also now clear that marital conflict contributes to children's poor SR via children's arousal regulation mechanisms (see El-Sheikh & Erath, 2011 for discussion). Consistent with conceptual work, empirical studies have reported relations between marital maladjustment, including IPV, and children's poorer SR (Crockenberg & Langrock, 2001; Davies & Cummings, 1994). Findings also are consistent in showing that adequate interparental relationship adjustment is related to aspects of children's behavioral and emotional SR, such as higher effortful control, and lower emotional reactivity and higher RSA (e.g., Bridgett et al., 2013; Crockenberg & Langrock, 2001; Davies & Cummings, 1998; Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009; Graham, Ablow, & Measelle, 2010; Gustafsson, Cox, & Blair, 2012; Rhoades et al., 2011). Finally, there is evidence that marital conflict affects children's bottom-up mediated undercontrolled (i.e., impulsivity) and overcontrolled (i.e., behavioral inhibition/fear) SR (see Cummings & Davies, 2002 for discussion). Thus, as with parenting, there is long-standing evidence of relations between interparental relationship adjustment and children's SR.

**Review of studies examining self-regulation and relational functioning.** Like work considering a relation between parent SR and parenting, research has accumulated that supports the importance of adult SR for relationship functioning, including links between SR and IPV (see Online Supplemental Table 3). Finkel, DeWall, Slotter, Oaten, and Foshee (2009) demonstrated that a SR intervention reduced violence in response to partner provocation, providing support for relations among SR and relational well-being. Similar to these findings, Pinto et al. (2010) reviewed studies (Cohen et al., 2003; Cohen, Rosenbaum, Kane, Warnken, & Benjamin, 1999; Schafer & Fals-Stewart, 1997; Stanford, Conklin, Helfritz, & Kockler, 2007; Teichner, Golden, Van Hasselt, & Peterson, 2001; Westby & Ferraro, 1999) that reported relations between lower executive functioning and engagement in IPV (also see Becerra-Garcia, 2014 who reported that men with a history of committing IPV had more difficulties with cognitive flexibility than a comparison group). Finally, one study, using a multimethod approach to assess behavioral SR, reported that higher maternal SR was related to better concurrently assessed dyadic adjustment, which mediated the longitudinal effects of maternal behavioral SR on infant emotional SR and negative affect (Bridgett et al., 2013), supporting the notion that interparental relational functioning may be a social mechanism in the intergenerational transmission of SR. However, whereas most studies have reported anticipated relations between behavioral SR and interpersonal functioning, we identified one study did not find anticipated relations (Schumacher, Coffey, Leonard, O'Jile, & Landy, 2013).

Research also has noted that the use of adaptive emotional SR strategies is related to better relationship functioning, and that problematic emotional SR is related to poorer relationship outcomes (Gross & John, 2003; Kerley, Xu, & Sirisunyaluck, 2008; Kinsfogel & Grych, 2004; Tharp, Schumacher, McLeish, Samper, & Coffey, 2013). Similarly, several longitudinal studies have found that poor emotional SR is related to IPV perpetration (Dankoski et al., 2006; Turcotte-Seabury, 2010). Studies employing physiological markers of emotional SR (e.g., vagal tone) have reported that better emotional SR is associated with better relationship adjustment (e.g., Diamond, Hicks, & Otter-Henderson, 2011; Gyurak & Ayduk, 2008; Murray-Close, Holland, & Roisman, 2012; Smith et al., 2011), consistent with the larger body of work that has employed mostly self-report measures of emotional SR. Nevertheless, Murray-Close (2011) did not find a significant relation between RSA and relational aggression in a sample of women. Finally, Kim et al. (2009) reported findings suggestive of intergenerational effects in a 20 year longitudinal study. Kim et al. reported that parent and child emotion dysregulation were related to parent and child relationship conflict, respectively, and that the relation between parent emotion dysregulation and their sons' relationship conflict was mediated by sons' emotion dysregulation. However, additional findings that would have provided further support for intergenerational transmission (e.g., parent relational conflict mediating relations between parent and child dysregulation) were not significant.

In addition to anticipated relations with top-down SR, studies have routinely noted that impulsivity is related to poor relational outcomes, including IPV. Cohen et al. (2012), Finkel et al. (2009; Study 1), Stuart and Holtzworth-Munroe (2005), Tharp et al. (2012), Schafer, Caetano, and Cunradi (2004), Kerley et al. (2008), and Berzenski and Yates (2010) reported concurrent associations between impulsivity and aspects of relationship adjustment (e.g., marital dissatisfaction and IPV perpetration). Likewise, Caetano and colleagues (Caetano, Vaeth, & Ramisetty-Mikler, 2008) found that men and women in violent relationships were more impulsive than participants in nonviolent relationships. However, one study did not find anticipated effects (Swogger, Walsh, Kosson, Cashman-Brown, & Caine, 2012) and other studies have reported mixed findings. For example, Newman, Caspi, Moffitt, and Silva (1997) examined impulsivity in 3-year-old children and their relationship adjustment when they reached 21 years of age. Newman et al. found that higher impulsivity was related to lower romantic relationship adjustment in some domains (power balance), but not in others (e.g., mutual interest and intimacy; also see Derefinko, DeWall, Metze, Walsh, & Lyman, 2011, who reported mixed findings similar to those noted by Newman et al.). Other studies have reported anticipated effects for men, but not women (Cunradi, Todd, Mair, & Remer, 2013; Mair, Cunradi, & Todd, 2012). However, one study found that womens', but not mens', higher impulsivity was related to more marital discord, but not psychological or physical aggression (O'Leary, Malone, & Tyree, 1994) and a separate study of women adjudicated for domestic violence reported a positive relation between higher impulsivity and more IPV perpetration (Shorey, Brasfield, Febres, & Stuart, 2011). Thus, although there are occasional within study inconsistencies in gender effects, across studies, evidence suggests that men's and women's high impulsivity contributes to relationship discord.

Finally, several studies have employed measures of SR that reflect combinations of problematic behavioral and emotional SR, and higher impulsivity. Payne, Higgins, and Blackwell (2010) assessed "self-control" using such a measure and found that higher self-control was related to less partner violence (see Cheung, Choi, & Cheung, 2014; Gover, Kaukinen, & Fox, 2008; Payne, Triplett, & Higgins, 2011; Verhoeven et al., 2007; Vohs, Finkenauer, & Baumeister, 2011 who reported similar findings using similarly defined measures of "self-control"). Although such studies are ambiguous in terms of the effects of specific SR processes on relationship adjustment, they provide further support for the role of SR in relationship adjustment.

**Section summary and recommendations for future work.** The studies noted in this section establish the importance of SR for relational functioning among adults,[6] including relationships that are experienced by children, which prior work has long noted to be related to children's SR. Two studies also provided more direct support for the idea that interparental relationship adjustment plays a role as a social influence in the intergenerational transmission of SR. In total, this body of work provides evidence that interparental relationship functioning, along with parenting behavior, acts as a social influence in the intergenerational transmission of SR.

Despite evidence in support of our conclusions, there are limitations in this body of work that should be considered in future studies. As with studies that have considered the role of SR in parenting behavior, studies of interparental relationships have not yet considered the interplay between multiple aspects of SR. Such studies are needed to provide insights into the unique, additive, or interactive effects of aspects of SR on relationship adjustment. Although important longitudinal studies have provided evidence of expected associations and the temporal ordering of effects, most studies are cross-sectional. Moreover, the field needs complete longitudinal studies that employ cross-panel methods to address limitations inherent in the more commonly used incomplete longitudinal designs that can incorrectly specify concurrent associations as longitudinal effects.

Finally, because the "spill over" hypothesis (i.e., interparental conflict spills over into the parent–child relationship, which then affects children's outcomes; Schoppe-Sullivan, Schermerhorn, & Cummings, 2007) has received support, the interplay between interparent conflict, parenting, parent SR and children's SR needs to be considered in future work. Although strong support exists in the literature of the direct relation of SR on interparental relationship quality and for the effects of interparental relationship functioning on children's SR, it may be that interparental functioning and parenting behavior together act as a mediators of the association between parent and child SR.

## Broader Rearing Context

**Overview of conceptual/empirical relations with children's self-regulation.** Similar to caregiving and interparental relationship functioning, other characteristics of the home environment, including home chaos (i.e., homes that are less structured, more crowded, and noisy; Wachs & Evans, 2010), lower socioeconomic status (SES), and the accumulation of risk factors (i.e., cumulative risk) may undermine or provide support for children's SR. Across developmental periods, studies have noted the adverse effects of high home chaos, low SES, and higher cumulative family risk factors on children's top-down SR (e.g., Buckner, Mezzacappa, & Beardslee, 2003; Dumas et al., 2005; Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005; Farah et al., 2006; Lengua, Honorado, & Bush, 2007; Lipina, Martelli, & Colombo, 2005; Martin, Razza, & Brooks-Gunn, 2012; Taylor, Sulik, et al., 2013). Findings in studies employing biomarkers of emotional SR also find that poorer rearing contexts can have a negative influence on children's emotional SR (Jacob, Byrne, & Keenan, 2009; but see Blair et al., 2013, who did not find a relation between cumulative risk and children's RSA).

Children's bottom-up SR also is affected by the broader rearing context. For instance, homes that are more chaotic are related to children's elevated impulsivity (e.g., Corapci, 2008; Dumas et al., 2005). Similarly, other aspects of the rearing context, such as family income, are related to children's behavioral inhibition/fear and impulsivity (e.g., Bush, Lengua, & Colder, 2010). Thus, in light of consistent, long standing findings showing relations between aspects of children's broader rearing context and their SR, children's rearing context, along with parenting and interparental relationship adjustment, can act as a strong contextual influence on children's SR.

**Studies examining relations between self-regulation and rearing context.** Given the importance of a stable, predictable rearing context for the development of children's SR, adequate parent SR may very well provide a critical basis for the provision of such a stable rearing context. Importantly, findings in the literature are emerging to support this possibility (see Online Supplemental Table 4). In the first study to examine this question, Valiente, Lemery-Chalfant, and Reiser (2007) found that better parent effortful control was related to lower home chaos, a finding partially replicated and extended by Deater-Deckard, Chen, Wang, and Bell (2012) who found that lower maternal executive functioning was associated with higher chaos in the lowest SES families. Similarly, Bridgett et al. (2013) reported an association between better maternal behavioral SR and lower home chaos. Moreover, chaos partially mediated the relation between maternal SR and infant frustration—a potential marker of poor emotional SR—several months later. However, chaos did not mediate the relation between maternal SR and infant falling reactivity, a more proximal marker of emotional SR.

Studies also have reported associations between parent SR and family SES and cumulative risk. Martini, Root, and Jenkins (2004) found that middle income mothers demonstrated better emotional SR than did low income mothers in response to their children's negative affect. Likewise, Bridgett et al. (2013) reported a negative association between better maternal behavioral SR and lower family cumulative risk, and Deater-Deckard et al. (2012) reported a positive relation between better maternal behavioral SR and higher family SES. Zalewski et al. (2014) also noted associations

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[6] It is also worth noting that studies of associations between children's self-regulation and their peer relationships and social competence (e.g., Eisenberg et al., 2003; Kim & Cicchetti, 2010; Spinrad et al., 2007) are consistent with, and provide support for, findings reported in the adult literature. We focused on findings in the adult literature given the more direct relevance of these studies for interparental relationships to which children are exposed.

between poorer maternal emotional SR and lower educational attainment, a risk factor often included in cumulative risk indices (also see Côté, Gyurak, & Levenson, 2010 for similar findings in a sample of nonparents, but see Samuelson et al., 2012 who did not find significant relations between maternal emotional SR and educational attainment). Similar to the findings reported by Zalewski et al. (2014), Creed, King, Hood, and McKenzie, (2009) reported a positive relation between better behavioral and emotional SR and more intense job seeking in a sample of unemployed Australian adults. Given that employment is usually a positive influence on family SES, Creed et al.'s (2009) findings also support the role of parent SR in influencing SES. Bottom-up impulsivity also has been related to aspects of children's broader rearing contexts, such as poorer parental educational attainment (Boutwell & Beaver, 2010). Finally, although one cross-sectional study (Verhoeven, Junger, Van Aken, Dekovic, & Van Aken, 2007) did not find relations between self-control and family size and SES when self-control was assessed using a measure examining aspects of behavioral SR, emotional SR, and impulsivity, another cross-sectional study using the same measure (Cheung et al., 2014) reported significant associations between poorer self-control and lower educational attainment and monthly income.

