The Development of Self-Regulation During Adolescence: Understanding the Effects by Pubertal Changes and Parenting

by

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Abstract

Self-regulation (SR) has been suggested to be a key developmental precursor to a host of developmental outcomes (e.g., externalizing and internalizing behaviors, academic achievement, task completion, etc.; Baumeister, Leith, Maraven, & Bratslavsky, 1998; Eisenberg et al., 2004; Silk et al., 2006). Steinberg (2004) suggests that during adolescence, there are substantial changes in SR capacity, as older teens make better choices by engaging in risk perception and risk appraisal and handle risky situations more appropriately than younger teens. This provides indirect evidence of developmental changes in SR during adolescence, and yet, no studies have directly tested whether this is empirically substantiated.

Dahl (2004) questioned, whether underlying neural systems can explain whether there is an analogous natural window of plasticity for learning SR in adolescence. The continued development of the adolescent brain might account for variability in SR; coupled with positive socialization experiences (e.g., high quality parent-adolescent relationships). Thus, the current proposed study sought to connect maturation and parenting and examined developmental changes in SR during adolescence.

Secondary data analyses were conducted on the NICHD Study of Early Child Care and Youth Development (SECCYD) study which is a longitudinal study using four phases of data; two of which were used in the current study(Phase III=2000-2004, through 6th grade, n = 1,061, and Phase IV=2005-2007, through 9th grade, n = 1,009).

Survey methods were utilized with multiple informants (i.e., mother, father/partner/other adult).

Conditional and Parallel Process Latent Growth Models were employed to assess whether SR continues to develop in adolescence, pubertal development is associated with SR development, and that parenting processes are associated with SR development. Findings indicated that, largely, SR does not continue to develop during adolescence (9.5-15.5 years) and that development of SR is not influence by parenting processes. There were some interesting findings regarding pubertal development in males where changes in puberty were significantly associated with changes in SR. By examining both biological markers of maturation (puberty) as well as parenting processes, the current study will advance scientific knowledge in a comprehensive and rigorous way, and will extend SR work from childhood into the adolescent period.

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Table of Contents

List of Tables

List of Figures

Introduction

The Conceptualization and Developmental Nature of Self-Regulation

Self-regulation (SR) is a complex construct that has been conceptualized and interpreted in different ways (e.g., Brody & Ge, 2001; Dishion & Stormshak, 2007; Eisenberg, Smith, Sadovsky, & Spinrad, 2004; Kopp, 1982; Moilanen, 2007; Purdie, Carroll, & Roche, 2004; Silk, Shaw, Forbes, Lane, & Kovacs, 2006; Silk, Shaw, Skuban, Oland, & Kovacs, 2006), even using different labels (e.g., effortful control – Eisenberg et al., 2004; Rothbart, 1989; emotion regulation – Silk et al., 2006; Silk et al., 2006; self-regulation - Brody & Ge, 2001; Colman, Hardy, Albert, Raffaelli, & Crockett, 2006; Dishion & Stormshak, 2007; Kopp, 1982; Moilanen, 2007; Purdie, Carroll, & Roche, 2004; and self-control – Finkenauer, Engles, & Baumeister, 2005; Jones, Cauffman, & Piquero, 2007; Vazsonyi & Huang, 2010). There exists consensus that SR is a construct that consists of multiple, inter-related elements, namely cognitive, emotional, and behavioral ones. There is also consensus that SR is a key developmental antecedent to a host of child and adolescent skills and/or outcomes (e.g., externalizing and internalizing behaviors, academic achievement, task completion, and goal setting and achieving Baumeister, Leith, Maraven, & Bratslavsky, 1998; Eisenberg et al., 2003; Eisenberg et al., 2004; Silk et al., 2006). Though scholarship has closely examined the development of SR during infancy (e.g., Calkins & Hill, 2007; Velling, Blandon, & Kolak, 2006), early childhood (e.g., Feldman, Masalha, & Alony, 2006; Karreman, van Tuijl, van Aken, & Dekovic, 2006) and childhood (e.g., Colman et al., 2006; Martel et al., 2007) over the course of the past two decades, it has not adequately considered potential developmental changes during adolescence. This is so salient because adolescents clearly change in their

ability to modulate cognition, emotions, and behaviors (e.g., Dishion & Stormshak, 2007; Moilanen, 2007; Moilanen, Rasmussen, & Padilla-Walker, 2014). One of the earliest useful *developmental* treatments of SR by Kopp (1982) captures the essence of "multi-dimensional" SR, namely the "ability to comply with a request, to initiate and cease activities according to situational demands, to modulate the intensity, frequency, and duration of verbal and motor acts in social and educational settings, to postpone acting upon a desired object or goal, and to generate socially approved behavior in the absence of external monitors" (p. 199). Extensions into adolescence by Moilanen (2007) have further refined it, again highlighting the interrelated nature of multiple SR elements: "the ability to flexibly activate, monitor, inhibit, persevere and/or adapt one's behavior, attention, emotions and cognitive strategies in response to direction from internal cues, environmental stimuli and feedback from others, in an attempt to attain personally-relevant goals" (p. 835). In fact, Moilanen argues that cognitive, emotional, and behavioral SR are interrelated and that these elements in effect "cooperate" when youth work to obtain desired goals; she also finds empirical support for this conceptualization based on cross-sectional data from youth. Other researchers have provided similar definitions for SR or social competence (Vaughn et al., 2009) young children. However, Moilanen's definition expands the definition into adolescence.

Dishion and Stormshak (2007) make the same point and suggest that self-regulation consists of multiple salient and overlapping dimensions, namely cognitive (e.g. attention control, goal-setting, self-monitoring), emotional (e.g., anger, desire, anxiety, and depression), and behavioral (e.g., inhibition and activation) factors. They also suggest that these in effect "work together" rather than independently to produce SR. A poignant

example by Moilanen (2007) shows adolescents that are faced with a situation in which friends are going out, but they have homework left to do, as emphasized by parents. The teen must then simultaneously regulate cognition, emotions, and behaviors to achieve their goal, namely to complete their homework, in order to be able to go out with friends. Thus, consistent with some of the most recent thinking and scholarship about SR, the current study on the potentially continued developmental changes of SR during the adolescent period both conceptualizes and measures SR as a "singular" construct, one that simultaneously considers cognitive, emotional, and behavioral elements. It is important to note that while the use of SR is used here, the literature reviewed will include other construct names such as self-control, effortful control, and emotion regulation to assure a more complete examination of the literature. Additionally, development or developmental changes refer to maturation or changes over time in the current study. Furthermore, the current study examines the development of SR using a social neuroscience perspective, one based on neuroscientific evidence (e.g., Ellis, Rothbart, & Posner, 2004; Masten, 2004; and Steinberg, 2004) where research considers the potential interplay between both "simple" pubertal/maturational processes in adolescence, manifested in brain development, as well as continued socialization pressures by parents on their adolescent children in the development of SR during the second decade of life.

Theoretical Considerations

In her seminal paper, Kopp (1982) proposed that SR is a construct that goes through distinct developmental changes from birth through early childhood, starting with neurophysiological modulation (birth to 2-3 months), which progresses to sensorimotor

modulation (3 to 9-12 months), to control (9-12 to 18+ months), to self-control (24+ months), and then self-regulation (24+ months). Kopp identifies caregiver's responsiveness as key during infancy to help the child master basic physical, emotional, and cognitive tasks. She also describes how as the infant matures, there is a shift from primarily external to internal sources of regulation. During this transition, the caregiver's role becomes more supportive rather than controlling. Thus, she highlights the key facilitating role by caregivers, as a process of interaction and exchange, during this period in the development of SR. In addition, Kopp suggests that social learning processes continue to affect the development of SR during early childhood, where parents and caregivers are models for the continued growth of SR. Kopp suggests that self-control emerges in the preschool years and self-regulation becomes refined later as children are better able to comply with other's requests and experience representational thought. However, Kopp suggests that self-regulation is slef-control, in kind rather than degree, as it involves a wider range of control mechanisms (Bronson, 2000). Though Kopp's discussion of SR development focused on infants and children, she highlighted the need for continued parental investment to facilitate SR development during adolescence. This idea has recently been reaffirmed by Eisenberg, Valiente, Morris, Fabes, Cumberland, Reiser, Gershoff, Shepard, and Losoya (2003) who argue that although individual differences, such as temperament (e.g., Rothbart & Sheese, 2007; Rothbart, Posner, & Kieras, 2006), exist early in life in SR, continued experiences from the social milieu contribute to the ongoing development and differences of SR. They suggest that a child's regulatory abilities are embedded in the social context of relationships and that their SR can be learned from caregivers or parents. Eisenberg (2012) explains that individual

differences in SR are also influenced by heredity, the prenatal environment, and environmental influences in childhood. Thus, in addition to considering whether and how pubertal milestones and associated maturational processes play into the development of SR during adolescence, the current study seeks to test to what extent continued socialization pressures by parents account for variability in SR development during the second decade of life.

Why Study Self-Regulation during Adolescence?

SR appears to be a key mechanism that enables individuals to succeed in life (e.g. Baumeister et al., 1998). SR capacity has been linked to controlling one's moods, improved concentration and task persistence, lower risk for alcohol and drug use, improved goal setting and follow-through as well as social competence. Conversely, low self-regulatory capacity has been linked to self-defeating behaviors, addiction, violence, low academic and social competence, anxiety, and depression (Baumeister et al., 1998; Eisenberg et al., 2003; Eisenberg et al., 2004; Martel et al., 2007; Silk et al., 2006). However, most scholarship to date on the development of SR has been carried out on children, though it is clear that SR is associated with a number of positive measures of adjustment during adolescence and adulthood (Martel et al., 2007; Silk et al., 2006; Sooyen, Brody, & Murry, 2003), what remains unknown is to what extent SR continues to develop during adolescence.

Steinberg (2004) suggests that over the course of adolescence there are substantial changes, and what appear to be improvements, in SR capacity as older teens make markedly better choices by engaging in risk perception and risk appraisal and handle risky situations more appropriately than younger teens. Steinberg identifies one candidate

source for these apparent changes in SR, namely maturational changes (e.g., brain development, puberty). Recent insights from neurosciences provide evidence that the brain, and specific brain structures in the frontal lobe area known to be key for SR, continues to develop during the teenage years, once thought to be completed in childhood. In fact, based on brain imaging and electroencephalography studies (e.g., Giedd, 2004; Giedd, 2008; Giedd et al., 1999; Lenroot & Giedd, 2006; Luna & Sweeney, 2004; Sowell, Thompson, Homs, Jernigan, & Toga, 1999; Spessot et al., 2004; Whitford et al., 2007) which provide the evidence of profound structural, and thus, by implication, functional changes during adolescence, a number of scholars have identified maturation and associated brain development as a potential contributor to SR developmental changes during this period (e.g., Ellis et al., 2004; Masten, 2004; Steinberg, 2004).

Additionally, Steinberg (2008) recently suggested gonadal hormones (e.g., estrogen and testosterone) are directly linked to the changes in the brain (i.e., brain maturation) that occur at the onset of puberty. These changes are suggested (Steinberg, 2008) to influence behavioral manifestations that are linked conceptually to the structural and functional changes in the brain over the course of adolescence. Bridging the two fields of behavioral science and neuroscience to study adolescent behavior has led to a new perspective, labeled a social neuroscience perspective (Steinberg, 2008). This perspective guides the current study which is interested in examining whether brain maturation and parenting influence the continued development of self-regulation during adolescence. Brain maturation was studied by using a proxy, namely puberty, as it has been conceptually linked to the structural and functional changes in the brain during the adolescent period. While the recent trend of bridging neuroscience and behavioral science

has provided indirect evidence of profound developmental changes in SR during adolescence, no to few studies to date have directly tested whether this is empirically substantiated. Therefore, the current study's aim is to examine whether developmental changes in SR occur during adolescence, and whether these changes are influenced by parental socialization and/or by maturational changes related to the onset of puberty. More specifically, the study examines whether parenting (e.g., support and monitoring) and maturation (e.g., pubertal development) contribute uniquely or additively to the continued developmental changes in SR during adolescence.

Literature Review

Eisenberg and colleagues (2004) noted that "regulated individuals should be able to respond in a spontaneous manner in contexts in which such reactions are acceptable and also able to rein in their approach or avoidant tendencies, when appropriate" (p. 261). For example, when a person experiences negative emotion they must regulate themselves so that they may respond in appropriate ways. There are many ways that one achieves this. Often, individuals distract themselves and shift their attention to something else, cutoff the input from the stimulus, mask the expression of the emotion, or inhibit impulses of aggression (Eisenberg et al., 2004). Eisenberg et al. (2004) explain that regulation is present early in life through infants' responses to stimuli and orientation responses which develop in the anterior portion of the brain. Furthermore, the authors suggest that regulation is stable over time. In other words, levels of regulation at age 2 will be correlated with levels of regulation at 22. However, this chapter notes that there are marked increases of regulation in preschool years and may continue to develop even

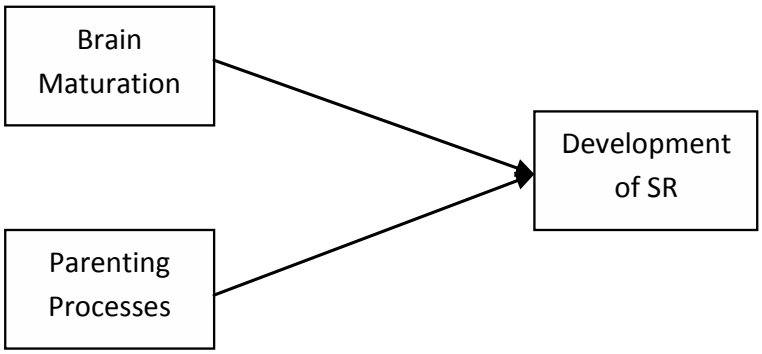
into adulthood. While Eisenberg and colleagues suggest that regulation is a stable trait, they do discuss the importance of socialization in regulation. For instance, the quality of parenting may contribute to the development of regulation where supportive behaviors (e.g., parent support and warmth) and supervision behaviors (e.g., helping child in social situations and positive discipline) are related to higher levels of regulation.

While SR is a relatively stable construct, there are variations between people regarding their levels of SR. Some individuals are simply better at regulating themselves than others. These variations in regulation are seen as early as preschool, something substantiated in a longitudinal study by Shoda, Mischel, and Peake (1990). In this study, the authors were interested in the relationship of delayed gratification in the preschool years (4-5 years) and later self-regulatory competencies in adolescence. Specifically, the authors conducted a series of experiments where the preschooler chose a preferable item over a nonpreferable item (e.g., marshmallow-two versus one, pretzels-two versus one). The experimenter then placed the nonpreferable item in front of the child with a bell and told them that they had to leave the room. The experimenter told the child that he/she could have the preferable item if they waited for the experimenter to return or they could ring the bell and have the non-preferable item. The authors found that over a decade later, when the children were an average age of 15, children with the best capacity for self-regulation during the experiment which occurred in preschool (or best at self-imposed delay of gratification) were the most successful young adults in terms of school performance, college readiness, social competence, and coping with stress (range of effect sizes -.30 - .58).

While childhood is a key developmental period in the understanding of self-regulation, new research in brain development among adolescents suggests that the brain continues to develop during the second decade of life. Dahl (2004) posed the question, regarding puberty and adolescence, whether underlying neural systems can explain whether there is an analogous natural window of plasticity for learning SR in adolescence. More recently, Dahl and Hariri (2005) suggest that the idea of hormones (e.g., "raging" hormones during adolescence due to puberty) affecting emotion and behavior are important during adolescence, but more current investigations need to examine how these pubertal hormones influence continued brain development and how these increases influence self-regulatory capacities relevant for adolescent development. Luna and Sweeney (2004) also point out that much work on environmental or contextual determinants of adolescent social behavior has been completed; however, little work exists on biological influences on cognition that are known to affect social behaviors during this developmental period. The continued development of the adolescent brain might account for variability in SR; coupled with positive socialization experiences (e.g., high quality parent-adolescent relationships) which might achieve better SR during the adolescent period (Dahl, 2004). Steinberg (2008) recently called for researchers that are interested in the physical and psychological wellbeing of adolescents to examine how contextual factors (e.g., parenting behaviors) and neural underpinnings of these processes influence the development of SR in adolescence (see Figure 1). Of course, socialization effects on SR by parents or caregivers have been well established, particularly among children, but not among adolescents. Thus, the current proposed study seeks to connect

maturation and parenting and to examine developmental changes in SR during adolescence, providing a novel approach in studying these key constructs.

Figure 1: Conceptual Model of Study Variables of Interest



Maturation and Self-Regulation

Although past research has suggested that brain development occurs during early childhood (e.g., Thompson, 2001), recent advances in brain research has shown continued brain development in the frontal lobe (i.e., the anterior portion of the brain and is known as the emotion control center and provides motor function, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, and social and sexual behavior) during adolescence and possibly into early adulthood (e.g., Giedd, 2004; Giedd, 2008; Giedd et al., 1999; Lenroot & Giedd, 2006; Luna & Sweeney, 2004; Sowell, Thompson, Homs, Jernigan, & Toga, 1999; Spessot et al., 2004; Whitford et al., 2007). Specifically, using magnetic resonance imaging (MRI), Whitford et al. (2007) found that gray matter (neural tissue in the brain that contains the nerve cell body and the

nerve fibers) in the adolescent brain decreases, while white matter (neural tissue in the brain that contains myelinated nerve fibers and underlies gray matter) in the frontal lobe increases in a sample of 138 healthy participants, ages 10-30 years (70 = females, 68 = males). Each participant had an MRI scan of their brain, and each scan was transformed into a standardized version where brain matter was compared. Changes in the relative amount of white matter were associated with improvement in language and memory skills (Whitford et al., 2007). Additionally, Giedd (2008) has studied maturational changes in the brain from childhood into adolescence using MRI scans as an ongoing, longitudinal project at the National Institute of Mental Health (NIMH Child Psychiatry Branch Longitudinal Brain Imaging Project). This project was started in 1989 and continues to follow participants. Giedd and colleagues (1999) examined MRI scans of N = 145 (89 males and 56 females) healthy children and youth, part of a larger project, that ranged in age from 4.2–21.6 years. This study compared the maturation of the brain, specifically how gray and white matter changed, during this developmental period; they found that white matter followed a linear growth pattern, while gray matter followed an inverted U shaped pattern (or nonlinear pattern). There was a net increase in white matter of 12.4% which did not differ significantly different by lobe. The gray matter, however, was regionally specific showing an increase in frontal lobe growth during preadolescence, with maximum size occurring at an average of 12.1 years for males and 11.0 years for females. Later, a decline in gray matter was observed in the post-adolescent developmental period. Interestingly, the article suggested that this developmental period and the changes in the brain could be important as they may suggest synapse eliminations, and thus, important synaptic pruning. Furthermore, it was

noted that early frontal peaks in brain development for females (one year earlier for females) may be related to pubertal onset as females typically enter puberty a year or more earlier than males. In turn, this suggests that brain development during adolescence might be "sparked" by gonadal hormones. More recently, the project completed over 5,000 brain scans based on over 2,000 participants (as of September, 2007; Giedd, 2008). At each assessment point, which is once every two years, participants undergo a structural MRI scan and answer questions related to behavior and mental health. Giedd (2008) found that by the time a child reaches age 6, their total cerebral volume reaches 95% of its peak size. However, during the adolescent period, synaptic pruning (neurological process where weak neurons are reduced to increase the efficiency of other synaptic configurations) and continued fine tuning of myelination (the wrapping of oligodencrocytes around axons which acts as an insulator and increases the efficiency of neuron signal transmission) occur in the frontal lobe which is the probable cause for continued brain development in the frontal lobe region. Kelley, Schochet, and Landry (2004) also suggest that the prefrontal cortex is last to undergo "fine tuning" of myelination and synaptic pruning and thus may be a cause for the fact that adolescents engage in risky behaviors during this time. Their brains have not matured in this area which is responsible for controlling executive functioning, impulse control, and decision-making. Consistent with this idea, Sowell et al. (1999) found that decreases in gray matter in the frontal lobe during adolescence were related to synaptic pruning occurring at this developmental period. Their sample consisted of normal young adults ages 23-30 years of age (n = 10, 5 male) and normal adolescents ages 12-16 years of age (n = 10, 5 male) that had undergone MRI scans of the brain to assess brain growth (both spatially

and temporally) during this period. They suggest that these findings, that the brain is continuing to develop during adolescence, are associated with known increases in cognitive development (e.g., emotion regulation) during adolescence.

To clarify, Luna and Sweeney (2004) have recently suggested that during adolescence, the process of synaptic pruning is associated with improved efficiency of local computations, and thus enhances the ability of specific brain regions to support high-level cognitive control of behavior. Additionally, they suggest that increased myelination speeds the neuronal transmission that in turn supports the collaboration of circuitry; the end result is that different regions of the brain are more effectively integrated and thus also support cognitive control of behavior as well. Based on neuroscientific evidence, Luna and Sweeney identify adolescence as a "critical period" where marked changes in brain performance are likely to be associated with qualitatively different abilities that manifest themselves in improved behavioral control (and thus, cognitive, emotional, and behavioral SR). Furthermore, Kelley et al. (2004) have noted that prefrontal cortex (PFC) functions interact with a number of other areas in the brain that are known contributors to attention, decision making, inhibition, and SR. The importance of this is that the prefrontal cortex in effect matures and improves capacity and functions as a potential coordinator and relay station of multiple brain systems that manifest themselves in improved SR over time, particularly during the adolescent developmental period.