Although most existing studies of relations between parent SR and broader rearing contexts that can affect children's SR are cross-sectional, several longitudinal studies provide key evidence of potential temporal patterns of associations. McClelland, Acock, Piccinin, Rhea, and Stallings (2013) reported relations between children's attention/behavioral SR and their educational outcomes 21 years later—outcomes that are frequently used as markers of SES or within cumulative risk indices. Controlling for early academic skills and maternal education, McClelland et al. (2013) found that better attention improved the odds of children graduating from college. In the only other longitudinal study identified that considered behavioral SR, Véronneau, Racer, Fosco, and Dishion (2014) reported that effortful control assessed in older adolescents was related to higher educational attainment when participants reached 23 to 25 years of age after accounting for notable covariates (e.g., parent educational attainment).

Bottom-up impulsivity also has been considered in longitudinal studies. In a study by Moffitt et al. (2011), poor self-control, assessed using measures that examined impulsivity, in childhood was related to lower income, diminished financial planning (e.g., less likely to own a home or have retirement savings), and difficulties with credit/money management when children reached 32 years of age. Among those who were parents, those originally low in self-control were more likely to be a single parent, a risk factor that is often included in indices of cumulative risk. Adolescent self-control, assessed as low impulsiveness, also appears to set in motion a chain of events leading to better educational and income outcomes by 25 years of age (Converse, Piccone, & Tocci, 2014). Finally, Nedelec and Beaver (2014) considered adolescent (Grades 9–12) low self-control, assessed using a scale that measured poor behavioral and emotional SR, and high impulsivity, in relation to outcomes when participants reached 24 to 34 years of age. These investigators reported that low self-control was related to fewer household assets, lower income and educational attainment, and to more employment difficulties (e.g., number of times fired)—outcomes potentially having consequences for children's SR—after controlling for IQ, age, race, gender, and parent's income (also see Fergusson, Boden, & Horwood, 2013 for similar findings).

**Section summary and recommendations for future work.** The evidence reviewed in this section suggests that parent SR influences key aspects of children's rearing contexts, such as home chaos, family SES, parent educational attainment, household assets, and cumulative risk, among others that have consistently demonstrated relations with children's SR. Furthermore, one study found that home chaos partially mediated the relation between parent SR and infant frustration. Although most studies are cross-sectional and have treated rearing contexts as covariates rather than testing mediation, longitudinal studies that assessed SR in children as young as 3 to 5 years of age (i.e., well before individuals bear any responsibility for maintaining an adequate rearing environment) and then followed them into adulthood, sometimes decades later, reported anticipated relations between SR and aspects of rearing environments (e.g., SES) that are known to be related to children's SR. Importantly, many of these studies controlled for the effects of parent education or SES (or related constructs) in the family of origin, which, along with the use of longitudinal methods, leads to more confidence in the temporal ordering of relationships. As such, the available evidence points to aspects of the broader rearing context as social influences in the intergenerational transmission of SR. Nevertheless, there is a key direction for the field to take that will further increase confidence by addressing the most salient limitation in existing studies. Given the existing evidence, the field now needs to employ longitudinal studies that test mediation by assessing parent SR, aspects of the broader rearing contexts in which children are raised, and SR in offspring.

## Supporting Evidence: Findings From the Personality Literature, Parents With ADHD, Animal Models, and Human Case Studies

Having connected parent SR to family dynamics and rearing contexts that have been consistently related to children's SR, we now turn to brief overviews of evidence in other literatures that support our tentative conclusion that the contextual influences we covered above are social mechanisms in the intergenerational transmission of SR. Although some of the supporting evidence to which we point is based on correlational findings, we also point to quasi-experimental and experimental findings, as well as case studies that help increase confidence that the relations to which we have pointed are causal in nature. We now turn to supportive correlational evidence in the human personality literature, focusing on conscientiousness. This is followed by correlational and quasi-experimental findings from adults with ADHD, experimental animal models, and finally, compelling qualitative evidence from human neuropsychological case studies.

### Evidence From Studies of Conscientiousness

Conscientiousness (i.e., the tendency toward self-discipline, planning, organization and attention to norms of conduct and behavior; Roberts, Jackson, Fayard, Edmonds, & Meints, 2009) has its roots in top-down SR, and specifically effortful control, which encompasses persistence and inhibitory control features that are critical to SR and conscientiousness alike across the life span

(Rothbart, Ahadi, & Evans, 2000). Over development, children's effortful SR influences the emergence of conscientiousness as they learn and apply regulation strategies in response to increasing demands for academic skills and compliance with cultural standards of behavior (Eisenberg, Duckworth, Spinrad, & Valiente, 2014). As Eisenberg, Rothbart, and their colleagues note, these early origins contribute to individual differences that arise from transactions between biological and contextual influences. More broadly, conscientiousness has been related to a range of outcomes, spanning academic/career success to interpersonal relationship longevity and satisfaction (Steel, Schmidt, & Shultz, 2008). Notably, these outcomes also include parenting and marital/family relationships (Bouchard, Lussier, & Sabourin, 1999; Vondra, Sysko, & Belsky, 2005). Given these findings and the origins of conscientiousness being rooted in top-down SR processes, this literature provides additional support for our conclusions about family processes that we identified as playing a role in the intergenerational transmission of SR.

## Evidence From Studies of Parents With ADHD

ADHD is an early emerging neurodevelopmental disorder that results in increased impulsivity, and often poorer behavioral and emotional SR (e.g., Barkley, 1997; Nigg, 2001; Woltering, Liu, Rokeach, & Tannock, 2013), stemming from abnormalities in bottom-up and top-down neurobiological mechanisms of SR (e.g., Rubia, 2011; Sonuga-Barke, 2005; Sonuga-Barke & Fairchild, 2012; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). As such, consideration of studies of parents with ADHD—a disorder that results in varying degrees of lifelong regulatory difficulties—could provide additional supporting evidence of the effects of SR on family dynamics. Turning first to parenting, Chen and Johnston (2007) found that more maternal symptoms of ADHD were related to less use of positive reinforcement and more inconsistent discipline. Other studies, using semistructured interviews to assess ADHD and observations of parenting, have reported similar findings (Chronis-Tuscano et al., 2008; Murray & Johnston, 2006; see Johnston, Mash, Miller, & Ninowski, 2012 for a review of studies considering relations between ADHD and parenting). Although studies have mostly considered mothers, several have reported associations between paternal ADHD and more frequent use of harsh parenting practices (Ellis & Nigg, 2009; Mokrova, O'Brien, Calkins, & Keane, 2010). Finally, one study reported that harsh and less positive parenting behavior mediated the relation between parent and child ADHD symptoms, offering further support that parenting can act as a social mechanism in the intergenerational transmission of SR (Tung, Brammer, Li, & Lee, 2014).

Supporting evidence in the ADHD literature extends beyond parenting to include marital relationship adjustment and the broader child rearing context. Eakin et al. (2004) reported that adults diagnosed with ADHD had lower marital adjustment than those without ADHD (also see Canu, Tabor, Michael, Bazzini, & Elmore, 2014; Moyá, Stringaris, Asherson, Sandberg, & Taylor, 2014). Similarly, adults who have ADHD are more likely to engage in IPV (e.g., Wymbs et al., 2012) and have multiple marriages (Murphy & Barkley, 1996). Regarding the broader rearing context, Mokrova, O'Brien, Calkins, and Keane (2010) noted an association between mothers' and fathers' ADHD symptoms and more home chaos—findings replicated by Farbiash, Berger, Atzaba-Poria, and Auerbach (2013). Adults with ADHD also are at higher risk of lower occupational attainment, poorer job performance, greater likelihood of being fired, and less educational attainment (e.g., Barkley, Fischer, Smallish, & Fletcher, 2006; Miller, Nevado-Montenegro, & Hinshaw, 2012). Thus, these findings provide further support for the role of parent SR in the provision of children's rearing contexts.

## Animal Models

**Evidence of self-regulatory effects on parenting and social relations.** Like findings in the human literature, there is a longstanding experimental animal literature indicating that sensitive caregiving (e.g., in rodents, greater frequency of lick/grooming, nest building, and pup retrieval) has lasting effects on offspring SR and physiological regulation of arousal (Feldman, Weller, Sirota, & Eidelman, 2002; Meaney, 2001). Of equal importance to our review, experimental animal studies also offer direct support for the importance of stress induced parent SR difficulties and specific neurobiological self-regulatory mechanisms in the provision of adequate parenting and social relationships, analogous to the mostly correlational evidence in the human literature.

Similar to the mostly behavioral evidence in humans we have covered to this point, several rodent studies have used experimental, but nonsurgical approaches to examine relations between maternal SR and parenting of pups. To demonstrate such effects, Lovic, Palombo, and Fleming (2011) induced impulsivity in rat pups through chronic early stress using an artificial rearing procedure (i.e., depriving pups of the types of caregiving that support the development of adaptive SR). After these pups became mothers, more impulsivity was associated with less pup licking. This finding extended and replicated several earlier studies in which rat attention and impulse control were impaired via early artificial rearing, which subsequently resulted in less frequent pup licking behavior (Lovic & Fleming, 2004; Palombo, Nowoslawski, & Fleming, 2010). Although these studies do not specify the neural origins of SR difficulties (e.g., impulsivity could reflect more bottom-up mediated impulsivity, poorer top-down inhibitory control, or both), the findings are critical complements to neurobiological experiments, discussed next, because they more closely approximate (though not perfectly) early human rearing experiences affecting self-regulatory development that in turn, appear to affect the rearing of the next generation.

In regard to specific top-down SR neurobiological mechanisms experimentally manipulated using lesion techniques, very early studies in which significant portions (i.e., $> 50\%$) of maternal rat cortical tissue were removed showed that severe parenting disturbances emerged, including the absence of or limited pup retrieval, licking/grooming, and nest building (e.g., Beach, 1937; Davis, 1939; Stone, 1938), but that other behaviors, such as mating, were spared (e.g., Davis, 1939). Studies of primates have reported similar findings. For instance, Franzen and Myers (1973) found that removal of PFC or anterior temporal cortex in Rhesus monkeys led to poorer parenting (e.g., less holding, cuddling, and infant retrieval from threat) and social deficits (e.g., increased aggression, and loss of grooming behaviors and dominance status) compared with controls and to those subjects wherein the ACC was removed (also see Myers, Swett, & Miller, 1973).

In addition to removal of specific areas, more precise lesion sites also have been considered. In one of the first studies to do so, Slotnick (1967) lesioned female rat cingulate cortices prior to mating and reported significant reductions in pup retrieval, and qualitatively described maternal behavior as more disorganized and inconsistent compared with rats with sham lesions. Similarly, Febo, Felix-Ortiz, and Johnson (2010) showed that chemically induced inactivation of the mPFC in rats resulted in impaired maternal pup retrieval, but not licking/grooming and hovering. However, in a separate study that employed surgical lesions to the mPFC, impairments in both pup licking and retrieval were observed (Afonso et al., 2007). Broader social difficulties (i.e., problematic social behavior that extends beyond parenting) also have been demonstrated. For example, in a study of Vervet monkeys (Raleigh, Steklis, Ervin, Kling, & McGuire, 1979) and another of Rhesus monkeys (Goursaud & Bachevalier, 2007), investigators reported that OFC lesions resulted in disturbed social behaviors. Goursaud and Bachevalier also found that hippocampal or amygdala lesions did not adversely alter social behaviors. Finally, Mass and Kling (1975) reported social impairments resulting from lesions to the dlPFC in a sample of stumptail macaques.

Animal models also have been employed to examine the role of subcortical areas important for bottom-up SR processes in parenting behavior. Lee and Brown (2007) demonstrated disruptions to parenting behavior in female and male California mice who received lesions to the basolateral amygdala. In this study, nucleus accumbens lesions resulted in mild pup retrieval disturbances only in male mice. However, the nucleus accumbens in male and female mice were not completely affected by lesions (a notable limitation), which may explain why such lesions did not result in wide-spread, more severe effects on parenting behavior. In an earlier study that contrasted lesions to the nucleus accumbens shell versus core, adverse effects on maternal pup retrieval, but not nest building, nursing, or licking, were reported only when the nucleus accumbens shell was affected (Li & Fleming, 2003). Similarly, other studies using a variety of blockade and lesion methods to examine the effects of striatal (including nucleus accumbens) and amygdala disruptions on parenting also have reported adverse effects on parenting behaviors (e.g., Keer & Stern, 1999; Numan et al., 2010; see Toscano, Bauman, Mason, & Amaral, 2009 for an example in Rhesus monkeys).

**Evidence of intergenerational transmission via parenting.** The findings from the animal literatures covered above provide experimental evidence that early stressful rearing contexts or disruption to the neurobiological mechanisms underlying SR results in parenting and social behaviors that in humans affect children's SR. Although supportive of findings in some studies that form the main foci of this review (e.g., relations between behaviorally assessed parent SR and caregiving behavior, and the emerging human fMRI findings), such studies do not provide direct evidence of intergenerational transmission processes that also are of importance. However, a different body of experimental work in the animal neuroscience literature does provide evidence that social processes, such as parenting, are important influences in the intergenerational transmission of SR. Specifically, to the degree that abuse/neglect can be construed as behavioral evidence of poor parent SR, we point to animal studies of abuse/neglect for experimental evidence that parenting acts as a social influence in the intergenerational transmission of SR. We believe that such studies provide the experimental evidence in which we are interested because (a) human studies have reported associations between parent SR deficits and risk for perpetrating or perpetration of child abuse (see Crouch et al., 2012; Henschel et al., 2014; Rohrbeck & Twentyman, 1986; and Smith et al., 2014 that were described earlier); and (b) abusive/neglectful parenting appears to adversely affect neural mechanisms of top-down and bottom-up SR (for reviews covering animal models or findings in human studies, see Hart & Rubia, 2012; Jaffee & Maikovich-Fong, 2013 or Parker & Maestripieri, 2011).

Consistent with our view that animal models of abuse/neglect provide experimental evidence of the intergenerational transmission of SR, Maestripieri (2005), using a cross-fostering design, reported that 56.25% of infant Rhesus monkeys who were abused by either biological or foster mothers engaged in abusive behavior toward their own infants, whereas 0% of the nonabused infants did so. Similar findings have been reported in other primates (e.g., Berman, 1990; Maestripieri, Lindell, & Higley, 2007) and in rodent models (e.g., Champagne, Francis, Mar, & Meaney, 2003; Francis, Diorio, Liu, & Meaney, 1999). As noted above, evidence from the animal abuse literature goes beyond behavioral findings, with work indicating that dysregulated, abusive or rejecting caregiving causes alterations to neural mechanisms of SR (e.g., mPFC; Blaze, Scheuing, & Roth, 2013) and in neurotransmitters with dense distributions within self-regulatory structures (e.g., Maestripieri et al., 2006). In sum, given existing evidence, findings from experimental animal studies are consistent with the proposed intergenerational transmission processes highlighted in our review.

## Evidence From Human Case Studies

Case studies in humans involving damage sustained to brain structures involved in top-down SR also provide evidence of relevance to our review. For instance, MH, who experienced bilateral frontal lobe damage at 4-years of age, had deficits in social behavior and neglected her infant as a young adult (Eslinger, Flaherty-Craig, & Benton, 2004; Price, Daffner, Stowe, & Mesulam, 1990). In another case, DT sustained bilateral damage to medial and polar prefrontal areas at an early age. As an adult, DT engaged in "impulsive, erratic, and immature behaviors [that] seriously compromised her abilities to participate in a marriage and care for her infant. These [difficulties] were expressed primarily by poor regulation of her emotions, disorganization in daily activities, and minimal anticipation of the needs of others" (Eslinger et al., 2004, p. 92). In a third case, "Subject A" sustained bilateral PFC damage at a young age. Anderson, Bechara, Damasio, Tranel, and Damasio (1999) reported that "her maternal behavior was marked by dangerous insensitivity to [her] infant's needs" (p. 1032). Finally, Anderson et al. also reported on "Subject B" who had early right frontal damage that resulted from resection of a tumor. Although able to graduate from high school with support, he was unable to hold a job and engaged in financially and sexually reckless behavior, the latter resulting in fathering a child, and the former resulting in "Subject B" being unable to fulfill child support-related obligations. In sum, these human case studies parallel findings from the supporting animal and human literatures that form core aspects of our review, and point to the importance of top-down mechanisms of SR for parenting, social functioning, and the provision of adequate rearing contexts—behaviors that in humans can have a powerful influence on children's SR.

## Section Summary, Limitations, Conclusions, and Directions for Future Work

In considering the methods employed across the supporting studies we have covered, more definitive support is provided for the influence of parent top-down and bottom-up neurobiological mechanisms of SR that shape caregiving and child rearing contexts that are known to contribute to children's development of SR. Of equal importance, animal models provide experimental evidence that caregiving indicative of poor SR (i.e., infant abuse) are transmitted across generations—findings that are consistent with the correlational evidence from similar intergenerational human studies (e.g., Kim et al., 2009; Pears et al., 2007). Thus, the supporting studies to which we have pointed provide additional weight of evidence as well as evidence of causal processes that is not available, due to a variety of factors, in the literature that is the primary focus of our review. Nevertheless, there are some limitations to the supporting works to which we have pointed.

Studies of conscientiousness are correlational, like most of the human studies covered in our review. Moreover, although top-down SR constitutes the developmental origins of conscientiousness, conscientiousness is broader than SR. Consequently, these studies only add weight to the evidence in support of our review, particularly in regards to aspects of top-down SR, but alone, would not constitute a sufficiently conclusive body of work. Studies of parents with ADHD (or that considered ADHD symptoms), some of which are correlational and others of which are quasi-experimental, reflect clinical levels of SR difficulties that emerge early in life and provide added support for our main points regarding the influence of parent SR on the social environment within the home. Moreover, because of the neurobiological origins of the SR difficulties experienced by those with ADHD (Rubia, 2011), these studies broadly complement findings from the animal models we discussed. However, because many (but not all) individuals with ADHD experience bottom-up and top-down SR difficulties, and many experience comorbid difficulties that are associated with top-down or bottom-up (or both) regulatory difficulties, studies of parents with ADHD are agnostic in terms of the influence of specific self-regulatory processes on family dynamics and on children. As such, like studies of conscientiousness, alone, studies of parents with ADHD would not constitute a sufficiently conclusive body of evidence.

Finally, although findings in the animal literatures are compelling, there are limitations that must be addressed and a avenue for future work that may provide even more direct evidence of intergenerational processes. One notable limitation is that the animal models we turned to in support of intergenerational transmission are in separate areas of neurobiological research. To address this limitation, it is feasible and necessary to conduct studies that (a) use lesion methods (or stressful early rearing paradigms) to disrupt neurobiological mechanisms of SR in Generation 1 (G1), resulting in impaired G1 parenting; (b) that follow offspring (i.e., Generation 2 [G2]) to document the probable G2 SR difficulties resulting from G1s parenting; and (c) that subsequently result in G2s poor parenting and transmission of poor SR to Generation 3. Finally, although there is considerable overlap across humans and other mammals in their neurobiology and physiology, there are important differences in the neurobiological processes of SR and caregiving between humans and other species. However, our consideration of human clinical case studies partly mitigates this concern because they revealed striking qualitative similarities with experimental animal lesion studies.

Although we have covered significant ground up to this point, before we can integrate separate pieces of existing evidence into a conceptual model of how SR is transmitted across generations, an additional question needs to be addressed: At what point in development does the intergenerational transmission of SR begin? Answering this question requires consideration of genetics and the environment from the point of conception—topics we turn to next.

# The Influence of the Prenatal Environment on Children's Self-Regulation

## Human Studies[7]

Evidence has now accumulated that suggests that maternal prenatal stress (often defined as being inclusive of perceived stress, anxiety, and depression) contributes to greater exposure of the fetus to maternal cortisol (e.g., Kapoor, Dunn, Kostaki, Andrews, & Matthews, 2006; Talge, Neal, Glover, & The Early Stress, Translational Research and Prevention Science Network: Fetal and Neonatal Experience on Child & Adolescent Mental Health, 2007), which when prolonged or chronic, may result in alterations to the fetal HPA axis (Van den Bergh, Mulder, Mennes, & Glover, 2005). For example, studies have reported higher baseline cortisol, heightened stress response during stressful tasks, and alterations in HPA axis functioning as evidenced by changes in diurnal cortisol in children who experienced chronic prenatal exposure to high maternal stress (e.g., de Bruijn, van Bakel, Wijnen, Pop, & van Baar, 2009; O'Connor et al., 2005), which in its own right may have negative effects on children's SR (we return to this point later in this review). Studies have demonstrated that changes extend beyond the HPA axis to include alterations to neural structures important for SR, including thinner ACC (Davis, Sandman, Buss, Wing, & Head, 2013) and reduced gray matter volume in the PFC (Buss, Davis, Muftuler, Head, & Sandman, 2010). In addition to neurobiological evidence, studies have reported behavioral evidence for links between prenatal exposure to elevated maternal stress on top-down and bottom-up SR, such as higher impulsivity and poorer inhibitory control, working memory, and attention (Buss, Davis, Hobel, & Sandman, 2011; Clavarino et al., 2010; Mennes, Stiers, Lagae, & Van den Bergh, 2006; Pesonen et al., 2006; Van den Bergh et al., 2005; for a review of mechanisms of prenatal stress exposure on child development, see Graignic-Philippe, Dayan, Chokron, Jacquet, & Tordjman, 2014).

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[7] In our review, we only focus on nonteratogenic processes. However, it is possible that other prenatal influences may play a role. For example, prenatal substance exposure may play a role because poor SR is a risk factor for substance use/abuse (e.g., Quinn & Fromme, 2010; Wills, Pokhrel, Morehouse, & Fenster, 2011), and maternal substance use while pregnant places offspring at risk of poor SR (see Bridgett & Mayes, 2011; Mayes, 2002; Mayes, Grillon, Granger, & Schottenfeld, 1998 for discussion related to prenatal cocaine exposure). Future work will want to empirically consider possibilities wherein parent SR may place children at greater risk of exposure to teratogens, either prenatally or postnatally.

Given the SR-related consequences of chronic prenatal exposure to elevated maternal stress, we believe that mothers who employ SR to modulate their own stress reduce the chances of such prenatal programming processes unfolding. Although studies have not yet directly considered such a possibility in pregnant women, several studies offer supporting evidence. Stawski et al. (2011) found that participants with better executive functioning had diurnal profiles of salivary cortisol indicative of healthier stress response systems. Similar findings were obtained by Hendrawan, Yamakawa, Kimura, Murakami, and Ohira (2012) and Compton, Hofheimer, and Kazinka (2013), who noted that better executive functioning was related to lower salivary cortisol in response to stressors. Several studies of emotional SR have reported that more adaptive emotion regulation strategies are associated with lower cortisol response to stress (e.g., Lam, Dickerson, Zoccola, & Zaldivar, 2009; Quirin, Kuhl, & Dusing, 2011). Likewise, RSA plays a role in inhibiting cortisol (see Thayer & Sternberg, 2006), producing negative relations between RSA or HRV and cortisol under stressful conditions, and between baseline cardiac indices and cortisol reactivity to stress (e.g., Bueno et al., 1989; Johnson, Hansen, Sollers, Murison, & Thayer, 2002; La Marca et al., 2011; Weber et al., 2010). Thus, these studies point to the role of top-down SR in the regulation of HPA axis activity in adults (for a review, see Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009).