In an attempt to understand the relationships between these variables Carlos, Crockett, Wolff, and Beal (2012) examined the effects of emotional reactivity, self-regulation, and puberty on prosocial behaviors concurrently and longitudinally. They

explained that adolescents who were low on emotional reactivity and high on self-regulation should have higher ratings of prosocial behaviors. Using hierarchical regression analyses they tested these variables independently and by using the interaction of the variables on prosocial behaviors. Results indicated there were significant effects of emotional reactivity and self-regulation on prosocial behaviors. Interestingly, they found that early maturers displayed higher levels of prosocial behaviors at home. The authors suggested that this result is due to puberty facilitating higher levels of prosocial behaviors by adolescents' emotional.. While this explanation is a plausible one, perhaps in light of recent brain development studies the hormones that are involved in the pubertal process are also increasing brain maturation in the frontal lobe which, in turn, increases the levels of self-regulation in adolescents. Those adolescents who are more regulated possibly rated higher on the prosocial behavior measure.

Recently Steinberg and colleagues (Albert, Chein, & Steinberg, 2013; Shulman, Harden, Chein, & Steinberg, 2014; & Steinberg, 2014) have presented a dual systems model for understanding self-regulation in adolescents in regards to risky behaviors. His recent research shows that pubertal hormones (i.e., testosterone and estrogen) trigger reward seeking/sensation seeking behaviors through the ventral striatum and orbitofrontal cortex. In some adolescents, this process of reward seeking behaviors manifests itself by engaging in risky behaviors which often occurs in the peer context. The peer social environment often rewards this sensation seeking behavior that is heightened during this time. However, self-regulation, the second component of the dual system, continues to mature during adolescence as a function of brain maturational changes, creating developmental period where we find a large gap between what amounts to a sensation

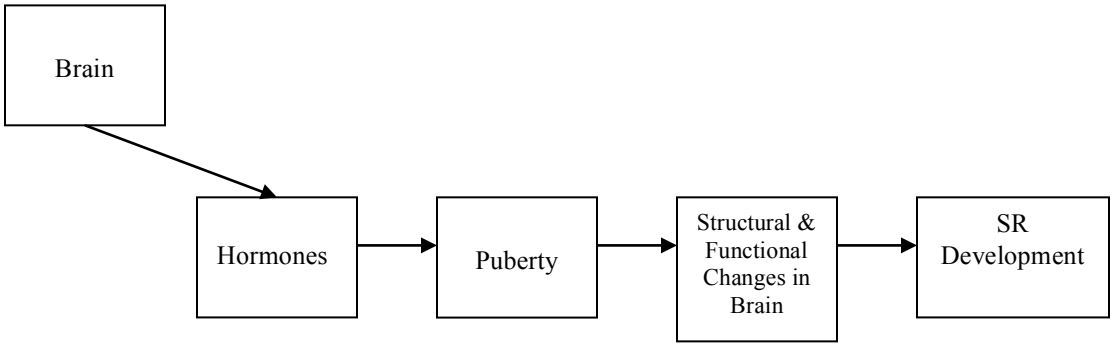
seeking drive and the tension with self regulatory skills . This period marks the time according to Steinberg and colleagues which places youth at heightened risk for making bad choices and for engaging in risky behaviors. Therefore, it is an important next step to develop our understanding of the continued development of self-regulation in adolescence as it changes as a function of brain maturation, measured for instance by puberty as a proxy.

Puberty as a Proxy for Brain Maturation

These brain developmental changes have been associated with increases in reproductive hormones (e.g., estrogen, progesterone, and testosterone) that are generally associated with and mark puberty (Cameron, 2004a; Cameron, 2004b). For example, Giedd et al. (2006) have found that in clinical populations (as part of the sample in the NIMH Child Psychiatry Branch Longitudinal Brain Imaging Project) where participants have hormone abnormalities, the normal maturational processes of the brain are altered. Additionally, Peper and Dahl (2013) explain that sex hormone surges during puberty actually create two effects in adolescent brain development. Specifically, organizational and activational effects. Organizational effects cause permanent change in the brain structure while activational effects create temporary changes in the neural systems that strengthen mating behaviors. Steinberg (2008) suggests that gonadal hormones (e.g., estrogen and testosterone) have a direct link to the changes in synaptic organization that occurs at the onset of puberty via oxytocin, one of the key neurotransmitters in the brain. Furthermore, he suggests that behavioral manifestations stemming from pubertal onset are logically linked to structural and functional changes in the brain. While puberty is not responsible for all maturational processes of the brain, there are some changes linked to

pubertal timing that lead to changes in brain maturation (Dahl, 2004; Dahl & Hariri, 2005, Peper & Dahl, 2013). More specifically, there are maturational brain changes that occur simply from the cascade of hormones that begin in the brain as this is where puberty first begins. Additionally, there are maturational changes in the brain that occur as a *consequence* of puberty (downstream changes). This occurs as pubertal hormones move through the blood-brain barrier, which bind to receptors in specific locations of the brain and stimulate changes (Dahl, 2004; Dahl & Hariri, 2005). Dahl and Hariri (2005) caution researchers that simply measuring the levels of a particular hormone via blood or saliva is insufficient as these levels only tap into one component of these maturational changes. Rather, it is the process of the hormone secretions along with their sequence and timing that are important in brain development during adolescence (see Figure 2).

Figure 2: Conceptual Model of Puberty as a Proxy for Brain Maturation



The importance of the interaction between puberty and brain development has also been recently addressed by Sisk and Foster (2004) who suggest that the brain-hormone interaction in adolescent brain growth are integral to behavioral maturation. In fact, when this interaction is altered in animal models (e.g., hamsters that have been castrated prior to puberty and steroid hormones introduced later), there are negative

consequences in brain development; specifically, altered brain development trajectories are connected to negative consequences in adult behavior of the animals. Therefore, it seems that there is a possible sensitive period during which brain growth, during adolescence, and puberty need to co-occur (and in a normal, healthy samples they seem to do so). Peper and Dahl (2013) suggest that future researchers on brain development foucs on puberty-specific effects in the adolescent developmental process. They note that in order to accomplish this task researchers must examine puberty longitudinally and using appropriate pubertal measures. Based on this interaction between the growth of the brain and pubertal onset, puberty is used as a proxy of brain development in the current study.

Sex Differences in Brain Maturation

Related to puberty, sex differences have been found in brain maturation during the adolescent period (e.g., Giedd, 2004; Giedd et al., 1999; Silveri et al., 2006), where frontal gray matter peaks a year earlier in females than in males. However, findings have also shown that the total brain volume is higher in males than females. Additionally, in a sample of 21 healthy adolescents (12 females and 9 males), Silveri et al. (2006) found in white matter microstructure using MRI scans of gray and white matter, where adolescent males had higher fractional anisotropy (FA) values (a measure of white matter coherence) in the white matter regions relative to females. These findings during the adolescent period may underlie some of the sex differences commonly found in research on emotional and cognitive changes. As mentioned previously (Sisk & Foster, 2004), these sex differences in brain development may be directly linked with sex differences in pubertal onset. Recently, Shulman, Harden, Chein, and Steinberg (2014) found that self-

regulatory or impulse control continues to increase in both male and female participants into their 20s with modest sex differences in the growth of impulse control and the rate in which this occurs in male and female youth. Specifically, females had higher levels of impulse control and significantly more rapid rates of growth than males. Based on these findings, it was necessary to examine whether any sex differences exist in the developmental changes examined in the current study.

Although neuroscience, puberty, and associated brain development changes in SR development are connected conceptually, to date no social scientific studies have effectively linked these areas. Cameron (2004) suggests that changes in hormones during puberty potentially modulate structure and function of neurons in the brain that are involved in regulation. She suggests that integrating these findings to study the effects of brain development, caused by pubertal hormones during adolescence, is an area that is ripe for investigation. Therefore, in an effort to approach this connection, Steinberg (2004) has suggested that numerous self-regulatory capacities (impulse control, planning, and foresight) continue to mature during early adolescence, and possibly even early adulthood. Steinberg has hypothesized that because of continued brain development, adolescents engage in fewer risky or problem behaviors over time as they traverse the second decade of life, largely because they are able to recognize the consequences to "negative" behaviors as they mature (Steinberg, 2004; Steinberg, 2008). Cameron (2004) has also pointed out that while research exists on how hormones affect the brain in adults (namely profound influences in regard to learning, memory, and regulation of emotion and behavior), there have been virtually none before or immediately after puberty during the adolescent developmental period. Dahl (2004) has also recently called for scientists to

build stronger bridges across disciplines to increase the knowledge base regarding the changes during adolescence. He also suggests that knowledge from such studies has a very high likelihood of informing numerous problems that affect youth. Thus, the current study examined these very changes in self-regulation as a function of adolescent maturation (e.g., pubertal markers) and of socialization efforts by parents and caregivers (e.g., parenting processes).

Parenting and Self-Regulation

Beyond maturation and individual differences (temperament), quality or parenting processes have been identified as important predictors in the development of SR. To date, most of this work has been completed on young children (e.g., Eisenberg et al., 2003; Grolnick & Farkas, 2002; Raikes, Robinson, Bradley, Raikes, & Ayoub, 2007; Silverman & Ragisa, 1992). Studies that have examined the link between parenting and SR suggests that supportive/nurturing parenting that includes an appropriate level of control (monitoring or behavioral control rather than psychological control) serves as a model to children on how to respond appropriately to people and situations. This modeling and learning by youth promotes and instills SR capacity (e.g., Brody & Ge, 2001; Gray & Steinberg, 1999; Moilanen, Rasmussen, & Padilla-Walker, 2014; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Steinberg, 2004; Steinberg, 2008). Morris et al. (2007) recently suggested that developmental sciences have established how parenting strategies influence SR; however, there are few studies that actually examine this relationship in adolescent samples and fewer that test this relationship longitudinally to ascertain whether there exist developmental changes in SR over time during the adolescent period. Steinberg (2008) suggests that youth from homes where parents are authoritative (warm

but firm) display higher levels of SR and engage in fewer risky behaviors. However, he goes further to explain that it is not clear whether these SR capacities are higher because of supportive parenting, which would provide modeling of appropriate responses to stimuli, or because of consistent discipline or parental monitoring, a more external constraint that provides appropriate limits on behaviors. More recently, Moilanen, Rasmussen, and Padilla-Walker (2014) studied bidirectional relationships between adolescents and their parents on measures of parenting (e.g., authoritative, authoritarian, and permissive) and adolescents' SR longitudinally. They found that there were no rank order changes in authoritative and permissive parenting on SR over time, meaning that the relative position of individuals on perceived parenting and SR continua do not appear to change during the developmental stage that was studied. They found that authoritarian parenting was negatively associated with SR over time. Specifically, authoritarian parenting was related to decreases in SR over time. While the authors did not find any associations between authoritative or permissive parenting on SR changes, longitudinally, they call for additional longitudinal research that examines specific dimensions of parenting (i.e., parental warmth and monitoring) as these parenting dimensions have been found to predict adolescent self-regulation. They suggest that new research use data with three or more time points to show if any developmental patterns exist. Therefore, the current study examines both the affective quality of the parent-adolescent relationship, namely supportive parenting, as well as the amount of discipline parents use, namely parental monitoring and their importance in the development of SR during adolescence in three waves of data.

Currently, few studies have examined whether parenting processes continue to be associated with and account for variability in the developmental course of SR during adolescence. Although most scholarship on the parenting-SR link is based on young children, these do provide important insights into how specific parenting processes may be related to the development of SR during adolescence. Thus, an important question is whether key parenting efforts during adolescence continue to affect the development of SR at a time when teens are striving for and are able to achieve greater independence from parents. Do ongoing parenting efforts during the teenage years uniquely foretell *the ongoing changes and development of SR*? In the following section, some key scholarship is reviewed, largely based on samples of children, on how parenting efforts account for variability in SR or for developmental changes in SR.

Parenting and Self-Regulation in Samples of Children

Parenting processes have consistently been found to be associated with higher levels of SR based on samples of children (e.g., Colman, Hardy, Albert, Raffaelli, & Crockett, 2006; Dennis, 2006; Eisenberg et al, 2003; Karreman, van Tuijl, van Aken, & Dekovic, 2006; Keller et al., 2004; Kopp, 1982). Though Posner and Rothbart (2000) argue that individual, temperamental differences exist early in life, and thus place children on different developmental SR trajectories; further differentiation among children emerges as a function of experiences, of how socialization pressures from parents or caregivers in effect further "constrain" behaviors over time. ~~In a study by~~ Eisenberg and colleagues (2003) ~~they~~ investigated the effects of maternal positive expressivity (as measured by combining overall positive affect and support/warmth) in a sample of 214 children from a longitudinal study that first obtained measures of parenting

and socioemotional development at an average age of 73 months (96 girls, 118 boys) and two years later in a follow-up study (n = 177 children; 80 girls, 97 boys). They found that maternal emotional expressivity was positively associated with children's adjustment (internalizing and externalizing behaviors) and social competence over a two year period, as mediated through SR. Specifically, using Structural Equation Modeling (SEM) they found that the path from maternal expressivity to regulation had a positive association while the path from regulation to externalizing and internalizing behaviors was negative. The path from regulation to social competence had a positive association. Furthermore, regulation significantly mediated the relationships between maternal positive expressivity and externalizing behaviors and social competence, while only marginally significant between maternal positive expressivity and internalizing behaviors.

Similarly, based on a sub-sample of 549 children from the National Longitudinal Study of Youth 1979 original sample, Colman et al. (2006) found that high levels of maternal warmth at Time 1 (ages 4-5) were associated with a higher SR capacity 4 years later at Time 2 (ages 8-9), even after controlling for initial levels of SR. Furthermore, based on a sample of 2,441 low-income toddlers (14-36 months) and their mothers that were part of an evaluation of Early Head Start, Raikes et al. (2007) found that high quality mother-child interactions (e.g., positive affective states), at 14 and 24 months, were associated with positive developmental changes or growth in SR by 36 months using growth modeling procedures. They also found that, over time, children's SR states changed over time and at final status. This variation in SR statuses was explained by the possible environmental effects of the mother-child interactions. They also found some

interesting gender differences in these changes, where girls had more modest increases than boys.

In an effort to examine for potential cultural differences in the parenting-SR link**,** Keller et al. (2004) found that toddlers from Cameroon (assessed at 3 months and then at 18-20 months), where more proximal parenting (warm, supportive interactions that fosters dependence) was found, developed SR earlier than children from Greece or Costa Rica, contexts characterized by more distal parenting (face to face interactions and object play that fosters independence). The total sample consisted of 116 families at first assessment (children were 3 months) and 90 families at the second one (at 18-20 months).

Thus, these two studies provide some preliminary evidence for potential gender differences (see also Kochanska, Murray, & Harlan, 2000 who found girls had greater SR capacity than boys) as well as contextual or cultural differences in the development of SR based on young children.

Finally, in an effort to provide some common language as well as consensus related to the developmental salience of parenting efforts for SR development**,** Karreman and colleagues (2006) completed a meta-analysis of 41 studies (3799 families average age of child = 38.35 months), largely based on preschool samples. They found that parental positive control was a significant predictor of SR across studies (effect size of weighted means are .08). Positive control was described as teaching, encouraging, and guiding children's behavior (e.g., support and monitoring). Based on the literature reviewed on children's SR capacities, it is evident that positive parenting contributes to SR development. Specifically, supportive parenting that is coupled with monitoring

strategies by parents predicts higher SR capacities and in one study (Raikes et al., 2007) showed linear growth in SR capacities over time.

More recently, Vazsonyi and Li (2010) examined whether parenting at 4.5 years predicted developmental changes over a 6 year timeframe using time points from 4.5, 8.5, and 10.5 years. The model they tested showed that self-control increased over time at a rate of .05 per year. Furthermore, parenting was statistically significant in predicting the intercept but not the slope of self-control. While this literature is on children rather than adolescents, it provides a useful model on how parenting may contribute to SR during adolescence.

Parenting and Self-Regulation in Samples of Adolescents

In examining the parenting-SR link in literature based on adolescents, a number of investigations have tested SR as a potential mediator between parenting processes and adjustment measures (e.g., externalizing or internalizing behaviors, academic achievement; Brody & Ge, 2001; Purdie et al., 2004; Sooyeon & Brody, 2005). These studies that take a variable centered approach, have not examined how and whether key parenting processes are important in the developmental changes of SR during adolescence. Furthermore, no previous work has explicitly tested how and whether brain development and the development of SR, or at least, whether biological markers for brain changes, such as pubertal changes, together with parenting processes explain SR development. Despite this dearth of scholarship in this area, a number of scholars have started to write about these links (Dahl, 2004; Luna & Sweeney, 2004; Steinberg, 2004; Steinberg, 2008). However, a number of studies appear to have provided successively more relevant information that can inform a study of these proposed links. For example,

Purdie and colleagues (2004**)** examined the relationship between authoritative parenting and academic and non-academic self-regulation in a sample of 14 to 16 year old youth (N = 214 families, 60% males, average age = 14.8) using mother, father, and youth responses to surveys. Results provided evidence of a strong link (path coefficient of .70) between high parental involvement (e.g., described as loving, accepting, and responsive; all indicative of parental support) and the development of academic (e..g, SR capacity related to academic pursuits) as well as non-academic (e.g., SR capacity to other pursuits not academic) self-regulation. Parental involvement, however, was only assessed as a mediator between parental self-efficacy and academic and non-academic SR. Similarly, Brody and Ge (2001) investigated the relationships between nurturant-responsive parenting behaviors (e.g., support, positive discipline, and monitoring) and SR over a two year period (Wave 1 at 12 years of age, Wave 2 occurred one year later) and how SR was associated with subsequent psychological adjustment (depressive symptoms, hostility, self-esteem) and alcohol (conducted at Wave 3/year 3). The sample consisted of 120 families (mother, father, and child; males = 62, females = 58) from the Southeast United States. Based, again, on a variable centered strategy using SEM, they found that SR was positively influenced by high levels of nurturant-responsive parenting (Wave 1, r=.54; Wave 2, r=.36) over a 2 year period between Wave 1 and Wave 2. This study found evidence that both mother's and father's parenting as well as the child's SR were stable over time, that SR was associated with mother and father nurturant-responsive parenting at Wave 2 which was associated with the child's SR at Wave 2, and that the child's SR was associated with later (Wave 3) adolescent adjustment. While this is article is informative of the relationships between parenting and self-regulation it is limited to two

timepoints. Based on a cross-sectional investigation of 1,359 of Dutch youth (males = 709, females = 650) between the ages of 10 and 14 years (average age = 12.3 years), Finkenauer, Engels, and Baumeister (2005) found that self-control, defined as regulating ones behaviors, thoughts, and emotions, was positively associated (Beta = .12) with parental acceptance (or support). Self-control partially mediated the effects by parenting on adolescents' externalizing and internalizing behaviors. Therefore, the authors suggest that there is a positive association between parenting and self-control and that self-control partially mediates the effects of parenting on adolescent's externalizing and internalizing behaviors. Furthermore, Kim and Brody (2005) studied the effects of family risk and maternal psychological functioning on parenting and externalizing/ internalizing behaviors as mediated by SR in African American early adolescents using a 5-wave longitudinal SEM model. The adolescents were 11 at the time of recruitment and were followed for four years. The authors found that the latent construct of parenting (e.g., competence promoting which included 3 manifest variables which were parental support, monitoring, and ineffective arguing) was associated with self-regulation, one year later (r = .27). This finding guides the assumption that parenting is related to increases in SR over time, although it was not tested in a growth modeling approach. In fact, Kim and Brody (2005) state that "involved and supportive parenting would be linked with rural African American youth's development of greater self-regulation" (p. 307). While this study only focused on African American youth, it guides research on the potential for supportive parenting to predict increases in the development of SR over time among youth. Finally, Buckner, Mezzacappa, and Beardslee (2003) studied characteristics of resilient youth and the role of SR processes in a sample of 155 low-income youth (45

were classified as resilient and 70 were classified as nonresilient). The sample included children ages 8-17 years of age and their mothers that were part of a larger longitudinal study. The authors were interested in identifying constructs that were predictive of resiliency and therefore used parental monitoring as one predictor. While not a focus of their study, they did find that parental monitoring was significantly and positively associated with SR ($r = .32$). While there is very little work on the effects of parental monitoring on the development of SR during adolescence and much more work on the effects of parental support (e.g., increases in support are associated with increases in SR), it is hypothesized that supportive parenting increases SR capacity and that controlling parenting decreases it during adolescence.