In addition to studies examining relations between top-down SR and cortisol, other studies have consistently shown the role of adequate top-down SR in protecting against stress, anxiety, and depression (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Carver et al., 2008; Moore, Zoellner, & Mollenholt, 2008). Given that stress, anxiety, and depression have been related to elevated maternal cortisol during pregnancy (e.g., Field & Diego, 2008; Field, Diego, & Hernandez-Reif, 2006; Sarkar, Bergman, Fisk, & Glover, 2006), these studies provide additional indirect support for the possibility that better maternal SR may protect children against prenatal exposure to chronically high levels of maternal cortisol. Next, we turn to animal models of the effects of prenatal stress on offspring SR, which helps to fill some gaps in the available human literature.

### Evidence From Animal Models

Animal models provide strong evidence for the effects of prenatal exposure to maternal stress on developing neurobiological mechanisms of SR (see Charil, Laplante, Vaillancourt, & King, 2010; Lupien, McEwen, Gunnar, & Heim, 2009; Kofman, 2002 or Weinstock, 2005, 2008 for reviews). In the rodent PFC, changes to spine density and dendritic complexity have been observed in prenatally stress exposed offspring (Murmu et al., 2006). Lateralized changes to right PFC dopamine function also have been noted (Fride & Weinstock, 1988). Differences in prefrontal connectivity also occur as a result of prenatal stress exposure, with reductions in interhemispheric coupling resulting in the diminished ability of the left PFC to inhibit activation of stress systems by the right PFC (Fride & Weinstock, 1987). In the mPFC, high prenatal stress contributes to alterations in the expression of dopamine and glutamate receptors (Berger, Barros, Sarchi, Tarazi, & Antonelli, 2002) and significant changes to the complexity and length of dendritic arbor, the density of excitatory spines, and the volume of neurons and glial cells (Mychasiuk, Gibb, & Kolb, 2012a). Similar changes have been observed in the OFC, with prenatal stress contributing to decreases in dendritic branching and length in the basilar branches of the OFC (Muhammad, Carroll, & Kolb, 2012). Changes to bottom-up neural mechanisms of SR also have been reported in rodent and primate studies (e.g., Bock, Murmu, Baila, Weinstock, & Braun, 2011; Converse et al., 2013; Lee et al., 2011), supporting the notion that prenatal stress affects offspring top-down and bottom-up SR at the level of neurobiology.

Following birth, antenatal stress exposure has persistent effects on the development of neurobiological mechanisms of SR via alterations to the offspring's HPA axis. As mentioned previously, prenatal stress exposure enhances sensitization of the offspring's stress response system (e.g., Henry, Kabbaj, Simon, Le Moal, & Maccari, 1994; Matthews, 2002; Weinstock, 1997). This heightened propensity for stress can lead to heightened physiological reactivity to stressors, an experience that, if chronic, can contribute to structural remodeling of the PFC, which may result in SR difficulties (Holmes & Wellman, 2009). Such postnatal SR-related difficulties include poorer inhibitory control and greater impulsivity (Son et al., 2007; Wilson, Schade, & Terry, 2012), reduced attention span (Schneider, Moore, Kraemer, Roberts, & DeJesus, 2002), and heightened emotional reactivity (Chapillon, Patin, Roy, Vincent, & Caston, 2002; Masterpasque, Chapman, & Lore, 1976; Schneider et al.). In sum, experimental animal models of prenatal stress exposure support the causal influence of prenatal stress exposure on offspring postnatal neurobiologically based deficits in SR.

### Summary and Directions for Future Work

The available evidence suggests that intergenerational transmission of SR begins in the prenatal environment. Mothers that have poor top-down SR may be inadequately equipped to cope with stressors, resulting in fetal exposure to heightened maternal cortisol. In turn, such exposure has programming effects on offspring HPA axis and neurobiological mechanisms of SR. As we pointed out, where human studies are limited, animal models fill some gaps. However, despite evidence in support of our tentative conclusion that the prenatal environment may be an important influence in the intergenerational transmission of SR, human studies are now needed that directly test the effects of maternal top-down SR on cortisol regulation during pregnancy and that subsequently consider children's SR postnatally—a critical gap to fill in the literature at this juncture.

## Genetic Influences

Up to this point, we have focused on evidence of relations between parent SR and rearing contexts. However, socialization and prenatal processes operate in tandem with genetic mechanisms that confer risks and benefits to children's developing SR. Next, we turn to key behavioral and molecular genetics studies pertaining to SR. Similar to the literature regarding links between behavioral and neurobiological factors reviewed above, there are relations between behavioral and molecular genetic substrates and individual differences in top-down and bottom-up SR (but see MacDonald, 2008, for some distinctions in the evolutionary roles of distinct aspects of SR). In addition to providing a comprehensive overview of the genetics of SR, we review studies on relations between parental gene polymorphisms for SR phenotypes and caregiving behaviors—that is, examination of relations between parental SR at the genetic level of analysis on parenting behavior.

## Behavioral and Emotional Self-Regulation

Behavioral genetic studies provide estimates of variance in individual differences that account for family member similarity attributable to heritable genetic factors and nongenetic factors (i.e., shared environment), as well as factors that do not contribute to family member resemblance (i.e., nonshared environment, including random measurement error). In terms of behavioral SR, studies of effortful control among children and adolescents point to evidence, across informants, of moderate levels (typically 40% to 60% range) of heritable and nonshared environmental variance and negligible shared environmental variance (Goldsmith, Buss, & Lemery, 1997; Lemery-Chalfant, Doelger, & Goldsmith, 2008; Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009; see Yamagata et al., 2005 for similar findings in adults). Similar results have been obtained for children's self-control (Wright & Beaver, 2005) with evidence accruing for assortative mating and heritable intergenerational transmission from parent-offspring designs (Boutwell & Beaver, 2010).

By comparison with the smaller literature on effortful control and self-control, there has been extensive genetically informative research on executive function. Results indicate moderate to high heritability (60% to 90% range), modest to moderate nonshared environmental variance (10% to 40% range), and negligible shared environmental variance based on general and domain-specific executive function performance using laboratory and clinical tasks. Heritability exceeds 70% when error variance is statistically removed, with substantial genetic overlap in heritable variance between inhibitory control, cognitive flexibility, and working memory (Doyle et al., 2005; Kuntsi et al., 2006; Luciano et al., 2001; Miyake & Friedman, 2012; Polderman et al., 2006; Wang, Deater-Deckard, Cutting, Thompson, & Petrill, 2012). Similar effects have been observed for ERP, fMRI, and brain volume endophenotypes during inhibitory and working memory tasks (e.g., ERP N2 and P3 waves, Anokhin, Heath, & Myers, 2004; ERP slow wave [SW], Hansell et al., 2010; fMRI BOLD response, Blokland et al., 2008, and Blokland et al., 2011; brain volume, Posthuma et al., 2002). However, research on young children suggests more ambiguous genetic and environmental variance estimates (Groot, de Sonneville, Stins, & Boomsma, 2004).

In terms of emotional SR, it appears that all of the research has focused on physiological indicators (i.e., to our knowledge, there is no relevant literature for reappraisal or suppression). With regard to RSA, 33% to 66% of the variance is heritable based on twin studies in childhood (Tuvblad et al., 2010), adolescence (de Geus, Kupper, Boomsma, & Snieder, 2007), and adulthood (Snieder, van Doornen, Boomsma, & Thayer, 2007; Snieder, Boomsma, van Doornen, & de Geus, 1997; Uusitalo et al., 2007). These studies also suggest that shared environmental variance may account for about 20% of the variance in childhood RSA, but is negligible by adulthood. A similar pattern is seen for measures of HRV (Kupper et al., 2004), with heritability highest when multiple indicators of HRV are considered simultaneously (Wang, Thayer, Treiber, & Snieder, 2005). Researchers also have reported evidence of a common genetic comorbidity shared between depression and low HRV (Su et al., 2010)—an important finding in regard to the intergenerational transmission of covarying cardiovascular and mood dysregulation symptoms. There also is evidence for additional genetic variance in sympathetic and parasympathetic cardiac variables when measured during physical and mental stressor tasks (de Geus et al., 2007; Wang et al., 2009). Finally, volumetric measures of neural structures important for behavioral and emotional SR show substantial heritability even in childhood (65% to 85% of the variance; see Batouli, Trollor, Wen, & Sachdev, 2014 for a review).

With respect to molecular genetics studies of behavioral SR, there has been extensive interest in candidate genes in dopamine and serotonin neurotransmitter systems that are involved in executive functioning and effortful control, based on evidence from association studies (Barnes, Dean, Nandam, O'Connell, & Bellgrove, 2011; Rothbart & Posner, 2005). The dopamine transporter (DAT1) gene includes a commonly studied 40-base pair repeat sequence; the 10-repeat allele has been associated with poorer inhibitory and attentional control (Gill, Daly, Heron, Hawi, & Fitzgerald, 1997; Roman et al., 2002; Waldman et al., 1998; Winsberg & Comings, 1999). Dopamine receptor 4 (DRD4) includes a commonly studied variant of a 48-base pair repeat sequence; individuals with the 7-repeat allele may have poorer inhibitory and attentional control (Auerbach, Benjamin, Faroy, Geller, & Ebstein, 2001; Ebstein, 2006; Fan, Fossella, Sommer, Wu, & Posner, 2003). Furthermore, there appears to be genetic dominance effects at this locus that emerge over childhood (Deater-Deckard & Wang, 2012).

Dopamine receptor 2 (DRD2) includes a Taq1A variant with common alleles A1 and A2. The A1 allele has been associated with variation in conflict/error monitoring (Klein et al., 2007). Serotonin transporter gene (5-HTT) has a 44 base-pair repeat insertion (long allele) versus deletion (short allele). The short allele has been related to poorer conflict/error monitoring and bias to negative emotions in automatic emotion processing (Canli, Ferri, & Duman, 2009). Similarly, a study in early childhood found that a combination of variants in the serotonin transporter gene was associated with ego resiliency, a personality dimension that represents resilience to environmental changes and stressors that has parallels with effortful control (Taylor, Spinrad, et al., 2013). However, there have been a number of nonreplications of these candidate gene associations for executive function performance (e.g., Kluger, Siegfried, & Ebstein, 2002; Palmer et al., 1999; Todd et al., 2001).

The mixed results that are typical of candidate gene studies are well exemplified in the comparison of two meta-analyses of executive function and the COMT gene. In one meta-analysis that included behavioral performance measures, there was no evidence of an association in spite of initial positive findings in the literature (Barnett, Scoriels, & Munafò, 2008). In contrast, another meta-analysis that included neuroimaged endophenotypes of frontal lobe activation indicative of cognitive control showed a substantial association with COMT (Mier, Kirsch, & Meyer-Lindenberg, 2010). For the foreseeable future, while meta-analytic studies seek to identify the systematic moderators of relations between behavioral SR phenotypes and genetic polymorphisms, caution is warranted when making generalizations about candidate gene association effects.

In regard to the molecular genetics of emotional SR, the literature is limited to physiological indicators of SR. One pedigree analysis of adults implicated a major recessive gene for variance in vagal tone (Sinnreich, Friedlander, Luria, Sapoznikov, & Kark, 1999). This was followed by discoveries of various candidate quantitative gene loci throughout the genome (Singh et al., 2002). A number of variants within the NOS1AP gene also have been implicated in heart rate functioning and dynamics, although it is unclear if these candidate markers are consistent across populations (Shah et al., 2013). Although mixed, genetic evidence also suggests that serotonin neurotransmission accounts for some variance in cardiorespiratory physiology (McCaffery et al., 2006). For instance, one study of young adults found that having one or two copies of the short allele of serotonin transporter gene (5-HTTLPR) was associated with lower resting RSA/vagal tone, compared with individuals with two copies of the long allele (Ellis, Beevers, Hixon, & McGeary, 2011). However, a subsequent study using more precise genotyping of a single nucleotide polymorphism within this gene yielded null findings (Vulturar, Chis, Ungureanu, & Miu, 2012). Finally, dopamine may be implicated as well. A study of changes in vagal tone from rest to stressor to recovery showed that children with the val/val allele of the COMT gene had stronger declines and weaker recoveries of vagal tone compared to met/met individuals (Mueller et al., 2012), results that now require replication.

More generally, molecular genetic studies of broader measures of cardiac stress reactivity, including changes in indices when shifting from resting to stressor states, point to several candidate gene variants involved in the neuromodulation of physiological stress reactivity and regulation (e.g., tyrosine hydroxylase, β1- and β2-adrenergic receptors, $\alpha_{2C}$-adrenergic receptor; see Wu, Snieder, & de Geus, 2010). Nevertheless, several caveats are necessary. Results are mixed, studies of youth are rare, and the literature has focused on indicators of sympathetic reactivity at the expense of examining parasympathetic regulation (e.g., Frigerio et al., 2009; Gilissen, Bakermans-Kranenburg, van IJzendoorn, & Linting, 2008; for an overview see Mueller et al., 2012).

## Impulsivity

Turning to bottom-up impulsivity, a meta-analysis (*N* of 27,147 across studies), which used a variety of behavioral genetic designs, reported a heritability estimate of 50%, with nonshared environmental influences also estimated to be 50% and negligible shared environmental variance (Bezdjian, Baker, & Tuvblad, 2011). Although not a formal component of their analysis, our own examination of their tables indicated a similar pattern of genetic and nonshared environmental variance components for measures focused on behavioral inhibition and those focused on impulsivity. Because of the age range of participants included in this meta-analysis, the authors were able to demonstrate that genetic effects were relatively stronger in children and males, and that nonshared environmental effects were important across development. Those results strongly corroborated findings from McCartney, Harris, and Bernieri's (1990) seminal meta-analysis of twin studies of personality and temperament who reported heritability estimates in the .50 range for impulsivity. An even higher heritability estimate of .73 (nearly all of it additive genetic variance) was reported for hyperactivity/impulsivity, in Nikolas and Burt's (2010) meta-analysis of ADHD symptoms. Finally, studies of the volume of deep brain structures most relevant to bottom-up regulation (e.g., putamen, caudate) also show substantial heritability even in childhood, with potentially modest decreases in genetic variance over the life span based on cross-sectional evidence (Batouli et al., 2014).

In addition to playing key roles in top-down mechanisms of SR, dopamine plays a critical role in bottom-up impulsivity. Hundreds of candidate gene studies have been published in the past 20 years that examine relations between dopamine system gene variants and the major components of impulsive behavior. A series of meta-analyses in the 2000s provided converging evidence of modest association with either or both of two variants in DRD4—the 7-repeat 48-bp VNTR and C-521T, a T/C single nucleotide polymorphism in the gene's promoter region (Munafò, Yalcin, Willis-Owen, & Flint, 2008). Beyond dopamine, studies of the impulsive, hyperactive and inattentive symptoms of ADHD have identified numerous candidate genes and regions across multiple chromosomes. However, none have reached statistical significance in genome-wide association analyses, suggesting that the genetic variation in impulsivity detected in behavioral genetic studies may reflect additive and interactive effects of numerous rare variants, rather than just a few common variants (for a meta-analysis and review see Neale et al., 2010).

## Studies Linking Regulation-Related Polymorphisms to Parenting Behavior

Given the literature that has established consistent relations between parent SR and parenting behavior at behavioral and neural levels, and the separate literature noting polymorphisms associated with SR, we considered the possibility that there may be evidence in the literature of relations between SR measured at the molecular genetic level and parenting behavior. As noted earlier, candidate genes involved in neuromodulation of dopamine and serotonin are of importance for considering genotypic associations with variation in well-regulated cognitions and behaviors. Therefore, given the role of SR in caregiving behavior, it is not surprising that the new literature of human studies on candidate gene associations with parenting behaviors has focused on the most widely studied, well understood candidate genes in those systems. For example, Lee et al. (2010) found that DAT1 was associated with harsher parenting. Polymorphisms in dopamine receptor 1 and 2, long implicated in initiation and regulation of caregiving behavior in rats (see Curry et al., 2013, for a review and experimental study showing interactions between dam and pup genotypes), also may be important for explaining variability in human maternal attention to an infant's needs (Mileva-Seitz et al., 2012). In addition, the presence of the commonly studied "short" allele of the serotonin transporter gene has been associated with less maternal sensitivity (Bakermans-Kranenburg & van IJzendoorn, 2008). However, at least in regards to alleic variations in the serotonin transporter gene, findings have been mixed (see Cents et al., 2014). Emerging evidence also suggests the presence of multiple statistical interactions involving other functional polymorphisms and the mothers' own child rearing history that may in turn affect parenting behavior (Mileva-Seitz et al., 2011).

More generally, maternal genotypic differences may matter most when considered in the face of stressors. For example, the presence of the 7-repeat allele of DRD4 has been related with greater maternal reactivity to challenging infant temperament (e.g., strong and frequent crying), with such mothers showing more sensitive caregiving for low-crying infants but the least sensitive caregiving for the fussiest infants (Kaitz et al., 2010). In a similar vein, the relation between DAT1 and harsh caregiving noted previously was stronger for mothers whose children were harder to manage (Lee et al., 2010). Furthermore, the combination of DRD4 and COMT alleles that are most strongly linked with poor neuromodulation of dopamine has been shown to interact with high parenting stress in the prediction of poor maternal sensitive caregiving (van IJzendoorn, Bakermans-Kranenburg, & Mesman, 2008). In sum, an emerging finding is clear, at least with respect to candidate genes in the dopamine system. Genetic markers of dopamine regulation that have been implicated in SR appear to explain variance in well versus poorly regulated caregiving. However, these effects may interact with stressors arising from the child's behavior, the broader rearing environment, or both.

## Section Summary and Recommendations for Future Work

Our overview of the behavioral and molecular genetics literature points to the long known importance of genetics in the intergenerational transmission of SR. Although findings of specific polymorphisms have at times been inconsistent across studies, the cumulative evidence provides the strongest support for dopamine and serotonin polymorphisms. Moreover, given associations between polymorphisms important for dopamine and serotonin neuromodulation and performance on behavioral measures of SR, we reviewed the emerging evidence that links genetic-level assessment of SR to variations in parenting behaviors. This evidence provides additional support, at a different level of analysis, for the role of SR in caregiving behavior that influences children's SR.

However, our overview of this literature also points to gaps to be addressed in future work. Existing work has focused almost entirely on endophenotypes involved in neuromodulation of top-down processes of behavioral SR and impulsivity. By comparison, studies have only considered cardiac biomarkers of emotional SR; no behavioral or molecular genetic studies have addressed questionnaire and task-based measures of cognitive emotional SR (e.g., reappraisal or suppression). Moreover, the emerging work that considers relations between parent genetic polymorphisms important for SR and parenting behavior is in need of replication, and extension to polymorphisms that have been identified as being important for cardiac indices of emotional SR. Finally, although behavioral genetic studies indicate that shared environmental variance in SR is modest, there is behavioral genetic evidence for familial similarity between and within generations in aspects of behavioral and emotional SR, and impulsivity, that is mediated in part by family dynamics including parenting behaviors (Deater-Deckard & Petrill, 2004; Harold et al., 2013). Therefore, genetic *and* social influences must be considered in any biologically plausible model of the intergenerational transmission of SR—a key implication for the model we introduce next.

## Self-Regulation Intergenerational Transmission Model

We have reviewed individual pieces of evidence along with supporting literatures that point to the role of prenatal programming, postnatal contextual mechanisms, and genetic influences in the intergenerational transmission of SR. As such, these factors play key roles in our presentation of the self-regulation intergenerational transmission model (see Figure 1). To tie individual pieces of evidence together and complete our formulation of the self-regulation intergenerational transmission model, next, we introduce how contextual mechanisms influenced by parent SR "get under the skin," including gene-environment interplay and potential epigenetic processes, to affect children's neurobiological mechanisms of SR. We also will note the transactional nature of the model, focusing on potential child and context effects on parent SR, representing feedback loops that may perpetuate intergenerational transmission processes.

## Stress Physiology Underlying Intergenerational Transmission Processes

It is unlikely that the contextual influences acting as intergenerational transmission mechanisms act directly on SR. Instead, these mechanisms appear to act on SR via stress physiology operating within neuroendocrine and neurotransmitter systems that influence the neurobiological mechanisms of SR at the physiological level. Such reasoning has already appeared in the literature on SR (Blair, 2010) and allostatic load (see McEwen, 1998 or Gunnar & Quevedo, 2007). The latter has been more often considered in association with HPA axis and stress dysregulation (e.g., Evans & Kim, 2007), but more recently has been noted as affecting self-regulatory mechanisms (see Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011 or Pechtel & Pizzagalli, 2011). Given the importance of stress physiology for understanding intergenerational transmission processes, we briefly note several key mechanisms, focusing on the effects of stress on the neurobiology of SR, and extend existing literature and theoretical reviews by pointing to neurotransmitter systems that have received less attention but are no less critical for SR.

**Glucocorticoids.** Although physiological stress responses to normal day-to-day variations in experiences are adaptive and help maintain homeostasis, among other important functions, chronically high and/or unpredictable extreme stressors disrupt self-regulatory systems in part via chronic exposure to stress hormones (e.g., cortisol). Consistent with this notion, existing evidence supports links between poor parenting and marital adjustment, mechanisms of transmission we reviewed earlier, and children's higher stress, including elevated cortisol (e.g., Blair et al., 2006; Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Evans & Kim, 2007; Grant et al., 2009; Pendry & Adam, 2007; Mills-Koonce et al., 2011; Repetti, Taylor, & Seeman, 2002; Taylor, Spinrad, et al., 2013; Webster-Stratton & Hammond, 1988). Such effects extend to the broader rearing context, with strong evidence for relations among lower SES, higher home chaos and elevations in children's cortisol (Bush, Obradovic, Adler, & Boyce, 2011; Chen, Cohen, & Miller, 2010; Laurent et al., 2013; Lupien, King, Meaney, & McEwen, 2001; Turner & Avison, 2003).

Chronically elevated stress during development sensitizes the stress-response system (e.g., Essex, Klein, Cho, & Kalin, 2002; Saltzman, Holden, & Holahan, 2005), which contributes to elevated cortisol that persists over time (see Hackman & Farah, 2009 and Raizada & Kishiyama, 2010 for overviews). In addition to other adverse effects (e.g., heightened reactivity to threat), chron-