The common theme that emerges in studies about SR is that positive parenting predicts higher self-regulation, and appears to remain salient in later years. Parental support and monitoring appear to have emerged as some of the key parenting constructs in the development of SR, and thus the current study also examined those effects in the development of SR during early and middle adolescence. Though the reviewed studies are informative for the current project, it remains unknown to what extent specific parenting processes account for variability in the development of SR during adolescence. Past research also identifies parental support as a key contributor to the development of SR. Dahl (2004) has recently argued that support by parents appears to be a prerequisite for the development of adolescent self-regulation skills. Additionally, he suggests that there is a need for a supportive social context with an appropriate amount of monitoring and support from parents so these youth can develop self-regulation skills in a supportive and protective context. He describes the adolescent as one that is developing emerging

skills and knowledge for adult roles and, yet, has an immature neurobehavioral system to deal with the ebbs and flows in emotions caused by hormones during this time. In addition, Masten (2004) noted that when monitoring is prematurely withdrawn from adolescents this leaves the adolescent to navigate situations alone or with peers at an early age as they have not yet achieved cognitive and emotional maturity. Finally, it is important to assess paternal parenting processes on the development of SR. Current studies on SR primarily focus on maternal contributions and leave the question of whether and how fathers affect the development of SR in children and adolescents. Recently, Meuwissen and Carlson (2015) test the effects of fathers' parenting on preschool children's effortful control. They suggest the importance of including fathers in studies that focus on regulation due to cultural shifts in parenting where fathers are more active in daily parenting tasks and spend more time with their children. Additionally, it is important to assess the relationship of fathers' parenting processes on SR as they may have differential contributions to the development of SR than mothers (Moilanen, Rasmussen, & Padilla-Walker, 2014). Thus, it appears that now is the time for scholarship to move forward by integrating insights from developmental and socialization research about parenting effects (i.e., maternal and paternal) and to combine these with recent advances from neuroscience about how maturation changes brain structure, and thus functioning, to develop a greater understanding of whether and how SR develops during the second decade of life. Thus, the current study aims to contribute to the scholarship on SR by assessing the unique and/or additive effects by maturation (e.g., puberty) and parenting (e.g., support and monitoring) in the development of SR during adolescence. Previous research has identified certain variables (e.g., race/ethnicity,

SES/income, family structure, marital status, and immigration status) as covariates of parenting processes and SR, and thus, as potential confounds (e.g., Bornstein, 2003; Coleman et al., 2006; French & Pitchall-French, 1998; Maton et al., 1996; Murry & Brody, 1999; Taylor, Casten, Flickinger, 1993; Pinderhughes, Dodge, Bates, Pettit, & Zelli, 2000; Shaw, Winslow, & Flanagan, 1999). For instance, SES or income level, is widely known to be associated with parenting and, therefore, must be controlled when examining the relationships between other predictors on parenting to remove the shared variance from SES/income level. Additionally, the influence of adolescents' Body Mass Index (BMI) must be controlled for when examining puberty and BMI as it has been identified as a confound and influences the timing of pubertal indicators (e.g., Schubert, Chumlea, Kulin, Lee, Himes, & Sun, 2005). By examining both biological markers of maturation (puberty) as well as specific parenting processes, the current study advances scientific knowledge in a comprehensive manner, and extends developmental work on SR from childhood into the adolescent period.

Research Questions and Hypotheses

1. Does SR continue to develop in the adolescent developmental period?

    Hypothesis 1: It was expected that positive linear growth in SR would be found over the 6 year period.

2. Do pubertal developmental changes influence possible SR changes during adolescence?

    Hypothesis 2: It was hypothesized that pubertal developmental changes (slope) would predict developmental changes in SR.

3. Does parental support (maternal and paternal) influence possible changes in SR during adolescence?

    Hypothesis 3: It was expected that there would be positive growth in parental support over time and that this change would be predictive of developmental changes in SR over time.

4. Does parental monitoring (maternal and paternal) influence possible changes in SR during adolescence?

    Hypothesis 4: The current question is exploratory in nature due to inconsistencies in the literature whether parental monitoring increases or decreases over time during adolescence.

5. Do parenting and pubertal development contribute to changes in SR during adolescence independently (uniquely) or simultaneously (additive); if additively which of the two influences changes in SR to a greater extent?

Hypothesis 5: It was expected that there would be independent and thus additive effects by developmental changes in parenting (support and monitoring) and by pubertal development on SR development.

Method

Participants

The current study utilized the Eunice Kennedy Shriver National Institute of Child Health and Human Development's (NICHD) Study of Early Child Care and Youth Development (SECCYD) which is a longitudinal, long-term study that follows children and their development ranging from infancy in 1991 (one month of age) to adolescence (15 years of age) across four phases of data collection (The NICHD obtained data from 10 data collection sites in the United States (i.e., Arkansas, California, Kansas, Massachusetts, Pittsburgh, PA, Philadelphia, PA, Virginia, Washington, North Carolina, and Wisconsin) by screening 8,986 families in 24 hospitals (5,416 families were eligible). Phase I (1991-1994; ages one month-36 months) had 1,364 participants, Phase II (1995-1999; ages 54 months-7 years) had 1,226 participants, Phase III (2000-2004; ages 8-12 years) had 1,061 participants, and Phase IV (2005-2007; ages 14-15 years) had 1,009 participants. While this a normative, longitudinal sample it is not nationally representative. There was an exclusion criterion for the study that included questions such as maternal age, multiple births, and whether the family was presently in a study. Family demographics at one month of age were: average maternal education = 14.4 years; mothers reported being single = 14%; mothers did not complete high school = 10%, average family income = 3.6 times the poverty threshold (24% between 0-1); families are racial/ethnic minorities = 24%. The current study included children and adolescents from Phase III-Phase IV (ages 9.5-15.5 years).

The rationale for targeting this age band is that (1) neuroscience evidence of brain changes and (2) known indirect evidence about changes in SR during early and middle

adolescence through decreasing rates of problem behaviors; and (3) most profound maturational changes, including markers such as menarche for girls, occur between the ages of 10 and 16 (mean age: 12 years; NIH, 2007). Thus, by targeting these age groups, it is maximizing the opportunity to study youth when they are undergoing the most profound maturational changes, particularly related to brain development (e.g., Steinberg, 2004). Furthermore, longitudinal studies are most "ideal for tracking changes in complex behaviors that occur over adolescence and identifying which of these changes are specifically associated with pubertal development" (Cameron, 2004, p. 120). However, Cameron notes that these studies are extremely rare. Additionally, she suggests that using several markers of pubertal development will provide a more accurate picture of pubertal stage and provide insight on the pubertal development for youth when assessed longitudinally.

Procedures

The primary objective of the NICHD SECCYD is to investigate the relationships between differing child care arrangements and measures of children's health, behavior, school performance and development in young children through adolescence. The factors that the NICHD SECCYD collect information on include the child care and family context while also assessing children's development (e.g., social, emotional, cognitive, and language), adjustment, and physical health using multiple methods (i.e., observation, interviews, questionnaires, and testing) and using multiple informants (i.e., mother, child/adolescent, teacher, and clinicians).

Measures

Demographic variables. When children were one month of age mothers reported the child's race/ethnicity. Family structure was rated as having two parents, two-parent extended or extended and augmented family, two-parent augmented, step-father family, single parent nuclear family, single parent extended or extended and augmented family, single parent augmented family, nontraditional nuclear family, nontraditional step-father family, nontraditional extended or extended and augmented family, nontraditional augmented family, nontraditional augmented family, two-parent alternate caregiver family, and single-parent alternate caregiver family. Sex of the child was reported as being male or female. SES was was measured by using mother reports of whether the family received food stamps from the government (Vazsonyi & Li, 2010).

Parenting. Parental support/warmth (see Appendix 1, Table 1) was assessed using the Parent-Child Relationship Scale (Pianta, 1994). For Phase III and IV a shortened form Parent-Child Relationship Scale (Pianta, 1994) was used that included 15 of 30 items. The scale used in the current study was positive parenting; items were rated by both mothers and fathers on a 5-point Likert scale ranging from 1= definitely does not apply to 5 = definitely applies (e.g., "children spontaneously shares personal information with me") at child ages 9.5, 10.5, 11.5 and 15.5. The reliability estimates (see Table 1) for positive parenting (e.g., parental support) ranged from $\alpha = .82$ to $\alpha = .85$ for mother ratings and $\alpha = .84$ to $\alpha = .87$ for father ratings.

Parental monitoring (see Appendix 1, Table 1) was assessed using the "Keeping Tabs" form from Phase III and IV of the study. The questionnaire included 11-items regarding parental supervision and monitoring of the child's activities as based on work

by Statin and Kerr (2000). Fathers only rated monitoring while there child was 9.5, therefore, mother ratings were used. The measure includes items such as "If your child didn't come home by a set time, would you know" and "Who your child spends time with?" Responses were rated on a 4-point Likert scale which include 1 = doesn't know at all, 2 = knows a little bit, 3 = knows a lot, 4 = knows everything. Reliability estimates (see Table 1) ranged from α = .67 to α = .76 for ages 10.5, 11.5, and 15.5. Monitoring at 9.5 years was dropped due to low reliability (α = .47).

Table 1

Demographics for Parental Support variables (Maternal and Paternal)

Variable	No. of Items	*N*	*M*	*SD*	α
Maternal Support					
	9	1022	4.20	.51	.82
9.5 yrs					
10.5 yrs	9	984	4.15	.52	.83
11.5 yrs	9	981	4.09	.56	.85
15.5 yrs	9	936	3.88	.61	.85
Paternal Support					
	9	698	4.10	.55	.84
9.5 yrs					
10.5 yrs	9	620	4.02	.54	.84
11.5 yrs	9	592	4.01	.58	.87
15.5 yrs	9	674	3.79	.62	.87
Maternal Monitoring					
10.5 yrs	10	972	3.65	.19	.67
11.5 yrs	10	980	3.69	.28	.75
15.5 yrs	10	672	3.56	.31	.76

Puberty. Puberty (see Appendix 1, Table 2) was measured by a questionnaire developed by Peterson and colleagues (1988) to provide an assessment that did not

contain pictures of pubertal stages and, therefore, permitted for use in schools. It was developed as a self-assessment but has been used many times by parents. The measure contains questions about physical development that are rated by the degree of a particular physical change (e.g., breast development, facial hair, genital development). There are separate questionnaires for males and females.

Table 2

Demographics of Pubertal Development Scale (Males and Females)

Variable	No.of Items	*N*	*M*	*SD*	α
Pubertal Development, M					
9.5 yrs	5	505	2.40	.31	.44
10.5 yrs	5	456	2.48	.37	.56
11.5 yrs	5	455	2.62	.43	.56
12.5 yrs	5	462	2.89	.57	.73
13.5 yrs	5	438	3.19	.61	.72
14.5 yrs	5	380	3.49	.51	.72
15.5 yrs	5	264	3.60	.43	.63
Pubertal Development, F					
9.5 yrs	5	493	2.66	.48	.65
10.5 yrs	5	447	2.94	.57	.72
11.5 yrs	5	462	3.24	.59	.77
12.5 yrs	5	445	3.45	.53	.73
13.5 yrs	5	399	3.60	.44	.57
14.5 yrs	5	324	3.72	.43	.56
15.5 yrs	5	228	3.79	.46	.60

Body Mass Index. To obtain each adolescent's Body Mass Index or BMI, height and weight were used, where weight was divided by height, based on the method

suggested by the Centers for Disease Control (CDC). Therefore, height was measured by a nurse practitioner or pediatric endocrinologist as his/her height in inches or in centimeters. Weight was assessed by his/her weight in kilograms or in pounds. Scores were categorized in the following according to the CDC Method: *(1) 3rd percentile, (2) 5th percentile, (3) 10th percentile, (4) 25th percentile, (5) 50th percentile, (6) 75th percentile, (7) 85th percentile, (8) 90th percentile, (9) 95th percentile, (10) 97th percentile, (11) Below the 3rd percentile.* BMI is used as a control for pubertal development in the current study as it has been shown to effect puberty in males and females, such as, obesity being related to earlier onset of puberty in males and females (DeLeonibus et al., 2014).

Self-Regulation. The use of the term self-regulation (SR) was used for the current main study construct, and this term was used through the current study; however, the survey items are in fact a measurement of self-control. This measure has been used recently by Vazsonyi and Li (2010) to assess developmental changes in SR in children. Therefore, self-control, which was measured by the Social Skills Rating System (SSRS, Gresham & Elliott, 1990; see Appendix 1). Mothers and fathers completed the assessment related to their child's SR (see Table 3). The correlations between maternal and paternal SR ratings of their adolescent ranged from r = .39 to r = .56. The SSRS-Parent Form consists of three parts, namely social skills, problem behaviors, and academic competence scales. The social skill component includes three subscales, namely cooperation, assertion and self-control. The current study only focused on the self-control subscale. It included 10 items on a 3-point Likert-type scale (0 = never, 1 = sometimes, 2 = very often; e.g., "controls temper when arguing with other child"). The

alpha ranged from α = .82 to α = .87 for mother reports and α = .83 to α = .86 for father reports.

Table 3

Demographics for Self-Regulation (SR) Variables (Maternal and Paternal Rated)

Variable	# of Items	*N*	*M*	*SD*	α
SR (Maternal)					
Grade 4 (9.5 yrs)	10	1022	1.39	.35	.83
Grade 5 (10.5 yrs)	10	985	1.39	.33	.81
Grade 6 (11.5 yrs)	10	980	1.39	.34	.83
15.5 yrs	10	935	1.40	.35	.82
SR (Paternal)					
Grade 4 (9.5 yrs)	10	698	1.39	.35	.84
Grade 5 (10.5 yrs)	10	619	1.39	.34	.83
Grade 6 (11.5 yrs)	10	592	1.40	.36	.85
15.5 yrs	10	674	1.42	.36	.84

Plan of Analyses

The current study intended to examine the developmental changes (growth trajectories) of adolescent self-regulation between the ages of 9.5 and 15.5 years and to examine to what extent pubertal development and/or parenting processes account for these changes over a 6 year period. Initial analyses (see Tables 4-6) included descriptive statistics and frequencies for each study scale or construct to examine variable distributions (including skew) as well as reliabilities prior to model building. Skewness of the main study variables ranged from -.11 to 1.33, resulting in an almost normal distribution of scores. Therefore, all original variable distributions were used in subsequent analyses. Secondly, variance from demographic variables (e.g., race, SES, family structure, and BMI for pubertal development) was removed from each of the main study variables (e.g., pubertal development, parenting measures, and SR) by residualizing scores through regression analyses. When sex was used as a covariate or models were tested by sex, sex was not included in computing the residualized variables. The advantages of residualizing study variables include conceptual considerations, namely that adding them to the model would be largely exploratory and thus not consistent with study hypotheses, but also analytic considerations, where more statistical power is retained for focal hypothesis testing. In a third step, Latent Growth Models (LGM) using conditional LGM and Parallel Process LGM were employed (see Tables 7 and 8) to test a series of hypotheses, along with some exploratory follow-up questions related to sex differences and non-linear changes over time. LGM is a Structural Equation Modeling (SEM) analytic strategy that permits tests of both inter- and intra-individual changes in the main study constructs – puberty, parenting processes, and adolescent SR. Furthermore, LGM allows the assessment of

initial status (mean) of the study parameters and of the slope (change or growth) over time (Tisak & Meredith, 1990). Next, the study hypotheses along with analytic strategies will be discussed.

Table 4

Maternal Support, Maternal Monitoring, Puberty, and Maternal Rated Self-Regulation Correlations

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1. M Sup, 9.5	-																								
2. M Sup, 10.5	.75**	-																							
3. M Sup, 11.5	.69**	.71**	-																						
4. M Sup, 15.5	.52**	.56**	.58**	-																					
5. M Mon, 10.5	.24**	.24**	.23**	.15**	-																				
6. M Mon, 11.5	.24**	.27**	.28**	.16**	.37**	-																			
7. M Mon, 15.5	.19**	.19**	.17**	.26**	.36**	.49**	-																		
8. Puberty, M, 9.5	-.03	-.05	.02	-.02	-.08	-.02	-.02	-																	
9. Puberty, M, 10.5	-.01	.00	.05	.06	.00	-.01	.01	.66**	-																
10. Puberty, M, 11.5	.02	.06	.03	.07	-.12*	-.07	-.03	.55**	.65**	-															
11. Puberty, M, 12.5	.08	.07	.03	.05	-.10*	.00	-.02	.43**	.48**	.67**	-														
12. Puberty, M, 13.5	.08	.08	.06	.07	-.03	.03	.00	.37**	.38**	.54**	.74**	-													
13. Puberty, M, 14.5	.03	.04	-.02	.01	.01	.09	.01	.28**	.22**	.38**	.55**	.73**	-												
14. Puberty, M, 15.5	.03	.03	-.04	-.02	.03	.02	-.03	.28**	.18**	.36**	.48**	.62**	.74**	-											
15. Puberty, F, 9.5	-.06	-.07	-.04	.04	-.13**	-.04	.08	-	-	-	-	-	-	-	-										
16. Puberty, F, 10.5	- .12**	-.11*	- .14**	-.02	-.09	-.04	.01	-	-	-	-	-	-	-	.74**	-									
17. Puberty, F, 11.5	-.11*	-.07	-.10*	-.01	-.09	-.03	-.04	-	-	-	-	-	-	-	.61**	.78**	-								
18. Puberty, F, 12.5	-.06	-.05	-.09	-.05	-.05	.01	.02	-	-	-	-	-	-	-	.48**	.67**	.78**	-							
19. Puberty, F, 13.5	-.07	-.04	-.05	-.05	.02	.02	-.05	-	-	-	-	-	-	-	.43**	.54**	.65**	.69**	-						
20. Puberty, F, 14.5	-.14*	-.13*	-.12*	-.12*	.04	-.03	-.03	-	-	-	-	-	-	-	.41**	.52**	.57**	.58**	.70**	-					
21. Puberty F, 15.5	-.09	-.08	- .19**	-.13	.11	-.01	.02	-	-	-	-	-	-	-	.19**	.32**	.44**	.48**	.60**	.66**	-				
22. Self-Reg, M, 9.5	.62**	.55**	.52**	.37**	.25**	.24**	.13**	-.07	-.03	-.02	-.01	.05	-.00	- .02	-.03	- .14**	-.09*	-.06	-.01	-.03	- .02	-			
23. Self-Reg, M, 10.5	.54**	.62**	.52**	.39**	.26**	.24**	.17**	-.05	-.05	.01	.03	.08	.01	.05	-.08	- .15**	-.09	-.08	-.03	-.09	- .05	.74**	-		
24. Self-Reg, M, 11.5	.50**	.55**	.60**	.39**	.31**	.26**	.17**	-.03	-.03	.02	.01	.07	-.03	- .05	-.06	- .14**	-.09	-.11*	-.02	-.04	- .04	.72**	.74**	-	
25. Self-Reg, M, 15.5	.42**	.45**	.46**	.56**	.24**	.19**	.13**	-.05	-.00	.05	.03	.13**	.11*	.08	-.05	- .13**	-.09	- .13**	-.04	-.04	- .05	.61**	.60**	.64**	-

Note. * = p <.05, ** = p = <.001.

Table 5

Paternal Support, Puberty, Paternal Self-Regulation

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. P Sup, 9.5	-																					
2. P Sup, 10.5	.75**	-																				
3. P Sup, 11.5	.70**	.72**	-																			
4. P Sup, 15.5	.53**	.60**	.62**	-																		
5. Puberty, M, 9.5	-.04	-.04	-.09	.03	-																	
6. Puberty, M, 10.5	-.05	-.03	-.04	.07	.66**	-																
7. Puberty, M, 11.5	.04	.05	-.07	.04	.55**	.65**	-															
8. Puberty, M, 12.5	.04	.05	-.02	.02	.43**	.48**	.67**	-														
9. Puberty, M, 13.5	.03	.01	-.02	.04	.37**	.38**	.54**	.74**	-													
10. Puberty, M, 14.5	-.02	-.03	-.06	-.03	.28**	.22**	.38**	.55**	.73**	-												
11. Puberty, M, 15.5	.02	.04	-.06	.01	.28**	.18**	.36**	.48**	.62**	.74**	-											
12. Puberty, F, 9.5	-.10	-.09	-.13*	-.12*	-	-	-	-	-	-	-	-										
13. Puberty, F, 10.5	-.14*	-.10	-.13*	-.11	-	-	-	-	-	-	-	.74**	-									
14. Puberty, F, 11.5	-.06	-.09	-.08	-.14*	-	-	-	-	-	-	-	.61**	.78**	-								
15. Puberty, F, 12.5	-.03	-.03	-.06	-.12*	-	-	-	-	-	-	-	.48**	.67**	.78**	-							
16. Puberty, F, 13.5	-.01	-.03	-.05	-.15*	-	-	-	-	-	-	-	.43**	.55**	.65**	.69**	-						
17. Puberty, F, 14.5	-.03	-.06	-.13	-.17**	-	-	-	-	-	-	-	.41**	.52**	.57**	.58**	.70**	-					
18. Puberty F, 15.5	-.03	-.06	-.19*	-.16*	-	-	-	-	-	-	-	.19**	.32**	.44**	.48**	.60**	.66**	-				
19. Self-Reg, P, 9.5	.60**	.52**	.50**	.39**	-.07	-.03	.01	.05	.08	-.05	.00	-.10	-.13*	-.12*	-.11	-.07	-.03	-.09	-			
20. Self-Reg, P, 10.5	.54**	.59**	.53**	.44**	-.06	-.06	.05	.07	.05	-.02	.01	-.07	-.09	-.07	-.09	-.05	-.04	-.10	.71**	-		
21. Self-Reg, P, 11.5	.51**	.53**	.62**	.44**	-.07	-.09	-.04	.03	.05	.01	-.02	-.08	-.11	-.08	-.04	-.07	-.03	-.17*	.69**	.74**	-	
22. Self-Reg, P, 15.5	.35**	.41**	.42**	.61**	.01	.04	.06	.06	.15**	.14*	.12	-.15**	-.11	-.07	-.07	-.10	-.10	-.09	.53**	.60**	.61**	-

Note. * = p <.05, ** = p = <.001.

Table 6

Maternal Support, Maternal Monitoring, and Paternal Support Correlations

Variable	1	2	3	4	5	6	7	8	9	10	11
1. M Sup, 9.5	-										
2. M Sup, 10.5	.75**	-									
3. M Sup, 11.5	.69**	.71**	-								
4. M Sup, 15.5	.52**	.56**	.58**	-							
5. M Mon, 10.5	.24**	.24**	.23**	.15**	-						
6. M Mon, 11.5	.24**	.27**	.28**	.16**	.37**	-					
7. M Mon, 15.5	.19**	.19**	.17**	.26**	.36**	.49**	-				
8. P Sup, 9.5	.42**	.41**	.36**	.31**	.15**	.10*	.07	-			
9. P Sup, 10.5	.39**	.45**	.37**	.34**	.16**	.12**	.14**	.75**	-		
10. P Sup, 11.5	.38**	.41**	.44**	.32**	.16**	.14**	.09*	.70**	.72**	-	
11. P Sup, 15.5	.33**	.38**	.37**	.51**	.11**	.08*	.15**	.13**	.60**	.62**	-

Note. * = p <.05, ** = p = <.001

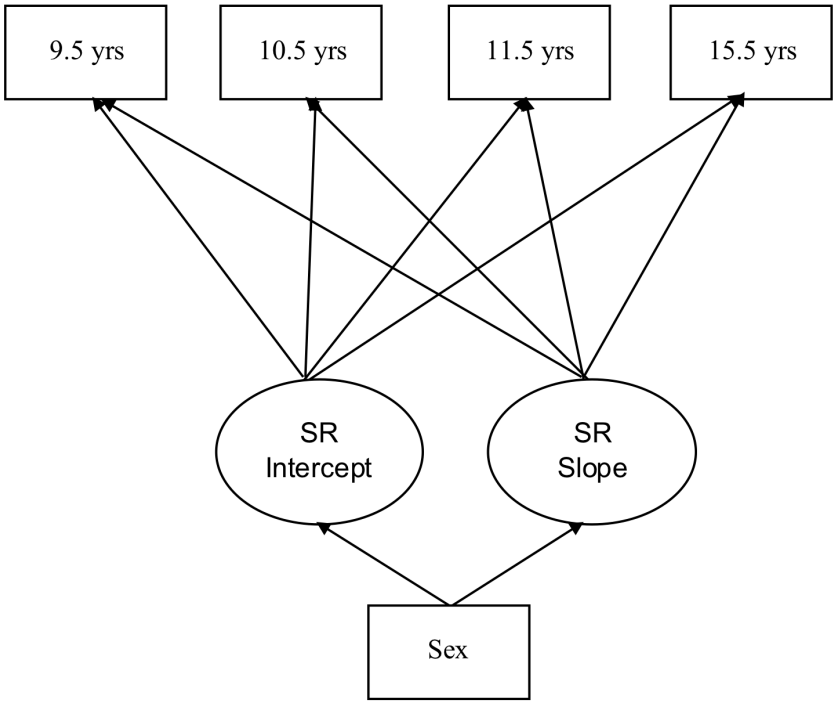
Hypothesis 1: It was expected that positive linear growth in SR would be found over the 6 year period.

A conditional LGM, where sex was used as a covariate in the model, was specified and tested in AMOS 22 (Arbuckle, 2005) to examine developmental changes in SR over the 6 year period (see Figure 3). The model hypothesized linear growth over time in SR, specified by 0, 1, 2, and 6 for slope parameter estimates to correspond with the ages 9.5, 10.5, 11.5, and 15.5 of the adolescents. The intercept parameter estimates were set to 1 and the intercept and slope factors were predicted by sex to test for sex differences. Mean differences at the intercept were expected to be associated with rates of change over time, where higher initial status is positively correlated with linear rates of change (slope). The positive growth in SR is expected largely due to recent neuroscientific evidence which suggests that in early adolescence, the brain is still developing which is related to the SR capacity. Therefore, as the adolescent gets older, their SR capacity should also improve (Luna & Sweeny, 2004; Steinberg, 2004; Steinberg, 2008). There also exists indirect evidence to support this as the likelihood of engaging in "risky" behaviors declines over time, presumably due to improved SR capacity (Steinberg, 2004, Steinberg, 2008). Factor loadings and standard fit indices (χ^2, χ^2 /df, CFI, RMSEA, and the associated *p* value) were used to evaluate model fit. Models also tested a quadratic growth function and declining growth to evaluate whether non-linear developmental changes exist in SR. Therefore, the paths for the quadratic term where identified with the following values: 0, 1, 4, and 36 and the paths for declining growth where set to 0, -1, -2, and -6. Although some evidence for sex differences in developmental changes in SR has been found in children (Kochanska, Murray, & Harlan,

2000), little empirical evidence based on adolescent samples exists; thus, it is unknown how and whether sex moderates developmental changes in SR (Brody & Ge, 2001; Buckner, Mezzacappa, & Beardslee, 2003; Purdie et al., 2004). Nevertheless, each model tested potential moderation effects by sex, where sex was first entered as a manifest variable predicting the intercept and the slope factors. Previous research has established important level differences in SR for male and female youth (e.g., based on children, Kochanska et al., 2000). However, in the focus was on whether sex moderated rates of change (e.g., slope) in SR during adolescence.

Figure 3

Hypothesis 1, Example of Unconditional Latent Growth Model for SR with Sex as a covariate



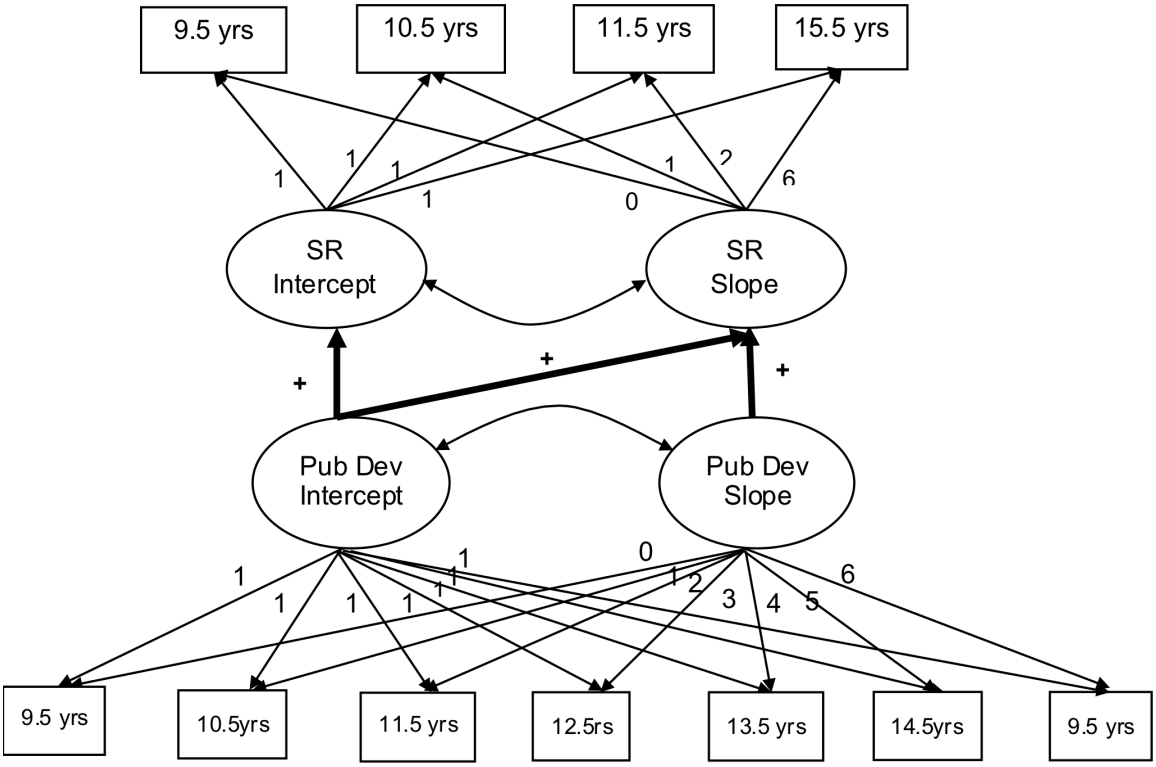
Hypothesis 2: It was hypothesized that pubertal developmental changes (slope) would predict developmental changes in SR.

A Parallel Process LGM was tested (see Figure 4) which modeled changes in pubertal development and how these affect developmental changes in SR over the 6 year period. The pubertal development indicators for each sex were combined to form a composite, consistent with previous work by Dick et al. (2001) and Wichstrom (2001). It was also expected that a positive linear relationship exists between the pubertal development intercept and the SR intercept as research has provided evidence that variability in pubertal development at age 11 is positively associated with variability in SR. Thus, higher levels of pubertal development were expected to predict higher initial status in SR (e.g., Steinberg, 2004). It was also expected that initial status of pubertal development would be positively associated with the developmental changes in SR over time, as high initial status also likely predicts greater subsequent changes in SR. Finally, the slope of pubertal development was expected to positively predict the SR slope (linear factor). Neuroscientific research has postulated that changes in pubertal development should impact the rate of change in SR over time (e.g., Cameron, 2004). Standard fit indices (χ^2, χ^2 /df, CFI, RMSEA, *p* value) were consulted to evaluate model fit; in addition, the three structural paths of interest were examined to see whether the predictions were supported. Based on previous empirical evidence (e.g., Cameron, 2004; Kelley et al., 2004; Luna & Sweeney, 2004; Steinberg, 2004; Whitford et al., 2007), it was expected that there would be positive growth in indicators of pubertal development over time and that these changes would predict developmental changes in SR over time

(positive, linear slope). As mentioned under hypothesis 1, posthoc analyses were conducted to test for potential sex differences as well as for non-linear growth.

Figure 4

Hypothesis 2, Example of Parallel Process Latent Growth Model for Pubertal Development and SR.



Hypothesis 3: It was expected that there would be positive growth in parental support over time and that this change would be predictive of developmental changes in SR over time.

Identical procedures were to be employed as in the previous set of analyses. However, due to complexities non-convergence issues of the model that included parental support in a Parallel Process LGM, a parental support variable was computed using the mean of both maternal support and paternal support, separately, from each time point. Therefore, maternal and paternal support were set as predictors of the intercept and the

slope of SR. It was expected that parental support would have a positive association with changes in SR as youth require more emotional support by parents as they attempt to develop adult roles (Barnes, Alan, & Farrell; 2000; Eisenberg et al., 2003; Grolnick & Farkas, 2002; Raikes et al., 2007; Silverman & Ragisa, 1992). Thus, higher levels of parental support would predict higher initial levels of SR as well as with developmental changes in SR over time. Factor loadings and standard fit indices (χ^2, χ^2 /df, CFI, RMSEA, *p* value) were consulted to evaluate model fit and the relationships of the paths. Consistent with previous model tests, sex and non-linear growth terms were also tested.

Table 7

Latent Growth Model Statistics for Maternal Models

Model	*X2*	*df*	*p*	CMIN	CFI	RMSEA	Unstandardized (b)	Variance (D)	Intercepts	Int to Slope Correlati
Maternal SR	9.19	6	.16	1.53	1.00	.020	Sex-SRi = .14** Sex-SRs = -.02	Di = .88*** Ds = .11***	SRi = -.23 ** SRs = .02	-.39***
Puberty→Maternal SR, M	269.58	54	0	4.99	.91	.075	Pubi->SRi = -.01 Pubs->SRs = .17** Pubi->SRs = .02*	Pub Di = .74*** Pub Ds = .14*** SR Di = .89*** SR Ds = .14***	Pubi = -.04 Pubs = .02** SRi = -.09** SRs = .01	SR = .42*** Pub = -.40***
Puberty→Maternal SR, F	178.37	54	0	3.30	.95	.06	Pubi->SRi = -.08 Pubs->SRs = -.13 Pubi->SRs = -.01	Pub Di = .74*** Pub Ds = .14*** SR Di = .89*** SR Ds = .12***	Pubi = .00 Pubs = .02** SRi = .06 SRs = -.01	SR = .38*** Pub = .41***
Maternal Support→ SR, M	5.12	7	.65	.73	1.00	0	MSup-SRi = .59*** MSup-SRs = -.01*	Di = .64*** Ds = .09**	SRi = -.08** SRs = .01	SR = -.47***
Maternal Support→ SR, F	10.60	6	.10	1.77	1.00	.03	Msup-SRi = .62*** MSup-SRs = -.00	Di = .64*** Ds = .12***	SRi = .06* SRs = -.01	SR = -.51***
Maternal Monitor→ SR, M	7.09	6	.31	1.18	1.00	.02	MMon-SRi = .29*** MMon-SRs = -.01**	Di = .82*** Ds = .09**	SRi = -.08** SRs = .01	SR = -.41***
Maternal Monitor→ SR, F	14.03	6	.03	2.34	.99	.05	MMon-SRi = .23*** MMon-SRs = .00	Di = .87*** Ds = .12***	SRi = .06 SRs = -.01	SR = -.39***

Note. Model was run separately for males and females. Male statistics are on the left/Female on the right. * = trend, ** = p <.05, *** = p = <.001, no * = non-significant.

Table 8

Latent Growth Model Statistics for Paternal Models

Model	*X2*	*df*	*p*	CMIN	CFI	RMSEA	Unstandardized (b)	Variance (D)	Intercepts	Int to Slop Correlatio
Paternal SR	12.00	6	.06	2.0	1.0	.03	Sex-SRi = .17** Sex-SRs = -.02*	Di = .87*** Ds = .08**	SRi = -.32** SRs =.03*	.39***
Puberty→ Paternal SR, M	213.83	54	0	3.96	.91	.07	Pubi->SRi = .00 Pubs->SRs = .21** Pubi->SRs = .03**	Pub Di = .77*** Pub Ds = .16*** SR Di = .91*** SR Ds = .16***	Pubi = -.04 Pubs = .02** SRi = -.15** SRs = .01	SR = .42** Pub = -.47***
Puberty→ Paternal SR, F	179.63	53	0	3.39	.94	.06	Pubi->SRi = -.11 Pubs->SRs = .03 Pubi->SRs = -.00	Pub Di = .86*** Pub Ds = .13*** SR Di = .86*** SR Ds = .13***	Pubi = .00 Pubs = .02** SRi = .02 SRs = -.02	SR = -.36* Pub = -.43***
Paternal Support→ SR, M	5.56	6	.47	.93	1.00	0	PSup-SRi = .71*** PSup-SRs = -.02**	Di = .60*** Ds = .07	SRi = -.14*** SRs = .02*	SR = -.49*
Paternal Support→ SR, F	17.79	6	.007	2.965	.986	.055	PSup-SRi = .697*** PSup-SRs = -.155	Di = .62*** Ds = .09**	SRi = -.026 SRs = -.006	SR = -.526***
Paternal Monitor→ SR, M	1.95	6	.92	.33	1.00	0	MMon-SRi = .22*** MMon-SRs = -.00	Di = .86*** Ds = .08**	SRi = -.14** SRs = .01	SR = -.48*
Paternal Monitor→ SR, F	16.97	6	.01	2.83	.98	.05	MMon-SRi = .17** MMon-SRs = -.02**	Di = .84*** Ds = .09**	SRi = .02 SRs = -.01	SR = -.32*

Note. Model was run separately for males and females. Male statistics are on the left/Female on the right. * = trend, ** = p <.05, *** = p = <.001, no * = non-significant.

Hypothesis 4: Does parental monitoring influence possible changes in SR during adolescence? The current question was exploratory in nature due to inconsistencies in the literature whether parental monitoring increases or decreases over time during adolescence.

As with hypothesis 3, a mean monitoring score was developed across time points due to model complexity and non-convergence; this terms was added into the model as a predictor of SR intercept and slope. Scholarship that includes longitudinal measures of monitoring are few in social sciences, leaving open the question of whether monitoring increases or decreases over time. Some research has provided evidence that monitoring increases over early adolescence into middle adolescence as this is a period of increased adolescent misbehaviors (Finkenauer et al., 2003; Kerr & Stattin, 2000), because adolescents spend more time with their peers than their parents. More recently, Pettit, Keiley, Laird, Bates, and Dodge (2007) found that maternal reported monitoring decreased over time when employing growth modeling procedures in a longitudinal sample of adolescents in grades 5, 6, 8, and 11. Thus, it was expected that youth who had higher monitoring would experience increased rates of developmental changes in SR. Factor loadings and standard fit indices (χ^2, χ^2 /df, CFI, RMSEA, *p* value) were consulted to evaluate the relationships between monitoring and the intercept and slope of SR as well as model fit. Once again, sex moderation effects as well as potential non-linear changes were also tested. Some research has shown sex differences in monitoring, where female adolescents experience greater monitoring by parents in more concerted effort to "control" their behaviors than male youth (e.g., Finkenauer et al., 2005; Smetana & Daddis, 2002).

Hypothesis #5. It was expected that there would be independent and thus additive effects by parenting (support and monitoring) and by pubertal development on SR development.

All main predictors (e.g., pubertal development, parental support, and parental monitoring) of SR were combined into a single model to test whether each maintained significant effects on the development of SR, and thus are additive, or whether they are non-additive. Identical procedures were employed as previously described. It was expected that significant associations between the slope of pubertal development and the slope of SR (a positive association) would be found; in addition, it was expected that parental support (positive association) and parental monitoring (positive association) would maintain a positive effect on the slope of SR. It is possible, however, that the effects would not be fully additive, but rather non-additive, or perhaps partially additive. For a non-additive model, there would only be one remaining significant effect from either pubertal development or parental support or parental monitoring. For partially additive effects, there might be at least two significant predictors of SR development. This might include any combination of the three constructs, such as pubertal development and parental support, or pubertal development and parental monitoring, or parental support and parental monitoring. In addition, if the quadratic exploratory analyses from hypotheses 2-3 are supported, then parallel models would be tested in the same manner, with the same expectation of additive effects. Finally, the above models and permutations would need to be repeated to examine for potential similarities or differences across models with different raters of parenting behaviors. Standard fit indices (χ^2, χ^2 /df, CFI, RMSEA, *p* value) were employed to evaluate model fit (e.g., Bentler, 1990; Bentler &

Bonnet, 1980); in addition, individual structural paths of interest were examined to determine whether predictions were supported. Again, sex as well as non-linear factors were tested as an exploratory analysis.

Results

Hypothesis 1

To test hypothesis 1 (see Figure 1) a conditional latent growth model (LGM) was specified that included sex as a covariate predicting the intercept and the slope for both maternal and paternal rated adolescent self-regulation (SR). The parameter estimates from the intercept to the latent variables (which included maternal and paternal ratings, separately, rated adolescent SR at ages 9.5, 10.5, 11.5 and 15.5) were fixed to a 1, while the parameter estimates from the slope to the latent variables were fixed to 0, 1, 2, and 6 in order to test for linear growth over the 6 year period, based on four assessment points. In order to stabilize betas in error terms, a correlation was specified between error terms 1 and 4.

Maternal-rated SR. For maternal rated adolescent SR, model fit was excellent ($\chi^2 = 9.19$, df = 6, $p = .16$, $\chi^2/df = 1.53$, CFI =.99, RMSEA = .02). The variance of the intercept was significant ($D_i = .88$, $p = <.001$) indicating significant differences in SR for individual adolescents at initial status (age 9.5). Additionally, the variance in the slope was also significant ($D_s = .11$, $p = <.001$), suggesting there were differences in the rates of change in adolescents during the 6 year period. The mean of the SR intercept was statistically significant ($\mu_i = -.23$, $p = < .05$); however, the mean of the slope was not significant ($\mu_s = .02$, $p = .11$), suggesting no linear growth in maternal rated SR over the 6 year period. The correlation between the intercept and slope was significant at r = -.39 ($p = <.001$) indicating that higher maternal ratings of SR at initial status were associated with lower rates of change in SR over time. Sex was significantly related to the intercept (b = .14, $p = <.05$), but was only a statistical trend related to the slope (b = -

.02, p = .08), suggesting possible sex differences in the development of SR in adolescence. This was further investigated in other hypotheses. Finally, no evidence was found of significant growth declining or non-linear changes in SR over the 6 year period. Therefore, the fundamental hypothesis was not supported with these data.

Paternal-rated SR. For paternal rated adolescent SR model fit was excellent (χ^2 = 12.00, df = 6, p = .06, χ^2/df= 2.00, CFI =.99, RMSEA = .027). The variance of the intercept was significant (D_i = .87, p = <.001) as was the variance in the slope (D_s = .08, p = <.05). This indicated individual differences in responses at initial assessment and differences in rates of adolescent SR development. This model produced a significant mean for the SR intercept (μ_i = -.32, p = <.05) but only a statistical trend effect in the mean of the SR slope (μ_s = .03, p = .09). This result suggested some modest evidence of potential linear growth in adolescent SR when rated by fathers. The correlation between the intercept and slope was significant at r = -.39 (p = <.001) indicating that individuals with higher paternal ratings of SR at initial status tended to experience lower slope scores or less growth in SR over time. Sex was significantly associated with SR intercept (b = .17, p = <.05); only a statistical trend for SR slope was found (b = -.02, p = .06). Due to some modest evidence of changes in the SR slope over time, a decision was made to continue testing the additional study hypotheses which examined how and whether measures of pubertal development and of parenting processes accounted for variability in the developmental changes of SR. As with maternal rated SR, declining as well as quadratic parameter estimates were specified and tested in follow-up analyses. However, no changes in model fit or in the slope term were found. Based on these results, neither maternal nor paternal rated SR latent growth models indicated growth or development in

SR over the 6 year period. Nevertheless, based on finding a trend in the paternal rated SR slope, the remaining hypotheses were tested.