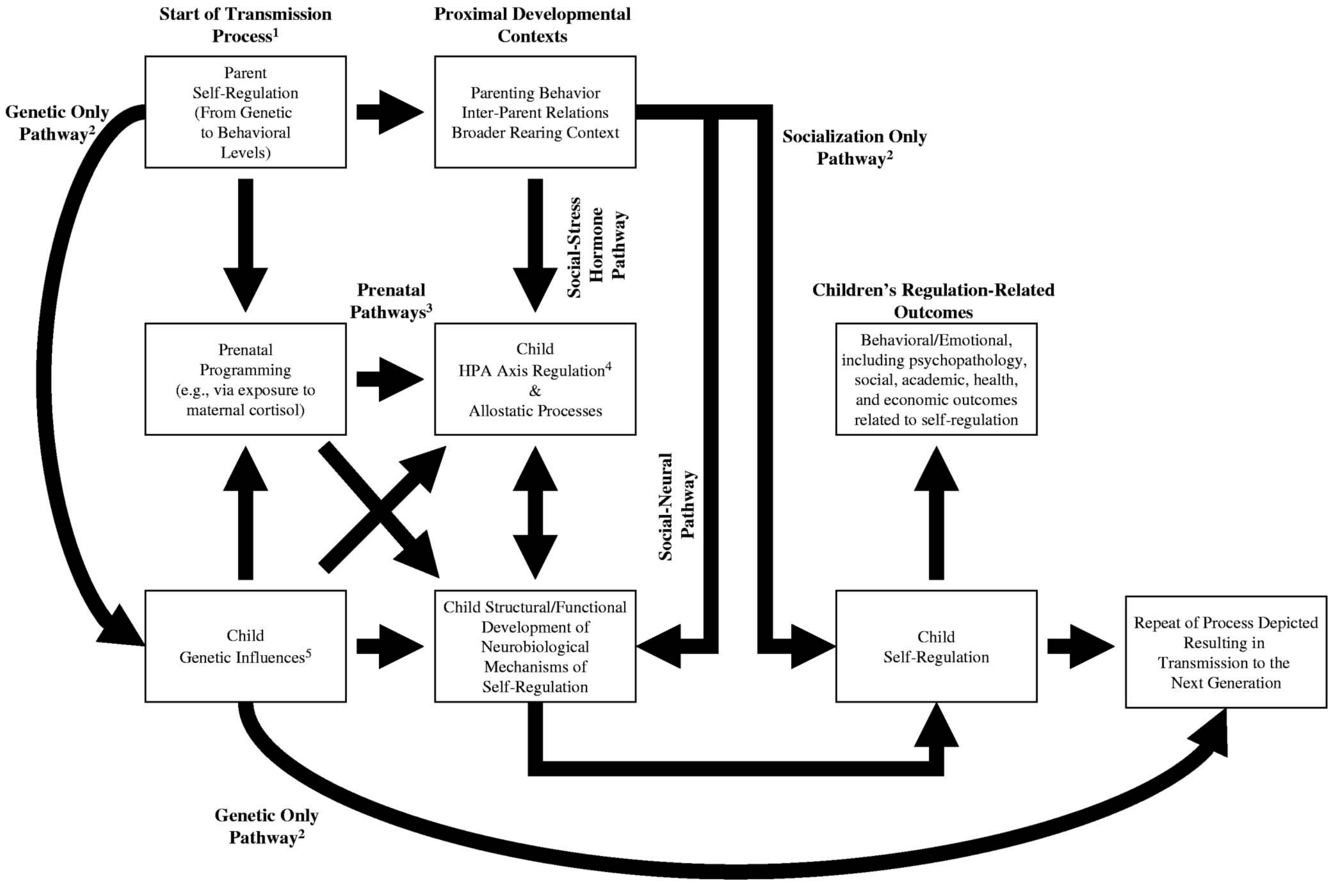

*Figure 1.* Self-regulation intergenerational transmission model. 1. All pathways lead back to parent self-regulation, reflecting the starting point of the intergenerational transmission process, as articulated throughout our review. For clarity, transactional processes, described in the text, are not depicted. Importantly, the model only applies to behavioral and emotional self-regulation, and to impulsivity. Although there is some evidence that suggests similar processes underlie the intergenerational transmission of behavioral inhibition/fear, as we have noted in the body of our review, there is currently insufficient empirical evidence to warrant inclusion of this aspect of bottom-up self-regulation in the model. 2. As we have depicted, genetic and socialization influences are critical components to our integrated model. Nevertheless, we selected the labels "Genetic Only" and "Socialization Only" for these pathways to illustrate more clearly that these pathways have historically been pointed to as representing the way in which self-regulation is transmitted across generations. 3. There are two prenatal pathways—one to children's HPA axis and allostatic processes, which then affect neurobiological mechanisms of self-regulation, and one directly to neurobiological mechanisms of self-regulation. 4. HPA axis refers to children's hypothalamic-pituitary-adrenal axis. 5. Genetic influence refers to genetic factors that make children more or less susceptible to the processes depicted. Potential epigenetic processes are not depicted for clarity, but are discussed in the text and can be inferred in the model. For example, epigenetic processes may be at play in neurobiological mechanisms given that the Genetic Influence arrow and the Social-Neural arrows are converging. Potential epigenetic modifications would be anticipated anyplace child Genetic Influence arrows converge with other influences.

ically elevated cortisol has been linked to lower effortful control in preschoolers (Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997; see Dettling et al., 2000 for similar findings), whereas more adaptive patterns of cortisol reactivity are related to better executive functioning (Blair, Granger, & Razza, 2005; Finy, Bresin, Korol, & Verona, 2014). Similarly, Blair et al. (2011) reported that relations among parenting, poverty, and young children's executive functioning were mediated by cortisol (also see Doan & Evans, 2011, who found that higher allostatic load was inversely related to working memory in adolescents). Likewise, adults reporting more negative affect appear to have elevated cortisol (Piazza, Charles, Sliwinski, Mogle, & Almeida, 2013), suggesting that poor emotional SR may be related with dysregulated HPA axis functioning—findings that are similar to those in other studies of adults (e.g., Carlson, Dikecligil, Greenberg, & Mujica-Parodi, 2012; Lam et al., 2009) and children (e.g., Scher, Hall, Zaidman-Zait, & Weinberg, 2010).

Evidence also suggests that elevated cortisol is related to higher impulsivity. Laceulle, Nederhof, van Aken, and Ormel (2014) reported that higher morning basal cortisol in adolescents was related to higher impulsivity and lower self-discipline; however, relations among impulsivity, self-discipline and other cortisol measures (e.g., obtained after a stressor) were not significant. Other studies have reported similar findings (i.e., higher cortisol related to more impulsivity) for boys but not girls (e.g., Dettling, Gunnar, & Donzella, 1999). Although these studies accentuate the potential complexity of the relations between cortisol and impulsivity that future work needs to routinely consider, other studies have reported direct relations between elevated cortisol and more impulsivity (e.g., Almeida, Lee, & Coccaro, 2010; Bruce, Davis, & Gunnar, 2002; Hatzinger et al., 2012).

At the neurobiological level, heightened exposure to glucocorticoids appears to contribute to structural and functional alterations to a range of neural systems (see Lupien et al., 2009 or de Kloet, Sibug, Helmerhorst, & Schmidt, 2005 for reviews). Systems that are adversely affected include those important for SR that contain high densities of glucocorticoid receptors, such as the cingulate gyrus (implicated in effortful control; e.g., Bush, Luu, & Posner, 2000), amygdala and hippocampus (involved in learning, memory, emotion processing, and behavioral inhibition/fear; e.g., McEwen, 1999; Phelps & LeDoux, 2005), nucleus accumbens (involved in impulsivity; Sinclair, Purves-Tyson, Allen, & Weickert, 2014), and frontal areas (involved in behavioral and emotional SR; Gold et al., 2005). Indeed, chronic elevations in cortisol appear to be associated with decreased frontal lobe volume and concomitantly poorer executive functioning (Gold et al., 2005; Meaney et al., 1996).

Although the evidence is compelling for the stress hormone process briefly articulated above, there are examples of nonsignificant findings (e.g., Taylor, Spinrad, et al., 2013) and of lower top-down SR and higher impulsivity being related to lower cortisol (see Davis, Bruce, & Gunnar, 2002; Lengua, Zalewski, Fisher, & Moran, 2013; Poustka et al., 2010; Zalewski et al., 2012). However, as suggested by Zalewski et al. (2012), profiles of blunted cortisol reactivity also may be consistent with HPA dysregulation. Given the sometimes inconsistent findings in the literature, more rigorous research is needed that addresses developmental timing, length of stress exposure, and potential social and biological moderating factors (see Sinclair et al., 2014 for a review in relation to sex hormones), in understanding the relations among stress hormones, such as cortisol, and SR mechanisms.

Finally, one study, although cross-sectional, has directly tested the patterns of relations that are generally supported by mostly separate literatures. Gonzalez, Jenkins, Steiner, and Fleming (2012) examined mother reports ($N = 89$) of their own stressful early life experiences, operationalized as consistency of care (i.e., parental loss) and maltreatment, diurnal cortisol, performance during measures of attention shifting and working memory (higher scores were indicative of poorer executive functioning), and sensitivity during interactions with their own infants. Gonzalez et al.'s (2012) key finding was that the effect of maternal stressful early life experience on parenting behavior was indirect through higher diurnal cortisol, which negatively affected maternal working memory (but not attention shifting), which in turn, was related to lower sensitivity during interactions with her own infant.

**Neurotransmitters.** In addition to stress hormones, the monoamine neurotransmitters dopamine, serotonin, and norepinephrine play integral roles in both SR and stress response. These neurotransmitter systems are spread throughout limbic, striatal and cortical regions, and intersect in the mPFC in particular (Flugge, van Kampen, & Mijnster, 2004)—areas that are critical for top-down and bottom-up SR. In brief, this process starts with threat detection and concomitant heightened emotional reactivity originating at the neural level in the amygdala, which engages the sympathetic nervous system (Goldstein, Rasmusson, Bunney, & Roth, 1996). Subsequently, adrenal steroids interact with serotonin and other neurotransmitters to signal modifications to existing neurons and creation of new neurons in limbic regions involved in the regulation of the stress response (McEwen, 2007), which is part of the central nervous system's attempt to regulate and maintain homeostasis. Likewise, the dopamine system is activated by stress preferentially in the mPFC for typical periodic stress and in the nucleus accumbens for chronic stress, with evidence also pointing to the role of mPFC in regulating dopamine response in the nucleus accumbens (e.g., Pascucci, Ventura, Latagliata, Cabib, & Puglisi-Allegra, 2007). Consequently, over time, stress can have deleterious effects on SR by decreasing top-down SR, and increasing impulsive behavior via effects at the neural level (see Gatzke-Kopp, 2011 for a comprehensive overview of the effects of stress on mesolimbic dopaminergic function, including some discussion of top-down dopaminergic functioning).

Despite the fact that most work has focused on animal models, an example in humans can be seen in the role of the attention network in vigilance and SR of emotion and behavior in the face of threat. Executive attention is vital to inhibitory control and resolution of cognitive conflict in the face of stressors. This process involves the interconnectivity of cortical and limbic regions via the ACC, which also is heavily regulated by dopamine. The development of electrophysical and biochemical connections, via dopamine in the frontal cortex, are genetically constrained yet operate in response to experience—and they are fundamental to individual differences in the top-down regulation of stress responses (Posner & Rothbart, 2009). Along similar lines, research has reported interactions between stress and genetic polymorphisms (e.g., COMT) influencing dopamine availability in the PFC, which in turn affected working memory processes (e.g., Buckert, Kudielka, Reuter, & Fiebach, 2012).

Although the monoamines have received the most attention, there are other neurotransmitters that are important for SR and show susceptibility to disruption by stress. Gamma-aminobutyric acid (GABA) and glutamate appear to be particularly important as both play roles in behavioral SR processes such as working memory (e.g., Aultman & Moghaddam, 2001; Lewis, Pierri, Volk, Melchitzky, & Woo, 1999) and inhibitory control (e.g., Jupp et al., 2013; Lesch, Merker, Reif, & Novak, 2013; Murphy et al., 2012). Glutamate and GABA also are important for emotional SR (e.g., Caballero, Thomases, Flores-Barrera, Cass, & Tseng, 2014; Sanacora, Treccani, & Popoli, 2012). Moreover, like the monoamines, GABA and glutamate are sensitive to chronic or uncontrollable stress (e.g., Knox, Perrine, George, Galloway, & Liberzon, 2010; Popoli, Yan, McEwen, & Sanacora, 2012), with effects found in the PFC when rodents are reared in stressful contexts (e.g., Melendez, Gregory, Bardo, & Kalivas, 2004). Thus, acute and/or prolonged stress early in life may disrupt development of top-down SR processes that are modulated in part by GABA and glutamate.

**Section summary and future directions.** Over time, chronic exposure to stressors, including those likely arising in part from poor parent SR, may lead to lasting disruption of children's neurobiological mechanisms of SR. Stress response at the physiological level, including hormones and neurotransmitters, contributes to such disruptions, providing a physiological basis for the perpetuation of poor SR across generations. However, this conclusion rests mostly upon a longstanding animal literature, particularly in regards to effects on neurotransmitter systems. Existing work in humans generally converges with findings in the animal literature, but additional work in human samples is needed. Specifically, although we have brought together separate pieces of well-supported literature (but see Gonzalez et al., 2012 for a study integrating these literatures empirically, and showing anticipated relations) in proposing a key role of stress physiology in the intergenerational transmission of SR, work is now needed that more directly and rigorously tests this premise by regularly considering the role of parent SR in the processes we have articulated here.