Hypothesis 2

To test hypothesis 2, a parallel process latent growth model was specified, where the LGM of pubertal development predicted the LGM of SR. This approach permits testing the main paths of interest, namely the paths from the pubertal development intercept to the SR intercept, the path from the pubertal development slope to the SR slope, and the path from the pubertal development intercept to the SR slope. This model was tested separately for male and female youth. For the SR LGM, all parameter estimates from the intercept to the latent variables were fixed to a 1, while the parameter estimates from the slope to the latent variables were fixed to 0, 1, 2 and 6in order to test for linear growth over the 6 year period. For the pubertal development LGM, all parameter estimates from the intercept to the latent variables were fixed to 1 while the parameter estimates from the slope to the latent variables were fixed to 0, 1, 2, 3, 4, 5, 6, and 7. The LGM for puberty had sex-specific latent variables. Therefore, four total models were tested for this hypothesis, specifically, maternal-male, maternal-female, paternal-male, and paternal-females.

Maternal-rated model (males). For males (see Table 3), model fit was acceptable ($\chi^2 = 268.32$, df = 54, $p = 0$, $\chi^2/df = 4.97$, CFI =.91, RMSEA = .08) when assessing maternal rated adolescent SR among male youth. There was a significant variance in both the intercept and the slope of pubertal development ($D_i = .74$, $p = <.001$; $D_{s=} .14$, $p = <.001$) indicating differences in scores of adolescents at initial assessment and over time. Additionally, significant variance was found in the intercept and slope of

SR (D_i = .89, p = <.001; $D_{s=}$ .14, p = <.001) indicating a significant difference among adolescents SR at initial assessment and in the developmental growth of SR over the 6 year period. While there was no significance in the mean of the pubertal intercept for males (μ_i = -.04, p = .38), the mean of the pubertal slope was significant (μ_s = .02, p = <.05) suggesting linear changes in pubertal development over the 6 year period. The mean of the intercept for maternal rated SR in males was significant (μ_i = -.09, p = <.05), while the mean of the slope was not (μ_s = .01, p = .54) indicating no growth in SR over the 6 year period for males when mothers rated their SR. The covariance between the intercept and slope of pubertal development was significant at r = -.40, p = <.001. This suggests that males with higher pubertal scores at initial assessment tended to have slower rates of change in puberty over time. The covariance between the intercept and slope of SR was also significant (SR, r = .42, p = <.001) indicating that higher maternal rated SR scores at initial status were related to more rapid changes in SR over time.

When examining the main paths of interest, namely the paths between the intercept and slope of the two latent growth models, no significant effect was found between the intercept of pubertal development and the intercept of maternal rated SR in males (b = -.01, p = .85). Additionally, no effect was found between pubertal development at initial status (i.e., intercept) and changes in maternal rated adolescent SR (slope) over time (b = .02, p =.11). This suggests that initial status of pubertal development is not associated to developmental changes in SR over time for males when mothers rate their SR. Additionally, a significant effect was found between the pubertal development slope and maternal rated adolescent SR slope over the 6 year period (b = .17, p = <.05). This indicated that changes in pubertal development were positively

associated with developmental changes in over the 6 year period as hypothesized. Specifically, there was a positive association suggesting that as adolescents matured (pubertal developmental changes), this was associated with positive developmental changes in mother-reported SR over the six year period among males.

Maternal-rated model (females). For this model (see Table 3), model fit was acceptable ($\chi^2 = 173.73$, df = 53, $p = 0$, $\chi^2/df = 3.28$, CFI =.95, RMSEA = .06). The variance for pubertal development at intercept was significant as well as the variance of the slope ($D_i = .85$, $p = <.001$; $D_{s=} .123$, $p = <.001$). Additionally, the variance of maternal rated SR at initial status was also significant ($D_i = .89$, $p = <.001$; $D_{s=} .12$, $p = <.001$) as well as the variance of the slope. This suggests a great amount of variance in individual scores for both pubertal development and maternal rated SR at initial status as well as variances in the scores over time for individuals. The mean of pubertal development intercept was not significant ($\mu_i = .00$, $p = .97$) but the mean of the slope for pubertal development was significant ($\mu_s = .02$, $p = <.05$). This suggests there was growth over time in pubertal development between the ages of 9.5 and 15.5. For SR, both the mean of the intercept and slope were non-significant ($\mu_{i=} .06$, $p = .20$; $\mu_s = -.01$, $p = .29$). Therefore, there was no evidence of growth or development in maternal rated SR in females over the 6 year period. The correlation between pubertal development intercept and slope was significant ($r = .41$, $p = <.001$) indicating that individual with higher scores in puberty at initial assessment tended to have higher slope scores in puberty over time. For SR, the correlation between the intercept and slope was significant ($r = .38$, $p = <.001$) suggesting that higher scores in SR at initial status tended to have higher scores in the slope over time. There were no statistical significant findings in the paths of interest

for female pubertal development LGM and maternal rated SR LGM. Based on these findings, hypothesis 2 for the maternal-female model is rejected as there are no significant effects of female pubertal developmental on maternal rated adolescent SR.

Paternal-rated model (males). For males (see Table 3), model fit was acceptable (χ^2 = 213.83, df = 54, p = 0, χ^2/df = 3.96, CFI =.91, RMSEA = .07) when assessing paternal rated adolescent SR. There was significant variance in both the intercept and the slope of pubertal development (D_i = .77, p = <.001; D_s = .16, p = <.001) indicating differences in scores of adolescents at initial assessment and over time. Additionally, significant variance was found in the intercept and slope of SR (D_i = .91, p = <.001; D_s = .16, p = <.001) indicating a significant difference among adolescents SR at initial assessment and in the developmental growth of SR over the 6 year period among adolescents. While there was no significance in the mean of the pubertal intercept for males (μ_i = -.04, p = .39), the mean of the pubertal slope was significant (μ_s = .02, p = <.05) suggesting slight linear changes in pubertal development over the 6 year period for males. The mean of the intercept for paternal rated SR in males was significant (μ_i = -.15, p = <.05) while the mean of the slope was not (μ_s = .01, p = .49). This indicates no growth in SR over the 6 year period for males when fathers rated their SR. The covariance between the intercept and slope of pubertal development was significant at r = -.47, p = <.001. This suggests that males with higher pubertal scores at initial assessment tended to have lower slope scores on puberty or less growth in puberty over time. The covariance between the intercept and slope of SR was also significant (SR, r = .42, p = <.001) indicating **that** higher paternal rated SR scores at initial status were related to higher scores or more positive growth over time for males.

When examining the paths of interest, namely the paths between the intercept and slope of the two latent growth models, there was **no significance** between the intercept of pubertal development and the intercept of paternal rated SR for males (b = .00, p = .99). However, there was a significant association between male pubertal development at initial status (i.e., intercept) and changes in paternal rated adolescent SR (slope) over time (b = .03, p = <.05). This suggests that pubertal development when first assessed is related, while minimally, to the development of paternal rated SR over time for males. Additionally, a significant effect did exist between male pubertal development slope and paternal rated adolescent SR slope over the 6 year period (b = .21, p = <.05) for males. This indicates that changes in male pubertal development have a positive association on the development of SR over the 6 year period. This supports hypothesis 2 namely that positive changes in puberty is associated with positive changes in SR. Based on this result, it appears that maturational changes (puberty) in adolescence is associated with positive growth/development in father-rated SR during adolescence among males.

Paternal-rated model (females). Model fit was acceptable (χ^2 = 179.63, df = 53, p = 0, χ^2/df = 3.39, CFI =.94, RMSEA = .06) for this model (see Table 3). The variance for female pubertal development at intercept was significant as well as the variance of the slope (D_i = .86, p = <.001; D_s = .13, p = <.001). Additionally, the variance of paternal rated SR at initial status was also significant (D_i = .86, p = <.001; D_s = .13, p = <.001) as well as the variance of the slope. This suggests a great amount of variance in individual scores for both pubertal development and paternal rated SR at initial status as well as variances in the scores over time for individuals. The mean of pubertal development intercept was not significant (μ_i = .00, p = .95) but the mean of the slope for pubertal

development was significant (μ_s = .02, p = <.05). This suggests there was growth over time in pubertal development for females. For SR, both the mean of the intercept and slope were non-significant (μ_i = .02, p = .67; μ_s = -.02, p = .11). Therefore, there was no evidence of growth or development in paternal rated SR in females over the 6 year period. The correlation between pubertal development intercept and slope was significant (r = .43, p = <.001) indicating that individual with higher scores in puberty at initial assessment tended to have higher slope scores in puberty over time. For paternal rated SR, the correlation between the intercept and slope was significant (r = -.36, p = <.001) suggesting that higher scores in SR at initial status tended to have lower scores in the slope over time for females. There were no statistical significant findings in the paths of interest for female pubertal development LGM and maternal rated SR LGM. However, there was a statistical trend between pubertal development at intercept and SR at intercept (-.11, p = .08). Based on these findings, hypothesis 2 for the paternal-female model is rejected as there are no significant effects of female pubertal development on paternal rated adolescent SR.

Hypothesis 3

To test hypothesis 3 a conditional latent growth model was created where a mean score for parental support was computed from assessment points in which the child was 9.5, 10.5, 11.5, and 15.5 and drawn as a predictor of the intercept and slope of SR. This model was tested by sex by selecting males and then females from the data file in which to run the model. Therefore, there were four models that were tested; namely, maternal-male, maternal-female, paternal-male, and paternal-female. All parameter estimates from the intercept to the latent variables were fixed to a 1 while the parameter estimates from

the slope to the latent variables were fixed to 0, 1, 2 and 6 in order to test for linear growth in SR over the 6 year period.

Maternal-rated model (males). Model fit was acceptable at $\chi^2 = 5.12$, df = 7, $p = .65$, $\chi^2/df = .73$, CFI = 1.00, RMSEA = 0 for the current model. The variances of the intercept and slope were both significant ($D_i = .64$, $p = <.001$; $D_s = .09$, $p = <.001$) indicating a large amount of variance among adolescent SR at initial status but only a slight, albeit, a significant one among adolescent SR over time. Only the mean of the intercept for maternal rated SR for males was significant ($\mu_i = -.08$, $p = <.05$; $\mu_s = .01$, $p = .28$) suggesting there is a very slight significant mean in the intercept of maternal rated SR for males in the current model. There was a significant and negative correlation between the intercept and the slope of maternal rated SR for males in this model ($r = -.47$, $p = <.001$) suggesting that higher levels of SR for males at initial assessment tended to have lower slope levels or less positive growth over time. Maternal support had a significant effect on the intercept of maternal rated SR in males ($b = .59$, $p = <.001$), however, it only produced a statistically significant trend effect on the slope of SR ($b = -.01$, $p = .06$). Based on the results, maternal support did not affect the development of maternal rated SR over the 6 year period for males.

Maternal-rated model (females). Model fit was acceptable at $\chi^2 = 10.60$, df = 6, $p = .10$, $\chi^2/df = 1.77$, CFI = .99, RMSEA = .03 for the current model. The variances of the intercept and slope were both significant ($D_i = .64$, $p = <.001$; $D_s = .12$, $p = <.001$) indicating a large amount of variance among maternal rated adolescent SR for females at initial status and a modest variance among maternal rated adolescent SR for females over time. The mean of the intercept for maternal rated SR for females was significant trend

(μ_i = .06, p = .08) while the mean of the slope for maternal rated SR for females was non-significant (μ_s = -.01, p = .13) suggesting there is a very slight significant mean of maternal rated SR at initial status for females in the current model. There was a significant and negative correlation between the intercept and the slope of maternal rated SR for females in this model (r = -.51, p = < .001) suggesting that higher scores of SR for females at initial assessment tended to have lower slope levels or less positive growth in maternal rated SR over the 6 year period. Maternal support had a large significant effect on the intercept of maternal rated SR in females (b = .62, p = < .001), however, it was not significantly related to the slope of maternal rated SR in females (b = -.00, p = .78). Based on the results, maternal support did not affect developmental changes in maternal rated SR over the 6 year period among females.

Paternal-rated model (males). Model fit was acceptable at χ^2 = 5.56, df = 6, p = .47, χ^2/df = .93, CFI = 1.00, RMSEA = 0 for the current model. The variances of the intercept and slope were both significant (D_i = .60, p = <.001; D_s = .07, p = <.05) indicating a large amount of variance among adolescent SR at initial status but only a slight, albeit, a significant one among paternal rated adolescent SR over time for males. Only the mean of the intercept for paternal rated SR for males was significant while the mean of the slope produced a statistical trend (μ_i = -.14, p = <.001; μ_s = .02, p = .08). This suggests there is a very slight significant mean in the intercept of maternal rated SR for males in the current model. There was a significant and negative correlation between the intercept and the slope of maternal rated SR for males in this model (r = -.49, p = <.05) suggesting that higher levels of paternal rated SR for males at initial assessment tended to have lower slope levels or less positive growth over time. Paternal support had

a large and significant effect on the intercept of paternal rated SR in males (b = .71, *p* = <.001). There was a slight significant effect of paternal support on the slope of paternal rated SR in males (b = -.02, *p* = <.05). This suggests that paternal support is associated with paternal rated SR in male adolescents at initial assessment and in the development of SR over the 6 year period. Based on the results and opposite to my hypotheses, paternal support was negatively associated with developmental changes in paternal rated SR over time for males.

Paternal-rated model (females). Model fit was good at $\chi^2 = 17.79$, df = 6, *p* = .01, $\chi^2/df = 2.97$, CFI = .99, RMSEA = .06 for the current model. The variances of the intercept and slope were both significant ($D_i = .62$, *p* = <.001; $D_s = .09$, *p* = <.001) indicating a large amount of variance among paternal rated adolescent SR for females at initial status and a modest variance among paternal rated adolescent SR for females. Neither the mean of the intercept nor the slope for paternal rated SR for females were significant. There was, however, a significant and negative correlation between the intercept and the slope of paternal rated SR for females (r = -.42, *p* = <.001), suggesting that higher scores of SR at initial assessment were associated with lower slope levels, changes or less positive changes/growth in father- rated SR over the 6 year period. Paternal support had a large significant effect on the intercept of paternal rated SR in females (b = .62, *p* = <.001); however, it was not significantly related to the slope of paternal rated SR in females (b = -.00, *p* = .48). Based on the results, paternal support did not appear to affect developmental changes in maternal rated SR over the 6 year period among females.

Hypothesis 4

To test hypothesis 4 a conditional latent growth model was hypothesized where a mean score for maternal ratings of parental monitoring (there was no measure for paternal monitoring during these assessment points) was computed from the following assessments: Age 9.5, 10.5, 11.5, and 15.5 years. Maternal monitoring was then specified as a predictor of the intercept and slope of adolescent SR. This model was then tested by sex.

Maternal-rated model (males). Model fit was good ($\chi^2 = 7.09$, df = 6, $p = .31$, $\chi^2/df = 1.18$, CFI =.99, RMSEA = .02) for the current model. The variances of the intercept and slope were both significant ($D_i = .81$, $p = <.001$; $D_s = .09$, $p = <.001$), indicating a large amount of variance in adolescent SR at initial status and the SR slope. Only the mean of the intercept for maternal rated SR for males was significant ($\mu_i = -.08$, $p = <.05$; $\mu_s = .01$, $p = .29$). There was a significant and negative correlation between the intercept and the slope of maternal rated SR for males in this model (r = -.41, $p = <.001$), suggesting that higher levels of SR at initial assessment were associated with lower rates of change over time. Maternal monitoring had a significant effect on the intercept of maternal rated SR in males (b = .29, $p = <.001$) and on the slope (b = -.01, $p = <.05$). This suggest that higher levels of maternal monitoring were associated with higher levels of maternal rated SR in adolescent males at initial status and that higher levels of maternal monitoring were associated with lower rates of change in SR or less positive growth. Thus, maternal monitoring had a small effect on developmental changes of SR over time for males and was opposite to the hypothesized effect.

Maternal-rated model (females). Model fit was excellent (χ^2 = 14.03, df = 6, p = .03, χ^2/df = 2.34, CFI = .99, RMSEA = .05 for the current model. The variances of the intercept and slope were both significant (D_i = .87, p = <.001; D_s = .12, p = <.001), indicating a large amount of variance among maternal rated adolescent SR for females at initial status and a modest amount of variance in the maternal rated slope of SR. Neither the mean of the intercept nor the one of the slope for maternal rated SR for females were significant. However, a significant and negative correlation between the intercept and the slope of maternal rated SR for females was found (r = -.39, p = <.001) suggesting that higher scores of SR for females at initial status was associated with a smaller rate of change or less positive growth in maternal rated SR over the 6 year period. Maternal monitoring had a significant effect on the intercept of maternal rated SR in females (b = .29, p = <.001) and on the slope of maternal rated SR in females (b = -.01, p = <.05). This suggests that maternal monitoring had a small, negative effect on the development changes of maternal rated SR for females which is opposite to the hypothesis. Thus, like for male youth, maternal monitoring also had a small negativeeffect on developmental changes of maternal rated SR over the 6 year period for females.

Paternal-rated model (males). As mentioned previously, maternal monitoring had to be used in the paternal rated SR models focused on testing monitoring as there were no paternal monitoring questions available. Model fit was acceptable at χ^2 = 1.95, df = 6, p = .92, χ^2/df = .33, CFI = 1.00, RMSEA = 0 for the current model. The variances of the intercept and slope were both significant (D_i = .86, p = <.001; D_s = .08, p = <.001), indicating substantial variance in the intercept term and modest variance in the slope terms of the model. Additionally, the mean intercept term for paternal rated SR among

males was significant (μ_i = -.14, p = <.001; μ_s = .01, p = .22), however, the mean of the slope was not.

There was a significant and negative correlation between the intercept and the slope of paternal rated SR for males in this model (r = -.48, p = <.05) suggesting that higher levels of paternal rated SR for males at initial assessment tended to be associated with lower rates of change or positive growth over time. In terms of predictive effects, maternal monitoring had a significant effect on the intercept of paternal rated SR in males (b = .22, p = <.001). There was no meaningful effect of maternal monitoring on the slope of paternal rated SR in males, even though it reach statistical significance (b = -.00, p = <.05). This suggests that maternal monitoring was associated with paternal rated SR in male adolescents at initial status but not with developmental changes of SR over the 6 year period. Based on the results, maternal monitoring does not have an effect on the development of paternal rated SR over time among males.

Paternal-rated model (females). Model fit was good at χ^2 = 16.97, df = 6, p = .01, χ^2/df = 2.83, CFI = .98, RMSEA = .05 for the current model. The variances of the intercept and slope were both significant (D_i = .84, p = <.001; D_s = .09, p = <.001). Neither the mean of the intercept nor the slope term reached significance. There was, however, a significant and negative correlation between the intercept and the slope of paternal rated SR for females (r = -.32, p = <.001), suggesting that higher scores of SR for females at initial assessment were associated with smaller slopes or less positive changes over time in paternal rated SR over the 6 year period. Maternal monitoring had a significant effect on the intercept of paternal rated SR in females (b = .17, p = <.05) and on the slope of paternal rated SR in females (b = -.02, p = <.05). This indicates that

higher scores of maternal monitoring are associated with higher scores of paternal rated SR in females at initial status and a negative association with developmental changes in SR for females which is opposite to the hypothesis. Based on the results, maternal monitoring did have a slight negative effect on the development of paternal rated SR over the 6 year period for females, where it predicted less positive changes.

Hypothesis 5

Finally, hypothesis 5 (see Figures 5 and 6) specified a conditional parallel process latent growth model, where all main study variables (whether latent variables or growth variables) were modeled together to assess whether or socialization processes vis-à-vis measures of maturation (puberty) provided unique and/or additive effects on developmental changes in SR during adolescence. As with hypotheses 3 and 4, a mean score for parental support (maternal and paternal) and monitoring (maternal) were computed from assessment points in which the child was 9.5, 10.5, 11.5, and 15.5 years old. These variables were then used as predictors of the intercept and slope terms of the intercept and slope term of adolescent SR. The model was tested separately by sex. Therefore, there were four models that were tested under hypothesis 5, namely, maternal rated SR focused on male youth, maternal rated SR focused on female youth, paternal rated SR focused on male youth, and paternal rated SR focused on female youth. Due to the complexity of the models, only significant findings will be highlighted here.

Maternal-rated model (males). Model fit was adequate in this additive model (χ^2 = 291.42, df = 72, p = 0, χ^2/df = 4.05, CFI = .92, RMSEA = .07). Significant intercept and slope variances were found for both pubertal development and maternal rated SR among males. Specifically, the variance of the intercept for pubertal development was large and

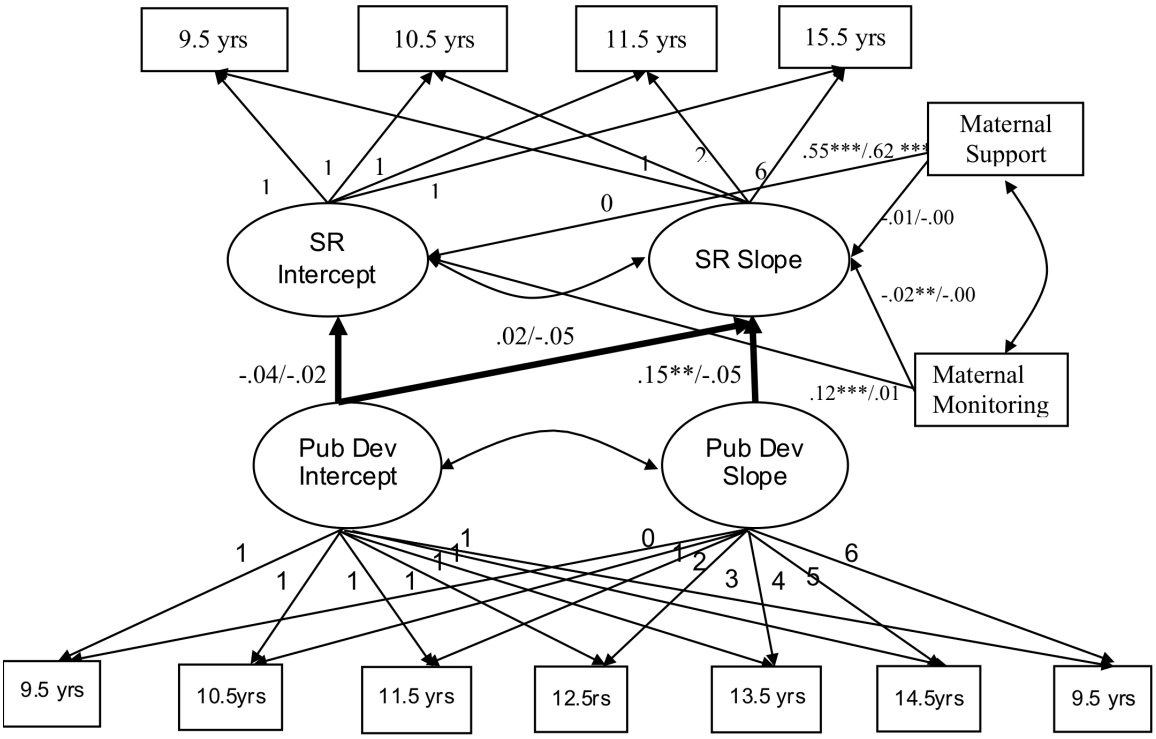
significant (D_i = .74, p = <.001), while the variance of the slope was also significant (D_s = .14, p = <.001). Additionally, the variance of the intercept for maternal rated SR was also significant (D_i = .65, p = <.001) as was the slope (D_s = .14, p = <.001). These results suggest a large amount of variance in individual scores of puberty and maternal rated SR at initial status and in scores among adolescents in puberty and maternal SR over time. The mean of the intercept of pubertal development was not significant but the slope was significant (μ_s = .02, p = <.05). The intercept of maternal rated SR was also significant for males in this model (μ_i = -.07, p = <.05). These findings indicate that there was slight positive growth in pubertal development over time for males but there was no growth in maternal rated SR for males. The correlations between the intercept and slope for puberty and SR were significant (r = -.40 and r = -.44, p = <.001; respectively). These correlations indicate that higher levels of puberty and maternal rated SR at initial status were associated with lower slope scores or less change over the 6 year period.

For the paths of interest between the two LGMs, all remained significant when maternal support and monitoring were added to the model, as previously found in Hypothesis 2, with some slight decreases in the magnitude of the relationships. Specifically, the pubertal development slope had a significant effect on the slope of maternal rated SR for males (b = .15, p = <.05). This finding indicated that even when the effects for support and monitoring are included in the model, changes in pubertal development over the 6 year period were associated with developmental changes in SR among males as rated by mothers. Maternal support maintained an effect on the SR intercept (b = .551, p = <.001), but not the slope. Additionally maternal monitoring continued to have an effect of the intercept and the slope of maternal rated SR (b = .13, p

= <.001 and b = -.02, *p* = <.05; respectively). Therefore, the current model was partially additive. Due to the fact that there were two parenting variables in the model, they were allowed to covary, by specifying an error term. The correlation between maternal support and maternal monitoring was significant (r = .32, *p* = <.001). As expected, this suggested that higher scores of maternal support are associated with higher scores of maternal monitoring.

Figure 5

Hypothesis 5, Conditional Parallel Process Latent Growth Model for Additive Model for Maternal Ratings



Note. Model was run separately for males and females. Male statistics are on the left/Female on the right. * = trend, ** = *p* <.05, *** = *p* = <.001, no * = non-significant.

Maternal-rated model (females). Model fit was acceptable in this additive model (χ^2 = 203.14, df = 72, p = 0, χ^2/df = 2.82, CFI = .95, RMSEA = .05). There were significant findings in the intercept and slope variances of both pubertal development and maternal rated SR for females. Specifically, the variance of the intercept for pubertal development was large and significant (D_i = .85, p = <.001), while the variance of the slope was also significant (D_s = .12, p = <.001). Additionally, the variance of the intercept for maternal rated SR was also significant (D_i = .65, p = <.001) as was the slope (D_s = .12, p = <.001). These results suggest a large amount of variance in individual scores for females in pubertal development and maternal rated SR both at initial status and in rates of change over time. The mean of the intercept of pubertal development was not significant but the slope was significant (μ_s = .02, p = <.05). A statistical trend was found for the intercept of maternal rated SR (μ_i = .06, p = .10), but the slope was not significant. These findings indicate that there was slight positive growth in pubertal development over time for females but no growth in maternal rated SR for females. The correlations between the intercept and slope for puberty and SR were significant (r = -.50 and r = -.41, p = <.001; respectively). These correlations indicated that higher levels of female puberty and maternal rated SR at initial status were associated with lower rates of change over the 6 year period. As with Hypothesis 2, none of the paths of interest between the two LGMs were significant in model which tested maternal ratings of SR among female youth. The relationship between maternal support and maternal rated SR at the intercept remained significant (b = .62, p = <.001), although the strength of the association weakened slightly once all constructs were included in this final model. However, the previous significant relationship between maternal monitoring and maternal rated SR intercept

became non-significant once all variables were added to the model. Again, the two parenting variables in the model were specified to covary (r = .33, p = <.001).

Paternal-rated model (males). Model fit was acceptable in the paternal-male final model (χ^2 = 235.29, df = 72, p = 0, χ^2/df = 3.27, CFI = .92, RMSEA = .06). There were significant findings in the intercept and slope variances of both pubertal development and paternal rated SR for males. Specifically, the variance of the intercept for male pubertal development was large and significant (D_i = .77, p = <.001), while the variance of the slope was also significant (D_s = .16, p = <.001). Additionally, the variance of the intercept for paternal rated SR was also significant (D_i = .63, p = <.001) as was the slope (D_s =.16, p = <.001). These results suggest a large amount of variance in individual scores of puberty among adolescent males and paternal rated SR at initial status and in scores among adolescents in puberty and paternal SR over time.

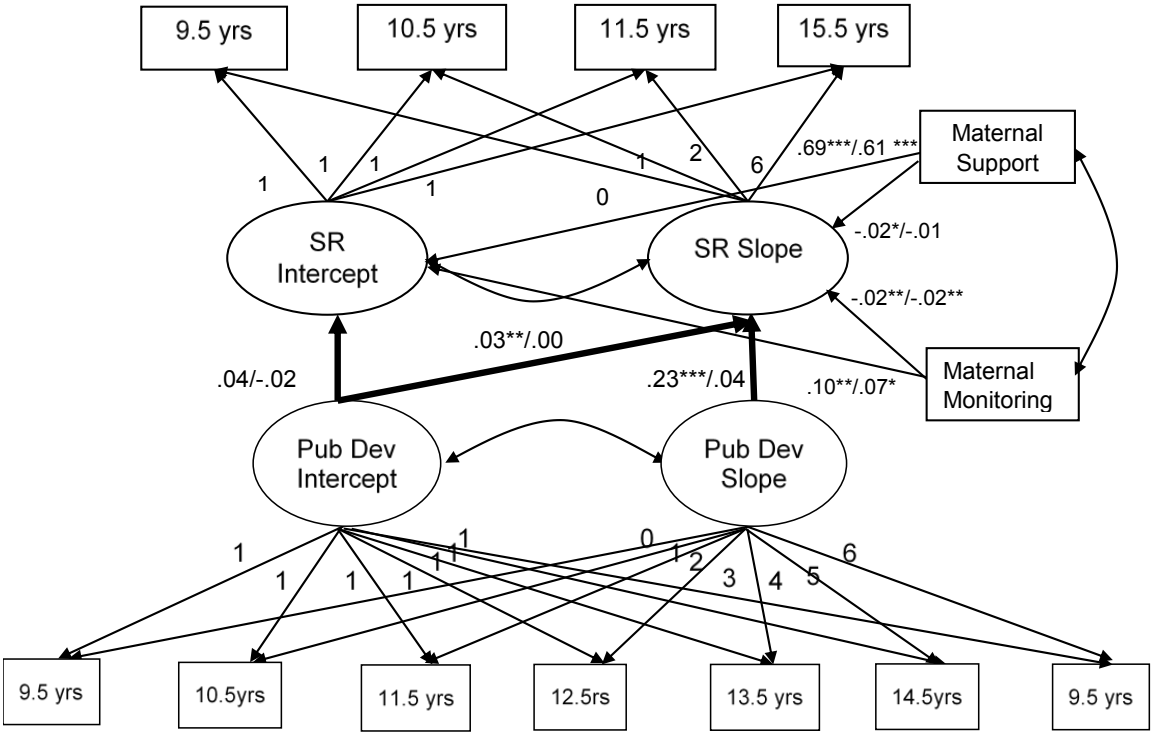
The mean of the intercept for male pubertal development was not significant but the slope was (μ_s = .02, p = <.05). The intercept of paternal rated SR was also significant for males in this model (μ_i = -.13, p = <.001). These findings indicate that there was slight positive growth in pubertal development over time for males but there was no growth in paternal rated SR for males. The correlations between the intercept and slope for puberty and SR were significant (r = -.46 and r = .49, p = <.001; respectively). These correlations indicate that higher levels of puberty were associated with lower slope scores or less growth over time and that for paternal rated SR higher scores of initial status were associated with higher slope scores or more positive growth over the 6 year period.

For the paths of interest between the two LGMs, all paths remained significant when paternal support and maternal monitoring were included that had been previously

found in Hypothesis 2, with some slight decreases in the strength of the relationships. Specifically, the male pubertal development slope had a significant effect on the slope of paternal rated SR for males (b = .23, p = <.001). This finding indicates that even when the effects for support and monitoring are included in the model, the changes in male pubertal development over the 6 year period were associated with developmental changes in father-rated SR among males. The intercept of male pubertal development was significantly associated with the slope of paternal rated SR (b = .03, p = <.05), indicating that scores from the initial assessment of puberty were modestly associated with developmental changes in SR over the 6 year period. Paternal support maintained a large and significant effect on the SR intercept (b = .69, p = <.001); however, the significant effect on the slope term was no longer significant, but only a trend (b = -.02, p = .06). Additionally, maternal monitoring continued to have an effect on the paternal rated SR intercept (b = .10, p = <.05), but was also slightly smaller than found in the model tested part of Hypothesis 4. No significant effect was found between maternal monitoring and the slope of paternal rated SR. The correlation between paternal support and maternal monitoring was significant (r = .21, p = <.001). This suggested that higher scores of paternal support were associated with higher scores of maternal monitoring. The current model was non-additive as pubertal development, only, remained to have a significant effect on the slope of SR.

Figure 6

Hypothesis 5, Conditional Parallel Process Latent Growth Model for Additive Model for Paternal Ratings



Note. Model was run separately for males and females. Male statistics are on the left/Female on the right. * = trend, ** = p <.05, *** = p = <.001, no * = non-significant.

Paternal-rated model (females). Model fit was acceptable in the paternal-female final model (χ^2 = 198.61, df = 72, p = 0, χ^2/df = 2.76, CFI = .94, RMSEA = .05). There were significant intercept and slope variances in both pubertal development and paternal rated SR for females. Specifically, the variance of the intercept for pubertal development was large and significant (D_i = .86, p = <.001), while the variance of the slope was also significant (D_s = .13, p = <.001). Additionally, the variance of the intercept for paternal rated SR was also significant (D_i = .63, p = <.001) as was the slope (D_s = .13, p = <.001). These results suggest a large amount of variance in individual scores for females in

pubertal development and paternal rated SR at initial status and in scores among female adolescents in puberty and paternal SR over time.

The mean of the intercept of pubertal development was not significant but the slope was (μ_s = .02, p = <.05). Neither the intercept of paternal rated SR nor the slope had was significant in the current model. These findings indicate that there was slight positive growth in female pubertal development over time, but there was no growth in paternal rated SR for females. The correlations between the intercept and slope for female puberty and paternal rated SR were significant (r = -.43 and r = -.44, p = <.001; respectively). These correlations indicated that higher scores at initial status in female puberty and paternal rated SR were associated with lower slope scores over the 6 year period. In comparison with findings from model tests as part of Hypothesis 2, the paths between puberty at initial status and the path from paternal rated SR at initial status has gone from a statistical trend to non-significant once all variables were entered into the model. Thus, none of the paths of interest between the two LGMs were significant in the model based on paternal ratings of female youth. The relationship between paternal support and paternal rated SR at the intercept remained significant (b = .61, p = <.001). This indicated that higher scores of paternal support were significantly associated with initial assessments of SR when rated by fathers. However, the previous significant relationship between maternal monitoring and paternal rated SR intercept became a statistical trend rather than significant (b = .07, p = .08) once all variables were added to the model. The relationship between maternal monitoring and paternal rated SR at the slope remained significant (b = -.02, p = <.05), suggesting there was a slight negative effect (less growth or change) on maternal monitoring and female adolescent development of SR when rated

by fathers. Therefore, this is a non-additive model. The correlation between paternal support and maternal monitoring for females was significant ($r = .16$, $p = <.05$), indicating that higher scores of paternal support were associated with higher reports of maternal monitoring.

Discussion

Neuroscience-SR connection

The integration of neurosciences and developmental psychology, and in particular related to adolescent development, has great potential for advances in understanding teens and their behaviors. This has already contributed, significantly, to the study of adolescent brain maturation (Giedd, 2004; Giedd, 2008; Giedd, 2015; Giedd et al., 1999; Lenroot & Giedd, 2006; Luna & Sweeney, 2004; Sowell, Thompson, Homs, Jernigan, & Toga, 1999; Spessot et al., 2004; Whitford et al., 2007). One of the most pivotal findings includes that adolescents' brains continue to mature and develop, particularly in the frontal lobe area where SR is housed, until the early to mid-twenties. In turn, this has provided unique research opportunities on how adolescents think, react, and make decisions. As such, research focusing on adolescent deviance and risk taking behaviors links developmental and criminological theories and explanatory frameworks to neurosciences, which provides a better understanding of why teens make certain choices and how they ascertain their rewards in doing so. In a recent article, Giedd (2015) suggested that

> Understanding that the adolescent brain is unique and rapidly changing can help parents, society and teens themselves to better manage the risks and grasp the opportunities of the teenage years. Knowing that prefrontal executive functions are still under construction, for example, may help parents to not overreact when their daughter suddenly dyes her hair orange and instead take solace in the notion that there is hope for better judgment in the future. Plasticity also suggests that constructive dialogue between parents and teens about issues such as freedoms and responsibilities can influence development. (p. 39)

Steinberg (2014) has further added to our understanding of how SR in adolescence is linked to the maturation of the brain during the teenage years in a pursuit to establish whether or not and when teens are able to fully rationalize serious situations

and consequences in order to inform policies that affect teens. Specifically, Steinberg (2014) provides cases of incarcerated men in their 30's and 40's that were found guilty of crimes committed in their teens. He proposes that life sentences for juveniles may not be appropriate as the teen brain is not fully mature and, thus, less able to utilize self-control during the events that led to the crimes. Additionally, he suggests that adolescence is a developmental period that has become increasingly longer due to earlier pubertal development and the later attainment of careers, marriage, and children. Therefore, he suggests that society needs to support teens by promoting the development of better SR; he suggests that SR is malleable, largely due to the plasticity of the brain regions that house SR. In turn, this would not only increase the well-being of the adolescent but society at large.

Is SR a stable trait or malleable?

Steinberg proposed that SR is changeable or modifiable during adolescence; thus, it is important to contextualize how SR is defined and to weigh whether it is a stable trait or a malleable construct. Researchers have had a long history of grappling with whether SR is a trait that we are born with and is, therefore, a stable construct (e.g., one child seems to be able to regulate him/herself better than another child from an early age), whether SR is a behavioral construct that is fostered and nurtured over time by parents and caregivers and can change over time, or both. If one takes the position that SR is a trait (like personality, for example), then we would expect few developmental changes (or growth) in SR over time, whether during childhood or adolescence. On the other hand, if SR is considered a malleable behavioral construct (like cooperation with others, for example), then changes in SR over time (childhood and possibly adolescence) would

be found, as a result of socialization processes and pressures. Interestingly, Eisenberg et al. (2004) explain that while SR/effortful control is seen as a temperamental construct, (e.g., trait) they do find that, it is influenced by parenting and socialization experiences, which contribute to increased growth, or development in SR during childhood as it becomes nurtured by parents. \In fact, based on dozens of studies over the course of two decades, Eisenberg and Sulik (2012) prose that there exist developmental changes in SR during childhood and adolescence, with small amounts of growth in effortful control occurring during adulthood. They identify SR is an important construct in understanding and predicting social competence and poor adjustment.

In addition to understanding whether SR is a trait or malleable construct, it is important to consider the definition of SR used in the current study. They further explain that it is necessary to understand and research the processes in normative development of SR as well as individual differences.

The current study utilized Moilanen's definition of SR in adolescence, namely "the ability to flexibly activate, monitor, inhibit, persevere and/or adapt one's behavior, attention, emotions and cognitive strategies in response to direction from internal cues, environmental stimuli and feedback from others, in an attempt to attain personally-relevant goals" was used in the current study (Moilanen, 2007, p. 835) and is, therefore, assessing SR from a behavioral standpoint, one that predicted changes or growth.

While very few studies have examined whether changes do occur in SR over the adolescent developmental period, numerous studies, mostly focused on children or early adolescents (e.g., Eisenberg et al., 2004; Meldrum, Young, & Weerman, 2012; Moilanen, Rasmussen, & Padilla-Walker, 2014; Raffaelli, Crockett, and Shen, 2005) provide

empirical evidence to support the idea that SR changes over time. Importantly, Piquero, Jennings, and Farrington (2009) conducted a meta-analysis of 34 studies which examined whether programmatic interventions focused on changing self-control in children and adolescents were successful. The studies included in the meta-analysis included parent ratings, teacher ratings, direct observation, and self-reports of self-control. In fact, as in the current study, some of this work assessed self-control with the SSRS self-control subscale. Findings provided strong evidence that self-control was malleable and that it could be improved during childhood and adolescence. Further still, the increases in self-control in the studies were associated with decreases in delinquency. Therefore, the authors recommend that intervention programs focused on children and adolescent well-being include self-control components as self-control/SR is able to increase, or develop, over time for children and adolescents.

Similarly, Moffitt et al. (2011, 2013) have suggested that SR is malleable as they explain that understanding the development of self-control and providing interventions to aid and increase the development of it would greatly benefit society. Specifically, Moffitt and colleagues have followed 1,000 participants over 40 years on various health and psychological factors as part of The Dunedin Multidisciplinary Heath and Development Study (Moffitt et al., 2011; 2013). They have found that self-control strongly predicted adult health, wealth, and happiness regardless of SES, race, and intelligence. They call for programs and interventions that focus on increasing self-control in all populations as a way to reduce costs to society (e.g., crime, healthcare, job loss) and to increase the health, wealth, and happiness of people in society. Based on their analysis of their data, they conclude that changes in self-control through intervention and prevention programs

aimed at improving it, would be most effective and beneficial during early childhood and childhood rather than adolescence. However, importantly, they maintain the importance of also intervening during adolescence to improve levels of self-control which in turn would support better decision making. They also note that the development of self-control appears to be related to both biology/genes and environmental effects.

While acknowledging the importance of both biological and environmental influences on SR, Vazsonyi, Roberts, and Huang (2015) discuss research on the development of self-control by presenting studies from seemingly opposite views on nature versus nurture and how these influence SR. While providing evidence for both socialization and biological influences, and suggesting that SR is clearly influenced by both, they advocate focusing on socialization or malleable influences in the development of SR as biological substrates cannot be readily changed. Related to this, Bridgett, Burt, Edwards, and Deater-Deckard (2015) recently provided a review of the intergenerational transmission of SR through various predictors important in the development of SR in children and adolescents, namely the environmental context, neurophysiology, parenting behaviors, or parents' SR levels, and so forth. They note

> 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. (p. 628)

The current study specifically tested the assertion that brain maturation (operationalized by puberty as a proxy) predicted changes in, or the development of, SR during adolescence. By utilizing LGM techniques, it was possible to assess whether there was any growth in SR between the ages of 9.5 and 15.5, whether changes in pubertal

development (tested separately for males and females) predicted developmental changes in SR during adolescence, and whether parenting processes predicted these changes over the 6-year period. Based on the findings from numerous studies, as mentioned above, SR was viewed a behavioral and emotional construct that is malleable and, therefore, has the potential to develop or increase over time for adolescents, as guided by research.