## Gene-Environment Interplay and Epigenetic Processes

Although we have emphasized the interplay between biological and contextual factors in the intergenerational transmission of SR, we have not yet discussed the role of gene-environment interplay, specifically gene-environment correlation (rGE), in such processes. This is an important consideration because parent SR genes that can be inherited by children also appear to be influencing the contextual factors discussed in this review (e.g., parenting). When genetic factors in children's SR also affect rearing contexts, evocative rGE (i.e., a heritable attribute evoking an environmental response) and passive rGE (i.e., association between the child's inherited genotype and their rearing) may be playing roles (see Avinun & Knafo, 2013 or Jaffee & Price, 2007 for discussion). For instance, the same genes that contribute to poor parental SR may evoke more conflict in marital relationships, with detrimental effects on children's SR. In terms of passive rGE, the genes that contribute to child and parent SR also appear to contribute to caregiving behaviors, with poorly regulated parents less able to provide parenting supportive of children's SR development. Thus, children who inherit genes that confer risk for SR difficulties are more likely to experience stressful contexts, compounding their risk for adverse self-regulatory outcomes.

Adoption-at-birth designs are well-suited for teasing apart the contributions of rGE to heritable phenotypes and to shedding light on questions regarding the intergenerational transmission of SR because passive rGE as an explanation for associations between adoptive parent and adopted child SR can be ruled out. As a recent example, Harold et al. (2013) reported associations between adoptive fathers' reports of adopted children's ADHD symptoms and adoptive mothers' reports of their own ADHD symptoms. Harold et al. also reported a positive relation between adoptive mother ADHD symptoms and hostility toward her adopted child, which, in turn, was related to adopted child ADHD symptoms. Finally, adopted children's earlier impulsivity evoked greater adoptive mother hostility, providing evidence of transactional processes (a topic we briefly turn to in the next section). These findings suggest that the intergenerational transmission of SR is not simply attributable to passive genetic transmission. However, although compelling, Harold et al. did not examine the aspects of SR (e.g., impulsivity and executive functioning) that may contribute to ADHD symptoms (Barkley, 1997; Nigg, 2001). As such, though supporting the framework proposed in our review, their findings require replication using direct measures of SR.

Nevertheless, there are limitations to adoption designs (e.g., children are not adopted into environments known to be adverse). However, such limitations can be addressed by animal analogs—cross-fostering designs in which offspring are raised by a nonbiological parent, eliminating the possibility of passive rGE. That is, rodents or other animals manipulated to be at high or low genetic risk can be "adopted out" to different types of mothers (e.g., sensitive vs. neglectful) in a way not possible with humans. Critically, cross-fostering animal studies test gene-environment interaction while controlling for passive rGE, and this literature demonstrates that maternal caregiving acts as an equal or more potent predictor, compared with genetic effects, of offspring behavior (e.g., Francis et al., 1999; Liu, Diorio, Day, Francis, & Meaney, 2000; Weaver et al., 2004). For example, when genetically high-reactive rat pups are cross-fostered to low-reactive dams that exhibit high levels of responsive caregiving, pups show lower reactivity relative to high-reactive pups raised by their reactive biological mothers (Caldji, Diorio, & Meaney, 2000). Such findings are usually interpreted within a stress reactivity framework (see Laurent et al., 2013 for a closely related example in a human adoption study). However, such findings also converge with the animal studies we described earlier in which poor SR was experimentally induced, leading to the very parenting behaviors that have negative effects on offspring stress reactivity independent of passive genetic transmission.

Moving beyond traditional population genetic studies, work is now emphasizing how caregiving contexts can alter gene expression—specifically, experiential imprinting on gene structure and function. Maternal care and stressful caregiving can affect offspring SR in part through epigenetic modifications that alter gene expression by silencing genes or enhancing transcription factor binding (Meaney, 2010). Currently, the most frequently studied epigenetic process is DNA methylation (see van IJzendoorn, Bakermans-Kranenburg, & Ebstein, 2011 for discussion in relation to child development), in which a methyl group binds to cytosine that follows guanine in "CpG sites," converting it to 5-methylcytosine. Greater methylation is related to less gene transcription, with only a subset of CpG sites being unmethylated, typically located in promoter regions (Razin, 1998). Another epigenetic mechanism is histone acetylation, which is the binding of a methyl and a carbonyl group that alters gene expression in those locations. Greater acetylation has been linked with greater binding of transcription factors (Roth, Denu, & Allis, 2001). These changes in gene expression operate as being responsive to information from the environment for each individual—changes that are presumed to best enable survival.

Much of the relevant work on epigenetic mechanisms has employed rodent models of stress reactivity (e.g., HPA axis), and SR to a lesser extent (Oberlander et al., 2008). As we previously discussed, in rodents there is wide variation in caregiving that affects pup stress response regulation (Champagne et al., 2003). Using cross-fostering and artificial caregiving designs to manipulate the quality of caregiving that pups receive, researchers have shown that early rearing plays a key role in the developing structure and function of neurobiological mechanisms of subsequent stress reactivity and regulation—above and beyond effects of shared genes between mother and pup (Francis et al., 1999). Epigenetic modifications explain some of this effect, with poor caregiving early in life being associated with greater methylation and lower acetylation of glucocorticoid receptor genes in the hippocampus. This process interferes with effective feedback in the

HPA axis, producing an exaggerated and prolonged stress reaction (Weaver et al., 2004) that can influence mechanisms of SR, as we described earlier. Critically, such findings in rodents have been extended to nonhuman primates (e.g., Patel, Katz, Karssen, & Lyons, 2008) and to humans (e.g., McGowan et al., 2009).

Of more importance for our review, recent work has extended findings regarding HPA axis reactivity and regulation to epigenetic influences on top-down mechanisms of SR. Blaze, Scheuing, and Roth (2013) reported methylation differences in genes expressed in the mPFC between rats exposed verses not exposed to maltreatment (also see Carlyle et al., 2012). Similar findings have been demonstrated within the PFC of nonhuman primates, including in the dlPFC and vlPFC (Patel et al., 2008; Provencal et al., 2012). Existing evidence from animal models also indicates epigenetic induced changes in response to contextual stressors in subcortical structures implicated in bottom-up SR processes (i.e., impulsivity and behavioral inhibition/fear) including the nucleus accumbens, hippocampus, and amygdala (see Zannas & West, 2014 for a review). There also is reason to believe that prenatal programming effects may arise in part from epigenetic mechanisms. For instance, studies in rodents have reported epigenetic effects in the hippocampus (e.g., Mychasiuk, Ilnytskyy, Kovalchuk, Kolb, & Gibb, 2011) and within the HPA axis (see Glover, O'Connor, & O'Donnell, 2010 for a review). Epigenetic processes also unfold in the frontal cortex in reaction to prenatal stress exposure in rodents (e.g., Mychasiuk, Gibb, & Kolb, 2012b; Mychasiuk et al., 2012a; also see Coulon, Wellman, Marjara, Janczak, & Zanella, 2013, who found effects of prenatal stress on PFC gene expression in lambs). Although these kinds of studies recently have been extended to humans in regards to epigenetic modifications to a glucocorticoid receptor gene (NR3C1; Hompes et al., 2013; also see Sinclair, Webster, Wong, & Weickert, 2011 for an example of epigenetic effects in human dlPFC in a cross-sectional, postmortem study), much more research in humans is needed that can identify prenatal and postnatal stress-induced epigenetic changes in relation to mechanisms of SR. Finally, there is mounting evidence that environmentally induced epigenetic alterations in one generation can be transmitted to the next generation (for a review, see Bohacek, Gapp, Saab, & Mansuy, 2013). However, studies have not yet considered such a possibility for SR, necessitating future work in animal and human models to identify intergenerational epigenetic effects on neurobiological mechanisms of top-down and bottom-up SR.

In sum, evidence suggests that the prenatal and postnatal rearing environment alters gene expression through epigenetic modifications, presumably to adaptively attune the nervous system to the frequency and intensity of stressors that are likely to be encountered by the offspring. Epigenetic alterations in neurobiological mechanisms that indirectly (e.g., HPA axis) and directly influence SR have been identified in animal models, with results now emerging in human samples that parallel some findings in the animal literature. Although these findings allow us to conclude that epigenetic modifications are *likely* involved in the intergenerational transmission of SR, stronger, more specific conclusions are premature until replication, especially with humans, has been achieved. As technological and methodological improvements arise, the field needs to move toward integration of molecular genetic and epigenetic methods with traditional human longitudinal correlational and experimental methods, to more rigorously test biologically plausible theories of intergenerational transmission via gene-environment interplay and epigenetic mechanisms.

## Transactional Processes

Our review has mostly focused on stable individual differences in SR. However, Baumeister's strength model of self-regulation (e.g., Baumeister, Vohs, & Tice, 2007; Muraven & Baumeister, 2000) proposes that SR is a resource that can be depleted as it is utilized (see Muraven & Baumeister, 2000; Vohs, Baumeister, & Ciarocco, 2005 for behavioral evidence and Wagner & Heatherton, 2013 for evidence at the neurobiological level). This model may offer one explanation regarding how parent SR could be affected by context and by children's SR. For example, children's dysregulated behavior or stressful family contexts (e.g., a conflictual interparental relationship) may deplete parent SR, representing transactional feedback loops within the intergenerational framework we have articulated. Such processes have the potential to offer new insights regarding "child effects" on parenting environments (including rGE) and how interparental conflict might spill over into parent–child interactions in ways that further promulgate risk for poor SR (see Buck & Neff, 2012; Schoppe-Sullivan et al., 2007; Stroud, Durbin, Wilson, & Mendelsohn, 2011).

Assuming that individuals with more regulatory resources also perform better on measures of SR, there is evidence that depletion of those resources plays a role in the intergenerational transmission process. Skowron, Cipriano-Essel, Benjamin, Pincus, and Van Ryzin (2013) obtained findings suggesting that interactions with children may be more taxing (i.e., SR depleting) for abusive parents, resulting in increases in negative parenting. Similarly, Deater-Deckard et al. (2010) reported that mothers with lower working memory were more likely to engage in negative parenting in the context of challenging child behavior (using a sibling quasi-experimental design, including a subsample of adoptive families to rule out passive rGE). In a subsequent study, this general pattern was found using a broader maternal executive function construct and maternal resting left-right frontal EEG alpha asymmetry (indicative of emotional reactivity and dysregulation), but only among mothers with few chronic socioeconomic and parenting stressors. In contrast, highly stressed mothers' SR had no effect in reducing harsh reactive parenting, suggesting that their regulatory resources may become overwhelmed in such contexts (Chen, Bell, & Deater-Deckard, 2014; Deater-Deckard, Chen, Wang, & Bell, 2012).

Though the above findings are cross-sectional, results are supportive of a transactional process that unfolds when framed within the resource model of SR. That is, stressful contexts can deplete parents' own SR, particularly in vulnerable individuals. As these processes affect children and they become more dysregulated, parent SR may be further depleted, leaving parents with limited SR resources with which to effectively assist children's regulatory efforts. Subsequently, this may result in dysregulated parenting behavior that continues to negatively affect children's SR, further perpetuating the intergenerational transmission of poor SR. Given the limitations of existing correlational cross-sectional work, longitudinal quasi-experimental and experimental intervention studies testing transactional processes (e.g., cascade models) are now needed to test such possibilities.

## Conclusion and Future Directions

We have described mechanisms, based on over 75 years of findings from diverse literatures, by which SR appears to be transmitted from parents to children. This evidence informed our articulation of the self-regulation intergenerational transmission model, a framework for understanding the complex connections between parent and child SR. Our model outlines the dynamic interplay between social mechanisms that at least in some cases (e.g., parenting behavior) appear to be causally influenced by parent SR, and children's neurobiological mechanisms that in turn affect children's SR. In short, parent SR exerts strong effects on variations in children's rearing contexts. When parent SR is poor, children are at high risk of being exposed to a chronically stressful rearing context, which in some instances may start prenatally. Children's genetic factors intersect with these stressful contexts via gene-environment interplay, likely inclusive of epigenetic mechanisms that eventually result in alterations to their neurobiological mechanisms of SR. These alterations result in dysregulated behavior and emotion that ultimately affect the rearing of the next generation, and to elevated risk of a diverse array of adverse outcomes within and across generations. Despite a strong evidence base for our model, in closing we point to future directions for filling critical gaps, and implications that stem from the model we have proposed.