Hypothesis 1

It was expected that growth in SR over the 6-year period would be found and that this growth would be predicted by pubertal development, a proxy for maturation and associated developmental changes in the prefrontal cortex; however, findings did not support this expectation. Rather, findings indicated (Hypothesis 1) that there were no developmental changes in SR as rated by mothers or fathers, and as separately tested among male or female youth between the ages of 9.5 and 15.5 years. This result was unexpected based on neuroscience research showing frontal lobe maturation in adolescence, Steinberg's conceptual work, and previous empirical work on increases in SR during childhood or early adolescence.

Specifically, adolescence is the developmental period that continued growth and maturation of the frontal lobe takes place which houses impulse control, decision-making, and SR. Based on this knowledge, Steinberg and others have suggested that SR should or does increase during the adolescent developmental period. However, conversely to the idea that SR should increase do to these maturational changes in the brain, the current study provides support that SR is stable during adolescence, with few changes, as most changes might have taken place earlier during the first decade of life, during childhood. While this finding stands in opposition to the current study's

conceptual framework, findings are, in fact, consistent with theoretical predictions made by self-control theory (Gottfredson & Hirschi, 1990). The theory predicts that self-control develops during childhood through socialization (e.g., parenting) processes and experiences, and that it fairly stable by the time a child reaches 10 years in age. The current study's findings seem more consistent with this theory's idea of SR being a stable construct, set by 10 as the current study's initial assessment age was 9.5, rather than developmental due to continued brain maturation in adolescence.

Hypothesis 2

Despite the fact that findings did not support that SR develops during adolescence, a number of interesting findings emerged. First, as part of Hypothesis 2, which examined the effects of puberty on SR development among males, the slope term of puberty was associated with the slope term of SR, both when rated by mothers and by fathers. This finding indicated that higher rates of change in pubertal development were associated with greater rates of change in SR over time. However, because the SR slope was not significant, it is difficult to interpret what this association reflects. Perhaps, a different measure of SR, specifically one that uses observational methods or adolescent self-reports rather than parental reports, would provide further insights.

Hypotheses 3 and 4

Additionally, the current study tested whether positive parenting processes (maternal and paternal support as in Hypothesis 3 and maternal monitoring as in Hypothesis 4) had a significant influence on developmental changes in SR during the adolescent years. Eisenberg (2012) suggests that while SR is, in part, temperamental it does continue to develop and is associated with positive parenting processes. Such

processes, as she explains, may be a contributor to individual differences in SR. Specifically, children that are raised with supportive/warm parents develop higher levels of SR than those that who are not. For example, research on development of SR in childhood has provided strong evidence on the link between positive parenting strategies and SR. For example, Vazsonyi and Li (2010) found that when entering parenting as a predictor into their LGM on SR from 4.5 years to 10.5 years there was an approximate 10% reduction in the unexplained variance of SR at initial status (4.5 years), however, parenting was not a significant predictor of the slope. This suggests that positive parenting is an important variable in explaining SR before the age 4.5 years. While the current study did find strong and significant associations between maternal/paternal support and maternal/paternal rated SR for both males and females on the initial status (9.5 years) of SR, only one significant association was found with the SR slope, namely between paternal support and the SR slope among male youth. However, the size of this significant association was very small (b = -.02, p = <.05) and is negative which suggests higher levels of paternal support were related to lower rates of growth in SR for adolescent males. Similarly, there was a significant association between maternal monitoring scores and initial status of maternal/paternal rated SR for both males and females, but only very small associations and non-significant associations on SR slope. It is important to note the large magnitude of the effects of parenting on SR at initial status (significant maternal parenting ratings b_{range} = .23 to .62; significant paternal parenting ratings b_{range} = .17 to .71). By finding such large effects, it reminds researchers of the importance of positive parenting processes during childhood that appear to be a key factor in childhood SR as proposed by Gottfredson and Hirschi (1990) and empirically

substantiated since then (e.g., Colman et al., 2006; Eisenberg et al., 2003; Karreman et al., 2006; Keller et al., 2004; Raikes et al., 2007; and Vazsonyi & Huang, 2010).

Although strong and significant effects of parental support and monitoring were found on the initial status of SR, which suggests that more positive parenting processes were associated with higher initial levels of SR at age 9.5, this effect is not maintained developmentally over time. This finding is, in fact, consistent with recent work by Moilanen, Rasmussen, and Padilla-Walker (2014) who found authoritative parenting assessed at wave 3 (when teens were 11-16 years of age) did not predict SR a year later. The authors suggest that perhaps children that are reared by authoritative parents have become successful at regulating themselves, and thus need less parental support during their teenage years. Another possible explanation for this finding could be that during the adolescent developmental period teens are becoming more independent and more engaged with peers, at the expense of parent-adolescent relationships (Farley & Kim-Spoon, 2014; Giedd, 2015). If so, the impact of parenting on the development of SR might be greater during early and middle childhood rather than during adolescence when teens spend less time with parents, when teens make more decisions on their own, and when teens spend most of their time with same-age peers (e.g., Meldrum & Hay, 2012).

Furthermore, Crosswhite and Kerpelman (2012) found that communication and autonomy support from parents was predictive of SR in children before the age of 10 and that overall effective parenting was significant before and after age 10 in a longitudinal examination of parenting and self-control in children ages 8-9 years and 12-13 years. However, there were no individual parenting characteristics that were found to significantly predict SR after age 10. The authors suggest that parents have a significant

impact on the development of their children's self-control by providing a positive parenting environment where the child will develop higher levels of self-control. These studies, as well as the findings from the current study that parenting has a significant association with SR intercept, provide evidence that parenting processes are important predictors or significantly associated with levels of SR, or changes in SR, during childhood. However, it seems that the effect of this association is absent when investigating whether parenting processes affect changes in SR during adolescence.

Hypothesis 5

By utilizing a conditional parallel process LGM, it was possible to attempt to assess whether the predictor variables (i.e., puberty and parenting) contributed uniquely or additively to developmental changes in SR during adolescence. Findings from the current study show that there was no growth or development in SR between ages 9.5 and 15.5 years, and that pubertal status did not predict the slope of SR during this time; this supports theoretical predictions by Goffredson and Hirschi's (1990) as noted, but also contradicts some recent research which attempts to connect prefrontal cortex maturation to SR during adolescence. However, it is important to note that significant associations between slope to slope paths between pubertal development and SR for males was maintained. This finding does provide evidence that there is some covariation between changes in pubertal development and changes in SR even when parenting is added to the model, despite a non-significant SR slope during adolescence. Furthermore, pubertal development covaried with the slope of SR to a greater extent than parenting, providing evidence that puberty appears to be more important for SR during adolescence than parenting. In addition to the study's findings of puberty on SR, results indicated that

parenting (i.e., support and monitoring) had minimal influences on the development of SR during adolescence; this was found for both male and female youth, but also as rated by either mothers or fathers.

Study Limitations

A number of study limitations require some mention, which contexualize current findings or a lack thereof. These include a non-generalizable sample and measurement and methodology limitations. First, the current study used data from a rather homogenous sample (a mostly European American, middle class) that is not nationally representative. Therefore, future research should assess whether SR changes during adolescence in other income and ethnic/racial groups compare the findings to the current ones. Second, the current study relied on parent-rated survey data for the main study variables. Surveys are extremely useful in such a large data collection process and when accessing information on adolescence; however, future research should include alternative methods (e.g., observational) to capture SR in process. Furthermore, the use of parent ratings was a limitation of the study. While using parent ratings for adolescent behaviors has been utilized in past research, it is an important limitation to mention. When parents rate their adolescent's behaviors, they in fact rate their perceptions of their teen's behaviors. Additionally, parents' perceptions are also influenced by the quality of the dyadic relationship. For instance, if the adolescent is displaying negative behaviors towards their parent, the parent may rate their child lower on SR. his might also contribute to the current findings of the current study. Perhaps one reason for this is that once children transition into adolescence, parents are not physically present as much anymore. Therefore, they may not observe their teen trying to self-regulate, independently, unlike

when their child was younger and required more support from them, including to regulate themselves. Future research should include a multi-method and multi-rater approach to provide a more thorough examination of the development of SR in adolescence and whether puberty and parenting are associated with the development.

Third, missing assessments between the ages of 12.5 to 14.5 years for parenting and SR constructs limited the analyses where more variation may have been found in these constructs over time. Specifically, SR only included assessments at 9.5, 10.5, 11.5 and 15.5. To fully test whether SR changes during the time when adolescents are going through the pubertal process, it would be important to have data at least annually or more frequently between the ages of 12.5 to 14.5. Parental support was also missing at ages 12.5, 13.5, and 14.5 years, both for maternal and paternal rated measures. Furthermore, paternal monitoring was not used at all and maternal monitoring was missing at ages 9.5, 12.5, 13.5, and 14.5 years of age. While slope parameters were specified in a manner that that manage this gap (0, 1, 2, and 6), the inclusion of more data points would have increased the possibility of finding significant developmental changes in SR over time as well as significant associations between parenting and SR development.

Fourth, SR was measured using the SSRS self-control subscale (Grisham & Elliot, 1990). The SSRS self-control subscale measures behavioral and emotional regulation and has been used to test the role of SR and the development of SR in children (e.g., Birmingham, 2013; Drake, Belsky, & Fearon, 2014; Vazsonyi & Li, 2010). Furthermore, it provided good reliabilities (α = .81-.83 for maternal ratings, α = .83-.85 for paternal ratings) in the current study. However, one notable limitation is that the response scale does not provide much variation as it utilizes a 3-point Likert type scale. If

the measure included 2-4 additional response categories, more variation in responses might have been found. In turn, this may have increased the ability to detect differences in SR over time. Raffaelli, Crockett, and Shen (2005) measured SR by using a 3-point response scale and found that SR changed/increased between 4/5 years of age and 8/9 years of age in a sample from the National Longitudinal Survey of Youth (NLSY); however, much like in the current work, SR did not change or increase at age 12/13 years. Again, this means that developmental changes over time in SR can be found, but were not found during the adolescent developmental period. Other assessments that measure similar or related SR constructs were available in the data set, but either did not measure SR as conceptualized in the current study (emotional/behavioral) or were not available at the assessment periods needed to conduct developmental tests (e.g., Tower of London-measures planning/executive functioning, only available in 9th grade; Tower of Hanoi-measures planning/executive functioning, only available in early school years; Emotion Regulation form-measures emotion regulation, only available at 4th, 5th, and 6th grades; Children's Behavior Questionnaire-measures temperament, only available once in childhood; Self-control Ratings-measures self-control-only available one time during childhood; and Delay of Gratification-measures self-regulation, only available once in childhood).

In addition to these measurement limitations, the current study was limited by in the sense that it used puberty as a proxy for brain maturation rather than having included functional MRI scans of the brain and use of hormonal levels during the pubertal process. On the other hand, with brain scans, it is unlikely that we could have studied this question in such a large and sample from across the United States due to the costs and issues of

attrition associated with such a study. Of course MRI scans combined with hormone level samples, SR tasks, observations, and surveys to simultaneously track increases in brain development with increases (or lack thereof) of SR during adolescence would be highly valuable research the could further provide insights into understanding these possible connections. According to Giedd (2015)

> The infrastructure for adolescent research is not well developed, funding for this work is meager and few neuroscientists specialize in this age group. The good news is that as researchers clarify the mechanisms and influences of adolescent brain developments, more resources and scientists are being drawn into the field, eager to minimize risks for teenagers and harness the incredible plasticity of the teen brain. (p. 39)

Finally, an important point to mention regarding the methods of the current study is that a broader sample of adolescents followed through late adolescence (e.g., 16-19) would have been beneficial for testing the main research questions in this study. Recent research (Giedd, 2015) has provided evidence that the prefrontal cortex continues to develop well into the 20's, but puberty occurs at a much earlier age. While the current sample included children from pre-adolescence and early adolescence (i.e., 9.5-12.5 years of age), it did not include middle to late adolescents, to support more thoroughly testing the influence of maturational changes on potential developmental changes in SR into the early 20s.

Conclusion

In conclusion, the current study hypothesized developmental changes in SR between the ages of 9.5 and 15.5 years; it also tested how and whether biological maturation as well as socialization effects contributed to such developmental changes in SR. Findings based on LGMs indicate that SR does not change much during early and middle adolescence, and thus, seems to be largely stable between 9.5 and 15.5 years. This was true for both male and female youth, and as rated both by mothers and by fathers on

measures of SR. In addition, although parenting (i.e., support and monitoring) appears highly salient for the developmental period preceding 9.5 years in age, it seems less important for developmental changes in SR over time subsequently. Some evidence was found which suggests that changes related to male pubertal development covary with changes in SR; however, it remains unclear the extent to which this relationship exists in light of mostly non-significant SR slopes during the age period tested.

The current study is important in the debate of whether SR is still developing in adolescence when many brain developments are occurring (Steinberg, 2008; Steinberg, 2014) or whether it is stable during as suggested by Gottfredson and Hirschi (1990). Implications of such studies as these may influence intervention programs and policies on teens. It is important to note that simply because the current findings did not support changes in SR during adolescence, this does not mean that intervention strategies in adolescence, aimed at increasing SR capacity, may not be useful. In fact, specific skills that are found in teens with higher levels of SR (e.g., thinking before reacting, goal setting, etc.) should be taught and encouraged in adolescents with lower levels of SR. As previously mentioned, Piquero, Jennings, and Farrington (2009) found that levels of SR in adolescents did change through programmatic interventions aimed at increasing self-control to deter teens from deviant behaviors. Subsequently, these SR increases were associated with decreased levels of deviance. Therefore, intervention strategies aimed at increasing SR during adolescence may be an important approach at reducing risk-taking behaviors, health compromising behaviors, and delinquency in teens.

References

Arbuckle, J. (2005). *AMOS 6.0 user's guide*. Chicago: SmallWaters.

Barnes, G. M., Alan, R. S., Farrell, M. P. (2000). The effects of parenting on the development of adolescent alcohol misuse: A six-wave latent growth model. *Journal of Marriage and the Family, 62,* 175-186.

Baumeister, R. F., Leith, K. P., Maraven, M. & Bratslavsky, E. (1998). Self-regulation as a key to success in life. In D. Pushkar, W. Bukowski, A. E. Schwartzman, D. M. Stack, D. R. White (Eds.), *Improving competence across the lifespan* (pp. 117-133). New York: Plenum Press.

Bentler, P.M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin,* 107, 238-246.

Bentler, P. & Bonnet, D. C. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin,* 88, 588-606.

Bentler, P. M. & Chou, C. (1987). Practical issues in structural modeling. *Sociological Methods and Research, 16*, 78-117.

Birmingham, R. S. (2013). Parenting, attachment and child outcomes: Self-regulation as a developmental mechanism. (Doctoral dissertation). Retrieved from Auburn University Electronic Theses and Dissertations. (2013-07-31T15:56:53Z)

Bornstein, M. H. & Bradley, R. H. (2003). Socioeconomic status, parenting, and child development. New Jersey: Lawrence Erlbaum Associates.

Bridgett, D. J., Burt, N. M., Edwards, E. S., & Deater-Deckard (2015). Intergenerational transmission of self-regulation: A multidisciplinary review and integrative conceptual framework. *Psychological Bulletin, 141*, 602-654.

Brody, G. H. & Ge, X. (2001). Linking parenting processes and self-regulation to psychological functioning and alcohol use during early adolescence. *Journal of Family Psychology, 15,* 82-94.

Bronson, M. (2000). *Self-Regulation in Early Childhood: Nature and Nurture.* New York, NY: Guilford Press.

Buckner, J. C., Mezzacappa, E., & Beardslee, W. R. (2003). Characteristics of resilient youths living in poverty: The role of self-regulatory processes. *Development and Psychopathology, 15,* 139-162.

Calkins, S. D. & Hill, A. (2007). Caregiver influences on emerging emotion regulation: Biological and environmental transactions in early development. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 229-248). New York: Guilford Press.

Cameron, J. L. (2004a). Interrelationships between hormones, behavior, and affect during adolescence: Understanding hormonal, physical, and brain changes occurring in association with pubertal activation of the reproductive axis. Introduction to Part III. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 110- 123). New York: Blackwell Publishing.

Cameron, J. L. (2004b). Interrelationships between hormones, behavior, and affect during adolescence: Complex relationships exist between reproductive hormones, stress-related hormones, and the activity of neural systems that regulate behavioral affect. Comments on Part III. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 134-142). New York: Blackwell Publishing.

Capaldi, D. M., Stoolmiller, M., Clark, S., & Owen, L. D. (2002). Heterosexual risk behaviors in at-risk young men from early adolescence to young adulthood: Prevalence, prediction, and association with STD contraction. *Developmental Psychology,* 38(3), 394-406.

Carlo, G., Crockett, L. J., Wolff, J.M. & Beal, S. J. (2012). The role of emotional reactivity, self-regulation, and puberty in adolescents' prosocial behaviors. *Social Development, 21*(4), 667-685.

Carter, M., McGee, R., & Taylor, B. (2007). Health outcomes in adolescence: Associations with family, friends, and school engagement. *Journal of Adolescence, 30,* 51-62.

Coleman, R. A., Hardy, S. A., Albert, M., Raffaelli, M., & Crockett, L. (2006). Early predictors of self-regulation in middle childhood. *Infant and Child Development, 15,* 421-437.

Cotter, R. B. & Burke, J. D. (2002). Innovative retention methods in longitudinal research: A case study of the developmental trends study. *Journal of Child and Family Studies, 11,* 485-498.

Crosswhite, J. M. & Kerpelman, J. L. (2012). Parenting and children's self control: Concurrent and longitudinal relations. *Deviant Behavior, 33,* 715-737.

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 1-22). New York: Blackwell Publishing.

Dahl, R. E. & Hariri, A. R. (2005). Lessons from G. Stanley Hall: Connecting new research in biological sciences to the study of adolescent development. *Journal of Research on Adolescence, 15*, 367-382.

de Kemp, R. A. T., Overbeek, G., & de Wied, M. (2007). Early adolescent empathy, parental support, and antisocial behavior. *Journal of Genetic Psychology, 168,* 5-18.

De Leonibus, C, Marcovecchio, M. L., Chiavaroli, V., de Giorgis, T., & Chiarelli, F. (2014). Timing of puberty and physical growth in obese children: A longitudinal study in boys and girls. *Pediatric Obesity, 9,* 292-299.

Dennis, T. (2006). Emotion self-regulation in preschoolers: The interplay of child approach reactivity, parenting, and control capabilities. *Developmental Psychology, 42,* 84-97.

Dick, D. M., Rose, R. J., Pulkkinen, L., & Kaprio, J. (2001). Measuring puberty and understanding its impact: A longitudinal study of adolescent twins. *Journal of Youth and Adolescence, 30,* 385-400.

Dishion, T. J. & Stomshak, E. A. (2007). Interventions with children and adolescents. In T. J. Dishion & E. A. Stomshak (Eds.), *intervening in children's lives: An ecological, family-centered approach to mental health care* (pp. 141-161). Washington, DC: American Psychological Association.

Drake, K., Belsky, J., & Fearon, R. M. P. (2014). From early attachment to engagement with learning in school: The role of self-regulation and persistence. *Developmental Psychology, 50*, 1350-1361.

Duncan, S. C., Duncan, T. E., & Hops, H. (1996). Analysis of longitudinal data within accelerated longitudinal designs. *Psychological Methods, 1,* 236-248.

Eisenberg, N. (2012). Temperamental effortful control (Self-regulation). Retrieved October 29, 2015 from the Encyclopedia on early childhood development Web site: http://www.child-encyclopedia.com/temperament/according-experts/temperamental-effortful-control-self-regulation.

Eisenberg, N., Smith, C. L., Sadovsky, A., & Spinrad, T. L. (2004). Effortful control: Relations with emotion regulation, adjustment, and socialization in childhood. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications (*pp. 259-282). New York: Guilford Press.

Eisenberg, N. & Sulik, M. J. (2012). Emotion-related self-regulation in children. *Teaching of Psychology, 39,* 77-83.

Eisenberg, N., Valiente, C., Morris, A. S., Fabes, R. A., Cumberland, A., Reiser, M., Gershoff, E. T., Shepard, S. A., & Losoya, S. (2003). Longitudinal relations among parental emotional expressivity, children's regulation, and quality of socioemotional functioning. *Developmental Psychology, 39*, 3-19.

Ellis, L. K., Rothbart, M. K., & Posner, M. I. (2004). Individual differences in executive attention predict self-regulation and adolescent psychosocial behaviors. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 337-340). New York: Blackwell Publishing.

Farley, J. P. & Kim-Spoon, J. (2014). The development of adolescent self-regulation: Reviewing the role of parent, peer, friend, and romtic relationships. *Journal of Adolescence, 37,* 433-430.

Feldman, R.L., Masalha, S., & Alony, D. (2006). Microregulatory patterns of family interactions: Cultural pathways to toddlers' self-regulation. *Journal of Family Psychology, 20,* 614- 623.

Finkenauer, C., Engels, R. C., & Baumeister, R. F. (2005). Parenting behaviour and adolescent behavioural and emotional problems: The role of self-control. *International Journal of Behavioral Development, 29,* 58-69.

French, L.A. & Picthall-French, N. (1998). The role of substance abuse among rural youth by race, culture, and gender. *Alcoholism Treatment Quarterly, 16*(3), 101-108.

Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 77-85). New York: Blackwell Publishing.

Giedd, J. N. (2008). The teen brain: Insights from neuroimaging. *Journal of Adolescent Health, 42,* 335-343.

Giedd, J. N. (2015). The amazing teen brain. *Scientific American, 312,* 32-37.

Giedd, J. N., Blumental, J. Jeffries, N. O., Castellanos, F. X., Liu, H. Zijdendbos, A. et al. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2,* 861-863.

Giedd, J. N., Clasen, L. S., Lenroot, R., Greenstein, D., Wallace, G. L., Ordaz, S., et al. (2006). Puberty-related influences on brain development. *Molecular and Cellular Endocrinology, 254,* 154-162.

Graham, J.W. (2003). Adding missing-data-relevant variables to FIML-based structural equation models. *Structural Equation Modeling, 10*, 80-100.

Granello, D. H. & Wheaton, J. E. (2004). Online data collection: Strategies for research. *Journal of Counseling and Development, 82,* 387-393.

Gray, M. R. & Steinberg, L. (1999). Unpacking authoritative parenting: Reassessing a multidimensional construct. *Journal of Marriage and the Family, 61,* 574-587.

Grasham, F. M., & Elliott, S. N. (1990). *The social skill rating system.* Circle pines, MN: American Guidance Service.

Grolnick, W. S. & Farkas, M. (2002). Parenting and the development of children's self-regulation. In M. H. Bornstein (Ed.), *Handbook of parenting: Vol. 5: Practical issues in parenting.* New Jersey: Lawrence Erlbaum Associates. (Vol 5, pp. 89-110).

Ho, C., Bluestein, D. N., & Jenkins, J. M. (2008). Cultural differences in the relationship between parenting and children's behavior. *Developmental Psychology, 44,* 507-522.

Jones, S., Cauffman, E., & Piquero, A. R. (2007). The influence of parental support among incarcerated adolescent offenders: The moderating effects of self-control. *Criminal Justice and Behavior, 34,* 229-245.

Karreman, A., van Tuijl, C., van Aken, M. A. G. (2006). Parenting and self-regulation in preschoolers: A meta-analysis. *Infant and Child Development, 15,* 561-579.

Keller, H., Yovsi, R., Borke, J., Kartner, J., Jensen, H., & Papaligoura, Z. (2004). Developmental consequences of early parenting experiences: Self-recognition and self-regulation in three cultural communities. *Child Development, 75,* 1745-1760.

Kelley, A. E., Schochet, T., & Landry, C. F. (2004). Risk taking and novelty seeking in adolescence. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 27-32). New York: Blackwell Publishing.

Kerr, M. & Stattin, H. (2000). What parents know, how they know it, and several forms of adolescent adjustment. *Developmental Psychology, 36,* 366-380.

Kim, S. & Brody, G. H. (2005). Longitudinal pathways to psychological adjustment among black youth living in single-parent households. *Journal of Family Psychology, 19*, 305- 313.

Kim, S., Brody, G. H., & Murry, V. M. (2003). Longitudinal links between contextual risks, parenting, and youth outcomes in rural African American families. *Journal of Black Psychology, 29,* 359-377.

Kochanska, G., Murray, K. T., Harlan, E. T. (2000). Effortful control in early childhood: Continuity and change, antecedents, and implications for social development. *Developmental Psychology, 36,* 220-232.

Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology, 18*, 199-214.

Lempers, J. D., Clark-Lempers, D., & Simons, R. L. (1989). Economic hardship, parenting, and distress in adolescence. *Child Development, 60,* 25-39.

Luna, B. & Sweeney, J. A. (2004). The emergence of collaborative brain function: fMRI studies of the development of response inhibition. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 296-309). New York: Blackwell Publishing.

Martel, M. M., Nigg, J. T., Wong, M. M., Fitzgerald, H. E., Jester, J. M., Puttler, L. I., Glass, J. M., Adams, K. M., & Zucker, R. A. (2007). Childhood and adolescent resiliency, regulation, and executive functioning in relation to adolescent problems and competence in a high- risk sample. *Developmental Psychopathology, 19,* 541-563.

Masten, A. S. (2004). Regulatory processes, risk, and resilience in adolescent development. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 110-123). New York: Blackwell Publishing.

Maton, K. I., Teti, D. M., Cornes, K. M., Vieira-Baker, C. C., Lavine, J. R., Gouze, K. R., & Keating, D. P. (1996). Cultural specificity of support sources, correlates and contexts: Three studies of African-American and Caucasian youth. *American Journal of Community Psychology, 24*(4), 551-587.

McCabe, S. E. (2004). Comparison of web and mail surveys in collecting illicit drug use data: A randomized experiment. *Journal of Drug Education, 34,* 61-72.

Meldrum, R. C. & Hay, C. (2012). Do peers matter in the development of self-control? Evidence from a longitudinal study of youth. *Journal of Youth and Adolescence, 41,* 691-703.

Miyazaki, Y. & Raudenbush, S. W. (2000). Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychological Methods, 5,* 44-63.

Moilanen, K. L. (2007). The Adolescent Self-Regulatory Inventory: The development and validation of a questionnaire of short-term and long-term self-regulation. *Journal of Youth and Adolescence, 36*, 835-848.

Moilanen, K. L., Rasmussen, K. E., Padilla-Walker, L. M. (2014). Bidirectional associations between self-regulation and parenting styles in early adolescence. *Journal of Research on Adolescence,* doi: 10.1111/jora.12125.

Moffitt, T. E., Arseneault, L., Belsky D., Dickson, N., Hancox, R. J., Harrington, H. L., Houts, R., Poulton, R., Roberts, B. W., Ross, S., Sears, M. R., Thomson, W., & Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *PNAS Proceedings to the National Academy of Sciences of the United States of America, 108*, 2693-2698.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in development of emotion regulation. *Social Development, 16,* 361-388.

Murry, V. M. & Brody, G. H. (1999). Self-regulation and self-worth of Black children reared in economically stressed, rural, single mother-headed families: The contribution of risk and protective factors. *Journal of Family Issues, 20*(4), 458-484.

Myrtveit, I., Stensrud, E., & Olsson, U. H. (2001). Analyzing data sets with missing data: An empirical evaluation of imputation methods and likelihood-based methods. IEEE Trans. Software End. 27

National Institutes of Health, NIH (2007). Pubertal development in adolescence. Retrieved September 27, 2007 from the National Institutes of Health Web site: http://www.nichd.nih.gov/health/topics/Puberty.cfm

Pianta, R. C. (1994). Ratings scales for parent-child interaction in preschoolers. University of Virginia.

Peper, J. S. & Dahl, R. E. (2013). The teenage brain: Surging hormones-Brain behavior interactions during puberty. *Current directions in Psychological Science, 22*(2), 134-139.

Petersen, A.C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth & Adolescence, 17*, 117-133.

Pettit, G. S., Keiley, M. K., Laird, R. D., Bates, J. E., & Dodge, K. A. (2007). Predicting the developmental course of mother-reported monitoring across childhood and adolescence from early proactive parenting, child temperament, and parents' worries. *Journal of Family Psychology, 21,* 206-217.

Pinderhughes, E. E., Dodge, K. A., Bates, J. E., Pettit, G. S., & Zelli, A. (2000). Discipline responses: Influences of parents' socioeconomic status, ethnicity, beliefs about parenting, stress, and cognitive-emotional processes. *Journal of Family Psychology, 14*, 380-400.

Piquero, A. R., Jennings, W. G., & Farrington, D. P. (2009). On the malleability of self-control: Theoretical and policy implications regarding a General Theory of Crime, *Justice Quarterly, 27*, 803-834.

Posner, M. I. & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Developmental psychopathology, 12,* 427-441.

Purdie, N., Carroll, A., & Roche, L. (2004). Parenting and adolescent self-regulation. *Journal of Adolescence, 27,* 663-676.

Raffaelli, M., Crockett, L. J. , Shen, Y. (2005). Developmental stability and change in self-regulation from childhood to adolescence. *The Journal of Genetic Psychology, 166,* 54-75.

Raikes, H. A., Robinson, J. L., Bradley, R. H., Raikes, H. H., & Ayoub, C. C. (2007). Developmental trends in self-regulation among low-income toddlers. *Social Development, 16,* 128-149.

Raudenbush, S. W. & Chan, W. (1992). Growth curve analysis in accelerated longitudinal designs. *Journal of Research in Crime and Delinquency, 29,* 387-411.

Roberts, G. C., Block, J. H., Block, J. (1984). Continuity and change in parents' child-rearing practices. *Child Development, 55,* 586-597.

Rothbart, M. K. & Sheese, B. E. (2007). Temperament and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 331-350). New York: Guilford Press.

Rothbart, M. K., Posner, M. I., Kieras, J. (2006). Temperament, attention, and the development of self-regulation. In K. McCarntey & D. Phillips (Eds.), *Blackwell handbook of early childhood development* (pp. 338-357). New York: Blackwell Publishing.

Rothbart, M. K. (1989). Temperament in childhood. In G. A. Kohnstamm, J. E. Bates, M. K. Rothbart (Eds.), *Temperament in childhood* (pp. 59-73). Oxford: John Wiley & Sons.

Ruiz, S. Y., Roosa, M. W., & Gonzales, N. A. (2002). Predictors of self-esteem for Mexican American and European American youths: A reexamination of the influence of parenting. *Journal of Family Psychology, 16,* 70-80.

Saris, W. E. & Satorra, J. D. (1987). Testing structural equation models. In P. Cuttance & R. Ecob (Eds.), *Structural modeling by example: Applications in education, sociological and behavioral research,* (pp. 202-220). New York: Cambridge University Press.

Savin-Williams, R. C. & Ream, G. L. (2005). Pubertal onset and sexual orientation in an adolescent national probability sample. *Archives of Sexual Behavior, 35,* 279-286.

Schaefer, E. S. (1965). Children's reports of parental behavior: An inventory. *Child Development, 36,* 413-424.

Schubert, C. M., Chumlea, W. C., Kulin, H. E., Lee, P. A., Himes, J. H., & Sun, S. S. (2005). Concordant and discordant sexual maturation among U.S. children in relation to body weight and BMI. *Journal of Adolescent Health, 37,* 356-362.

Shaw, D. S., Winslow, E. B. & Flanagan, C. (1999). A prospective study of the effects of marital status and family relations on young children's adjustment among African American and European American families. *Child Development, 70,* 742-755.

Silk, J. S., Shaw, D. S., Forbes, E. E., Lane, T. L., & Kovacs, M. (2006). Maternal depression and child internalizing: The moderating role of child emotion regulation. *Journal of Clinical Child and Adolescent Psychology, 35,* 116-126.

Silk, J.S., Shaw, D. S., Skuban, E. M., Oland, A. A., & Kovacs, M. (2006). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *Journal of Child Psychology, 47,* 69-78.

Silveri, M. M., Rohan, M. L., Pimentel, P. J., Gruber, S. A., Rosso, I. M., Yurgelun-Todd, D. A. (2006). Sex differences in the relationship between white matter microstructure and impulsivity in adolescents. *Magnetic Resonance Imaging, 24,* 833-841.

Sisk, C. L. & Foster, D. L. (2004). The neural basis of puberty and adolescence. *Nature Neuroscience, 7*, 1040-1047.

Shoda, Y., Mischel, W., & Peake, P. K. (1990). Predicting adolescent cognitive and self-regulatory competencies from preschool delay of gratification: Identifying diagnostic conditions. *Developmental Psychology, 26,* 978-986.

Shulman, E. P., Harden, K.P., Chein, J. M., & Steinberg, L. (2014). Sex differences in the developmental trajectories of impulse control and sensation-seeking from early adolescence to early adulthood. *Journal of Youth and Adolescence,* 1-17. doi: 10.1007/s10964-014-0116-9.

Smetana, J. G. & Daddis, C. (2002). Domain-specific antecedents of parental psychological control and monitoring: The role of parenting beliefs and practices. *Child Development, 73,* 563-580.

Stevens, J. (1996). *Applied multivariate statistics for the social sciences* (3rd ed.). Mahwah, NJ: Lawrence Erlbaum Associates.

Soenens, B., Vansteenkiste, M., Luychx, K., & Gossens, L. (2006). Parenting and adolescent problem behavior: An integrated model with adolescent self-disclosure

and perceived parental knowledge as intervening variables. *Developmental Psychology, 42,* 305-318.

Spessot, A. L., Kerstin, J. P., & Peterson, B. S. (2004). Neuroimaging of developmental psychopathologies: The importance of self-regulatory and neuroplastic processes in adolescence. In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 86-104). New York: Blackwell Publishing.

Stattin, H. & Kerr, M. (2000). Parental monitoring: A reinterpretation. *Child Development, 71,* 1072-1085.

Steinberg, L. (2004). Risk taking in adolescence: What changes, and why? In R. Dahl & L. Spear (Eds.). *The Annals of the New York Academy of Sciences, Adolescent Brain Development: Vulnerabilities and Opportunities* (Vol. 1021, pp. 110-123). New York: Blackwel Publishing.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28,* 78-106.

Steinberg, L. (2014). *Age of opportunity: Lessons from the new science of adolescence.* Boston, MA: Houghton Mifflin Harcourt. Tanner, J. M. (1962). Growth at adolescence, 2nd Edition. Oxford: Blackwell.

Taylor, R. D., Casten, R., & Flickinger, S. M. (1993). Influence of kinship social support on the family processes experiences and psychosocial depression of African-American adolescents. *Developmental Psychology, 29(2), 382-388.*

Teleki, J. K., Powell, J. A., & Claypool, P. L. (1984). Parental child-rearing behavior perceived by parents and school-age children in divorced and married families. *Family and Consumer Sciences, 13,* 41-51.

Thompson, R. A. (2001). Development in the first years of life. *The Future of Children, 11,* 21- 33.

Tisak, J. & Meredith, W. (1990). Descriptive and associative developmental models. In A. von Eye (Ed.), *Statistical models in developmental research, Vol II: Time series and categorical and longitudinal data* (pp. 387-406). New York: Academic Press.

Vazsonyi, A. T. & Huang, L. (2010). Where self-control comes from: On the development of self-control and its relationship to deviance over time. *Developmental Psychology, 46*(1), 245-257.

Vazsonyi, A. T., Roberts, J. W., & Huang, L. (2015). Why focusing on nurture made and still makes sense: The biosocial development of self-control. In M. DeLisi & M. G. Vaughn (Eds.), *The Routledge International Handbook of Biosocial Criminology,* (pp. 263-280). New York, NY: Routledge.

Velling, B. L., Blandon, A. Y., Kolak, A. M. (2006). Marriage, parenting, and the emergence of early self-regulation in the family system. *Journal of Child and Family Studies, 15,* 493-506.

Whitford, T. J., Rennie, C. J., Grieve, S. M., Clark, C. R., Gordon, E., & Williams, L. M. (2007). Brain maturation in adolescence: Concurrent changes in neuroanatomy and neurophysiology. *Human Brain Mapping, 28,* 228-237.

Wichstrom, L. (2001). The impact of pubertal timing on adolescent's alcohol use. *Journal of Research on Adolescence, 11,* 131-150.

Wothke, W. (2000). Longitudinal and multigroup modeling with missing data. In T. D. Little, K. U. Scnabel, & J. Baumert (Eds.) *Modeling longitudinal and multilevel data: Practical issues, applied approaches, and specific examples.* New Jersey: Lawrence Erlbaum 219-240, 269-281.

Youngoh, J. (2015). Stability of self-control: Hirschi's redefined self-control. *International Journal of Offender Therapy and Comparative Criminology, 59,* 51-67.

Youngoh, J. & Bouffard, L. (2014). Stability of self-control and gender. *Journal of Criminal Justice, 42,* 356-365.

Appendix 1: Measures

CHILD-PARENT RELATIONSHIP SCALE

Robert C. Pianta

Child:_____ Age:_____

Parent:_____

Please reflect on the degree to which each of the following statements currently applies to your relationship with your child. Using the scale below, circle the appropriate number for each item.

Definitely does not apply 1	Not really 2	Neutral, not sure 3	Applies somewhat 4	Definitely applies 5

1.	I share an affectionate, warm relationship with my child.	1	2	3	4
2.	My child and I always seem to be struggling with each other.	1	2	3	4
3.	If upset, my child will seek comfort from me.	1	2	3	4
4.	My child is uncomfortable with physical affection or touch from me.	1	2	3	4
5.	My child values his/her relationship with me.	1	2	3	4
6.	When I praise my child, he/she beams with pride.	1	2	3	4
7.	My child spontaneously shares information about himself/herself.	1	2	3	4
8.	My child easily becomes angry at me.	1	2	3	4
9.	It is easy to be in tune with what my child is feeling.	1	2	3	4
10.	My child remains angry or is resistant after being disciplined.	1	2	3	4
11.	Dealing with my child drains my energy.	1	2	3	4
12.	When my child is in a bad mood, I know we're in for a long and difficult day.	1	2	3	4
13.	My child's feelings toward me can be unpredictable or can change suddenly.	1	2	3	4
14.	My child is sneaky or manipulative with me.	1	2	3	4
15.	My child openly shares his/her feelings and experiences with me.	1	2	3	4

Pubertal Development Scale

1. Would you say that your growth **in** height **has** not yet begun to spurt, **has** barely started, is definitely underway, or **does** growth **seem** completed? (if asked, **spurt** = more growth than usual)
1**2**3**4** NO Yes (Barely) Yes (Definintely) Development Completed

2. And how about the growth of body hair? **Would** you say that your body hair **has** not yet started growing, **has** barely ***started*** growing, or does growth seem completed? (if asked, body hair = underarm or pubic hair)
NO Yes (Barely) Yes (Definitely) Development Completed

3. Have you noticed any skin changes, especially pimples?
No Yes (Barely) Yes (Definitely)

4. Have you noticed a deepening of your voice? Boys only.
No Yes (Barely) Yes (Definitely) Development Completed

5. Have you begun to grow hair on your face? Boys only.
No Yes (Barely) Yes (Definitely) Development Completed

6. Have your breasts begun to grow? Girls only.
No Yes (Barely) Yes (Definitely) Development Completed

7. Have you begun to menstruate (get your period)? Girls only.
No **Yes** (Barely) **Yes** (Definitely)

8. What was the date of your first period: Month_____ Year_____

If you don't know the date, how old were you?
Age_____

9. How tall are you?
Feet_____ inches_____

10. How much do you weigh? Pounds_____

11. Do you think your development is earlier or later than most other boys/girls your age?
1. much earlier 2.somewhat earlier 3. about the same 4.somewhat later
5. much later

Social Skills Rating System (SSRS) (P)

0	1	2
Never	Sometimes	Very Often

1. Uses free time at home in an acceptable way.
2. Keeps room clean and neat without being reminded.
3. Speaks in an appropriate tone of voice at home.
4. Joins group activities without being told to.
5. Introduces herself or himself to new people without being told.
6. Responds appropriately when hit or pushed by other children.
7. Asks sales clerks for information or assistance.
8. Attends to speakers at meetings such as in church or youth group.
9. Politely refuses unreasonable request from others.
10. Invites others to your home.
11. Congratulates family members on accomplishments.
12. Make friends easily.
13. Shows interest in a variety of things.
14. Avoids situations that are likely to result in trouble.
15. Puts away toys or other household property.
16. Volunteers to help family members with tasks.
17. Receives criticism well.
18. Answers the phone appropriately.
19. Helps you with household tasks without being asked.
20. Appropriately questions household rules that may be unfair.
21. Attempts household tasks before asking for your help.
22. Controls temper when arguing with other children.
23. Is liked by others.
24. Starts conversations rather than waiting for others to talk first.
25. Ends disagreements with you calmly.
26. Controls temper in conflict situations with you.
27. Give compliments to friends or other children in the family.
28. Completes household tasks within a reasonable time.
29. Asks permission before using another family member's property.
30. Is self-confident in social situations such as parties or group outings.
31. Avoids situations that are likely to result in trouble.
32. Responds appropriately to teasing from friends or relatives of his or her own age.
33. Uses time appropriately while waiting for your help with homework or some other task.
34. Accepts friends' ideas for playing.
35. Easily changes from one activity to another.
36. Cooperates with family members without being asked to do so.
37. Acknowledges compliments or praise from friends.
38. Reports accidents to appropriate persons.

Grasham, F. M., & Elliott, S. N. (1990). *The social skill rating system.* Circle pines, MN: American Guidance Service.

Keeping Tabs

KEEPING TABS

The next questions are about how much you know about what your child does.
How much do you know about…

1 = Don't know at all 2 = Know a little bit 3 = Know a lot 4 = Know everything

1. Who your child spends time with?
1 2 3 4
2. How your child spends his/her free time?
1 2 3 4
3. How your child spends his/her money?
1 2 3 4
4. Where your child goes right after school?
1 2 3 4
5. Where your child goes throughout the day on the weekend?
1 2 3 4
6. Problems your child is having at school?
1 2 3 4

1 = Never 2 = Sometimes 3 = Often 4 = Always

7. Do you tell your child what time he/she has to be home on school nights? 1 2 3 4
8. Do you tell your child what time he/she has to be home on weekend nights? 1 2 3 4
9. If your child didn't come home by the set time, would you know? 1 2 3 4
10. If you aren't home and your child leaves the house, does your child leave a note or call to say where he/she is going? 1 2 3 4
11. When you aren't at home, does your child know how to get in touch with you? 1 2 3 4