First, our model is intended to be adapted as new knowledge emerges. We only focused on the four contextual mechanisms that have the clearest supporting evidence. However, there are likely other mechanisms not yet identified or for which there is too little evidence to warrant inclusion at this time. For instance, exposure to neighborhood disadvantage is related to children's stress reactivity, including cortisol (e.g., Hackman, Betancourt, Brodsky, Hurt, & Farah, 2012), which affects their SR. Given that parent SR seems to affect factors (e.g., SES) that influence where families reside, and that those neighborhood factors may influence children's SR, there may be links between parent SR and the neighborhoods in which children reside that act as another contextual mechanism in the intergenerational transmission of SR. Future work will need to consider this and other possibilities. As new candidates for social and/or neurobiological pathways are identified, the self-regulation intergenerational transmission model can be adapted accordingly.

There also are avenues for future work on other genetic, epigenetic, and physiological processes influencing the intergenerational transmission of SR that have yet to be considered or are just emerging as possibilities. Many single genetic polymorphisms that may contribute small but detectable statistical effects on SR, or combinations of polymorphisms (i.e., haplotypes) that collectively contribute more variance, remain unidentified. Likewise, the field has just begun to scratch the surface of gene-by-gene interactions at the same or multiple loci and their role in individual differences (including SR) in humans. Further insights will come from epigenetic processes during distinct developmental periods where intergenerational transmission may be relatively stronger or weaker (see Sinclair et al., 2011, who identified periods early in life and during late adolescence for stress pathway development in the dlPFC). Consideration of hormones that are not traditionally regarded as part of the HPA axis (e.g., androgens) and their interplay with stress hormones and neurotransmitters will be important in future work given existing evidence from animal and human studies (e.g., Mehta & Beer, 2010; Wang, Neese, Korol, & Schantz, 2011). Neuropeptides (e.g., NYP and oxytocin) also may play a role (e.g., Bos, Panksepp, Bluthé, & van Honk, 2012; Ito, Dumont, & Quirion, 2013; Quirin et al., 2011), but have been rarely considered in the SR literature. Inquiry into such biological processes will shed new light on individual differences in SR and may lead to conclusions about why the same chronic stressor (e.g., poor parenting) affects different aspects of SR across individuals (i.e., multifinality). The possibilities noted here represent important next steps in further refining the science of SR at the physiological level.

More research using genetically informed designs is needed, but may face challenges. Given the role of passive and evocative rGE that could lead to overestimates of heritability in twin studies, adoption and step-family sibling and offspring studies are well-suited to identifying rGE when used in conjunction with twin and nontwin sibling designs. However, adoption studies are expensive, and many family contexts into which children are adopted are less stressful compared with their biological parents' family contexts. This can result in range restriction of measures (resulting in attenuated effects) of parent SR and the intergenerational transmission mechanisms noted in this review. Nevertheless, such studies will be critical. Genetic factors and children's rearing contexts *together* play key roles in the intergenerational transmission of SR, including passive and evocative rGE. Studies that employ genetically sensitive designs in human (e.g., adoption designs) and animal samples (e.g., cross fostering) are needed to move the field forward in terms of its understanding of the additive and interactive contributions of genetic and environmental factors in relation to the intergenerational transmission of SR.

In a broader context, consideration of parent SR has implications for understanding relations between seemingly disparate outcomes. For example, Miller, Chen, and Parker (2011) proposed a model of bio-behavioral mechanisms that link childhood maltreatment and poverty to a biological cascade of responses that over time result in compromised immune function and proinflammatory physiological responses, ultimately contributing to chronic disease states (e.g., vascular diseases and autoimmune disorders). Miller et al. (2011) noted that maltreatment and poverty often co-occur, and that there may be common factors that underlie both. Our review points to parent SR and its connection with parenting (including abuse) and SES as one potential factor underlying these often co-occurring stressors (e.g., Crouch, Hanson, Saunders, Kilpatrick, & Resnick, 2000; Herrenkohl & Herrenkohl, 2007), potentially linking parent SR with children's chronic disease states, perhaps decades later, through stressful rearing contexts and through links with children's SR and their engagement in unhealthy behaviors. Even more broadly, our review provides a parsimonious account of why risks for a wide range of adverse outcomes accumulate within families and persist across generations. If support for these possibilities is garnered by future work, the transmission of SR across generations may explain, perhaps in large part, the transmission across generations of harsh reactive parenting practices, poorer relationship quality, IPV, and socioeconomic disadvantage that have been demonstrated in prior work (Conger, Belsky, & Capaldi, 2009; Doumas, Margolin, & John, 1994; Harper, Marcus, & Moore, 2003; Simons, Whitbeck, Conger, & Wu, 1991; Stith et al., 2000).

Although our conclusions are well supported by existing work, many developmental studies have not measured parent SR. A key example illustrates some of the implications that stem from this observation. That is, because nearly all forms of psychopathology can be characterized in part as reflecting one or more aspects of poor SR, and given that parent psychopathology has been of long-standing interest to the field, it may be that the effects of parent psychopathology on family processes and on children are accounted for in part by parent SR. Future studies should consider this possibility, and also consider if parent psychopathology accounts for variance in family processes after controlling for parent SR. Doing so would more unequivocally partition variance that can be attributed to parent psychopathology. Similarly, given evidence of the potent effects of parent SR on rearing contexts and child outcomes, we recommend that studies routinely assess the most relevant aspects of parent SR for the outcomes of interest. In addition, more studies examining children's SR need to consider neurobiological mechanisms (e.g., HPA axis regulation) that can help explain the links between parent and child SR (see Blair et al., 2011 for a partial empirical example).

Next, although the literature indicates substantial gender similarity in the effects of mother's *and* father's SR on families and children, most work has only considersed mothers. Consequently, the effects of paternal SR on children and families are areas in need of additional focus. This includes the father's role in prenatal programming. Recent rodent models have demonstrated adverse effects of paternal preconception stress on offspring HPA axis regulation via modified spermatogenesis (Niknazar et al., 2013) and epigenetic alterations of sperm microRNA (Rodgers, Morgan, Bronson, Revello, & Bale, 2013). Additional work has reported changes in DNA methlyation in offspring hippocampus and frontal lobes resulting from paternal preconception stress (e.g., Mychasiuk, Harker, Ilnytskyy, & Gibb, 2013)—findings similar to those reported in the maternal prenatal programming literature. This early work suggests that paternal preconception stress may affect offspring SR, but new animal and human studies need to replicate and extend these early findings.

Other methodological issues also need to be considered in future work. As we noted earlier, existing work has yet to consider how top-down and bottom-up SR might interact to affect children and their rearing contexts. Interactive effects of poor top-down and bottom-up impulsivity (i.e., low scores on measures of top-down SR, and high scores on measures of impulsivity) may have particularly potent adverse consequences for children and the contexts in which they are raised. More precise measurement of SR also will need to be carefully considered. For instance, we caution against the use of measures that assess parent or child SR that are ambiguous because they contain a mix of items pertaining to top-down and bottom-up SR processes. Moreover, most studies of impulsivity have relied on questionnaires, and future studies will need to employ behavioral tasks to rigorously replicate and extend existing findings based on surveys. Future studies also need to consider the different facets of impulsivity that have recently been described in the literature (see Sharma et al., 2014) when testing intergenerational transmission processes described in our review.

It also is important to emphasize that not all parents raising children in stressful conditions (e.g., high chaos, lower SES) possess inadequate SR. Variability in parental SR is wide even in high-stress contexts, and it may be that in such contexts parent SR serves as a critical moderator of the effects of stress on children's SR, perhaps through its effects on parenting—something to consider in future research (see Doan & Evans, 2011 for evidence in partial support). Furthermore, we have focused on chronic, moderate to high stress or acute events (e.g., abuse) that lead to the highest likelihood of the maladaptive processes we have described. However, the effect of a "normal" frequency and intensity of stress on SR is an area that has received little attention. Future work should focus on identifying the function of stress over the entire continuum, and whether that function is nonlinear with specific thresholds beyond which there are deleterious implications for children's SR.

Next, as we noted early in our review, there is little work regarding the intergenerational transmission of behavioral inhibition/fear. Only a handful of studies have considered relations between manifestations of parent and child behavioral inhibition/fear, and results are mixed (Arroyo et al., 2012; Coplan et al., 2008; Daniels & Plomin, 1985; Degnan et al., 2008; Gartstein et al., 2010; Kiel & Buss, 2011; Rickman & Davidson, 1994). Likewise, only two studies have considered parent behavioral inhibition/fear and parenting behavior (Desjardins, Zelenski, & Coplan, 2008; Kiel & Maack, 2012); however, it is notable that these studies reported relations that might be anticipated on the basis of the framework proposed in our review. Similarly, although there is evidence that those higher in behavioral inhibition/fear experience poorer relationship quality (Nelson et al., 2008; Tackett et al., 2013), some studies have not found this association (e.g., Kubzansky, Martin, & Buka, 2004). However, relative to the modest literature that considers behavioral inhibition/fear, a larger literature has considered relations between parent anxiety and parenting and relationship functioning (Caughlin, Huston, & Houts, 2000; Murray et al., 2012; Przeworski et al., 2011; van der Bruggen, Stams, & Bogels, 2008). Nevertheless, because only 33% to 50% of those higher in behavioral inhibition/fear manifest clinical levels of anxiety (Clauss & Blackford, 2012; Kagan & Snidman, 1999), and because those with anxiety also may have difficulties with top-down SR (Ansari & Derakshan, 2011; Cisler, Olatunji, Feldner, & Forsyth, 2010), we view such evidence as supportive but not conclusive of the potential effects of parental behavioral inhibition/fear on mechanisms of intergenerational transmission of this aspect of SR. Thus, future work should consider behavioral inhibition/fear, in addition to or separately from anxiety, to see if a consistent pattern emerges.

There are other parallels between our review and the literature on behavioral inhibition/fear. Behavioral inhibition/fear is moderately heritable (Emde et al., 1992; Smith et al., 2012), neural mechanisms of behavioral inhibition/fear are sensitive to stress (e.g., Cohen et al., 2013), and there is evidence from animal models that stressful experiences result in epigenetic modifications within the amygdala and hippocampus (see Champagne, 2013 for an overview). As such, in light of existing evidence, we speculate that many of the processes we have articulated in relation to top-down SR and impulsivity are relevant for understanding the intergenerational transmission of behavioral inhibition/fear. Going forward, much more empirical work is needed to test this supposition.

Finally, there are implications for intervention. In the short-term, targeting parent SR may improve parenting behavior and other aspects of children's rearing contexts, reducing stress experienced in the home and conferring benefits for children's SR.

Several interventions exist that may be employed for such purposes. For instance, mindfulness interventions appear to improve attention and other aspects of SR (e.g., Holzel et al., 2011; Tang et al., 2007). There also is evidence that executive functioning can be improved via systematic training (e.g., Holmes et al., 2010; Klingberg, 2010; Takeuchi et al., 2010). Other methods (e.g., dialectical behavior therapy; Linehan, Bohus, & Lynch, 2007; Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006) have been effective in reducing difficulties with emotional SR. However, in order to maximize benefits for children's SR, interventions targeting parent SR will likely need to be combined with those targeting family dynamics (e.g., the Triple P parenting program; Nowak & Heinrichs, 2008; Sanders, 2012).

In the long-term, disrupting the intergenerational transmission of poor SR and promoting adaptive SR across generations is critical to improving human capital and health. Programs that strengthen children's SR may result in lasting changes and subsequent enhancement of regulation-related outcomes (e.g., educational, behavioral, emotional, economic, and health outcomes, as well as future family dynamics) at the same time as potentially promoting the transmission of adaptive SR to subsequent generations. Programs reaching large numbers of children, perhaps implemented in educational settings, may be particularly potent (Blair & Diamond, 2008). Although evidence of real world efficacy is not yet available, the Canadian Self-Regulation Initiative (2014) that started in 2012 in British Columbia may be the type of wide-spread effort that could lead to lasting improvements in children's SR and affect SR in the next generation, if implemented with sufficient intensity and duration. Indeed, given evidence for key developmental periods for SR in early childhood as well as in adolescence, taking the long view and employing wide-spread prevention and early intervention efforts early and consistently may yield the best outcomes within and across generations. This approach is the most likely to produce improvements in health, academic, and social well being, and contribute to reductions in the significant costs to society that stem from poor SR.

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